

Perspective

Can Psychedelics help with Alcohol Use Disorder (AUD): For now, the data says no

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Abstract

Aim: The aim of this perspective article is to evaluate the potential of Psychedelics in the management of Alcohol Use Disorder (AUD) vis-a-vis pre-existing therapeutic modalities and to elucidate practical directions for policy in AUD Addiction medicine, supported by scientific evidence, while prioritizing safety and accessibility.

Methods: We performed a narrative synthesis of past and current literature and incorporated findings from randomized controlled trials (RCTs), meta-analyses, and policy reports in relation to pharmacological and psychedelic-assisted treatments for AUD. PubMed was utilized for this search.

Results: While there is budding interest in psychedelics- LSD, MDMA, Psilocybin- existing evidence supporting their efficacy in treating AUD is sparse and in some studies require more evidence in terms of study types (placebo controlled blind) and methods, with high expectancy bias and limited follow-up periods. On the contrary, current FDA-approved medications such as Naltrexone, Acamprosate, and Disulfiram are aimed at minimizing relapse, moderating craving, and promoting continued abstinence. However, the clinical application of these drugs is alarmingly low at 2%. Policy measures such as telehealth-based prescribing, long-acting

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formulations, and potential over-the-counter access to naltrexone may improve treatment accessibility and reduce stigma. Psychedelics may be considered during the management of treatment-resistant AUD, but should be limited to methodologically sound clinical trials until clear safety and efficacy are demonstrated.

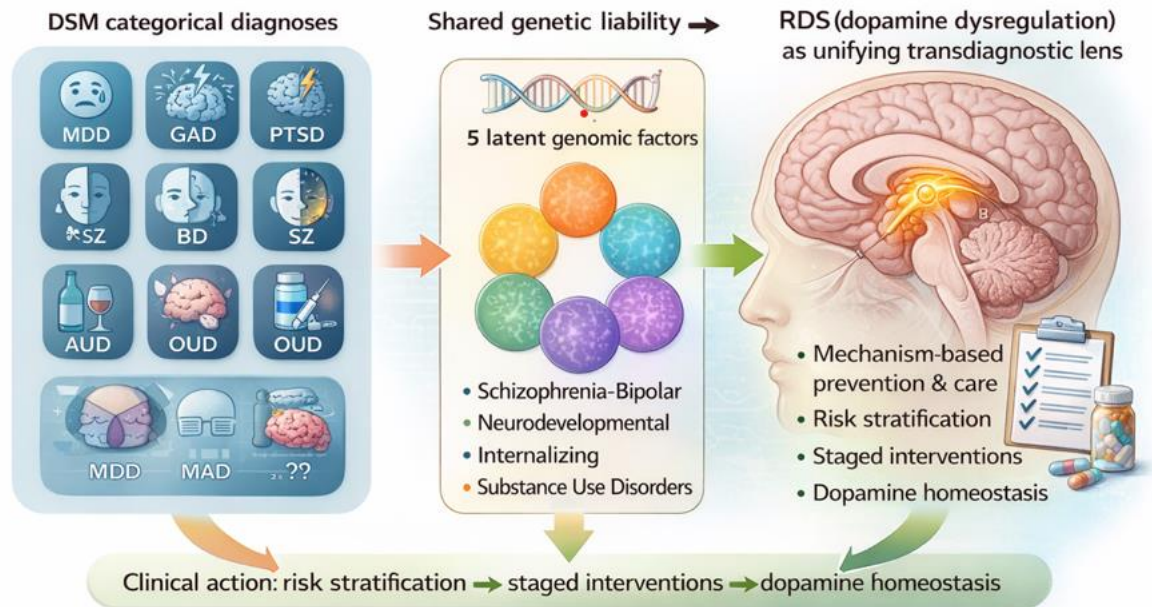
Conclusion: Current therapeutic modalities for AUD remain severely underused. Policy should focus primarily on integrating and improving patient access to existing multimodal approaches, such as pharmacotherapy in primary and telehealth care, and behavioral supports such as AA. While psychedelics do present an intriguing prospect, the priority should be implementing what works now while rigorously testing what may enhance care in the future.

Keywords: Alcohol Use Disorder; Naltrexone; Acamprosate; Disulfiram; Psychedelics; Psilocybin; Addiction Policy.

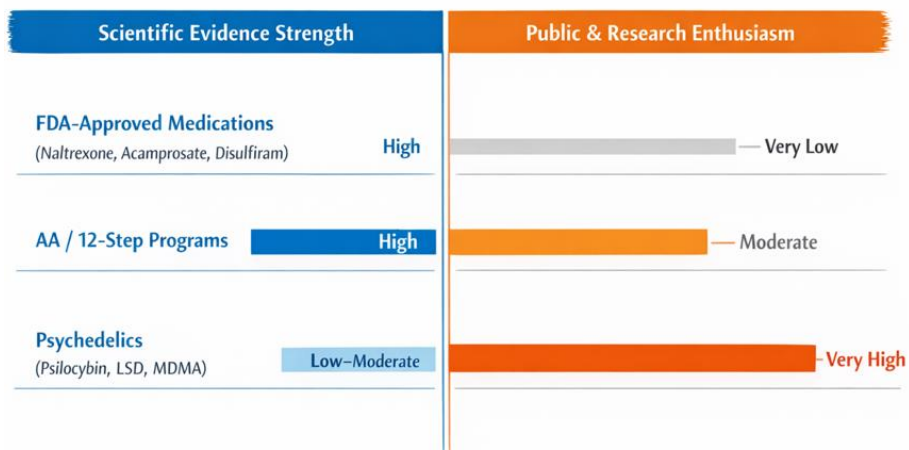
Key points

- Alcohol Use Disorder (AUD) affects 280 million people and 5% of deaths worldwide.
- Enthusiasm for psychedelics in AUD treatment feels novel, but the scientific interest dates back to the 1950s.
- Psilocybin is moving forward in promising clinical trials for a number of psychiatric illnesses but not for AUD.

Graphical Abstract



Evidence vs. Enthusiasm in Alcohol Use Disorder Treatments



Mismatch between evidence strength and therapeutic enthusiasm in Alcohol Use Disorder (AUD)

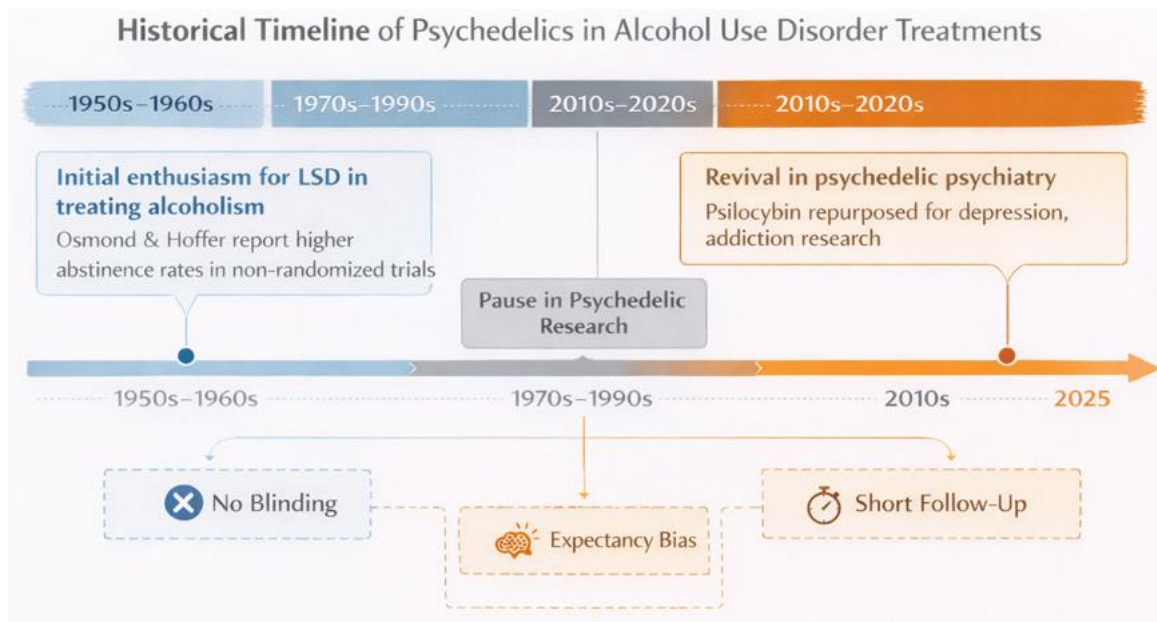
In this perspective, we utilized PUBMED to obtain relevant references carefully cited in this manuscript. Characterized by compulsive drinking, loss of control, and negative emotional states when not using, Alcohol Use Disorder (AUD) was linked to dementia [1]. Despite

advances in neuroscience and genetics, relapses approach 60-70 percent within a year of treatment cessation [2]. Stress is a risk factor for the development of AUD and for relapse, and furthermore, chronic alcohol use leads to adaptations in central and peripheral stress biology. Medications like naltrexone, acamprosate, and disulfiram were FDA-approved decades ago, and demonstrate consistent benefits—yet are rarely utilized [3]. Studies have shown that Naltrexone is beneficial by attenuating of craving via "psychological extinction" and reducing relapse [4].

Psychedelics like psilocybin, LSD, and MDMA have been considered potential breakthrough treatments for major depression, borderline personality disorder, eating disorders, Generalized Anxiety Disorder (GAD), end-of-life anxiety, and substance use disorders or reward deficiency [5,6]. Previously, the RDS Consortium theorized that Psychedelic Assisted Therapy (PAT) may play a role in the treatment of trauma-induced personality disorders. The authors identified Psychedelic Assisted Therapy (PAT) as a pathway for treating both BPD and PTSD, proposing that reframing BPD as PTSD could lead to more effective, personalized interventions, ultimately improving the quality of life for those affected by trauma. Such reclassification might also mitigate stigma, enhance our understanding of the underlying mechanisms, and optimize therapeutic interventions for a broader range of diagnostic categories characterized by anhedonia, negative affective states, hypervigilance, and dissociation [7].

“The bad news is, this enthusiasm, particularly around psilocybin, sometimes outpaced the data, raising critical questions about whether psychedelics are suited for treating AUD and if the field could be succumbing to a wave of therapeutic hype,” said A. Benjamin Srivastava [8]. Moreover, “The evidence for their role in treating AUD is limited and much of it is quite flawed. Meanwhile, we have extensive, high-quality evidence supporting the efficacy and safety of already-existing AUD treatments, especially when paired with structured behavioral support such as Alcoholics Anonymous (AA). As addiction psychiatrists, our immediate priority should be advocating wider use of treatments we already know work, rather than being swept away by incomplete and largely uninterpretable data on psychedelics. The last thing we

want is for patients to self-administer psilocybin for AUD when existing treatments are much safer and more effective.” [7]. Figure 1.



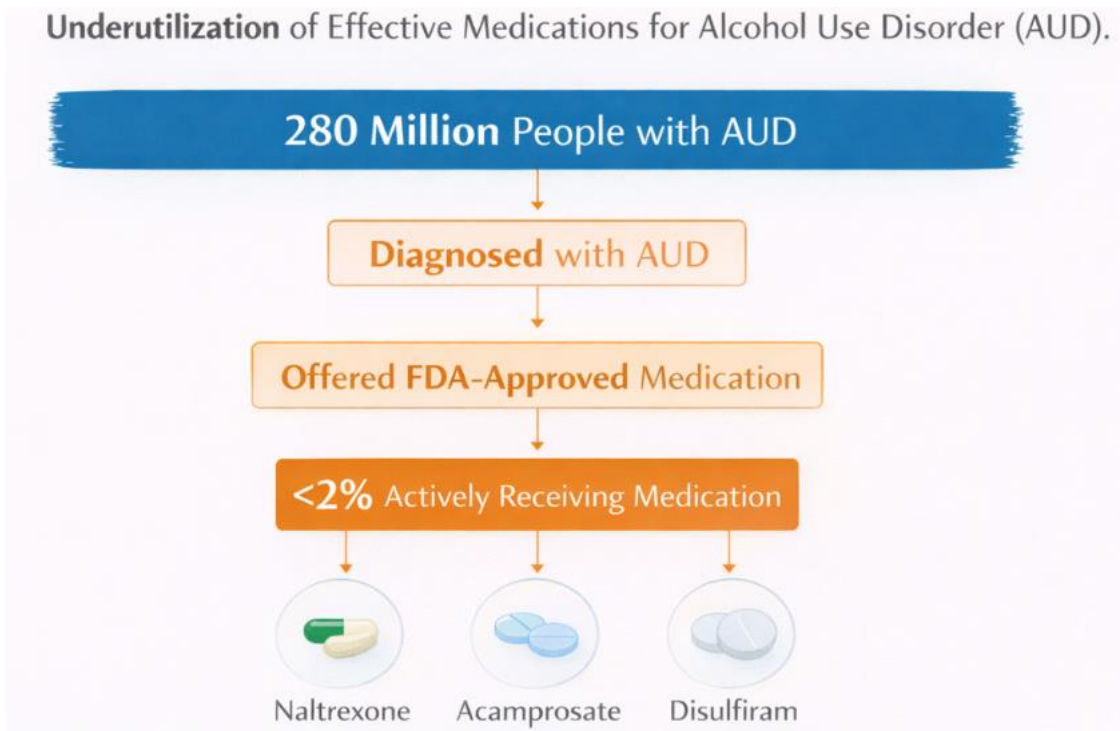
Canadian psychiatrists Humphry Osmond and Abram Hoffer experimented with LSD to treat alcoholism in the 1960s [8]. Osmond sought to reproduce “spiritual bottoming out” described by Alcoholics Anonymous (AA), which many recovered individuals cited as a turning point in recovery. In small studies, Osmond and Hoffer reported higher rates of abstinence among patients receiving LSD versus traditional therapy—sparking curiosity from an AA founder, Bill Wilson. But early studies were methodologically weak, without randomization, blinding, or standardized measures, and Wilson also abandoned his advocacy [9]. As Srivastava and Gold emphasize, enthusiasm around psychedelics is high, but shouldn’t substitute for quality evidence. Nor do we want people to try psilocybin on their own for AUD. In a recent article in JAMA, noted nonclinical psilocybin use has sharply increased in the US, particularly among adults 19-50 years, with more than 7 million individuals reporting past-year use [10].

In a phase 2 trial by Bogenschutz and colleagues, psilocybin combined with psychotherapy reduced heavy drinking days—but only in the final four weeks of a 36-week study, and after most assessments showed no difference between psilocybin and diphenhydramine placebos.

Up to 90 percent of study participants accurately guessed if they were given a psychedelic or a placebo, due to the unmistakable effects of psilocybin and LSD [11,12].

Psychedelic-assisted therapy may find a role in severe/refractory AUD cases [13]. Some studies hypothesize that psychedelics exert effects by promoting neuroplasticity or facilitating mystical experiences. Its potential lies in disrupting rigid network activity, potentially allowing for greater emotional and cognitive flexibility, and addressing identity in ways conventional treatments do not [14]. Overall, these various lines of research suggest that classic psychedelics might hold strong potential as therapeutics and as tools for experimentally investigating mystical experiences and behavioral-brain function more generally. But current evidence is insufficient to recommend psychedelics over or even alongside standard care [15].

Existing medications for AUD remain under-prescribed, although the director of the National Institute on Drug Abuse, Nora Volkow, and the National Institute on Alcohol & Alcohol Abuse and Alcoholism's (NIAAA) Dr. George Koob emphasize the need to use these medications. However, there is indeed an urgent need for potent effective, and non-addictive pharmaceuticals or nutraceuticals to treat all addictive behaviors or reward deficiency. One example is that oral naltrexone reduces relapse risk and heavy drinking days [16]. Acamprosate supports abstinence maintenance after detox, and disulfiram is highly effective under supervision—especially when abstinence is the goal. [17]. In addition, Acamprosate was also associated with significantly higher rates of treatment completion ($p = 0.004$) and medication compliance ($p < 0.001$) than placebo. Men and women did not differ on any measure of acamprosate efficacy, safety, or tolerability [18]. Figure 2.



The AUD treatment medication naltrexone can be administered orally or as an extended-release injection, Vivitrol (monthly). The injectable form addresses a major treatment barrier: adherence. Patients struggling with taking a daily medication may benefit from long-acting once-a-month injectable Naltrexone instead [19].

Approved in the United States in 2004, acamprosate modulates glutamate and GABA receptors, helping stabilize the neurochemical imbalance created by chronic alcohol use. It is particularly effective at supporting abstinence after detoxification [20].

Disulfiram (Antabuse) is often underused. While it doesn't reduce cravings, it acts powerfully through aversive conditioning. Anyone who drinks alcohol becomes severely nauseous because disulfiram blocks the ability to process alcohol. Open-label and supervised trials demonstrate that disulfiram has among the highest abstinence rates. [21]

Direct-to-Consumer Models

We have three FDA-approved medications, decades of data, and validated models for combining them with behavioral support. Yet <2 percent of people with AUD are actually in spite of recent efforts to provide increased access to Naltrexone using a Viagra-like direct-to-patient sales model, delivering naltrexone via telehealth, shipping medications to homes. Normalizing medication through direct-to-consumer marketing and flexible, patient-controlled paths may bring evidence-based treatments to public consciousness. Randomized clinical trials support the efficacy of MDMA in the treatment of PTSD and psilocybin in the treatment of depression and cancer-related anxiety. The research to support the use of LSD and ayahuasca in the treatment of psychiatric disorders is preliminary, although promising. Overall, the database is insufficient for FDA approval of any psychedelic compound for routine clinical use in psychiatric disorders at this time, but continued research on the efficacy of psychedelics for the treatment of psychiatric disorders is warranted.

In a November 2025 JAMA article, Mass General Hospital's Harvard psychiatrists advocate for making oral naltrexone available Over the Counter (OTC) to address unhealthy alcohol use and AUDs [22]. Both are under-treated in the U.S. Naltrexone reduces alcohol cravings and heavy drinking, but is underutilized primarily due to stigma, shame, and lack of awareness that Naltrexone works. OTC availability is a logical idea that could reduce harm, high-intensity drinking, and mirror successful public health strategies like OTC nicotine replacement products for smokers [23].

Not everyone wants to quit drinking—and that's okay. Research supports a step-care, harm-reduction model where reduced or controlled drinking goals are valid. Naltrexone works in this model by reducing the “buzz” [psychological extinction] of alcohol, allowing patients to regain control before they are ready for abstinence [24].

A large Cochrane review confirmed that AA and 12-step facilitation were more effective in maintaining abstinence than other psychological treatments. That makes AA a great addition

to Naltrexone treatment. AA, after all, is one of the most cost-effective tools for promoting abstinence and reducing alcohol-related harm, especially with pharmacotherapy [25].

Psychedelics may not deserve the therapeutic spotlight in AUD, but with depression, treatment-resistant anxiety, eating disorders, Post-Traumatic Stress Disorder (PTSD), and existential distress in terminal illnesses, there is compelling data for psilocybin, LSD, and others [26,27].

Randomized controlled trials at Johns Hopkins, Imperial College London, and other leading institutions have shown that one to two sessions of psilocybin-assisted therapy can rapidly and dramatically reduce depressive symptoms. In some cases, it has effects that last a month. On November 4, 2025, psychedelic drug developer Compass Pathways said it's accelerating the FDA process to move its experimental psilocybin-based depression therapy up 9-12 months. The decision follows completion of enrollment for a late-stage study for the psychedelic-based therapy, COMP360, as well as a positive meeting with the FDA [28].

Studies in GAD and obsessive-compulsive disorder reinforce the idea that psychedelics may “reset” entrenched cognitive patterns. Given that these conditions often involve repetitive thought loops and maladaptive rumination, psychedelics may hold a unique role where antidepressants and anxiolytics fail [29].

Additionally, in terminally ill patients facing anxiety and depression, psychedelics have demonstrated rapid, enduring reductions in distress, decreases in death anxiety, and greater spiritual well-being persisting six months or longer. These outcomes are compelling because conventional medications offer modest relief and don't address existential suffering [30]. Finally, a single dose of psilocybin combined with five psychotherapy sessions may not be sufficient to reduce relapse rates and alcohol use in severely affected AUD patients following withdrawal treatment [31]. However, given the limited sample size of our study, larger trials are needed in the future to confirm these findings [SEE TABLE 1].

Policy

Precision and Compassion in Addiction Care

Principle

For alcohol use disorder (AUD), policy should prioritize interventions with proven safety, scalability, and real-world effectiveness while maintaining scientific openness to innovation. The question is not psychedelics or standard care, but how to deploy what works now while testing what may augment care later. Emerging RCTs of psilocybin in AUD show mixed and methodologically limited findings (expectancy and psychotherapy confounds), reinforcing a trial-only posture with stringent blinding, active comparators, and longer follow-up [7,10,31].
Table 1.

1) Scale What Already Works

- **Make medication the default.** Embed opt-out prescribing of FDA-approved AUD medications in primary care, emergency departments, and behavioral-health settings.
- **Normalize stepped care.** Support both abstinence and harm-reduction goals. Oral naltrexone for reduction, acamprosate for abstinence, supervised disulfiram for aversive support.
- **Adherence solutions.** Expand access to extended-release naltrexone.
- **Behavioral scaffolding.** Integrate AA/12-Step or SMART Recovery handoffs.

2) Reduce Access Frictions

- **OTC pathway for oral naltrexone.** Authorize pharmacist protocols and pilot state programs to assess feasibility [22].
- **Tele-AUD coverage parity.** Require payer parity for telehealth initiation and maintenance visits.
- **Prior-authorization reform.** Remove PA for first-line AUD medications.

3) Accountability & Outcomes

- **Measure what matters.** Tie reimbursement to pharmacotherapy initiation, reduction in heavy-drinking days, liver-function improvement, and patient-reported outcomes.
- **Quality reporting.** Add AUD medication metrics to HEDIS and public dashboards.

4) Equity & Stigma Reduction

- **Community delivery.** Fund mobile and re-entry programs using peer navigators.
- **Messaging reset.** Recast medication as brain-health treatment, not “willpower failure.”

5) Guardrails for Psychedelic Innovation

- **Trial-only for AUD.** Limit psychedelics to IRB-approved trials until robust RCT evidence shows clear benefit.
- **Methodological rigor.** Require active-comparator blinding, multi-site designs, and long follow-up.
- **Transdiagnostic science.** Study mechanisms (neuroplasticity, affective flexibility) without displacing established care.

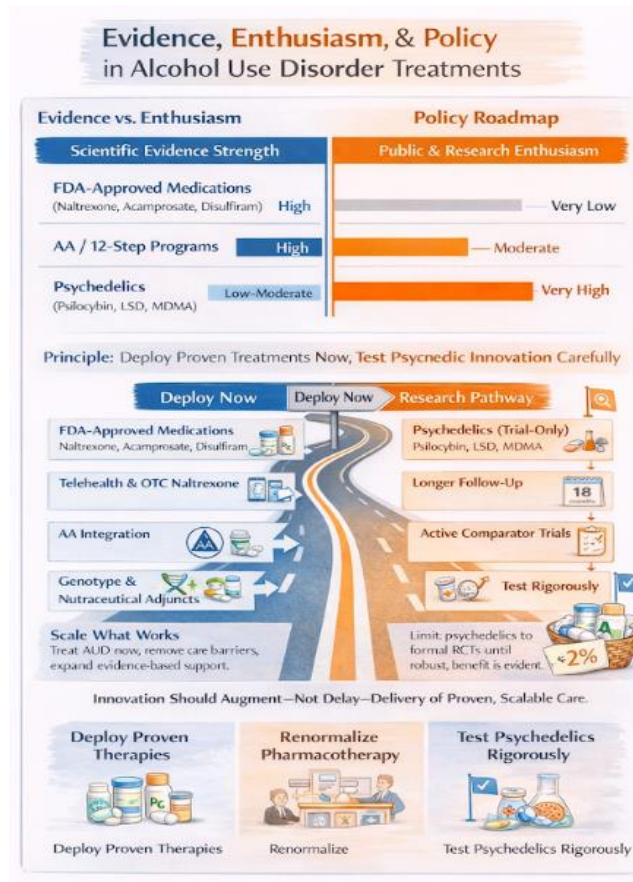
6) Precision & Next-Gen Adjuncts

- **Genotype-guided care.** Support **GARS** and other neurogenetic tools to stratify risk and inform treatment choice.
- **Coverage for nutraceuticals.** Include **KB220-class pro-dopamine regulators** and other evidence-based nutraceuticals as non-addictive dopaminergic modulators, paired with outcomes tracking.
- **Integrated addiction–liver pathways.** Unite hepatology and addiction clinics around shared metrics.

Bottom line

For AUD, deploy what works now, remove friction, and measure results—while advancing precision neurogenetic adjuncts and validated nutraceuticals within structured research and value-based care (Figure 3).

Genetic Variant	Mechanism / Neurobiology	Behavioral & Substance Predisposition Risks
COMT (Catechol-O-Methyltransferase, G allele)	Enzyme regulating dopamine breakdown in prefrontal cortex; variants affect dopamine tone and decision-making.	Alcohol, Cannabis, Nicotine, Opioids, Stimulants, Glucose; ↑ Impulsivity & addiction vulnerability.
DRD1 (Dopamine Receptor D1, A allele)	Dopamine receptor signaling in reward circuits.	Alcohol, Nicotine.
DRD2 (Dopamine Receptor D2, A variant; associated with ANKK1)	Reduced receptor density → ↓ reward sensitivity; linked with ANKK1 variant altering dopamine signaling.	Cocaine, Alcohol, Nicotine, Glucose; ADHD/ADD, OCD, Pathological Aggression; ↑ Substance abuse vulnerability.
DRD3 (Dopamine Receptor D3, C variant)	Altered dopamine receptor binding, affecting motivation/reward.	Cocaine, Opioids, Glucose.
DRD4 (Dopamine Receptor D4, C variant)	Modulates dopamine-related novelty-seeking and risk-taking.	Alcohol, Cannabis, Nicotine, Opioids; Non-Substance Behaviors: Overeating, PTSD.
DAT1 / SLC6A3 (Dopamine Transporter, G allele / polymorphisms)	Regulates dopamine reuptake; altered transporter activity impacts impulsivity.	Alcohol, Cannabis, Cocaine, Heroin, Nicotine; ↑ Impulsivity.
5-HTTLPR / SLC6A4 (Serotonin Transporter Gene, S or LG allele)	Affects serotonin transport; influences mood, depression risk, and impulsivity.	Alcohol, Cocaine, Heroin, Nicotine; ADD/ADHD, Depression (Anhedonia), PTSD.
MAOA (Monoamine Oxidase A, 4R variant)	Enzyme degrades dopamine & serotonin; variants affect neurotransmitter balance.	Alcohol, Cannabis, Cocaine, Nicotine, Glucose; ↑ Aggression, Harm Avoidance issues.
GABRB3 (GABA Receptor Subunit β3, over-expressed 181 allele)	GABAergic inhibitory control; dysregulation contributes to anxiety/addiction risk.	Alcohol; Anxiety-related substance abuse.
OPRM1 (Opioid Receptor Mu 1)	Modulates opioid system & response to natural/reinforcing stimuli.	Alcohol, Opioids; ↑ Dependence risk, altered pain/reward sensitivity.
BDNF (Brain-Derived Neurotrophic Factor)	Neuroplasticity & adaptability of reward circuits.	General addiction vulnerability; stress-related relapse risk.
TH (Tyrosine Hydroxylase)	Rate-limiting enzyme in dopamine synthesis.	Altered dopamine production → ↑ Addiction risk.
CHRNA4 (Nicotinic Acetylcholine Receptor Subunit Alpha-4)	Regulates acetylcholine signaling; critical in nicotine reward.	Nicotine dependence, other substance use disorders.



Limitations

Alcohol is a harmful drug, and reducing its consumption is a significant challenge for users. Furthermore, alcohol dependence is often treatment-resistant, and no completely effective treatment model is available for chemical dependence. Classic psychedelics, such as LSD, psilocybin, and ayahuasca, have been used in different clinical and pre-clinical trials, demonstrating promising pharmacotherapeutic effects in the treatment of treatment-resistant psychopathological conditions, such as addiction, especially related to alcohol dependence. Although many participants achieve positive results with only one treatment dose in clinical studies, great inter-individual variability exists in the duration of these effects. Therefore, further studies using different doses and experimental protocols should be conducted to enhance evidence about psychedelic substances [32]. For decades, psychedelics have been investigated for the treatment of psychiatric disorders. Specifically, evidence suggests that

psychedelics may have therapeutic potential for the treatment of alcohol use disorder. Several studies with classic psychedelics, including LSD and psilocybin, show promising results, with psychedelics decreasing alcohol drinking and promoting abstinence in individuals with alcohol use disorder. [33]. The 5-HT_{2A} receptor (5-HT_{2A}R) is a key regulator of meso-corticolimbic DA release and controls cellular mechanisms underlying cocaine effects. 5-HT_{2A}R actions contribute importantly to psychedelic mechanisms of action. The psychedelic 5-HT_{2A}R agonist (-)-2,5-dimethoxy-4-iodoamphetamine [(-)-DOI] was found to devalue cocaine reward and motivation to take cocaine in a 5-HT_{2A}R-dependent manner. Psychedelic use among people who use methamphetamine may improve mood and social function and reduce substance use [34]. In one study, the main results reported psychedelic users as less depressed (Patient Health Questionnaire-9; PHQ-9) ($d = -0.29$) and having more use of drugs (Drug Use Disorders Identification Test; DUDIT) ($d = 1.27$). In the Big Five personality traits, openness differed notably ($d = 1.72$), and the between-group effects in PHQ-9 were explained by lower neuroticism [35].

In addition, results from the pattern recognition task showed that current ecstasy users produced significantly more errors than the other two groups ($p < 0.01$). When results were combined for both the letter and pattern tasks, once again current ecstasy users produced significantly more errors than non-ecstasy users ($p < 0.01$). Working memory deficits obtained were statistically significant with both ecstasy using groups performing significantly worse than non-users on the computation span measure ($p < 0.01$). Moreover, ANCOVA with measures of processing speed as covariates failed to eliminate the group difference in computation span ($p < 0.01$) [36]. Indeed, there is some evidence that supports the use of psilocybin in alleviating symptoms of depression and anxiety; psychedelic compounds also have potential as alcohol use disorder treatments and may help reduce symptoms tied to opioid withdrawal [37]. Psychedelic use among people who use methamphetamine may improve mood and social function and reduce substance use. Post-experience, participants reported improved mood (59.3 %) and social functioning (49.6 %), and reduced use of methamphetamine (34.0 %) and other substances (35.1 %). Forward planning and fewer challenging experiences were linked to improved mood

(aOR 1.84, $p = 0.048$; aOR 2.21, $p = 0.012$) and reduced substance use (aOR 2.27, $p = 0.008$; aOR 3.58, $p < 0.001$) [38].

Importantly, several drug classes (MDMA metabolites or analogs, anesthetics, muscle relaxants, amphetamines and stimulants, benzodiazepines, ethanol, opioids), four antidepressants (bupropion, sertraline, venlafaxine and citalopram) and olanzapine demonstrated increased odds ratios for the reported risk of death [39].

One important question that still needs to be investigated - Why might psychedelics demonstrate efficacy in depression, PTSD, or end-of-life distress, but not consistently in AUD? It is well known that ethanol works on the brain to produce its desired effects, e.g., sociability and intoxication, and hence the brain is an important organ for exploring subsequent harm. These come in many different forms such as the consequences of damage during intoxication, e.g., from falls and fights, damage from withdrawal, damage from the toxicity of alcohol and its metabolites and altered brain structure and function with implications for behavioral processes such as craving and addiction. On top of that are peripheral factors that compound brain damage such as poor diet, vitamin deficiencies leading to Wernicke-Korsakoff syndrome. Prenatal alcohol exposure can also have a profound impact on brain development and lead to irremediable changes of fetal alcohol syndrome [40].

Conclusion

Psychedelic use grew sixfold among U.S. adults 35–50 years old from 2014 to 2024, based, in part, on positive controlled research clinical trials. Clinical trials are highly controlled medical procedures designed for scientific rigor and patient safety, while self-medication occurs in uncontrolled, non-medical settings, with unapproved "street drugs", and carries significant immediate and long-term risks.

The path forward isn't about choosing between psychedelics and standard medications — it's about prioritizing treatments, comparing to existing treatments, saving lives while supporting innovative research. The utility of psychedelics in depression, end-of-life distress, GAD, eating

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disorders, and trauma are compelling. But with AUD, hype runs ahead of science. Clinicians seek to reduce harm, save lives, and restore function. For AUD, we have the tools. The next step is ensuring they are actually used.

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Conflict of Interest: KB owns patents linked to both GARS and KB220

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References

1. Monnig, M., Shah, K. (2024). Linking alcohol use to Alzheimer's disease: Interactions with aging and APOE along immune pathways. *Med Res Arch*, 12(8): 1. <https://doi.org/10.18103/mra.v12i8.5228>
2. Milivojevic, V., Sinha, R. (2025). Laboratory and real-world experimental approaches to understanding alcohol relapse. *Curr Top Behav Neurosci*, 72: 427–451. https://doi.org/10.1007/7854_2023_456
3. Blum, K., Lott, L., Baron, D., Smith, D.E., Badgaiyan, R.D., Gold, M.S. (2020). Improving naltrexone compliance and outcomes with putative pro-dopamine regulator KB220, compared to treatment as usual. *J Syst Integr Neurosci*, 7: 1. <https://doi.org/10.15761/JSIN.1000229>
4. Blum, K., Modestino, E.J., Badgaiyan, R.D., Baron, D., Thanos, P.K., Elman, I., Siwicki, D., Febo, M., Gold, M.S. (2018). Analysis of evidence for the combination of pro-

- dopamine regulator (KB220PAM) and naltrexone to prevent opioid use disorder relapse. *EC Psychol Psychiatr*, 7(8): 564–579. [PubMed](#)
5. Martire, G., Sipple, D., Baron, D., Gold, M.S., Lewandowski, K.U., Dennen, C.A., Sharafshah, A., et al. (2024). Theorizing that psychedelic assisted therapy may play a role in the treatment of trauma-induced personality disorders. *J Addict Psychiatry*, 8(2): 161–165. [PubMed](#)
 6. Jowett, S., Karatzias, T., Albert, I. (2020). Multiple and interpersonal trauma are risk factors for both post-traumatic stress disorder and borderline personality disorder: A systematic review. *Psychol Psychother*, 93(3): 621–638. <https://doi.org/10.1111/papt.12248>
 7. Srivastava, A.B., Gold, M.S. (2025). A tragedy of errors: The state of psychedelic research in the treatment of alcohol use disorder. *Brain Sciences*, 15(11): 1190. <https://doi.org/10.3390/brainsci15111190>
 8. Dyck, E. (2005). Flashback: Psychiatric experimentation with LSD in historical perspective. *Can J Psychiatry*, 50(7): 381–388. <https://doi.org/10.1177/070674370505000703>
 9. Novak, S.J. (1997). LSD before Leary: Sidney Cohen's critique of 1950s psychedelic drug research. *Isis*, 88(1): 87–110. <https://doi.org/10.1086/383628>
 10. Hutchison, K.E., Hooper, J.F., Karoly, H.C. (2025). Psilocybin outside the clinic: Public health challenges of increasing publicity, accessibility, and use. *JAMA Psychiatry*: 1. <https://doi.org/10.1001/jamapsychiatry.2025.3038>
 11. Bogenschutz, M.P., Pommy, J.M. (2012). Therapeutic mechanisms of classic hallucinogens in the treatment of addictions: From indirect evidence to testable hypotheses. *Drug Test Anal*, 4(7–8): 543–555. <https://doi.org/10.1002/dta.1376>
 12. Bogenschutz, M.P., Johnson, M.W. (2016). Classic hallucinogens in the treatment of addictions. *Prog Neuropsychopharmacol Biol Psychiatry*, 64: 250–258. <https://doi.org/10.1016/j.pnpbp.2015.03.002>
 13. Johnson, M.W., Hendricks, P.S., Barrett, F.S., Griffiths, R.R. (2019). Classic psychedelics: An integrative review of epidemiology, therapeutics, mystical experience, and brain

- network function. *Pharmacol Ther*, 197: 83–102.
<https://doi.org/10.1016/j.pharmthera.2018.11.010>
14. Lyvers, M., Meester, M. (2012). Illicit use of LSD or psilocybin, but not MDMA or nonpsychedelic drugs, is associated with mystical experiences in a dose-dependent manner. *J Psychoactive Drugs*, 44(5): 410–417. <https://doi.org/10.1080/02791072.2012.736842>
15. Romeo, B., Kervadec, E., Fauvel, B., Strika-Bruneau, L., Amirouche, A., Bezo, A., Piolino, P., Benyamina, A. (2025). The intensity of the psychedelic experience is reliably associated with clinical improvements: A systematic review and meta-analysis. *Neurosci Biobehav Rev*, 172: 106086. <https://doi.org/10.1016/j.neubiorev.2025.106086>
16. Morley, K.C., Kranzler, H.R., Luquin, N., Jamshidi, N., Adams, C., Montebello, M., et al. (2024). Topiramate versus naltrexone for alcohol use disorder: A genotype-stratified double-blind randomized controlled trial. *Am J Psychiatry*, 181(5): 403–411. <https://doi.org/10.1176/appi.ajp.20230666>
17. Hu, W., Morris, B., Carrasco, A., Kroener, S. (2015). Effects of acamprosate on attentional set-shifting and cellular function in the prefrontal cortex of chronic alcohol-exposed mice. *Alcohol Clin Exp Res*, 39(6): 953–961. <https://doi.org/10.1111/acer.12722>
18. Mason, B.J., Leher, P. (2012). Acamprosate for alcohol dependence: A sex-specific meta-analysis based on individual patient data. *Alcohol Clin Exp Res*, 36(3): 497–508. <https://doi.org/10.1111/j.1530-0277.2011.01616.x>
19. Ndegwa, S., Pant, S., Pohar, S., Mierzwinski-Urban, M. (2017). Injectable extended-release naltrexone to treat opioid use disorder. *CADTH Issues in Emerging Health Technologies*, 163: 1. [PubMed](#)
20. Florence, L., Lassi, D.L.S., Kortas, G.T., Lima, D.R., de Azevedo-Marques Périco, C., Andrade, A.G., Torales, J., Ventriglio, A., De Berardis, D., De Aquino, J.P., Castaldelli-Maia, J.M. (2022). Brain correlates of the alcohol use disorder pharmacotherapy response: A systematic review of neuroimaging studies. *Brain Sci*, 12(3): 386. <https://doi.org/10.3390/brainsci12030386>
21. Peachey, J.E. (1981). A review of the clinical use of disulfiram and calcium carbamide in alcoholism treatment. *J Clin Psychopharmacol*, 1(6): 368–375. <https://doi.org/10.1097/00004714-198111000-00004>

22. Terechin, O., Matta, S.E., Suzuki, J. (2025). Over-the-counter naltrexone to address unhealthy alcohol use. *JAMA Psychiatry*: 1. <https://doi.org/10.1001/jamapsychiatry.2025.3035>
23. Blum, K., Gold, M., Clark, H.W., Dushaj, K., Badgaiyan, R.D. (2016). Should the United States government repeal restrictions on buprenorphine/naloxone treatment? *Subst Use Misuse*, 51(12): 1674–1679. <https://doi.org/10.1080/10826084.2016.1200097>
24. Blum, K., Lott, L., Baron, D., Smith, D.E., Badgaiyan, R.D., Gold, M.S. (2020). Improving naltrexone compliance and outcomes with putative pro-dopamine regulator KB220, compared to treatment as usual. *J Syst Integr Neurosci*, 7: 1. <https://doi.org/10.15761/JSIN.1000229>
25. Blum, K., Thompson, B., Demetrovics, Z., Femino, J., Giordano, J., Oscar-Berman, M., et al. (2015). The molecular neurobiology of twelve steps program & fellowship: Connecting the dots for recovery. *J Reward Defic Syndr*, 1(1): 46–64. <https://doi.org/10.17756/jrds.2015-008>
26. Correa da Costa, S., Bormann, N.L., Oesterle, T., McGinnis, M.T., Ho, M.F., Vetteson-Trutza, S.A., Rummans, T., Gold, M.S. (2025). The role of psychedelics in the treatment of substance use disorders: An overview of systematic reviews. *Brain Sci*, 15(10): 1056. <https://doi.org/10.3390/brainsci15101056>
27. Blum, K., Gondré-Lewis, M.C., Modestino, E.J., Lott, L., Baron, D., Siwicki, D., McLaughlin, T., et al. (2019). Understanding the scientific basis of post-traumatic stress disorder (PTSD): Precision behavioral management overrides stigmatization. *Mol Neurobiol*, 56(11): 7836–7850. <https://doi.org/10.1007/s12035-019-1600-8>
28. Goodwin, G.M., Aaronson, S.T., Alvarez, O., Atli, M., Bennett, J.C., Croal, M., DeBattista, C., et al. (2023). Single-dose psilocybin for a treatment-resistant episode of major depression: Impact on patient-reported outcomes. *J Affect Disord*, 327: 120–127. <https://doi.org/10.1016/j.jad.2023.01.108>
29. Kim, V., Wilson, S.M., Woesner, M.E. (2025). The use of classic psychedelics for depressive and anxiety-spectrum disorders: A comprehensive review. *J Clin Psychopharmacol*, 45(1): 37–45. <https://doi.org/10.1097/JCP.0000000000001941>

30. Jain, M.K., Gumper, R.H., Slocum, S.T., Schmitz, G.P., Madsen, J.S., Tummino, T.A., et al. (2025). The polypharmacology of psychedelics reveals multiple targets for potential therapeutics. *Neuron*, 113(19): 3129–3142. <https://doi.org/10.1016/j.neuron.2025.06.012>
31. Rieser, N.M., Bitar, R., Halm, S., Rossgoderer, C., Gubser, L.P., Thévenaz, M., et al. (2025). Psilocybin-assisted therapy for relapse prevention in alcohol use disorder: A phase 2 randomized clinical trial. *EClinicalMedicine*, 82: 103149. <https://doi.org/10.1016/j.eclinm.2025.103149>
32. Lodetti, G., de Bitencourt, R.M., Rico, E.P. (2024). Classic psychedelics and the treatment for alcoholism. *Prog Neuropsychopharmacol Biol Psychiatry*, 135: 111129. <https://doi.org/10.1016/j.pnpbp.2024.111129>
33. Marinho, E.A.V., Serra, Y.A., Oliveira-Lima, A.J., Marcourakis, T., Berro, L.F. (2024). Ayahuasca for the treatment of alcohol use disorder. *Int Rev Neurobiol*, 178: 283–300. <https://doi.org/10.1016/bs.irn.2024.07.007>
34. Bahceci, D., Siefried, K., Steele, M., Harrod, M., Bell, G., Barratt, M.J., et al. (2025). Exploring psychedelic experiences among people who regularly use methamphetamine: Findings from an international survey. *Drug Alcohol Depend*, 272: 112699. <https://doi.org/10.1016/j.drugalcdep.2025.112699>
35. Sjöström, D.K., Claesdotter-Knutsson, E., Kajonius, P.J. (2024). Personality traits explain the relationship between psychedelic use and less depression in a comparative study. *Sci Rep*, 14(1): 10195. <https://doi.org/10.1038/s41598-024-60890-1>
36. Wareing, M., Fisk, J.E., Montgomery, C., Murphy, P.N., Chandler, M.D. (2007). Information processing speed in ecstasy (MDMA) users. *Hum Psychopharmacol*, 22(2): 81–88. <https://doi.org/10.1002/hup.827>
37. Jordan, A. (2023). Emerging perspectives in addiction psychiatry. *J Clin Psychiatry*, 84(4): 1. [PubMed](#)
38. Bahceci, D., Siefried, K., Steele, M., Harrod, M., Bell, G., Barratt, M.J., et al. (2025). Exploring psychedelic experiences among people who regularly use methamphetamine: Findings from an international survey. *Drug Alcohol Depend*, 272: 112699. <https://doi.org/10.1016/j.drugalcdep.2025.112699>

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- 39.** Cohen, I.V., Makunts, T., Abagyan, R., Thomas, K. (2021). Concomitant drugs associated with increased mortality for MDMA users reported in a drug safety surveillance database. *Sci Rep*, 11(1): 5997. <https://doi.org/10.1038/s41598-021-85389-x>
- 40.** Nutt, D., Hayes, A., Fonville, L., Zafar, R., Palmer, E.O.C., Paterson, L., Lingford-Hughes, A. (2021). Alcohol and the brain. *Nutrients*, 13(11): 3938. <https://doi.org/10.3390/nu13113938>