ACT Legislative Assembly
Standing Committee on Health, Ageing, Community and Social Services
GPO Box 1020
CANBERRA ACT 2601

Inquiry into exposure draft of the Drugs of Dependence (Cannabis Use for Medical Purposes) Amendment Bill 2014 and related discussion paper

I have enclosed for your information and consideration a letter I had sent to the ACT Chief Minister and the Leader of the ACT Opposition, in relation to the proposal to legalise marihuana for 'medical use.' In response, I have received a letter (6.1.2015) from Mr Simon Corbell, MLA in his capacity as Minister for Health suggesting I make my views known to your committee, so I am formally submitting the above letter to your committee. I am making this submission on the basis that its contents are for the Committee only and not available to those outside government. However, I understand from a recent phone call to the secretariat that the Committee may choose to make parts of it available in the public domain and if that were to occur I would not want my name associated with such a release. If you are unable to accede to my request for privacy relating to public release, then I withdraw the submission.

Since the 1980s our society has become blighted by drug abuse, including marihuana. Between the mid-80s and 1993 governments ceased to rely on the expert independent advice on public health matters from the NH&MRC. Before this cessation, public health did not have the potential to become a political football and I have outlined the reasons for this in the accompanying letter.

In 1992 I undertook an NH&MRC Travelling Fellowship which involved researching the history of drug regulation (both licit and illicit) in Australia. The present legislation on illicit drugs is based on the two UN Conventions to which Australia is a signatory. These modern Conventions have their origins in the concerns of the Chinese government in the late 19th century over the harmful effects resulting from the unregulated movement of opium between countries. In the Queensland Hansard – "Sale and Use of Poisons Bill, 26 October 1888, Second Reading,"(pp 1734-1739), it states, inter alia, "In addition, he (*an MP*) said Mr Cheong had quoted a letter from the Prime Minister of China in which he said 'If the opium trade continues for another 50 years there will be no China'".

I feel the same way about Australia today in relation to drug abuse, particularly if we follow the American and Mexican model, where there is a lot of correct emphasis on *supply reduction*, but very little on *demand reduction*. I would suggest some sort of national get-together of governments to face up to the problem without it being politicised. Re-naming it 'mental health' and having TV stations suggesting one rings a helpline if they are feeling depressed is not addressing drug abuse properly. Some years back, when APC powders were causing people to die from renal failure there was a Senate Enquiry I believe was entitled "Australia – a drug affected society". It complemented

two separate State enquiries into the same issue and it resulted in excellent outcomes and the problem went away as a result of the recommendations arising out of these enquiries.

Yours sincerely

[redacted]

23 January 2015

The Chief Minister &
Leader of the Opposition
ACT Legislative Assembly
GPO Box 1020
CANBERRA ACT 2601

I am writing to express views opposing the establishment of the use marihuana for "medical use". I would like to stress that I am not some sort of "wowser" trying to condemn people who use or have used marihuana. I know that not everyone who has experimented with marihuana in their youth has a problem, particularly if the use was limited. I am looking at the issue from a rational scientific aspect in relation to an individual's health and from experience as a 'coal face' health professional. I will also be sending copies of this letter to relevant Ministers and Leaders of the Opposition in other jurisdictions because of the need for a common approach to marihuana.

However, I was disappointed recently to see that the local paper announced on a Tuesday that there was going to be a public forum in Canberra 'on Tuesday' about this issue but no date was given. I assumed it would be the next Tuesday. The next day I picked up the paper only to see that the forum had been conducted the day before and the article was all about Dr Wodak and his opinions. It was obvious that the forum was stage managed to support only one point of view. The concept of medical marihuana was promoted some years back by the drug abuse lobby. I think it was in the mid 1990s. The strategy then was much the same as it is today, manipulation of a particular group of sick people to further the aims of the drug abuse lobby. At that time they used people with Multiple Sclerosis, particularly women, and the media portrayed patients in an advanced stage. This time it is people with a terminal illness. On the first occasion this posed a problem for the regulators and politicians who did not want to be painted as monsters refusing treatment to seriously ill people. The drug abuse lobby and their media allies thought this would be enough to make the government back down, but they were wrong. Marihuana is made up of approximately 60 *active* compounds. Most of them are toxic, but one was found to be a safe sedative/tranquilliser which could be beneficial, and to counter the drug abuse lobby pressure for an unsafe product, the Australian Drug Evaluation Committee (ADEC) approved a standardised cannabinoid derived from marihuana that satisfied regulatory standards and could be prescribed by doctors on approval . This held the drug abuse lobby back for a while but now they are ramping up the pressure again nearly 20 years on because drug abusers now represent a significant number of voters and they are hoping politicians will wilt this time and give in to them. As far as I know marihuana has no analgesic properties, so I wonder what is its therapeutic purpose in terminal illness where pain control is the major issue. Therapeutic drugs are not administered by smoking because of the possibility of cancer as with cigarettes and other tobacco products.

I have also enclosed a clinical review of marihuana and a collection of abstracts from newspaper articles and other documentation outlining the dangers of marihuana use. Whilst I realise the current discussion relates to its use in terminal patients I have considered its general use as well, on the basis that general use is the next step after 'medical' use, as is happening today in the USA. The clinical review was part of a WHO program I was working on in Geneva in 1995, called INTOX. INTOX has been largely wound down because of budget cuts. This program, a sub-program of the International Program on Chemical Safety, conducted international conferences which drew together the world's leading clinical toxicologists who reviewed chemicals to which humans may be exposed (including domestic and agricultural chemicals, licit and illicit drugs, herbal medicine etc) and produced a PIM (Poisons Information Monograph) on each substance. This PIM was designed to contain information to assist doctors treating patients admitted to Emergency Departments with toxic symptoms following exposure to a potential poison. It enabled them to access quickly on the Internet the information necessary to diagnose and treat the patient.

I have nothing against herbs as a useful source of medicines. Digitalis (foxglove) was used for heart conditions in Europe for millennia and it contains cardiac glycosides. However as civilization progressed it was realised that if you used the whole herb you may get inconsistent results. That was because the mix and concentration of glycosides varied from year to year, depending on weather patterns, the soil it was grown in, and other uncontrollable natural factors. By extracting the various glycosides and determining which of them had the most beneficial effect you could establish a safe and effective standardised dose for the active principle in the herb so that the doctor would know exactly what the patient was getting and could alter the dose up or down to get the desired therapeutic response. In the case of digitalis the best glycoside was digoxin and this is still widely prescribed. Similarly, there are so many variations of the marihuana plant, so the same problem of varying concentrations of active principles in the herb would occur as it had with the digitalis herb and other herbs with recognised therapeutic effect.

Mainstream medicines and clinical practice cover end of life problems very well. There is no need for marihuana – it is just another gimmick towards the ultimate aim of the drug abuse lobby to have it legalised. You have seen that in the USA. Initially it was allowed for 'medical use'. I assumed this meant it was authorised by medical practitioners, but I saw a documentary about the medical use in America and people were consulting what looked like drug-ravaged dealers supplying it to healthy looking young people. I asked an American friend who were they? The reply was that they were 'registered' dealers. (At least, it seems the current proposal here is to use medical practitioners to prescribe it). Having achieved that first step of 'medical marihuana' the next step in America was to legalise it for everyone and this has been achieved in several states. At the moment in the USA, supply seems to be the domain of small time producers but in time it will be fully commercialised and promoted as a safe product for mainstream use – with legalistic warnings on the packaging, of course.

Some time back the public, the media and governments were outraged that companies could sell a product (tobacco/nicotine) knowing that its purpose was to get people addicted and hence produce never-ending “repeat sales” and knowing that it was dangerous to health. Yet the media and governments are virtually promoting the use of a similar product, marihuana. Heavy use of marihuana is known to cause schizophrenia and other serious effects. Lung cancer is another by-product. So now we are getting a bipolar approach to medicines – ordinary citizens get medicines that require full safety scrutiny while other patients, targeted by the drug-abuse lobby, become second class citizens whose health and welfare does not matter, because they will be using medication that does not satisfy the internationally recognised standards of quality, safety and efficacy that are required for marketing approval.

Several years ago a doctor from the Swedish Ministry of Health was invited to Canberra to speak with politicians and health professionals on the system that they were now using to minimise drug abuse. They had spent years using the “human rights/no stigmatisation” approach, only to see their country going down the gutter, so they scrapped the “Harm Minimisation” approach and beefed up the “demand reduction” strategy by bringing in abusers, caught with possession by the police, at an early stage for counselling with family members present and giving them a choice of either going to jail or getting on a program to get them off the illicit drug. As well, they had hard hitting information campaigns warning against the dangers of using illicit drugs, including marihuana. It appears to be a success but it costs a lot of money and other governments are reluctant to spend real money on reducing drug abuse. In Australia, the laissez-faire ‘harm minimisation’ model is the approach to drug abuse. One example of this is the strategy in Australia for opiate abusers which is to stabilise the patients on methadone and then do nothing – just keep them on methadone so they don’t ‘play up’. But these people are real people who need help to get back to normal and very few get off the methadone program here because there is no back up support program as in Sweden. Even in Britain, where I worked in methadone clinics, methadone patients are carefully monitored and their doses reduced by 2.5mg at a time at regular intervals so they can eventually be free of addiction. As a health professional you get to know them as real people with real needs and hopes, like anybody else. Some of my methadone patients used to take jobs driving interstate trucks – not a good look for road safety. In relation to marihuana and the amphetamine based drugs such as “ice” there seems to be no programs to help the users get off them or not use them in the first place. If marihuana is legalised there would be nothing to stop airline pilots smoking it and I would not fly on an airline whose management supported marihuana use.

According to the Swedish doctor and others the trouble with the non-opiate drugs such as amphetamines and marihuana is that they damage the receptor sites in the body where our normal body chemicals have their action. The synthetic chemicals have a greater affinity for these receptor sites than do the natural body chemicals, so they suppress the natural

chemicals and with regular use damage the receptor sites. In the case of “ice” it also destroys a proportion of the receptor sites. This means that when the abuser stops taking the drug and the natural chemicals try to come back they cannot exert their full effect because of the damage to or reduction of receptor sites. This results in a mental health deficit (caused by the drug). In the case of heavy use of marihuana it often manifests in schizophrenia and with “ice” a neurosis develops which manifests as erratic violent behaviour. As you can see in Appendix A, depression is commonly associated with marihuana use.

Contrast the current governments’ (Federal and State) ‘harm minimisation’ approach to drug abuse with the demand-reduction strategy for stopping tobacco usage (or obesity):- With illicit drugs you must not ‘stigmatise’ the product or the user because it’s not nice. With tobacco you stigmatise the user by banning smoking in restaurants, offices etc and make them go outside into the cold. With illicit drugs the school education programs are designed to help the students use illicit drugs ‘safely’. With tobacco you try and scare them into not using tobacco by having graphic health warnings on the packaging. I am all in favour of the demand reduction strategy for smoking tobacco, so why can’t we use the same thing for reducing demand for illicit drugs? The problems associated with drug abuse in our community are spiralling out of control. The previous Federal and NSW governments recognised this and neatly solved the problem by changing the name of drug abuse to “mental health” and creating separate departments to look at the problem. It must be bad if you have to have a whole expensive department devoted to it, but such a decision has only increased drug abuse because it is now considered a disease state in its own right, rather than resulting from the same kind of peer pressure associated with the uptake of smoking or alcohol use. It’s getting ridiculous where we are being told about the horrors of having sugar in your coffee or tea or having soft drinks but it’s OK to smoke marihuana. A minority of people may turn to marihuana as a result of a pre-existing mental health problem but the majority smoke it because of peer pressure and ‘fashion’. Marihuana causes mental health problems, not the other way around. It has been my experience that those who abuse drugs will never admit that their resultant poor health status is a result of their drug use, and blame something else. The latest fashion is wheat. I spoke recently to a chronic marihuana smoker who was in terrible physical and psychological shape and he told me he had taken wheat out of his diet because it was bad for his health. He still looked terrible.

I have been keeping a “scrapbook” of newspaper clippings and other documentation since 1997 – not comprehensive, but enough to be informative. I have abstracted some of these articles and documents in Appendix B attached to this letter. One thing that I have noticed is that since 1997 there has been a lot of repetitive similarities. I remember in 2007 I bought a copy of “The International Herald Tribune” somewhere in Europe and read an article in support of legalising marihuana. It was almost word for word as a similar article I had read a few weeks prior in London in the Financial Times, and as well as before and since in other

publications. It makes one question the objectivity of journalism as it appears someone is being paid by interested parties to have such articles published. The common themes in these articles are:

- The “War on Drugs” is unwinnable therefor legalise them.
- Current policies on drugs are not working, therefore legalise them
- Laws prohibiting drugs have been a failure, therefore legalise them
- Tobacco and alcohol are worse than illicit drugs, therefore legalise them
- Stigmatising drug users is an affront to human rights, therefore legalise drugs
- Legalising drugs will afford better control and cut out crime
- Humans have always turned to mind-altering drugs, therefore legalise them

In relation to the above, the “War on Drugs” is an American construct, not Australian. Authorities in Australia have never formalised their approach with such a term. The driving force for legalising marihuana comes from American organisations such as NORML, with branches in Australia. The promotional material appears to be generated in America and given to journalists here who publish it uncritically, so it appears what is happening in America is the same here.

“Current policies are not working”. I agree. Since the Drugs of Dependence Branch in Canberra in the 1980s was told to forget what they were doing and to implement the new ‘harm minimisation” policy, drug abuse, particularly smoking marihuana, has become an epidemic. These policies are definitely not working. The Police and Customs are doing a marvellous job but there is no effective policy on demand reduction to complement their supply reduction efforts. I suggest we start using in a co-ordinated way the recommendation that came out of the “Visby Accord” following the HNN World Conference on Drug Related Issues in Sweden in 2001, namely “We support prevention, treatment and enforcement/interdiction as the 3 tenets of effective drug policy.”

“Prohibition” does not work and humans have always turned to drugs. That’s true in absolute terms, but the same could be said about rape, child molestation, theft, murder, corruption etc but one does not see massive campaigns to overturn laws that prohibit these activities. People who push this ‘prohibition does not work’ line are hardly credible in the face of the stark reality of the drug abuse problem. It is not a ‘human rights’ issue, it is a ‘health’ issue, both for the individual and the country.

Legalising drugs of abuse does not cut out crime. Legal drugs with abuse potential such as oxycodone and methadone are freely sold by dealers in nightclubs and bars etc, alongside the illicit drugs. Legalising marihuana will only increase its use, because it will become ‘respectable.’

The question asked by many people who have to deal with the outcomes of drug abuse at the coalface is "When are politicians going to draw the line on listening to well funded drug abuse advocates and instead take notice of mainstream public health advice and law enforcement agencies?" The pressure from these activists, often supported by wealthy and/or influential public figures has been relentless since the 1980s. We often see newspaper articles where ex-presidents, wealthy businessmen, etc are trotted out to lend their support for marihuana use. Up until 1993 the Federal and State governments relied on public health advice from the National Health and Medical Research Council. It was formed in 1936 and grew out of the Australian Health Council which was formed in 1928 in order to harmonise the response to and address public health issues that transcended State boundaries. If a problem was detected, the NH&MRC had access to the best mainstream health expertise and after thorough investigation, would arrive at a recommendation which was given to the various governments. The governments would collectively and invariably adopt the recommendation on the basis of this objective advice and the various pressure groups (Industry or activist based) had to wear it. In 1993, this role of the NH&MRC was effectively dismantled and governments no longer used a common and central point of reference to deal with public health issues. Following this, there arose a plethora of privately funded "Public Health" bodies, run by people known to support aspects of drug abuse, including marihuana smoking, and who get positive and regular press coverage. I consider it critical for governments to establish and fund as a government entity a credible national public health advisory body along the lines of the pre-1993 NH&MRC, if we are to come to terms with drug abuse.

The other question asked is: "Why do politicians allow activist groups to promote drug abuse but act against drug dealers and users?" The *promotion* of drug abuse, which includes using marihuana, should be outlawed as well, otherwise we will drift on for another 20 years, by which time drug abuse will be out of control and Australia will start looking like Mexico with students getting killed because they can't pay their drug bills to the dealers. This is not a matter for 'moral equivalence", defined by a student in the last Q&A audience as – ("There is no good and evil, only differences of opinion"). Drug abuse is bad for the individual and bad for the country. You will never eradicate it but you can take steps to reduce the number of users to a level where the very well-being of society is not threatened. If you compare the number of drug-implicated violent crimes in Sydney that one sees nightly on TV to the time when I was growing up in Sydney, there is no comparison. It is destroying families. How many times do you see the situation where an adult child will return home and murder a family member because they refuse to give them money for drugs. The media blandly reports the action but never the cause… You must not 'stigmatise' illicit drugs or the users. I have had the benefit of a span of years covering Australian society from the 1950s to now and Australia is a much more dangerous place to live in today because of the inappropriate approach to drug abuse by our elected representatives. Several years ago I made a conscious decision not to work in community

pharmacies any longer because in two pharmacies I found aluminium baseball bats under the counter to be used in case self-defence was needed. It's got to that stage.

Please don't give in to the drug abuse lobby and allow marihuana for 'medical' use.

Yours sincerely

[redacted]

7 December 2014

Attachment A

WHO Poison Information Monograph on Marihuana

1.1 Scientific name

Cannabis Sativa L. (var Indica)

1.2 Family

Cannabinaceae

1.3 Common name(s) of the plant and synonyms (in each country)

Marihuana (Latin America)
Marijuana (Latin America)
Cañamo de la India (Latin America)
Maconha (Latin America)
Grifa
Marijane (USA, UK)
Indian hemp (USA, UK)
Guaza
Chanvre (France)
Herbe (France)
Hanfkraut (Germany)
Kif
Ganja
Hemp
Juana
Pot
Yerba

Note: Cannabis resin may receive different names such as: Hashish, Charas, Chira, Hasis, Gauja and others.

There are numerous "street" names for the plant or the resin that can be added (e.g. grass, tea, weed, reefer).

2. SUMMARY

2.1 Main risks and target organs

The main target organ is the CNS.
Acute or prolonged psychotic states, distress and anxiety may occur.
Traffic and occupational accidents may be related to acute effects of marihuana use.

Larynx and bronchopulmonary neoplasm have been attributed to smoking marihuana.

The possibility of immunologic, chromosomal, reproductive and foetal abnormalities is under research.

2.2 Summary of clinical effects

Acute intoxication - depends upon several facts such as (e.g. emotional state, past experiences, psychological state, association with other drugs. Marihuana may produce: apprehensive states, panic, anxiety, hallucinations and prolonged psychotic states (acute delirious psychosis, paranoid reactions, flashbacks, excitation with auto or heteroagressiveness, mental confusion and depersonalization). Severe headaches and abdominal discomfort are common.

On clinical examination: motor incoordination, reduction of reflex responses, distortion of the perception of time and space, conjunctival irritation, dryness of mouth and throat, pulmonary irritation (hacking cough, bronchial hypersecretion, bronchospasm) tachycardia and tremor.

Note: The effect sought by the consumer is a disinhibition state with euphoria, unmotivated laugh, relaxed sensation and pleasant drowsiness. After the lethargic phase, changes in humour and depression may be observed.

Chronic use may be associated with the induction of "amotivational syndrome" and loss of memory, amongst many other possible effects (see section 9).

2.3 Relevant laboratory analyses/sample collection

Sample collection:
specimen of the plant or seeds for identification.

marijuana cigarettes.

urine for determination of cannabinoids (can be positive even three days after a single use and four weeks or more after chronic use).

saliva for determination of cannabinoids (experimental)

Toxicological analysis: detection of cannabinoids in urine by thin layer chromatography, immunoassay or EMIT test.

2.4 First-aid measures and management principles

Treat psychiatric symptoms: calm the patient, make him feel protected and safe, prescribe rest in a noise-free shady room. Induce a sense of security and trust using a soft and monotonous voice ("talking down"). In case of severe excitation, administer diazepam 10 mg IM. When psychotic phenomena predominate, haloperidol 5 mg IM is recommended. The patient should be kept well hydrated.

2.5 Poisonous parts

The active principles are contained in all parts of the plant, but mostly in the brown resin secreted by hairs in female inflorescences.

2.6 Main toxins

Marihuana contains approximately 60 active compounds, or cannabinoids; the most important is delta-9-tetrahydrocannabinol.

Other cannabinoids are: cannabinol, cannabidiol, cannabinolic acid, cannabigerol, cannabicyclol, different isomers of tetrahydrocannabinol.

3. CHARACTERISTICS

3.1 Description of the plant

3.1.1 Special identification features

Only macroscopic details will be described.
Indian hemp is an annual dicotyledon herb that can reach up to 3 metres high or more with suitable humidity and soil. The stem is covered by rigid hairs, rough to the touch. Species are male and female; the latter has more leaves. The leaves are long-stalked, palmate, with 3-7 narrow and toothed leaflets. The upper leaves are alternate and the low ones, opposite.

Flowers are very small, green and have axillary branches (Hardin & Arena, 1974). The fruit is oval, flat, 5 to 6 mm long and 4 mm wide, with a light green colour. It has an herbaceous strong smell and taste.

3.1.2 Habitat

It grows in well-irrigated lands with warm weather, blooming in the spring and summer.

3.1.3 Distribution

The plant is originally from the Caspian and Black Sea area, and was taken to Persia and India eight centuries ago.

The Cannabis sativa native to Central Asia has a world wide distribution. It is cultivated in North, Central and South America, in Asia, Europe and North and Central Africa. Major producers include Mexico, Brasil, Paraguay, Colombia, Peru, New Zealand and Arabia.

The Indian variety is cultivated in the Orient, Asia and North Africa.

3.2 Poisonous parts of the plant

The active principles of this plant are contained in the brown resin secreted by hairs located in female inflorescences. Although the active principles are located in the whole plant, the major concentration is in the blooming buds. The so called "seed" or fruit, has a lower concentration, but leaves and blooming parts have a higher concentration. When they are new, they contain more

active principles and are called "Kifi". The dried leaves and buds (less rich), are called "bhang". The resin of the buds, the part with major pharmacological activity, is called "hashish"; this can be extracted and stored in blocks.

The common "street" marihuana is usually a mixture of dried flowers, leaves and occasionally seeds. It has a brownish green colour, depending on dryness and maturity of the plant.

3.3 The toxin

3.3.1 Name

Indian hemp contains more than 60 cannabinoids including: cannabinol, cannabidiol, cannabinolic acid, cannabigerol, cannabicyclol, and various tetrahydrocannabinol isomers, the most important is **delta-9-tetrahydrocannabinol (delta-9-THC).**

3.3.2 Description, chemical structure stability

CAS number = 1972-08-3

Molecular weight = 172

Structural formula:

Solubility: Practically insoluble in water, soluble 1 in 1 in alcohol and ketones, and 1 in 3 of glycerol. It is soluble in fixed oils.

Stability: decreases with time, especially in the case of hashish, but may resist high temperatures without inactivation.

The concentration of delta-9-THC in a sample depends upon the genetic structure of the plant, local conditions of growth, storage methods and time elapsed between harvest and use.

Street marihuana, called "sin semilla" (seedless) has usually higher concentrations of delta-9-THC of 7 - 14%.

Hashish, the oily dark substance obtained after dissolving the marihuana paste contains 20 to 30% of delta-9-THC. One drop induces an effect similar to one cigarette. It is generally commercialized as fragments of solid resin called "chocolate" (Reynolds, 1982; Jaffe, 1986).

3.3.3 Other physico-chemical characteristics

It has a penetrating, sweetish aroma, very persistent, that impregnates clothes and hair of users.

Cannabinoids are soluble in alcohol, ether and other organic solvents, but non-soluble in water and mineral acids.

3.4 Other chemical contents of the plant considered relevant

No data available.

4. USES/CIRCUMSTANCES OF POISONING

4.1 Uses

Medicinal: delta-9-THC and some synthetic analogues are used therapeutically, e.g. for nausea and vomiting produced by antineoplastic chemotherapy. Synthetic cannabinoids used therapeutically include "Dronabinol", nabilone and "Levonamtradol".

A further possible indication is to reduce intra-ocular pressure in the treatment of open angle glaucoma.

Some synthetic cannabinoids are undergoing clinical trial as analgesics and anticonvulsants.

Cannabis sativa has been mixed with other plants in the preparation of anti-asthma cigarettes.

Medical uses have been limited by adverse effects similar to those observed after smoking marihuana, but Dronabinol and nabilone have been approved by regulatory authorities in the United States for therapeutic use (Reynolds, 1982).

Abuse: marihuana is the most frequently abused drug in the world after alcohol and tobacco, and it is the main illegal drug of abuse.

Religious: some cultures accept its use for a defined religious purpose (e.g. some Buddhist and Tibetan sects, and groups in the north of Africa).

4.2 High risk circumstances

As in every case of drug abuse, several determining factors should be considered at individual, family and social levels. Therefore, the high risk circumstances of use and abuse will vary according to the country or region.

4.3 High risk geographical areas

Abuse of marihuana is worldwide, but it is more common in cities than in rural areas.

5. ROUTES OF ENTRY

5.1 Oral

Not common in cases of abuse but it is the usual route of administration for medical purposes.

5.2 Inhalation

The commonest way of consuming marihuana and hashish. The inhaled smoke of one cigarette ("joint") contains 0.5-0.7 g delta-9-THC.

Marihuana can be smoked directly or through small pipes or "bongs". They are similar to those used with opium, where refrigeration by air or water reduces the irritant effects on the tracheo-bronchial tract allowing a deeper and prolonged inhalation.

The usual technique consists in inhaling very deeply and maintaining the smoke in the lungs for 20 or 30 seconds to maximise the absorption of cannabinoids; the extraction is about 50% of the cannabinoid content.

5.3 Dermal

No data available.

5.4 Eyes

No data available.

5.5 Parenteral

There are some reports of experimental intravenous injection of marihuana solutions and of cannabinoids in clinical trials.

5.6 Others

No data available.

6. KINETICS

6.1 Absorption by route of exposure

Oral administration

Absorption from the gastrointestinal tract is almost complete. Peak blood levels and maximal pharmacological effects occur later after oral administration than after inhalation (Cone, 1988). Symptoms become apparent within 30 - 120 minutes, reaching a peak after 2 - 3 hours (Schwartz, 1987; Jaffe, 1985).

Inhalation

After inhalation, peak plasma concentrations are achieved within a 7 - 10 minutes; subjective effects appear in 20 or 30 minutes but rarely persist for more than 2 - 3 hours.

No data are available on other routes of absorption.

6.2 Distribution by route of exposure

Delta-9-THC is metabolised by the liver. It is intensively lipophilic and high concentrations accumulate in fatty tissues in great quantities; these are liberated slowly into the circulation (Jaffe, 1986).

6.3 Biological half-life by route of exposure

The half-life of delta-9-THC is 3 days. Jaffe (1985) reported that plasma concentrations of delta-9-THC and 11 hydroxydelta-9-THC fall rapidly (in a few minutes) due to their redistribution in the fatty tissues; afterwards there is a slow decline with a half-life of 30 hours due to the metabolism and gradual elimination of the drug. The half-life maybe increased in chronic users to 4.1 days (range 2.9 and 5.0 days) (Johansson, 1988).

6.4 Metabolism

After oral administration but not after inhalation, delta-9-THC undergoes first-pass hepatic metabolism via enzymatic hydroxylation and carboxylation to the active metabolite 11-hydroxy-delta-9-THC, then carboxylation to the more polar inactive metabolite, 11-nordelta-9-THC acid. Enterohepatic circulation occurs and only 35% is excreted in the urine.

Around 80 cannabinoid metabolites can be identified from a similar metabolic pathway; the most important one is 11-hydroxy-delta-9-THC which is metabolised to non-cannabinoid metabolites such as terpenes and alkenes.

Delta-9-THC and its metabolites persist in human plasma for several days or weeks (Jaffe, 1986) but repetitive ingestion or smoking over weeks is not followed by clinically apparent accumulation; this suggests that the persistent metabolites are inactive.

Chronic marihuana smokers metabolise delta-9-THC more rapidly than non-smokers.

6.5 Elimination by route of exposure

35% of delta-9-THC and its metabolites is eliminated in the urine compared with 65% in the faeces.

Metabolites can be detected in urine even 2 - 3 days after one exposure and, in cases of chronic use, after 4 - 5 weeks of abstinence.

7. TOXICOLOGY/TOXINOLOGY/PHARMACOLOGY

7.1 Mode of action

No specific mechanism or site of action has been demonstrated. The effects on the CNS can be determined by a diminution of cholinergic activity at neuronal level. Psychological effects do not depend on dopaminergic or noradrenergic action.

The cardiovascular effects (tachycardia, decubitus systolic hypertension and orthostatic hypotension) are counteracted by propranolol (Jaffe, 1986).

7.2 Toxicity

7.2.1 Human data

7.2.1.1 Adults

The average toxic dose is 0.035 mg/kg (Schwartz, 1987). The minimal effective dose of delta-9-THC is 5 mg. A 0.5 - 1 g marihuana cigarette contains 0.5 - 11% delta-9-THC, (Jaffe, 1986). Assuming that the average concentration is 5% delta-9-THC and that 50% is destroyed by pyrolysis during smoking, the total inhaled dose is approximately 25 mg; of this, approximately 60% is absorbed by inhalation.

Nahas (1975) estimates that the lethal dose by intravenous injection is 2 g for a 70 kg person.

The minimum plasma concentration of delta-9-THC which produces psychotropic effects is 25 ng/ml (Hollister, 1988).

7.2.1.2 Children

No data available.

7.2.2 Animal data

With Cannabis extract, the LD50 in mice is:

oral	21.6 g/kg
dermal	11g/kg
IV	0.18g/kg

(Valbuena Briones, 1987)

7.2.3 In vitro data

No data available.

7.3 Carcinogenicity

Marihuana smoking and hashish abuse produce histological changes and affect the bronchial epithelium in the young animal.

In animals, the tar produced by marihuana pyrolysis is more carcinogenic than that of tobacco (Jaffe, 1986). High temperature burning corresponding with deep inhalation into the lungs, together with smoking until the end of the cigarette, all increase the carcinogenic risk of delta-9-THC and polyaromatic hydrocarbons.

7.4 Teratogenicity

Cannabinoids cross the placental barrier and may affect foetal development. When mothers are exposed to cannabis during pregnancy, both the human and animal newborn may show postpartum behavioural effects such as altered response to stimuli and impairment of learning (Jaffe, 1986).

In a prospective study of 1226 mothers, of which 27% were users, urine metabolites of cannabis were found in 16% of cases, and there were alterations in foetal development, weight and height, with values lower than those of a child born to non-smoking mothers (Zucherman, 1989).

7.5 Mutagenicity

Comparative studies between marihuana smokers and non-smokers show that more than 60% of the former have a significant increase of chromosomal alterations: in smokers the average was 3.4% of leucocytes, and in non-smokers only 1.2%.

7.6 Interactions

Abuse of marihuana may lead to the use of other drugs and alcohol. It is important to determine whether other drugs have been consumed in case of acute intoxication. Unfortunately, only a few

interactions are well-known, so the physician must be alert to the possible variety of clinical presentations.

Clinical features other than the typical symptoms should be evaluated in order to determine the possible association with other drugs.

Delta-9-THC enhances the metabolism of barbiturates, antipyrine, and ethanol.

The combination of cocaine and marihuana reportedly significantly increases heart rate and arterial pressure (Foltin, 1987). This finding is important if we consider the frequent association between marihuana and cocaine abuse. The authors conclude that this combination in a non-controlled situation and in high doses may cause severe cardiovascular toxicity.

8. TOXICOLOGICAL/TOXINOLOGICAL ANALYSIS

8.1 Sample

8.1.1 Collection

Collect sample of the plant, seed, leaves, dried preparation, cigarettes or other suspected specimen (e.g. resin) for pharmacognostical or analytical identification.

Biological specimens (urine) should be collected in clean flasks and sent to the laboratory.

8.1.2 Storage

No special requirements

8.1.3 Transport

No special requirements

8.2 Toxicological/Toxinological analysis method

(In preparation)

8.2.1 Test for active ingredient

(In preparation)

8.2.2 Test for biological sample

(In preparation)

8.3 Other Laboratory analyses

(In preparation)

8.3.1 Haematological investigations

(In preparation)

8.3.2 Biochemical investigations

(In preparation)

8.3.3 Arterial blood gas analysis

(In preparation)

8.3.4 Other relevant biomedical analyses

(In preparation)

9. CLINICAL EFFECTS

9.1 Acute poisoning by:

9.1.1 Ingestion

The clinical presentation is similar to that produced by inhalation (see section 9.1.2) though there are differences in the time to onset of symptoms, which may be delayed by 30 minutes to 1 hour.

9.1.2 Inhalation

The acute effects of cannabis depend on:

. the concentration of delta-9-THC in plasma

. the concentration of delta-9-THC in the marihuana cigarette

. the inhalation technique (prolonged and deep inhalation; use of bong, pipes, others),

. individual and environmental conditions (set and setting)

Setting
Conditions depending upon the person, such as previous experiences, attitude, expectations for the actual experience, and personality. In this way, inexperienced young persons, fearing to be discovered, can present acute anxiety reactions and panic, fear of losing self-control and unpleasant sensations. Young people with unstable personalities and acute affective disorders, such as

depression or a psychotic background (unrelated to drugs), have a higher risk of developing adverse effects and permanent psychosis.

Setting
Conditions due to the environment (confidence in partners, link between users and other participants, comfort, safety, etc).

Interactions between cannabis and other drugs of abuse and alcohol may provoke unpredictable effects in individuals and the response to delta-9-THC may be very complex.

The usual clinical picture appears after smoking a cigarette with 2% of THC, or after a 20 mg dose of delta-9-THC. After a few minutes, the first effects on humour, motor coordination, sensitivity, auto-perception, cognitive capacity, attention and time perception occur. Feelings of well-being, euphoria, laughing and relaxation are common; somnolence is observed when the individual is alone.

Tasks that require intermediate steps before reaching the objective are difficult to perform (time misperception). There is a tendency to mix past, present and future with a strange feeling of unreality and depersonalization. Motor coordination (balance and reaction time) is always affected, even at low doses, and there is therefore a higher risk of accidents. Perception of colour, distance, and depth, and visual acuity is impaired. These effects are more persistent than the subjective disorders, and they last for 4 - 24 hours.

Marihuana smokers frequently experience hunger, dryness of the mouth and throat, vivid visual images, hyperacusia, and increased sensations of touch, taste and smell. There is a reduction of empathy and perception of other people's emotions, conversation may be unclear and communication may be interrupted by unrelated words and ideas.

At higher doses the patient may have: hallucinations, delirium, paranoia and variable degrees of anxiety culminating in panic and toxic psychosis.

There is an increase in heart rate, with high supine systolic blood pressure and orthostatic hypotension. Conjunctival irritation is usually seen.

The patient's body temperature is increased by inhibition of sweating.

Respiratory effects are related to the chronic use of marihuana; the acute effect is bronchodilatation both in healthy and asthmatic individuals. But in the latter, irritative effects may precipitate an asthmatic crisis. A painful, itching or burning sensation of mouth and throat produces irritating cough.

Hashish smokers develop inflammation and swelling of the uvula.

The cannabis "trip" may be interrupted easily voluntarily, so that the person may look normal, even in his affective relations and in the speed and contents of speech.

After 2 or 3 hours, the user may gradually "leave" the intoxicated state and develop clumsiness (physical and mental), irritability (that may turn into rage), somnolence and deep sleep. Depression may occur.

During this "coming down" phase, the avidity for food rich in carbohydrates, sweets and cola soft drinks is common. Once the "trip" is over, there is practically no hangover.

Only two physical signs persist: tachycardia and conjunctival irritation (although the latter can be avoided by use of eye drops).

Although the effects of marihuana are usually pleasant, adverse effects may be observed even when the consumer is experienced, and may be seen also after a single dose, even if it is low.

Adverse effects are:

acute toxic psychosis with: excitation, confusion, disorientation, illusions, depersonalization, visual hallucinations and delirium.

acute panic reactions ("bad trip") accompanied by abdominal pain, headaches, anxiety, depression with excessive fear of being discovered, fear of dying and uncontrollable aggressive feelings with paranoid ideas.

"flashback" reactions are not frequently associated with cannabis but some cases have been reported (Schwartz, 1986).

affective disorders

chronic psychosis

9.1.3 Skin exposure

No data available.

9.1.4 Eye contact

Conjunctival irritation is usually seen due to the direct effects of smoke contact.

9.1.5 Parenteral exposure

In cases where parenteral administration has been observed experimentally, the clinical picture is similar to that produced by social use, although the symptoms develop faster and more intensely.

9.1.6 Other

No data available.

9.2 Chronic poisoning by

9.2.1 Ingestion

No data available.

9.2.2 Inhalation

The chronic effects of Cannabis are controversial, but believed to be more important the younger the patient starts with the abuse. They may consist of:
amotivational syndrome: although marihuana may be a primary or secondary factor inducing amotivational syndrome, it is not the main one: a drug that produces passiveness is effective only in the predisposed individual (Cohen, 1982).

The syndrome consists of:

loss of interest, apathy, passiveness

lack of interest in work and productivity without any concern

lassitude and loss of energy

lack of tolerance and easy frustration

melancholy, bad temper and whims.

loss of concentration and inability to process any new information.

shabby look

a life style that is based on a search for the drug.

Use of other drugs: some authors believe that marihuana may lead to abuse of other drugs, a phenomenon that has been called "escalation".

Impairment of memory, loss of concentration, a loss of faith in themselves, in judgement and ambitions; deterioration of relationships with family, teachers and other authorities has been recognized by most chronic abusers after treatment.

crime: chronic abusers may be involved more frequently in illegal activities.

Tolerance and dependence are mainly due to functional or pharmacodynamic adaptations of the CNS, rather than to faster metabolism and excretion. Tolerance develops to emotional changes, tachycardia, body temperature and psychomotor tasks; tolerance of the cardiac effects may develop in just a few days.

Experienced abusers may have more intense subjective effects than beginners, but will have lesser deterioration of perceptive and motor functions.

After chronic abuse at high doses, sudden discontinuation produces: irritability, restlessness, nervousness, loss of weight, insomnia, tremors, rise of body temperature and shivering.

Symptoms may start a few hours after withdrawal, and last a few days (Schwartz, 1987; Jaffe, 1986).

Car accidents may be an indirect consequence of acute abuse. In those who smoke marihuana more than 6 times per month, the risk of car accidents is increased 2.4-fold (Schwartz, 1987).

Endocrine effects have been reported following chronic use, including impairment of gonadotrophin secretion (FSH and LH), reduction in testosterone levels and a direct effect on cytochrome P 450 of the Leydig cells with inhibition of testosterone synthesis. Both mechanisms will lead to olisgospermia and possibility of subfertility (Nahas, 1975).

In the woman, alteration of menstrual cycles (amenorrhoea and anovulatory cycles), and also a reduction in plasma levels of prolactine have been reported.

Chromosomal alterations have been observed in germinal cells and lymphocytes.

Effects on the respiratory system:

chronic bronchitis, sinusitis, asthma, rhino-pharyngitis, uvular inflammation.

bronchopulmonary histological changes that may lead to squamous cell metaplasia and hyperplasia of basal cells with changes in subepithelial glandules and infiltration of mononuclear leucocytes in the alveoli and pulmonary interstitium. These anomalies are similar to those considered as pre-cancerous in tobacco smokers and have been found in marihuana smokers who do not smoke tobacco.

Wu (1988) reported higher levels of carboxyhaemoglobin in marihuana smokers than in tobacco smokers.

Histological alterations at cerebral levels have been described by some authors (Jaffe, 1986).

Alterations on the immune system are possible, although evidence is not conclusive. The immune response may be suppressed due to a reduction of T-lymphocytes secondary to alteration in DNA production.

chronic diarrhoea, abdominal pain and loss of weight are frequent in marihuana abusers.

Note: It is important to stress that some of the symptoms described as "chronic effects" may be experienced by teenagers undergoing an 'adolescent crisis' and should not, therefore, be readily attributed to use of marihuana.

9.2.3 Skin exposure

No data available.

9.2.4 Eye contact

No data available.

9.2.5 Parenteral

No data available.

9.2.6 Others

No data available.

9.3 Course, prognosis, cause of death

The normal course of acute poisonings is usually uneventful, once the subjective effects have ceased. Motor coordination and reflexes may take a few hours to come back to normal.

The prognosis may be uncertain in case of psychotic reactions and only follow-up of the patient will allow diagnosis of a permanent psychosis. Adequate treatment and avoidance of further exposure usually gives a favourable prognosis which is more dependent on the psychiatric features of the patient than on the marihuana use.

Tachycardia will rarely exceed 140 or 150/min, but patients with previous cardiovascular impairment may be at risk of acute cardiac failure.

The evolution and prognosis of chronic abuse depends upon adequate treatment and consideration of other factors associated with the use of drugs. Most effects are reversible once abuse has ceased.

Chronic use of cannabis may cause bronchopulmonary cancer which may appear earlier than in tobacco smokers (Valbuena Briones, 1987).

The clinical course and prognosis will be worse if other drugs and alcohol have also been taken.

The usual cause of death is by accident or clinical complications, as it is extremely rare to have lethal over-dose (the lethal dose is very high).

Cases of acute marihuana poisoning and overdose by other more dangerous drugs should be managed as a poisoning by the associated drug.

9.4 Systematic description of clinical effects

9.4.1 Cardiovascular

Tachycardia, increased systolic pressure (in decubitus); orthostatic hypotension, vasodilatation.

9.4.2 Respiratory

Acute irritative effects, cough, and asthmatic crisis in predisposed patients.

Chronic use may produce chronic bronchitis, sinusitis, rhinopharyngitis, bronchopulmonary and laryngeal cancer.

Acute on chronic use may also lead to acute bronchitis and pneumonia.

9.4.3 Neurologic

9.4.3.1 Central Nervous System

Acute intoxication: motor incoordination, alterations of emotional states, impairment of self-perception and perception of the environment (hallucinations, illusions), impairrment of cognitive functions, memory deficits and decreased attention. Chronic use may produce amotivational syndrome and chronic psychosis. Acute use in chronic abuser can produce adverse reactions if the patient was already a psychosis or amotivational syndrome.

9.4.3.2 Peripheral nervous system

No data available.

9.4.3.3 Autonomic nervous system

No data available.

9.4.3.4 Skeletal and smooth muscles

No data available.

9.4.4 Gastrointestinal

Acute abuse induces dry mouth, thirst and a desire to eat carbohydrates and sweet soft drinks. Chronic abuse may produce diarrhoea, abdominal pain and loss of weight.

9.4.5 Hepatic

Chronic use may induce the hepatic metabolism of ethanol, barbiturates and antipyrine.

9.4.6 Urinary

9.4.6.1 Renal

No data available.

9.4.6.2 Others

No data available.

9.4.7 Endocrine and reproductive system

Chronic effects on man are: infertility due to oligospermia, impotence, and chromosomal alterations in germinal cells. Theoretically, aggravated oligospermia may occur as an acute on chronic effect.

In the woman: amenorrhoea, anovulatory cycles, chromosomal alterations in germinal cells and diminuition of prolactin serum levels.

Endocrine effects have been described after chronic use. Impairment of gonadotrophin secretion (FSH and LH), reduction in testosterone levels and also a direct effect on cytochrome P 450 of the Leydig cells with inhibition of testosterone synthesis. Both mechanisms will lead to olisgospermia and possibility of subfertility (Nahas, 1975).
In the woman, alteration of menstrual cycles (amenorrhea and anovulatory cycles), and also a reduction in plasma levels of prolactin have been reported.

9.4.8 Dermatological

Skin dryness occurs during acute poisoning.

9.4.9 Eyes, ears, nose, throat: Local effects

In acute poisoning: conjunctival irritation, itch and burning sensation of throat, dryness of mouth.

In chronic poisoning: swelling and inflammation of the uvula.

9.4.10 Hematologic

Anaemia by nutritional deficiency has been reported in chronic abusers.

9.4.11 Immunological

Immune deficiency due to diminished numbers of T-lymphocytes has been reported in chronic abusers.

9.4.12 Metabolic

9.4.12.1 Acid Base disturbances

No data available.

9.4.12.2 Fluids and electrolytes disturbances

No data available.

9.4.12.3 Others

Hyperthermia may occur if the patient is in a hot environment (Jaffe, 1985).

9.4.13 Allergic reactions

No data available.

9.4.14 Other clinical effects

Abstinence syndrome:

After chronic abuse at high doses, sudden discontinuation produces: irritability, restlessness, nervousness, loss of weight, insomnia, tremors, rise of body temperature and shivering.

Symptoms may start a few hours after withdrawal, and last a few days (Schwartz, 1987; Jaffe, 1986).

9.4.15 Special risks

In drug abusing patients it is necessary to investigate HIV serological positivity, because there may be a possible association with intravenous use of other drugs as well as with other high risk situations related to drug abuse.

Pregnancy: impaired foetal development and decreased in growth have been described (Zuckerman, 1989).

9.5 Others

Cannabis may be contaminated with:

- Paraquat: abuse of contaminated marihuana without secondary ill-effects has been reported (Schwartz, 1987).

- Aspergillus fumigatus: a fatal case of pulmonary aspergillosis was reported in a heavy marihuana smoker; the fungus found in the marihuana and obtained from pulmonary biopsy was the same (Hamadeh, 1988).

10. MANAGEMENT

10.1 General Principles

Management of psychiatric disturbances is based on sedation and antipsychotic treatment.

There is no risk of death in cases of pure acute Cannabis intoxication. However, the cardiovascular and respiratory functions should be monitored carefully in order to detect possible severe tachycardia or bronchial asthma.

If abuse of other drugs or alcohol is suspected, vital functions should be monitored and drug treatment should be avoided or used with care due to the risk of potential interactions.

In cases of chronic abuse, patient management should have two main objectives: i) evaluation and treatment of organic consequences, and ii) psychological and social assistance by an interdisciplinary staff.

10.2 Relevant laboratory analysis

10.2.1 Sample collection

Blood: if general evaluation of the patient is required, blood should be collected as usual for routine exams.
Urine for detection of cannabinoids: first-voided urine contains the highest concentration of cannabinoids. It can be refrigerated, frozen or even stored at room temperature for up to three days until it can be sent to a reliable and carefully supervised medical laboratory for testing (Schwartz, 1987).

Saliva samples can be tested for cannabinoids in some countries.

Cigarettes and suspected marihuana should be kept for macroscopical, microscopical and chemical identification.

10.2.2 Biomedical analysis

No biomedical analysis is considered relevant for diagnosis, prognosis and treatment of this acute intoxication. If the patient is a chronic abuser of drugs, the following could be necessary for prognosis and therapy:

Chest X-ray
Otorhinolaryngological evaluation and laryngoscopy according to the clinical picture
Serological studies for HIV
Echographic diagnosis and monitoring of pregnancy

10.2.3 Toxicological analysis

(In preparation)

10.2.4 Other investigations

As required by the patient's clinical condition.

10.3 Life-support procedures and symptomatic treatment

Life-support procedures are not usually necessary, but some clinical states may require them:

If cardiovascular effects (tachycardia) are present, cardiac monitoring is necessary and propranolol can be administered in the usual dosage (80-180 mg orally or IV, according to the clinical situation).

In case of bronchoconstriction, inhalation of bronchodilators may be necessary. Dosage will depend upon the drug and clinical severity of the case.

The psychological and psychiatric consequences may require:

(i) hospitalization in a quiet and comfortable room

(ii) if sedative measures are necessary, the first step will be the "talking down" technique in order to give confidence and calm down the patient. This may be followed, if needed, with diazepam 5 - 10 mg IM.

(iii) If psychotic reactions occur, haloperidol 5 mg IM may be administered.

It may be useful to consult a psychiatrist, especially in difficult cases.

Physical measures of restraint are not recommended because psychological support giving security and confidence to the patient in a monotonous and smooth voice is frequently enough to overcome the panic crisis.

10.4 Decontamination

No procedures for decontamination are available.

10.5 Elimination

There is no procedure to accelerate elimination.

10.6 Antidote treatment

10.6.1 Adults

No data available.

10.6.2 Children

No data available.

10.7 Management discussion: alternatives, controversies, research needs

The management of acute marihuana intoxication is symptomatic and depends upon the experience of the treating physician. Differences in treatment will be seldom important. Perhaps the only symptoms that may influence prognosis are those related to some psychotic reactions, in which case a wrong evaluation and treatment can lead into a chronic psychiatric illness.

The main controversy is over the treatment of chronic users: some consider that hospitalization is needed in every case; others consider that users of marihuana do not need treatment. Between these extremes, various possibilities include: individual psychotherapy, out-patient management, group therapy, and uni-, multi- or interdisciplinary treatment.

Further information is required on the possible teratogenic, mutagenic and oncogenic effects of marihuana. Bearing in mind the extended use and abuse of marihuana, clinical and biochemical research is important but it should be scientifically sound and well documented.

11. ILLUSTRATIVE CASES

11.1 Case reports from literature

Note: published case reports do not describe the usual cases of marihuana smoking, except when abnormalities arise due to complications or associated abuse of other drugs.

A 21 year old man from Angola who used great amounts of hashish from the age of 11. He used other drugs only occasionally. He was hospitalized in the psychiatric unit of a prison hospital with a

severe depressive syndrome and suicide attempt. When he gave up hashish abuse, all the clinical evidences of depression disappeared (Valbuena Briones, 1986).
Fatal pulmonary aspergillosis occurred in a patient who had undergone bone marrow transplantation who smoked contaminated marihuana. He was 34 years old; he developed pulmonary aspergillosis on the 75th day after bone marrow transplantation for chronic myelocytic leukemia. He had smoked marihuana heavily for several weeks prior to hospital admission. The cultures of marihuana revealed aspergillosis fumigatus with identical morphology and growth to a sample grown from an open lung biopsy specimen (Hamadeh, 1988).

11.2 Internally extracted data on cases (from the writer of the monograph)

A 21 old woman who was a heavy marihuana abuser (five or more cigarettes per day for 5 years) asked for help to give up the abuse. She had also used amphetamines and smoked tobacco (less than 10 cigarettes a day). The most relevant clinical findings were: loss of weight, amotivational syndrome, irritability, frequent problems with the police and paranoid ideation. All the biological parameters were normal and she was HIV negative. Three months later she began treatment and presented a severe dysphonia resistant to usual therapy. The evaluation by otorhinolaryngologist showed a chronic inflammatory process of vocal cords with squamous metaplasia. Unfortunately, as the patient gave up the treatment, we were unable to follow-up the case.

The management of this patient was based upon symptomatic treatment and psychological assistance. No antipsychotic drugs were needed as she had an acceptable evolution when gave up the drug with psychological support. Ketazolam 12 mg twice a day were needed to reduce anxiety at the beginning of treatment (one month).

11.3 Internal case

To be added by the PCC.

12. ADDITIONAL INFORMATION

12.1 Availability of antidotes

No data available.

12.2 Specific preventive measures

Not relevant.

12.3 Other

No data available.

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14. AUTHOR(S), REVIEWER(S), DATE(S) (INCLUDING EACH UPDATING) COMPLETE ADDRESSES

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Attachment B
Abstracts from Newspaper Clippings and other Documents

Articles relating to marihuana

The Telegraph 13 June 2000, by Rachel Morris and Lillian Saleh

"Dr …. (*neuro psychologist*) has reviewed medical research on marijuana over the past 4 years. 'In the past four years there has been a significant increase in the number of people being admitted to psychiatric units in NSW as a result of cannabis-induced psychosis with figures having increased from 15 per cent in 1993 to 26 percent in 1998.' 'If 26 per cent of people are fronting with serious mental illness and manic depression, then I hardly see it as a soft recreation drug.' Dr …. said yesterday.

He said the biggest rise in cannabis use was among those 14 to 19. 'Thirty nine per cent of that age group admitted to using cannabis in the past 12 months while 10 per cent admitted using it on a regular weekly basis.' The second largest group was their parents – those 40 to 49 years old. Dr .. said that marijuana was 70 per cent more carcinogenic than tobacco.

…. Will look at ways to encourage children and young people to "Just Say No" to drugs rather than use the harm minimisation approach favoured by the State Government

The Telegraph 5 March 2001, letter by Father John George

… The UN International Narcotics Control Board is somewhat cautious in regarding the therapeutic use of cannabis. On December 12, 2000 the Board replied … "… any decision on the medical use of cannabis should be based on clear scientific and medical evidence ..there have been so far no results made available to the Board …. " .. The International Narcotics Control Board has refused to accept mere anecdotal evidence in place of solid extensive scientific research on the medical use of cannabis…… "There have been so far no results made available to the board from research undertaken in the Netherlands, the United Kingdom and in the US."

3-6 May 2001 – Report on HNN World Conference on Drug Related Issues, Sweden

… – Chairman, the Institute of Global Drug Policy- "The first behaviour to disappear is judgement and therefore responsibility. It shows a misunderstanding of the addictive personality to expect that people receiving free needles or 'medical' marijuana will now behave responsibly. Marijuana is made up of 140 different chemicals. One of those 140 is a sedative/tranquilliser which could be beneficial, but no other medicine is taken via smoking – not only a very inefficient way to administer a tranquilliser but also very toxic. Serious consideration of using marijuana medically would be to extract the sedative ingredient from the other harmful ingredients and take it in oral form."

Mr ….. - member of the Dutch Parliament – 'He spoke against the direction his government has taken in liberalising drug laws and spoke of the reasoning they have for their decisions and the

inconsistencies. E.g. It is legal (in Holland) for a drug café to supply a small amount of marijuana for personal use to its customers, but illegal to be found with a large cache, or to supply in bulk any marijuana. But obviously the cafes have to deal with someone so they deal with blackmarket suppliers. To get around this contradiction the government will have to either legalise the drug dealers or become the drug dealers themselves.' (*Note: It is illegal in Holland to smoke tobacco products in these cafes, but OK to smoke marihuana!!??)*

June, 1998 – Swedish Drug Policy (Extract) presented to UN General Assembly

(In Sweden).. great efforts are made to limit the experimental use of cannabis, for 3 reasons.

1. ..'research has shown that cannabis can cause serious harm, especially to teenagers. A comprehensive survey of current research into the harmful effects of cannabis, *Adverse Health Consequences of Cannabis Use – A survey of scientific studies into the range of damage to health caused by cannabis* (Swedish National Board of Health and Welfare, 1998) shows that teenagers' psychological development is liable to be stunted or retarded by cannabis use.'
2. 'research shows that, even if cannabis does not *automatically* lead to abuse of other drugs, cannabis use does constitute a heightened risk of more severe drug abuse to occur at a later stage.'
3. 'There is a statistical connection between cannabis and other drugs. Over the past thirty years we have seen that when cannabis use declines, abuse of other drugs does the same. And when cannabis use increases, there is a corresponding upsurge, for example, in heroin and amphetamine abuse.'

28 February 2010 – Sydney Morning Herald, by Nicole Ostrow

''Young adults who used marijuana as teens were more likely than those that didn't to develop schizophrenia and psychotic symptoms, a seven-year old study has found. Those who use the drug six or more years were twice as likely to develop a psychosis such as schizophrenia or to have delusional disorders than those who never used it. Research involving more than 3800 young adults …. found long term users were also four times more likely to have psychotic-like experiences." ….

"Dr …. said even those who had used marijuana for fewer than three years still had an increased risk of scoring higher than those who had not."

(The above study by the Queensland Brain Institute at the University of Queensland was funded by the National Health and Medical Research Council of Australia)

2 March 2011 - BBC News – 'Cannabis use raises psychosis risk – study'

'Using cannabis as a teenager or young adult increases the risk of psychosis, a report suggests. The study published in the British Medical Journal involved tracking 1,900 people over a period of 10 years. …. This research strongly suggests that cannabis use comes first, rather than people taking it for their symptoms……. It found that cannabis use "significantly" increased the risk of psychotic

symptoms, even when other factors such as socio-economic status, use of different drugs and other psychiatric conditions were taken into account. Sir…. , professor of psychiatric research at the Institute of Psychiatry said the study added "a further brickwall of evidence" showing that traditional use of cannabis is a contributory cause of psychoses like schizophrenia.'

Canberra Times, 17 November 2011, by Christopher Knaus

….. Canberra is the last jurisdiction in Australia to introduce drug driving testing……. ACT Policing's Sergeant … said the laws would improve road safety in the ACT.

Canberra Times, 7 August 2012, Kate Hagan

Teenagers who smoke cannabis weekly are more than twice as likely as non-users to develop an anxiety disorder in their late 20s, even if they stop using the drug, new research has shown. The research, published in the journal *Addiction*, drew on the results of a landmark 15-year study of nearly 2000 Victorian secondary students. An analysis of data collected between 1992 and 2008 found teenagers who smoked cannabis once a week or more for at least six months doubled their risk of having an anxiety disorder for up to a decade afterwards. About 12 per cent of teenagers in the study – or one in eight – smoked cannabis at that level. ….. Lead author Professor … said the study showed cannabis use during adolescence had "a persistent association with anxiety disorders" that continued into adulthood. "Given that anxiety is the most prevalent mental health disorder in the Australian population … we need to investigate the findings further because it is highly possible that that early cannabis use causes enduring mental health risks". Co-author Professor …. said it was possible that cannabis was causing lasting changes to the brain at a time when it was developing rapidly. "During the teen years the parts of the brain that are involved in managing emotions are still developing rapidly and it is highly possible that heavy use of cannabis at this sensitive point could have long-lasting effects."

The Australian 15-16 September 2012 , by Adam Cresswell

…. For …. and some other experts … who believe that loosening legal restrictions would be disastrous, sending a permissive message they believe would only increase consumption, at a time when evidence of cannabis harms is continuing to mount. "What other product would we legalise and put on the market that causes eight cancers and is linked to schizophrenia and bipolar disorder?"

… evidence has strengthened in the past 6 years that it can increase the risk of psychosis schizophrenia and impaired thinking among regular users. …. A review written by Australian drug experts … published in *The Lancet* in 2009 found that the most probable adverse effect of cannabis included dependence syndrome, increased risk of car accidents, breathing difficulties, heart disease and in the case of regular users, impaired psychosocial development and mental health.. Just last week a study published in in US journal *Cancer* found men who had used cannabis faced twice the risk of the more aggressive forms of testicular cancer.

Canberra Times, 17 April 2014, reprinting an article from *The Telegraph*, London

Experimenting with cannabis on a casual basis damages the brain permanently, research has found. It is far from being a 'safe' drug… experts said. People who had used it once or twice a week for a matter of months were found to have changes in the brain that govern emotion, motivation and addiction. Researchers from Harvard Medical School carried out 3D scans on students who used cannabis casually and were not addicted and compared them with those who had never used it. …. Research author .. professor of psychiatry and behavioural sciences … said: "This study raises a strong challenge to the idea that casual marijuana use isn't associated with bad consequences. Some people only used marijuana to get high once or twice a week. … People think a little recreational use shouldn't cause a problem, if someone is doing OK with work or school. Our data directly says that this is not the case."

…. , chief executive of Rethink Mental Illness, said: "For too long cannabis has been seen as a safe drug, but his study suggests it can have a really serious impact on your mental health.. Research also shows that when people smoke cannabis before the age of 15, it quadruples their chance of developing psychosis."

Sydney Morning Herald, 24 August 1998, letter from Athol Moffitt support

… Of course, the legalisation of heroin for addicts would be a step towards the legalisation of drugs more generally. Liberalisation (legalisation, etc) of drugs by amendment ("reform") of the laws is the basic aim of most members of the Australian Drug Law Reform Foundation…

The Telegraph 12 October 1997, by Piers Ackerman

… Harm minimisation advocate … has been among the loudest advocates of such shooting galleries. … is one of the number of Australians who have received grants from international financier George Soros, who wants to see all drugs made available to those who want them. ACT MP …. who led the charge to have Canberra declared the smack capital of Australia is another beneficiary of the erratic financier's largesse…… Media manipulation is another trait of the pro-drug group and the parliamentary committee hearing evidence into the shooting gallery proposal has not been immune. Those witnesses who support the harm minimisation group have been given wide access to the press, those who support abstinence have been quietly presented and permitted to leave without any media attention at all.

Other information about marihuana

The Telegraph 13 June 2000, by Heather Gilmore

New laws introduced in April allow under-18s to escape with a caution if they are found with up to 30g of cannabis, or about 12 joints, for personal use. Adults are allowed to carry 15g of cannabis under the caution system……. The Kings Cross police drug unit is aware that drugs are still sold inside several Roslyn St cafes. ….."…they are obviously cafes frequented by backpackers and tourists who

feel quite comfortable smoking cannabis there", the unit's head Sgt. … said." "(Cannabis) is still a prohibited drug and people are committing a crime by using it, but obviously heroin and powdered drugs like cocaine, are our priority."

4 December 2011 – Sydney Morning Herald Online

… Marijuana is by far the most popular drug: more than 7 million Australians have tried it at some point in their lifetime. Ecstasy is our number-two drug of choice: 2 million of us have tried it at some point have given it a go and another half a million have of us have used the dance drugs GHB and ketamine. By the time Australians turn 20, 37 per cent of us have already tried illicit drugs and that figure rises to a shade less than 60 per cent by the time we turn 40…… Publically the dominant community attitude on drugs in Australia is disapproval and fear, and this feeling seems to be growing. The figures bear that out. The latest Australian Institute of Health and Welfare research shows us as implacably hostile to dealers and suppliers: 80 per cent of us would like even harsher punishment for those caught pushing heroin, cocaine, amphetamines and ecstasy. And support for legalising those drugs doesn't reach double figures…..

Newspaper clipping, date and name of paper not known

Australia has the worst drug problem in the English-speaking world, with marijuana use outstripping even the US a drugs expert claimed yesterday. Justice …., who headed a royal commission into organised crime and drug links in the early 1970s, blasted current governments for the extent of the problem. Justice …. said Australia had lost the chance to be drug free nation in the late 1970s. Talk of legalisation, decriminalisation or relaxing penalties for drug use would not work, he said. "Prohibition has been undermined and degraded. Unless you change the public perception of drug use you can't stop it." (he) said.