Justin Sinclair

AMCREC Lead Coordinator; Research Fellow

Medicinal Cannabis Expert and Pharmacognosist

Saturday, August 10, 2019

(Plenary)

11:20 - 11:40 Myths and Misconceptions Around Cannabis

NICM Health Research Institute

WESTERN SYDNEY UNIVERSITY

AUSTRALIAN MEDICINAL CANNABIS RESEARCH & EDUCATION COLLABORATION

A NICM HEALTH RESEARCH INITIATIVE

Myths & misconceptions around medicinal cannabis.

South GP CME Conference, New Zealand Medical Association, Christchurch NZ

10th August 2019

JUSTIN SINCLAIR

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Disclosures

Justin Sinclair (Pharmacognosist)

PhD student (WSU) MHerbMed (USyd) BHSc DBM DNut

FNHAA MICRS MSCC

- Research Fellow – NICM Health Research Institute (Western Sydney University).
- Coordinator, Australian Medicinal Cannabis Research & Education Collaboration (AMCREC).
- Former Scientific Advisory Board Member – Bioceuticals (Resigned April 2019).
- Scientific Advisory Board Member – United in Compassion (Registered Charity in MC patient advocacy – *pro bono*).

NICM Campus, WSU (Westmead NSW)

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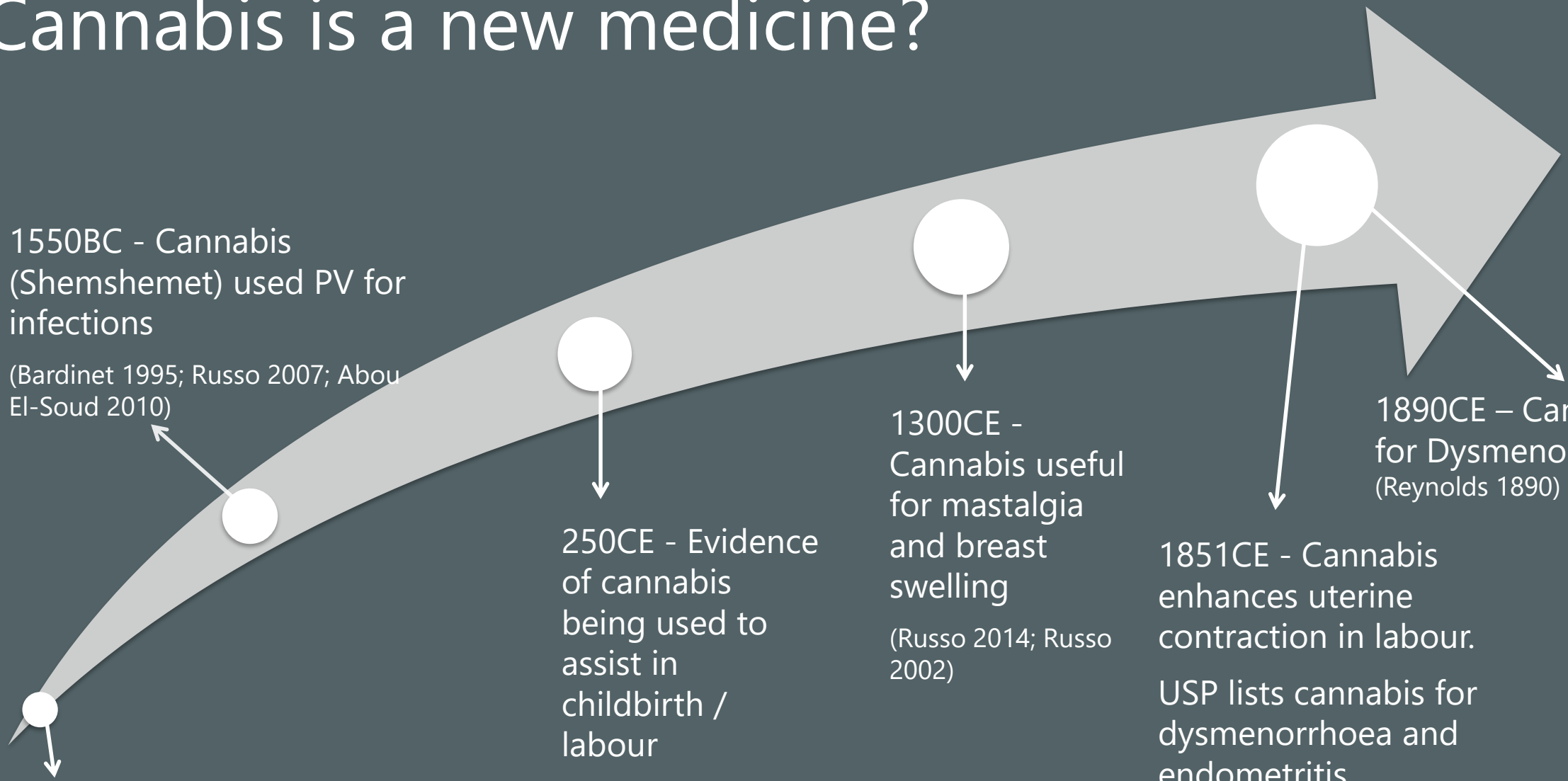
Cannabis is a new medicine?



2800BC - Cannabis mentioned as treatment for disorders of the female reproductive system

(Touwn 1981; Russo 2014)

1550BC - Cannabis (Shemshemet) used PV for infections

(Bardinet 1995; Russo 2007; Abou El-Soud 2010)

250CE - Evidence of cannabis being used to assist in childbirth / labour

(Russo 2014; Zias 1993)

1300CE - Cannabis useful for mastalgia and breast swelling

(Russo 2014; Russo 2002)

1851CE - Cannabis enhances uterine contraction in labour.

USP lists cannabis for dysmenorrhoea and endometritis

(Christison 1851; Russo 2014)

1890CE – Cannabis used for Dysmenorrhoea (Reynolds 1890)

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Cannabis is a new medicine? 1870-1890

Condition (Cannabis used)	Reference
Melancholia, obsession, anxiety	Polli (1870)
Mental depression with insomnia	Strange (1883)
Migraine prophylaxis, dysuria, dysmenorrhoea and urinary retention	Ringer (1886)
Advantages over opiates in pain.	Hare (1887)
Chronic daily headache	Mackenzie (1887)
Superiority in migraine & tremor (Parkinson's Disease)	Gowers (1888)
Migraine, senility, dysmennorhoea, childhood convulsions (epilepsy)	Reynolds (1890)

Table 1: Summary of the noted medical findings of Cannabis use between the years 1870 and 1890 (Russo 2014)

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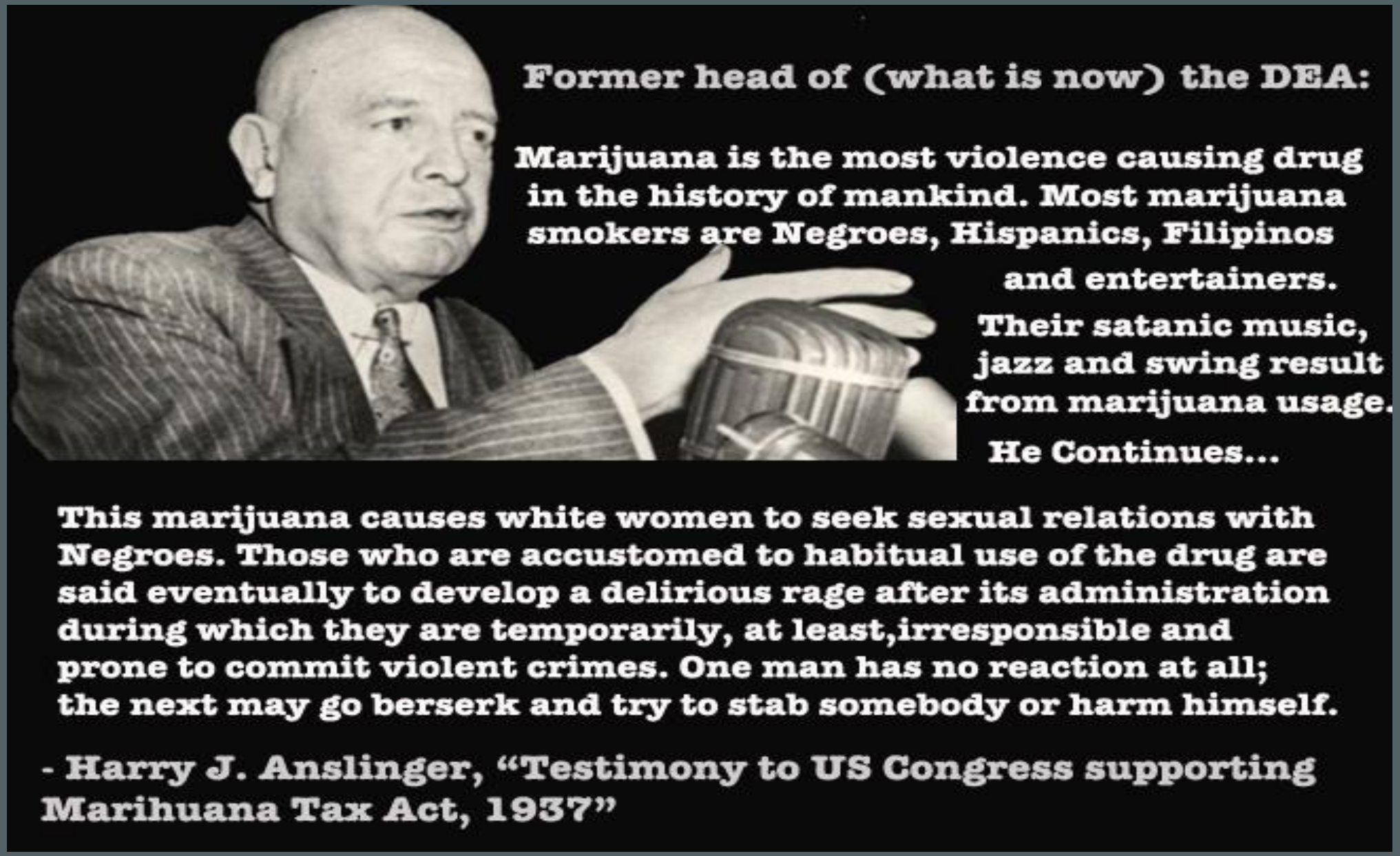
Cannabis prohibition 1920-1937



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Cannabis prohibition 1920-1937

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Cannabis lowers your IQ?

Figure 1: The female inflorescence (bud) of *Cannabis spp.*

Cannabis may cause short term memory impairment whilst under the influence.

This is considered reversible upon cessation...not permanent.

Certain Cannabis phytochemicals such as Cannabidiol (CBD) actually exhibit neuroprotective activity.

Animal studies are now showing :

- Foetal hypoxia (Alvarez et al. 2008)
- Multiple sclerosis
- Hypoxic brain injury (Ischaemic stroke)
- Alzheimer's Disease (Ramirez et al. 2005)

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Cannabis lowers your IQ?

Proc Natl Acad Sci U S A. 2016 Feb 2;113(5):E500-8. doi: 10.1073/pnas.1516648113. Epub 2016 Jan 19.

Impact of adolescent marijuana use on intelligence: Results from two longitudinal twin studies.

Jackson NJ[1], Isen JD[2], Khoddam R[3], Irons D[4], Tuvblad C[5], Iacono WG[4], McGue M[4], Raine A[6], Baker LA[3].

⊕ **Author information**

Abstract

Marijuana is one of the most commonly used drugs in the United States, and use during adolescence--when the brain is still developing--has been proposed as a cause of poorer neurocognitive outcome. Nonetheless, research on this topic is scarce and often shows conflicting results, with some studies showing detrimental effects of marijuana use on cognitive functioning and others showing no significant long-term effects. The purpose of the present study was to examine the associations of marijuana use with changes in intellectual performance in two longitudinal studies of adolescent twins (n = 789 and n = 2,277). We used a quasiexperimental approach to adjust for participants' family background characteristics and genetic propensities, helping us to assess the causal nature of any potential associations. Standardized measures of intelligence were administered at ages 9-12 y, before marijuana involvement, and again at ages 17-20 y. Marijuana use was self-reported at the time of each cognitive assessment as well as during the intervening period. Marijuana users had lower test scores relative to nonusers and showed a significant decline in crystallized intelligence between preadolescence and late adolescence. However, there was no evidence of a dose-response relationship between frequency of use and intelligence quotient (IQ) change. Furthermore, marijuana-using twins failed to show significantly greater IQ decline relative to their abstinent siblings. Evidence from these two samples suggests that observed declines in measured IQ may not be a direct result of marijuana exposure but rather attributable to familial factors that underlie both marijuana initiation and low intellectual attainment.

KEYWORDS: adolescence; intelligence; longitudinal; marijuana use; twins

(Jackson *et al* 2016)

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Cannabis lowers your IQ?

Addiction. 2018 Feb;113(2):257-265. doi: 10.1111/add.13946. Epub 2017 Sep 5.

Associations between adolescent cannabis use and neuropsychological decline: a longitudinal co-twin control study.

Meier MH[1], Caspi A[2,3,4], Danese A[4,5,6], Fisher HL[4], Houts R[2,3], Arseneault L[4], Moffitt TE[2,3,4].

⊕ Author information

Abstract

AIMS: This study tested whether adolescents who used cannabis or met criteria for cannabis dependence showed neuropsychological impairment prior to cannabis initiation and neuropsychological decline from before to after cannabis initiation.

DESIGN: A longitudinal co-twin control study.

SETTING AND PARTICIPANTS: Participants were 1989 twins from the Environmental Risk (E-Risk) Longitudinal Twin Study, a nationally representative birth cohort of twins born in England and Wales from 1994 to 1995.

MEASUREMENTS: Frequency of cannabis use and cannabis dependence were assessed at age 18. Intelligence quotient (IQ) was obtained at ages 5, 12 and 18. Executive functions were assessed at age 18.

FINDINGS: Compared with adolescents who did not use cannabis, adolescents who used cannabis had lower IQ in childhood prior to cannabis initiation and lower IQ at age 18, but there was little evidence that cannabis use was associated with IQ decline from ages 12-18. For example, adolescents with cannabis dependence had age 12 and age 18 IQ scores that were 5.61 ($t = -3.11$, $P = 0.002$) and 7.34 IQ points ($t = -5.27$, $P < 0.001$) lower than adolescents without cannabis dependence, but adolescents with cannabis dependence did not show greater IQ decline from age 12-18 ($t = -1.27$, $P = 0.20$). Moreover, adolescents who used cannabis had poorer executive functions at age 18 than adolescents who did not use cannabis, but these associations were generally not apparent within twin pairs. For example, twins who used cannabis more frequently than their co-twin performed similarly to their co-twin on five of six executive function tests ($Ps > 0.10$). The one exception was that twins who used cannabis more frequently than their co-twin performed worse on one working memory test (Spatial Span reversed; $\beta = -0.07$, $P = 0.036$).

CONCLUSIONS: Short-term cannabis use in adolescence does not appear to cause IQ decline or impair executive functions, even when cannabis use reaches the level of dependence. Family background factors explain why adolescent cannabis users perform worse on IQ and executive function tests.

(Meier *et al* 2018)

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Cannabis lowers your IQ?

Associations between adolescent cannabis use frequency and adult brain structure : A prospective study of boys followed to adulthood.

Madeline H. Meier, Roberta A. Schriber, Jordan Beardslee, Jamie Hanson, Dustin Pardini

Drug and Alcohol Dependence, 2019, 202, 191-199

PMID : 31357120
DOI : 10.1016/j.drugalcdep.2019.05.012

Abstract

BACKGROUND : Few studies have tested the hypothesis that adolescent cannabis users show structural brain alterations in adulthood. The present study tested associations between prospectively-assessed trajectories of adolescent cannabis use and adult brain structure in a sample of boys followed to adulthood.

(Meier *et al* 2019)

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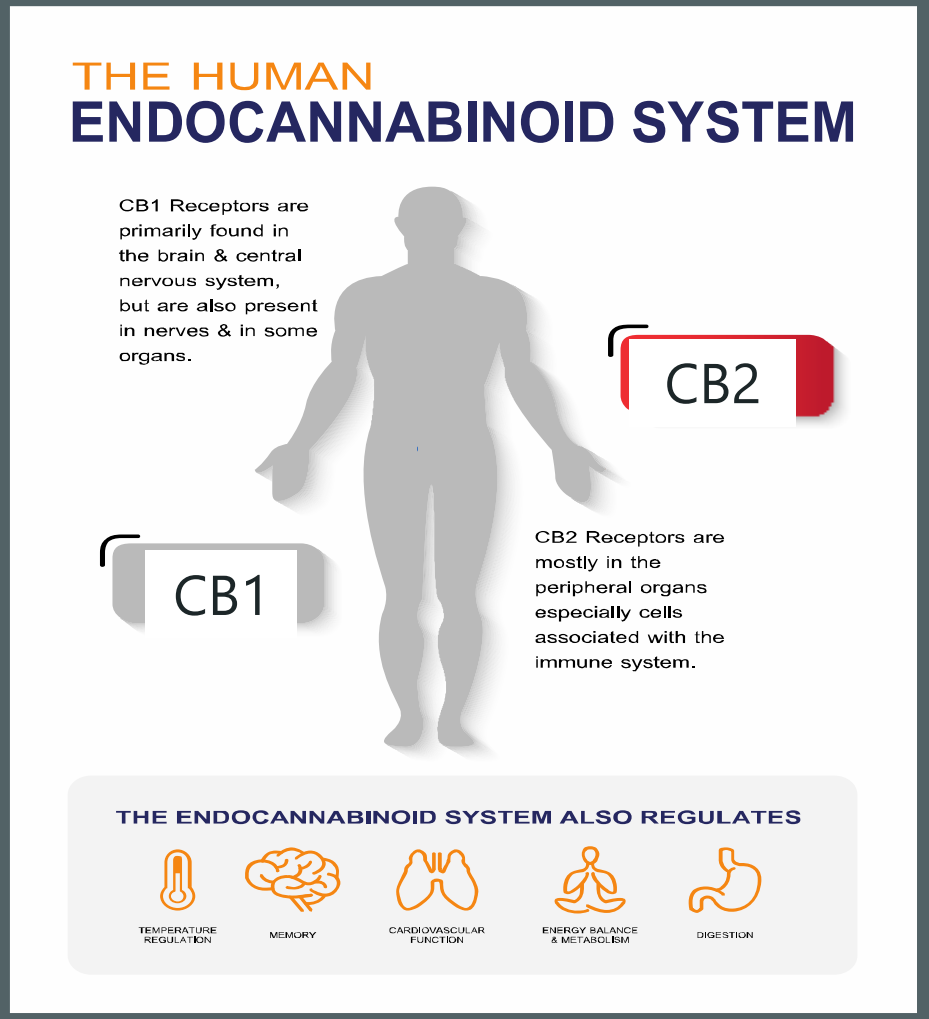
All Cannabis gets you “high”?

- The intoxicating effect of cannabis is largely dependent on the phytochemistry exhibited in the specific chemovar of the plant.
- There exist numerous chemovars of Cannabis that have been selectively bred to be low in THC, but higher in other phytochemicals such as CBD.
- Individual dosing (titration) and appropriate Cannabis chemovar selection / dosage form is key to reducing intoxicating effects.



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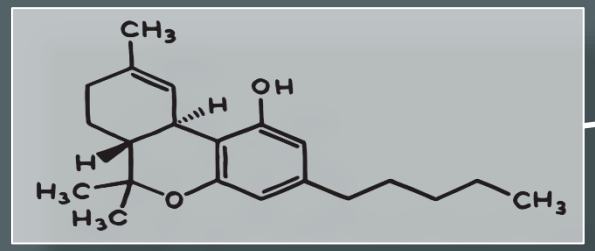
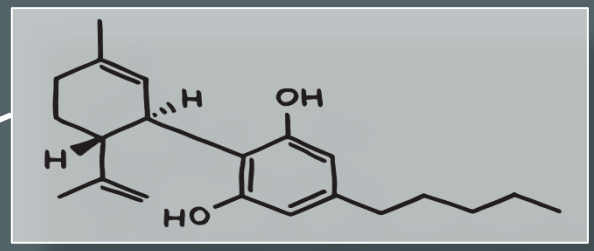
CBD is medicinal & THC is recreational?



Tetrahydrocannabinol (THC) Structure

Partial agonist
CB1 / CB2



Cannabidiol (CBD) Structure

Agonist of:

5HT1A

TRPV1

TRPV2

modulates:

α1 & α3 Glycine Receptors

TRPA1

PPARs

Pharmacological actions of THC
Analgesic (Rahn & Hohmann, 2009)
Antiemetic (Haney et al. 2007; Hollister 1971; Machado et al. 2008)
Anti-inflammatory (Hampson et al. 1998)
Antipruritic (Neff et al. 2002)
Bronchodilator (Williams et al. 1976)
Muscle relaxant (Kavia et al. 2010)
Antioxidant, Neuroprotective (Hampson et al. 1998)
↓ symptoms of Alzheimer's (Eubanks et al. 2006)

Pharmacological actions of CBD
Anticonvulsant (Jones et al. 2010)
Analgesic (Davis & Hartoum, 1983)
Anti-inflammatory (Booz, 2011)
Antiemetic / Antinausea (Rock et al. 2010)
Anxiolytic (Russo et al. 2005; Campos & Guimares, 2008)
Antioxidant (Hampson et al. 1998)
Neuroprotective (Hampson et al. 1998)

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CBD is non-psychoactive?

Neurochemical Research
August 2005, Volume 30, Issue 8, pp 1037–1043 | Cite as

Agonistic Properties of Cannabidiol at 5-HT1a Receptors

Authors | Authors and affiliations

Ethan B. Russo, Andrea Burnett, Brian Hall, Keith K. Parker

Neuropharmacology. 2016 Apr;103:16-26. doi: 10.1016/j.neuropharm.2015.12.017. Epub 2015 Dec 19.

Cannabidiol induces rapid-acting antidepressant-like effects and enhances cortical 5-HT/glutamate neurotransmission: role of 5-HT1A receptors.

Linge R[1], Jiménez-Sánchez L[2], Campa L[2], Pilar-Cuéllar F[1], Vidal R[1], Pazos A[1], Adell A[3], Díaz Á[4].

CNS Neurol Disord Drug Targets. 2014;13(6):953-60.

Antidepressant-like and anxiolytic-like effects of cannabidiol: a chemical compound of Cannabis sativa.

de Mello Schier AR, de Oliveira Ribeiro NP, Coutinho DS, Machado S, Arias-Carrión O, Crippa JA, Zuardi AW, Nardi AE, Silva AC[1].

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Cannabis is a drug of dependence?

Experimental and Clinical Psychopharmacology
1994, Vol. 2, No. 3, 244-268

Comparative Epidemiology of Dependence on Tobacco, Alcohol, Controlled Substances, and Inhalants: Basic Findings From the National Comorbidity Survey

James C. Anthony, Lynn A. Warner, and Ronald C. Kessler

Studying prevalence of *Diagnostic and Statistical Manual* (3rd ed., rev., American Psychiatric Association, 1987) drug dependence among Americans 15–54 years old, we found about 1 in 4 (24%) had a history of tobacco dependence; about 1 in 7 (14%) had a history of alcohol dependence; and about 1 in 13 (7.5%) had a history of dependence on an inhalant or controlled drug. About one third of tobacco smokers had developed tobacco dependence and about 15% of drinkers had become alcohol dependent. Among users of the other drugs, about 15% had become dependent. Many more Americans age 15–54 have been affected by dependence on psychoactive substances than by other psychiatric disturbances now accorded a higher priority in mental health service delivery systems, prevention, and sponsored research programs.

The aim of this article is to report basic descriptive findings from new research on the epidemiology of drug dependence syndromes, conducted as part of the National Comorbidity Survey (NCS). In this study, our research team secured a nationally representative sample and applied standardized diagnostic assessments in a way that allows direct comparisons across prevalence estimates and correlates of tobacco dependence, alcohol dependence, and dependence on other psychoactive drugs (Kessler et al., 1994).

For this overview of the survey's findings, a primary goal has been to answer two basic epidemiologic questions about drug dependence involving tobacco, alcohol, controlled drugs such as cocaine, and inhalants: First, in the population under study, what proportion of persons now qualifies as a currently active or former case of drug dependence? Second, where are the affected cases more likely to be found within the sociodemographic structure of the study population?

In addition, population estimates presented in this article shed light on the epidemiology of dependence on tobacco, alcohol, and the following individual drugs and drug groups: cannabis; heroin; cocaine; psychostimulants other than cocaine; analgesic drugs; a drug group consisting of anxiolytic, sedative, and hypnotic drugs; psychedelic drugs; and inhalant drugs. The following population estimates are presented for each of these listed drugs, including tobacco and alcohol: (a) lifetime prevalence of drug dependence, evaluated in relation to criteria published in the *Diagnostic and Statistical Manual of Mental Disorders*, Third Edition, Revised (DSM–III–R; American Psychiatric Association, 1987); (b) lifetime prevalence of extramedical drug use, defined to encompass illicit drug use as well as patients taking prescribed medicines to get

James C. Anthony, Etiology Branch, Addiction Research Center, National Institute on Drug Abuse and Johns Hopkins University; Lynn A. Warner and Ronald C. Kessler, Institute for Social Research and Department of Sociology, University of Michigan.

The National Comorbidity Survey (NCS) is a collaborative epidemiologic investigation of the prevalence, causes, and consequences of psychiatric morbidity and comorbidity in the United States. The NCS is supported by U.S. Public Health Service Grants MH 46376 and MH 49098 with supplemental support from the National Institute on Drug Abuse and W. T. Grant Foundation Grant 90135190.

Preparation of this article was supported by the National Institute on Drug Abuse Addiction Research Center. We acknowledge H. Chilcoat for valuable research assistance.

Correspondence concerning this article may be addressed to James C. Anthony, P. O. Box 5180, Addiction Research Center, National Institute on Drug Abuse, Baltimore, Maryland 21224. Electronic mail may be sent to anthony@jhuhyg.sph.jhu.edu.

(Anthony, Warner & Kessler 1994).

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Cannabis is a drug of dependence?

COMPARATIVE EPIDEMIOLOGY OF DRUGS

Table 2
Estimated Prevalence of Extramedical Use and Dependence in Total Study Population and Lifetime Dependence Among Users

Drug categories	Proportion with a history of dependence		Proportion with a history of extramedical use		Dependence among extramedical users	
	P	*SE*	*P*	*SE*	*P*	*SE*
Tobacco[a]	24.1	1.0	75.6	0.6	31.9	–
Alcohol	14.1	0.7	91.5	0.5	15.4	0.7
Other drugs	7.5	0.4	51.0	1.0	14.7	0.7
Cannabis	4.2	0.3	46.3	1.1	9.1	0.7
Cocaine	2.7	0.2	16.2	0.6	16.7	1.5
Stimulant	1.7	0.3	15.3	0.7	11.2	1.6
Anxiolytics, etc.[b]	1.2	0.2	12.7	0.5	9.2	1.1
Analgesics	0.7	0.1	9.7	0.5	7.5	1.0
Psychedelics	0.5	0.1	10.6	0.6	4.9	0.7
Heroin	0.4	0.1	1.5	0.2	23.1	5.6
Inhalants	0.3	0.1	6.8	0.4	3.7	1.4

Note. Weighted estimates from the National Comorbidity Survey data gathered in 1990–1992 for persons 15–54 years old (n = 8,098). Dash indicates data not estimated. P = Estimated prevalence proportion.
[a]n = 4,414. [b]Anxiolytics, sedatives, and hypnotic drugs, grouped.

(Anthony, Warner & Kessler 1994).

Cannabis dependency does exist but is also dependent on the individual.

Factors such as individual polymorphic expression, individual variability, the strain of Cannabis being utilised and the dosage taken are also important contributing factors.

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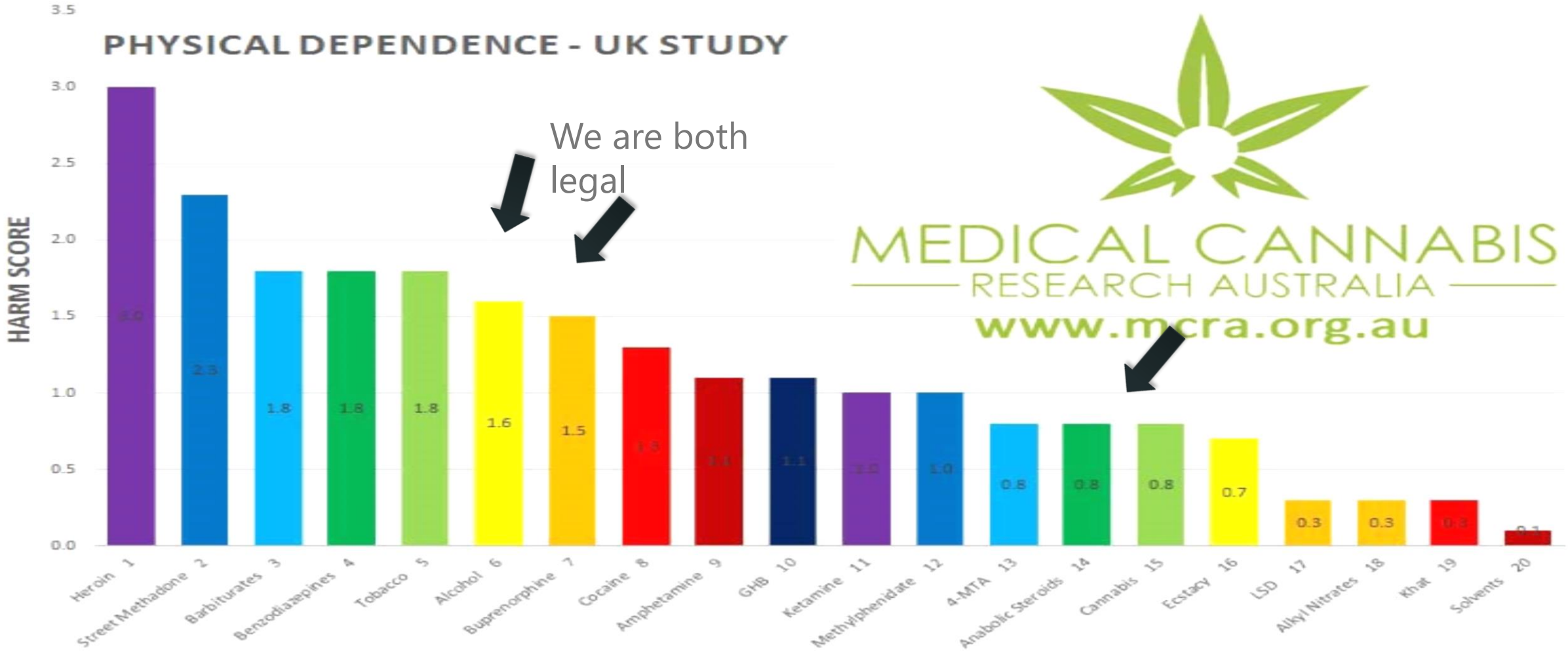
Cannabis is a drug of dependence?



Cannabis is low on the addiction scale. Heroin is the most addictive substance with a harm score of 3. Cannabis is half as addictive than alcohol (1.6) and slightly lower than caffeine/coffee (not on this scale but around (0.9).

Cannabis is a gateway drug?

Harm Reduction Journal

BioMed Central

Research

Open Access

Cannabis as a substitute for alcohol and other drugs

Amanda Reiman

Address: School of Social Welfare, University of California, Berkeley, 120 Haviland Hall, Berkeley, CA 94720, USA

Email: Amanda Reiman - areiman@berkeley.edu

Published: 3 December 2009

Harm Reduction Journal 2009, **6**:35 doi:10.1186/1477-7517-6-35

Received: 28 September 2009

Accepted: 3 December 2009

This article is available from: http://www.harmreductionjournal.com/content/6/1/35

Abstract

Background: Substitution can be operationalized as the conscious choice to use one drug (legal or illicit) instead of, or in conjunction with, another due to issues such as: perceived safety; level of addiction potential; effectiveness in relieving symptoms; access and level of acceptance. This practice of substitution has been observed among individuals using cannabis for medical purposes. This study examined drug and alcohol use, and the occurrence of substitution among medical cannabis patients.

Methods: Anonymous survey data were collected at the Berkeley Patient's Group (BPG), a medical cannabis dispensary in Berkeley, CA. (N = 350) The sample was 68% male, 54% single, 66% White, mean age was 39; 74% have health insurance (including MediCal), 41% work full time, 81% have completed at least some college, 55% make less than $40,000 a year. Seventy one percent report having a chronic medical condition, 52% use cannabis for a pain related condition, 75% use cannabis for a mental health issue.

Results: Fifty three percent of the sample currently drinks alcohol, 2.6 was the average number of drinking days per week, 2.9 was the average number of drinks on a drinking occasion. One quarter currently uses tobacco, 9.5 is the average number of cigarettes smoked daily. Eleven percent have used a non-prescribed, non OTC drug in the past 30 days with cocaine, MDMA and Vicodin reported most frequently. Twenty five percent reported growing up in an abusive or addictive household. Sixteen percent reported previous alcohol and/or drug treatment, and 2% are currently in a 12-step or other recovery program. Forty percent have used cannabis as a substitute for alcohol, 26% as a substitute for illicit drugs and 66% as a substitute for prescription drugs. The most common reasons given for substituting were: less adverse side effects (65%), better symptom management (57%), and less withdrawal potential (34%) with cannabis.

Conclusion: The substitution of one psychoactive substance for another with the goal of reducing negative outcomes can be included within the framework of harm reduction. Medical cannabis patients have been engaging in substitution by using cannabis as an alternative to alcohol, prescription and illicit drugs.

Background

It has been observed that those who use large amounts of cannabis frequently use other drugs as well, especially alcohol. This can create a potential synergistic effect, resulting in increased harms [1-4]. Economic research has looked at the substitution and complimentarity of particular substances by modelling the effects of price fluctuation on use, although the limits of such research have

(Reiman 2009)

A Gateway drug is defined as "one that apparently can lead to the use of harder, more addictive or dangerous drugs".

Examples of hard drugs may include:

- Heroine
- Methamphetamine
- Cocaine

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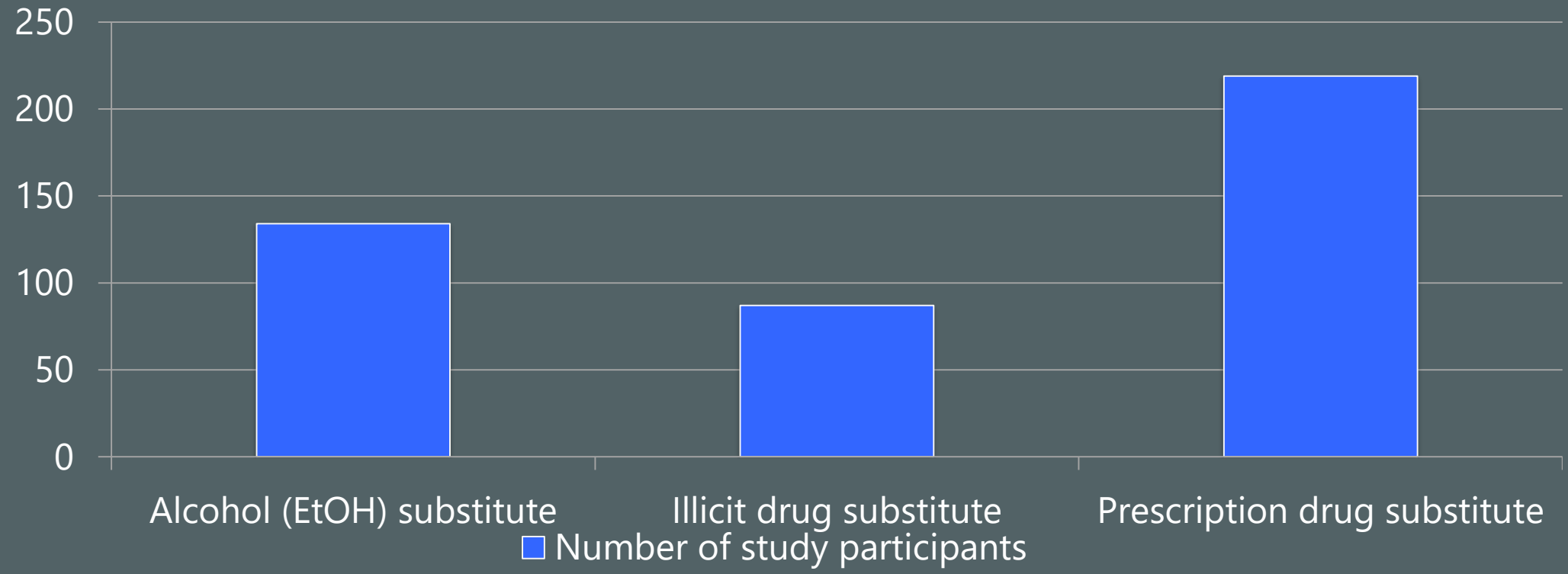
Cannabis is a gateway drug?

Table 2: Percent of sample reporting using cannabis as a substitute. (Reiman 2009)

Type of substitution	# of Participants	% of Participants
EtOH substitute	n = 134	40%
Illicit drug substitute	n= 87	26%
Prescription drug substitute	n= 219	65.8%



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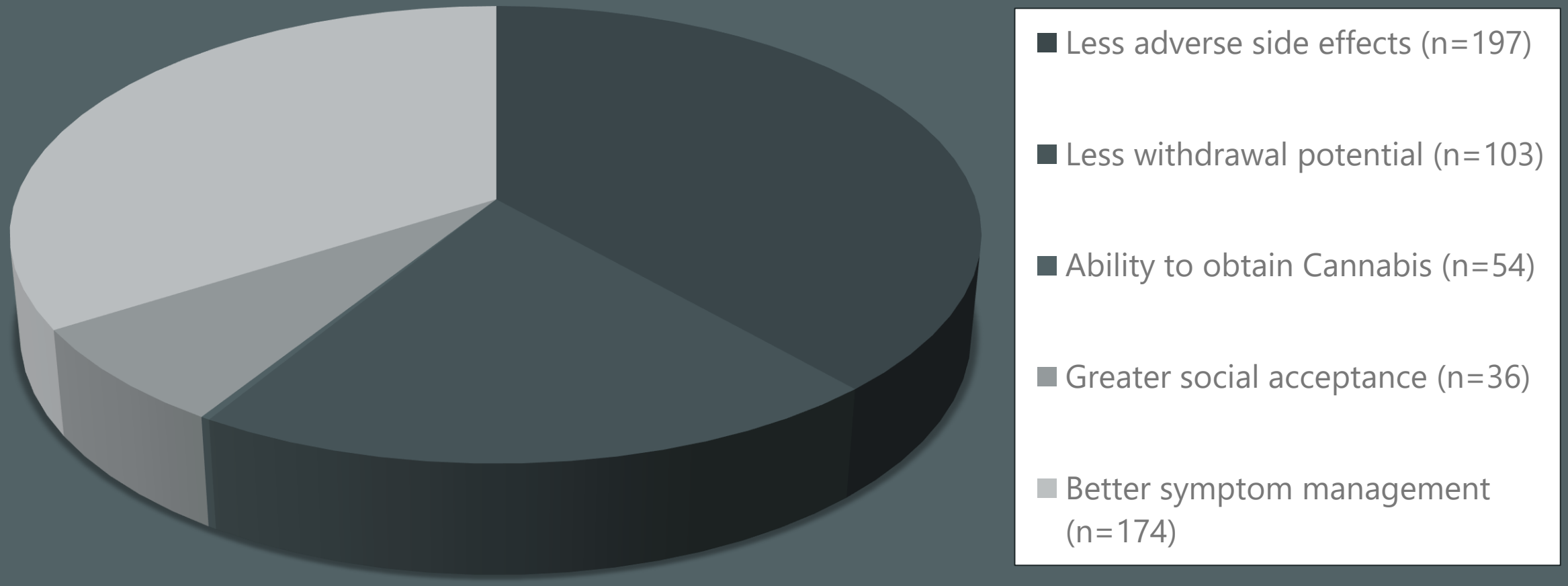
Cannabis is a gateway drug?

Reasons for using cannabis as a substitute



(Reiman 2009)

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Cannabis is a gateway drug?

Drug types	2009	2010	2011	2012	2013	2014	2015
All overdose deaths	379	342	362	367	380	387	453
Pharmaceutical	295	266	275	306	313	316	358
Illegal	147	149	153	133	166	164	227
Alcohol	94	85	88	80	94	94	106

Table 2. Overdose deaths by individual contributing drugs 2009–15, opioids and benzodiazepines[8]

Drug types	2009	2010	2011	2012	2013	2014	2015
All overdose deaths	379	342	362	367	380	387	453
Contributing drug: benzodiazepines	**160**	**169**	**180**	**199**	**212**	**215**	**238**
Diazepam	104	109	124	133	164	169	192
Oxazepam	18	19	44	41	17	19	34
Alprazolam	62	56	43	57	45	28	23
Clonazepam	7	9	14	18	19	25	33
Contributing drug: opioids	**177**	**145**	**183**	**212**	**192**	**186**	**199**
Oxycodone	41	39	46	46	61	46	58
Codeine	76	57	66	93	71	54	64
Morphine	22	11	10	13	7	12	8
Methadone	50	55	72	75	70	67	67
Buprenorphine	3	4	14	4	3	7	4

Reproduced from Coroners Court of Victoria. Findings Case 408012, Attachment C. Coroners Prevention Unit, Coroners Prevention Unit Data Summary, Re: Victorian Overdose Death 2009–2015. Last revised 30th August 2016. Available at www.coronerscourt.vic.gov.au/home/coroners+written+findings [Accessed 6 November 2016].

Cannabis is a gateway drug?

JAMA Intern Med. 2014 Oct;174(10):1668-73. doi: 10.1001/jamainternmed.2014.4005.

Medical cannabis laws and opioid analgesic overdose mortality in the United States, 1999-2010.

Bachhuber MA[1], Saloner B[2], Cunningham CO[3], Barry CL[4].

⊕ Author information

Erratum in
JAMA Intern Med. 2014 Nov;174(11):1875.

Abstract

IMPORTANCE: Opioid analgesic overdose mortality continues to rise in the United States, driven by increases in prescribing for chronic pain. Because chronic pain is a major indication for medical cannabis, laws that establish access to medical cannabis may change overdose mortality related to opioid analgesics in states that have enacted them.

OBJECTIVE: To determine the association between the presence of state medical cannabis laws and opioid analgesic overdose mortality.

DESIGN, SETTING, AND PARTICIPANTS: A time-series analysis was conducted of medical cannabis laws and state-level death certificate data in the United States from 1999 to 2010; all 50 states were included.

EXPOSURES: Presence of a law establishing a medical cannabis program in the state.

MAIN OUTCOMES AND MEASURES: Age-adjusted opioid analgesic overdose death rate per 100 000 population in each state. Regression models were developed including state and year fixed effects, the presence of 3 different policies regarding opioid analgesics, and the state-specific unemployment rate.

RESULTS: Three states (California, Oregon, and Washington) had medical cannabis laws effective prior to 1999. Ten states (Alaska, Colorado, Hawaii, Maine, Michigan, Montana, Nevada, New Mexico, Rhode Island, and Vermont) enacted medical cannabis laws between 1999 and 2010. States with medical cannabis laws had a 24.8% lower mean annual opioid overdose mortality rate (95% CI, -37.5% to -9.5%; P = .003) compared with states without medical cannabis laws. Examination of the association between medical cannabis laws and opioid analgesic overdose mortality in each year after implementation of the law showed that such laws were associated with a lower rate of overdose mortality that generally strengthened over time: year 1 (-19.9%; 95% CI, -30.6% to -7.7%; P = .002), year 2 (-25.2%; 95% CI, -40.6% to -5.9%; P = .01), year 3 (-23.6%; 95% CI, -41.1% to -1.0%; P = .04), year 4 (-20.2%; 95% CI, -33.6% to -4.0%; P = .02), year 5 (-33.7%; 95% CI, -50.9% to -10.4%; P = .008), and year 6 (-33.3%; 95% CI, -44.7% to -19.6%; P < .001). In secondary analyses, the findings remained similar.

CONCLUSIONS AND RELEVANCE: Medical cannabis laws are associated with significantly lower state-level opioid overdose mortality rates. Further investigation is required to determine how medical cannabis laws may interact with policies aimed at preventing opioid analgesic overdose.

Comment in
What ecologic analyses cannot tell us about medical marijuana legalization and opioid pain medication mortality. [JAMA Intern Med. 2015]
What ecologic analyses cannot tell us about medical marijuana legalization and opioid pain medication mortality--reply. [JAMA Intern Med. 2015]
Legalization of medical marijuana and incidence of opioid mortality. [JAMA Intern Med. 2014]

PMID: 25154332 PMCID: PMC4392651 DOI: 10.1001/jamainternmed.2014.4005

[PubMed - indexed for MEDLINE] **Free PMC Article**

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Cannabis versus opioid medication

Harm Reduct J. 2017; 14: 58.
Published online 2017 Aug 18. doi: 10.1186/s12954-017-0183-9
PMCID: PMC5563007

Rationale for cannabis-based interventions in the opioid overdose crisis

Philippe Lucas[1,2,3]

Author information ► Article notes ► Copyright and License information ►

Abstract

Go to:

Background

North America is currently in the grips of a crisis rooted in the use of licit and illicit opioid-based analgesics. Drug overdose is the leading cause of accidental death in Canada and the US, and the growing toll of opioid-related morbidity and mortality requires a diversity of novel therapeutic and harm reduction-based interventions. Research suggests that increasing adult access to both medical and recreational cannabis has significant positive impacts on public health and safety as a result of *substitution effect*. Observational and epidemiological studies have found that medical cannabis programs are associated with a reduction in the use of opioids and associated morbidity and mortality.

Aims and Methods

This paper presents an evidence-based rationale for cannabis-based interventions in the opioid overdose crisis informed by research on *substitution effect,* proposing three important windows of opportunity for cannabis for therapeutic purposes (CTP) to play a role in reducing opioid use and interrupting the cycle towards opioid use disorder: 1) prior to opioid introduction in the treatment of chronic pain; 2) as an opioid reduction strategy for those patients already using opioids; and 3) as an adjunct therapy to methadone or suboxone treatment in order to increase treatment success rates. The commentary explores potential obstacles and limitations to these proposed interventions, and as well as strategies to monitor their impact on public health and safety.

Conclusion

The growing body of research supporting the medical use of cannabis as an adjunct or substitute for opioids creates an evidence-based rationale for governments, health care providers, and academic researchers to consider the implementation and assessment of cannabis-based interventions in the opioid crisis.

(Lucas 2017)

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Cannabis is a gateway drug?

Health Aff (Millwood). 2016 Jul 1;35(7):1230-6. doi: 10.1377/hlthaff.2015.1661.

Medical Marijuana Laws Reduce Prescription Medication Use In Medicare Part D.

Bradford AC[1], Bradford WD[2].

⊕ Author information

Abstract

Legalization of medical marijuana has been one of the most controversial areas of state policy change over the past twenty years. However, little is known about whether medical marijuana is being used clinically to any significant degree. Using data on all prescriptions filled by Medicare Part D enrollees from 2010 to 2013, we found that the use of prescription drugs for which marijuana could serve as a clinical alternative fell significantly, once a medical marijuana law was implemented. National overall reductions in Medicare program and enrollee spending when states implemented medical marijuana laws were estimated to be $165.2 million per year in 2013. The availability of medical marijuana has a significant effect on prescribing patterns and spending in Medicare Part D.

Project HOPE—The People-to-People Health Foundation, Inc.

KEYWORDS: Medicare Part D; medical marijuana; state policy

PMID: 27385238 DOI: 10.1377/hlthaff.2015.1661

[PubMed - in process]

(Bradford & Bradford 2016)

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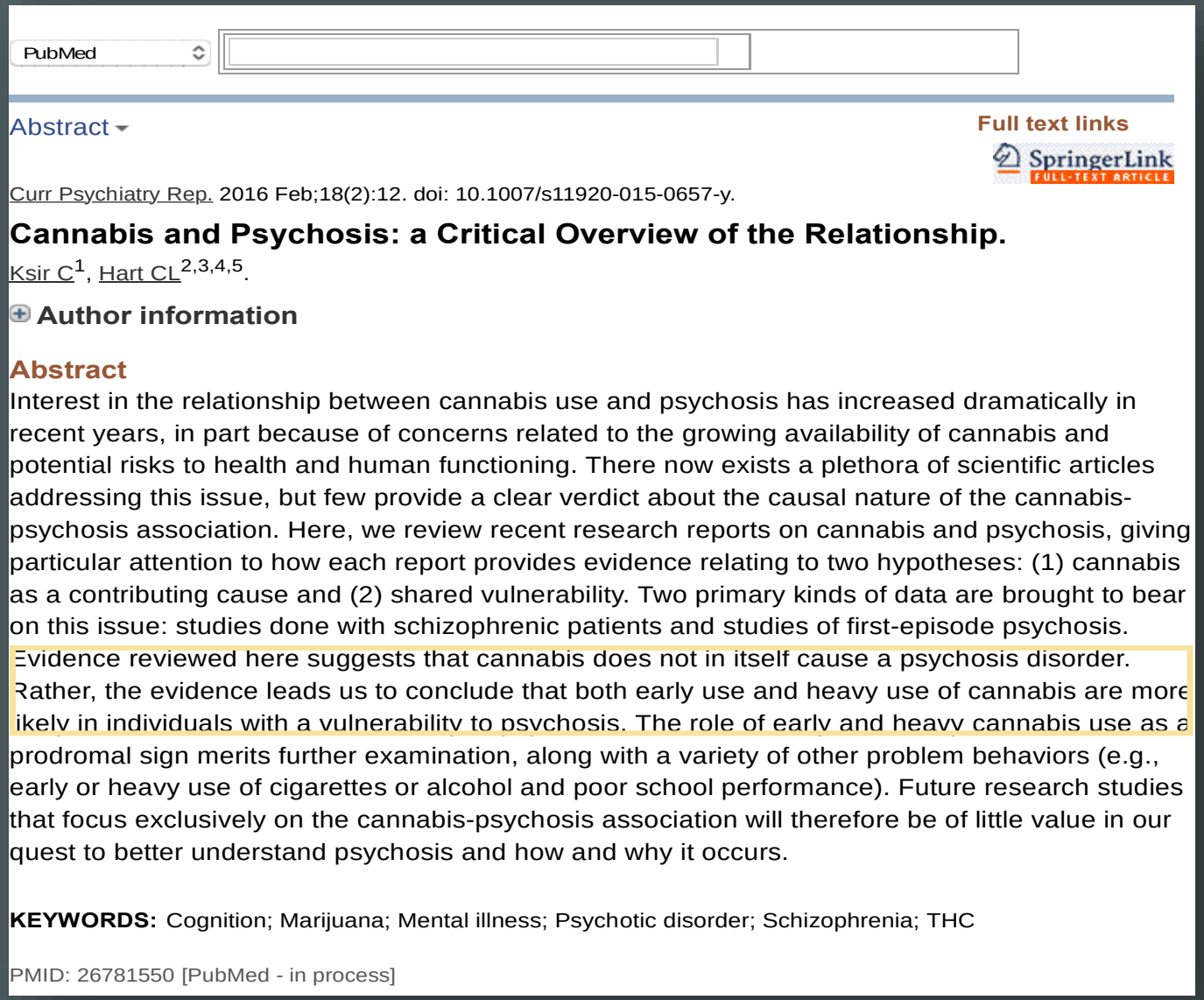
Cannabis can cause psychosis?



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Curr Psychiatry Rep. 2016 Feb;18(2):12. doi: 10.1007/s11920-015-0657-y.

Cannabis and Psychosis: a Critical Overview of the Relationship.

Ksir C[1], Hart CL[2,3,4,5].

Author information

Abstract

Interest in the relationship between cannabis use and psychosis has increased dramatically in recent years, in part because of concerns related to the growing availability of cannabis and potential risks to health and human functioning. There now exists a plethora of scientific articles addressing this issue, but few provide a clear verdict about the causal nature of the cannabis-psychosis association. Here, we review recent research reports on cannabis and psychosis, giving particular attention to how each report provides evidence relating to two hypotheses: (1) cannabis as a contributing cause and (2) shared vulnerability. Two primary kinds of data are brought to bear on this issue: studies done with schizophrenic patients and studies of first-episode psychosis. Evidence reviewed here suggests that cannabis does not in itself cause a psychosis disorder. Rather, the evidence leads us to conclude that both early use and heavy use of cannabis are more likely in individuals with a vulnerability to psychosis. The role of early and heavy cannabis use as a prodromal sign merits further examination, along with a variety of other problem behaviors (e.g., early or heavy use of cigarettes or alcohol and poor school performance). Future research studies that focus exclusively on the cannabis-psychosis association will therefore be of little value in our quest to better understand psychosis and how and why it occurs.

KEYWORDS: Cognition; Marijuana; Mental illness; Psychotic disorder; Schizophrenia; THC

PMID: 26781550 [PubMed - in process]

(Ksir & Hart 2016)

- Psychosis is an inability to distinguish what is real and can include delusions and hallucinations.
- Psychosis can be a brief episode or longer term as seen in psychiatric conditions such as schizophrenia.
- The exact cause of psychosis is unknown but likely involves a complex interplay of physical, genetic, psychological and environmental factors.

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Cannabis can cause psychosis?

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Addiction. 2009 Nov;104(11):1856-61. doi: 10.1111/j.1360-0443.2009.02736.x.

If cannabis caused schizophrenia--how many cannabis users may need to be prevented in order to prevent one case of schizophrenia? England and Wales calculations.

Hickman M[1], Vickerman P, Macleod J, Lewis G, Zammit S, Kirkbride J, Jones P.

Author information

Abstract

BACKGROUND: We consider how many cannabis users may need to be prevented in order to prevent one case of schizophrenia or psychosis [defined as number needed to prevent (NNP)].

METHOD: Calculation for England and Wales using best available estimates of: incidence of schizophrenia; rates of heavy and light cannabis use; and risk that cannabis causes schizophrenia.

RESULTS: In men the annual mean NNP for heavy cannabis and schizophrenia ranged from 2800 [90% confidence interval (CI) 2018-4530] in those aged 20-24 years to 4700 (90% CI 3114-8416) in those aged 35-39. In women, mean NNP for heavy cannabis use and schizophrenia ranged from 5470 (90% CI 3640-9839) in those aged 25-29 to 10 870 (90% CI 6786-22 732) in 35-39-year-olds. Equivalent mean NNP for heavy cannabis use and psychosis were lower, from 1360 (90% CI 1007-2124) in men aged 20-24 and 2480 (90% CI 1408-3518) in women aged 16-19. The mean and median number of light cannabis users that would need to be prevented in order to prevent one case of schizophrenia or psychosis per year are four to five times greater than among heavy users.

CONCLUSIONS: The number of young people who need to be exposed to an intervention to generate NNP and prevent one case of schizophrenia will be even larger. The public health importance of preventing cannabis to reduce schizophrenia or psychosis remains uncertain. More attention should be given to testing the hypothesis that cannabis is related causally to psychotic outcomes, and to considering what strategies will be the most effective in reducing heavy cannabis use among young people.

In this UK study, it was estimated that to prevent one case of psychosis approximately 2000 young men would need to stop using Cannabis.

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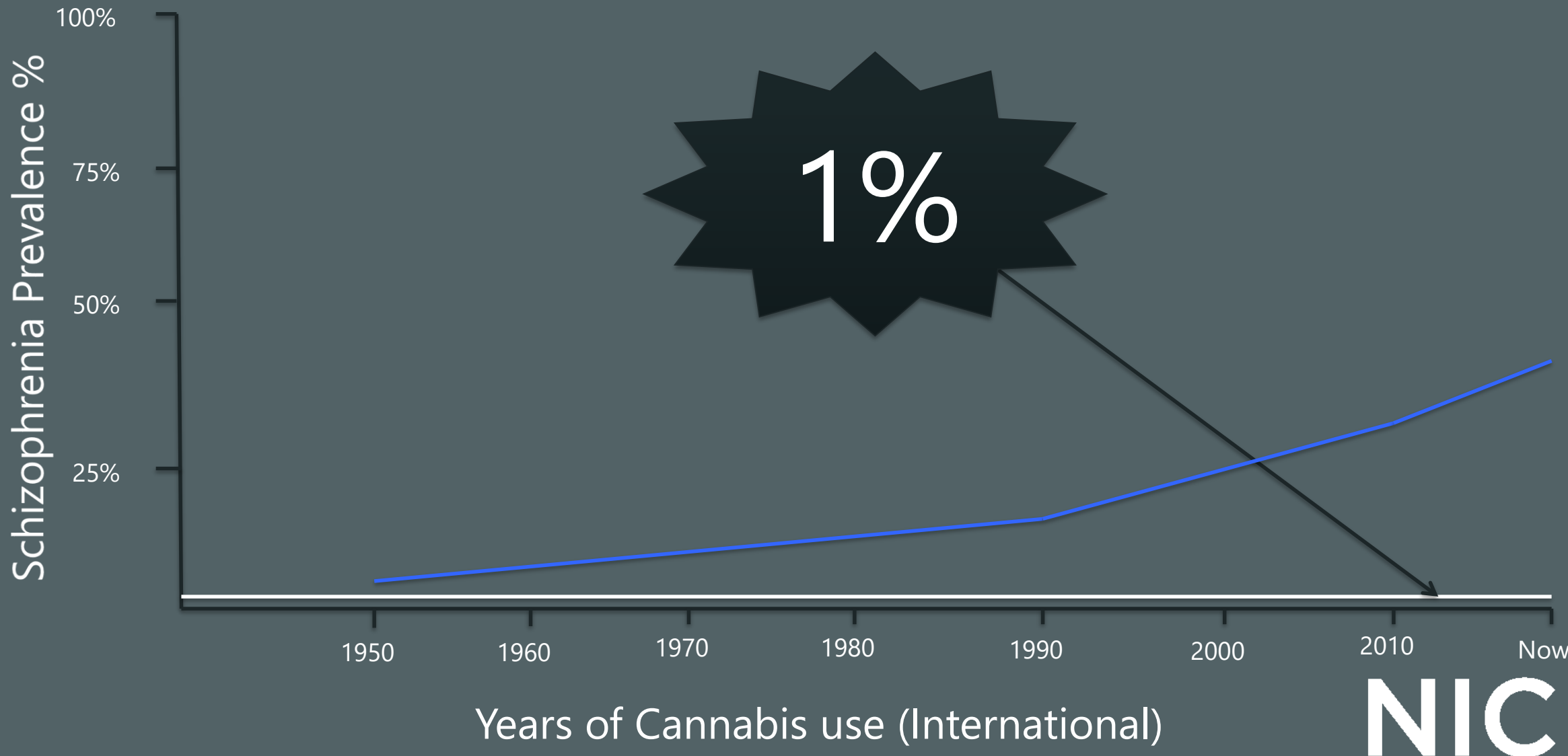
Cannabis can cause psychosis?



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Cannabis can cause psychosis?

Medicines (Basel). 2018 Sep; 5(3): 86.
Published online 2018 Aug 8. doi: 10.3390/medicines5030086
PMCID: PMC6164121
PMID: 30096776

The Role of Cannabis within an Emerging Perspective on Schizophrenia

Jegason P. Diviant,[1] Jacob M. Vigil,[1,*] and Sarah S. Stith[2]

Abstract

Go to:

Background: Approximately 0.5% of the population is diagnosed with some form of schizophrenia, under the prevailing view that the pathology is best treated using pharmaceutical medications that act on monoamine receptors. **Methods:** We briefly review evidence on the impact of environmental forces, particularly the effect of autoimmune activity, in the expression of schizophrenic profiles and the role of *Cannabis* therapy for regulating immunological functioning. **Results:** A review of the literature shows that phytocannabinoid consumption may be a safe and effective treatment option for schizophrenia as a primary or adjunctive therapy. **Conclusions:** Emerging research suggests that *Cannabis* can be used as a treatment for schizophrenia within a broader etiological perspective that focuses on environmental, autoimmune, and neuroinflammatory causes of the disorder, offering a fresh start and newfound hope for those suffering from this debilitating and poorly understood disease.

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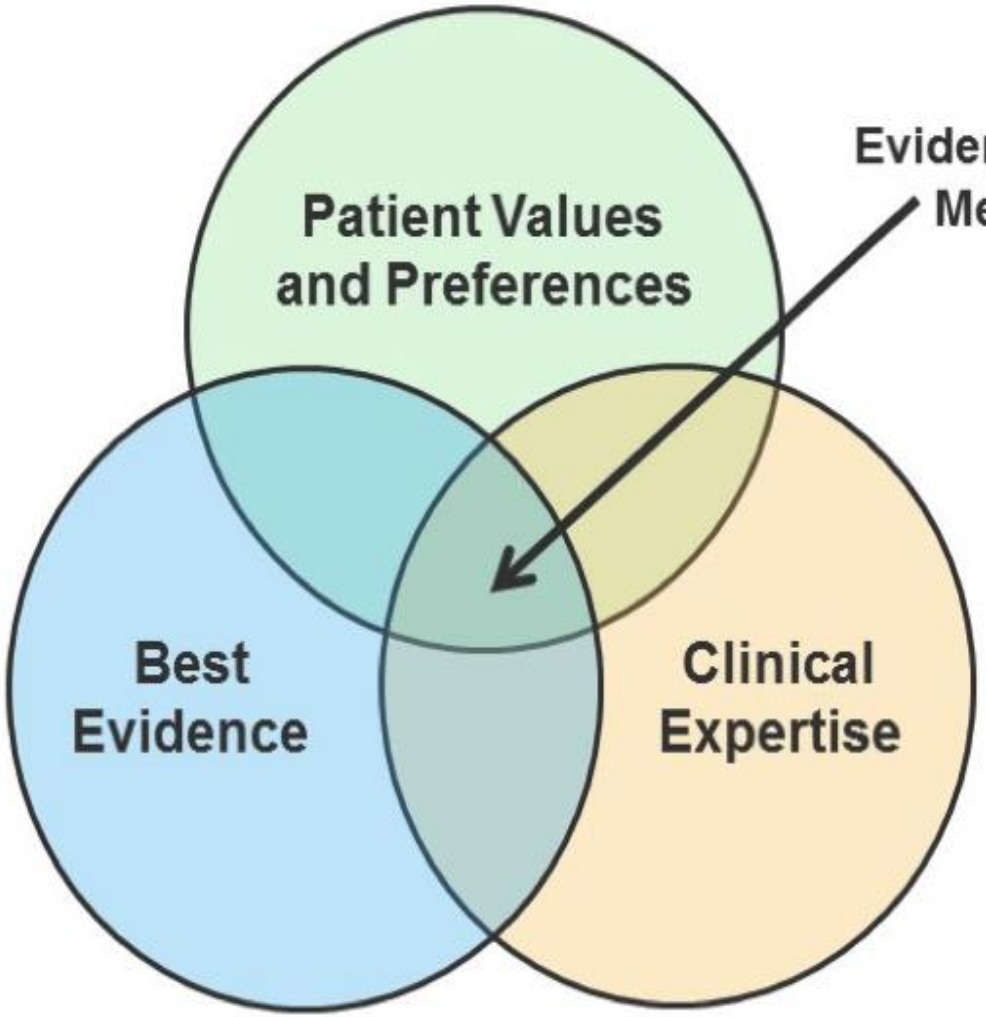
Components of Evidence-Based Medicine



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