

Review Article

Psychedelics and neuroanesthesia: A narrative review of converging mechanisms

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Abstract

Current neuroscience research calls into question the binary model of anesthesia as a state of consciousness shifted by unconsciousness, replacing it with an idea of consciousness as a continuum created by interactions between large-scale cortex and subcortical regions. Meanwhile, psychedelic drugs are making a comeback as potential treatments for treatment-resistant depression, post-traumatic stress disorder, anxiety during life-threatening illness and substance use disorders. Classical serotonergic psychedelics (like psilocybin and lysergic acid diethylamide) have a primary target on 5-hydroxytryptamine 2A receptors, while the dissociative anesthetic ketamine inhibits N-methyl-D-aspartate receptors, and the weak gamma-aminobutyric acid-A receptor modulation is a component of the action of nitrous oxide. These agents interact differently with receptors, but have similar network-level effects on the human cortex, such as disruption of integrity of the default-mode network, loss of frontoparietal directed connectivity, increased between-network interactions, and characteristic EEG signatures that are strikingly similar to those seen during emergence from general anesthesia. The anesthesiology paradigm offers a very unique and well controlled platform for dissecting the relative contribution of drug pharmacology, expectancy, and the conscious subjective experience – something that has been recently shown by ketamine masked by surgical anesthesia trials. Here, the current narrative review aims to bring together the latest available evidence on the intersection of psychedelic pharmacology and clinical anesthesia, focusing on common molecular targets, neural-network signatures, EEG correlates, and the therapeutic relevance of anesthetic emergence in specific psychiatric patient populations. The implications for perioperative monitoring, perioperative sedation in vulnerable patients, and designing future placebo-controlled studies of psychedelics are discussed. These two literatures bring the neuroanesthesiologist into a crucial role in the future of clinical psychedelic science.

Keywords: Anesthesia, Consciousness, Electroencephalography, Hallucinogens, Ketamine, Neuroimaging, Nitrous oxide, Psilocybin.

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1. Introduction

For over 100 years the practitioners of anesthesia have assumed consciousness is dichotomic: awake or sleep, and the purpose of a general anesthetic was to make a patient reliably unconscious. Yet, in the last 20 years, this conceptual framework has undergone a major transformation. Modern neuroscience has shifted the definition of consciousness to a graded, multidimensional phenomenon, depending on the dynamic interplay of large-scale systems in the cortex and subcortical circuitry, and susceptible to the effects of pharmacological manipulation and contextual modulation; even seemingly unresponsive patients under GA maintain residual frontoparietal connectivity and covert cortical processing, and the depth of subjective unconsciousness is represented by multiple orthogonal dimensions—such as arousal, awareness, connectedness, and the capacity for integrated information processing.^{1,2}

Alongside this reimagining of consciousness has been the revival of clinical studies of psychedelics. Classical psychedelics, dissociative and entactogens have been shown to have several psychiatric indications and are now being explored in a modern fashion under scientific rigor after decades of regulatory silence since their ban in the late 1960s, such as treatment-resistant major depression, post-traumatic stress disorder, anxiety in life-threatening illness, and substance use disorders. The major modern study with healthy volunteers that showed that one dose of psilocybin could lead to mystical-type experiences of significant personal meaning that lasted for at least fourteen months opened the door for the current series of phase 2 and phase 3 studies using classical serotonergic psychedelics in psychiatric patients.³

The agents of interest can be grouped into three general categories, all of which share common neurobiological endpoints, in terms of pharmacology. Partial agonists at the 5-HT_{2A} receptor, such as psilocybin and lysergic acid diethylamide, and dissociative anesthetics (prototype ketamine) antagonize the N-methyl-D-aspartate (NMDA) receptor, particularly on gamma-aminobutyric acidergic interneurons of the cortex, resulting in a paradoxical disinhibition of the glutamatergic output of pyramidal cells, acute glutamate surge, and rapid downstream signaling through the mammalian target of rapamycin and brain-derived neurotrophic factor pathways.^{4,5}

The association of anesthesia and altered states of consciousness is actually much older than is often thought. Inhaled nitrous oxide was used as a psychedelic drug by Sir Humphry Davy in the late 18th century, and the dissociative effects of ketamine were described from the earliest use as an anesthetic on the battlefield in the 1960s. In each of these pharmacological groups, the same compound produces subjective psychedelic effects as well as surgical anesthesia, with the major factors being the dosage, route of administration, and the pharmacokinetic profile. This pharmacological continuum is the conceptual link that is essential to the literatures of clinical psychedelic therapy and neuroanaesthesia.⁶

The next level of conceptual convergence lies in the large-scale brain networks. The two literatures thus explore complementary states of the same underlying network architecture, with disruption of the DMS in the volunteers receiving psilocybin or lysergic acid diethylamide contrasting broadly with the anteriorization of alpha oscillations in frontal cortex, slow-wave coherence and disruption of thalamocortical

alpha dynamics associated with loss of consciousness during propofol-induced anaesthesia.⁷

The therapeutic benefits that have been historically observed with the use of psychedelic substances are notoriously hard to distinguish from the expectancy, set and setting that is associated with administration. Given the recent randomized controlled trial (RCT) masking the drug with surgical anesthesia in patients suffering from major depressive disorder (MDD), results of which has been one of the most rigorous attempts to date to dissociate drug pharmacology and the subjective experience of the psychedelic trip, the results has substantially narrowed the field understanding just how much of the observed antidepressant signal in conventional psychedelic trials is due to expectancy.⁸

The aim of this narrative review was to draw together the existing neurobiological and electrophysiological evidence linking psychedelics to neuroanesthesia, with a particular focus on shared molecular targets, common large-scale network signatures, the electroencephalographic correlates, and the growing clinical evidence base, and to discuss the methodological, ethical, and translational implications for routine perioperative practice and future clinical trial design.

2. Review of the Literature

2.1. Molecular and receptor-level convergence

The three main classes of agents discussed in this chapter overlap in downstream signaling pathways, but have distinct primary targets at the molecular level.^{9,10} It is noteworthy that only serotonergic agonists which are known to engage specific intracellular signaling pathways coupled to the 5-HT_{2A} receptor, such as phospholipase A₂ and arachidonic acid pathways, generate hallucinogenic behavioral phenotypes, while non-hallucinogenic 5-HT_{2A} agonists, which do not engage this biased signaling, do not.¹¹ This biased agonism is an important concept as it opens the door to a potentially simplified model of future 5-HT_{2A} agonists with only the neuroplastic and antidepressant effects of the classical psychedelics and without the potential for the long-term subjective effect. Both pathways converge on activity-dependent synaptic plasticity, defined by dendritic spine growth in the prefrontal cortex, activation of the mammalian target of rapamycin (mTOR) and increased activity of brain-derived neurotrophic factor (BDNF) signaling, and these effects are induced by drugs with widely different chemical structures, which explains why they produce similar rapid-onset, lasting antidepressant effects in different patient populations.¹² Another is dampening down the production of pro-inflammatory

cytokines and microglial activation – both effects are known for ketamine and psilocybin, and are associated with the pathophysiology of treatment-resistant depression. Recent studies also indicate that metabolites of ketamine, including (2R,6R)-hydroxynorketamine, may be responsible for its own antidepressant effect via AMPA rather than NMDA receptor antagonism, thus expanding the therapeutic window far beyond.¹³ Pharmacological diversity of these compounds, however, gives rise to a remarkable similarity at the molecular and cellular level, leaving a solid ground for integrated research of psychedelic and anesthetic pharmacology in common experimental paradigms.¹⁴

Table 1 summarizes the principal receptor targets, downstream signaling pathways, and clinical indications of the major psychedelic and anesthetic agents relevant to this review.

2.2. Network-level and electrophysiological signatures

Classical psychedelics are known to cause a stereotyped acute reorganization of the functional architecture of the cortex in functional neuroimaging studies. Decreased regional cerebral blood flow and decreased blood-oxygen-level-dependent signal in critical regions of the default-mode network (DMN) were observed in the earliest task-free functional magnetic resonance imaging (fMRI) studies with intravenous psilocybin, and were accompanied by a decrease in within-network coherence and an increase in between-network connectivity in this

brain network.¹⁵ The same regions of the midline of the brain are characterized by hyperconnectivity in major depressive disorder, suggesting a direct mechanistic explanation for the antidepressant effect of psilocybin-induced breakdown of the default-mode network. The size of the default-mode disintegration also predicts the subjective experience of ego-dissolution as reported by participants, indicating that network-level properties are predictive of phenomenologically meaningful changes in conscious experience, not just an epiphenomenon of receptor occupancy.¹⁶ A complementary temporally precise window into the same phenomena is provided by EEG studies. In all three drugs, recordings demonstrate broadband cortical desynchronization, low-frequency oscillatory power reduction (especially in the alpha band), and increase of measures of neural signal diversity and complexity (Lempel-Ziv complexity, perturbational complexity index), which are seen as electrophysiological correlates of an enriched repertoire of conscious states, in contrast to the impoverished and stereotyped repertoire characteristic of pharmacological unconsciousness. Of particular interest to the anesthesiologist, the enhancement of signal diversity is reliably abolished during deep general anesthesia and sharply diminished during burst-suppression, suggesting that the same electrophysiological measure applies to both ends of the spectrum of psychedelics and anesthetics, and could indeed provide a common measure for future translational studies.¹⁷

Table 1: Pharmacological and clinical comparison of major psychedelic and anesthetic agents

Agent	Primary receptor target	Downstream pathway	Anesthetic role	Psychiatric indication
Psilocybin / psilocin	5-HT2A partial agonist	Glutamate release; BDNF/mTOR; spine growth	None	TRD; existential anxiety
LSD	5-HT2A partial agonist (broader serotonergic)	DMN disintegration; entropic cortical activity	None	Depression; anxiety
Ketamine	NMDA receptor antagonist	GABA-ergic disinhibition; glutamate surge; mTOR/BDNF	Dissociative anesthetic; analgesic	TRD; acute suicidality
Nitrous oxide	NMDA antagonist; weak GABA-A	Glutamate modulation; possible opioid component	Inhalational adjunct; sedation	TRD (off-label)
Propofol	GABA-A receptor positive modulator	Alpha anteriorization; thalamocortical disruption	Intravenous general anesthetic	Investigational antidepressant
MDMA	Serotonin / norepinephrine releaser	Prosocial signaling; oxytocin; reduced amygdala reactivity	None	PTSD

5-HT2A, 5-hydroxytryptamine 2A; BDNF, brain-derived neurotrophic factor; DMN, default-mode network; GABA, gamma-aminobutyric acid; LSD, lysergic acid diethylamide; MDMA, 3,4-methylenedioxymethamphetamine; mTOR, mammalian target of rapamycin; NMDA, N-methyl-D-aspartate; PTSD, post-traumatic stress disorder; TRD, treatment-resistant depression.

In many ways, these signatures are the opposite of what is generated in general anesthesia. The loss of posterior alpha rhythms, the anteriorization of alpha and beta rhythms, and emergence of large amplitude low-frequency power are stereotypic signatures of induction of and emergence from propofol-induced unconsciousness with high temporal resolution, which can also be used at the bedside to estimate proximity to the recovery of consciousness with a good amount of reliability.¹⁸ During the induction and emergence from general anesthesia, however, the brain passes through intermediate states of activity where the diversity of signals, frontoparietal connectivity, and large-scale integration of networks can briefly resemble states observed in psychedelic experience, affording a momentary resemblance between the two states.¹⁸ This convergence has paved the way for systematic research on intraoperative dreaming and slow-emergence techniques in psychiatric populations, where the anesthetic experience and the psychedelic experience is most clinically relevant, and in which case-control studies point to the potential for long-term therapeutic benefit in certain patients with pre-existing trauma-related disorders.¹⁹

Table 2 summarizes representative neuroimaging and electrophysiological findings across the major psychedelic and anesthetic states relevant to this convergence.

3. Discussion

These findings with nitrous oxide are especially significant because they came from the anesthetic community, with standard inhalational delivery, and because a lower concentration of nitrous oxide, 25%, was as effective as the higher concentration of 50% with much greater tolerability, showing that a routine perioperative agent can be effective as an antidepressant without the use of scheduled or experimental agents.²⁰⁻²² While not diminishing the antidepressant effect of ketamine, it does highlight that belief in being administered ketamine, and the therapeutic context were sufficient to explain a significant portion of the observed antidepressant response in conventional unblinded trials of psychedelic drugs.⁹ These trials provide a good overall picture of both the potential for these psychedelic and anesthetic drugs as fast-acting antidepressants and the methodological hurdle of functional unmasking and expectancy effects in placebo-controlled experiments, a hurdle that will need anesthesiology informed methodological innovation to overcome.²³

The review of the mechanistic literature, as well as the review of the clinical literature above shows a consistent story that has come together in nearly 30 years. In the late 1990s, the earliest electrophysiological evidence that hallucinogens specifically target the apical dendrites of pyramidal cells in the brain, recorded in slices of the rodent prefrontal cortex, appeared, followed by demonstrations that activation of 5-HT_{2A} by hallucinogens specifically suppresses low-frequency oscillations in the cortex in vivo, and that this effect is reversible by antipsychotic drugs.²⁴

Table 2: Representative neuroimaging and electrophysiological signatures of psychedelic and anesthetic states

State	fMRI / network features	EEG features	Behavioral correlate
Psilocybin	DMN integrity; between-network connectivity	Broadband desynchronization; alpha power	Mystical experience; ego dissolution
LSD	Increased global connectivity; entropic dynamics	Alpha power; signal diversity	Altered perception of time and self
Subanesthetic ketamine	Global functional connectivity; salience network changes	Alpha; gamma; Lempel-Ziv complexity	Dissociation; rapid antidepressant effect
Nitrous oxide (subanesthetic)	Reduced functional differentiation in frontoparietal cortex	Frontal delta; transient theta on washout	Analgesia; antidepressant effect
Propofol GA	Disrupted frontoparietal connectivity; covert islands of activity	Frontal alpha anteriorization; slow waves; burst suppression	Unresponsiveness; rare intraoperative dreams
Emergence	Transient re-integration of large-scale networks	Loss of frontal alpha; complexity	Variable: confusion, dreams, psychedelic-like reports

DMN, default-mode network; EEG, electroencephalography; fMRI, functional magnetic resonance imaging; GA, general anesthesia; LSD, lysergic acid diethylamide.

At the same time, two-photon microscopy in rodents revealed that a single dose of psilocybin causes rapid and lasting increases in dendritic spine density in the frontal cortex, an effect that persists for at least one month, suggesting a possible candidate for the cellular basis of the long-lasting clinical effects seen in human trials.^{24,25}

In the same year, human studies showed that a frequently used anesthetic, nitrous oxide, also had effects on functional differentiation in frontoparietal and somatomotor cortical networks, which were remarkably similar to that of classical psychedelics, lending additional support to a unified cortical signature for the psychedelic class.²⁶

This stream of research provides a compelling argument for a common circuit model that links classical psychedelics, dissociative and some anesthetic agents and for their combined study within an anesthesiology-guided framework.

There are a few caveats to temper enthusiasm. Most of the network-level and electrophysiological data come from small sample sizes, heterogeneous protocols and specialized research centers, and direct extrapolation from mechanistic studies in rodents to human cortical dynamics has to be done with great care.²⁷ The clinical trials analyzed are almost exclusively carried out in targeted, often very homogenous patient groups, and the role of expectancy effects seen in the anesthesia-masked ketamine paradigm has yet to be sufficiently addressed in psilocybin or MDMA clinical trials. There are also no standardized outcome measures for psychedelic anesthetic interaction, no objective real-time biomarkers of subjective psychedelic state and no consensus protocols for slow-emergence techniques. The solution to these restrictions will depend on the continued use of multicentric cooperation and collaboration of anesthesiologists, psychiatrists, neuroscientists, and methodologists, along with the creation of prospective registries that would collect dream content, emergence phenomena, and post-dream mental-health outcomes. The neuroanesthesiologist has the unique opportunity to take the lead in this field, given his or her routine use of the controlled altered state of consciousness, high level infrastructure for neuromonitoring, and the long experience of safe pharmacological manipulation of the human brain.^{28,29}

4. Conclusion

Classical psychedelics, dissociative anesthetics, and inhalational agents such as nitrous oxide exhibit convergent network-level and electrophysiological

signatures despite divergent receptor pharmacology. The anesthesiology paradigm provides a uniquely controlled environment in which to dissect drug pharmacology from expectancy and subjective experience. Emerging clinical evidence supports rapid antidepressant and anti-PTSD effects across this pharmacological continuum, with intraoperative dreaming representing a novel therapeutic target. Neuroanesthesiologists are uniquely positioned to lead the next phase of clinical translation. Future research should integrate multimodal neuromonitoring with carefully designed perioperative protocols to harness this convergence safely and ethically.

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Kunal Kumar Sharma: Conceptualization, literature search, data interpretation, manuscript drafting, critical revision of the manuscript, and final approval of the version to be published.

Ethical Approval

Ethical approval was not required for this review article.

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Conflict of Interest

None.

Use of Artificial Intelligence (AI)

The authors declare that no artificial intelligence (AI) tools were used in the generation, analysis, interpretation, or writing of this manuscript.

References

1. Mashour GA, Hudetz AG. Neural correlates of unconsciousness in large-scale brain networks. *Trends Neurosci.* 2018;41(3):150–60. <https://doi.org/10.1016/j.tins.2018.01.003>
2. Huang Z, Vlisides PE, Tarnal VC, Janke EL, Keefe KM, Collins MM, et al. Brain imaging reveals covert consciousness during behavioral unresponsiveness induced by propofol. *Sci Rep.* 2018;8(1):13195. <https://doi.org/10.1038/s41598-018-31436-z>
3. Griffiths RR, Richards WA, McCann U, Jesse R. Psilocybin can occasion mystical-type experiences having substantial and sustained personal meaning and spiritual significance. *Psychopharmacology (Berl).* 2006;187(3):268–83. <https://doi.org/10.1007/s00213-006-0457-5>

4. Shao LX, Liao C, Davoudian PA, Savalia NK, Jiang Q, Wojtasiewicz C, et al. Pyramidal cell types and 5-HT_{2A} receptors are essential for psilocybin's lasting drug action. *Nature*. 2025;642(8067):411–20. <https://doi.org/10.1038/s41586-025-08813-6>
5. Zanos P, Gould TD. Mechanisms of ketamine action as an antidepressant. *Mol Psychiatry*. 2018;23(4):801–11. <https://doi.org/10.1038/mp.2017.255>
6. Nagele P, Duma A, Kopec M, Gebara MA, Parsoei A, Walker M, et al. Nitrous oxide for treatment-resistant major depression: a proof-of-concept trial. *Biol Psychiatry*. 2015;78(1):10–8. <https://doi.org/10.1016/j.biopsych.2014.11.016>
7. Carhart-Harris RL, Muthukumaraswamy S, Roseman L, Kaelen M, Droog W, Murphy K, et al. Neural correlates of the LSD experience revealed by multimodal neuroimaging. *Proc Natl Acad Sci U S A*. 2016;113(17):4853–8. <https://doi.org/10.1073/pnas.1518377113>
8. Lii TR, Smith AE, Flohr JR, Okada RL, Nyongesa CA, Cianfichi LJ, et al. Randomized trial of ketamine masked by surgical anesthesia in patients with depression. *Nat Ment Health*. 2023;1(11):876–86. <https://doi.org/10.1038/s44220-023-00140-x>
9. Yip HYK, Papa A. Signaling Pathways in Cancer: Therapeutic Targets, Combinatorial Treatments, and New Developments. *Cells*. 2021;10(3):659. <https://doi.org/10.3390/cells10030659>
10. Denomme N, Heifets BD. Ketamine, the first associative anesthetic? Some considerations on classifying psychedelics, entactogens, and dissociatives. *Am J Psychiatry*. 2024;181(9):784–6. <https://doi.org/10.1176/appi.ajp.20240552>
11. González-Maeso J, Weisstaub NV, Zhou M, Chan P, Ivic L, Ang R, et al. Hallucinogens recruit specific cortical 5-HT_{2A} receptor-mediated signaling pathways to affect behavior. *Neuron*. 2007;53(3):439–52. <https://doi.org/10.1016/j.neuron.2007.01.008>
12. Homayoun H, Moghaddam B. NMDA receptor hypofunction produces opposite effects on prefrontal cortex interneurons and pyramidal neurons. *J Neurosci*. 2007;27(43):11496–500. <https://doi.org/10.1523/JNEUROSCI.2213-07.2007>
13. Ly C, Greb AC, Cameron LP, Wong JM, Barragan EV, Wilson PC, et al. Psychedelics promote structural and functional neural plasticity. *Cell Rep*. 2018;23(11):3170–82. <https://doi.org/10.1016/j.celrep.2018.05.022>
14. Carhart-Harris RL, Erritzoe D, Williams T, Stone JM, Reed LJ, Colasanti A, et al. Neural correlates of the psychedelic state as determined by fMRI studies with psilocybin. *Proc Natl Acad Sci U S A*. 2012;109(6):2138–43. <https://doi.org/10.1073/pnas.1119598109>
15. Dai R, Larkin TE, Huang Z, Tarnal V, Picton P, Vlisides PE, et al. Classical and non-classical psychedelic drugs induce common network changes in human cortex. *Neuroimage*. 2023;273:120097. <https://doi.org/10.1016/j.neuroimage.2023.120097>
16. Schartner MM, Carhart-Harris RL, Barrett AB, Seth AK, Muthukumaraswamy SD. Increased spontaneous MEG signal diversity for psychoactive doses of ketamine, LSD and psilocybin. *Sci Rep*. 2017;7:46421. <https://doi.org/10.1038/srep46421>
17. Purdon PL, Sampson A, Pavone KJ, Brown EN. Clinical electroencephalography for anesthesiologists: part I — background and basic signatures. *Anesthesiology*. 2015;123(4):937–60. <https://doi.org/10.1097/ALN.0000000000000841>
18. Ching S, Cimenser A, Purdon PL, Brown EN, Kopell NJ. Thalamocortical model for a propofol-induced alpha-rhythm associated with loss of consciousness. *Proc Natl Acad Sci U S A*. 2010;107(52):22665–70. <https://doi.org/10.1073/pnas.1017069108>
19. Chow HS, Hack LM, Kawai M, Heifets BD. Anesthetic-induced intraoperative dream associated with remission of a psychiatric disorder: a case report. *AA Pract*. 2022;16(8):e01613. <https://doi.org/10.1213/XAA.0000000000001613>
20. Goodwin GM, Aaronson ST, Alvarez O, Arden PC, Baker A, Bennett JC, et al. Single-dose psilocybin for a treatment-resistant episode of major depression. *N Engl J Med*. 2022;387(18):1637–48. <https://doi.org/10.1056/NEJMoa2206443>
21. Mitchell JM, Bogenschutz M, Lilienstein A, Harrison C, Kleiman S, Parker-Guilbert K, et al. MDMA-assisted therapy for severe PTSD: a randomized, double-blind, placebo-controlled phase 3 study. *Nat Med*. 2021;27(6):1025–33. <https://doi.org/10.1038/s41591-021-01336-3>
22. Nagele P, Palanca BJ, Gott B, Brown F, Barnes L, Nguyen T, et al. A phase 2 trial of inhaled nitrous oxide for treatment-resistant major depression. *Sci Transl Med*. 2021;13(597):eabe1376. <https://doi.org/10.1126/scitranslmed.abe1376>
23. Aghajanian GK, Marek GJ. Serotonin induces excitatory postsynaptic potentials in apical dendrites of neocortical pyramidal cells. *Neuropharmacology*. 1997;36(4–5):589–99. [https://doi.org/10.1016/s0028-3908\(97\)00051-8](https://doi.org/10.1016/s0028-3908(97)00051-8)
24. Celada P, Puig MV, Díaz-Mataix L, Artigas F. The hallucinogen DOI reduces low-frequency oscillations in rat prefrontal cortex: reversal by antipsychotic drugs. *Biol Psychiatry*. 2008;64(5):392–400. <https://doi.org/10.1016/j.biopsych.2008.03.013>
25. Carhart-Harris RL, Leech R, Hellyer PJ, Shanahan M, Feilding A, Tagliazucchi E, et al. The entropic brain: a theory of conscious states informed by neuroimaging research with psychedelic drugs. *Front Hum Neurosci*. 2014;8:20. <https://doi.org/10.3389/fnhum.2014.00020>
26. Tagliazucchi E, Roseman L, Kaelen M, Orban C, Muthukumaraswamy SD, Murphy K, et al. Increased global functional connectivity correlates with LSD-induced ego dissolution. *Curr Biol*. 2016;26(8):1043–50. <https://doi.org/10.1016/j.cub.2016.02.010>
27. Shao LX, Liao C, Gregg I, Davoudian PA, Savalia NK, Delagarza K, et al. Psilocybin induces rapid and persistent growth of dendritic spines in frontal cortex in vivo. *Neuron*. 2021;109(16):2535–44.e4. <https://doi.org/10.1016/j.neuron.2021.06.008>
28. Vejmla Č, Tyliš F, Piorecká V, Koudelka V, Kadeřábek L, Novák T, et al. Psilocin, LSD, mescaline, and DOB all induce broadband desynchronization of EEG and disconnection in rats with robust translational validity. *Transl Psychiatry*. 2021;11(1):506. <https://doi.org/10.1038/s41398-021-01624-z>

29. Dai R, Huang Z, Larkin TE, Tarnal V, Picton P, Vlisides PE, et al. Psychedelic concentrations of nitrous oxide reduce functional differentiation in frontoparietal and somatomotor cortical networks. *Commun Biol.* 2023;6(1):1284. <https://doi.org/10.1038/s42003-023-05619-y>
30. Hack LM, Sikka P, Zhou K, Kawai M, Chow HS, Heifets B. Reduction in trauma-related symptoms after anesthetic-induced intra-operative dreaming. *Am J Psychiatry.* 2024;181(6):563–4. <https://doi.org/10.1176/appi.ajp.20230908>