IN THE HIGH COURT OF SOUTH AFRICA

(GAUTENG DIVISION - PRETORIA)

CASE NO. 58668/11

In the matter between:

JULIAN CHRISTOPHER STOBBS First

Plaintiff

KATHLEEN (MYRTLE) CLARKE Second Plaintiff

CLIFFORD ALAN NEALE THORP Third Plaintiff

and

NATIONAL DIRECTOR OF PUBLIC PROSECUTION First Defendant

MINISTER OF JUSTICE AND CONSTITUTIONAL DEVELOPMENT Second

Defendant

MINISTER OF HEALTH Third

Defendant

C

MINISTER OF SOCIAL DEVELOPMENT Fourth Defendant

MINISTER OF INTERNATIONAL RELATIONS

AND CORPORATION Fifth

Defendant

MINISTER OF TRADE AND INDUSTRY Sixth Defendant

MINISTER OF POLICE Seventh

Defendant

DOCTORS FOR LIFE INNTERNATIONAL

INCORPORATED Eighth Defendant

NOTICE IN TERMS OF RULE 36 (9) (a) AND (b)

KINDLY TAKE NOTICE THAT at the hearing of the above matter the first to seventh defendants will call the following as an expert witness to give evidence:-

PROFESSOR SHABIR BANOO

TAKE NOTICE FURTHER THAT the report attached hereto represents a summary of the evidence which the said witness will gave at the trial of the matter and the reasons therefor.

DATED AT PRETORIA ON THIS THE 25TH DAY OF JANUARY 2016.

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To: THE REGISTRAR OF THE ABOVE HONOURABLE COURT
PRETORIA

And to: **SCHINDLERS ATTORNEYS**
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Report on Cannabis and its Use in South Africa

Shabir Banoo

Medicines Control Council

Introduction

Globally, cannabis is the most widely consumed illicit drug, with an estimated 4.5 percent of the world's adult population using it.[1] Although cannabis may not be the principal problem drug of abuse in Europe, the Americas, Australia, or Asia, it remains the main problem drug of abuse in Africa.[2] Cannabis is produced in nearly every country worldwide today, and is the most widely produced illicit drug. The highest levels of cannabis herb production – approximately 25% of global production – take place in Africa, particularly in Morocco, South Africa, Lesotho, Swaziland, Malawi, Nigeria, Ghana, Senegal, Gambia, Kenya, and Tanzania.[3]

Cannabis is a genus of flowering plants that includes three different species, *Cannabis sativa*, *Cannabis indica* and *Cannabis ruderalis,* all of which are indigenous to Central and South Asia. The two species that are most commonly grown are *Cannabis sativa* and *Cannabis indica*. *Cannabis ruderalis* produces only trace amounts of psychoactive substances, and thus is not commonly grown. The terms 'dagga', 'marijuana' (and other street names such as 'dope', 'weed', 'blow', 'grass', etc.) are frequently used to refer to preparations of leaves, stalks, flowers and seeds of the plant. The unpollinated female plants are called *hashish.* Cannabis oil is a concentrate obtained by solvent extraction of the crude plant material, or of the resin secreted by the female plant.

Cannabis contains over 400 compounds, including over 60 cannabinoids which are unique to the plant[4]. The pharmacology of most of the cannabinoids is largely unknown but the most potent psychoactive constituent, delta-9-tetrahydrocannabinol (THC), has been isolated, synthesized, and most studied. Other plant cannabinoids include delta-8-THC, cannabinol and cannabidiol (see below). These have additive, synergistic or antagonistic effects with THC and may modify its actions. Non-cannabinoid constituents of the plant are similar to those found in tobacco (with the exception of nicotine) and are associated with similar harms when smoked. Cannabis is generally inhaled by smoking, but may also be

[1] UNODC, Cannabis: A Short Review, 2012
[2] UNODC, World Drug Report, 2000.
[3] UNODC, The Cannabis Market, 2011
[4] Ashton C.H. (2001). Pharmacology and effects of cannabis: a brief review. BJP 2001, 178:101-106

ingested. Peak intoxication through smoking is reached within 15–30 minutes and the effects last for 2–6 hours. Cannabinoids remain in the body for long periods and accumulate after repeated use. Cannabinoids may be found in the urine for 2–3 days after smoking a single cigarette and for up to 6 weeks after the last use in heavy users.

The THC content varies significantly between different sources and preparations of cannabis. Moreover, over the past 20 years sophisticated cultivation methods (such as hydroponic farming) and plant-breeding techniques have greatly increased the potency of cannabis products. This process of selective growing has substantially increased THC content over the years, from 1–3% in the 1970s to 6–13% and higher today.[5] Sinsemilla and Netherwood varieties of cannabis may have a THC content of up to 20%.[6] Thus, current users of cannabis may have very different experiences to those of the past. This view is considered to be important, since the effects of THC are dose-related and most of the research on cannabis was carried out in the 1970s using doses which were much lower (10-fold) than are found in preparations available today.[7]

Several varieties of cannabis, known as hemp, have a very low cannabinoid content, and are instead grown for their fiber and seed.

The Legal Status of Cannabis

Cannabis is classified as a narcotic drug under Schedules I and IV of the 1961 United Nations (UN) Single Convention on Narcotic Drugs, making it subject to special restrictions. South Africa is a signatory to this UN convention and is required to ensure that drug-related activities such as cultivation, production, trade, possession and use are prohibited by law.[8] Article 2 of the 1961 Single Convention provides for the following, in reference to Schedule IV drugs:

"A Party shall, if in its opinion the prevailing conditions in its country render it the most appropriate means of protecting the public health and welfare, prohibit the production, manufacture, export and

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[5] World Health Organisation. (1997). Programme on substance abuse. Cannabis: A health perspective and research agenda. Geneva.

[6] Adams IB, Martin BR. (1996). Cannabis: pharmacology and toxicology in animals and humans. Addiction. 91: 1585-1614.

[7] Gold M.S. (1991). Marijuana. In Comprehensive Handbook on Alcohol and Drug Addiction (ed N.S. Miller), pp 353-376. New York. Marcel Decker.

[8] Single Convention on Narcotic Drugs, 1961 as amended by the 1972 Protocol. United Nations.

import of, trade in, possession or use of any such drug except for amounts which may be necessary for medical and scientific research only, including clinical trials therewith to be conducted under or subject to the direct supervision and control of the Party".

This provision, whilst allowing member countries to determine the most appropriate measures required to protect public health, provides for the limitation of cannabis to medical and scientific research purposes only.

The current legal position in the South Africa is that possession and supply of cannabis is illegal. In South Africa, cannabis is controlled in line with the 1961 Single Convention and is listed as a controlled substance in Schedule 7 of the Schedules to the Medicines and Related Substances Act, 1965 (Act 101 of 1965). Section 22A (9)(a)(i) of the Act provides that no person may acquire, use, possess, manufacture or supply cannabis as the whole plant or any portion or product thereof, and includes synthetic cannabinoids (see below). This section of the Act also makes provision for the Director-General to issue a permit authorising a medical practitioner, analyst, researcher or veterinarian to use cannabis, on the prescribed conditions, for the treatment or prevention of a medical condition in a particular patient, or for the purposes of education, analysis or research.

In terms of the Medicines Act, cannabis may be used as hemp fibre or in a processed product provided that the hemp fibre does not contain more than 0,1 percent THC or in the case of a processed product, not more than 0,001 percent THC and is in a form that does not contain cannabis seeds and is not suitable for ingestion, smoking or inhaling purposes.

The current inscription for cannabis in the Schedules to the Medicines Act reads as follows:

SCHEDULE 7

Preamble:

"All preparations or mixture of such substances containing or purporting to contain substances referred to in this Schedule include the following (unless expressly excluded or unless listed in another Schedule):

(i) the isomers of such substances, where the existence of such isomers is possible within the chemical designation;

(ii) *the esters and ethers of such substances and of the isomers referred to in (i), as well as the isomers of such esters and ethers, where the existence of isomers of such esters, or ethers is possible;*

(iii) *the salts of such substances and of the isomers referred to in (i), as well as the salts of the esters, ethers and isomers referred to in (ii), where the existence of such salts is possible;*

(iv) *the isomers of any of the salts referred to in (iii), where the existence of such isomers is possible;*

(v) *all preparations and mixtures of any of the above.*

(vi) *all homologues of listed substances unless listed in another Schedule*

Schedule 7 inscription for Cannabis:

Cannabis (dagga), the whole plant or any portion or product thereof, except:

a. *when separately specified in the Schedules; (S6) or*
b. *processed hemp fibre containing 0.1 percent or less of tetrahydrocannabinol and products manufactured from such fibre, provided that the product does not contain whole cannabis seeds and is in a form not suitable for ingestion, smoking or inhaling purposes; or*

c. processed product made from cannabis seeds containing not more than 10 milligram per kilogram (0,001 percent) of tetrahydrocannabinol and does not contain whole cannabis seeds."

["Processed" means treated by mechanical, chemical or other artificial means but does not include- (a) harvesting; or (b) the natural process of decay"].

Additionally, Schedule 7 also contains the following inscription relating to synthetic cannabinoids:

"Synthetic cannabis substances (synthetic cannabinoids) included but not limited to:

- *cannabicyclohexanol;*
- *JWH-018; JWH-073; JWH-200; CP-47497; CP 47497-C6; CP 47497-C7; CP 47497-C8; CP 47497-C9; HU-210"*

In its 1994 annual report, the International Narcotic Control Board (INCB) recommended that member countries establish licensing and registration regulations, define the control systems for hemp cultivation and also properly define which cannabis varieties are authorised for cultivation. Further, it is

worth noting that the INCB continues to oppose cannabis reform at the international level which refers to efforts to ease restrictions on cannabis use under international treaties.

The Pharmacology and Biochemistry of Cannabis

As mentioned above, the most prevalent psychoactive substances in cannabis are cannabinoids, most notably THC. Some varieties, having undergone careful selection and growing techniques, can yield as much as 29% THC.[9] Another psychoactive cannabinoid present in *Cannabis sativa* is tetrahydrocannabivarin (THCV), but it is only found in small amounts and is a cannabinoid antagonist.[10] In addition, there are also similar compounds contained in cannabis that do not exhibit any psychoactive properties and include cannabidiol (CBD), cannabinol (CBN), cannabivarin (CBV), cannabidivarin (CBDV), and cannabinolic acid. How these other compounds interact with THC is not fully understood. Studies suggest that CBD may alter the metabolism of THC by inactivating cytochrome P450, an important class of enzymes that metabolize drugs, resulting in a substantial increase in brain concentrations of THC and its major metabolites.[11] Most cannabinoids are lipophilic (fat soluble) compounds that are easily stored in fat, thus yielding a long elimination half-life relative to other recreational drugs. Additionally, THC is rapidly converted to 11-hydroxy-THC, which is also pharmacologically active, resulting in the drug effect outlasting measurable THC levels in blood. Cannabis also contains many terpenoids compounds which may synergize with the cannabinoids to produce their unique effects.

Cannabinoids act on specific receptors that are widely distributed in the brain regions involved in cognition, memory reward, pain perception, and motor coordination. These receptors respond to an endogenous ligand, anandamide, which is much less potent and has a shorter duration than THC. There are two main types of cannabinoid receptors, namely CB1 and CB2 receptors.

The CB1 receptor, is highly abundant in the brain and is responsible for mediating the psychoactive effects of THC. The CB2 receptor is structurally different and is found only on cells of the immune system. Cannabinoids act as immunomodulators at CB2 receptors, meaning that they increase some immune responses and decrease others.[8] THC and endogenous anandamide additionally interact with

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[9] H.K. Kalant & W.H.E. Roschlau (1998). Principles of Medical Pharmacology (6th ed.). pp. 373–375

[10] Turner, C.E.; Bouwsma, et al. (1980). Constituents of Cannabis sativa – Electron voltage selected ion monitoring study of cannabinoids. Biological Mass Spectrometry 7 (6): 247–56

[11] J.E. Joy, S. J. Watson, Jr., and J.A. Benson, Jr, (1999). Marijuana and Medicine: Assessing the Science Base. Washington D.C: National Academy of Sciences Press. ISBN 0-585-05800

glycine receptors. There is also evidence that cannabinoids can affect pain transmission through an interaction with the opioid and dopamine systems in the brain.[12]

When smoked, the effects of cannabis manifest within seconds and are fully apparent within a few minutes, typically lasting for 1–3 hours, varying by the person and the strain of cannabis.[2] After oral ingestion of cannabis, the onset of effect is delayed relative to smoking, taking between 30 minutes to 2 hours, but the duration is prolonged due to continued slow absorption. The duration of noticeable effects has been observed to diminish due to prolonged, repeated use and the development of a tolerance to cannabinoids[2].

While there are similarities between cannabis and tobacco smoke, recent evidence also shows that cannabis smoke contains higher amounts of ammonia, hydrogen cyanide, and nitrogen oxides, but lower levels of polycyclic aromatic hydrocarbons.[13] Directly inhaled cannabis smoke contains 20 times as much ammonia and 5 times as much hydrogen cyanide as tobacco smoke. Cannabis smoke also contains thousands of organic and inorganic chemical compounds. Additionally, over fifty known carcinogens have been identified in cannabis smoke.[14] These include nitrosamines, reactive aldehydes, and polycylic hydrocarbons, including benz[a]pyrene.

Synthetic Cannabinoids

Synthetic cannabinoids are a heterogeneous group of compounds developed to probe the endogenous cannabinoid system or as potential therapeutic agents and have been widely published. However, illicit laboratories subsequently utilized this published data to develop synthetic cannabinoid variations marketed as abusable designer drugs.[15] In the early 2000s, these became popular as "legal highs" under brand names such as Spice and K2, in part due to their ability to escape detection by standard cannabinoid screening tests. The majority of synthetic cannabinoid detected in herbal products have greater binding affinity to the cannabinoid CB1 receptor than does THC and greater affinity at the CB1 than the CB2 receptor. *In vitro* and animal *in vivo* studies show their pharmacological effects to be 2-100 times more potent than THC, including analgesic, anti-seizure, weight-loss, anti-inflammatory, and anti-

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[12] H. Abadinsky (2004). Drugs: An Introduction (5th ed.). pp. 62–77; 160–166. ISBN 0-534-52750-7

[13] Moir, D., Rickert, W.S., et al. (2008). A Comparison of Mainstream and Sidestream Marijuana and Tobacco Cigarette Smoke Produced under Two Machine Smoking Conditions. Chemical Research in Toxicology 21 (2): 494–502.

[14] Does smoking cannabis cause cancer? Cancer Research UK. 2010.

[15] Castanetoa M.S, Gorelickc D.A, et al. (2014) Synthetic cannabinoids: Epidemiology, pharmacodynamics, and clinical implications. Drug Alcohol Depend. pii: S0376-8716(14)01033-3.

cancer growth effects. Synthetic cannabinoid produce physiological and psychoactive effects similar to THC, but with greater intensity, resulting in medical and psychiatric emergencies[10]. Human adverse effects include nausea and vomiting, shortness of breath or depressed breathing, hypertension, tachycardia, chest pain, muscle twitches, acute renal failure, anxiety, agitation, psychosis, suicidal ideation, and cognitive impairment. Long-term or residual effects are, as yet, unknown. Due to these public health consequences, many synthetic cannabinoids are classified as controlled substances. However, frequent structural modification by illicit laboratories continue to result in a stream of novel synthetic cannabinoids that may not be legally controlled in all countries or detectable by routine laboratory tests.

In South Africa, the inscription for cannabis in Schedule 7 of the Medicines Act (Act 101 of 1965) has been updated to include all chemically related derivatives of THC and synthetic cannabinoids.

Patterns of Cannabis Use Globally and in South Africa

In many countries, cannabis use increased during the 1990s and early 2000s, but is now generally stabilizing. Rates of use, however, are not low and it is estimated that between 2.8% and 4.5% of the world population aged 15-64 used cannabis at least once during the past year in 2009.[1] The annual prevalence of cannabis use in North America is approximately 10.7% of the population aged 15-64, and use among the youth has risen over the past four years. Africa has the third highest cannabis prevalence rate in the world, after the Oceania region and North America, with estimates ranging from 3.8% to 10.4% of the population.[1]

In South Africa, cannabis remains the most common illicit drug used, especially among youth attending specialist treatment centres. Additionally, cannabis is the most common primary substances of abuse for patients younger than 20 years, with up to 78% of patients being Black.[16] Additionally, treatment admissions with cannabis as the primary drug of abuse has increased significantly in almost all provinces and regions in the country. Cannabis is the illicit drug most likely to be consumed by high-school students and is commonly used by young rave club attendees. Although many young people do not perceive cannabis to be a problem, the South African Community Epidemiology Network on Drug Use (SACENDU) adolescent treatment demand, trauma, and arrestee data clearly reflect the burden that cannabis use has to the health, social welfare, and criminal justice systems in South Africa.[16]

[16] SACENDU Report. Phase 34. February 2014.

Peltzer and Ramlagan recently reviewed cannabis use trends (over a period of 12 years) in the South African population, by sourcing data from surveys, specialised alcohol and drug treatment centres, cannabis-related trauma unit admissions, and arrestee studies.[17] They concluded that cannabis was the most common illicit substance used, with current self-reported cannabis use of 5 - 10% among adolescents and 2% among adults. Furthermore, it was higher among men than women, higher in urban than rural areas, higher in the urban provinces of Western Cape and Gauteng than the other provinces, and higher among coloureds and whites than other racial groups.

Heavy cannabis use is generally defined as daily or near daily use and this pattern of use over years places users at greatest risk of adverse health and psychological consequences. Daily cannabis users are more likely to be male, to be less well educated, to use alcohol and tobacco regularly, and to use amphetamines, hallucinogens, psychostimulants, sedatives, and opioids.

Combination of Cannabis with other Drugs

An obvious confounding factor in cannabis research is the concomitant use of other recreational drugs, especially alcohol and tobacco.[18] Such complications demonstrate the need for studies on cannabis that have stronger controls, and investigations into effects of cannabis that may also be caused by other drugs. The Australian National Household Survey of 2001[19] showed that cannabis is rarely used without other drugs: 95% of cannabis users also drank alcohol; 26% took amphetamines; 19% took ecstasy and only 2.7% reported not having used any other drug with cannabis. Evidence also suggests that alcohol causes THC to be absorbed more rapidly into the blood plasma of the user.[20] Of interest, data from the Australian National Survey of Mental Health and Wellbeing found that three-quarters of recent cannabis users reported using alcohol when cannabis was not available.[21]

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[17] Peltzer K, Ramlagan S. (2007). Cannabis use trends in South Africa. South African Journal of Psychiatry. 13(4): 126-131.

[18] Zhang Z, Morgenstern H, et al. (1999). Marijuana use and increased risk of squamous cell carcinoma of the head and neck. Cancer Epidemiology, Biomarkers and Prevention 8 (12): 1071–8.

[19] National Drug Strategy Household Survey (2001). Australian Institute of Health and Welfare.

[20] Lukas SE, Orozco S. (2001). Ethanol increases plasma Δ9-tetrahydrocannabinol (THC) levels and subjective effects after marihuana smoking in human volunteers. Drug and Alcohol Dependence 64 (2): 143–9.

[21] Hall L, Degenhardt W. (2001). The relationship between tobacco use, substance-use disorders and mental health: results from the National Survey of Mental Health and Well-being. Nicotine & Tobacco Research 3 (3): 225–34.

In South Africa, cannabis is often mixed with other substances including methaqualone[22] (Mandrax), heroin, cocaine and the antiretroviral (ARV) agent efavirenz[23]. In addition, mixtures having street names such as 'Nyaope', 'Sugars', 'Whoonga', etc., contain varying amounts of cheap heroin and cocaine, and are usually smoked with cannabis. These street concoctions often also contain other substances such as rat poison, cleaning detergents, efavirenz, methamphetamine and other illicit drugs. Street use of the ARV efavirenz may exploit its well-known central nervous system effects which may enhance the effects of cannabis, heroin, methamphetamine, and other illicit drugs. Recent data suggest that an increasing number of young people are presenting for assistance at treatment centres relating to abuse of 'Nyaope', 'Sugars' and 'Whoonga'. A total of 1537 patients were treated across the seven treatment centres during the second half of 2008 alone.[24]

Cannabis smoked with methaqualone is known by the street name 'white pipe' in South Africa and is associated with a number of negative health outcomes, including dependence.[25] Methaqualone users, in particular, have a high risk of becoming physically and psychologically dependent on the drug.[26] In addition, the frequent and prolonged use of cannabis in this population of users may lead to psychological dependence, tolerance, and withdrawal symptoms on cessation of use.[27] The prolonged use of cannabis and white pipes may also lead to respiratory problems, including pre-cancerous changes in lung tissue, and significantly more abnormalities in the pulmonary bronchi, respiratory illnesses, and symptoms of chronic bronchitis than occur among non-users.[28]

Acute Effects of Cannabis

The psychoactive effects of cannabis, known as a "high", are subjective and can vary based on the person and the method of use. Cannabis produces euphoria and relaxation, perceptual alterations, time distortion, and the intensification of ordinary sensory experiences, such as eating and listening to music.[6] When used in a social setting it may produce infectious laughter and talkativeness. Short-term

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[22] de Miranda S. Drugs and Drug Abuse in Southern Africa. Pretoria: JL Van Schaik. 1987: 32-35.
[23] Larkin F, van Wyk B, et al. (2010). Of Remedies and Poisons: Recreational Use of Antiretroviral Drugs in the Social Imagination of South African Carers. African Sociological Review 14(2): 62-73
[24] SACENDU Research Brief Vol 12(1) 2009.
[25] Bhana A, Parry CDH, et al. (2002). The South African Community Epidemiology Network on Drug Use (SACENDU) Project, Phases 1-8 – Cannabis and Mandrax. SAMJ. 92(7): 542-547.
[26] Faught E. (1986). Methaqualone withdrawal syndrome with photo-paroxysmal responses and high amplitude visual evoked potentials. Neurology. 36: 1127-1129.
[27] Budney AI, Navy PL, Hughes JR. (1999).Marijuana withdrawal among adults seeking treatment for marijuana dependence. Addiction. 94: 1311-1322.
[28] Hall W. (1998). The respiratory risks of cannabis smoking. Addiction. 93: 1461-1463.

memory and attention, motor skills, reaction time, and skilled activities are impaired while a person is intoxicated.

The most common unpleasant side-effects of occasional cannabis use are anxiety and panic reactions. These effects may be reported by naïve users, and they are a common reason for discontinuation of use; more experienced users may occasionally report these effects after receiving a much larger than usual dose of THC.[6]

Cannabis smoking or ingestion of THC increases heart rate by 20—50% within a few minutes and may last for up to 3 hours. Blood pressure is increased while the person is sitting, and decreased while standing. These effects are of negligible clinical significance in healthy young users because tolerance develops to them. Other effects may also include dry mouth, reddening of the eyes and a reduction in intra-ocular pressure.[6]

At higher doses, effects can include acute psychosis, auditory and/or visual illusions, pseudo-hallucinatory responses, and ataxia from selective impairment of reflexes. In some cases, cannabis can lead to dissociative states such as depersonalization and derealization.[29] Acute episodes of psychosis usually abate after 6-8 hours, but in rare instances, may continue for days, particularly in heavy users.[30] While many psychoactive drugs clearly fall into the category of either stimulant, depressant, or hallucinogen, cannabis exhibits a mix of all properties. Concerns have been raised about the potential for long-term cannabis consumption to increase risk for schizophrenia, depersonalization disorder, bipolar disorders, and major depression.

Neurological Effects of Cannabis

Brain regions where cannabinoid receptors are most prevalent are believed to be involved with the behavioral effects produced by cannabis[31]. These include the *basal ganglia*, associated with movement control; the *cerebellum*, associated with body movement coordination; the *hippocampus*, associated with learning, memory, and stress control; the *cerebral cortex*, associated with higher cognitive

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[29] Johnson, B.A. (1990). Psychopharmacological effects of cannabis. British journal of hospital medicine 43 (2): 114–6, 118–20, 122.

[30] Barceloux, D.G. (2012). Marijuana (Cannabis sativa L.) and synthetic cannabinoids. Medical Toxicology of Drug Abuse: Synthesized Chemicals and Psychoactive Plants. John Wiley & Sons. p. 915. ISBN 978-0-471-72760-6.

[31] Pertwee, R. (1997) Pharmacology of cannabinoid CB1 and CB2 receptors. Pharmacology & Therapeutics 74 (2): 129–80.

functions; and the *nucleus accumbens*, regarded as the reward center of the brain. Other regions where cannabinoid receptors are moderately concentrated are the *hypothalamus*, which regulates homeostatic functions; the *amygdala*, associated with emotional responses and fears; the *spinal cord*, associated with peripheral sensations like pain; the brain stem, associated with sleep, arousal, and motor control; and the *nucleus of the solitary tract*, associated with visceral sensations like nausea and vomiting.[16] Cannabinoids, depending on the dose, inhibit the transmission of neural signals through the basal ganglia and cerebellum. At lower doses, cannabinoids seem to stimulate locomotor activity, while greater doses inhibit it, most commonly manifested by lack of steadiness (body sway and hand steadiness) in motor tasks that require a lot of attention. Other brain regions are also involved in the control of movement and contain abundant cannabinoid receptors, suggesting their possible involvement as well.[16]

Effects of Cannabis on Driving

Cannabis usage has been shown to have a negative effect on driving ability[32] with acute cannabis consumption being associated with an increased risk of motor vehicle accidents, particularly fatal collisions.[18] This study found that drivers who consume cannabis within three hours of driving are nearly twice as likely to cause a vehicle collision as those who are not under the influence of drugs or alcohol.[33] Kelly et al[34] show similar results, with laboratory studies examining the effects of cannabis on skills utilised while driving showing impairments in tracking, attention, reaction time, short-term memory, hand-eye coordination, vigilance, time and distance perception, and decision making and concentration.

A review conducted by the European Monitoring Centre for Drugs and Drug Abuse concluded that the acute effect of moderate or higher doses of cannabis impairs the skills related to safe driving and injury risk, specifically attention, tracking and psychomotor skills.[35] In their review of driving simulator studies, Kelly et al conclude that there is evidence of dose-dependent impairments in cannabis-affected drivers' ability to control a vehicle in the areas of steering, headway control, speed variability, car following,

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[32] Li MC, Brady JE, et al. (2012). Marijuana use and motor vehicle crashes. Epidemiol Rev. 34(1):65-72.) 34 (1): 65–72.

[33] Asbridge M, Hayden JA, Cartwright JL. (2012). Acute cannabis consumption and motor vehicle collision risk: systematic review of observational studies and meta-analysis. BMJ. 2012 Feb 9;344:e536.

[34] Kelly E; Darke S; Ross J. (2004). A review of drug use and driving: epidemiology, impairment, risk factors and risk perceptions. Drug and Alcohol Review 23 (3): 319–44.

[35] Sznitman SR, Olsson B, Room R, eds. (2008). A cannabis reader: global issues and local experiences 2. Lisbon: European Monitoring Centre for Drugs and Drug Addiction. ISBN 978-92-9168-312-3.

reaction time and lane positioning.[19] The researchers note that "even in those who learn to compensate for a drug's impairing effects, substantial impairment in performance can still be observed under conditions of general task performance.

A recent meta-analysis of crash risk and drug use found that cannabis was associated with minor, but not statistically significant increased odds of injury or fatal accident.[36] The study concluded that "*...the increase in the risk of accident involvement associated with the use of drugs must be regarded as modest...Compared to the huge increase in accident risk associated with alcohol, as well as the high accident rate among young drivers, the increases in risk associated with the use of drugs are surprisingly small.*"[21]

Effects on the Respiratory System

Chronic heavy cannabis smoking is associated with increased symptoms of chronic bronchitis, such as coughing, production of sputum, and wheezing.[37] Lung function is significantly poorer and there are significantly greater abnormalities in the large airways of marijuana smokers than in non-smokers. Tashkin and colleagues have reported evidence of an additive effect of marijuana and tobacco smoking on histopathological abnormalities in lung tissue.[27] Similar additive effects on bronchitic symptoms were reported in an epidemiological study of the respiratory effects of smoking "non-tobacco" cigarettes in 990 individuals aged under 40 years in the USA.[38] Non-tobacco smokers reported more coughing, phlegm production, and wheeze than non-smokers, irrespective of whether they also smoked tobacco. Those who had never smoked any substance had the best respiratory functioning, followed in order of decreasing function by current tobacco smokers, current non-tobacco smokers, and current smokers of both tobacco and non-tobacco cigarettes. Non-tobacco smoking alone had a larger effect on respiratory function than tobacco smoking alone, and the effect of both types of smoking was additive. A follow-up of this cohort,[39] showed a greater rate of decline in respiratory function among marijuana-only smokers

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[36] Elvik, R (2013). Risk of road accident associated with the use of drugs: a systematic review and meta-analysis of evidence from epidemiological studies. Accident Analysis and Prevention 60: 254–67.

[37] Tashkin DP, Fligiel S, et al. (1990). Effects of habitual use of marijuana and/or cocaine on the lung. In: Chiang CN, Hawks RL, eds. Research findings on smoking of abused substances. National Institute on Drug Abuse Research Monograph 99. Rockville, Maryland.

[38] Bloom JW, Kaltenborn WT, et al. (1987). Respiratory effects of non-tobacco cigarettes. BMJ 295: 1516-1518.

[39] Sherrill DL, Krzyzanowski JW, et al. (1991) Respiratory effects of non-tobacco cigarettes: a longitudinal study in general population. Int J Epidemiol. 20: 132-137.

than in tobacco-only smokers and additive effects of tobacco and marijuana smoking. The study also showed that long-term cannabis smoking increased bronchitic symptoms.

In view of the adverse effects of tobacco smoking, the similarity between tobacco and cannabis smoke, and the evidence that cannabis smoking produces histopathological changes that precede lung cancer,[40] long-term cannabis smoking may also increase the risks of respiratory cancer. There have been reports of cancers in the aerodigestive tract in young adults with a history of heavy cannabis use.[41] These reports are worrying since such cancers are rare among adults under the age of 60, even those who smoke tobacco and drink alcohol.

Cardiovascular Effects of Cannabis

Short term effects on the cardiovascular system can include increased heart rate, dilation of blood vessels, and fluctuations in blood pressure.[42] The cardiovascular effects of cannabis are not associated with serious health problems for most young, healthy users. Cannabis use by older people, particularly those with some degree of coronary artery or cerebrovascular disease, may pose greater risks.[43]

Reproductive Effects of Cannabis

Chronic administration of high doses of THC to animals lowers testosterone secretion, impairs sperm production, motility, and viability, and disrupts the ovulatory cycle.[44] Whether cannabis smoking has these effects in human beings is uncertain since the published evidence is small and inconsistent.

Cannabis use in pregnancy is associated with restrictions in growth of the fetus, miscarriage, and cognitive deficits in offspring.[45] A recent systematic review of substance exposure *in-utero* and developmental outcomes in adolescence found some evidence that prenatal exposure to cannabis was

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[40] Fligiel SEG, Roth MD,et al. (1997). Tracheobronchial histopathology in habitual smokers of cocaine, marijuana and/or tobacco. Chest. 112: 319-326.

[41] Sridar KS, Raub WA, et al. (1994). Possible role of marijuana smoking as a carcinogen in the development of lung cancer at an early age. J Psychoactive Drugs. 26: 285-288.

[42] Jones RT. (2002). Cardiovascular system effects of marijuana. Journal of Clinical Pharmacology 42 (11): 58–63.

[43] Aranya A, Williams M. (2007). Marijuana as a trigger of cardiovascular events: Speculation or scientific certainty? International Journal of Cardiology 118 (2): 141–147.

[44] Bloch E. (1983). Effects of marijuana and cannabinoids on reproduction, endocrine function, development and chromosomes. In: Fehr KO, Kalant H, eds. Cannabis and health hazards. Toronto: Addiction Research Foundation.

[45] Fonseca BM, Correia-da-Silva G, et al. (2013). The Endocannabinoid System in the Postimplantation Period: A Role during Decidualization and Placentation. Int J Endocrinol (Review) 510-540.

associated with deficits in language, attention, areas of cognitive performance, and delinquent behavior in adolescence.[46]

Furthermore, three studies have shown an increased risk of non-lymphoblastic leukaemia,[47] rhabdomyosarcoma,[48] and astrocytoma[49] in children whose mothers reported using cannabis during their pregnancies. None of these was a planned study of the association and cannabis use was one of many potential confounders included in statistical analyses of the relation between the exposure of interest and childhood cancer. However, replication of these findings is seen as a priority.

Behavioural Effects of Cannabis in Adolescence

There is a cross-sectional association between heavy cannabis use in adolescence and the risk of leaving high-school education and of experiencing job instability in young adulthood.[50] However, the strength of this association may be reduced in longitudinal studies when statistical adjustments are made for the fact that, compared with their peers, heavy cannabis users have poor high-school performance before using cannabis. There is some evidence that heavy use has adverse effects on family formation, mental health, and involvement in drug-related crime.[49] In each case, the strong associations in cross-sectional studies are more modest in longitudinal studies after statistical control for associations between cannabis use and other pre-existing characteristics that independently predict these adverse outcomes.

A recent study investigating the association between the maximum frequency of cannabis use before age 17 years and seven developmental outcomes assessed up to age 30 years, recorded clear and consistent associations and dose-response relations between the frequency of adolescent cannabis use and all adverse young adult outcomes.[51] Compared with individuals who had never used cannabis, those who were daily users before age 17 years had clear reductions in the odds of high school completion, degree attainment, and substantially increased odds of later cannabis dependence, use of other illicit drugs, and suicide attempt.[50]

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[46] Irner, TB. (2012). Substance exposure in utero and developmental consequences in adolescence: A systematic review. Child Neuropsychology (Review) 18 (6): 521–49.

[47] Robison LI, Buckley JD, et al. (1989). Maternal drug use and the risk of childhood nonlympholastic leukemia among offspring: an epidemiologic investigation implicating marijuana. Cancer. 63: 1904-1911.

[48] Grufferman S, Schwartz AG, et al. (1993). Parent's use of cocaine and marijuana and increased risk of rhabdomyosarcoma in their children. Cancer, Causes & Control. 4: 217-224.

[49] Kuitjen RR, Bunin GR, et al. (1992). Parental occupation and childhood astrocytoma. Cancer Res. 52: 782-786.

[50] Newcombe T, Bentler P. (1988). Consequences of adolescent drug use: impact on the lives of young adults. Newbury Park, California: Sage Publications.

[51] Silins E, Horwood LJ. (2014). Young adult sequelae of adolescent cannabis use: an integrative analysis. Lancet Psychiatry. 1: 286–93.

A consistent finding in the USA has been the regular sequence of initiation into drug use in which cannabis use has typically preceded involvement with "harder" illicit drugs such as stimulants and opioids. The interpretation of this sequence remains controversial. It appears, however, that there may be a selective recruitment into cannabis use of non-conforming adolescents who have a propensity to use other illicit drugs, and that once recruited to cannabis use, social interaction with drug-using peers, and greater access to illicit-drug markets, they are more likely to use other illicit drugs.[52]

Dependence Syndrome

Animals develop tolerance to the effects of repeated doses of THC and studies suggest that cannabinoids may affect the same reward systems as alcohol, cocaine, and opioids.[53] Heavy smokers of cannabis also develop tolerance to its subjective and cardiovascular effects and some report withdrawal symptoms on the abrupt cessation of cannabis use.[54] There is evidence that a cannabis dependence syndrome occurs with heavy chronic use in individuals who report problems in controlling their use and who continue to use the drug despite experiencing adverse personal consequences.[55] There is some clinical evidence of a dependence syndrome analogous to that for alcohol. In the USA, cannabis dependence is among the most common forms of illicit-drug dependence in the population. About one in ten of those who ever use cannabis become dependent on it at some time during their 4 or 5 years of heaviest use. This risk is more like the equivalent risk for alcohol (15%) than for nicotine (32%) or opioids (23%).[56]

Cannabis Use and Psychosis

Substance abuse and/or dependence are common problems among the general population, and evidence indicates that the incidence is even higher in individuals with mental health problems. Experimental studies and surveys of users provide strong evidence that cannabis intoxication can

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[52] Fergusson D, Horwood J. (1997). Early onset cannabis use and psychosocial adjustment in young adults. Addiction. 92: 279-296.

[53] Wickelgren I. (1997). Marijuana: harder than thought? Science. 276: 1967-1968.

[54] Weisbeck GA, Schuckit MA, et al. (1996). An evaluation of the history of marijuana withdrawal syndrome in a large population. Addiction. 91: 1469-1478.

[55] Stephens RS, Roffman RA, et al. (1993). Adult marijuana users seeking treatment. J Consult Clin Psychol. 61: 1110-1204.

[56] Anthony JC, Warner LA, et al. (1994). Comparative epidemiology of dependence on tobacco, alcohol, controlled substances and inhabitants: findings from the National Comorbidity Study. Clin Exp Psychopharmacol. 2: 244-268.

produce transient, and usually mild, psychotic and affective experiences.[57] In their systematic review, Theresa Moore and colleagues found "an increase in risk of psychosis of about 40% in participants who had ever used cannabis", and a clear dose-response effect with an increased risk of 50–200% in the most frequent users.[58] Additionally, a number of prospective studies also provide support for the hypothesis that cannabis abuse increases the subsequent risk for developing schizophrenia.[59] These findings also suggest that cannabis use can exacerbate the symptoms of schizophrenia or may precipitate schizophrenia in vulnerable individuals.

Koen et al,[60] in a study aimed at identifying the demographic and clinical correlates of cannabis abuse in a large South African schizophrenia group, found the prevalence of cannabis use/abuse in this population to be high, consistent with findings of previous international studies. Abuse/use started mainly in the teenage years, was more prevalent among males than females, and was associated with negative overall outcomes.

Conclusion

The available data on cannabis use presented in this report clearly highlights the adverse sequelae of cannabis use, particularly in adolescents. These effects are wide ranging and its use is linked to addiction, cognitive impairment, motor skills deficiency, respiratory, cardiovascular and mental health problems.

Additionally, recent emerging evidence showing an increased risk of adverse developmental outcomes in adolescents, is of particular concern. Both local and international experience confirm cannabis as being the most commonly abused drug among adolescent treatment seekers.

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[57] D'Souza DC, Perry E, et al. (2004). The psychotomimetic effects of intravenous delta-9-tetrahydrocannabinol in healthy individuals: implications for psychosis. Neuropsychopharmacology 29: 1558–72.

[58] Moore THM, Zammit S, et al. (2007). Cannabis use and risk of psychotic or affective mental health outcomes: a systematic review. Lancet. 370: 319-328.

[59] Arseneault L, Cannon M, et al. (2004). Causal association between cannabis and psychosis: Examination of the evidence. Br J Psychiatry. 184: 110-117.

[60] Koen L, Jonathan R, et al. (2009). Cannabis use and abuse correlates in a homogeneous South African schizophrenia population. South African Journal of Psychiatry. 15 (1): 8-12.

Prevention of cannabis abuse therefore remains a necessity and a public health priority. This will contribute to achieving broad health and social benefits through the concurrent implementation of evidence-based prevention and treatment strategies to effectively reduce use, abuse and addiction.

Whilst cannabinoids have been shown to be useful in a few medical indications, research in this area is ongoing and its use for this purpose is already enabled in current legal provisions.