Therapeutisch gebruik van illegale drugs

Marianne Destoop

MD, PhD



09/08/2019

Cannabisolie nu toch in apotheken

Binnenkort is cannabisolie op voorschrift verkrijgbaar in Belgische apotheken. Mensen die het al gebruiken, handelen niet langer illegaal.

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Pijnartsen waarschuwen: “Medicinale cannabis kan meer kwaad dan goed doen”

YV | 14 juni 2019 | 05u00

83 | 34 REACTIES



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Paddo's gaan depressie tegen

Een enkele dosis psilocybine, het actieve ingrediënt van hallucinogene paddenstoelen, helpt de angst en de depressie die mensen met kanker ondervinden gedurende een halfjaar of langer te verlichten. Dat schrijven Amerikaanse wetenschappers in het *Journal of Psychopharmacology*.



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VS keuren neusspray tegen depressie goed

AW | 06 maart 2019 | 07u13 | Bron: Belga

DEEL REAGEER



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Amerikaanse burgers zullen binnenkort toegang hebben tot esketamine, een nieuw geneesmiddel dat wordt voorgesteld als een revolutie in de behandeling van depressies. Het geneesmiddelenagentschap van de VS, de FDA, zette het licht op groen voor de verkoop van het product dat in de vorm van een neusspray op de markt komt.

Pijnartsen waarschuwen: "Medicinale cannabis kan meer kwaad dan goed doen"

YV | 14 juni 2019 | 05u00

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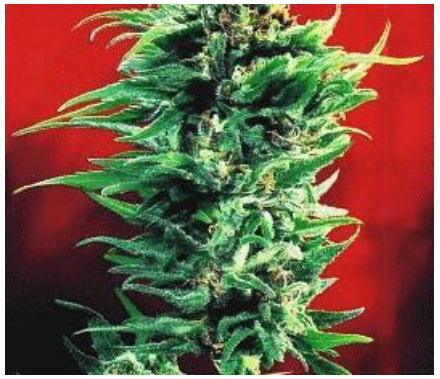
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Cannabinoiden



- Hennep- of cannabisplant
- THC (delta-9-TetraHydroCannabidinol) en CBD (CannaBiDiol)
- Gerookt in sigaretten (joint of pijp), gegeten (spacecake) of gedronken (thee)
- Ontspannend, geestesverruimend en





- Tussen 2009 en 2015 schommelde THC-gehalte van de in Nederland gekweekte wiet tussen 14 en 18%
- Synthetische cannabinoïden: K2, Spice, Sence








image







CBD



Review

ACCP

A Review of Human Studies Assessing Cannabidiol's (CBD) Therapeutic Actions and Potential

The Journal of Clinical Pharmacology
2019;00(0):1–13
© 2019, The American College of Clinical Pharmacology
DOI: 10.1002/jcph.1387

C. Michael White, PharmD[1,2]

Abstract

Cannabidiol (CBD) is a highly touted product for many different disorders among the lay press. Numerous CBD products are available, ranging from a US Food and Drug Administration (FDA)-approved product called Epidiolex to products created for medical marijuana dispensaries and products sold in smoke shops, convenience stores, and over the Internet. The legal status of the non-FDA-approved products differs depending on the source of the CBD and the state, while the consistency and quality of the non-FDA-approved products vary markedly. Without independent laboratory verification, it is impossible to know whether the labeled CBD dosage in non-FDA-approved CBD products is correct, that the delta-9-tetrahydrocannabinol content is <0.3%, and that it is free of adulteration and contamination. On the Internet, CBD has been touted for many ailments for which it has not been studied, and in those diseases with evaluable human data, it generally has weak or very weak evidence. The control of refractory seizures is a clear exception, with strong evidence of CBD's benefit. Acute CBD dosing before anxiety-provoking events like public speaking and the chronic use of CBD in schizophrenia are promising but not proven. CBD is not risk free, with adverse events (primarily somnolence and gastrointestinal in nature) and drug interactions. CBD has been shown to increase liver function tests and needs further study to assess its impact on suicidal ideation.

Keywords

anxiety, cannabidiol, cannabis, CBD, psychosis, seizures

Cannabis sativa (marijuana) has been evaluated for the treatment of many diseases and disorders, but the altered sensory and time perception (the "high") provided by the delta-9-tetrahydrocannabinol (delta-9-THC) component has been a barrier to wider adoption of the product by patients and medical professionals.[1] Employers may test workers or prospective workers for delta-9-THC in their hair, nails, or urine, with positive findings resulting in suspension, termination, or not being hired.[1] Finally, regulatory restrictions at the federal level and in some states make delta-9-THC–containing products illegal to possess or to transport across state lines, even for legitimate medical purposes such as seizures, nausea/vomiting, and pain/spasticity.[2,3]

Cannabidiol (CBD) is the second most prevalent bioactive constituent of the *Cannabis sativa* plant, and unlike in some animal species, does not convert to delta-9-THC in the human body.[2] In vitro, animal, and human studies suggest mechanisms of action for CBD directly or tangentially related to the endocannabinoid system, as delineated in Table 1.[2–10] There has been an incredible amount of media coverage on the health benefits of CBD, with CBD products heralded as medical breakthroughs or even miracles for all types of diseases.[11,12]

Medical professionals may be interested in CBD as a new therapy for patients with limited traditional options or are asked about CBD products by patients, family, and acquaintances for a host of ailments. Similarly, medical professionals might be interested in recommending CBD to patients in lieu of the medical marijuana the patient is seeking. Finally, patients may not be forthcoming about using CBD products in addition to or as a substitute for your prescribed therapy. This article provides the information medical professionals need to understand quality control issues with CBD products, CBD's pharmacokinetics and drug interaction potential, and the benefits and risks associated with using CBD for seizures, anxiety, schizophrenia, pain/spasticity, and Parkinson disease. This review will not assess the efficacy or safety of combined delta-9-THC–CBD products because the specific impact of CBD in the products cannot be determined.

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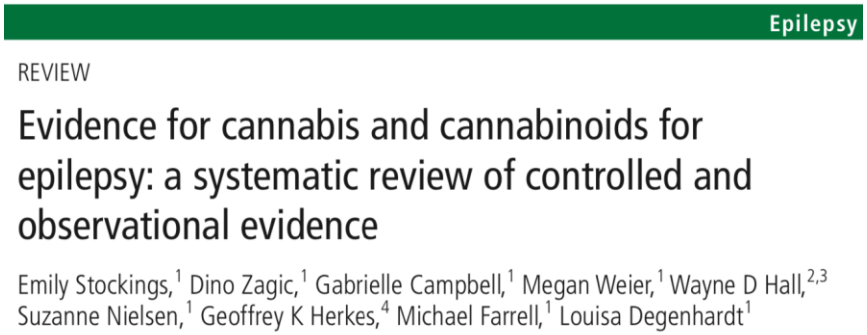
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Effect en indicatie

1. Epilepsie

6 RCT's (n=555)



Epilepsy

REVIEW

Evidence for cannabis and cannabinoids for epilepsy: a systematic review of controlled and observational evidence

Emily Stockings,[1] Dino Zagic,[1] Gabrielle Campbell,[1] Megan Weier,[1] Wayne D Hall,[2,3] Suzanne Nielsen,[1] Geoffrey K Herkes,[4] Michael Farrell,[1] Louisa Degenhardt[1]

- CBD verhoogt het percentage patiënten met een vermindering van aanvallen met ≥ 50% (n= 2 trials, RR 1.74)
- CBD verhoogt het percentage van volledig aanvalsvrije patiënten (n= 3 trials, RR 6.17)
- CBD verbetert de health-related QOL (n= 2 rials, RR 1.73)

Maar

- het eerste resultaat gaat in 2 trials enkel over patiënten met Dravet syndroom en Lennox-Gastaut syndroom
- sterkte van evidentie beperkt

DUS

- CBD is een effectieve optie als adjuvante therapie bij refractaire aanvallen bij Dravet syndroom en Lennox-Gastaut syndroom
- CBD is veelbelovend in de behandeling van andere vormen van epilepsie, maar meer onderzoek is nodig

2. Angst

12 kleine studies met single-dose CBD, vnl bij gezonde vrijwilligers

Table 2. CBD Effects on Public Speaking Anxiety[40-42]

Study Name and Subject Number	Study Design and Duration	CBD and Control Dose and Route
Public Speaking Stress Studies		
Bergamaschi 2011 (n = 24)	Subjects (n = 24) with untreated social anxiety disorder; R, DB, PC; single dose. CBD or placebo was also included for comparative purposes. Evaluations: baseline, 80 minutes after ingestion (pretest), immediately before speech (anticipatory), during speech, and 15 and 35 minutes post speech	CBD 600 mg (STI Pharmaceuticals & THC Pharmaceuticals) or placebo; oral
Results: CBD 600 mg significantly reduced VAMS anxiety, VAMS discomfort, and VAMS cognitive impairment during the speech vs placebo. No significant benefits on VAMS occurred vs placebo during the pretest, anticipatory phase, and 15 and 35 minutes after speech. CBD 600 mg significantly reduced SSPS-N scores vs placebo both in the anticipatory phase and during the speech but not at other time periods. *Conclusions:* Premedicating with CBD 600 mg reduced anxiety.		
Zuardi 2017 (n = 60)	Normal volunteers. R, DB, PC, AC; single dose. Evaluations: baseline, 80 minutes after ingestion (pretest), during speech, and 60 minutes after speech	CBD 100, 300, 900 mg (BSPG-Pharm), placebo or clonazepam 1 mg; oral
Results: VAMS anxiety significantly reduced with clonazepam during and 1 hour after the speech, and it was reduced with CBD 300 mg 1 hour after speech vs placebo. CBD 300 mg subjects had higher SBP and DBP during public speaking than with clonazepam. No other differences observed between groups at other times. *Conclusion:* Premedicating with CBD 300 mg reduced anxiety after public speaking.		
Linares 2018 (n = 57)	Normal volunteers. R, DB, PC; single dose. Evaluations: baseline, 90 minutes after ingestion (pretest), immediately before speech (anticipatory), during speech, immediately after speech, and 30 minutes after speech	CBD 150, 300, 600 mg or placebo; oral
Results: VAMS anxiety significantly reduced with CBD 300 mg during the speech vs placebo but not with the other 2 doses of CBD. No other VAMS anxiety differences observed between groups at other times, although all of the CBD groups were qualitatively lower than the placebo group at all evaluable time points. *Conclusion:* Premedicating with CBD 300 mg reduced anxiety during public speaking.		

AC, active controlled; DB, double blind; CBD, cannabidiol; DBP, diastolic blood pressure; PC, placebo controlled; R, randomized; SBP, systolic blood pressure; SSPS-N, Negative Self-Statement Public Speaking Scale; VAMS, Visual Analog Mood Scale.

Table 3. CBD Efficacy With Other Stressor That Can Induce Anxiety[43-48]

Study Name and Subject Number	Study Design and Duration	CBD and Control Dose and Route
Normal Volunteer Stress Induced Anxiety Model Studies		
Crippa 2004 (n = 10)	R, DB, PC, CO; single dose. Evaluations of the VAMS made 30 minutes before drug ingestion, at the time of drug ingestion, and at 60 minutes (at the time of stressor (cannula insertion) and 75 minutes afterwards.	CBD 400 mg (THC Pharmaceuticals) or placebo; oral
Results: CBD 400 mg significantly reduced VAMS anxiety scores and increased VAMS mental sedation scores significantly at both 60 and 75 minutes after ingestion versus placebo. *Conclusions:* CBD provided anxiolytic and sedative properties.		
Bhattacharyya 2010 (n = 15)	R, DB, AC; single dose. Drugs taken 1 hour before fMRI scanning and scanning lasted for 1 hour. Assessments made at baseline (drug admin), 1 hour (begin scan), 2 (end scan), and 3 hours (after scan). Viewing fearful faces and visual and auditory stimulation tasks were given.	CBD 600 mg (THC Pharmaceuticals), placebo, THC 10 mg, or THC 10 mg + CBD 600 mg; oral
Results: CBD alone did not impact the PANNS, STAI, or VAMS scores vs placebo at any time point, but THC alone did. In a second phase, pretreatment with CBD attenuated THC induced PANNS score increases significantly at 30 minutes. *Conclusions:* CBD did not alone provide antianxiety or psychotic effects but did attenuate the psychotic effects of THC.		
Das 2013 (n = 48)	R, DB, PC; single dose. Subjects were shocked according to a room-specific pattern, and then the pattern was repeated but no shocks were given, both on the same day CBD was given either before the second pattern occurred or after. Then subjects came back another day to repeat the procedure. The MRS was determined before and after the procedures on both days.	CBD 32 mg (STI Pharmaceuticals) or placebo; inhaled (vaporized)
Results: CBD and placebo both reduced the MRS anxiety score, but no significant differences occurred between them. *Conclusions:* CBD did not provide antianxiety effects to electrical shocks.		
Arndt 2017 (n = 38)	R, DB, PC, CO; single dose. Drug administered 2.5 hours before testing began. Mood evaluations conducted over 4.75 hours.	CBD 300, 600, 900 mg (Intys Therapeutics) or placebo; oral
Results: POMS, IAPS, DEIT, ABT, CB, ES scales showed no differences between groups. *Conclusion:* CBD did not provide antianxiety effects after emotional, anxiety-producing stimuli.		
Hundal 2018 (n = 32)	R, DB, PC; single dose. Three-dimensional virtual reality provided the stressor. Evaluations conducted 130 minutes after taking CBD or placebo. Mood evaluations conducted at baseline and immediately after leaving the virtual reality scenario. Cortisol, SBP, DBP, HR taken periodically throughout.	CBD 600 mg (GW Pharmaceuticals) or placebo; oral
Results: Anxiety outcomes (BAI, UMACL) and persecutory ideation outcomes (SPSS, CAPE) showed no significant differences between groups after the stressor. CBD showed trend toward increased anxiety via the BAI post-stressor ($P = .09$). No differences in cortisol, SBP, DBP, or HR between groups. *Conclusions:* CBD provided no antianxiety or persecutory ideation effects.		
Cannabis User Stress-Induced Anxiety Models		
Hindocha 2015 (n = 48)	R, DB, PC, CO; single dose. Assessments: Subjects completed an emotional facial affecter cognition task including fearful, angry, happy, sad, surprised, and disgusted varying in intensity from 20% to 100%. Baseline evaluations conducted followed by drug inhalation and retesting began 10 minutes thereafter.	CBD 16 mg (STI Pharmaceuticals), THC 8 mg or placebo; inhaled (vaporized)
Results: CBD and THC had no effect on VAS scores for anxiety. CBD enhanced correct facial recognition for facial images at 60% intensity but not the others while THC reduced facial recognition at 40% intensity. *Conclusions:* CBD did not provide beneficial antianxiety effects.		
Studies in Patients Diagnosed With Social Anxiety Disorder		
Crippa 2011 (n = 10)	R, DB, PC, CO; single dose. Evaluations of the VAMS made 30 minutes before drug ingestion, at the time of drug ingestion, and at 60 minutes (cannula insertion), 75 minutes (stress period), and 140 minutes (after stress).	CBD 400 mg or placebo; oral
Results: Subjects had severe baseline anxiety via the SPIN and BSPS scores of >52 and >54, respectively. CBD group had lower VAMS anxiety scores at 60, 75, and 140 minutes after dosing vs placebo, but there were no differences between groups for VAMS mental or VAMS physical sedation scores vs placebo. *Conclusion:* CBD therapy reduces anxiety during a stressful event.		

ABT, Attentional Bias Task; AC, active controlled; BAI, Beck Anxiety Inventory; BSPS, Brief Social Phobia Scale; CAPE, Community Assessment of Psychic Experiences; CB, Cyberball; CBD, cannabidiol; DB, double blind; DBP, diastolic blood pressure; DEIT, Dynamic Emotion Identification Task; ES, Emotional Stroop; fMRI, functional magnetic resonance imaging; HR, heart rate; IAPS, International Affective Picture System; MRS, Mood Rating Scale; PANNS Positive and Negative Syndrome Scale; PC, placebo controlled; POMS, Profiles of Mood State; R, randomized; SBP, systolic blood pressure; SPIN, Social Phobia INventory; SSPS, State Social Paranoia Scale; SSPS-N, Negative Self-Statement Public Speaking Scale; STAI, Spielberger State Trait Anxiety Inventory; UMACL, University of Wales Mood Adjective Checklist; VAMS, Visual Analog Mood Scale.

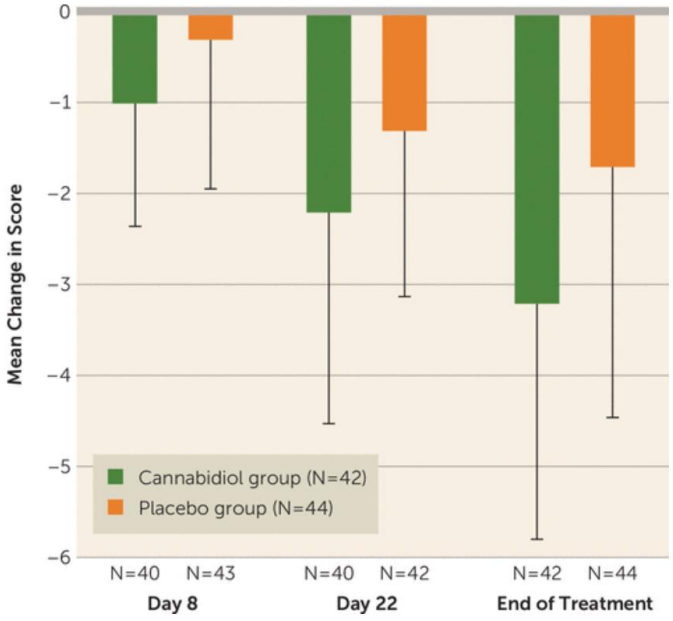
DUS

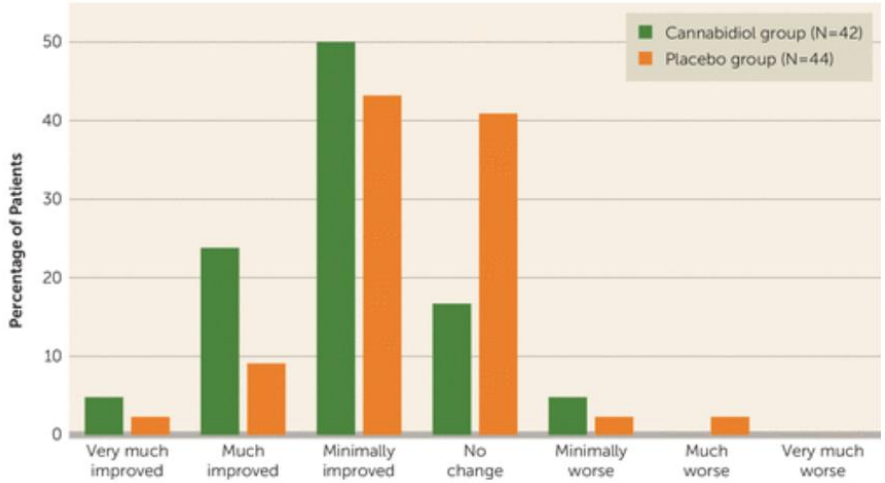
Beloftevol, maar niet bewezen als premedicatie bij stressvolle gebeurtenissen die angst kunnen veroorzaken bv. publiek spreken

3. Schizofrenie en psychose

a) 3 RCT's met patiënten met schizofrenie:

- N=88, 1000 mg/d add-on gedurende 6w (Mc Guire, 2018)





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- N=36, 600mg/d add-on gedurende 6w (Boggs, 2018)
- N=39, 200-800mg/d gedurende 4w vs amisulpiride (Leweke, 2012)

b) Single-dose CBD bij patiënten met schizofrenie (n= 28), plecebo vs 300 vs 600mg CBD (Hallak, 2010)

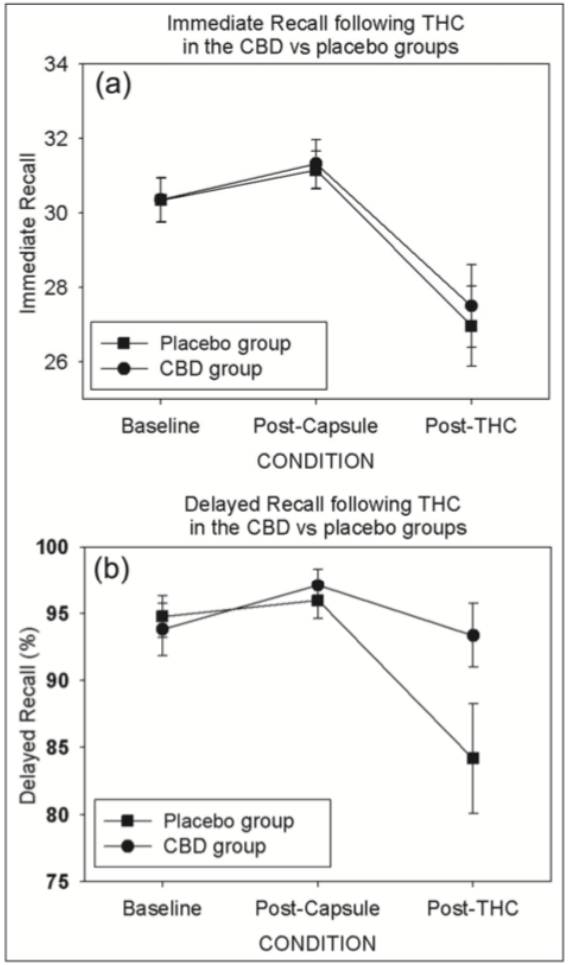
c) 2 dubbel-blinde studies die het effect van single-dose CBD meten

- Bhattacharyya (2010): n= 15

placebo vs THC 10mg vs CBD 600mg vs THC 10mg + CBD 600mg

- Englund (2013): n=48

IV 1.5mg THC + 600mg CBD vs placebo



DUS

(veel) belovende resultaten , doch niet bewezen in de chronische behandeling van schizofrenie

4. Pijn en spasticiteit

- 1 RCT, n=24, CBD vs THC vs CBD + THC vs placebo (Wade 2003)
- Open-label studie met CBD-verrijkte hennepolie, n=12, sublinguaal150mg/ml (Palmieri, 2017)
- Open-label studie, n=7, 100-300mg/d (Cunnettia, 2018)

DUS: beperkte en zwakke evidentie

5. Parkinson

1 RCT, n=21, placebo vs 75mg CBD vs 300mg CBD (Chagas, 2014)

Bijwerkingen

- Somnolentie en vermoeidheid
- GI-bijwerkingen (diarree, braken, verminderde eetlust)
- Pyrexia
- Gestegen leverfunctietesten (vnl ALT)
- Suïcidaliteit

- Minder EPS, gewichtstoename en prolactinestijging in vgl tot amisulpiride?

Farmacokinetiek

- Metabolisatie door cytochroom P450 (CYP2C19 en CYP3A) en uridine 5´-diphopho-glucuronosyltransferase (UGT1A7, UGT1A9 en UGT2B7)
- Risico op interactie met CYP en UGT substraten (vnl CYP2C19), CYP inducers en CYP 3A inhibitoren

Risico's

- Vele verkochte producten bevatten weinig tot geen CBD.

Sample ID	CBD(A)			THC(A)		
	label, %	measured, %	deviation, rel. %	label, %	measured, %	deviation, rel. %
1	27	2.3	–91.5	17	0.1	–99.4
2	25	0	–100	35	4.6	–86.9
3	12	0.2	–98.3	–	0	*
4	10.9	2.8	–74.3	–	0.1	*
5	10	2.2	–78	10	4	–60
6	8	0.6	–92.5	4	0.2	–95
7	8	0.6	–92.5	4	0.1	–97.5
8	6	0.2	–96.7	5	0.1	–98
9	5	0	–100	40	3.4	–91.5
10	4	4.7	+17.50	–	0.2	*
11	4	5.4	+35	–	0.3	*
12	4	4	0	–	0	*
13	4	4.2	+5	–	0	*
14	3	3.1	+3.3	–	0.2	*
15	2.75	2.8	+1.8	–	0.1	*
16	0.1	0.1	0	4	6.3	+57.5
17	–	0.1	*	7	7.9	+12.9
18	–	0	*	5	0.7	–86
19	–	0	*	5	0.9	–82
20	–	0.1	*	20	15.8	–21
21	–	0	*	7	6.4	–8.6

CBD, cannabidiol; THC, tetrahydrocannabinol; CBD(A), total sum of CBD plus CBD-acid; THC(A), total sum of THC plus THC-acid. * Not applicable because no label claim was made.

Wetgeving

- België heeft in 2018 onder druk van Europa een wet goedgekeurd die toelaat om producten met een THC percentage lager dan 0,2% THC op de markt te brengen. Bijgevolg worden producten die voldoen aan deze vereiste niet langer onder de drugswetgeving bekeken.

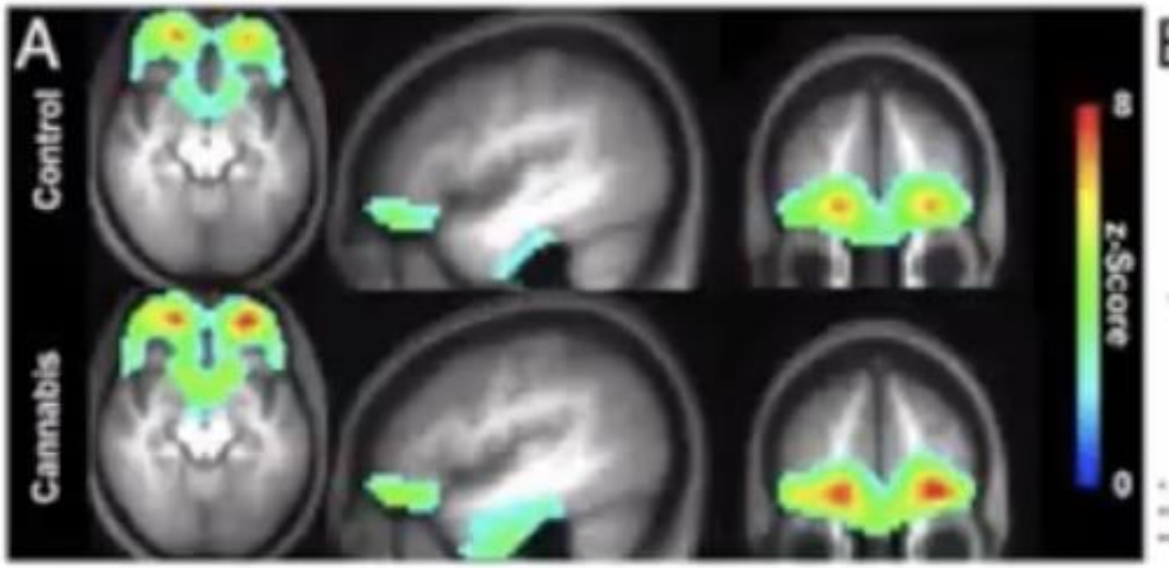
- Echter waren de Belgische autoriteiten niet voorbereid op deze maatregel... Hierdoor ontbreekt tot op vandaag een juridisch kader voor het commercialiseren van CBD hennepbloemen en producten op basis van CBD.

THC

- Misselijkheid, braken en eetlustvermindering bij kanker (Bowles, 2012; Philips, 2016)
- HIV/aids (Lutge, 2013)
- Epileptische insulten bij bepaalde vormen van E bij kinderen (Hussain, 2015)
- Chronische neurogene pijn (Andreae, 2015; Aviram, 2017)
- Spasticiteit bij MS (Lakhan, 2009)



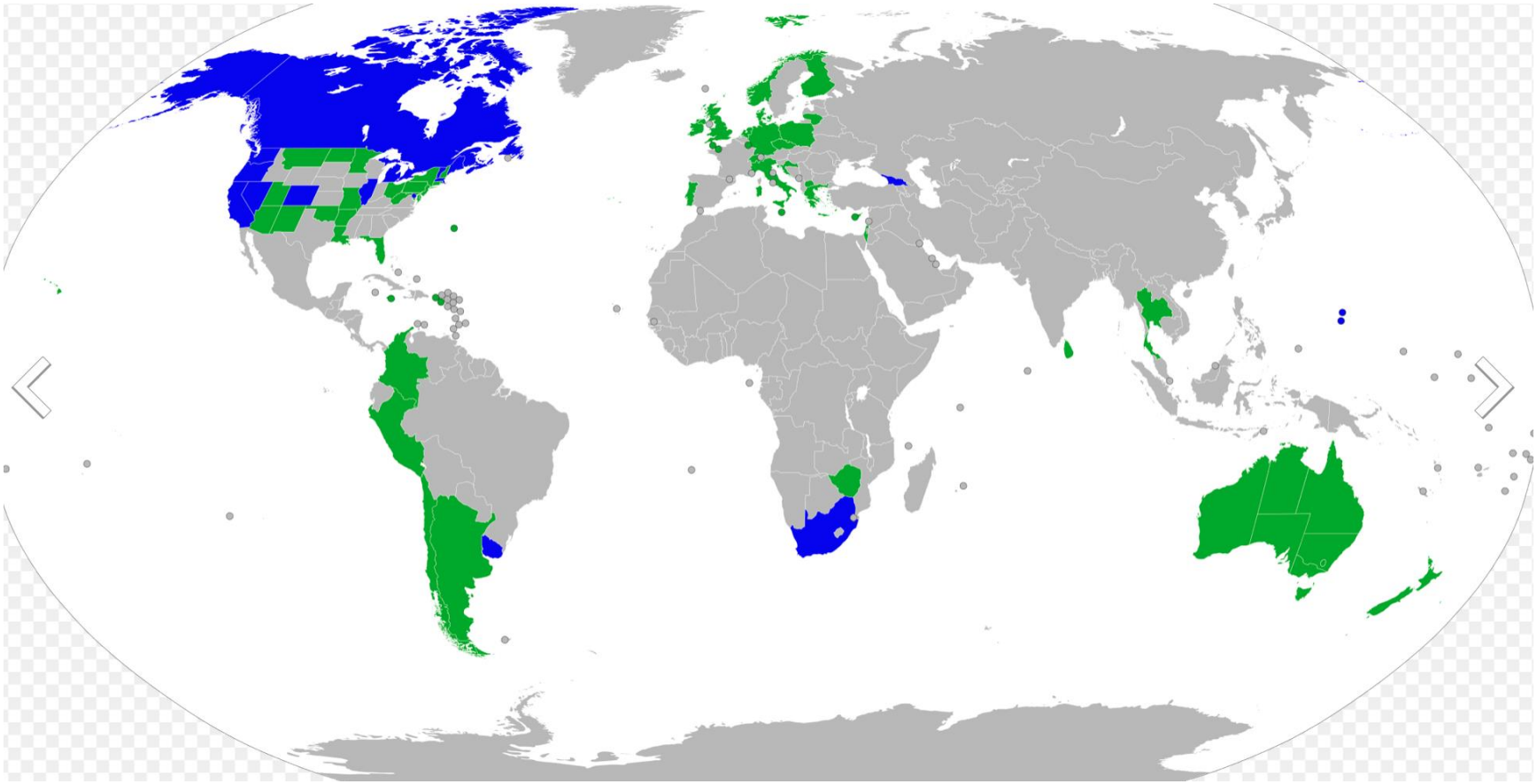
Cannabis increase OFC connectivity



Bijwerkingen en risico’s

- Vermoeidheid
- Duizeligheid en braken
- Hallucinaties
- Cognitieve problemen
- Afhankelijkheid
- Psychose

Medicinale cannabis



JAMA Psychiatry | Original Investigation

US Adult Illicit Cannabis Use, Cannabis Use Disorder, and Medical Marijuana Laws
1991-1992 to 2012-2013

Deborah S. Hasin, PhD; Aaron L. Sarvet, MPH; Magdalena Cerdá, DrPH; Katherine M. Keyes, PhD; Malka Stohl, MS; Sandro Galea, MD, DrPH; Melanie M. Wall, PhD

Figure 1. Medical Marijuana Laws (MML) and Marijuana Use in the Past 12 Months

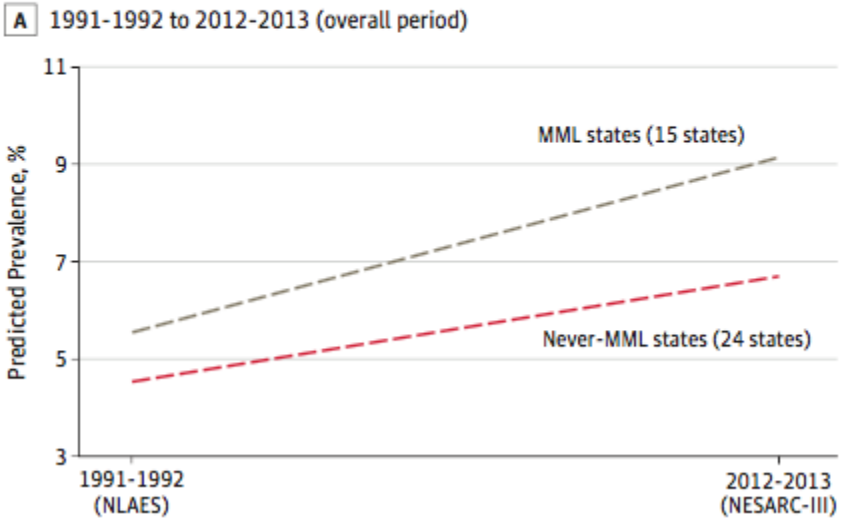
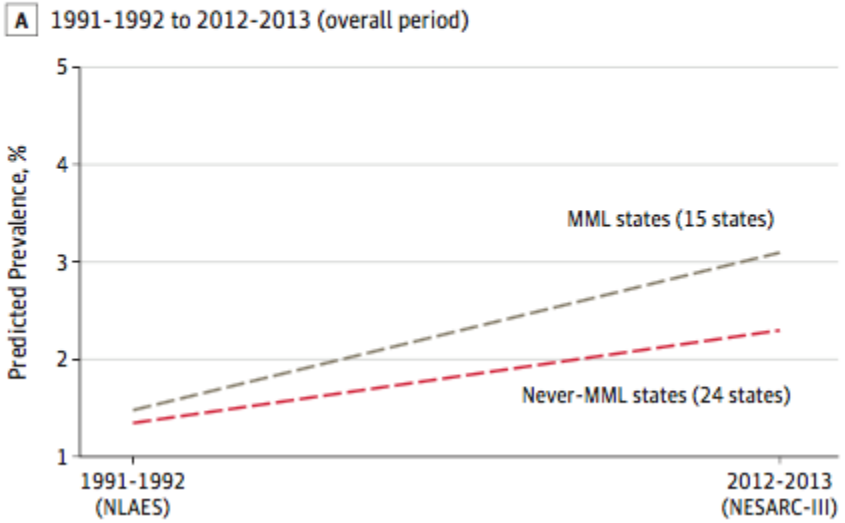
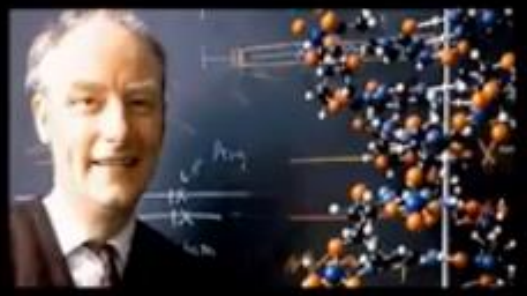


Figure 2. Medical Marijuana Laws (MML) and *DSM-IV* Cannabis Use Disorder in the Past 12 Months



Hallucinogenen

Two revolutionary Nobel prizewinners who valued the psychedelic experience



Francis Crick
Double Helix



Kary Mullis (PCR)
Polymerase Chain Reaction

Effect en indicatie

Neuropharmacology xxx (2017) 1–19

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journal homepage: www.elsevier.com/locate/neuropharm

Invited review

Psychiatry & the psychedelic drugs. Past, present & future

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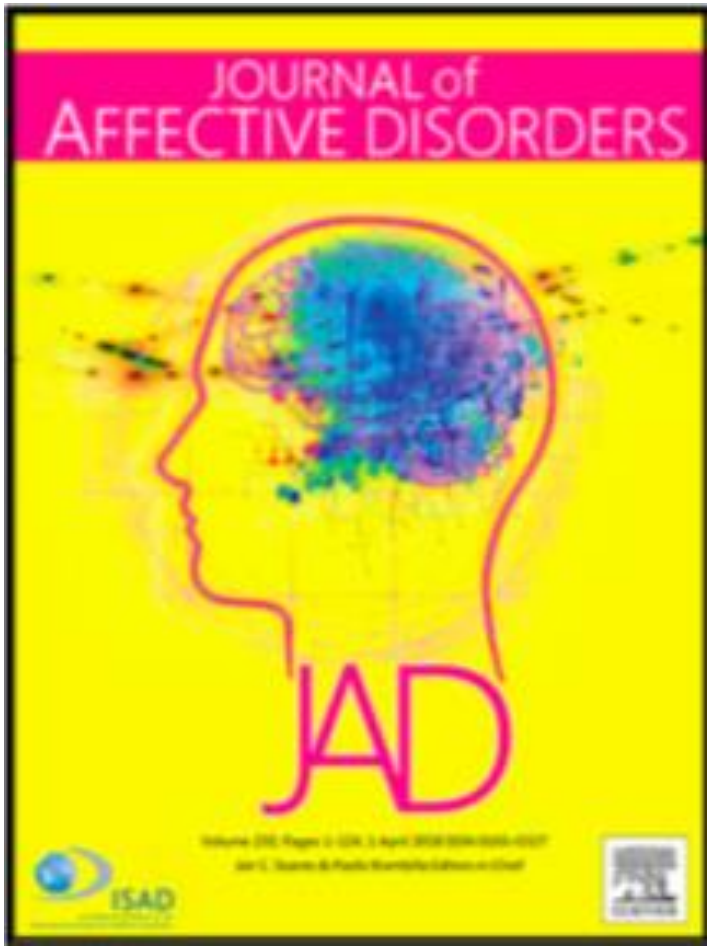
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Psychiatric disorders
Clinical trials

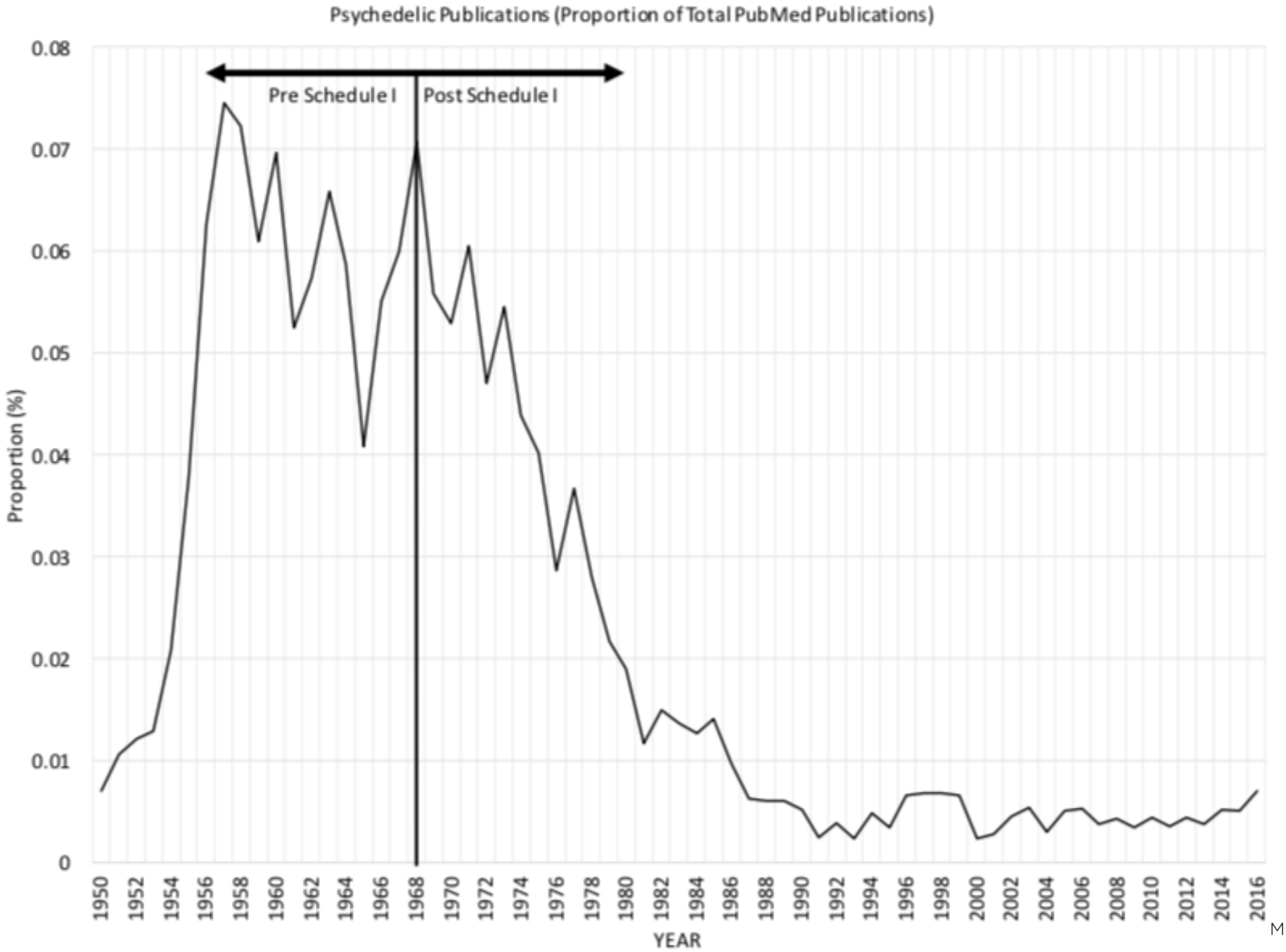
ABSTRACT

The classical psychedelic drugs, including psilocybin, lysergic acid diethylamide and mescaline, were used extensively in psychiatry before they were placed in Schedule I of the UN Convention on Drugs in 1967. Experimentation and clinical trials undertaken prior to legal sanction suggest that they are not helpful for those with established psychotic disorders and should be avoided in those liable to develop them. However, those with so-called 'psychoneurotic' disorders sometimes benefited considerably from their tendency to 'loosen' otherwise fixed, maladaptive patterns of cognition and behaviour, particularly when given in a supportive, therapeutic setting. Pre-prohibition studies in this area were sub-optimal, although a recent systematic review in unipolar mood disorder and a meta-analysis in alcoholism have both suggested efficacy. The incidence of serious adverse events appears to be low. Since 2006, there have been several pilot trials and randomised controlled trials using psychedelics (mostly psilocybin) in various non-psychotic psychiatric disorders. These have provided encouraging results that provide initial evidence of safety and efficacy, however the regulatory and legal hurdles to licensing psychedelics as medicines are formidable. This paper summarises clinical trials using psychedelics pre and post prohibition, discusses the methodological challenges of performing good quality trials in this area and considers a strategic approach to the legal and regulatory barriers to licensing psychedelics as a treatment in mainstream psychiatry.

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Psychedelic Publications (Proportion of Total PubMed Publications)
Pre Schedule I
Post Schedule I
Proportion (%)
0.08
0.07
0.06
0.05
0.04
0.03
0.02
0.01
0
1950
1952
1954
1956
1958
1960
1962
1964
1966
1968
1970
1972
1974
1976
1978
1980
1982
1984
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2012
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2016
YEAR

Mescaline

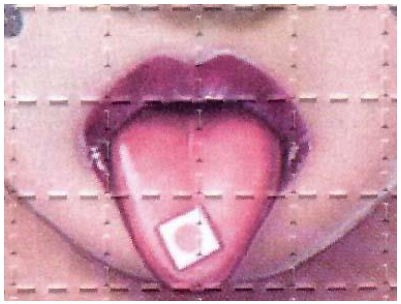
- Eerste experimenten 1895 – 1940:
  - Prentiss & Morgan, 1895
  - Mitchell, 1896
  - Arthur Heffter, 1897
  - Alwyn Knauer, 1913
  - Heinrich Kluver, 1926
  - Erich Guttman, 1936



1940-1970: studies bij psychose

- 1943: Hofmann synthetiseerde LSD
- 1950: Busch & Johnson: LSD aan manische en psychotische patiënten (n=21)
- 1952: Hoch: LSD en mescaline bij schizofrenie (n=59)
- 1955: Denber & Merlis: 500mg mescaline bij schizofrenie(n= 25)
- 1957: Merlis: 500 en 700mg mescaline bij schizofrenie (n=24)
- 1968: Langs & Barr: schizofrenie vs controles, LSD vs placebo





1940-1979 bij neurose

- 1954: Sandison: variabele dosissen LSD (startdosis 25mcg) met variabele intervallen (meestal wekelijks) (n=36)
- 1957: Sandison: 6m FU (n=93)

- 1960: Chandler & Hartman: 690 psychotherapie sessies met LSD (tussen 50 en 150mcg) (n=110)
- 1964: Whitaker: gemmiddeld 3 LSD psychotherapie sessies (n=100) vs controles (n=100)
- 1967: Savage: LSD (200-300mcg) en mescaline (200-400mg) zonder psychotherapie (n=243) met FU tot 12m

1940-1979 bij alcoholisme

Meta-analyse (Krebs & Johansen, 2012)

- 1961 Maclean: 400-1500mcg LSD aan AUD (n=61) en andere patiënten (n=39), FU 18m
- 1962 Jensen: LSD aan AUD (n=58) vs psychotherapie aan AUD (n=35) vs TAU aan AUD (n=45)
- 1966 Smart: AUD (n=30): TAU vs TAU+60mg ephedrine sulphate vs TAU+ 800mcg LSD, FU 6m
- 1969 Hollister: AUD (n=72): LSD 600mcg vs 60mg dextroamfetamine, FU 12m
- 1969 Ludwig: AUD (n=176): LSD (3mcg/kg) + hypnose + psychotherapie vs LSD + psychotherapie vs LSD + geen therapie, FU 12m

DUS: LSD effect op 1-maand abstinentie, na 6m geen sign effect meer

Besluit studies voor het verbod

- Methodologie van suboptimale kwaliteit
- Geen effectiviteit van psychedelica bij patiënten met psychotische stoornissen en omwille van toename van psychotische stoornissen zijn psychedelica relatief gecontra-indiceerd
- Bij unipolaire stemmingsstoornissen een verbetering tot 80%, maar meta-analyse niet mogelijk omwille van beperkte kwaliteit
- Mogelijks effectiviteit bij AUD

Bijwerkingen (Cohen, 1960)

- Beperkte verhoging van polsslag en bloeddruk
- Hoofdpijn
- Gastro-intestinale klachten
- Misselijkheid
- Veranderingen in temperatuur gewaarwordingen, soms ook veranderingen in lichaamstemperatuur (met bibberen of zweten)
- Bad trip
- SI-poging 0.12%, geslaagde SI 0.04%
- Psychotische symptomen langer dan 48u (0.18%)
- Geen melding van afhankelijkheid

- In 1967 werden een aantal meldingen gemaakt van toxische psychologische reacties (Keeler & Reifler) wat werd overgenomen door de media. Daarenboven deed de CIA onderzoek deed naar LSD als waarheidsdrugs. Dit alles leidde tot negatieve socio-politieke attitude tov psychedelica. Bovendien eisten 151 patiënten in Denemarken compensatie voor psychiatrische symptomatologie die te wijten zou zijn aan LSD behandeling (niet bewezen) soms onder dwang en soms zonder informed consent (tussen 1980 en 1990).

- In 1967 werden de psychedelica (mescaline, psilobycine en LSD) geclassificeerd onder schedule 1 van de UN convention on drugs, wat betekent dat er geen aanvaardbaar medisch gebruik is toegestaan en ze beschouwd worden als schadelijk en risicovol voor afhankelijkheid op het hoogste niveau.

Moderne studies

1. OCD

25, 100, 200 of 300 mcg/kg psilocybine gedurende 1w(n=9) (Moreno, 2006)

2. Nicotine afhankelijkheid

20mg vs 30 mg psilocybine in een 15-w programma voor rookstop, 6m FU (n=15) (Johnson, 2014)

3. AUD

0.3 mg/kg vs 0.4 mg/kg psilocybine (n=10) (Bogenschultz, 2015)

4. MDD

- 10mg vs 25mg psilocybine, FU 6m (n=20) (Carhart-Harris, 2017)
- ayahuasca (2ml/kg) bij recurrente depressie, FU 3w (n=4) (Osorio, 2015)
- ayahuasca (2ml/kg) bij recurrente depressie (n=17) (Sanches, 2016)

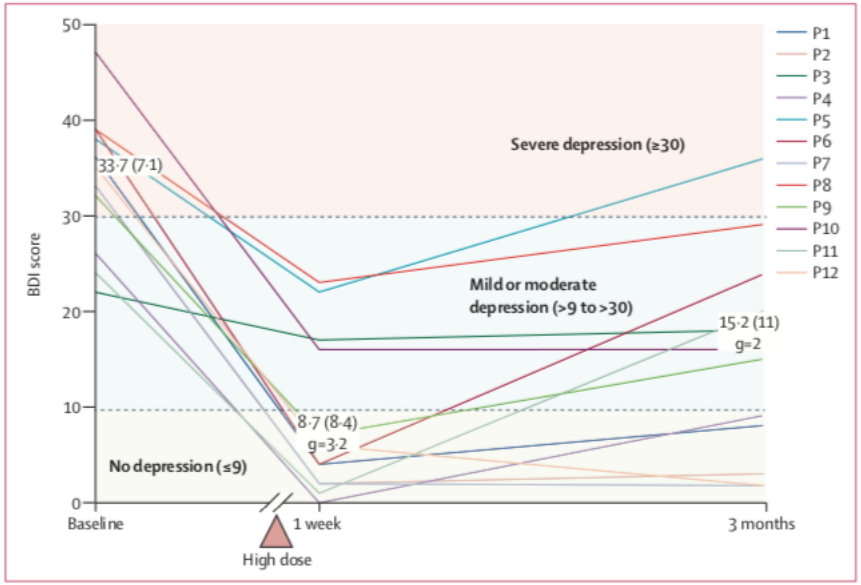


Figure 4: **Depression severity (BDI) over time, by patient**
Figure shows depression severity (BDI) over time plotted for each of the 12 patients. Mean values (SD) are shown as well as the relevant effect sizes (Hedges' g) versus baseline. BDI=Beck Depression Inventory.

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5. Angst

- psilocybin (0.2mg/kg) vs placebo (niacin 250mg) aan patiënten met angsten geassocieerd met een levensbedreigende aandoening, FU 3m (n=12) (Grob, 2011)
- 200mcg vs 20 mcg LSD, FU 12m

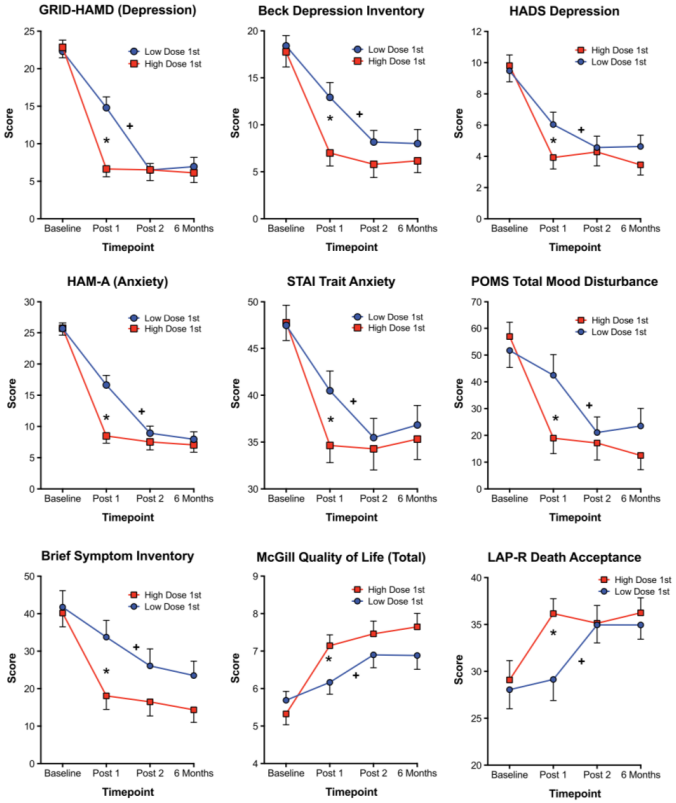
(n=12) (Gasser, 2014)

- 1 of 3mg psilocybin vs 22 of 30mg

psilocybine (n=51) (Griffiths, 2016)

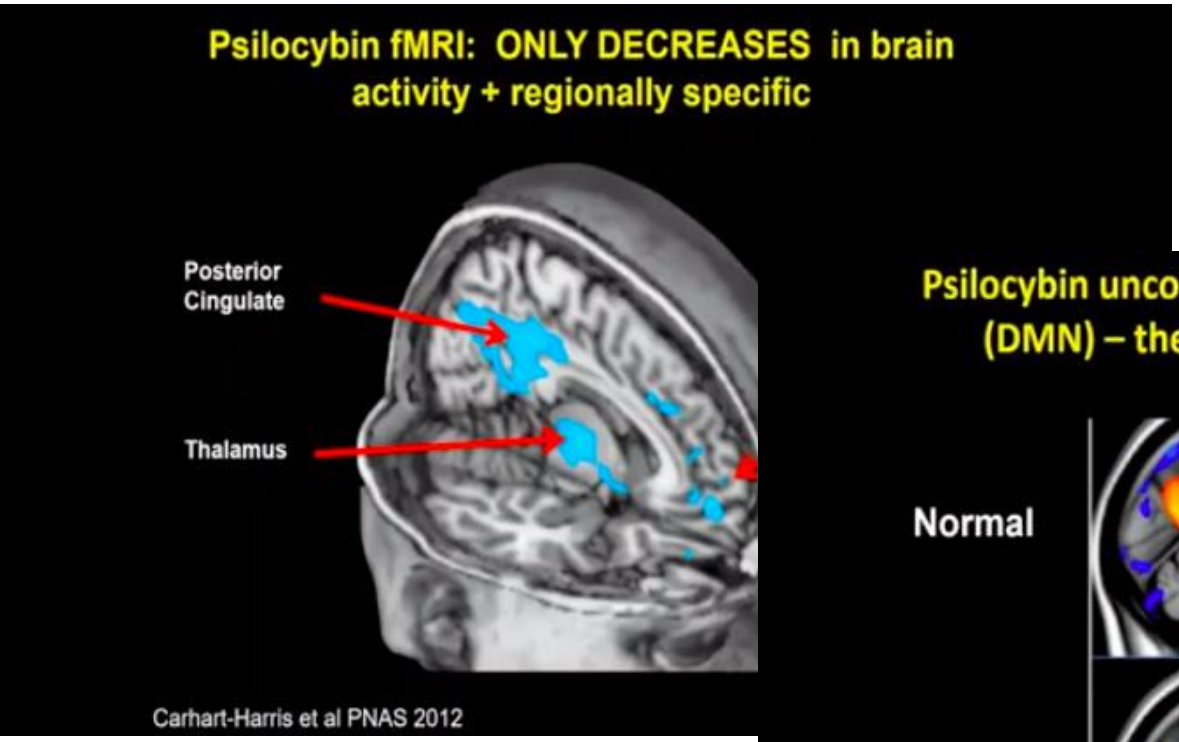
- 0.3mg/kg psilocybin vs 250mg

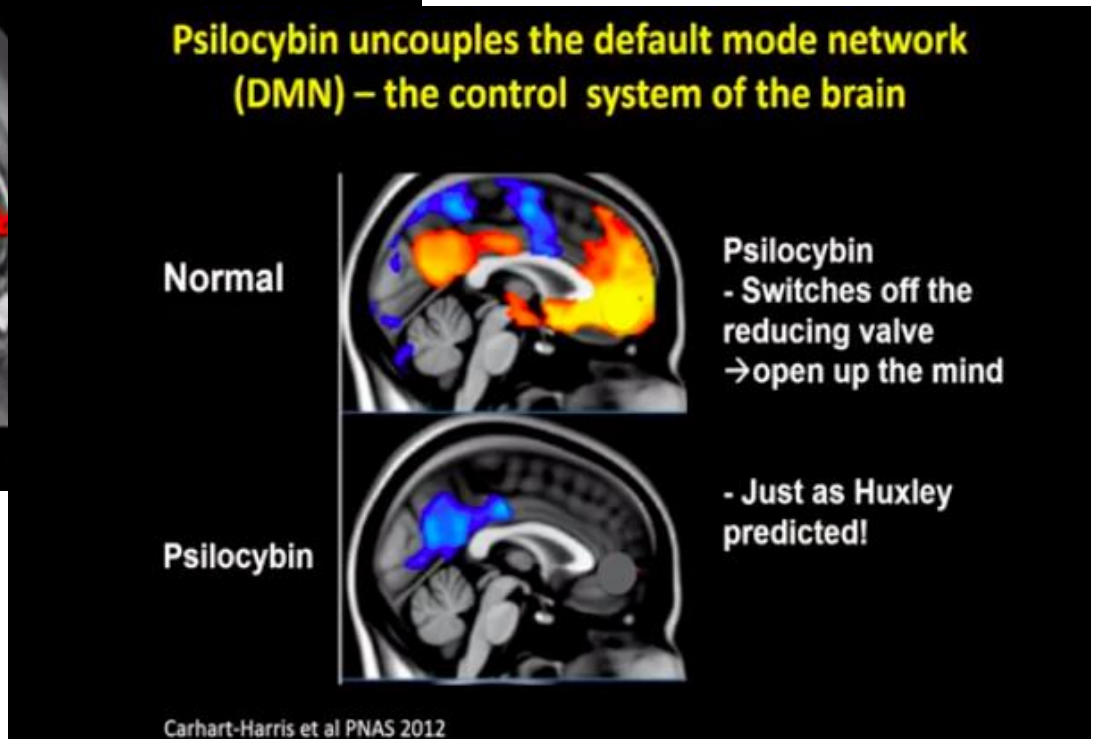
niacin (n=29) (Ross, 2016)

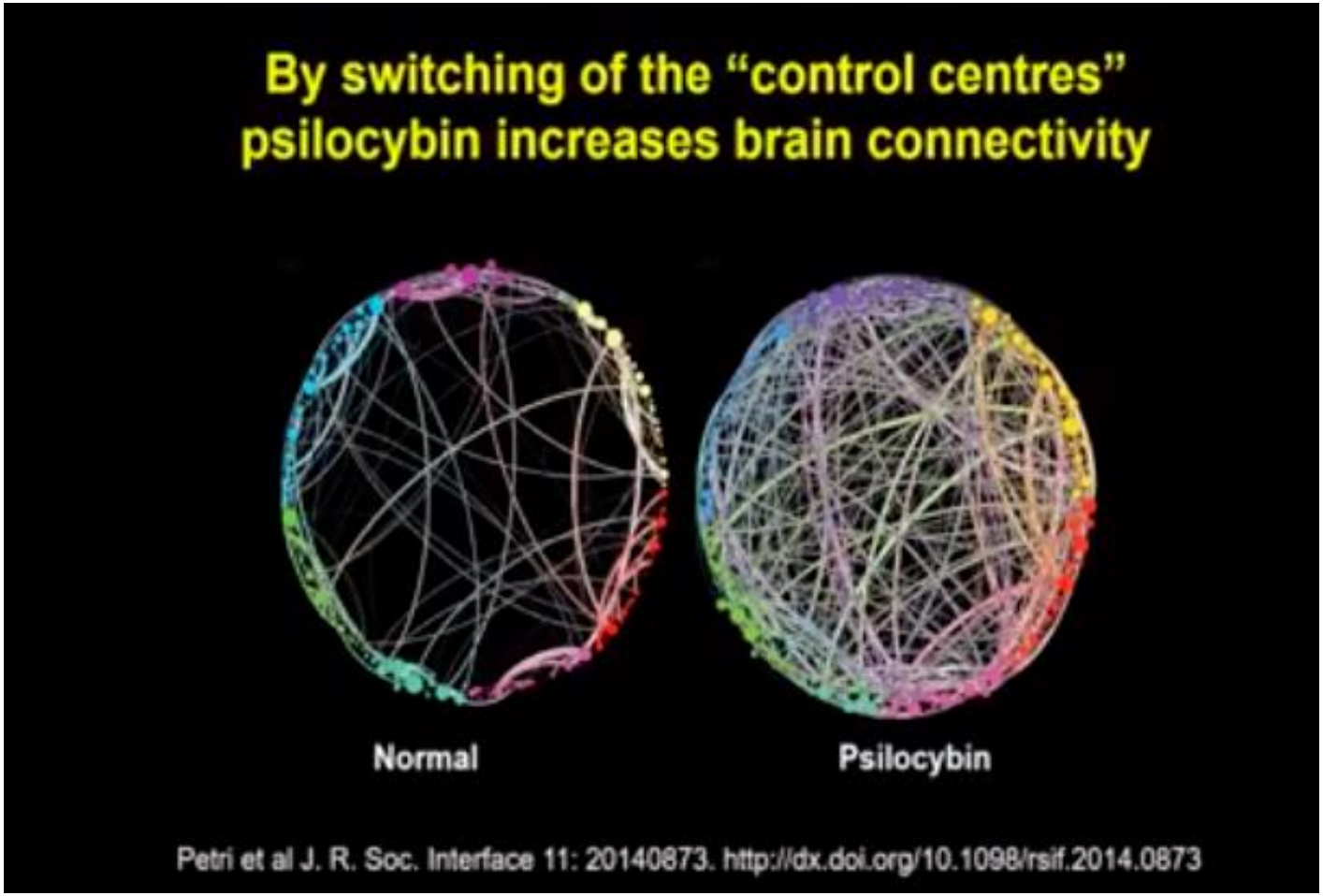


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Psilocybine

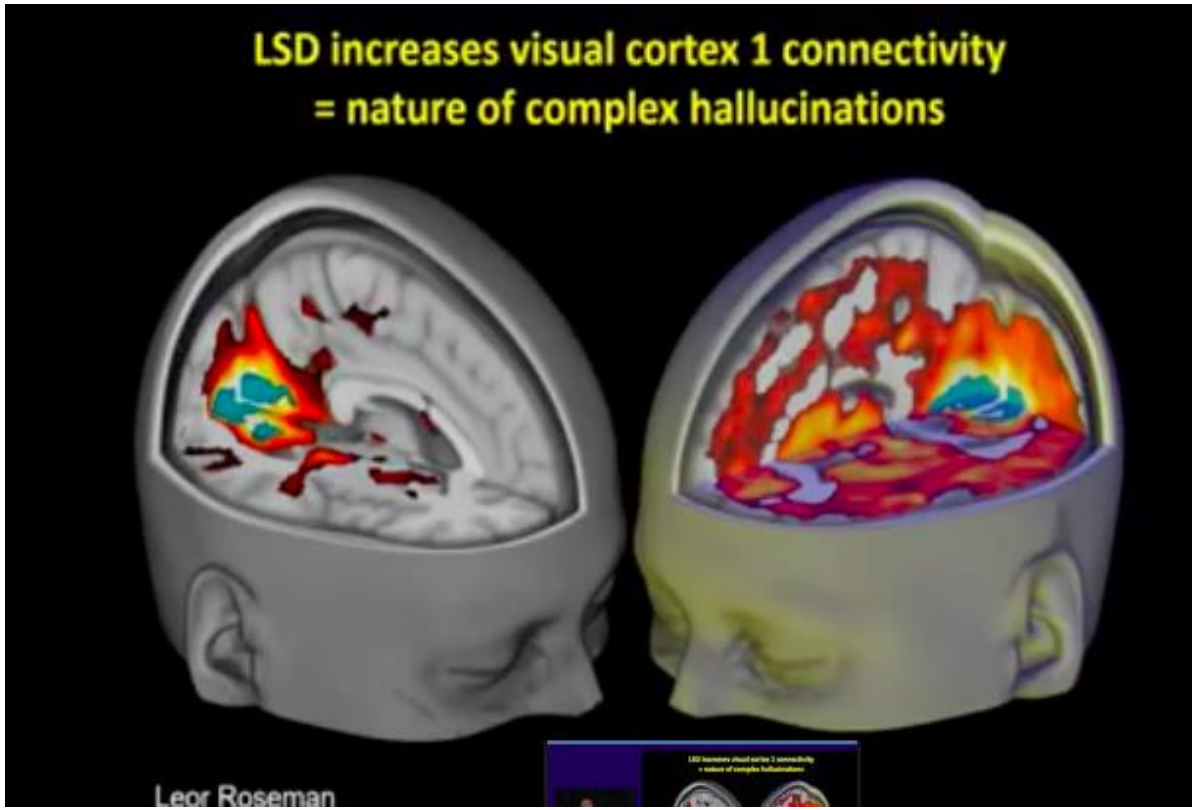






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LSD

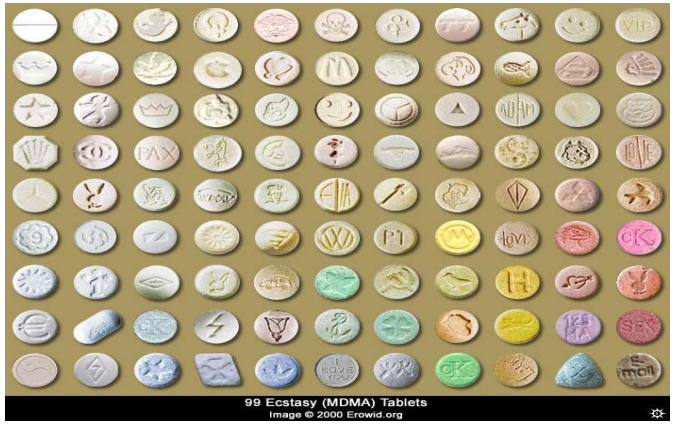

LSD increases visual cortex 1 connectivity
= nature of complex hallucinations
Leor Roseman

Bijwerkingen

- Voorbijgaande angstgevoelens
- Nausea en braken
- Milde verhogingen in bloeddruk en hartslag
- Hoofdpijn
- Beperkte tolerantie en weinig neiging tot abusus (Brunton, 2011)

MDMA

- 5-HT1A en 5-HT1B receptoren
- Dopamine en noradrenaline
- Alpha-2 receptoren
- Oxytocine



MDMA

- Jaren 60:



Alexander Shulgin



Leo Zeff



David Nichols

- Jaren 80: verschillende niet-gecontroleerde case studies en 1 grote follow-up studie (n≥100) (Gasser, 1995)
- Verboden middel

frontiers in Psychiatry

REVIEW
published: 20 March 2019
doi: 10.3389/fpsyt.2019.00138

A Review of 3,4-methylenedioxymethamphetamine (MDMA)-Assisted Psychotherapy

Ben Sessa, Laurie Higbed and David Nutt*

Neuropsychopharmacology Unit, Department of Medicine, Imperial College London, London, United Kingdom

This paper provides a brief review of the history, proposed pharmacological mechanisms, safety issues, and clinical applications of the medicine 3,4-methylenedioxymethamphetamine (MDMA). Most clinical MDMA research in patients to date has focused on MDMA-assisted psychotherapy to treat posttraumatic stress disorder (PTSD). In this review paper other potential therapeutic applications for MDMA therapy are described, including contemporary studies treating anxiety associated with autism and the authors' ongoing study exploring the potential role for MDMA-assisted psychotherapy to treat alcohol use disorder. MDMA therapy for PTSD is now entering the final Phase 3 stage of drug development, with a target set for licensing by the FDA and EMA in 2021. This means that if clinical efficacy criteria are achieved, MDMA would become a medicine.

Keywords: MDMA (3, 4-methylenedioxymethamphetamine), trauma, addiction, psychotherapy, alcohol

OPEN ACCESS

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THE EARLY THERAPEUTIC USE OF MDMA

In the late 1960's, after lysergic acid diethylamide (LSD) was banned, some psychedelic therapists began exploring other drugs as tools to enhance psychotherapy. One, Leo Zeff, was initially introduced to MDMA in 1976 by psychedelic chemist, Alexander "Sasha" Shulgin, who had been studying psychedelics since the early 1960s (1). Zeff went on to successfully and safely give MDMA, then legal, to many thousands of patients (2). Shulgin, alongside chemist David E. Nichols, published the first report into the effects and pharmacology of MDMA in humans (3).

Not a "classic" psychedelic drug, but an "entactogen" (4), MDMA produces a more gentle and easily tolerated state compared to LSD. It is shorter-acting, which makes it more clinically manageable, it enhances feelings of empathy and bonding and allows users to access and process memories of emotional trauma (5).

Psychotherapists using MDMA in the early 1980s, when was called "Adam" or "Empathy," wished to keep it within the clinical research community. But MDMA became rebranded as the more marketable "Ecstasy" and its non-clinical use spread—especially in the club scene or in large parties called raves. In 1984, in response to rising police seizures of the drug, the DEA announced that it intended to ban the compound. The clinical MDMA research community requested a hearing to debate the DEA's intention, but in May 1985 MDMA was initially placed in an emergency Schedule One category and subsequently became permanently scheduled thereafter, where it has stayed ever since—hugely restricting opportunities for its research (6). Due to this Catch 22 situation, very little clinical research was able to take place. This prompted the formation of the US-based research organization, *The Multidisciplinary Association for Psychedelic Studies* (MAPS), which today is spearheading global clinical research of MDMA.

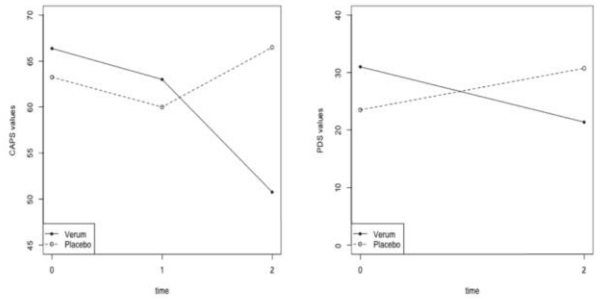
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Effect en indicatie

1. PTSD

a) 2 RCT's

- N= 22, placebo vs 125mg MDMA, 12m-FU (Mithoefer, 2010)
- N= 12, 25 vs 125mg MDMA, 12m-FU(Oehen, 2013)



CAPS mean total scores by group for time TO-T2 (SD)

TO: Baseline < 4 weeks before MDMA and after discontinuation of psychtropic medication
Active Placebo: 63.4 (7.9) Full Dose: 66.4 (13.6)
T1: 3 weeks post MDMA-session 2
Active Placebo: 60.0 (6.8) Full Dose: 63.0 (17.8)
T2: 3 weeks post MDMA-session 3 (end of treatment)
Active Placebo: 66.5 (7.6) Full Dose: 50.8 (19.7)
CAPS Change scores (SD):
T0-T1: Active Placebo: -3.3 (9.9) Full dose: -3.4 (12.0)
T1-T2: Active Placebo: 6.5 (10.3) Full dose: -12.2 (8.1)
T0-T2: Active Placebo: -3.2 (15.3) Full dose: -15.6 (18.1)

PDS mean scores by group for time TO-T2 (SD)

TO: Baseline < 4 weeks before MDMA and after discontinuation of psychtropic medication
Active Placebo: 23.5 (1.9) Full Dose: 30 (6.3)

T2: 3 weeks post MDMA-session 3 (end of treatment)
Active Placebo: 30.8 (6.2) Full Dose: 21.4 (11.9)

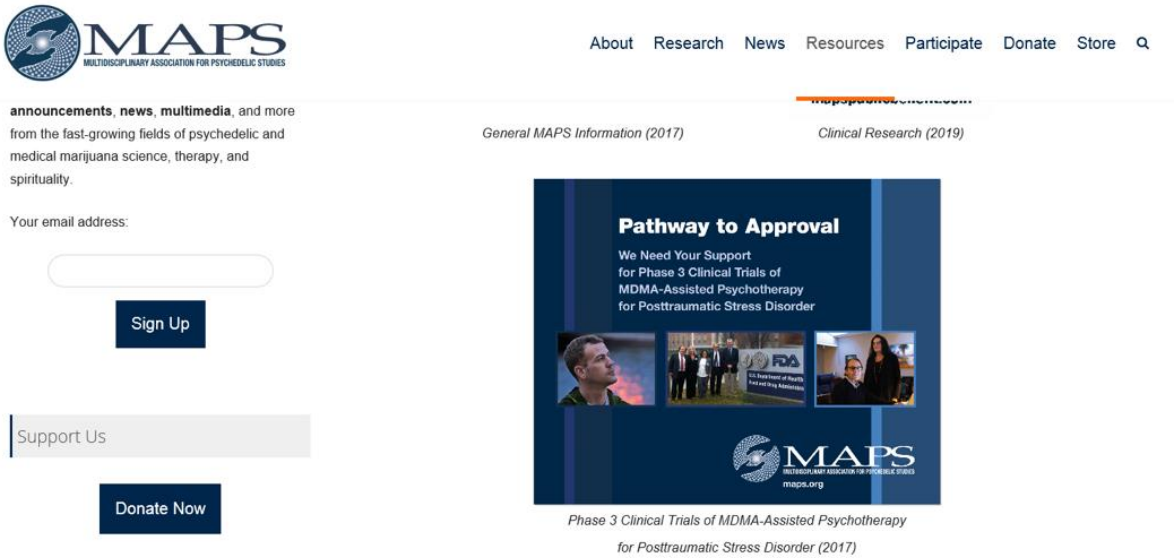
Changes scores T0-T2 (SD):
Active Placebo: 7.3 (6.2) Full dose: -8.6 (13.0)

Figure 2. CAPS and PDS scores by group for time T0-T2.

b) Fase-2 studies in USA, Israel en Canada (Ota'lora, 2018 en Mithoefer, 2018)

c) In 2019 starten de fase-3 studies in UK, Nederland, Duitsland en Tsjechië

d) Goedgekeurde behandeling in 2021?



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Developing MDMA into a Prescription Medicine

MAPS' top priority is developing MDMA-assisted psychotherapy into a legal, Food and Drug Administration (FDA) and European Medicines Agency (EMA)-approved treatment for PTSD caused by sexual assault, war, violent crime, or any other trauma. Our Phase 2 studies have shown that MDMA in conjunction with psychotherapy can help people overcome chronic, treatment-resistant PTSD. MDMA (3,4-methylenedioxymethamphetamine) is a semi-synthetic compound patented by Merck in 1914 and now in the public domain. MDMA decreases activity in the left amygdala (associated with fear and traumatic memories); increases activity in the frontal cortex; and promotes the release of oxytocin and prolactin, hormones associated with love and bonding. MDMA can decrease fear and defensiveness while increasing trust and empathy. In MDMA-assisted psychotherapy, MDMA is administered up to three times, in the context of a several month process of non-drug psychotherapy, unlike most medications for mental illnesses which are often taken daily for years.

Phase 3 Trials: MDMA-Assisted Psychotherapy for PTSD

Our FDA-regulated Phase 3 clinical trials of MDMA-assisted psychotherapy for PTSD are taking place at approximately 15 locations across the United States, Canada, and Israel. Preparations for Phase 3 trials in Europe are also underway through Phase 2 studies and negotiations with EMA. The Phase 3 clinical trials are assessing the efficacy and safety of MDMA-assisted psychotherapy in adult participants with severe PTSD. Over a 12-week treatment period, participants receive 12 non-drug preparatory and integration sessions, lasting 90 minutes each, along with three day-long psychotherapy sessions assisted by either MDMA or placebo. The primary endpoint is the Clinician Administered PTSD Scale (CAPS-5), as assessed by a blinded pool of independent raters. The trials are the final phase of research required by the FDA to gather adequate efficacy and safety data that could potentially lead to approval of MDMA as a legal prescription treatment for PTSD. If approved, MDMA will be required to be used in conjunction with psychotherapy under direct observation in an outpatient setting.

Completed Phase 2 Trials: MDMA-Assisted Psychotherapy for PTSD

On May 7, 2019, the overall results of six Phase 2 clinical trials of MDMA-assisted psychotherapy for the treatment of posttraumatic stress disorder (PTSD) were published in the peer-reviewed journal *Psychopharmacology*. The publication is the most comprehensive analysis of the safety and efficacy of MDMA-assisted psychotherapy for treatment of PTSD. Sponsored by MAPS, the double-blind, controlled Phase 2 trials in 103 subjects found that 54% of participants in the active treatment group (75–125 mg MDMA) did not qualify for a diagnosis of PTSD, compared to 23% in the control group (0–40 mg MDMA). In the published analysis, the course of double-blind treatment included two eight-hour MDMA-assisted psychotherapy sessions spaced three to five weeks apart, combined with weekly non-drug psychotherapy sessions. The trials were conducted by independent investigators in Charleston, South Carolina (two trials); Boulder, Colorado; Vancouver, British Columbia; Solothurn, Switzerland; and Beer Yaakov, Israel. Trial participants included men and women with chronic PTSD from a variety of causes.

Breakthrough Therapy Designation: MDMA-Assisted Psychotherapy for PTSD

On August 16, 2017, the FDA granted Breakthrough Therapy Designation to MDMA for the treatment of PTSD. The FDA grants this designation for treatments that (1) are intended alone or in combination with one or more other drugs to treat a serious or life-threatening disease or condition; and (2) preliminary clinical evidence indicates substantial improvement over existing therapies. A Breakthrough Therapy Designation signals that this treatment may have a meaningful advantage and greater compliance over available medications for PTSD.

Participate in Research

MAPS sponsors clinical trials around the world that offer eligible volunteers the opportunity to participate in our research studies. Our studies have strict enrollment criteria based on the goal of the study and the condition the study is investigating. To learn more, visit **maps.org/participate**.

Phase 3 Study Locations

Phase 3 clinical trials of MDMA-Assisted Psychotherapy for PTSD began in late 2018. The study aims to enroll 200–300 participants across approximately 15 sites in the U.S., Canada and Israel. Results will be submitted to the FDA in 2021 with approval expected in 2022.



Phase 3 Study Design



Timeline for Regulatory Approval



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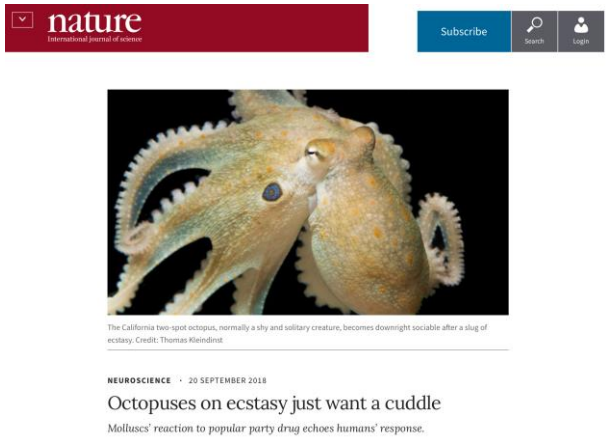
2. AUD

Bristol-Imperial MDMA in alcoholism study: 2 sessies met 125mg (+ 62,5mg) MDMA, 8w FU (Sessa, 2017)

Resultaten eind 2019???

3. Sociale angst bij ASS

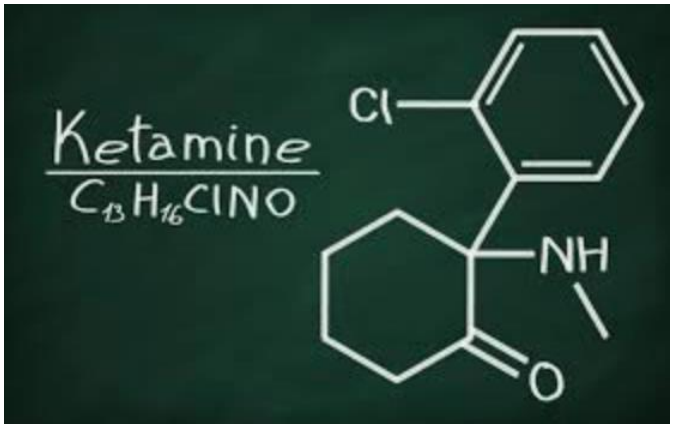
1 RCT, 75mg MDMA vs 125mg MDMA vs placebo, FU 6m (n=12) (Danforth, 2018)



Bijwerkingen

- Mortaliteit: 3/j bij illegaal gebruik, geen bij klinische studies (Schifano, 2003)
- Transiënte neurocognitieve effecten (geheugen deficits, PMV, verminderd executief functioneren, concentratiestoornissen)
- Afhankelijkheid zeldzaam
- Verhoogde bloeddruk en harstslag, verhoogde temperatuur, tandenknarsen, verminderde eetlust, verstoord evenwicht
- Vermoeidheid, gevoelens van irritatie en depressie zelden in klinische settings

Ketamine



- NMDA-receptor antagonist

- Stadia:
  - Empathogene ervaring (25-50mg IM): opgewekte stemming, verhoogd bewustzijn van lichaam
  - Out-of-body ervaring (75-125mg IM): onthechting van het lichaam
  - Ego-dissolving transcendentale ervaring (150-200mg IM): verdwijnen van de grenzen met de realiteit en van zz, bijna-dood ervaring

Effect en indicatie

1. Depressie

1. Kleine ongecontroleerde studies met orale ketamine (0.5 – 3mg/kg) van enkele tot 28 toedieningen
2. Positieve resultaten op vlak van effectiviteit en veiligheid
3. Systematic review en meta-analyse (McGirr, 2015): 7 RCT

   1 trial met ketamine intranasaal

   6 IV ketamine

2. Pijn

1. Orale toediening (tot 660 keer)
2. Bijwerkingen: misselijkheid, hoofdpijn, verwardheid, angst, hallucinaties

REDACTIONEEL

Offlabelbehandeling met ketamine voor therapieresistente depressie: mogelijkheden en knelpunten

S.Y. SMITH-APELDOORN, J.K.E. VERAART, R.A. SCHOEVERS

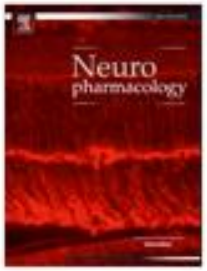
ARTIKEL

Dat toediening van ketamine een snel en robuust antidepressief effect kan hebben bij patiënten met een therapieresistente depressie is inmiddels via meerdere (meta-analytische) studies aangetoond (Han e.a. 2016; Kishimoto e.a. 2016), en is reeds in een eerder Redactioneel in dit tijdschrift beschreven (Vrieze 2018). Professor Vrieze beschreef tevens dat het effect van eenmalige toediening van ketamine bij het merendeel van de patiënten ongeveer een week aanhoudt, waarna terugval optreedt. De effectiviteit en de veiligheid van herhaalde toediening van ketamine zijn daarom onderwerp van onderzoek (o.a. Aan het Rot e.a. 2010).

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3. Verslaving

Accepted Manuscript



Ketamine for the treatment of addiction: Evidence and potential mechanisms

I. Ivan Ezquerra-Romano, W. Lawn, E. Krupitsky, C.J.A. Morgan

PII: S0028-3908(18)30017-0

DOI: 10.1016/j.neuropharm.2018.01.017

Reference: NP 7036

To appear in: *Neuropharmacology*

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- Salvador Roquet (Barney, 1977)

- Krupitsky et Grinenko



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Ketamine Psychedelic Therapy (KPT): A Review of the Results of Ten Years of Research

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Published online: 06 Sep 2011.

- Recent ontwende alcoholici 2.5mg/kg ketamine (n=111) vs. recent ontwende alcoholici met TAU (n=100): na 1 jaar was 66% abstinent tov 24% (Krupitsky et Grinenko, 1997)

- Recent ontwende heroine-afh patiënten (n=70): 0.2 mg/kg IM (n=35) vs. 2.0 mg/kg IM (n=35) (Krupitsky, 2002)
- Recent ontwende heroine-afh patiënten (n=59) kregen 1 KPT sessie, nadien 2 KPT sessies (n=26) vs 2 counseling sessies (n=27) (Krupitsky, 2007)

- Ongemotiveerde cocaine afhankelijke mannen (n=8) kregen 0.41mg/kg ketamine vs 0.71 mg/kg ketamine vs 2mg lorazepam (Dakwar, 2014)
- Ongemotiveerde cocaine afhankelijke deelnemers (n=20): 0.025 mg/kg midazolam vs 0.71 mg/kg ketamine (Dakwar, 2016)

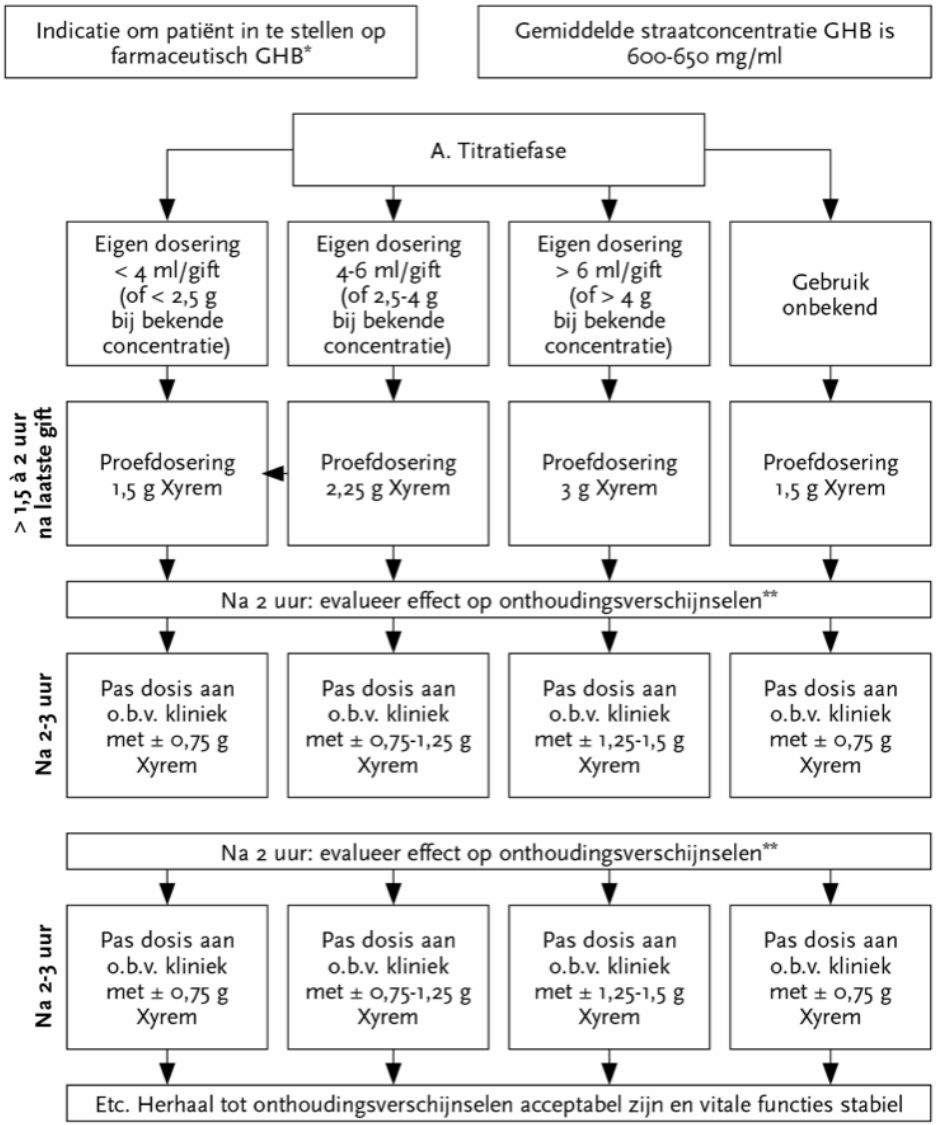
Bijwerkingen

- Tachycardie, palpitaties
- Hypertensie
- Nystagmus
- Mydriase
- Braken
- Cognitieve problemen
- Urologische problemen

GHB

- Xyrem (sodium gamma-hydroxybutyraat) voor kataplexie bij narcolepsie sinds 2004 (Owen, 2008)
- Ontwenningsschema GHB
  - Alleen in ziekenhuizen met voldoende ervaring
  - Startdosering ongeveer 75% van het gerapporteerde gebruik
  - .Verlaag per dag Xyrem met 3mL (=300-450mg)
  - Pas eventueel aan op geleide van het beeld en meet bloeddruk en pols tenminste vier maal daags

Figuur 18.1 Stroomschema Instellen op farmaceutische GHB (Bron: Van Noorden, et al. 2012)



* GHB zoals gebruikt in dit schema heeft een concentratie van 500 mg/ml (Xyrem)

** Onthoudingsverschijnselen: craving, agitatie, tremoren, zweten, moeheid, angst, koude rillingen, spiertrekkingen, loopneus, buikpijn, braken, hallucinaties, delier.

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Heroïne

- Nederland, Denemarken en Duitsland: heroïne op medisch voorschrift
- Zwitserland: ruim palet van opiaat-agonisten (methadon, buprenorfine, vertraagde afgifte morfine, medicinale heroïne
- Canada: 2 trials (NAOMi en SALOME) met heroïne op medisch voorschrift (ook hydromorfone-Diluadid beschikbaar)



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Telling our stories: heroin-assisted treatment and SNAP activism in the Downtown Eastside of Vancouver

Susan Boyd, Dave Murray, SNAP & Donald MacPherson

Harm Reduction Journal 14, Article number: 27 (2017) | Download Citation

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- Noorwegen: heroïne op voorschrift staat hoog op de (politieke/gezondheidszorg) agenda

Besluit

- Effectiviteit aanwezig
- Bijwerkingen beperkt
- Studies beperkt

- Individuele klinische aanpak?
- Maatschappelijke houding?
- Onderzoek?

REDACTIONEEL

Therapeutisch gebruik van illegale drugs

M. DESTOOP

ARTIKEL

Twee maanden geleden zette een patiënt me aan het denken over het therapeutisch gebruik van illegale drugs. De jongeman van 23 jaar met blijvende psychotische en cognitieve symptomen na jarenlang misbruik van cannabis vroeg tijdens de consultatie mijn advies betreffende het gebruik van cannabidiol(CBD)-olie. Ondanks behandeling met amisulpride bleef hij last hebben van paranoïde symptomen en auditieve hallucinaties, die verminderden na inname van CBD. Via internet vond hij artikelen die wijzen op de gunstige effecten van CBD op het cognitief functioneren, wat hem zou kunnen helpen in de voorbereiding van zijn universitaire studies.

Tot mijn verbazing vond ik inderdaad gedegen wetenschappelijke evidentie voor het therapeutisch gebruik van illegale drugs, waaronder cannabidiol. Tijd voor een korte stand van zaken over het therapeutisch gebruik van illegale drugs:

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British Journal of Pharmacology

British Journal of Pharmacology (2017) **174** 3257–3267 3257

Themed Section: Pharmacology of Cognition: a Panacea for Neuropsychiatric Disease?

REVIEW ARTICLE

Lifestyle use of drugs by healthy people for enhancing cognition, creativity, motivation and pleasure

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Drug harm ranking

Nutt et al Lancet 2007

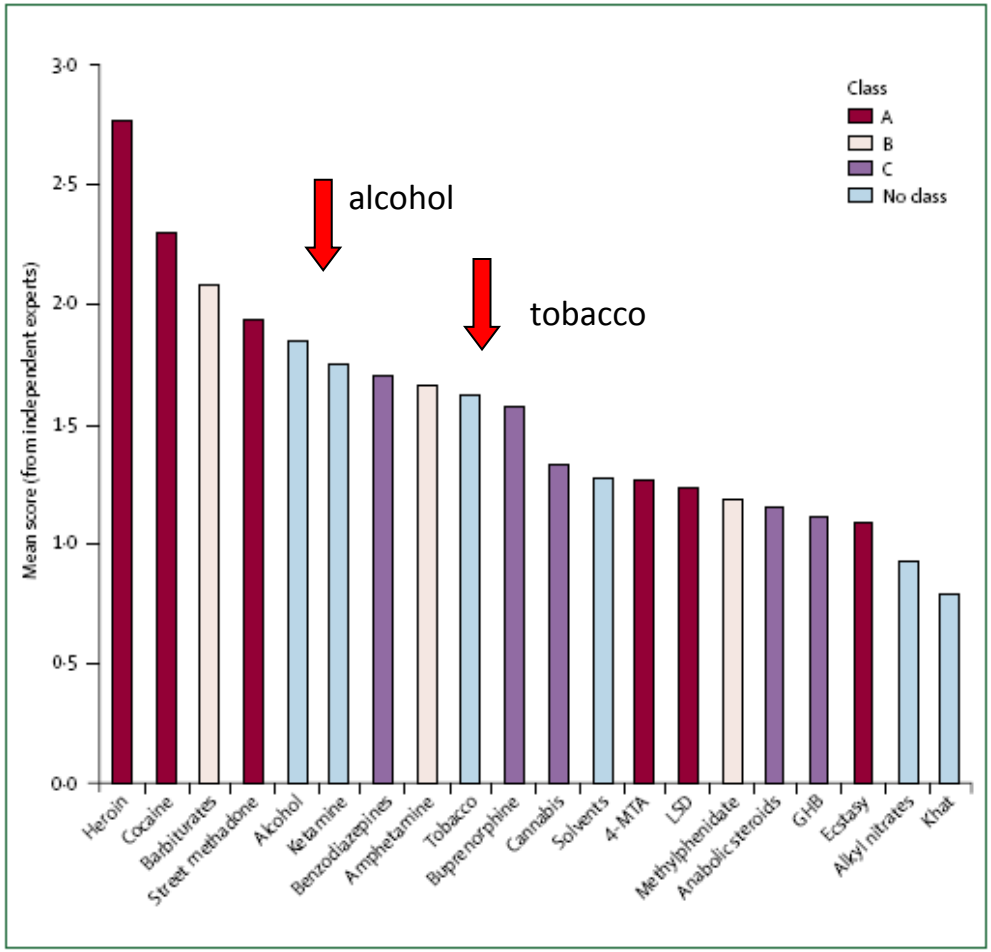


Figure 1: **Mean harm scores for 20 substances**
The respective classification under the Misuse of Drugs Act, where appropriate, is shown above each bar. Class A drugs are indicated by black bars, B by dark grey, and C by light grey. Unclassified substances are shown as unfilled bars.

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Drugs: regulation & harm. Prof D Nutt

https://www.youtube.com/watch?v=2TookjqAqF4



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