Psychiatric Aspects of Marijuana

Professor David J. Castle

Chair of Psychiatry, St. Vincent's Mental Health
St. Vincent's Hospital & The University of Melbourne
Level 2, 46 Nicholson Street, Fitzroy 3065
Tel: (03) 9231 4145 Fax: (03) 9231 4802 Email: david.castle@svha.org.au

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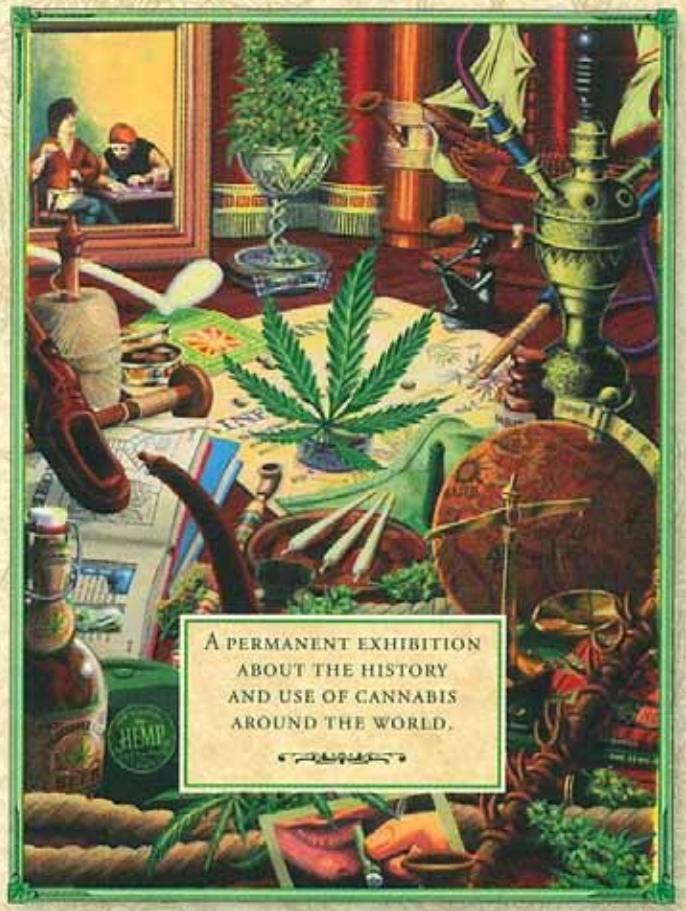
The Hash Marihuana & Hemp Museum

AMSTERDAM

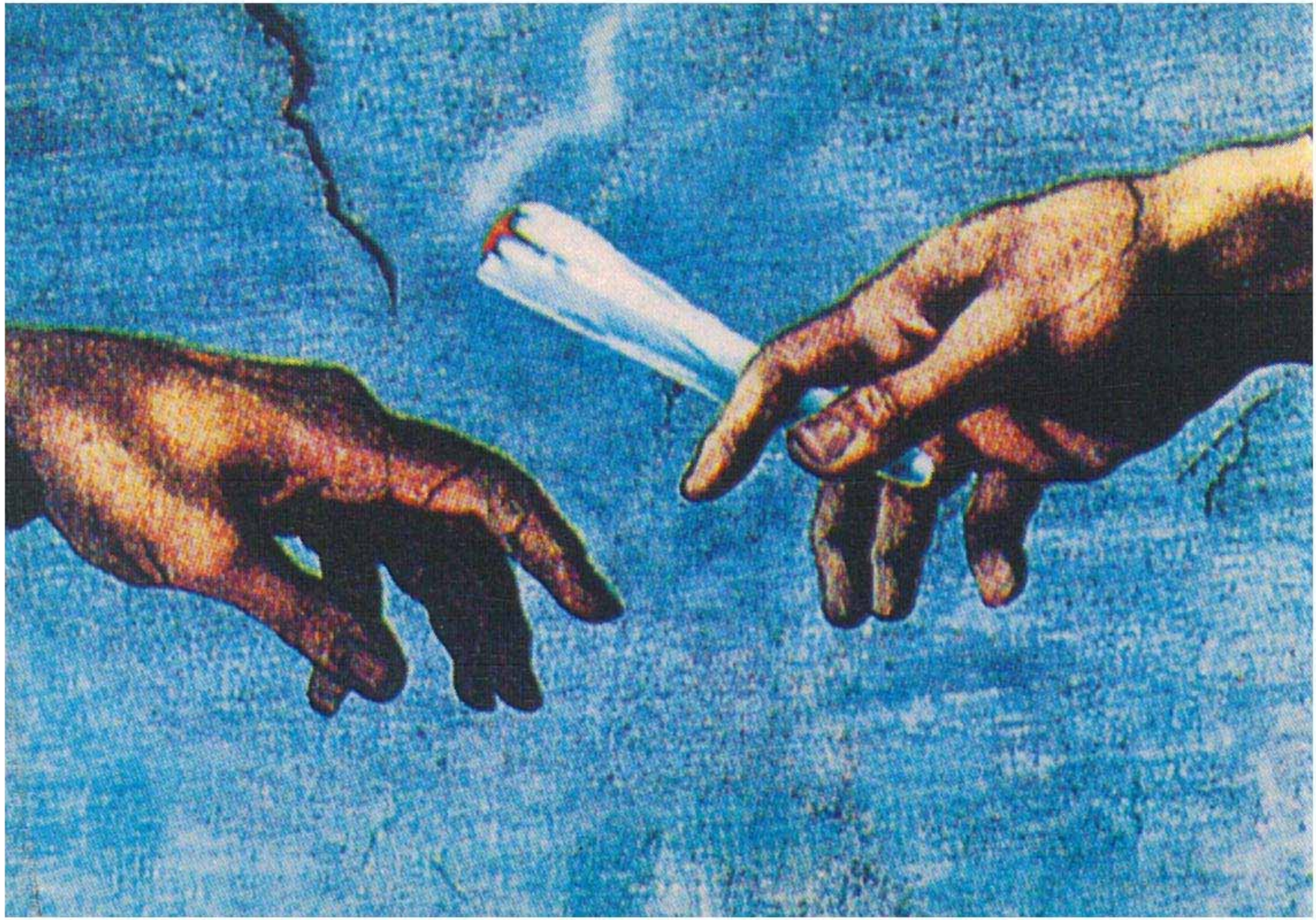


Oudezijds Achterburgwal 130 Amsterdam.

Next to the 'Sensi Seed Bank' Grow Shop.

Open all week from 11.00 until 22.00 hours.

www.hashmuseum.com



Rates of Cannabis Use in Europe

(Choquet et al, 2007)

- study of 16 year olds
- 21% lifetime use, 9% prior month
- highest (22%) France, lowest Sweden (1%)
- not related to knowledge of harmful effects, but to parents knowing where kids are on a Saturday night

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Effects of THC on the Developing Brain (1)

- crosses placenta
- excreted in breast milk
- adolescent exposure increasing

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Effects of THC on the Developing Brain (2)

Infant rats exposed to THC (Viveros, 2007)

- **↑ cortisol response on weaning**
- **In ♀ high dose anxiogenic response – reversed by nicotine**
- **In ♂ → ↑ morphine self administration**
- **Interactions between nicotinic, opiate and cannabinoid systems**

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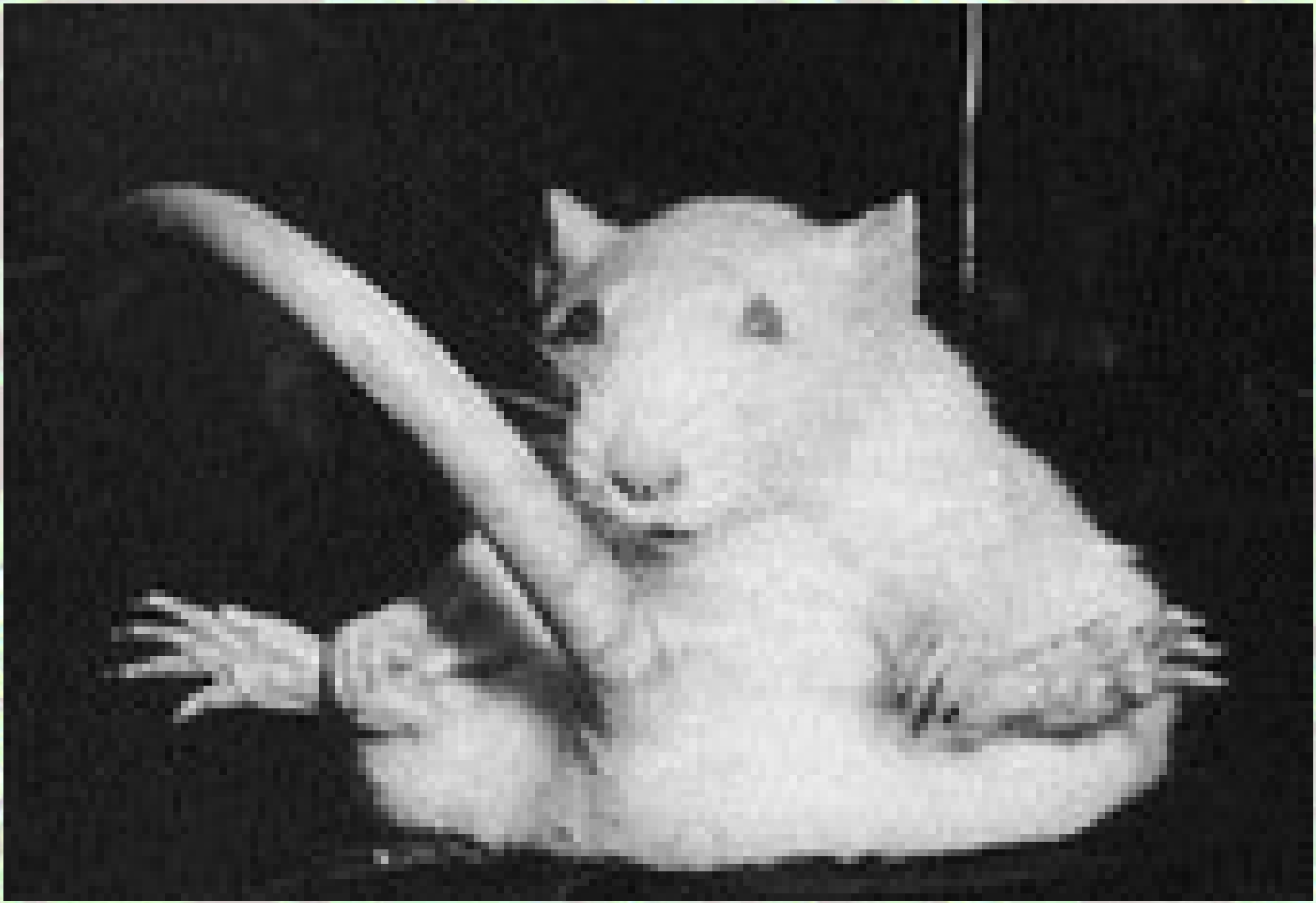

Effects of THC on the Developing Brain (3)

Verrico et al, (2014)

- **6 month exposure of adolescent monkeys, to THC (equivalent 1-2 cigarette 5 x weekly)**
- **→ impaired spatial working memory**

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Effects of THC on the Developing Brain (4)

In adolescent humans (Block, 2007; Jager, 2007)

- **US/Nederlands sample of teenage males (n=20)**
- **No differences in brain structures**
- **In US sample**
  - **↓ verbal recall (Bushke's Test)**
  - **↓ abstraction (WCST)**
  - **↓ selective attention**
- **Function imaging**
  - **Hypoactivity**
  - **↓ prefrontal activity on cognitive task**
  - **No difference selective abstraction, but users showed ↑ activity in ® fusiform gyrus (? ↑ effort?)**

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The Human Cannabinoid System (1)

- CB1 receptor diffusely in brain
- CB2 receptor spleen, testis, etc.

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The Human Cannabinoid System (2)

CB1 Receptors

- expressed only in brain
- anandamine natural substrate
- diffusely in cortex (esp. frontal), basal ganglia, cerebellum, hypothalmus, anterior cingulate, hippocampus.
- axons & nerve terminals (ie. presynaptic)
- modulate release of neurotransmitters from axon terminals
  - Inhibits glutamate, GABA, NA, DA, 5HT, ACh
  - Effects blocked by rimonabant

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Effects on CNS Function & Psychomotor Control

(Iversen, 2012)

- CB1 receptors in basal ganglia, cerebellum
- In rats, low dose THC → ↓ activity

  high dose THC → ↑ activity & catalepsy and "popcorn effect"
- In humans, impair balance, fine motor control

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Effects on Memory

(Iversen, 2012)

- acute intoxication → impaired STM
- reversed by rimonabant
- mediated by hippocampus? Via GABA, glutamate
- ? role in extinction of aversive memories

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Effects on Neo Cortex

(Iversen, 2012)

- changes on EEG (sleep-like) reversed by rimonabant
- effects on sleep/wake cycle
- psychomimetic effects
- effects on experience of passage of time

Effects on Appetite

(Iversen, 2012)

- ↑ appetite ("munchies") especially for sweet foods
- Mediated by hypothalamus
- THC (dronabinol) → ↑ appetite and weight gain in AIDS patients
- Rimonabant → ↓ appetitite ↓ weight

Effects on Pain

(Iversen, 2012)

- Interaction between cannabinoid & opioid systems
- THC & morphine act synergistically in test of acute & chronic pain
- ? Act via activation of a brainstem circuit also required for opiate analgesia
- ? Role in reward aspects of THC

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Tolerance & Dependence

(Iversen, 2012)

- tolerance to THC well demonstrated in animals
- withdrawal syndrome established, including craving, ↓ appetite, ↓ sleep, irritability, restlessness
- probable links with opiate dependence
  - cannabis-dependent rats sensitised to heroin
  - opiate withdrawal partly relieved by THC

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Effects on Anxiety

(Iversen, 2012)

- Immediate effect of tachycardia

  → can precipitate panic

- Low doses paradoxically anxiolytic

  (? Related to opioid system involvement)

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Effects on Mood – (1) Mania

(Castle & Ames, 1996)

- euphoria with contagious laughter part of intoxication
- manic symptoms part of "cannabis psychosis"
- some people with bipolar claim it helps moderate manic symptoms (? helps sleep)
- no consistent antidepressant effect in depression

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Effects on Mood – Depression (1)

- **Clinical association between cannabis & depression**

- **4 cohort studies of depression & cannabis use**
  - Zurich (Angst, 1996): 2.3 x risk
  - NZ (Ferguson & Horwood, 1997): 36% in “heavy” users vs. 11% in non users
  - Melbourne (Patton et al, 2002): 8.6 x risk in daily users vs. non users (2.0 x after confounders – in females only)
  - Baltimore (Bovasso, 2001): 4.5x risk over 14-16 year follow-up

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Effects on Mood – Depression (2)

- **Interpretation (Degenhardt et al, 2012):**
  - (i) Cannabis → depressed mood
  - (ii) Depressed mood → cannabis use
  - (iii) common factors → both depression & cannabis use

- **Low PAF (~1.9% estimate in Bovasso study)**

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PER SERVING	100ml	355ml
ENERGY	199kj	706kj
CARBOHYDRATE (SUGAR)	11g	39g
FAT	NIL	NIL
PROTEIN	NIL	NIL
SODIUM	47mg	167mg
CALCIUM	10mg	36mg
POTASSIUM	4mg	14mg
MAGNESIUM	4mg	15mg
TAURINE	400mg	1420mg
CAFFEINE	32mg	113mg
GUARANA EXTRACT	22mg	79mg
VITAMIN B3	2mg	7mg
VITAMIN B6	2mg	7mg
VITAMIN B12	2mcg	7mcg
INOSITOL	2mg	7mg
PANTOTHENIC ACID	2mg	7mg
GLUCURONOLACTONE	10mg	36mg

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355ml

Energy Drink Energy Drink

H.E.M.P The Serious Stimulant - A packed full sparkling energy charge specially developed to help you reach past your boundaries.

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100% maximum daily intake, 1.5 bottles
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Ingredients: Carbonated Water, Sucrose, Invert Sugar, Citric Acid, Sodium Citrate, Taurine, Guarana Extract, Flavours, Preservative (211) Minerals: (Calcium Chloride, Magnesium Sulphate, Potassium Chloride), Caffeine, Glucuronolactone, Vitamins (Pantothenic Acid, B3, B6, B12) Inositol, Colour (150).

5C REFUND AT COLLECTION DEPOTS WHEN SOLD IN S.AUST.

Psychomimetic Properties of Cannabis Sativa (1)

- Psychoactive moiety is delta-9-tetrahydrocannabinol
  - Action via CB1 receptors (Huestis et al, 2001)
- Dopamine release in limbic system
  - Blockade of Dopamine D2 receptors blocks some but not all psychotic effects (D'Souza et al, 2012)

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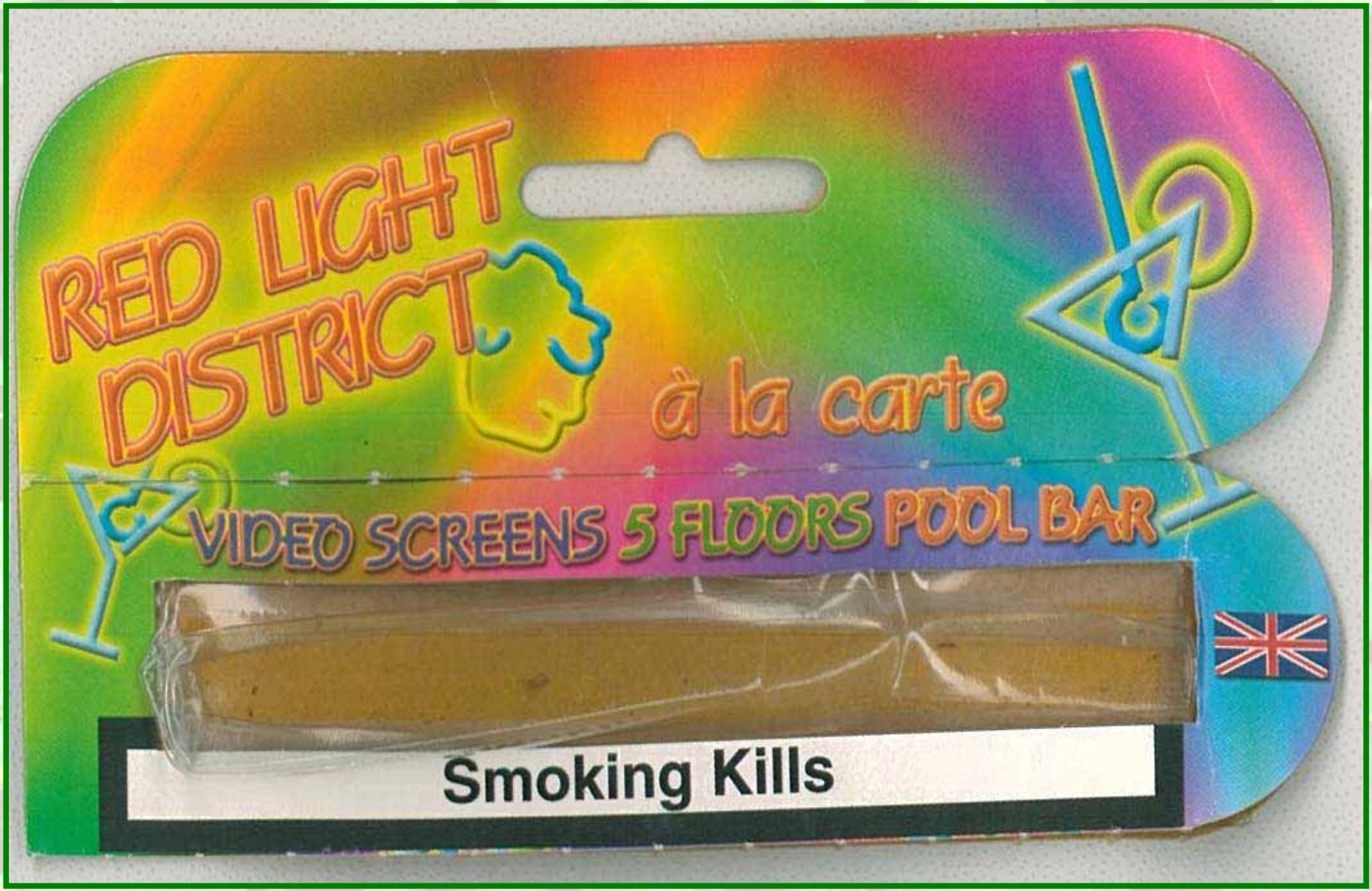
RED LIGHT DISTRICT
à la carte
VIDEO SCREENS 5 FLOORS POOL BAR
Smoking Kills

Psychomimetic Properties of Cannabis Sativa (2)

(Ames et al, 1958): 12 medical volunteers

- alteration in sense of self
- altered sense of passage of time
- euphoria
- transient paranoid ideation
- visual hallucinations (eyes closed)

(Isbell et al, 1967): delta-9-THC in human subjects

- Dose-response relationship
- Some idiosyncratic response at low dose

Psychomimetic Properties of Cannabis Sativa (3)

(Verdoux et al, 2003)

- **Experience sampling**
- **Acute effects of cannabis more extreme in people with "psychosis proneness"**

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Cannabis Psychosis

= discrete temporally related to cannabis exposure

(Rottanburg et al, 1982)

20 psychotic admissions with urinary cannabinoids

20 patients matched on age & clinical Dx

PSE within 24 hours

Repeat PSE in 7 days

cannabis patients:

- **more hypomania, agitation**
- **less auditory hallucinations, affective flattening, incoherence of speech**
- **better clinical response**

Note: a third had clouding of consciousness

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Cannabis-induced Psychosis & Subsequent Schizophrenia

(Arendt et al, 2005)

- **535 patients with cannabis-induced psychosis**

  ⬇

  followed for 3 years

  ⬇

  44.5% schizophrenia spectrum disorders

Note: - young males most vulnerable
- diagnosis > 1yr in 47.1% of patients

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Cannabis Psychosis

(Thornicroft et al, 1991): Review of epidemiological evidence, concluded not a distnct entity

Note: tests of the hypothesis (Robins & Guze, 1970):

(1) phenomenology
(2) response to medication
(3) longitudinal course
(4) familial aggregation

(McQuire et al, 1995): Study of 23 psychotic patients with cannabis vs 46 controls without:

- FH in cannabis group 7.1% vs 0.7% in controls

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Cannabis and Schizophrenia Onset

(Stefanis et al et al, 2013)

- **997 participants for Australian SHIP study**
- **Linear association between age of cannabis initiation and age of psychosis ($p < 0.001$)**
- **7-8 year 'cumulative toxic effect' – shorter with higher cannabis exposure**

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Cannabis and the Course of Schizophrenia (1)

(Negrete et al, 1986): 137 (25 cannabis users) patients with schizophrenia over 6 months.

Cannabis patients:

- higher relapse rate
- more delusions/hallucinations

(Linszen et al, 1994): 24 cannabis users vs 69 non users

cannabis users:

- more an earlier relapse

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Cannabis and the Course of Schizophrenia (2)

Causal Pathways:

- **More severely affected patients might be independently more prone to cannabis use**
- **Other substances often also involved**
- **Cannabis worsens course of illness**

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KEEP OFF THE GRASS

Motivation for Use

Why such high rates of use?

Spencer et al (2012)

- **Determine reasons for substance use amongst people with psychosis (n=68)**
- **Determine the influence these motives have on quantity, context, problems and dependence**
- **Determine the role motives play in mediating the relationship between psychotic symptoms and substance use**

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Why Do People With Psychotic Illness Abuse Drugs? (1)

Factor Analysis

Coping with Unpleasant Affect – 37% of variance

- Because it helps when you feel nervous
- It helps when you feel depressed
- To forget your worries
- To feel more motivated
- To make it easier to sleep
- To help me concentrate
- Because you feel more self confident and sure of yourself
- To relieve boredom
- To decrease restlessness
- To slow down racing thoughts

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Why Do People With Psychotic Illness Abuse Drugs? (2)

Factor Analysis

Enhancement – 10% of Variance

- Because it makes you feel good
- Its what most of your friends do when you get together
- Because its fun
- To get high
- Because it makes a social gathering more enjoyable
- As a way to celebrate
- To relax

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Why Do People With Psychotic Illness Abuse Drugs? (3)

Factor Analysis

Conformity & Acceptance – 8% of variance

- So you won't feel left out
- To be liked
- To help you talk to others
- To be sociable
- To be part of a group

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Why Do People With Psychotic Illness Abuse Drugs? (4)

Factor Analysis

Relief of positive symptoms and side effects – 6%

- To get away from the voices
- To reduce side effects of medication
- Because your friends pressure you to do it
- To decrease suspiciousness and paranoia

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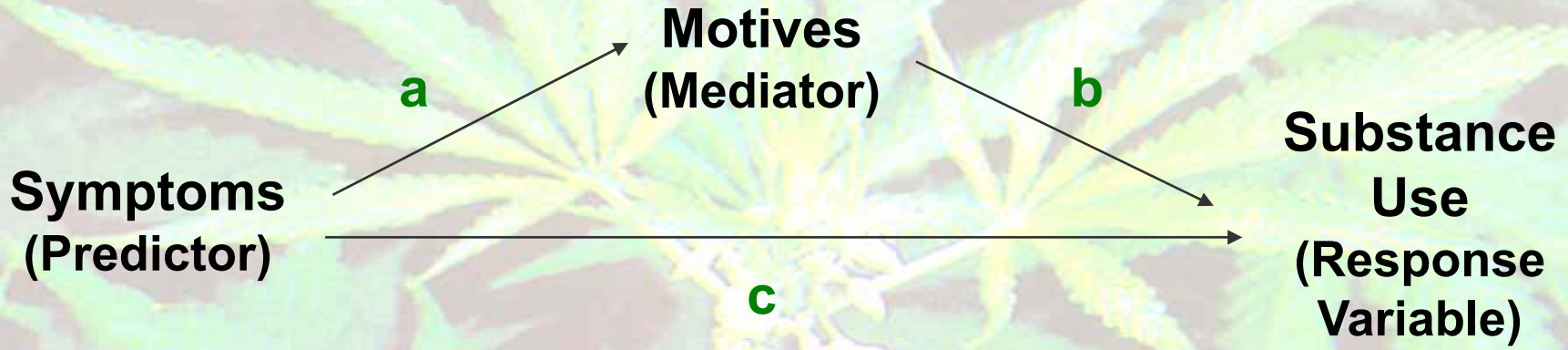
Model of Symptoms Motives For Use & Substance Use



- Path *a* = symptoms (predictor) significantly predict motives for use (Mediator)
- Path *b* = motives (mediator) significantly predict substance use (response variable
- Path *c* = symptoms (predictor) must significantly predict substance use (response variable)

Path *a* and *b* outweigh path *c*, suggesting motives are the mediator between symptoms and substance use.

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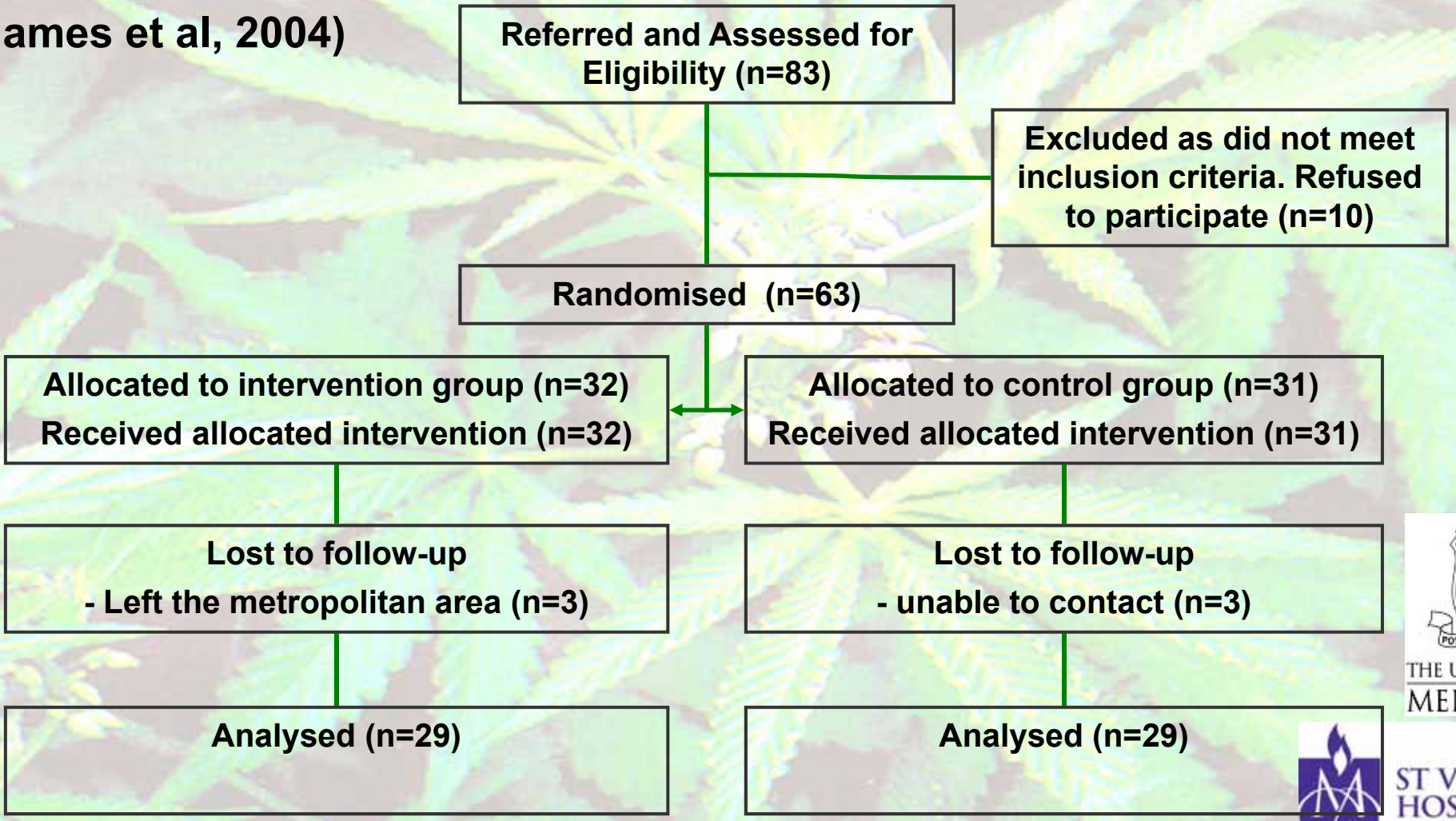
Project Flow Diagram of Subjects Progress Through the Trial

(James et al, 2004)



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Intervention for the Experimental Group

Module 1: Education and Exploration of Motivations for Use: This module develops an awareness of impact on substance use on mental and physical health and lifestyle. It also explores individual reasons for substance abuse, allowing these to be incorporated into the treatment package, by way of discussion and problem-solving in the sessions, as well as being targeted in individualised homework exercises.

Module 2: Motivational Enhancement and Goal Setting: Participants are asked to explore their expectations, review the advantages and disadvantages of substance use and to consider goals for management.

Module 3: Cutting Down, Harm Reduction and Self-Monitoring: Strategies are introduced for harm minimisation, cutting down and stopping drug use. This is complemented by self-monitoring strategies.

Module 4: Coping with High Risk Situations: This module assists participants to identify high-risk situations and help find alternative ways to cope. High-risk situations also include factors likely to induce deterioration in psychiatric symptoms.

Module 5: Review and Future Planning: The final module reviews previous sessions and addresses each participant's goals. Plans for the future are decided and local community drug and alcohol services are introduced.

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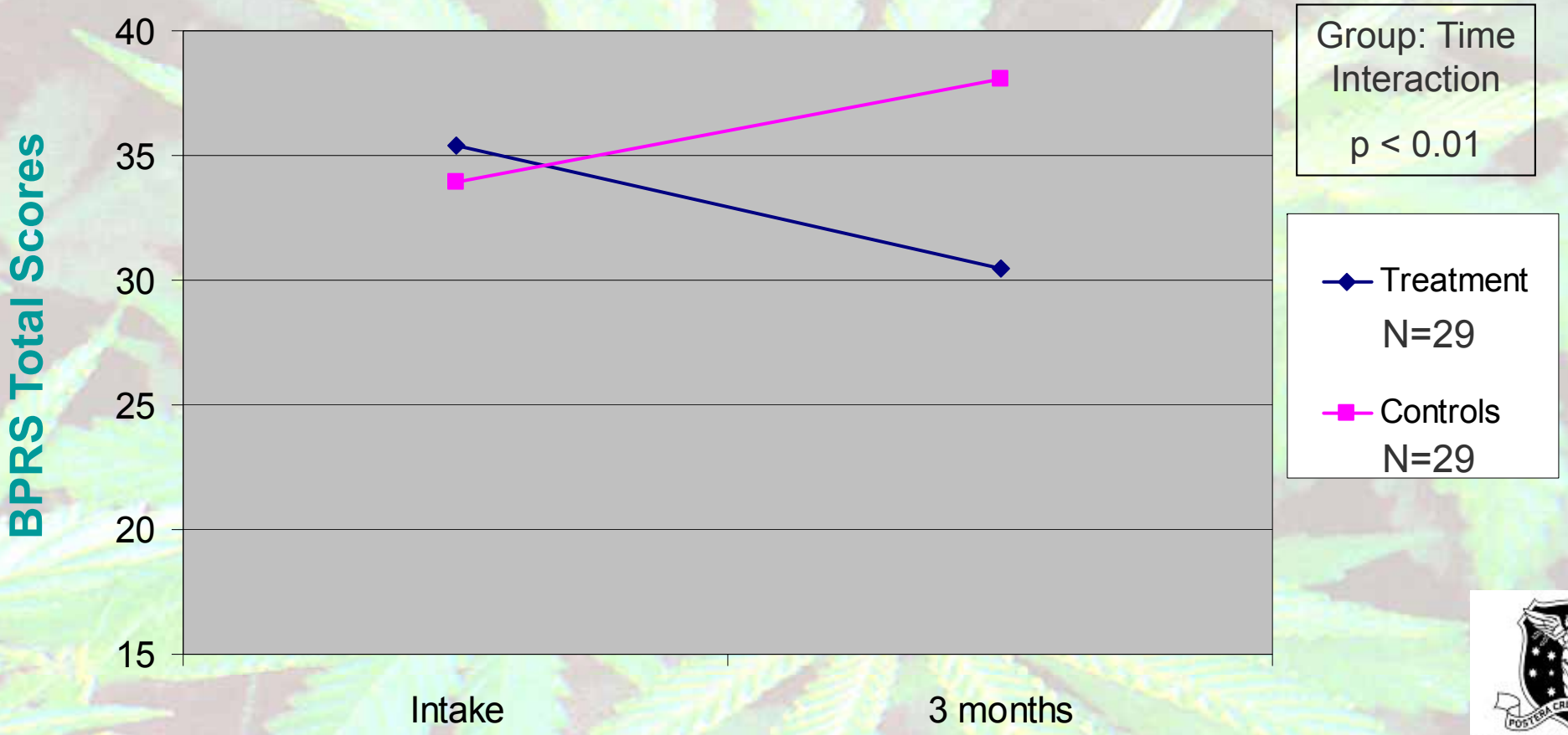
BPRS Total Scores



	Intake	3 months
Treatment	35.41	30.48
Controls	33.93	38.10

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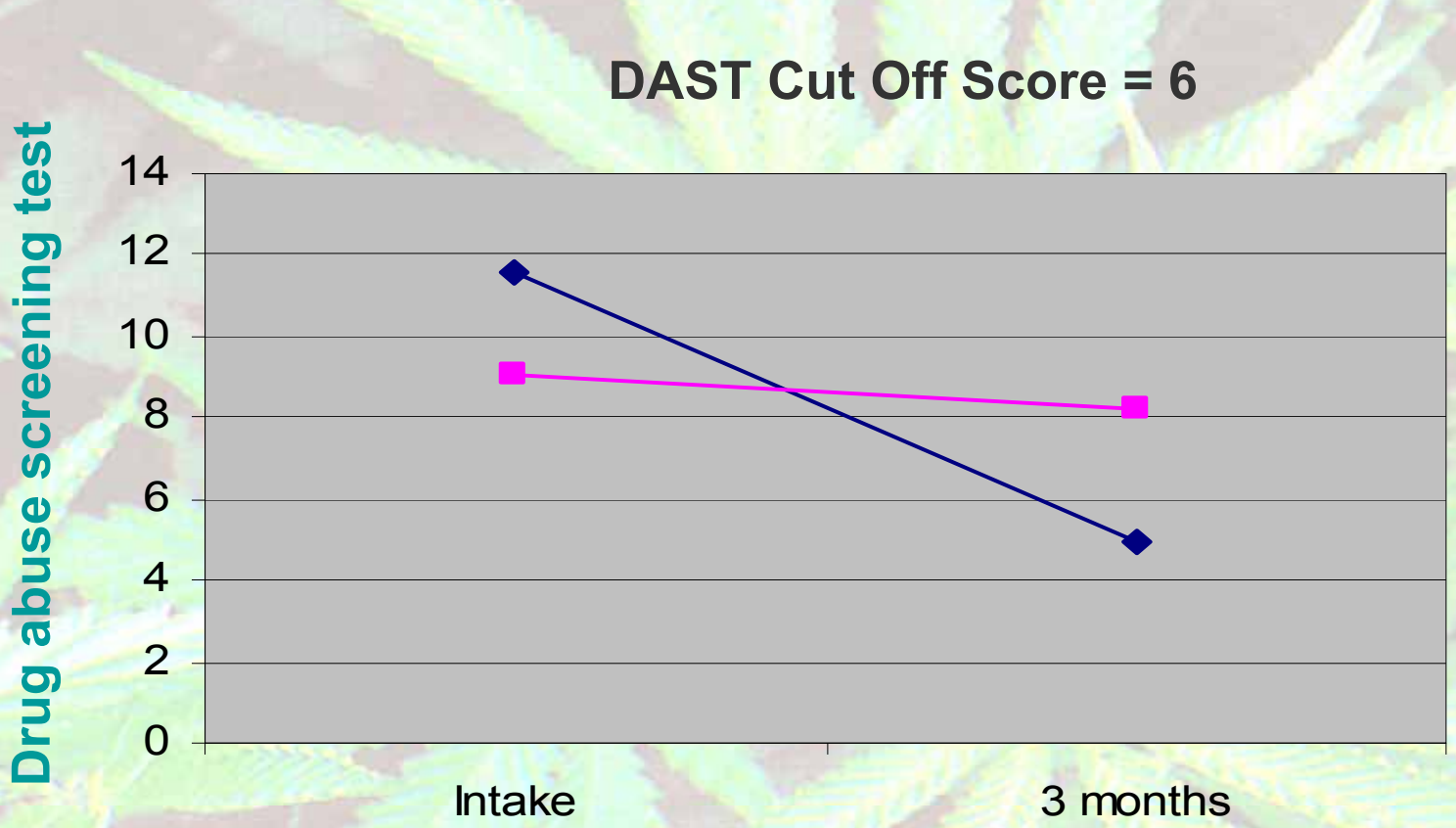
Drug Abuse Screening Test (DAST)



Interaction Effect

Time p<0.01

Group Time

p < 0.01

Treatment
n = 29

Controls
n = 29

Treatment	**11.58**	**4.96**
Controls	**9.10**	**8.24**

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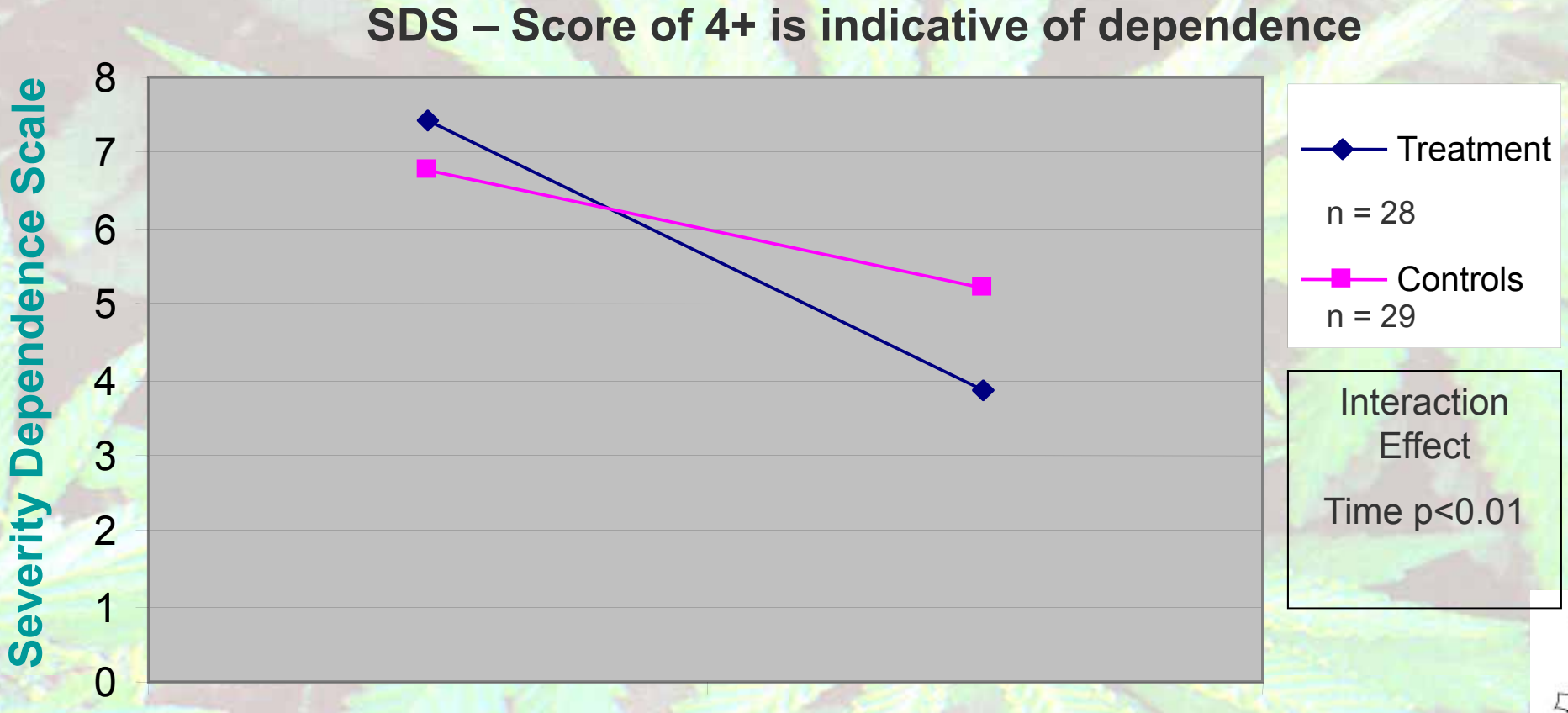
Severity of Dependence Scale



	Intake	3 months
Treatment	**7.42**	**3.85**
Controls	**6.75**	**5.20**

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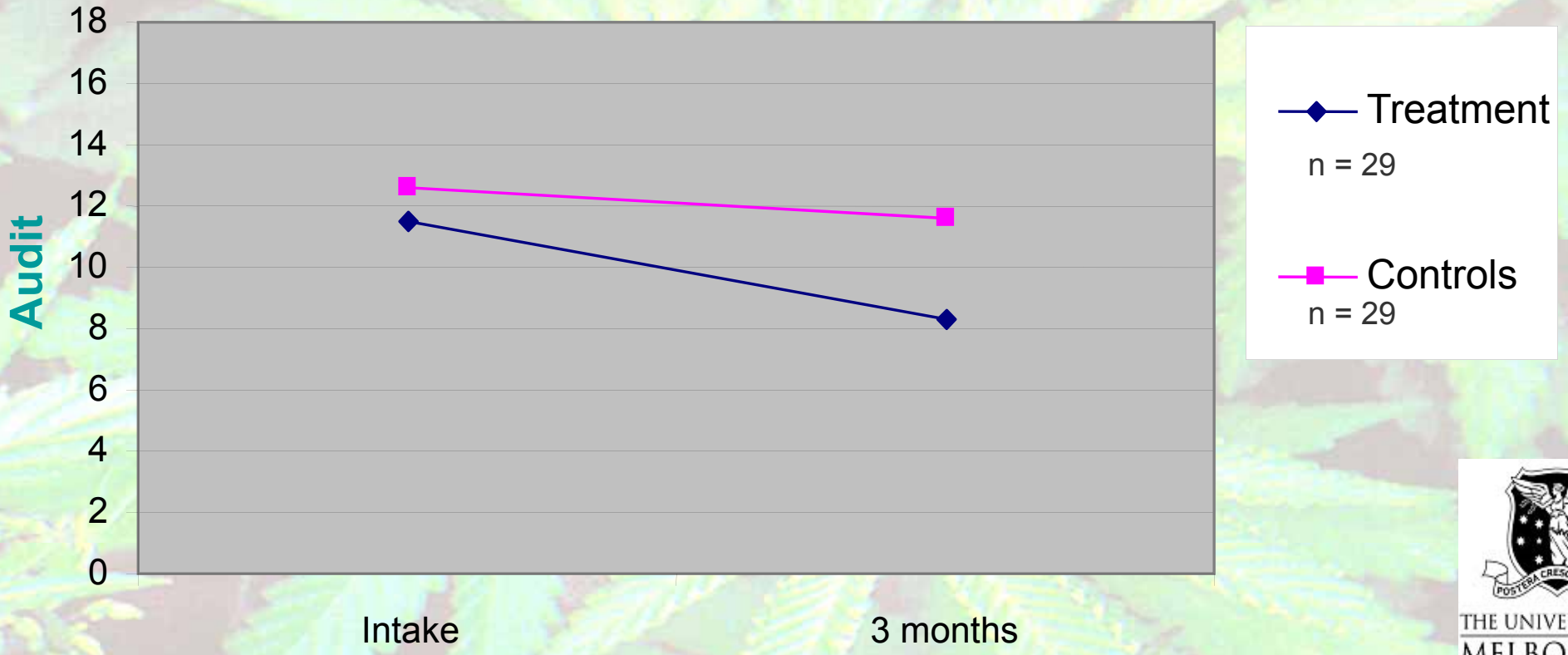
Audit

AUDIT score of 8+ is indicative of harmful/hazardous drinking, 13+ = dependence



	Intake	3 months
Treatment	**11.91**	**7.08**
Controls	**12.7**	**16.2**

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Does Cannabis Cause Schizophrenia?

Bradford Hill's criteria for causality:

- **Strength of association**
- **Consistency of association**
- **Specificity of association**
  - **specificity of cause**
  - **specificity of effect**
- **Temporality of association**
- **Biological gradient**
- **Experimental evidence**
- **Plausible hypothesis**

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Swedish Conscript Study

(Andreason et al, 1987)

45,570 male Swedish conscripts 1969/70

↓

questionnaire re background & drugs

(7% refusal rate on drug question)

↓

psychological assessment

↓

follow-up through 1987

(register of psych care; death register)

↓

cannabis best predictor (RR 2.3) of later sz after

- psych Dx at conscription
- parental divorce

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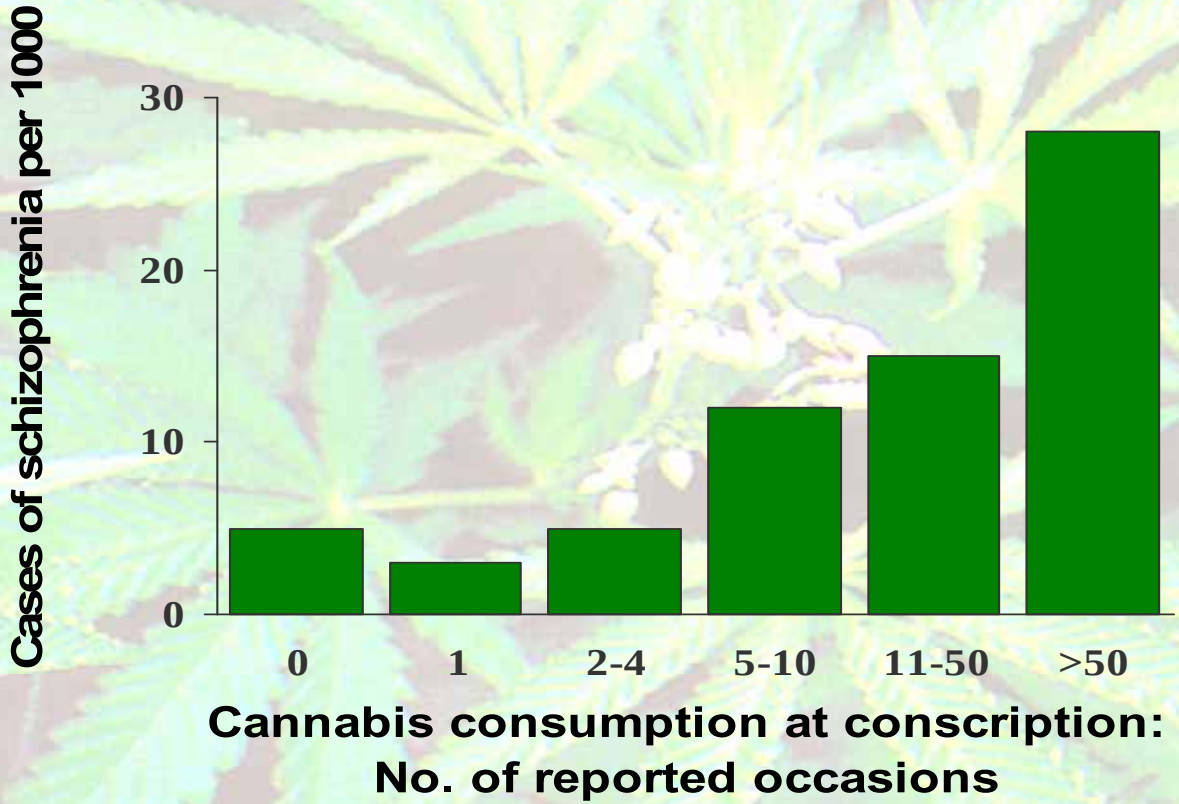
Dose – Response Relationship



Rates of schizophrenia after different levels of cannabis consumption

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Criticisms of the Swedish Conscript Study

Methodology:

- ? validity of self-report drug use
- ? complete ascertainment of cases
- ? accuracy of diagnosis
- ? role of toxic psychosis/cannabis psychosis
- ? role of other drugs
- ? effect of controlling for all potential confounders

Interpretation:

- does not show temporal link
- might merely reflect underlying disposition to both cannabis use and schizophrenia

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NEMESIS Study

(Van Os et al, 2002)

- 5104 m & f Dutch general population (59 had psychosis) assessed at 1 and 3 years
- Cannabis use at baseline associated with psychotic symptoms (RR 2.76; 1.2 - 6.5)
- Dose-response effect
- Confounders controlled for:
  - Age & sex
  - Marital status
  - Urban dwelling
  - Ethnicity & discrimination

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Dunedin Study

(Arsenault et al, 2002)

- Cohort study 1037 males & females followed from birth to 26yrs (96% follow-up)
- Cannabis use at 15yrs associated with later schizophreniform psychosis (10% vs 3%); RR 3.12 (0.7 – 13.3)
- Confounders controlled for:
  - social class
  - prior psychotic symptoms (age 11yrs)
- Interaction between psychotic symptoms at age 11, Cannabis use at 18, and psychotic symptoms at 26

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Genes, Cannabis & Schizophrenia (1)

Gene-environment interaction

- **COMT Val 158 Met Allele**
  - **Dunedin Cohort Study (Caspi et al. 2005)**
  - **Experiential sampling (Henquet et al, 2009)**

  <u>But</u> not replicated in psychotic patients (Zammit et al, 2007)

- **AKT1 (Van Winkel et al, 2011)**

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Genes, Cannabis & Schizophrenia (2)

(Power et al, 2014)

- **2082 participants**
- **Association between schizophrenia risk alleles (polymetric risk scores) and cannabis use (7% of variance)**
- **"part of the association between schizophrenia and cannabis is due to a shared genetic aetiology"**

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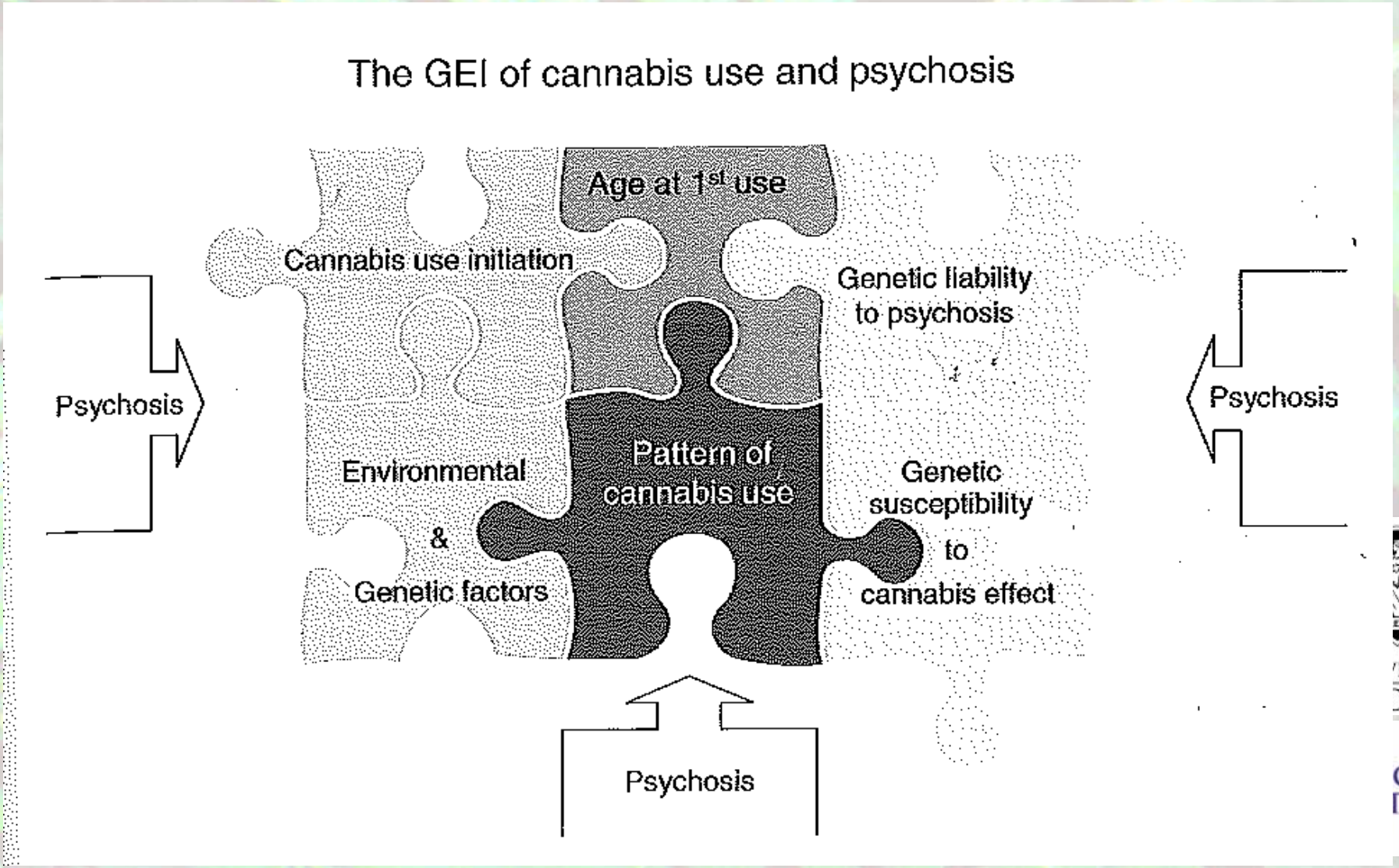
Genes, Cannabis & Schizophrenia (3)

(Di Forti et al, 2012)



An Amotivational Syndrome? (1)

Components of the putative 'amotivational syndrome'

- **loss of interest in things in general, with associated apathy and passivity**
- **loss of desire to work, and loss of concern with work performance, resulting in loss of productivity**
- **loss of energy and easy fatigability**
- **moodiness and irritability**
- **impaired concentration**
- **lack of concern for personal appearance and hygiene**
- **a lifestyle that prioritizes cannabis procurement and consumption**

(Schwartz, 1987)

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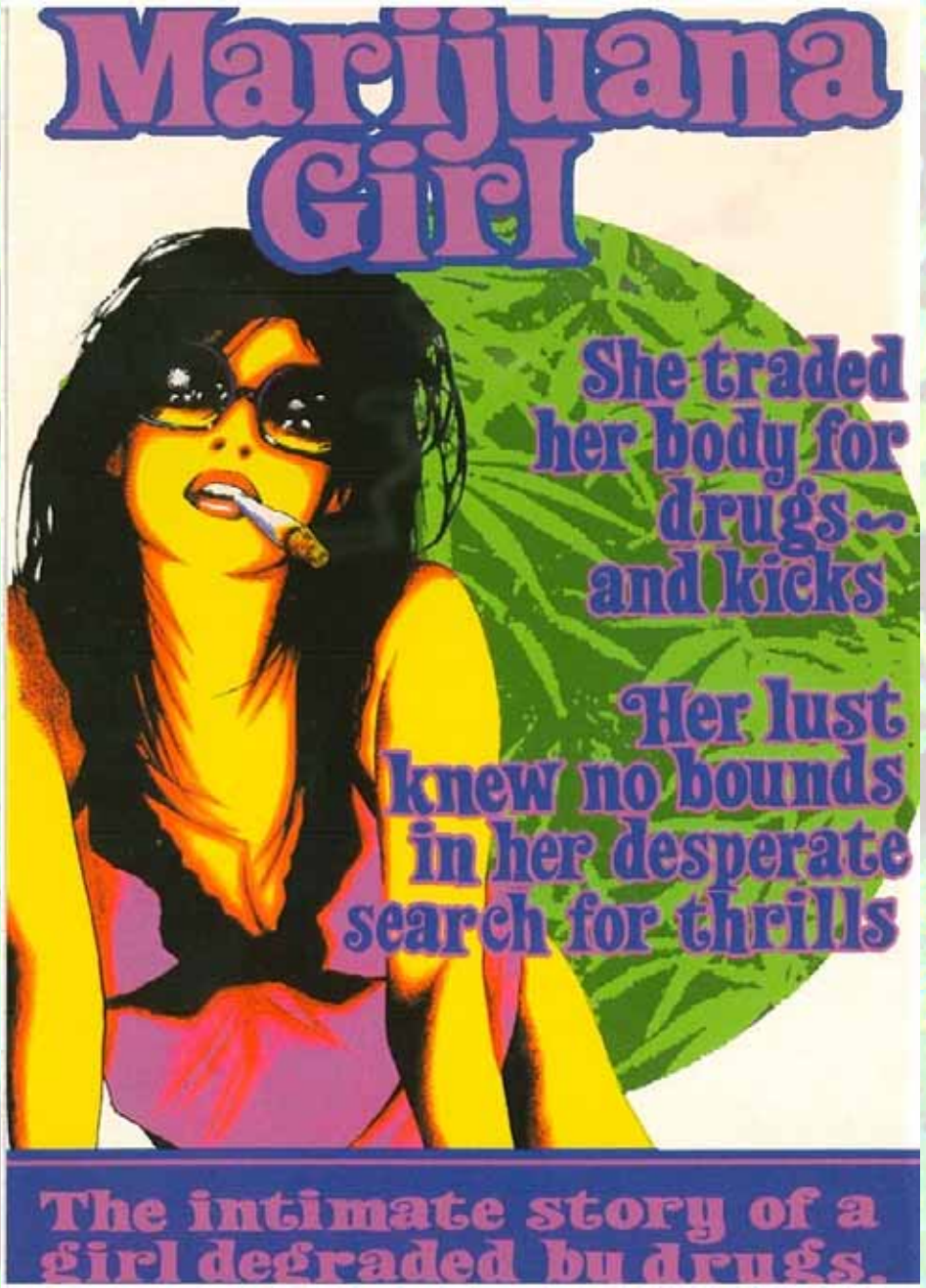

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An Amotivational Syndrome? (2)

(Tennant & Groesbeck, 1972): Study of US soldiers in West Germany (45% habitual cannabis users)

- no discernible effect on 392 subjects of smoking up to 10g per month

- 110 men smoking 50-600g month (500-6000 joints):
  - apathy
  - lethargy
  - impaired concentration
  - impairment of STM

(9 followed up for 2 years – 6 showed no residual Sx)
conclusion: reversible sub-acute encephalopathy

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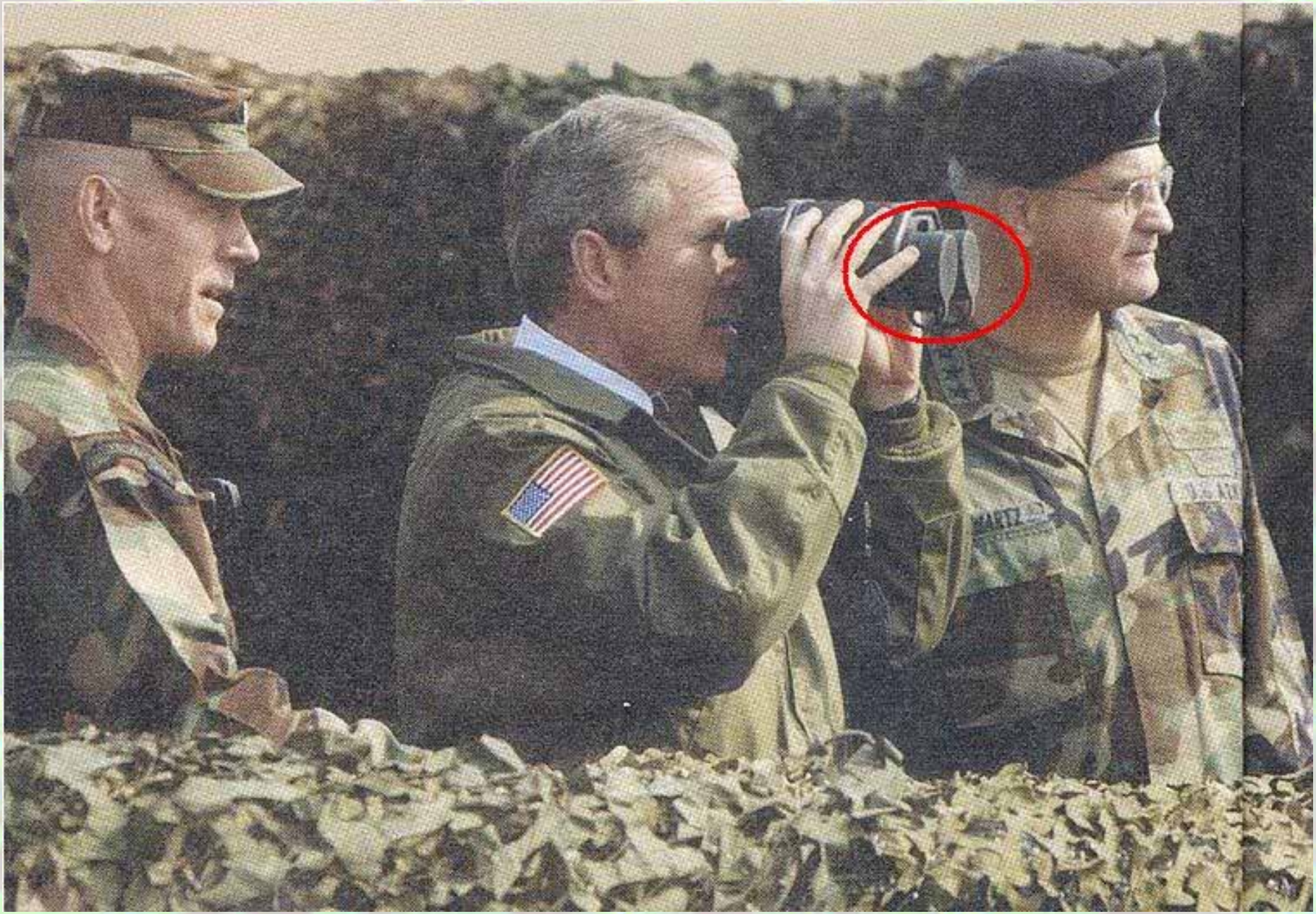

Cognitive Effects

(Pope et al, 2001)

- 77 Cannabis users (5000 – 18,500 times)

  vs 87 controls (<50 times; median 10)
- Tested on abstinent days 0, 1, 7, 28
- By day 28, no significant difference between groups

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Effects on Brain Structure (1)

(Ames et al, 1979)

- early reports of CT scan abnormalities not confirmed in unconfounded samples
- primate studies – no evidence of neuropathological damage over long term but ? long term effects

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Effects on Brain Structure (2)

(Yucel et al, 2008)

- 15 long term heavy (>5/day) users (mean duration 20yrs) with no polydrug use vs controls
- Bilateral ↓ hippocampal volume (12%) and amygdala volume (7%)
- (L) hippocampal volume inversely associated with cumulative exposure
- +ve psychotic symptoms also associated with cumulative exposure

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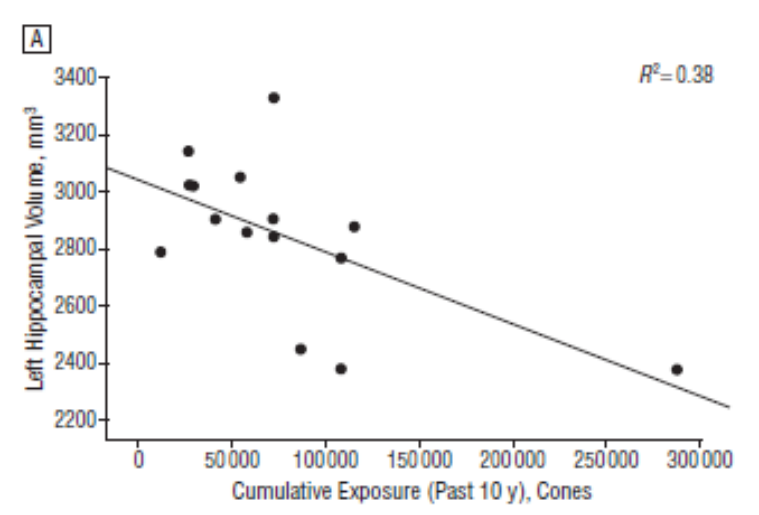
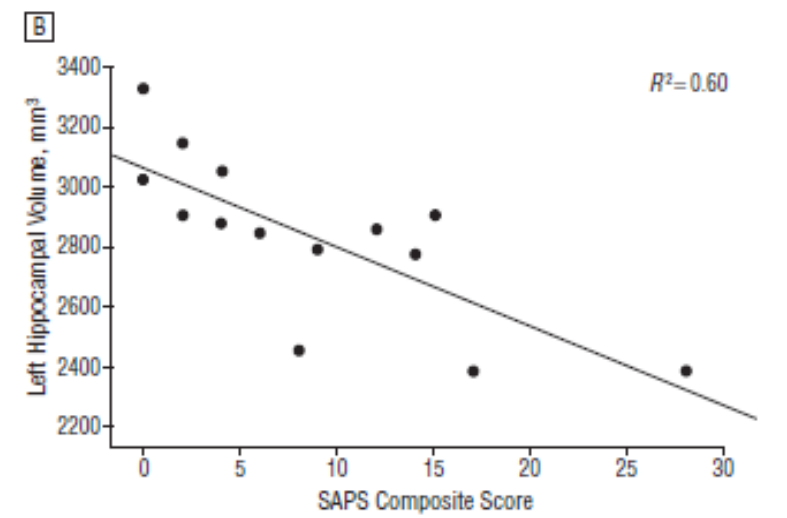
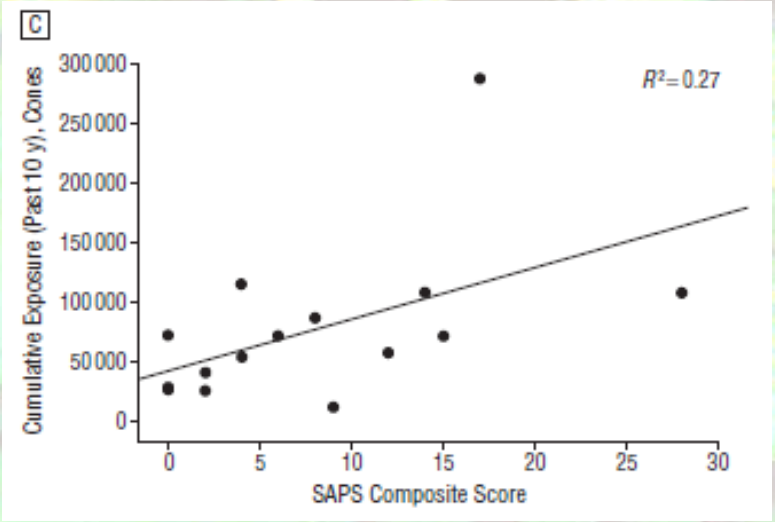
Effects on Brain Structure (3)

(Yucel et al, 2008)







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Effects on Brain Structure (4)

- recent review of all neuroimaging studies in cannabis use (Martin-Santos et al, 2010)
  - 8 structural imaging studies
  - only 3 showed any difference between users and controls
  - Variety of sites (hippocampus, parahippocampal grey, amygdala, parietal white matter)
  - Conclusion: “Minimal evidence of major effects of cannabis on brain structure has been reported”

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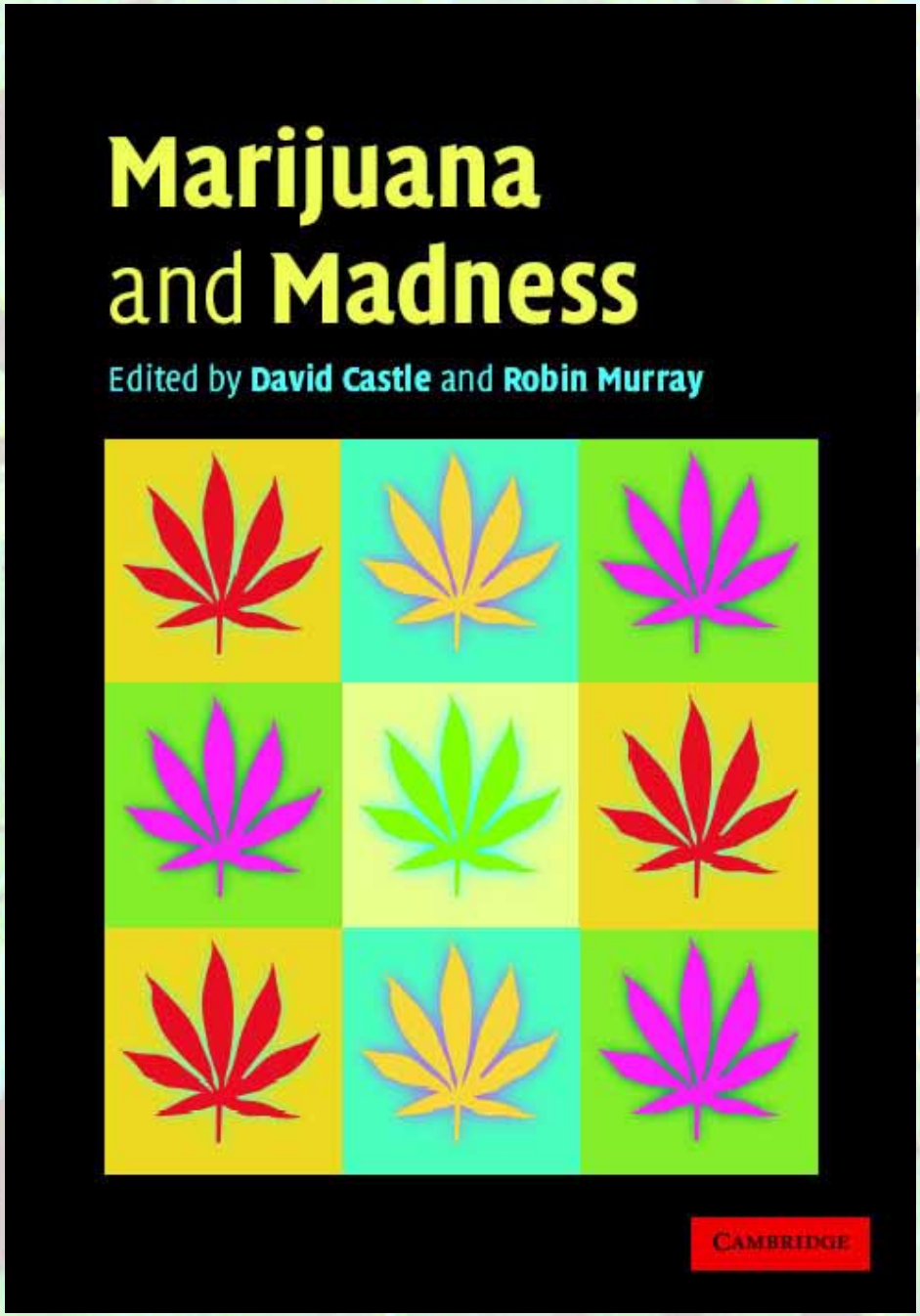

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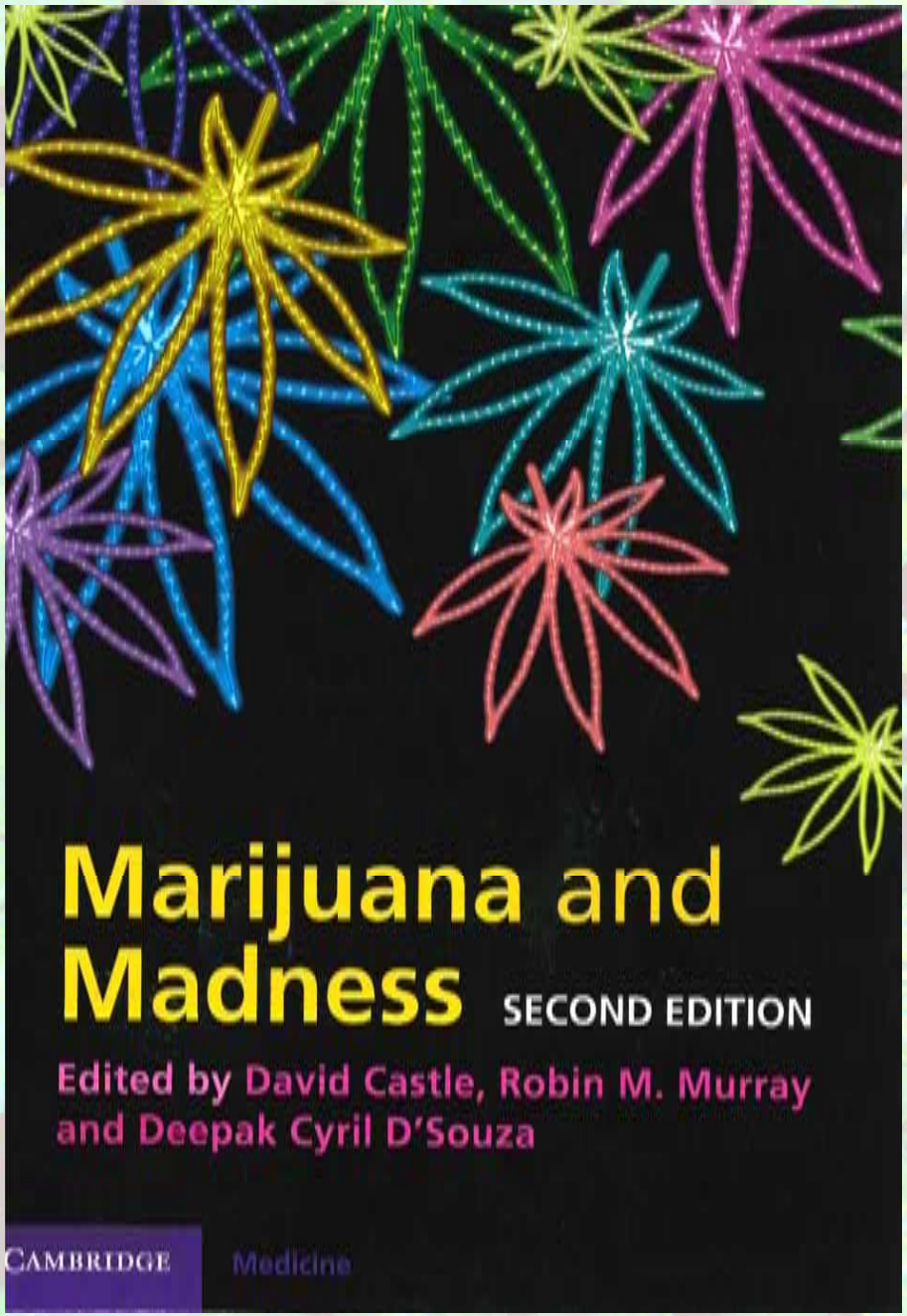

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