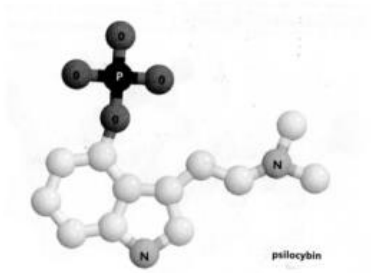
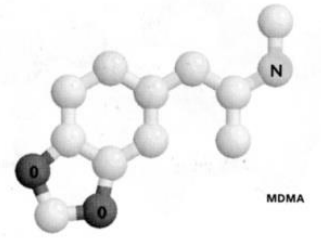
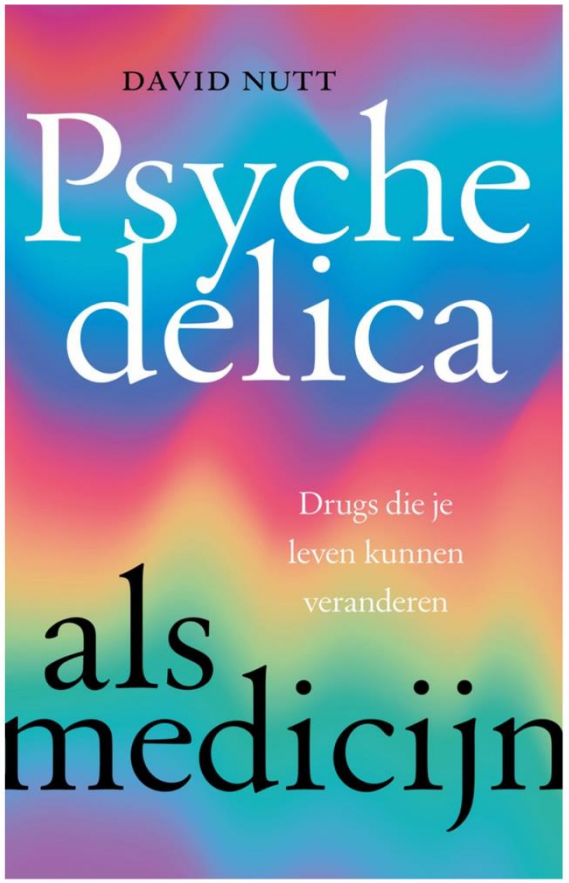


Psychedelics Masterclass





David Nutt FMedSci DLaws

Prof of Neuropsychopharmacology

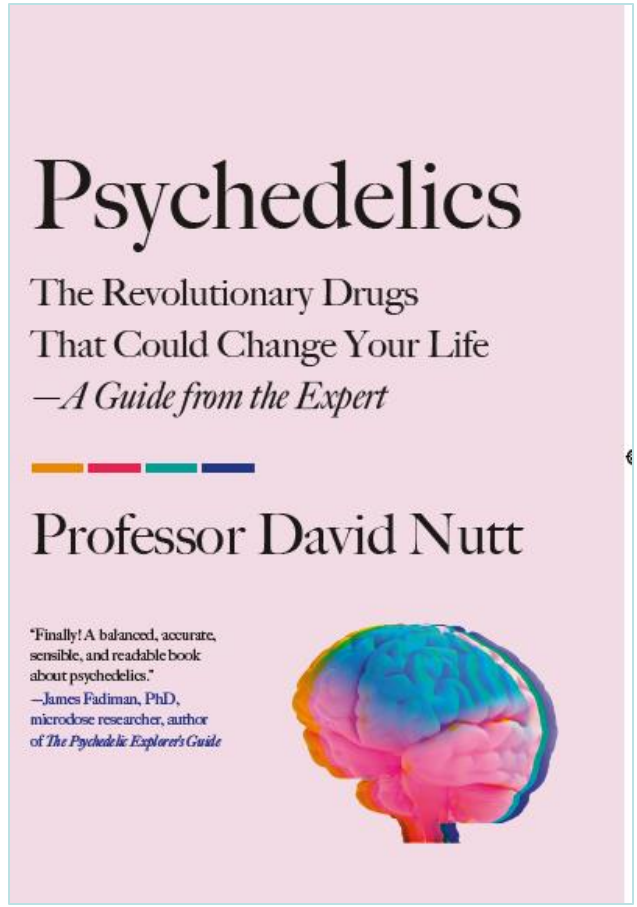
Imperial College London

d.nutt@imperial.ac.uk

CRO Awaknlifesciences

profdavidnutt@twitter.com

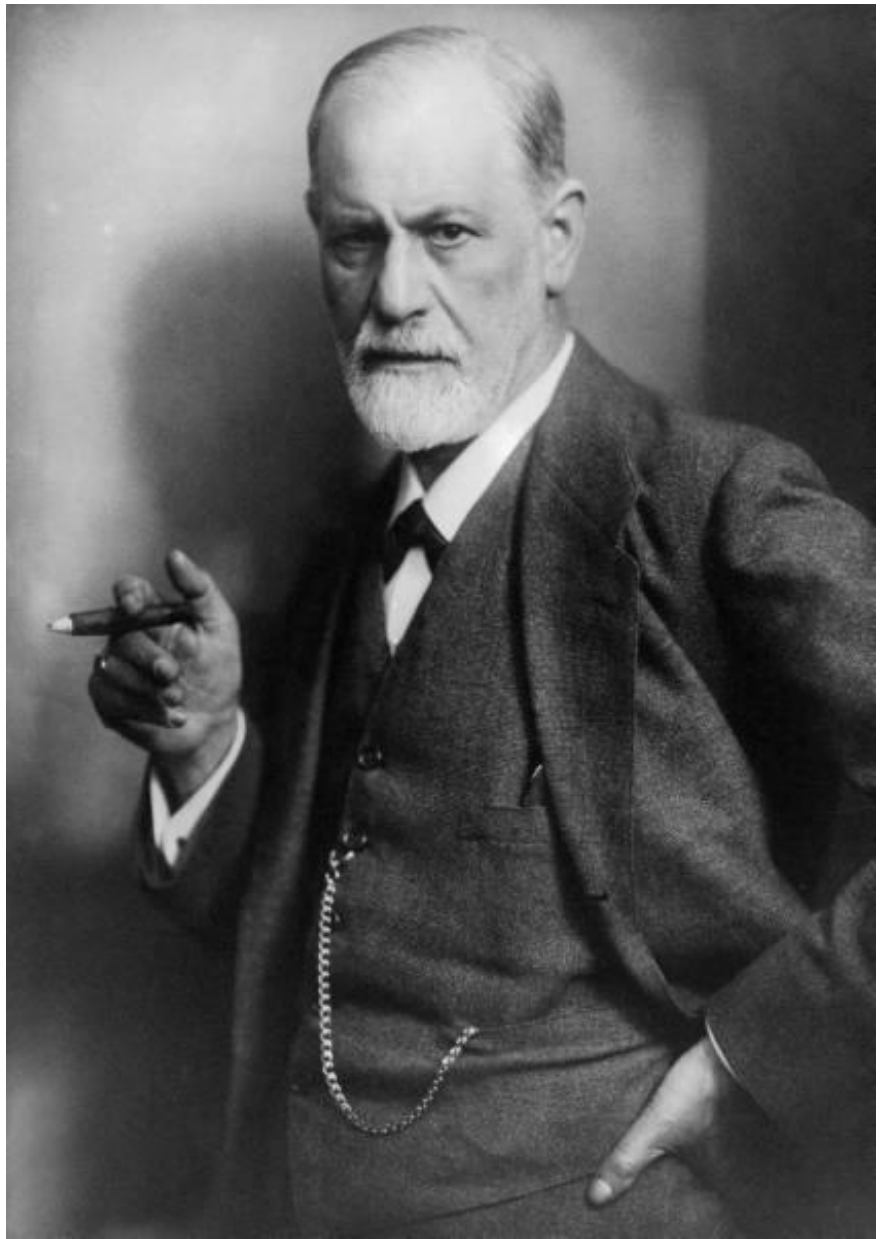
Podcasts on www.drugscience.org.uk



Declaration of interests – 2022-2024

- **Chair – DrugScience [UK] - & PAREA Europe (Psychedelic Access and Research European Alliance)**
- Member International Centre for Science in Drug Policy
- Editor of the journal Drug Science policy and law
- Advisory Boards - AWAKN, Psyched Wellness, Neural Therapeutics
- Speaking honoraria - Lundbeck, BMS/Otsuka, Janssen, Takeda
- Grants and clinical trial support - Wellcome Trust, MRC, Compass Pathways, Usona, Filament
- Director Equasy Enterprises and GABA Labs. Share options – Psyched Wellness
- Expert witness in some legal cases relating to psychotropic drugs
- Edited/written 40 books - some purchased by pharma companies

The Forefather of Psychedelic Therapy?

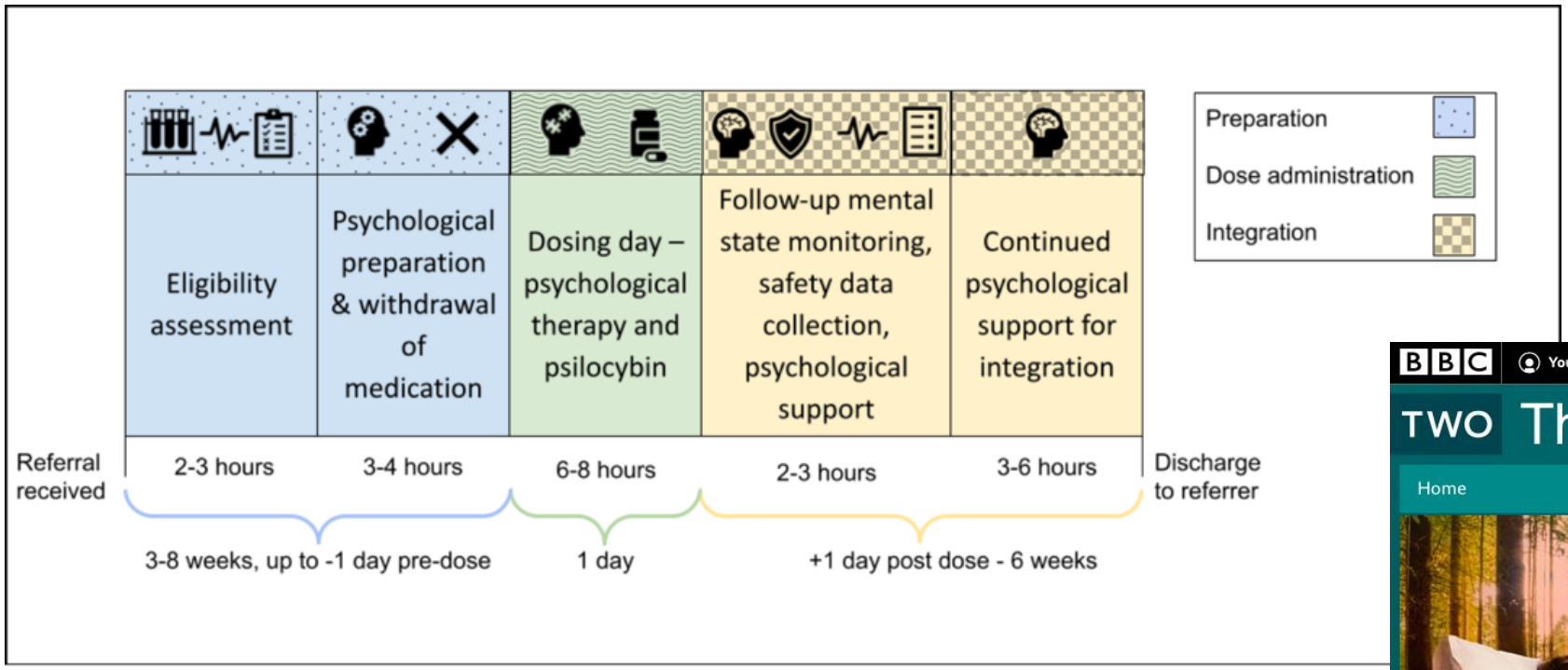


"The future may teach us how to exercise a direct influence, by means of particular chemical substances, upon …the neural apparatus. It may be that there are other still undreamt of possibilities of therapy."

From *An Outline of Psychoanalysis*

Sigmund Freud
London 1938.

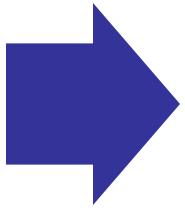
How we do psychedelic therapy



Question:
Is therapy necessary?

Subjective experience of recovery
Some of our patients' quotes-

How do psychedelics heal the mind ?



Watts et al Journal of Humanistic Psychology 2017, Vol. 57(5) 520

Resetting thinking…

It was like when you defrag the hard drive on your computer, I experienced blocks going into place, things being rearranged in my mind, I visualized as it was all put into order and I thought my brain is bring defragged, how brilliant is that! p11

***My outlook has changed significantly. I'm more aware now that it's pointless to get wrapped up in endless negativity. I feel as if I've seen a much clearer picture*.** **p1**

Watts et al Journal of Humanistic Psychology 2017, Vol. 57(5) 520

Better connections to the world

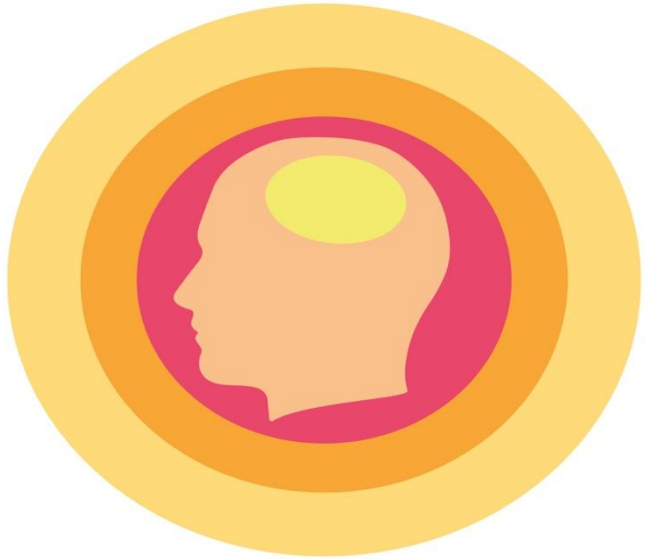
After [the session] when I went outside, everything was very bright and colourful and it felt different. I noticed things I didn't notice usually, the leaves on the trees and the birds, small details. (P7)

[For months] I would look at the park across the road from my flat: so green, a type of green I'd never experienced before. Being among the trees was incredible, like experiencing them for the first time, so vibrant, so alive. Things look different even now. (P12)

Watts et al Journal of Humanistic Psychology 2017, Vol. 57(5) 520

Better connections to …. emotions

[I] became myself at age 7, after my [grandparent] had died. I totally was back there, so vivid, so real, I had the emotions that I would have felt at the time: fearful, why did this happen, the naivety, the shock. (P12)



I have felt a sense of acceptance; more acceptance of agony, boredom, loneliness, and also appreciation of the wonderful times. [A] willingness to try to accept the negative times. (P5)

Watts et al Journal of Humanistic Psychology 2017, Vol. 57(5) 520

Better connections to …..body

It helped me realise why I felt the pain in my chest, I saw it visually and felt it emotionally, then I felt so much lighter, like something had been released. (P11)

I lost a lot of weight just purely because I didn't want to eat badly and that went on for some months. I couldn't eat what I knew wasn't good for me. (P19)

Watts et al Journal of Humanistic Psychology 2017, Vol. 57(5) 520

Better connections to to self

Self worth/self love: *'I had an experience of tenderness towards myself, a feeling of true compassion I'd never felt before (P16)*

Inner therapist: 'Its like ingesting your own psychotherapist' (P19)

.....spiritual

I felt the presence of God: I have always thought that he was a man because of the way I was raised, reading the bible, but it felt like a female energy. (P7)

I was taken in a rapture and was floating in mid air, completely in a state of awe and ecstasy. It's a very powerful message to take away. (P18)

Watts et al Journal of Humanistic Psychology 2017, Vol. 57(5) 520

Other treatments → disconnection

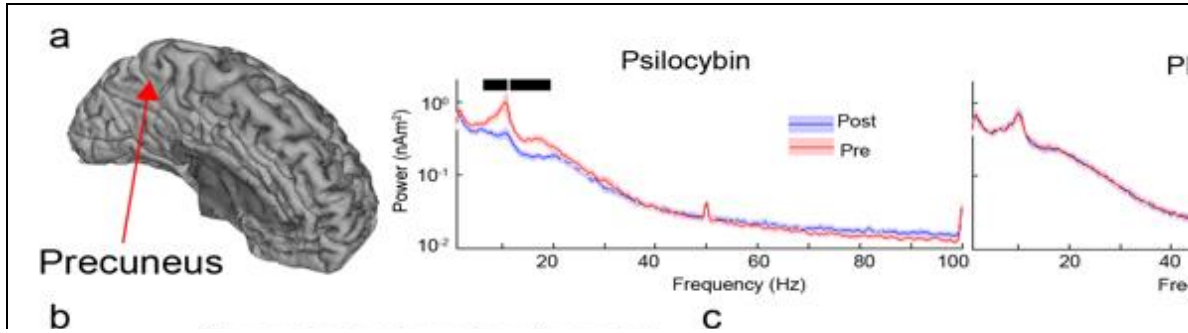
- **Antidepressants – blunt emotions** *"Where escitalopram nulled me to be less scared, psilocybin has made me realise that fear is not something to be feared*

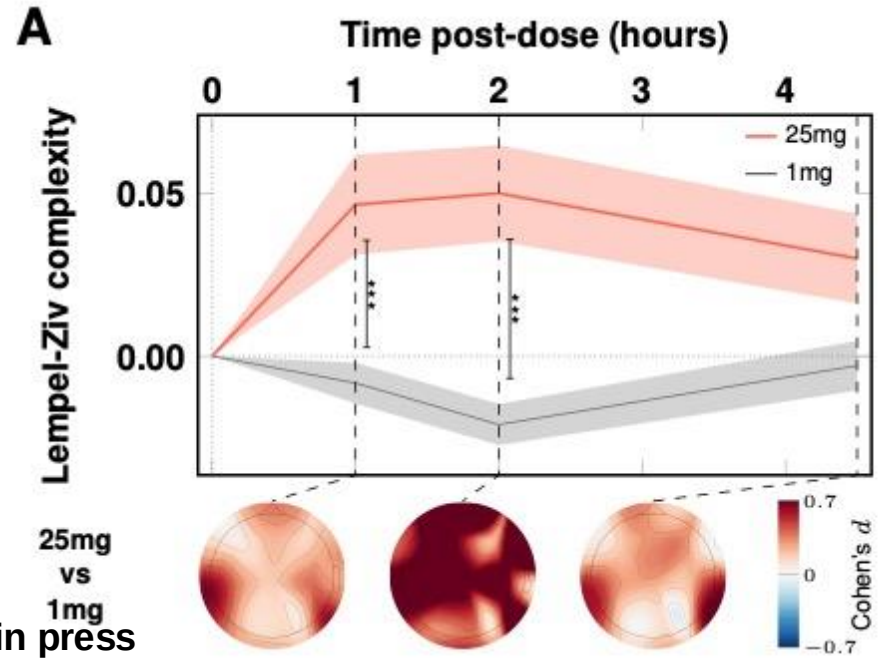
- **CBT – didn't work -→ just reinforces sense of failure**
  - **And doesn't address trauma** *" I got up the courage to tell him, I'd never told anyone. And [the psychiatrist] just looked at his shoes.' (P16)*

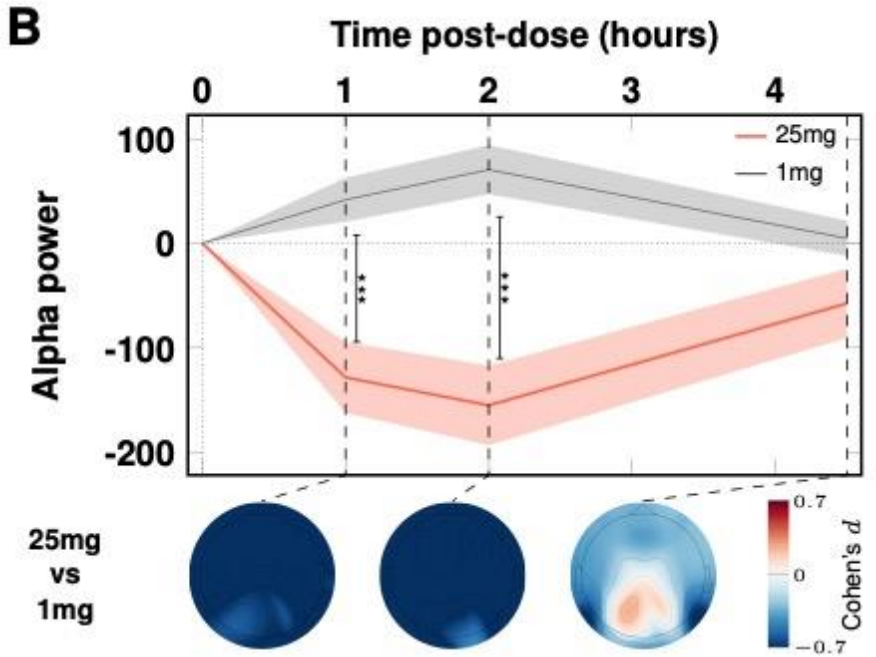
Watts et al Journal of Humanistic Psychology 2017, Vol. 57(5) 520

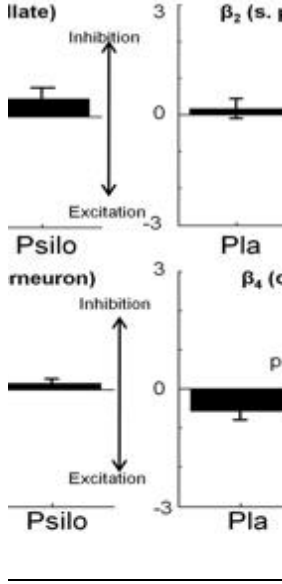
How does the trip experience and the effect between high-dose and micro-dose psychedelic compare on fMRI and therapeutic efficacy?

Psilocybin → EEG dose effect 25-v-1 mg









Imperial College in press

6.6. Figures S26. Adapted version of 'Persisting Effects Questionnaire' of Griffiths et al. 2006.

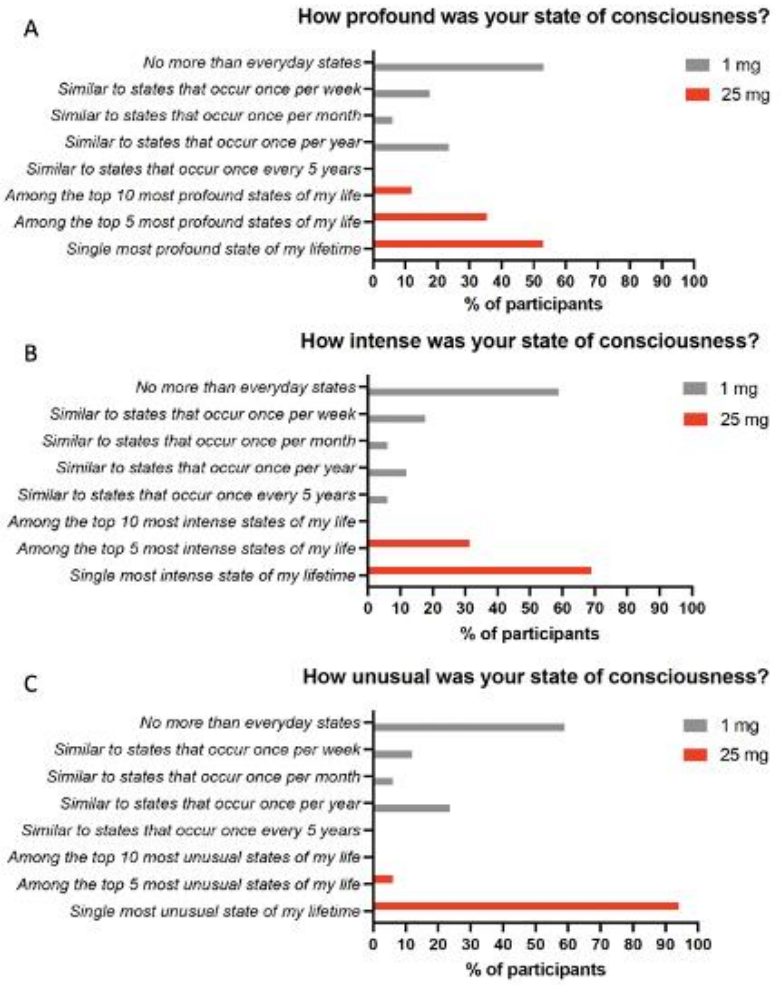


Figure S26. One-month after each dosing session, we asked all participants to assess the quality of the state of consciousness they had experienced during each dosing session using the labelled ranking criteria shown on the y-axis of the above charts. Participants ranked how: **A)** *profound*, **B)** *intense*, and **C)** *unusual* their state of consciousness was in relation to their life up to that moment. They did this after both the 1mg (gray) and 25mg psilocybin experiences (red). This scale was an adapted version of the Persisting Effects Questionnaire used in [4].

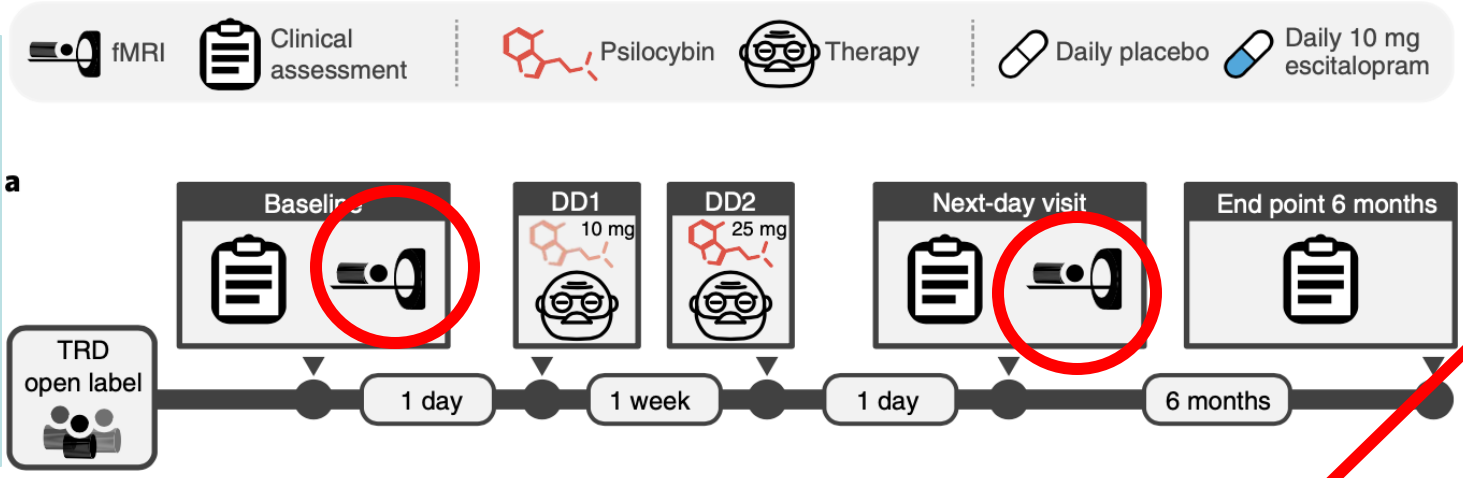
Insight study

1mg –v- 26 mg of psilocybin

Lyons in prep

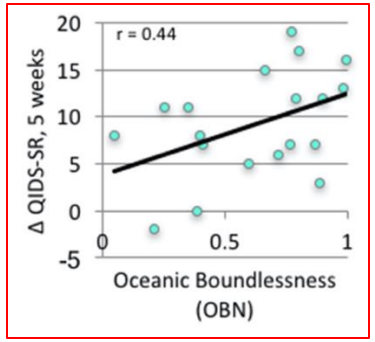
Mechanisms - explored via fMRI across our two depression trials

Imperial N=20 depression study 1



Post psilocybin MRI on day 1

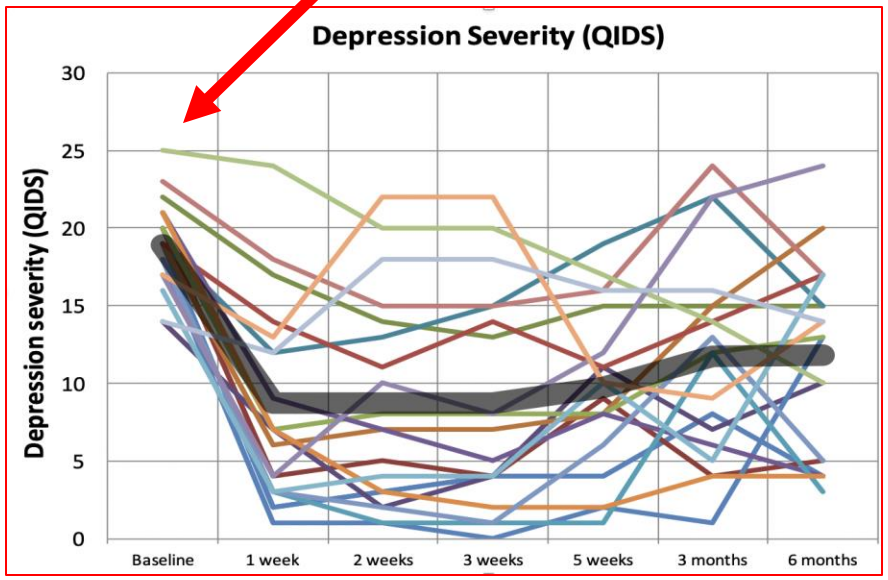
Acute state predicting outcomes



Psilocybin with psychological support for treatment-resistant depression: an open-label feasibility study

Robin L Carhart-Harris, Mark Bolstridge, James Rucker*, Camilla M J Day*, David Erritzoe, Mendel Kaelen, Michael Bloomfield, James A Rickard, Ben Forbes, Amanda Feilding, David Taylor, Steve Pilling, Valerie H Curran, David J Nutt

Imperial's open label pilot study in 20 patients with treatment-resistant moderate-severe major depression[4]

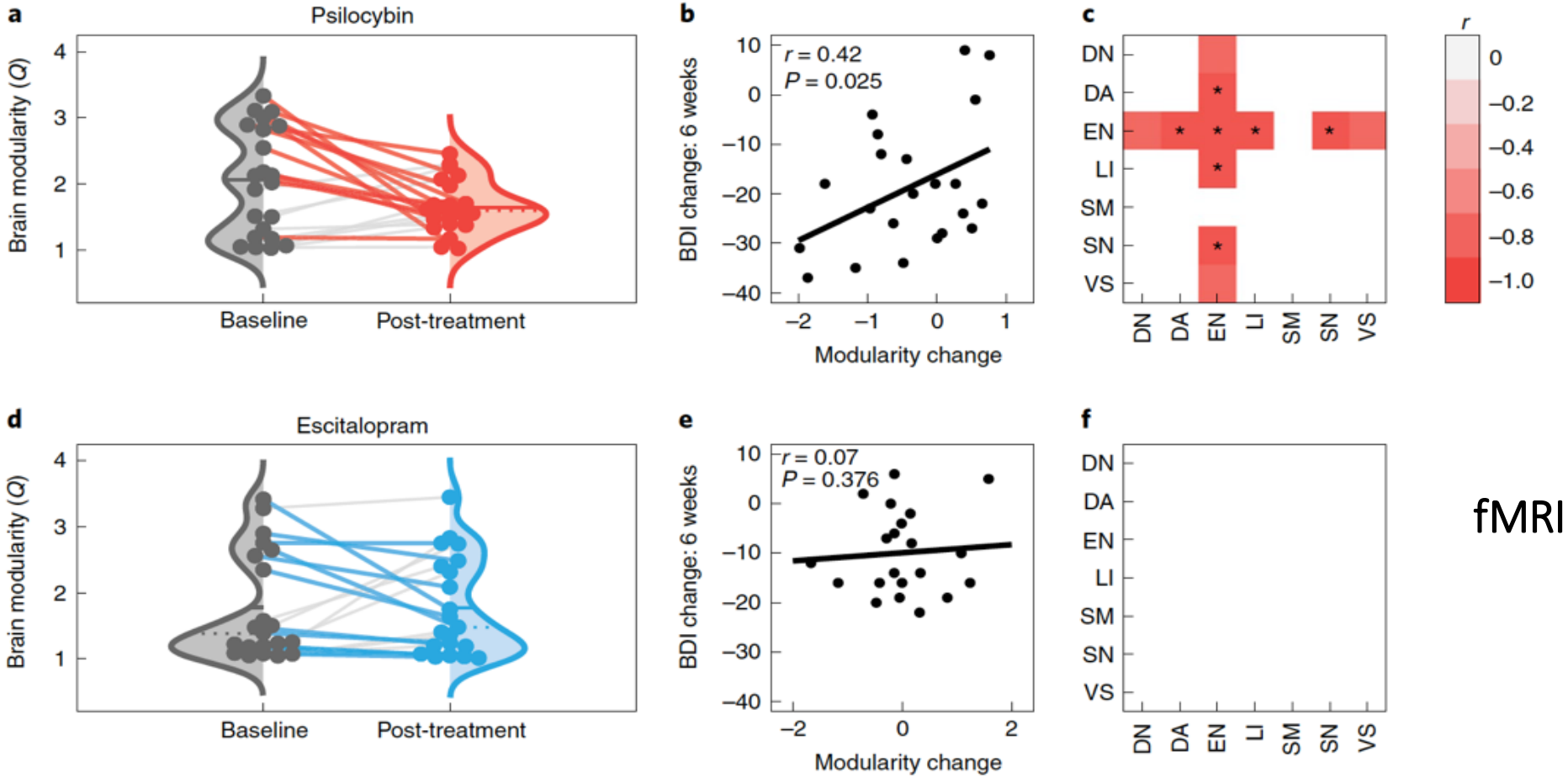


Roseman et al 2018

For similar data from recent depression study see Brandon Weiss talk

d.erritzoe@imperial.ac.uk

Increased network flexibility (reduced brain modularity) after psilocybin but not escitalopram in depression



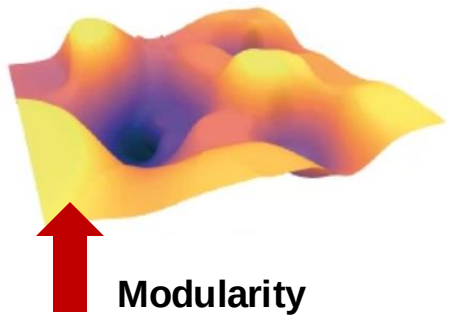
fMRI

Fig. 5 | Increased global brain network integration correlates with treatment response following psilocybin, but not following escitalopram. a, Significant

e Medicine

A model of psychedelic psychotherapy

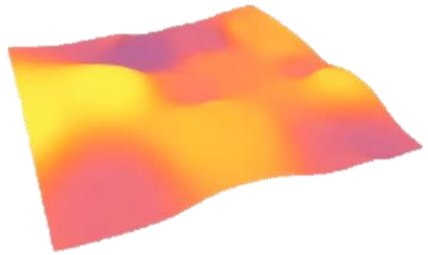
Depression/Addiction



Modularity

Patients locked into repetitive thought loops

Psychedelic state

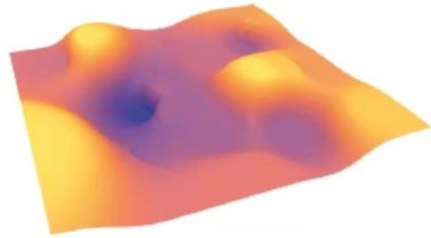


Global functional integration, entropy, forward waves, flattening of energy landscape

+

Psychotherapy

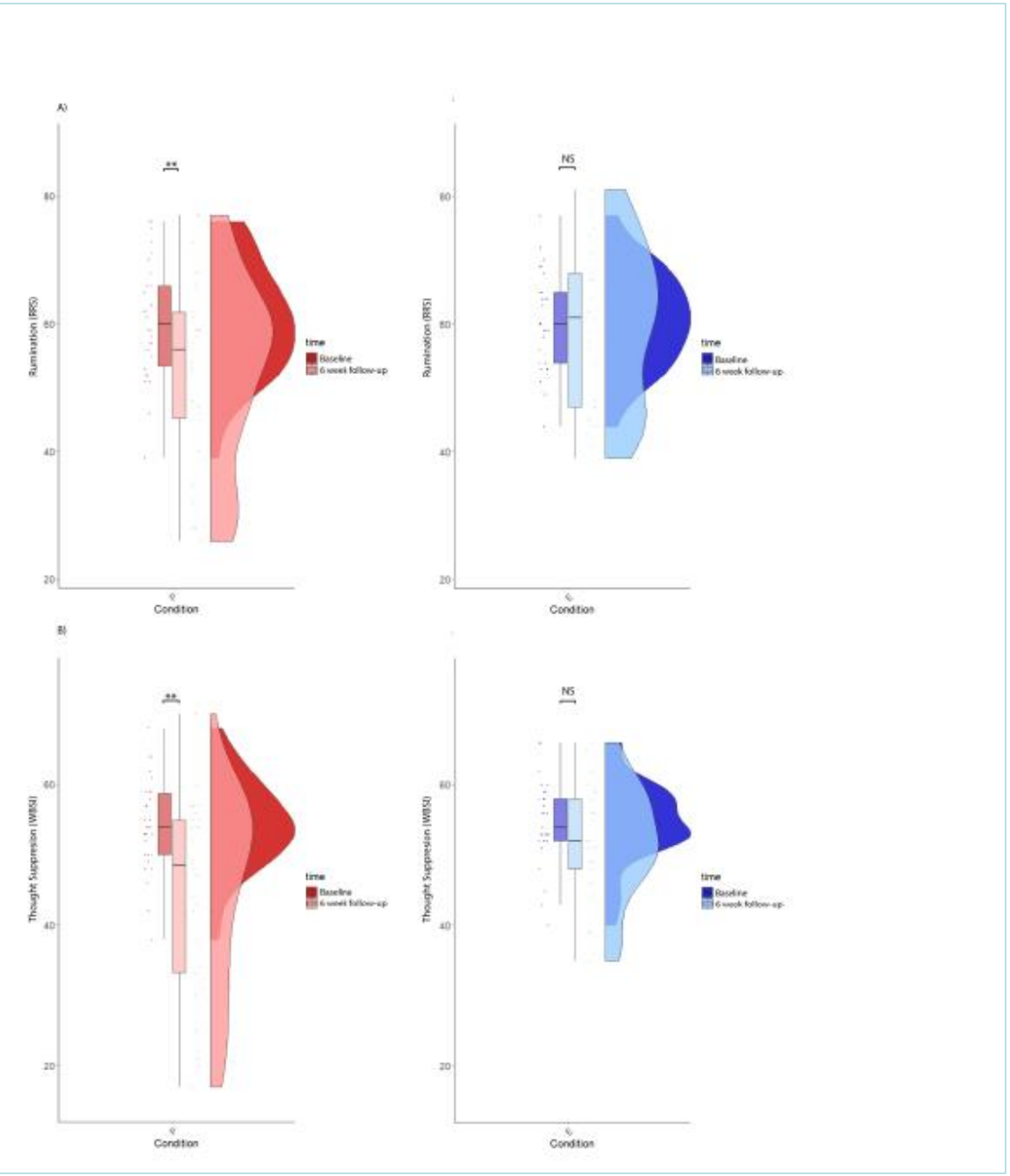
Post-treatment



Modularity

More flexible thinking - escape from ruminations

From Daws et al Nature Medicine 2022

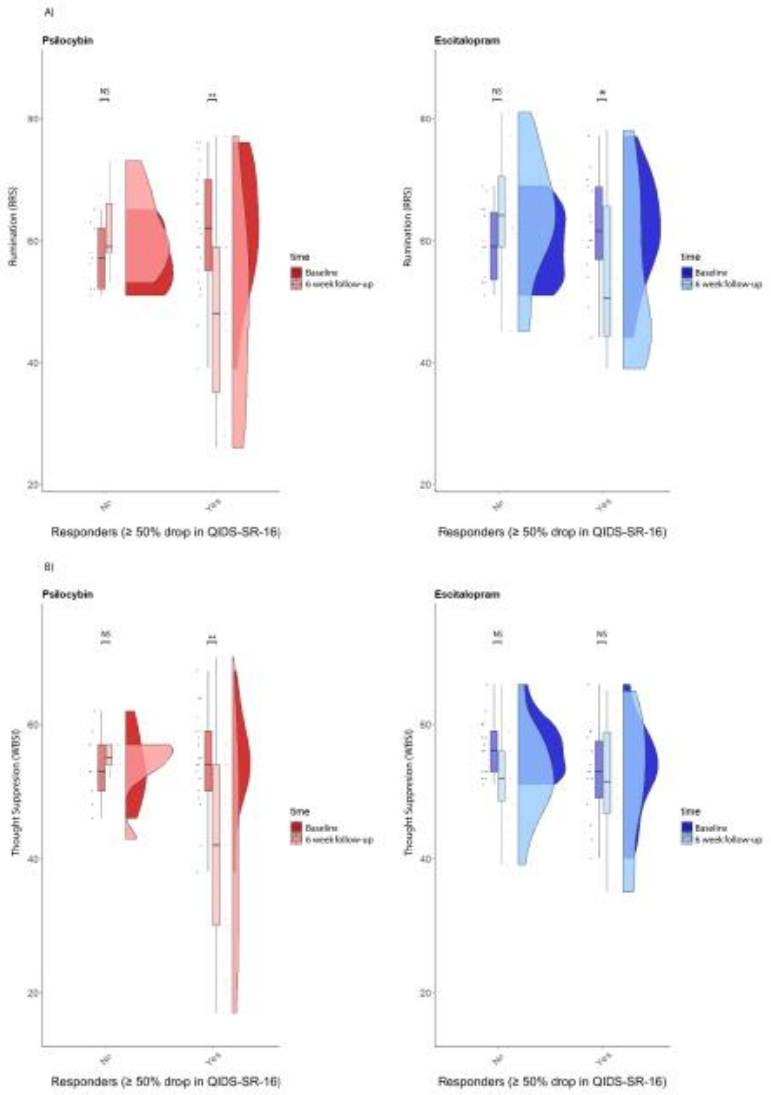


Rumination (top) and thought suppression (bottom) scores before and after treatment

Psilocybin – red
Escitalopram – blue

Dark colour before
Light colour at 6 weeks

Barba T et al (2022) BJPsych Open



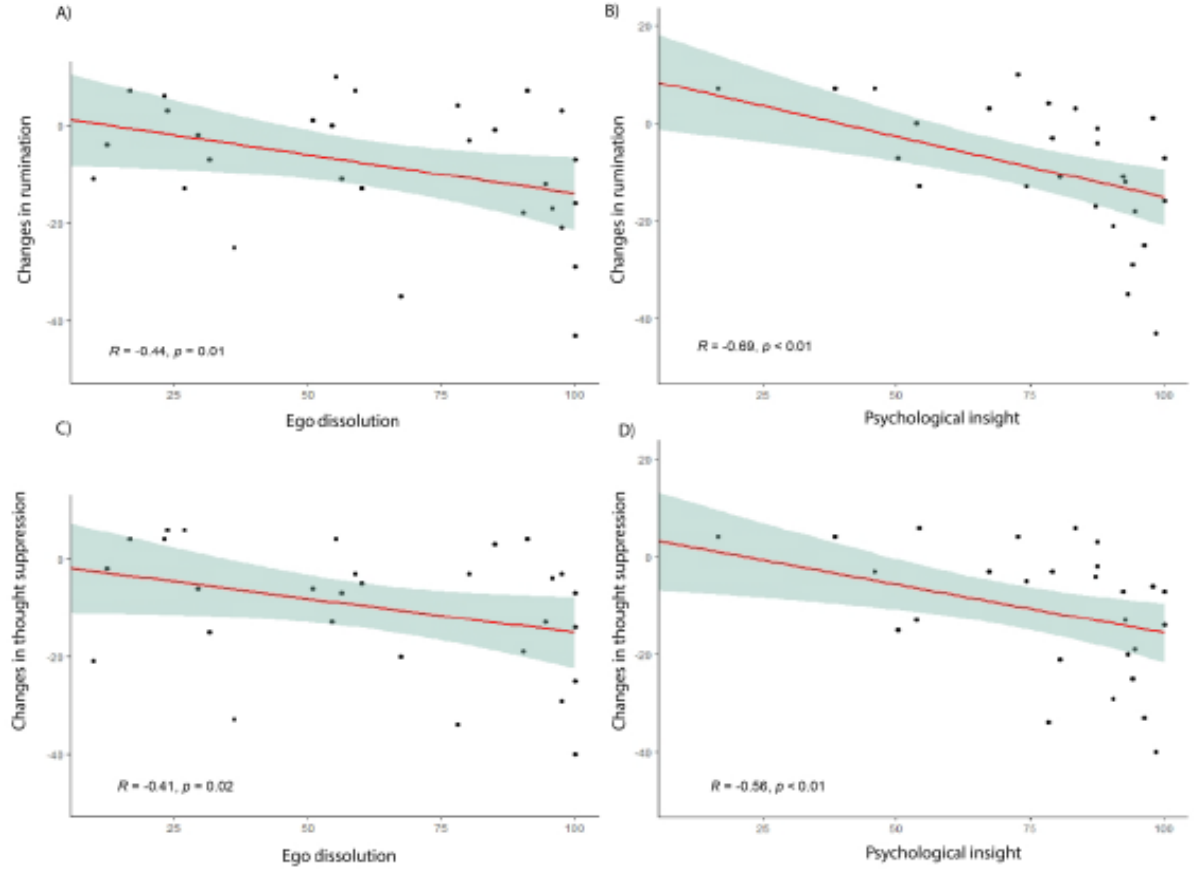
Responder analysis non-responders

Psilocybin – red
Escitalopram – blue

Dark before –
Light at 6 weeks

Barba T et al (2022) BJPsych Open

Acute psilocybin effects predict outcomes



Psilocybin-escitalopram trial

Barba T et al (2022) BJPsych Open

Learnings from our studies

Predictors of good outcomes	No or negative effect
Breakthrough / mystical type experiences **++**	Expectation for psilocybin to work (BUT does for escitalopram)
? Single trauma aetiology → longer remission	Anxiety during trip **- -**
Non-psychedelic (midi) doses eg 5-10mg psilocybin can have significant emotional releasing effects → OCD and impulsive cutting reframing studies	? Stopping SSRIs may reduce effect of psilocybin

What about other disorders?

A (cholinergic) psychedelic experience led to the founding of AA in 1933 by Bill Wilson

"Suddenly the room lit up with a great white light. I was caught up in an ecstasy which there are no words to describe. It seemed to me in my mind's eye, that I was on a mountain and that a wind not of air but of spirit was blowing. ***And then it burst upon me that I was a free man."***

Years after this psychedelic-induced sobriety conversion Bill Wilson experienced LSD (with Huxley) and came to believe that it could help "cynical alcoholics" achieve spiritual awakening …. more later

6 LSD trials in alcoholism

1970	problems and treatment intentions	quiet room	group therapy

	Follow-up (months)	LSD (n/N)	Control (n/N)	Weight	Odds Ratio (95% CI)
First follow-up					
Smart *et al.*, 1966	6	[a]/10	[a]/20	7.2%	1.41 (0.36-5.60)
Hollister *et al.*, 1969	2	18/36	11/36	14.7%	2.27 (0.87-5.94)
Ludwig *et al.*, 1969	1	88/132	31/44	27.3%	1.88 (0.93-3.81)
Bowen *et al.*, 1970	12	9/22	7/22	8.9%	1.48 (0.43-5.10)
Pahnke *et al.*, 1970	6	34/73	13/44	21.6%	2.08 (0.94-4.60)
Tomsovic & Edwards, 1970	3	30/52	17/45	20.4%	2.25 (0.99-5.10)
Total		**325**	**211**	**100%**	**1.96 (1.36-2.84)**

Test for heterogeneity: $\tau^2 = 0.00$; $\chi^2 = 0.65$, df = 5 ($P = 0.99$); $I^2 = 0\%$

Test for overall effect: Z = 3.59 ($P = 0.0003$)

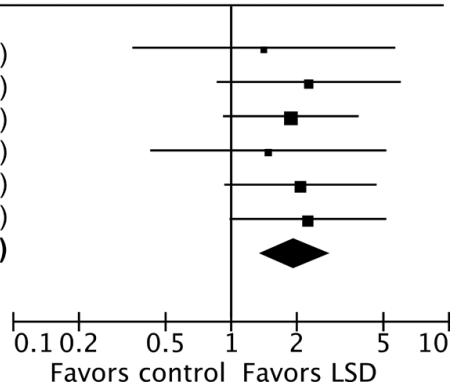


Figure 2. Improvement on alcohol misuse at the first available follow-up after LSD versus control treatments.

[a]Continuous outcome data.

Effect size >= all current therapies

Journal of Psychopharmacology 2012 Krebs and Johanssen

A thought experiment – what if psychedelics had not been banned?

Assumptions: since 1970 @ 100million alcohol related deaths globally

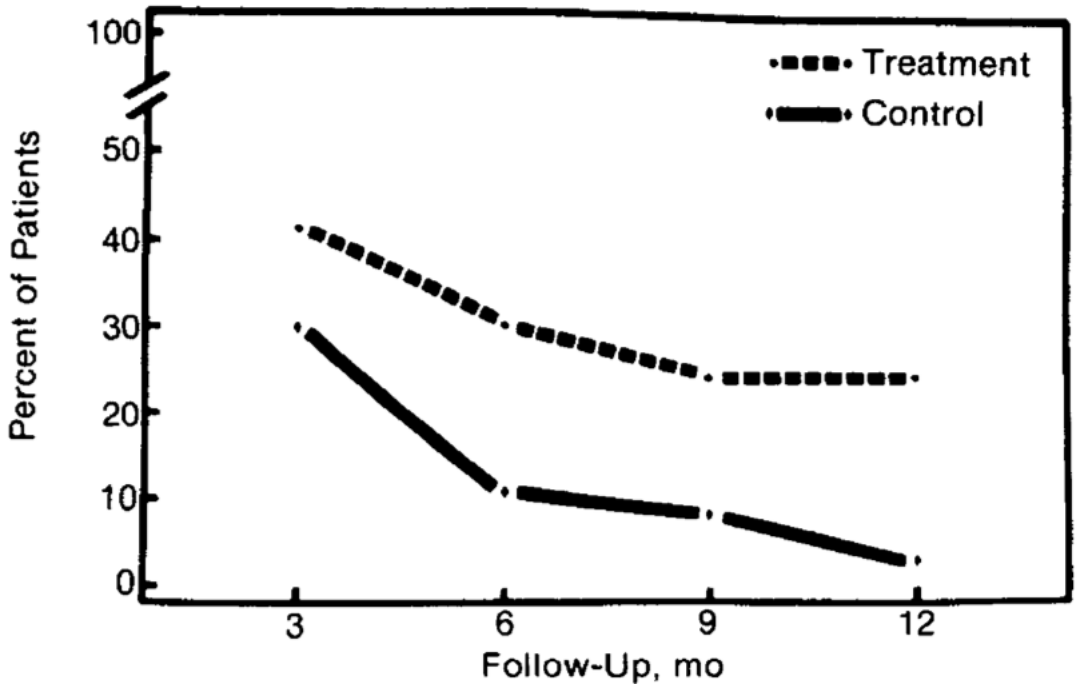
If LSD healed just 10% = 10 million premature deaths prevented

How many lives saved by the psychedelic ban?
- likely none – say 1000….. 10,000…..

Benefit-risk equation very much in favour of treatment …….

LSD for heroin addiction

Fig 1.—Percent of patients maintaining total abstinence at 3-, 6-, 9-, and 12-month follow-up.



Leonard N

The two experiences of heroin and LSD are like night and day. Heroin is night, a time to sleep and with sleep nothing comes. But with LSD it is like dawn, a new awakening, it expands your mind, it give you a brand-new outlook on life.

Arch Gen Psych 1973

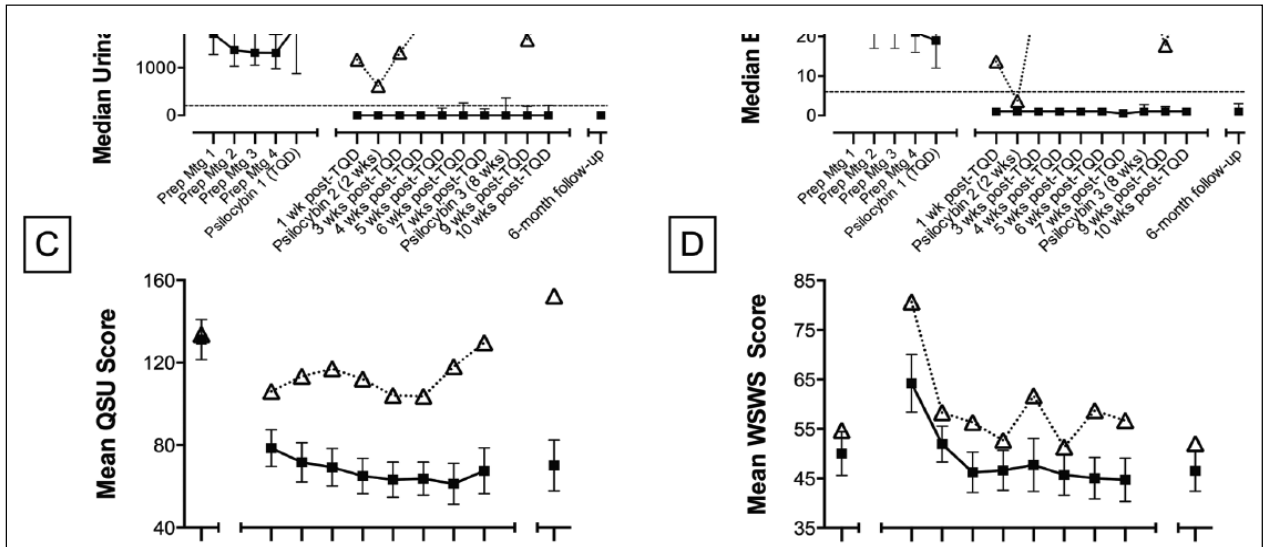
Residential Psychedelic (LSD) Therapy for the Narcotic Addict

A Controlled Study

Charles Savage, MD, O. Lee McCabe, PhD, Baltimore

Psilocybin for tobacco quitting

Tobacco quitting – Johnson 2014 – J of Psychopharmacology



Johnson – unpublished data on 82 treatment- resistant smokers
1 dose psilocybin -v- nicotine patch
52% psilocybin – abstinent -v- 25 patch at 6 months
Improved cognition in oddball task = less cognitive interference = less automaticity

Stops mood symptoms of nicotine withdrawal – no effect on physical ones

US National. Institute for Drug Abuse now funding a new larger quitting study

Psilocybin for alcohol dependence

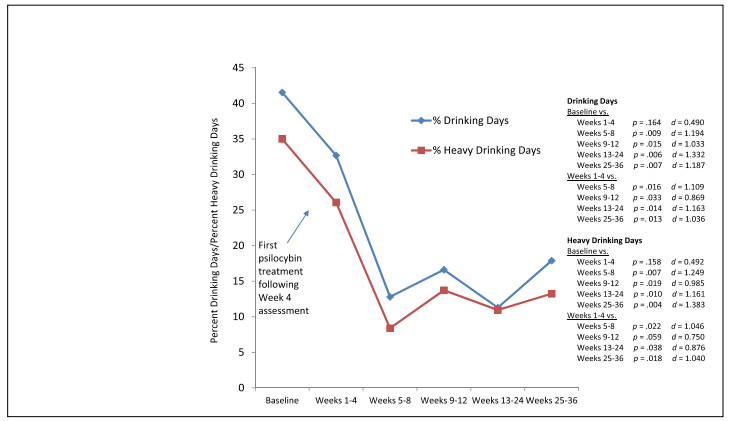


Figure 3. Drinking outcomes and effect sizes.
Means shown are for all available data (n = 10 at baseline, n = 9 at all other time points). p-values are from paired t-tests (df = 8). Cohen's d is shown for the contrast between baseline or weeks 1–4 and each follow-up time point.

Bogenschutz et al
J of Psychopharmacology 2015

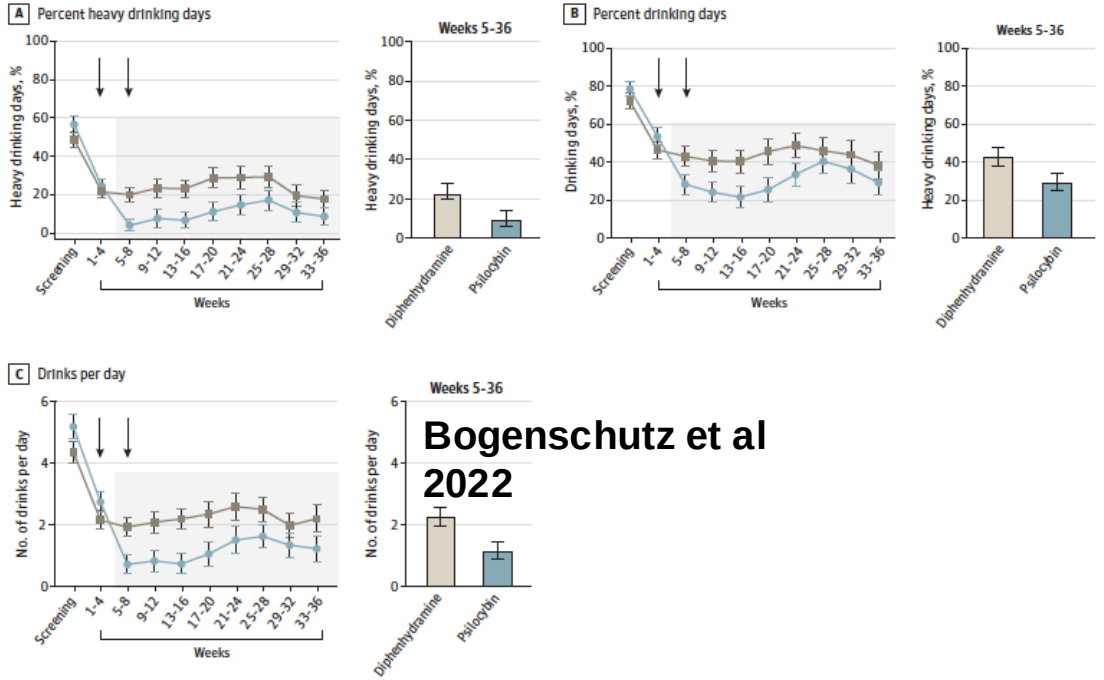
Research

JAMA Psychiatry | Original Investigation

Percentage of Heavy Drinking Days Following Psilocybin-Assisted Psychotherapy vs Placebo in the Treatment of Adult Patients With Alcohol Use Disorder
A Randomized Clinical Trial

Michael P. Bogenschutz, MD; Stephen Ross, MD; Snehal Bhatt, MD; Tara Baron, MA; Alyssa A. Forcehimes, PhD; Eugene Laska, PhD; Sarah E. Mennenga, PhD; Kelley O'Donnell, MD, PhD; Lindsey T. Owens, MA; Samantha Podrebarac, MA; John Rotrosen, MD; J. Scott Tonigan, PhD; Lindsay Worth, MA

Figure 2. Effects of Treatment on Continuous Drinking Outcomes



Bogenschutz et al 2022

Mean (SE) estimates for screening (84 days prior to screening), weeks 1-4 (28 days prior to first double-blind medication session; covariate in the model), and eight 28-day bins following the first double-blind medication session (shaded area: weeks 5-8, 9-12, 13-16, 17-20, 21-24, 25-28, 29-32, and 33-36). Arrows represent double-blind medication sessions 1 and 2.

New studies starting at Imperial College

1. Psilocybin for maintaining abstinence in opioid dependence

i4i Addiction Mission: Innovation for Treatment and Recovery (AMI) - Stage 1

NIHR | National Institute for Health and Care Research

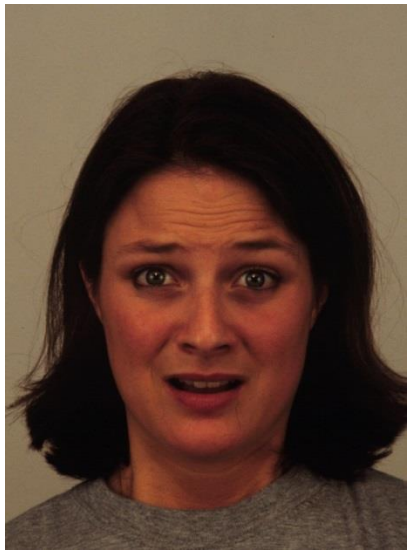
Reference number	NIHR206736
Programme	Invention for Innovation
Call	i4i Addiction Mission: Innovation for Treatment and Recovery (AMI) - Stage 1
Lead Applicant	Dr David Erritzoe
Host organisation	Imperial College of Science, Technology and Medicine
Research Title	A Phase 2a dose ascending, placebo randomised controlled trial, to investigate psilocybin therapy and brain reward mechanisms in opiate use disorder (OUD).

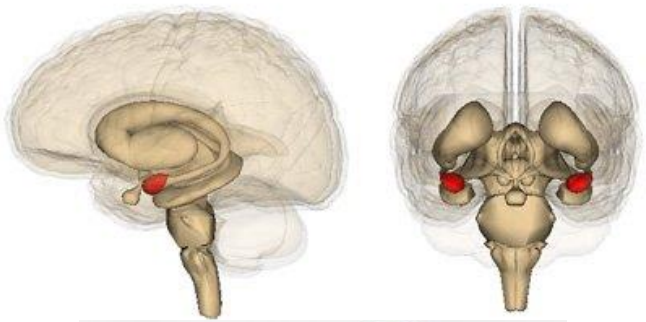
Clinical and imaging outcomes after single 25 mg trip dose

2. Psilocybin for gambling disorder
   – Pilot study for proof of principle

But what about SSRIs? Amygdala – Faces - SSRIs

Fearful



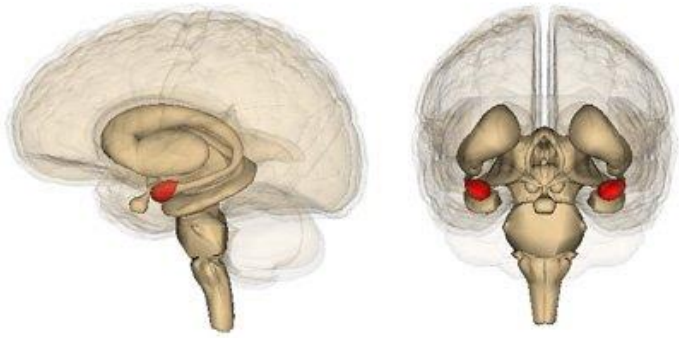


Depression > “Healthy”

SSRIs reduce activity

Amygdala in psilocybin or escitalopram assisted therapy

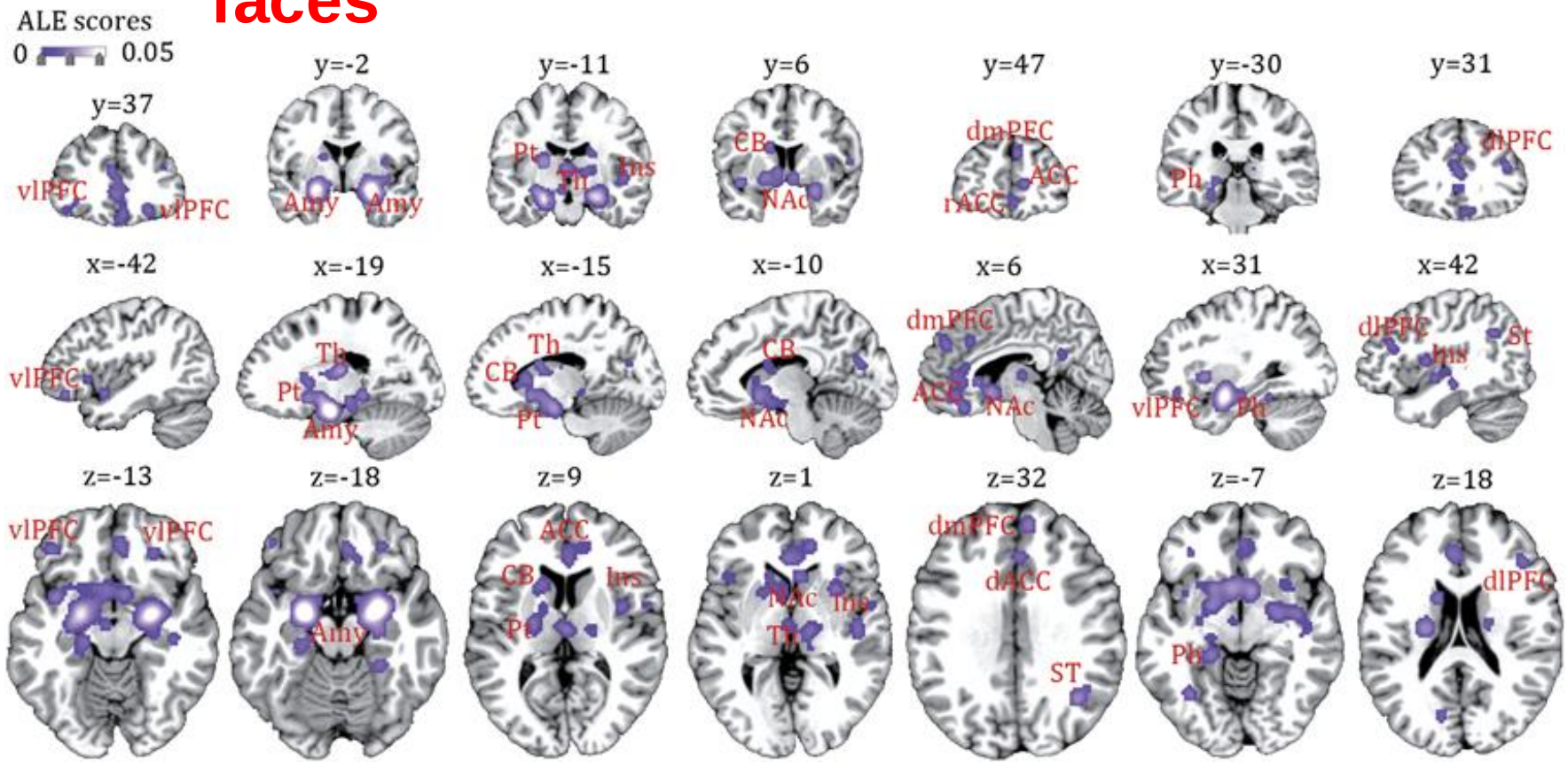
Baseline and after therapy
Passively viewing
Blocks of 15 seconds



Roseman et al 2018

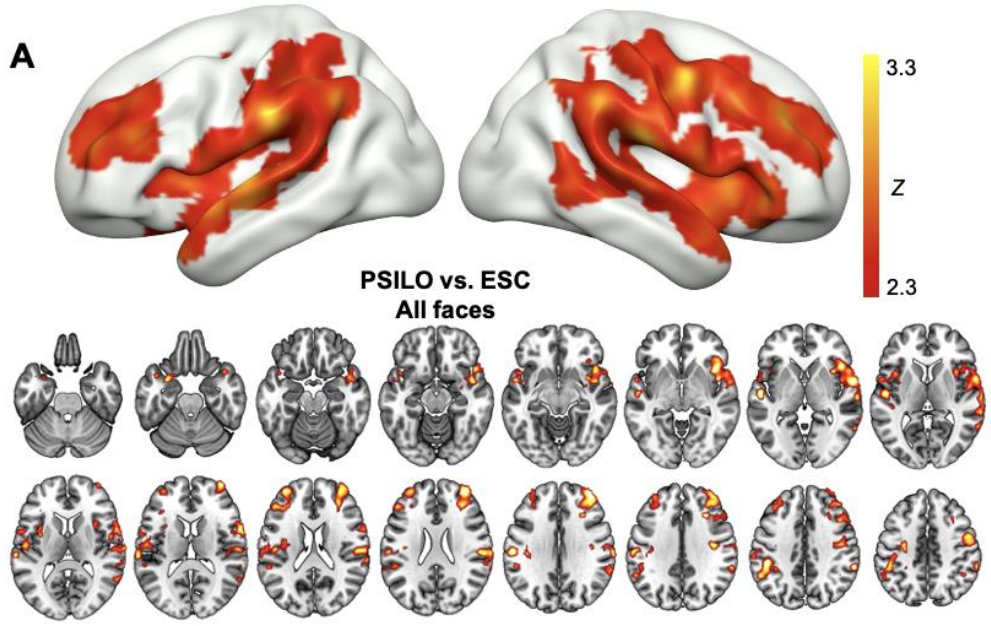
Antidepressants and amygdala - meta-analysis

→ **Reduced activity to negative emotional faces**

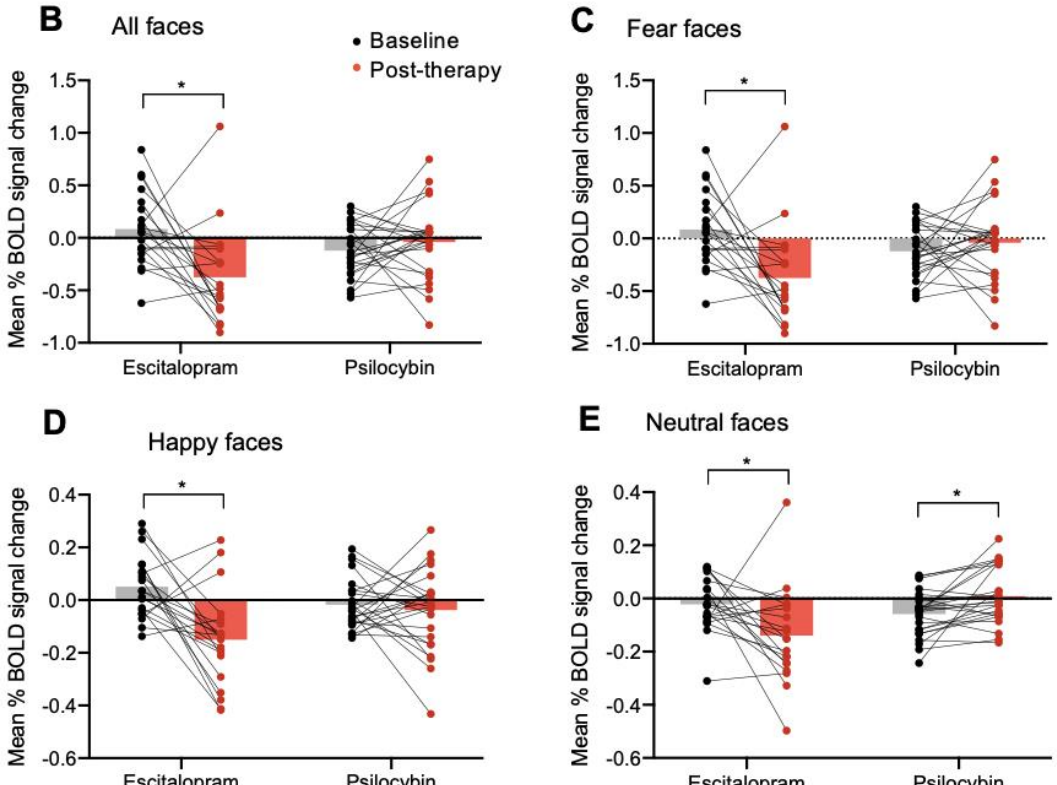


Ma et al. (2015), *Molecular psychiatry*

Psilocybin-v- escitalopram fMRI – faces



Panel A – regions of significantly greater activation in psilocybin group (all faces)

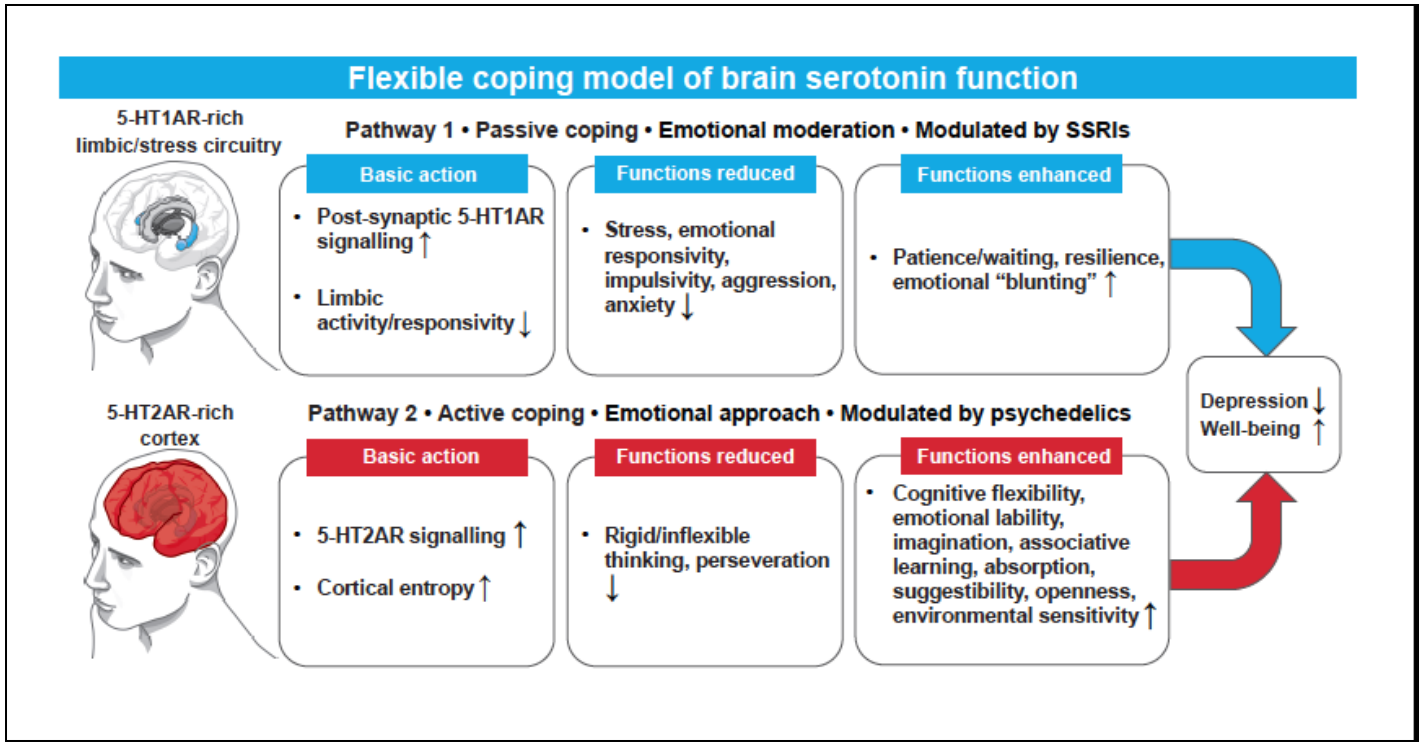


Escitalopram blunts with all emotions

Psilocybin does not blunt and may enhance neutral

Wall et al submitted

There are now two ways to lift depression
Different brain regions and different 5-HT receptors



At last depression on par with other common disorders e.g. hypertension and cancer that benefit from mechanistically-different treatment options

Carhart-Harris and Nutt – Journal of Psychopharmacology 2017 – free download

"Where escitalopram nulled me to be less scared, psilocybin has made me realise that fear is not something to be feared"

Other learnings in our studies

Predictors of good outcomes	No or negative effect
Breakthrough / mystical type experiences **++**	Expectation for psilocybin to work (BUT does for escitalopram)
? Single trauma aetiology → longer remission	Anxiety during trip **- -**
Non-psychedelic (midi) doses eg 5-10mg psilocybin can have significant emotional releasing effects → OCD and impulsive cutting reframing studies	? Stopping SSRIs may reduce effect of psilocybin

Clinical considerations re other medications

5-HT2A receptor blocking drugs block effects of psilocybin

- **Antipsychotics e.g. olanzapine quetiapine risperidone**
- **Antidepressants e.g. amitriptyline mirtazapine trazodone**

- **SSRIs can blunt effects – ideally stop**
- **Lamotrigine also though little research**

Other medicines to be cautious with

- **Lithium – risk of seizures**
- **MAOIs – possible risk of serotonin syndrome [though note ayahuasca is DMT/MAOI combo**

What we don't yet know and need to research Part 1

1. **Optimal trip duration – DMT/5-MEO = mins –v- psilocybin/LSD = hrs**

2. **Why some patients have enduring benefits but in others the effects wear off**
   - **Can current relapse preventing drugs such as SSRIs or lithium delay depression returning?**
   - **Or top up doses?**
   - **Or microdosing?**

3. **How much therapeutic benefit does psychotherapy add**
   - **And if it does, what is the best form to use**
   - **Does blind-breaking and expectancy affect outcomes**

4. **Why some people on SSRI antidepressants and some other non-5-HT2A blocking drugs e.g. lamotrigine have blunted response - and the clinical implications of this**
   i.e. do we need to stop SSRIs?

What we don’t yet know and need to research Part 2

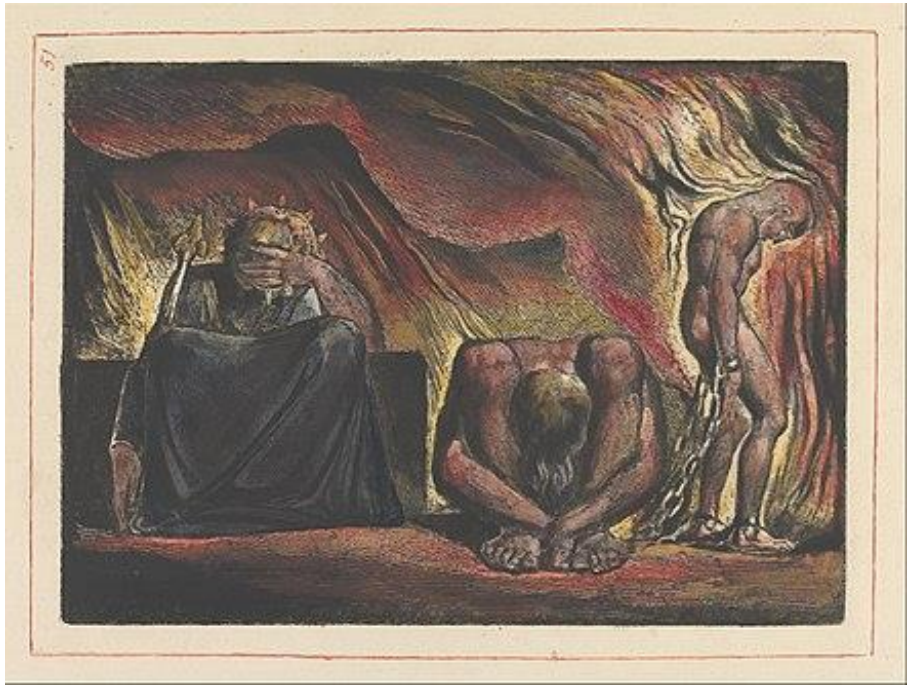
5. **Are the brain mechanisms of treatment effects in addiction or other disorders the same as in depression?**

6. **Whether non-psychedelic 5-HT2A agonists can be as effective antidepressants as psychedelic ones in humans: they can have antidepressant-like effects in rodent**
   - **And do they work in animal models of addiction ?**

7 . What are the parameters of the psychedelic plasticity window?
- **How long does it last?**
- **Is it different for different drugs?**
- **How can we best utilise this window therapeutically?**
- **How to measure it?**

Why psychedelics for addictions?

William Blake – Mind-forged manacles control our behaviour and thoughts
= deep canalization of mental content



Neuropharmacology 226 (2023) 109398

Contents lists available at ScienceDirect

Neuropharmacology

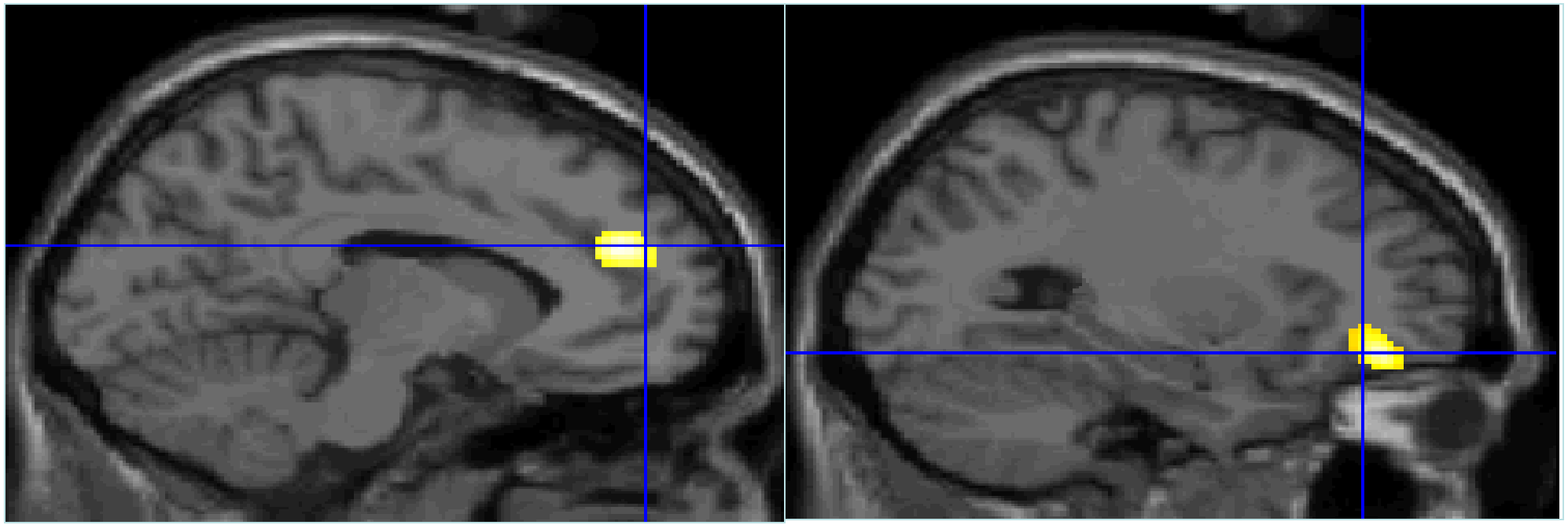
journal homepage: www.elsevier.com/locate/neuropharm

Canalization and plasticity in psychopathology

R.L. Carhart-Harris [a,b,*], S. Chandaria [b,c,d], D.E. Erritzoe [b,e], A. Gazzaley [a], M. Girn [f], H. Kettner [a,b], P.A.M. Mediano [g,h], D.J. Nutt [b], F.E. Rosas [b,c,i,j], L. Roseman [b,e], C. Timmermann [b,e], B. Weiss [b,e], R.J. Zeifman [b,k], K.J. Friston [l]

The Skofeld plate
in *Jerusalem: The Emanation of the Giant Albion*

Addictions share a common brain circuit

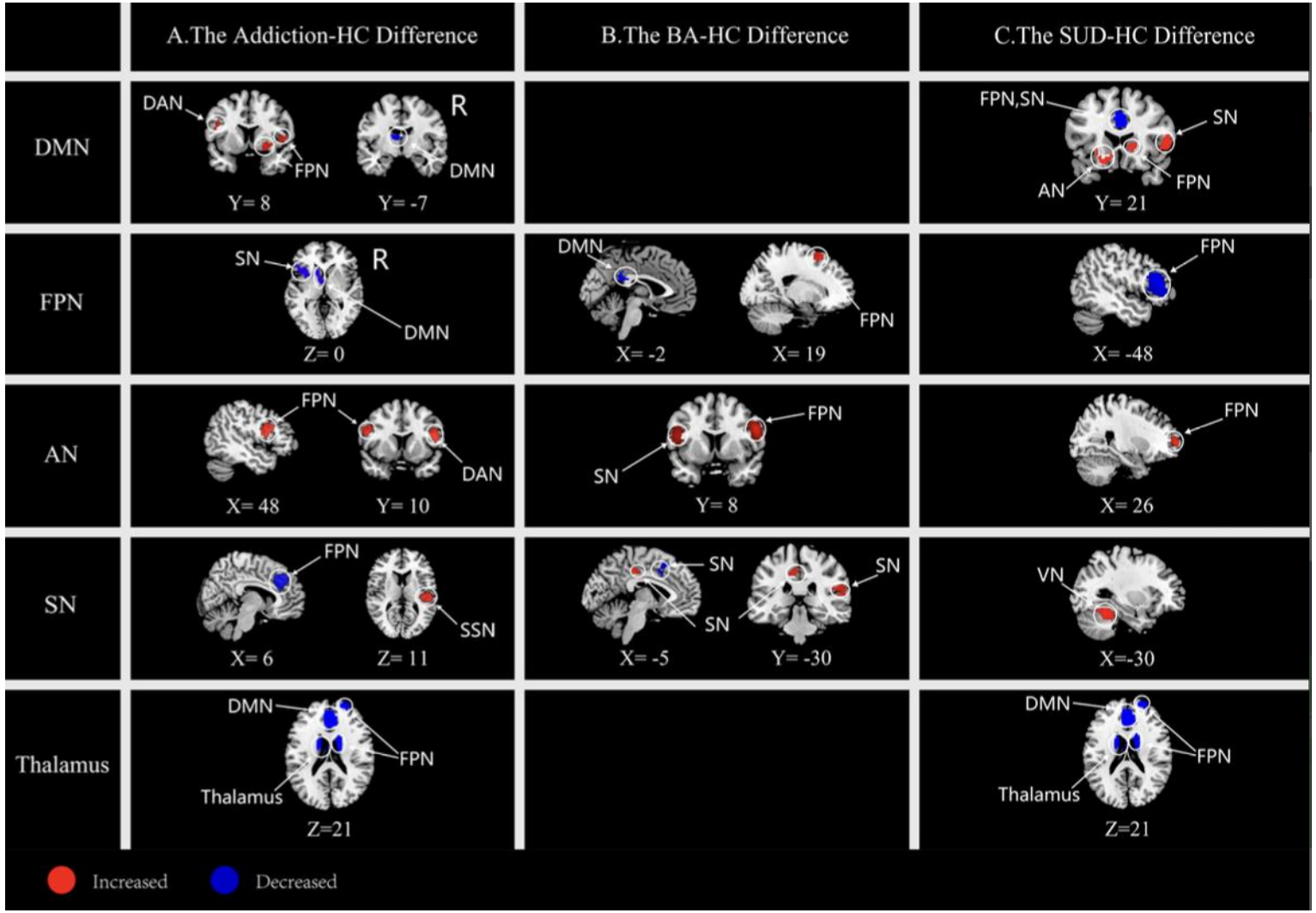


Remembering use **Urge to use**

Daglish et al 2001 Am J Psychiatry Heroin addiction

$^{15}O2$ water PET

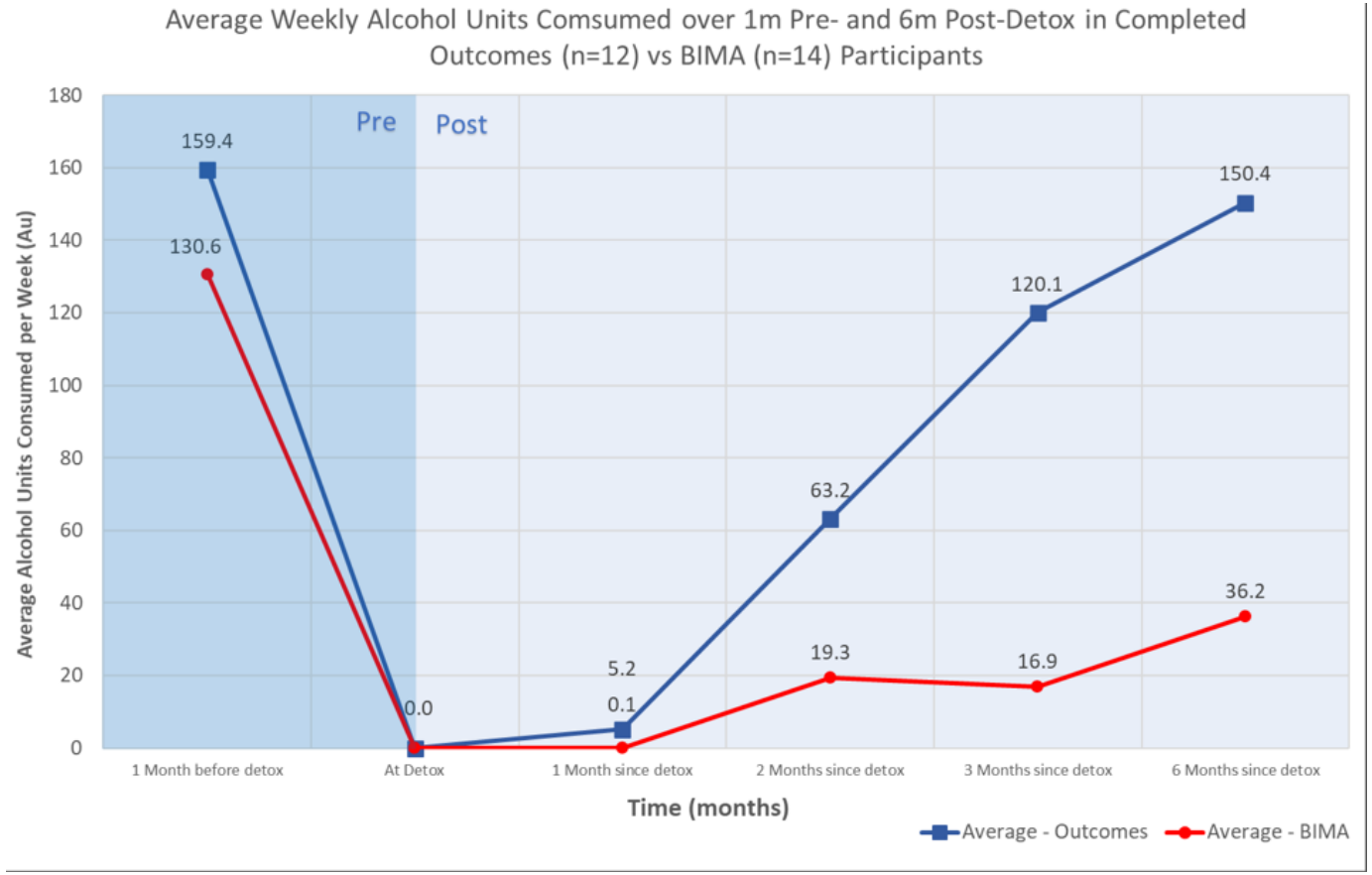
Network metanalysis in addiction



Zeng X, Han X, Zheng D, Jiang P, Yuan Z (2024). Psychological Medicine 54, 473–487. https://doi.org/10.1017/S0033291723003434

DMN – default mode network
FPN – fronto-parietal network
SN – salience network
AN – affective network

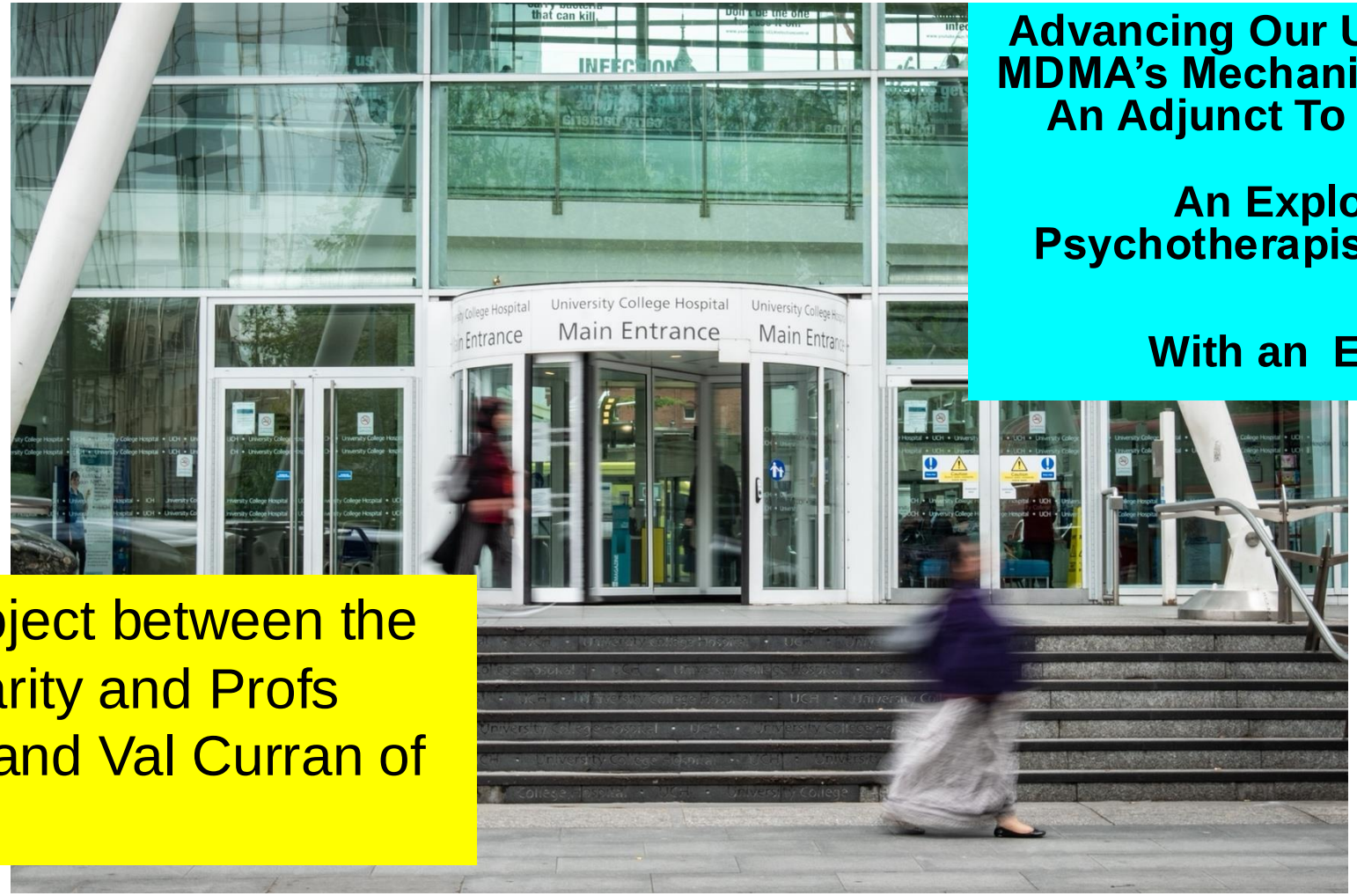
MDMA for trauma-related alcoholism - the BIMA study



2 sessions within a standard abstinence-based psychotherapy programme → highly significant decrease in alcohol use over next year

Sessa et al 2021 Journal of Psychopharmacology

New MDMA research project starting in UK



Advancing Our Understanding Of MDMA's Mechanisms Of Action As An Adjunct To Psychotherapy

An Exploration Of Psychotherapists' Experiences

With an Entactogen

New research project between the Drug Science charity and Profs Sunjeev Kamboj and Val Curran of UCL

https://www.drugscience.org.uk/uclmdma

Current theories of how psychedelics work therapeutically

➔ Disrupt the brain networks underpinning addiction

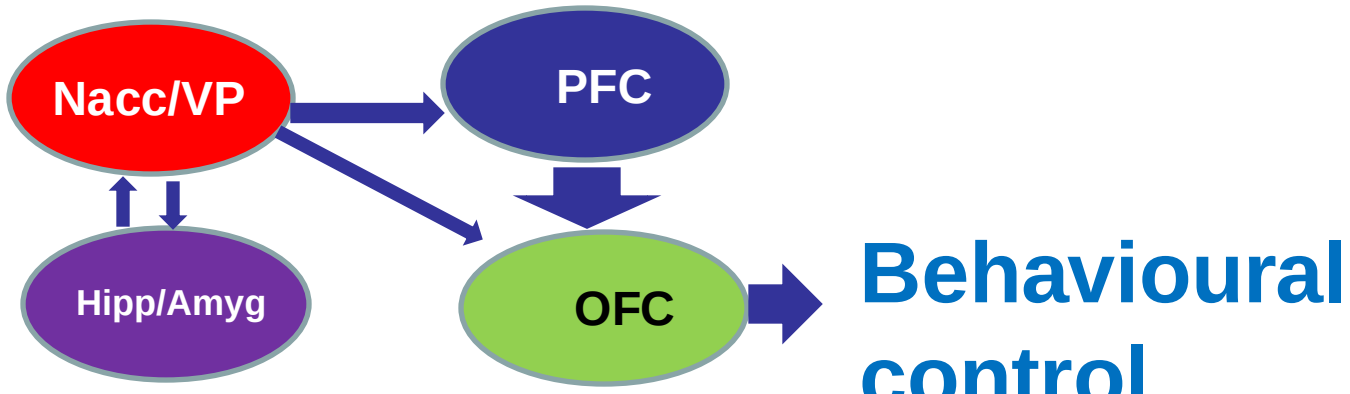
➔ Open critical windows for new learning

➔ Allow reframing/rewriting/extinction of addiction memories

Not mutually exclusive

Balanced brain state – before addiction

PFC controls final decision making



Behavioural control

Red = reward prediction and pleasure

Blue = cognitive control

Green = motivation drive and salience attribution

Purple = learning and memory

PFC – prefrontal cortex
OFC = orbitofrontal cortex
Nacc = nucleus accumbens
VP = ventral pallidum
Hipp = hippocampus
Amy = amygdala

Adapted from Baler and Volkow 2006

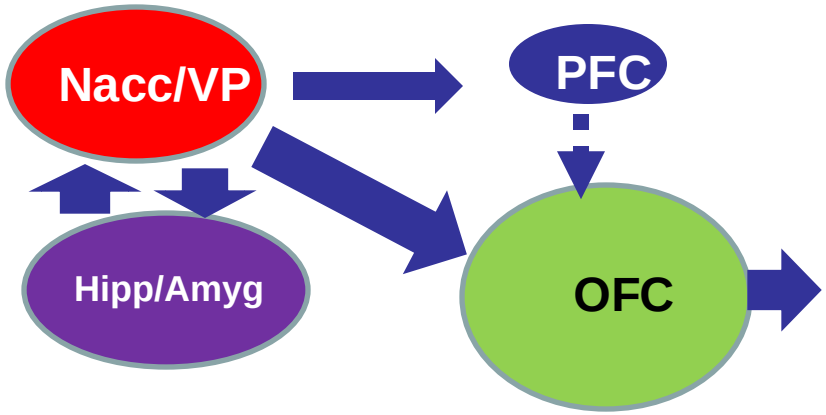
Addicted brain

Red = reward prediction and pleasure

Blue = cognitive control

Green = motivation drive and salience attribution

Purple = learning and memory



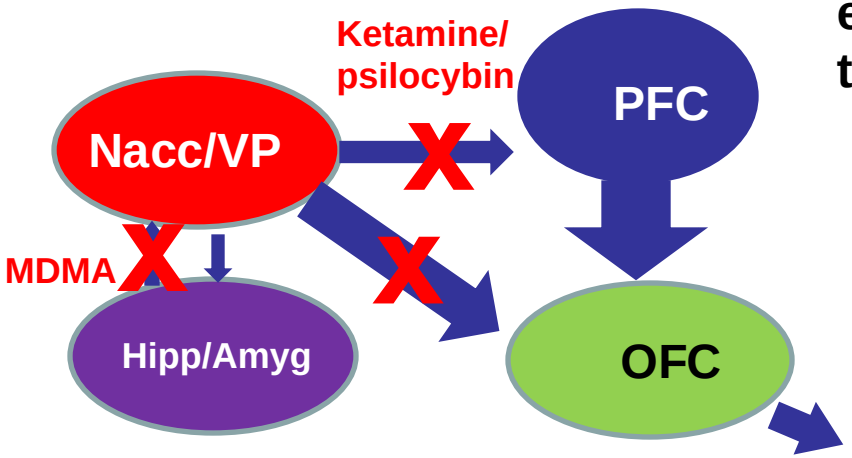
Go →drugs and other addictive behaviours

Excessive drives from memory and reward circuits depress PFC and enhance OFC functioning so PFC no longer controls behaviour

PFC – prefrontal cortex
OFC = orbitofrontal cortex
Nacc = nucleus accumbens
VP = ventral pallidum
Hipp = hippocampus
Amy = amygdala

Adapted from Baler and Volkow 2006

Disrupting these overactive circuits can restore balance in the brain



+ Psychotherapy
expands capacity of PFC to control behaviour

Control over use

PFC – prefrontal cortex
OFC = orbitofrontal cortex
Nacc = nucleus accumbens
VP = ventral pallidum
Hipp = hippocampus
Amy = amygdala

Red = reward prediction and pleasure

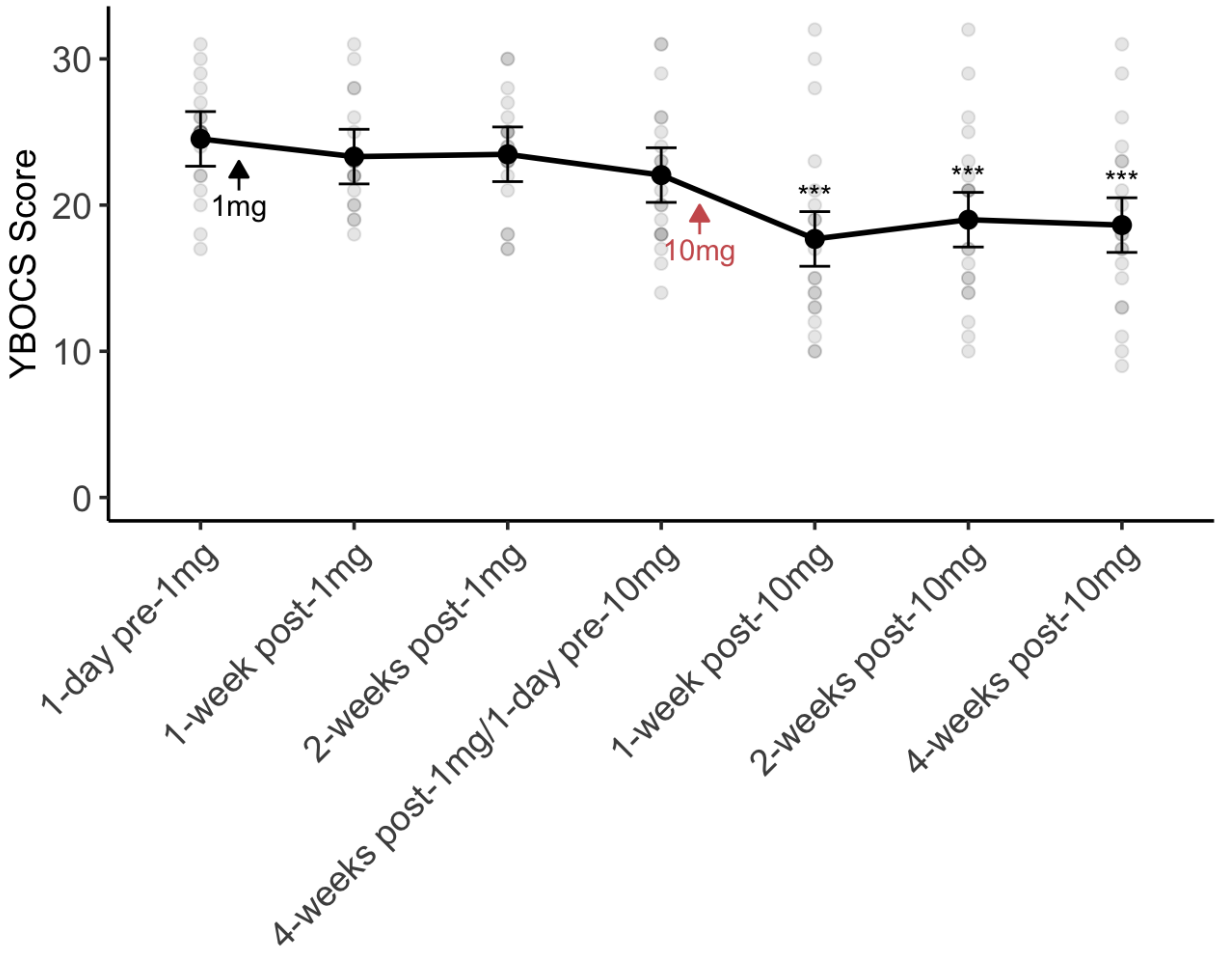
Blue = cognitive control

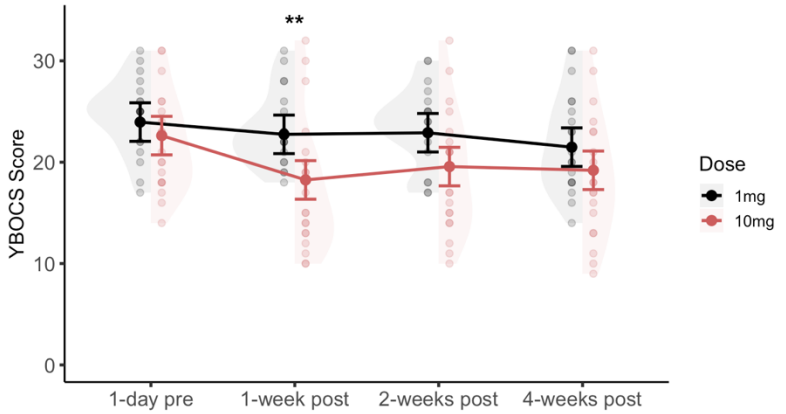
Green = motivation drive and salience attribution

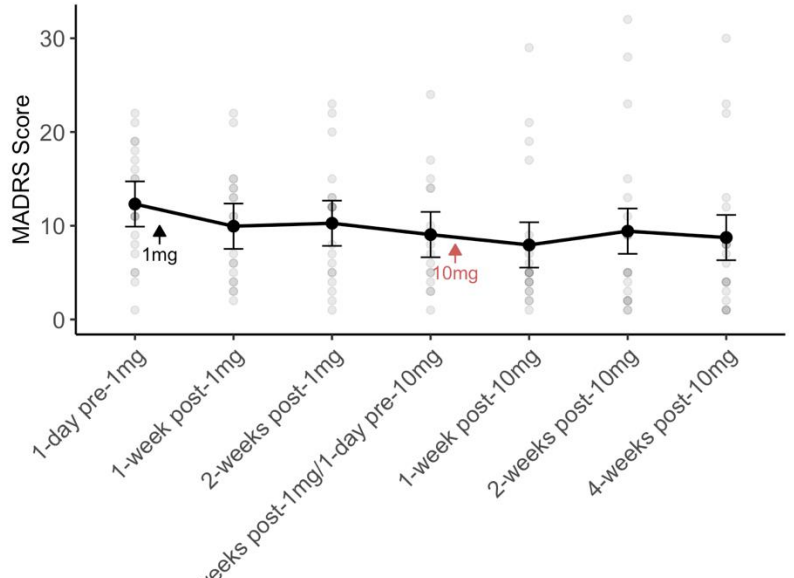
Purple = learning and memory

Adapted from Baler and Volkow 2006

OCD – psycholytic psilocybin treatment







1mg (placebo) v 10mg (psycholytic) doses of psilocybin

What we don't yet know and need to research Part 1

1. **Optimal trip duration – DMT/5-MEO = mins –v- psilocybin/LSD = hrs**

2. **Why some patients have enduring benefits but in others the effects wear off**
   - **Can current relapse preventing drugs such as SSRIs or lithium delay depression returning?**
   - **Or top up doses?**
   - **Or microdosing?**

3. **How much therapeutic benefit does psychotherapy add**
   - **And if it does, what is the best form to use**
   - **Does blind-breaking and expectancy affect outcomes**

4. **Why some people on SSRI antidepressants and some other non-5-HT2A blocking drugs e.g. lamotrigine have blunted response - and the clinical implications of this**
   i.e. do we need to stop SSRIs?

What we don’t yet know and need to research Part 2

5. **Are the brain mechanisms of treatment effects in addiction or other disorders the same as in depression?**

6. **Whether non-psychedelic 5-HT2A agonists can be as effective antidepressants as psychedelic ones in humans: they can have antidepressant-like effects in rodent**
   - **And do they work in animal models of addiction ?**

7 . What are the parameters of the psychedelic plasticity window?
- **How long does it last?**
- **Is it different for different drugs?**
- **How can we best utilise this window therapeutically?**
- **How to measure it?**

Acknowledgements, further reading and questions

THE CENTRE FOR PSYCHEDELIC RESEARCH
Imperial College London

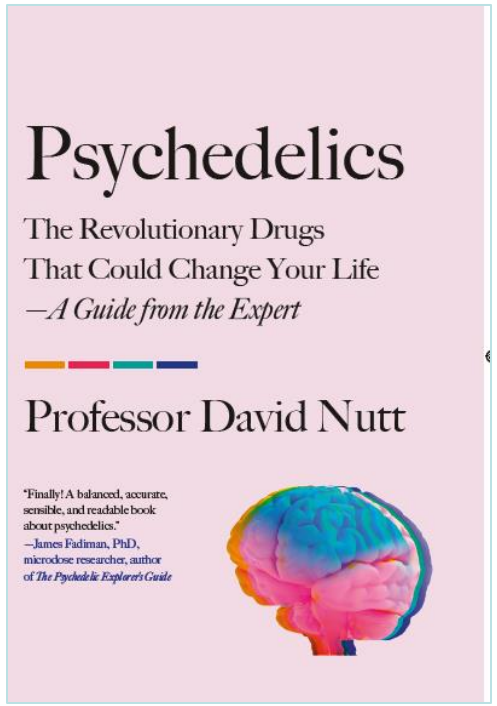
Alexander Mosley Charitable Trust

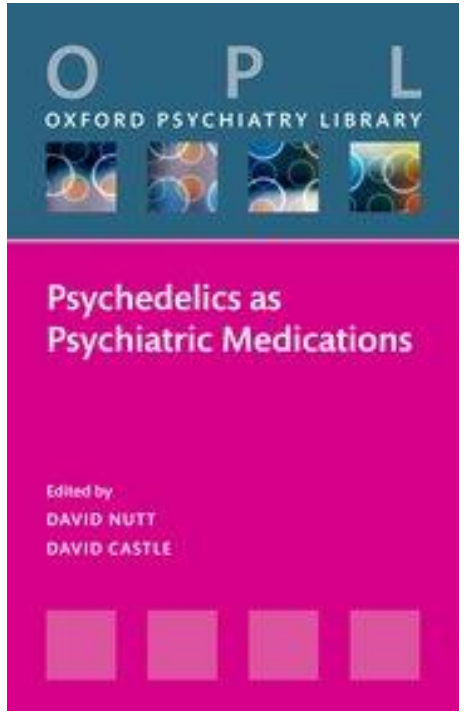
MRC Medical Research Council

NIHR UK addictions mission

ICCAM
Imperial College London
UNIVERSITY OF CAMBRIDGE
MANCHESTER 1824 The University of Manchester

BECKLEY FOUNDATION





Enhanced connectivity between amygdala and dopamine nuclei in alcohol use disorder → excess urge → loss of control?

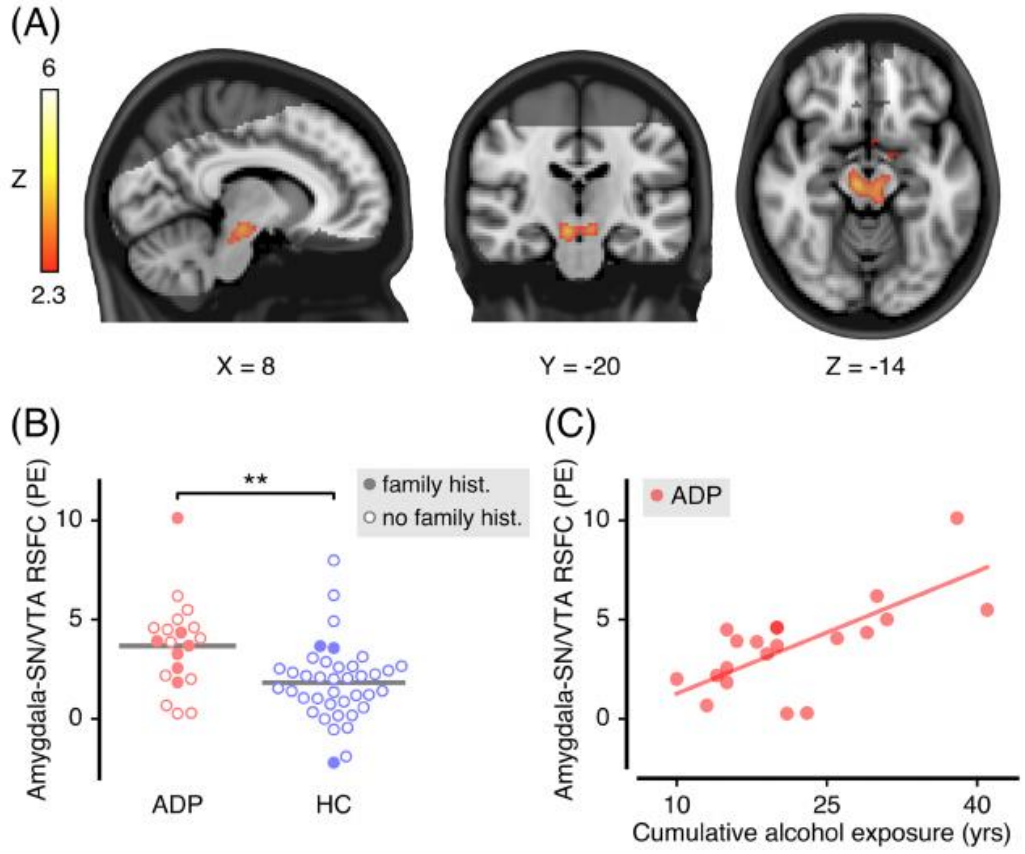
Received: 21 July 2019 | Revised: 31 July 2020 | Accepted: 5 October 2020
DOI: 10.1111/adb.12980

Addiction Biology WILEY

ORIGINAL ARTICLE

Chronic alcohol exposure differentially modulates structural and functional properties of amygdala: A cross-sectional study

Csaba Orban[1,2,3] | John McGonigle[1] | Remy S.A. Flechais[1] | Louise M. Paterson[1] | Rebecca Elliott[4] | David Erritzoe[1] | Karen D. Ersche[5,6] | Anna Murphy[4] | Liam J. Nestor[1,6] | Filippo Passetti[1,5,6] | Laurence J. Reed[1] | Andre S. Ribeiro[1] | Dana G. Smith[5,7] | John Suckling[5,6,8] | Eleanor M. Taylor[4] | Adam D. Waldman[9] | Victoria C. Wing[1] | J.F. William Deakin[4] | Trevor W. Robbins[5,7] | David J. Nutt[1] | Anne R. Lingford-Hughes[1] | ICCAM Platform*



ICCAM

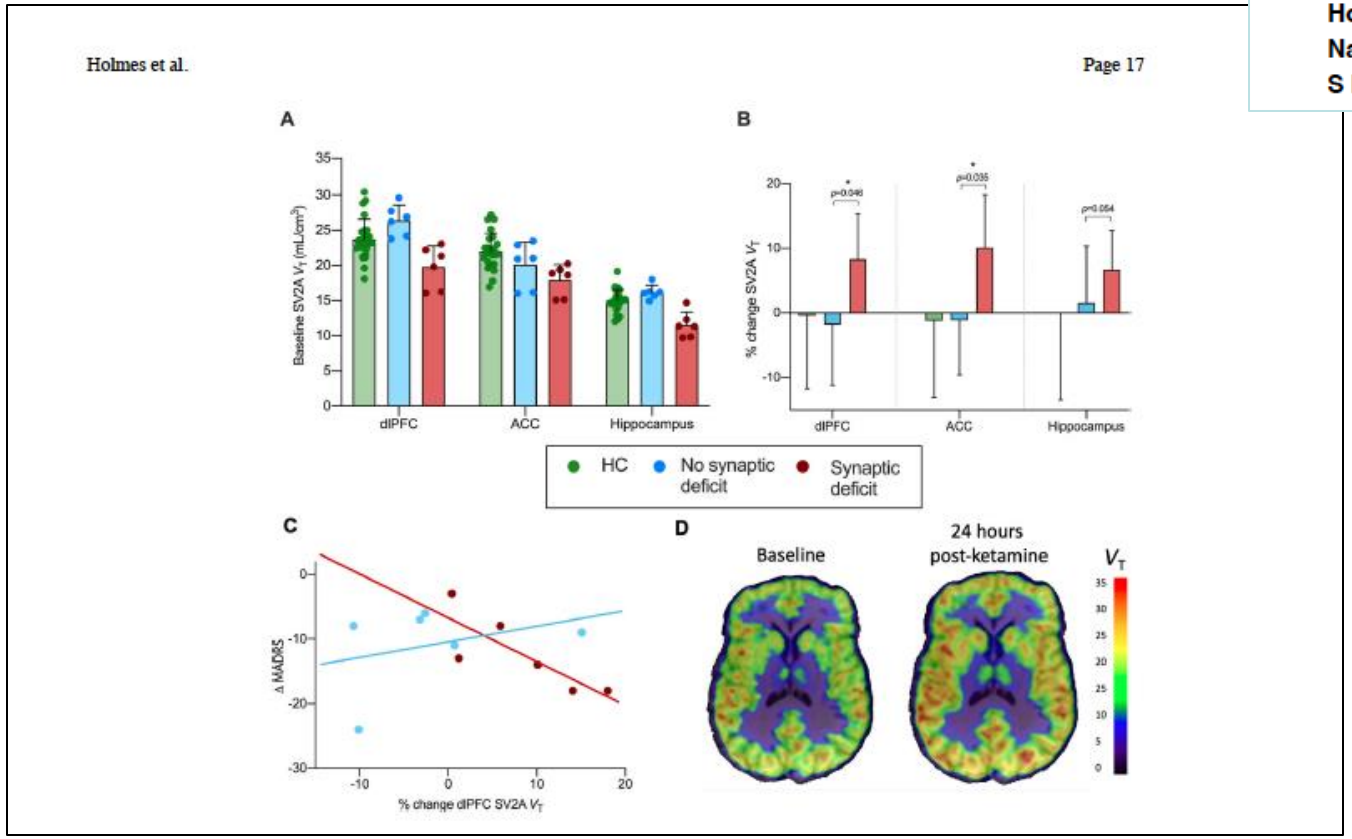
Imperial College London | UNIVERSITY OF CAMBRIDGE | MANCHESTER 1824 The University of Manchester

Ketamine neuroplasticity?
11C- UCB-J PET

Published in final edited form as:
Mol Psychiatry. 2022 April ; 27(4): 2273–2281. doi:10.1038/s41380-022-01465-2.

Imaging the effect of ketamine on synaptic density (SV2A) in the living brain

Sophie E Holmes[1,*], Sjoerd J Finnema[2], Mika Naganawa[2], Nicole DellaGioia[1], Daniel Holden[2], Krista Fowles[2], Margaret Davis[1], Jim Ropchan[2], Paul Emory[2], Yunpeng Ye[2], Nabeel Nabulsi[2], David Matuskey[1,2], Gustavo A Angarita[1], Robert H Pietrzak[1,3], Ronald S Duman[1], Gerard Sanacora[1], John H Krystal[1,3], Richard E Carson[2], Irina Esterlis[1,3,*]



Imperial College studies

We find no change following ketamine in healthy volunteers

DMT study underway