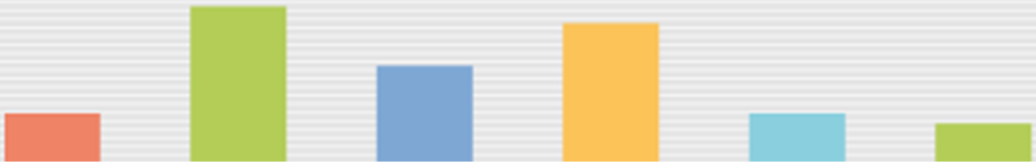
European Monitoring Centre
for Drugs and Drug Addiction

Cannabis policy
history, legacy and evidence



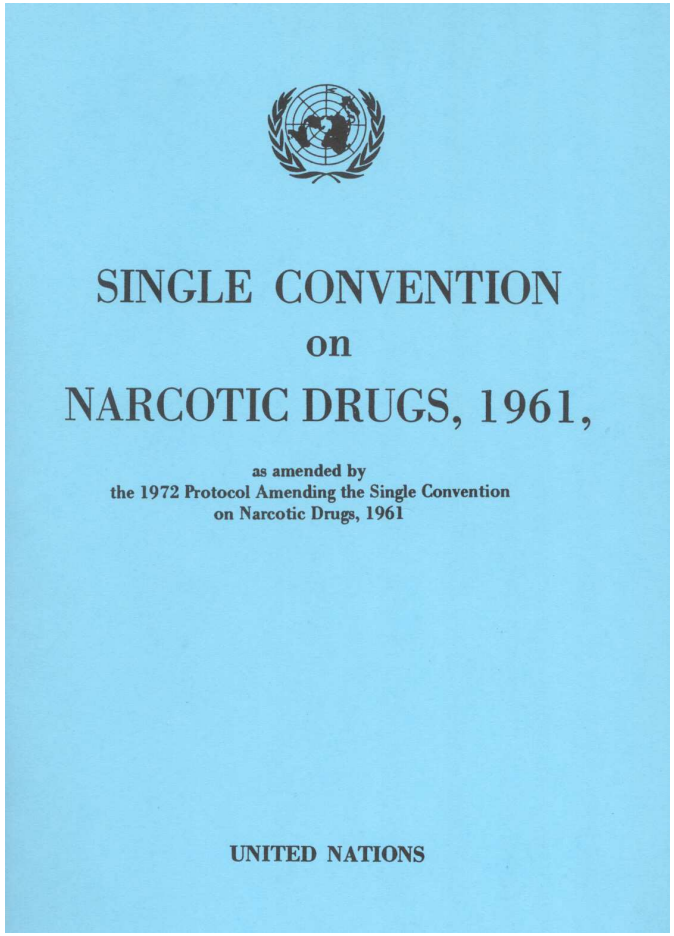
Origins of cannabis control

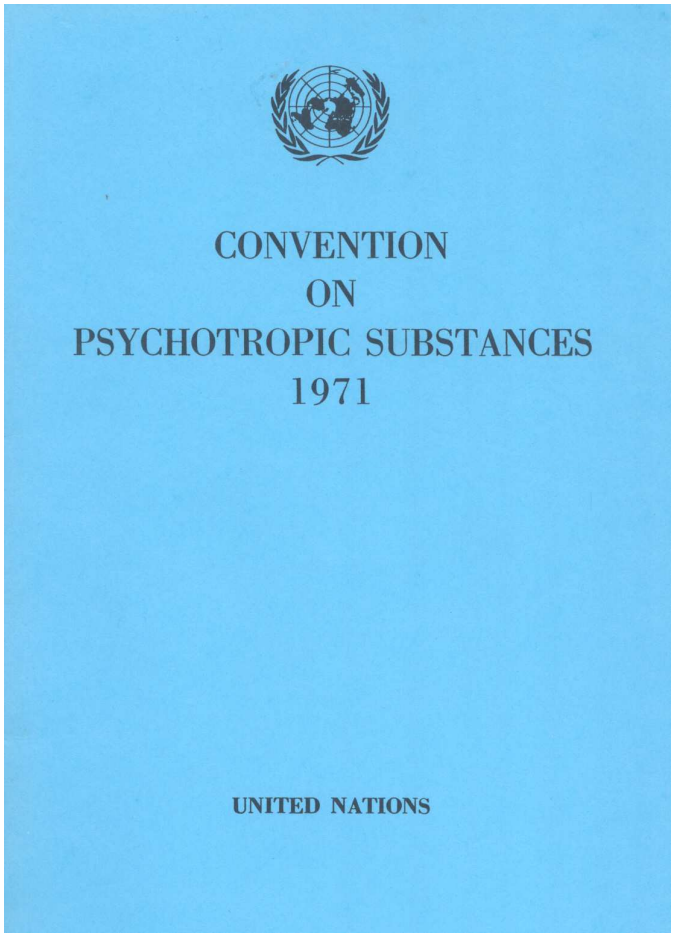
1925 Geneva Convention on Opium

1930s prohibitionist movement

US influence in the UN diplomacy after world war II

Cannabis
Classified as one of the most dangerous substances



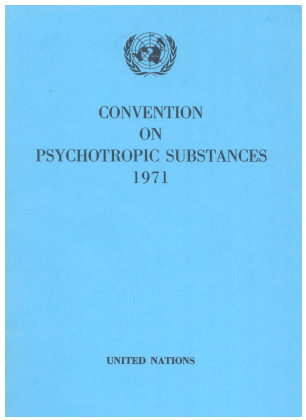


UN Single Convention 1961

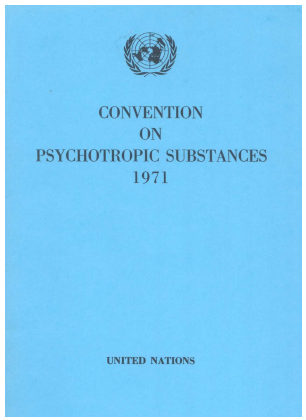
Schedule I	Schedule II	Schedule III	Schedule IV
Acetorphine AcetorphineAcetyl-alpha-methylfentanyl Alphaprodine Betacetylmethadol Beta-hydroxy-3-methylfentanyl **Cannabis and Cannabis resin** Coca leaf Cocaine Desomorphine Dipipanone Fentanyl Etorphine Heroin Metazocine Methadone Morphine Opium Oxycodone	Acetyldihydrocodein Codeine Dextropropoxyphen Dihydrocodeine Ethylmorphin Nicocodine Nicodicodine Norcodeine Pholcodine Propiram	Preparations of Acetyldihydrocodein Codeine, DihydrocodeineEthylmorphine, Nicodicodine, Norcodeine, Pholcodine Preparations of Propiram Preparations of Cocaine	AcetorphineAcetyl-alpha-methylfentanyl Beta-hydroxy-3-methylfentanyl Beta-hydroxyfentan **Cannabis and Cannabis resin** Desomorphine Etorphine Heroin Ketobemidone 3-methylfentanyl 3-methylthiofentanyl MPPP Para-fluorofentanyl PEPAPThiofentanyl

List of Substances in the Schedules

Substances in Schedule I



International non-proprietary name (INN)	Other non-proprietary or trivial name	Chemical name
BROLAMFETAMINE	DOB	(±)-4-bromo-2,5-dimethoxy-*alpha*-methylphenethylamine
CATHINONE		(x)-(*S*)-2-aminopropiophenone
Not available	DET	3-[2-(diethylamino)ethyl]indole
Not available	DMA	(±)-2,5-dimethoxy-*alpha*-methylphenethylamine
Not available	DMHP	3-(1,2-dimethylheptyl)-7,8,9,10-tetrahydro-6,6,9-trimethyl-6*H*-dibenzo[*b*,*d*]pyran-1-olo
Not available	DMT	3-[2-(dimethylamino)ethyl]indole
Not available	DOET	(±)-4-ethyl-2,5-dimethoxy-*alpha*-phenethylamine
ETICYCLIDINE	PCE	*N*-ethyl-1-phenylcyclohexylamine
ETRYPTAMINE		3-(2-aminobutyl)indole
(+)-LYSERGIDE	LSD, LSD-25	9,10-didehydro-*N*,*N*-diethyl-6-methylergoline-8*beta*-carboxamide
Not available	MDMA	(±)-*N*,*alpha*-dimethyl-3,4-(methylene-dioxy)phenethylamine
Not available	mescaline	3,4,5-trimethoxyphenethylamine
	methcathinone	2-(methylamino)-1-phenylpropan-1-one
Not available	4-methylaminorex	(±)-*cis*-2-amino-4-methyl-5-phenyl-2-oxazoline
Not available	MMDA	2-methoxy-*alpha*-methyl-4,5-(methylenedioxy)phenethylamine
Not available	N-ethyl MDA	(±)-N-ethyl-*alpha*-methyl-3,4-(methylenedioxy)phenethylamine
Not available	*N*-hydroxy MDA	(±)-*N*-[*alpha*-methyl-3,4-(methylenedioxy)phenethyl]hydroxylamine
Not available	parahexyl	3-hexyl-7,8,9,10-tetrahydro-6,6,9-trimethyl-6*H*-dibenzo[*b*,*d*]pyran-1-ol
Not available	PMA	*p*-methoxy-*alpha*-methylphenethylamine



Not available	N-ethyl MDA	(±)-N-ethyl-*alpha*-methyl-3,4-(methylenedioxy)phenethylamine
Not available	*N*-hydroxy MDA	(±)-*N*-[*alpha*-methyl-3,4-(methylenedioxy)phenethyl]hydroxylamine
Not available	parahexyl	3-hexyl-7,8,9,10-tetrahydro-6,6,9-trimethyl-6*H*-dibenzo[*b*,*d*]pyran-1-ol
Not available	PMA	*p*-methoxy-*alpha*-methylphenethylamine
Not available	psilocine, psilotsin	3-[2-(dimethylamino)ethyl] indol-4-ol
PSILOCYBINE		3-[2-(dimethylamino)ethyl]indol-4-yl dihydrogen phosphate
ROLICYCLIDINE	PHP, PCPY	1-(1-phenylcyclohexyl)pyrrolidine
Not available	STP, DOM	2,5-dimethoxy-*alpha*,4-dimethylphenethylamine
TENAMFETAMINE	MDA	*alpha*-methyl-3,4-(methylenedioxy)phenethylamine
TENOCYCLIDINE	TCP	1-[1-(2-thienyl)cyclohexyl]piperidine
Not available	tetrahydrocannabinol, the following isomers and their sterochemical variants: 7,8,9,10-tetrahydro-6,6,9-trimethyl-3-pentyl-6*H*-dibenzo[*b*,*d*] pyran-1-ol (9*R*,10a*R*)-8,9,10,10a-tetrahydro-6,6,9-trimethyl-3-pentyl-6*H*-dibenzo[*b*,*d*]pyran-1-ol (6a*R*,9*R*,10a*R*)-6a,9,10,10a-tetrahydro-6,6,9-trimethyl-3-pentyl- 6*H*-dibenzo[*b*,*d*]pyran-1-ol (6a*R*,10a*R*)-6a,7,10,10a-tetrahydro-6,6,9-trimethyl-3-pentyl-6*H*- dibenzo[*b*,*d*]pyran -1- ol 6a,7,8,9-tetrahydro-6,6,9-trimethyl-3-pentyl-6*H*-dibenzo[*b*,*d*] pyran-1-ol (6a*R*,10a*R*)-6a,7,8,9,10,10a-hexahydro-6,6-dimethyl-9-methylene- 3-pentyl-6*H*-dibenzo[*b*,*d*]pyran-1-ol	
Not available	TMA	(±)-3,4,5-trimethoxy-*alpha*-methylphenethylamine

The salts of the substances listed in this Schedule whenever the existence of such salts is possible

100 years of Governments or Parliaments studies on the effects of cannabis

1894 United Kingdom The Indian Hemp Drugs Commission

1925 Panama The Panama Canal Zone Report

1944 USA La Guardia Report

1969 United Kingdom the Wootton Report

1970 Canada Le Dain Report

1971 &1972 Netherlands Baan & Hulsman Commissions

1973 USA Shafer Commission

1971 & 1977 Australia Marriott Committee and the Baume Committee

1982 United Kingdom Report of the Expert Group on the Effects of Cannabis Use, Home Office Advisory Council on the Misuse of Drugs, Home Office

1994 Australia Commonwealth of Australia Legislative options for cannabis use

1998 United Kingdom House of Lord Science and Technology Select Committee, Ninth Report, Cannabis: the scientific and medical evidence,

1998 New Zealand Inquiry into the Mental Health Effects of Cannabis, Report of the Health Committee, AJHR, I.6A New Zealand,

100 years of Governments or Parliaments studies on the effects of cannabis

1999 Switzerland Swiss Federal Commission for Drug Issues, Cannabis Report, Federal Office of Public Health, Bern

2001 Jamaica A Report of the National Commission on Ganja to Rt. Hon. P.J. Patterson, Q.C., M. P. Prime Minister of Jamaica,

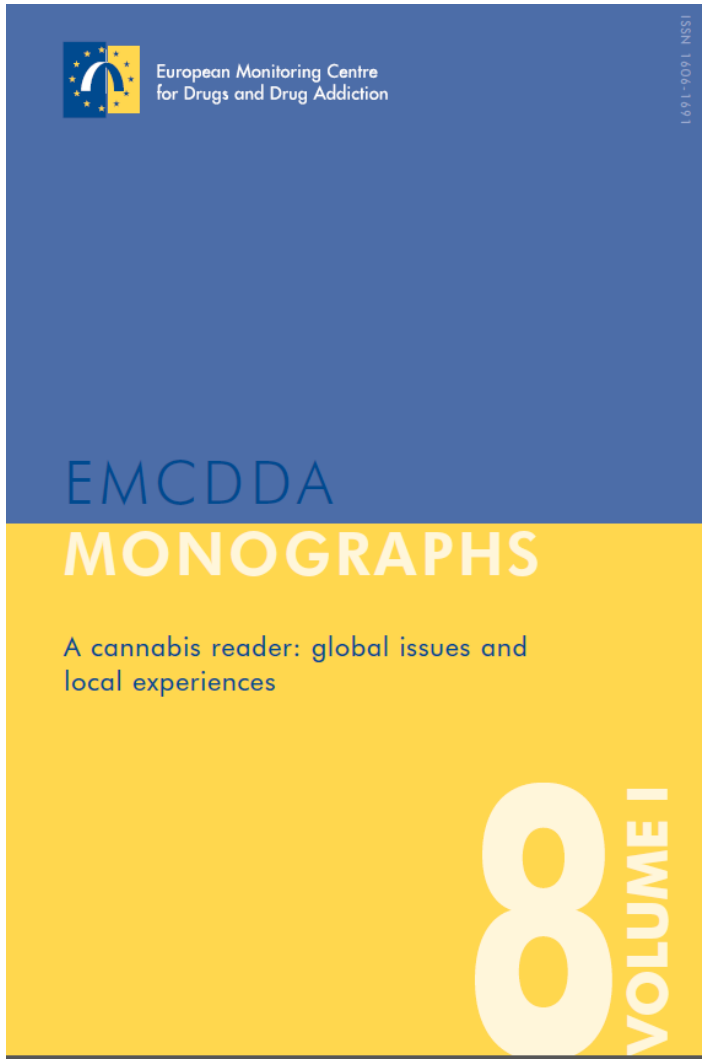
2002 United Kingdom Reports by the Advisory Council on Misuse of Drugs, Home Office, The Classification of Cannabis under the Misuse of Drugs Act 1971

2002 Canada The Senate Special Committee On Illegal Drugs, Cannabis: Our Position For A Canadian Public Policy,

2003 France Rapport de la Commission d'enquête su Sénat français sur la politique nationale de lutte contre les drogues illicites, N°321,

2005 United Kingdom Further Consideration of the Classification of Cannabis under the Misuse of Drugs Act 1971

Review of 100 years of official studies



1. **Cannabis is not an harmless substance**
2. **The dangers have been overstated**
3. **Personal use offences do not require criminal sanctions**

Lancet 2010: Drug harms in the UK: a multicriteria decision analysis *David J Nutt, Leslie A King, Lawrence D Phillips, on behalf of the Independent Scientific Committee on Drugs*

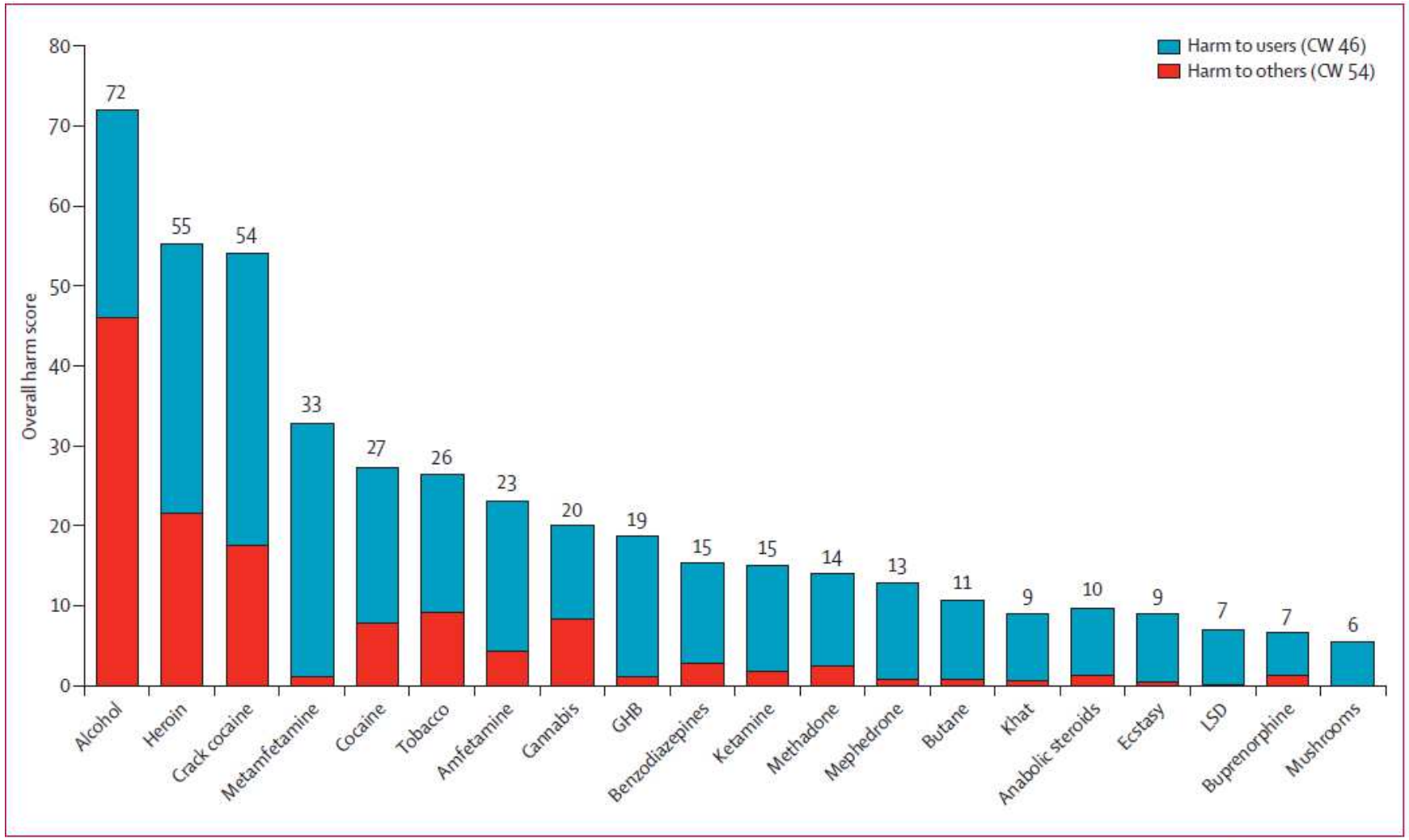
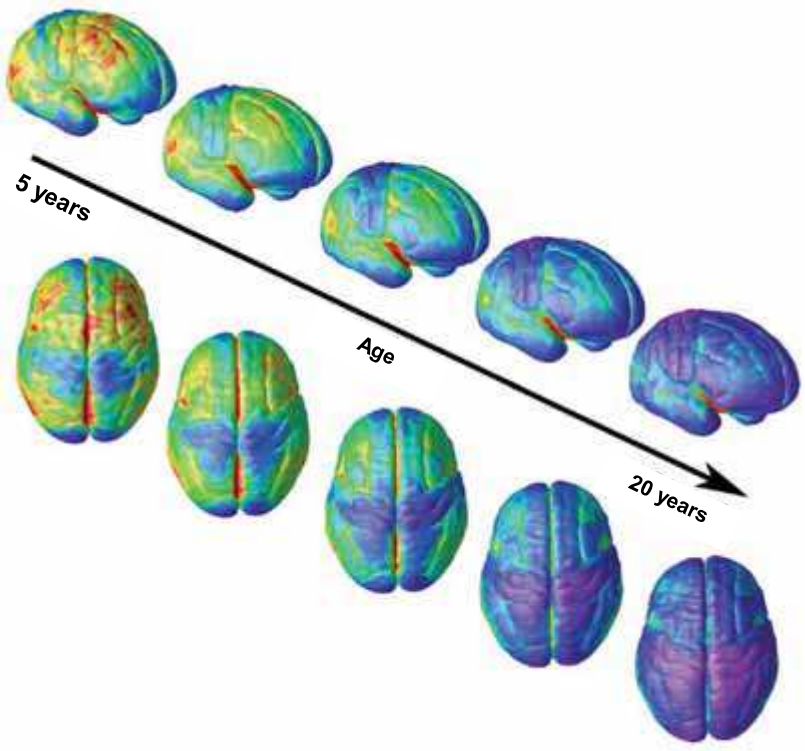


Figure 2: **Drugs ordered by their overall harm scores, showing the separate contributions to the overall scores of harms to users and harm to others**
The weights after normalisation (0–100) are shown in the key (cumulative in the sense of the sum of all the normalised weights for all the criteria to users, 46; and for all the criteria to others, 54). CW=cumulative weight. GHB=γ hydroxybutyric acid. LSD=lysergic acid diethylamide.

Studies adverse effects from cannabis use



The Journal of Neuroscience, April 16, 2014 • 34(16):5529–5538 • 5529

Neurobiology of Disease

Cannabis Use is Quantitatively Associated with Nucleus Accumbens and Amygdala Abnormalities in Young Adult Recreational Users

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[1]Laboratory of Neuroimaging and Genetics, Department of Psychiatry, [2]Mood and Motor Control Laboratory, [3]Center for Morphometric Analysis, Department of Psychiatry, and [4]Athinoula A. Martinos Center in Biomedical Imaging, Department of Radiology, Massachusetts General Hospital, Charlestown, Massachusetts 02129, [5]Harvard Medical School, Boston, Massachusetts 02115, and [6]Warren Wright Adolescent Center, Department of Psychiatry and Behavioral Sciences, Northwestern University Feinberg School of Medicine, Chicago, Illinois 06011

Marijuana is the most commonly used illicit drug in the United States, but little is known about its effects on the human brain, particularly on reward/aversion regions implicated in addiction, such as the nucleus accumbens and amygdala. Animal studies show structural changes in brain regions such as the nucleus accumbens after exposure to Δ9-tetrahydrocannabinol, but less is known about cannabis use and brain morphometry in these regions in humans. We collected high-resolution MRI scans on young adult recreational marijuana users and nonusing controls and conducted three independent analyses of morphometry in these structures: (1) gray matter density using voxel-based morphometry, (2) volume (total brain and regional volumes), and (3) shape (surface morphometry). Gray matter density analyses revealed greater gray matter density in marijuana users than in control participants in the left nucleus accumbens extending to subcallosal cortex, hypothalamus, sublenticular extended amygdala, and left amygdala, even after controlling for age, sex, alcohol use, and cigarette smoking. Trend-level effects were observed for a volume increase in the left nucleus accumbens only. Significant shape differences were detected in the left nucleus accumbens and right amygdala. The left nucleus accumbens showed salient exposure-dependent alterations across all three measures and an altered multimodal relationship across measures in the marijuana group. These data suggest that marijuana exposure, even in young recreational users, is associated with exposure-dependent alterations of the neural matrix of core reward structures and is consistent with animal studies of changes in dendritic arborization.

Key words: **cannabis; gray matter density; marijuana; multimodal imaging; reward; topology/shape**

Introduction

Marijuana (cannabis) is the most commonly used illicit drug in the United States (15.2 million past-month users; US Department of Health and Human Services, 2008). It is also the most widely used illicit drug on college campuses (Mohler-Kuo et al., 2003). Moreover, its use is increasing among adolescents and young adults (Henry et al., 2003), partially due to society's changing beliefs about cannabis use and its legal status.

Cannabis use is associated with impairments of cognitive functions, including learning and memory, attention, and decision-making. Animal studies show structural changes in brain regions underlying these functions after exposure to Δ9-tetrahydrocannabinol (THC), the main psychoactive component of cannabis (Lawston et al., 2000; Downer et al., 2001). In the nucleus accumbens, the length of the dendrites and number of dendritic spines increases with THC exposure in rats (Kolb et al., 2006). Less is known about the relationship between cannabis use and brain structure in humans. Although some studies have shown volume reductions in the hippocampus, amygdala, and cerebellum, others have not shown such effects (see Lorenzetti et al., 2010 for review). Differences in methodology may have contributed to these mixed results, suggesting that using a variety of structural methods together to quantify brain morphology may

Received Nov. 6, 2013; revised March 9, 2014; accepted March 11, 2014.

Author contributions: J.M.G., M.J.L., B.K., N.M., A.J.v.d.K., A.B., and H.C.B. designed research; J.M.G., J.K.K., S.L., and A.J.v.d.K. performed research; M.J.L., B.K., A.B., and H.C.B. contributed unpublished reagents/analytic tools; J.M.G., J.K.K., S.L., M.J.L., N.M., A.B., and H.C.B. analyzed data; J.M.G., A.B., and H.C.B. wrote the paper.

This work was supported by the National Institute on Drug Abuse (Grants 14118, 026002, 026104, and 027804 to H.C.B. and Grant 034093 to J.M.G.), the Office of National Drug Control Policy, Counterdrug Technology Assessment Center (Grants DABK39-03-0098 and DABK39-03-C-0098), and the National Institute of Neurological Disorders and Stroke, National Institutes of Health (Grant 052368 to A.J.B.). H.C.B. was also supported by the Warren Wright Adolescent Center at Northwestern Memorial Hospital and Northwestern University, Chicago. J.M.G. was supported by a Harvard Medical School Norman E. Zinberg Fellowship in Addiction Psychiatry Research.

The authors declare no competing financial interests.

*J.K.K., S.L., and M.J.L. contributed equally to this work.

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Cannabis – a political hot topic

Debates and controversies

Most stringent classification

Exaggeration and banalisation

Centre of diplomatic attention among nations and scientific controversies