

Investigating Therapeutic Targets for Alcohol Use Disorder: Pharmacological View of ClinicalTrials.gov Data

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Background: Alcohol Use Disorder (AUD) is a pervasive and chronic condition that causes considerable individual and societal burden. Therapeutic interventions remain limited, highlighting the critical need for the development of more effective interventions.

Objective: This study aims to explore therapeutic targets in AUD by analyzing interventional clinical trials registered on ClinicalTrials.gov. It seeks to identify prevailing pharmacological and behavioral treatment strategies and emerging areas of investigation.

Methods: A comprehensive search of ClinicalTrials.gov using the keywords “Alcohol Use Disorder” identified 209 completed interventional trials. Trials were categorized by intervention type, pharmacological targets, study phase, and participant demographics. Descriptive statistics were used to assess the prevalence and characteristics of the therapeutic strategies under investigation.

Results: Pharmacological interventions were predominant, targeting systems such as cholinergic (nicotinic receptors), opioid, GABAergic, and glutamatergic pathways. Behavioral interventions were also common, frequently combined with pharmacological treatments. Trials investigating novel targets such as neuroinflammation and neuromodulation are emerging. Most studies were small in scale and were at early to mid-phase stages. Overall, the treatment outcomes showed modest to moderate success in reducing alcohol cravings, increasing abstinence duration, and preventing relapse, though larger confirmatory trials remain limited.

Conclusion: The clinical trials for AUD reflects a strong focus on neurotransmitter-targeted pharmacotherapy, supplemented by behavioral approaches. Newer interventions targeting neuroimmune pathways and brain stimulation techniques show promise but require further investigation in larger, more advanced trials.

Keywords: alcohol use disorder, pharmacological interventions, behavioral therapy, clinical trials, neurotransmitter systems, neuroinflammation, neuromodulation

Introduction

Alcohol Use Disorder (AUD) is a chronic, prevalent mental disorder that affects millions worldwide. A national survey on drug use and health revealed that almost 29 million people aged 12 and above had AUD in the past year in the United States.¹ This condition is associated with several negative consequences affecting quality of life.^{2,3} These effects increase the risk of damage to the nervous, endocrine, pulmonary, gastrointestinal, cardiovascular, and other systems.^{4–6} AUD can specifically contribute to multiple complications such as periodontal problems, nutritional deficiencies, liver cirrhosis, brain stroke, coronary artery disease, sexually transmitted infections, hypothyroidism, and diabetes complications.^{4,7–9} AUD has also been associated with the development of different types of cancer.^{10–12} Moreover, AUD patients are more likely to place an economic burden on health systems.^{13,14}

AUD is associated with significant alterations in neurotransmitter systems, leading to acute and chronic neuroadaptive changes.^{15–17} The key neurotransmitters involved include Gamma-aminobutyric acid (GABA), glutamate, dopamine, serotonin, and endogenous opioids.^{18–20} These alterations contribute to the behavioral, emotional, and cognitive symptoms of AUD. For instance, chronic alcohol consumption enhances GABAergic inhibition and suppresses

glutamatergic excitation, leading to tolerance and withdrawal symptoms upon cessation.^{21–23} Moreover, dysregulation of dopamine pathways in the mesolimbic system reduces sensitivity to natural rewards, reinforcing alcohol-seeking behavior.^{24,25} Serotonin deficits contribute to mood instability and impulsivity, while alterations in the endogenous opioid system amplify alcohol’s rewarding effects.^{26,27} Based on that, these neurotransmitters are classified as major targets for pharmacological treatments. The diverse pharmacological mechanisms of action involve inhibition of aldehyde dehydrogenase, dopamine β-hydroxylase, serotonin, and opioid receptor antagonist,^{28,29} and modulating GABA activity in the brain.^{5,8,16}

AUD affects several key brain regions, leading to significant neuroadaptive changes. Dysfunctions in the prefrontal cortex, which plays a role in decision making and impulse control, may contribute to compulsive alcohol-seeking behaviors and difficulties with regulating consumption.^{18,30} The amygdala, which plays a crucial role in stress and emotional regulation, exhibits heightened reactivity, leading to increased anxiety, emotional dysregulation, and a greater susceptibility to relapse.^{31–33} The nucleus accumbens, a key component of the brain’s reward system, undergoes changes in dopamine signaling, resulting in altered reward processing and intensified cravings for alcohol.^{34–36} Additionally, the hippocampus, critical for learning and memory, experiences neurotoxicity and structural damage, leading to cognitive deficits.^{37–39} These neurobiological alterations collectively contribute to the progression and persistence of AUD, making it challenging for individuals to control their alcohol consumption and maintain long-term abstinence.

Different therapeutic options have been used to manage AUD, including pharmacological and behavioral treatments.^{40–42} Precisely, the most effective management strategy for AUD involves both pharmacological and psychosocial interventions.^{43,44}

The Food and Drug Authority has approved only a limited number of medications for use with addiction, including disulfiram, naltrexone, and acamprosate.^{29,42,45} These medications play a crucial role in the treatment of substance use disorders by targeting different aspects of addiction management.^{29,46,47} Disulfiram works by creating an adverse reaction to alcohol consumption, while naltrexone helps reduce cravings and the rewarding effects of alcohol and opioids.^{48–51} Acamprosate is primarily used to maintain abstinence by restoring the balance of neurotransmitters in the brain.^{52,53} Table 1 provides a summary of AUD medications’ pharmacology.

Emerging interventions for AUD include novel pharmacotherapies such as GLP-1 receptor agonists, for example semaglutide, liraglutide, and tirazepide. These have shown promise in reducing alcohol intake through appetite and reward modulation.^{54–57} Additionally, psychedelic-assisted therapies using psilocybin, MDMA, and other psychedelics might facilitate behavior change, reduce cravings, and support long-term abstinence when combined with psychotherapy.^{58–60} These innovative and emerging approaches may offer alternatives to traditional treatments, particularly for individuals with treatment-resistant AUD.^{61,62}

Psychosocial interventions play a crucial role in the comprehensive management of AUD, aiming to address the behavioral, emotional, and social aspects of the condition.^{63,64} These interventions include cognitive-behavioral therapy (CBT), motivational interviewing (MI), contingency management, and group-based therapies such as 12-step programs

Table 1 Medication, Pharmacological Class, Mechanism of Action, Uses in AUD

Medication	Pharmacological Class	Mechanism of Action	Uses in Alcohol Use Disorder (AUD)
Disulfiram	Aldehyde dehydrogenase inhibitor	Inhibits aldehyde dehydrogenase, leading to accumulation of acetaldehyde.	Used as an aversion therapy to discourage alcohol consumption.
Naltrexone	Opioid antagonist	Blocks opioid receptors, reducing the rewarding effects of alcohol and decreasing cravings.	Reduces alcohol cravings and prevents relapse.
Acamprosate	Glutamate modulator	Modulates glutamatergic neurotransmission to restore balance in the brain.	Helps maintain abstinence by stabilizing neurotransmitter activity.

(eg, Alcoholics Anonymous).^{65–67} They help individuals identify and change maladaptive drinking behaviors, develop coping skills to handle triggers and stressors, and enhance motivation for maintaining abstinence.

The pathophysiology of AUD is complex and current research reflects efforts to target specific neurotransmitter systems, such as GABA, glutamate, and the opioid system. Additionally, neuromodulatory techniques, immune-targeting therapies, and behavioral interventions continue to gain traction as adjunctive or standalone treatments. This paper explores the diverse therapeutic strategies identified in clinical trials and examines their potential to improve outcomes for patients with AUD. As AUD becomes increasingly recognized as a neurobiological disorder, there is growing interest in identifying new therapeutic targets that can address the diverse mechanisms contributing to addiction. Advances in clinical research, particularly trials cataloged on platforms such as ClinicalTrials.gov, provide a rich resource for understanding the evolving landscape of therapeutic strategies for AUD.

Methods

The data for this paper were obtained from ClinicalTrials.gov using the search term “Alcohol Use Disorder” to identify relevant clinical trials up to 26 March 2025 (see [Supplementary File](#)). The dataset included information on trial status, interventions, targeted mechanisms, and outcomes. For the purpose of this review, the trials were filtered based on their intervention type (pharmacological, behavioral, or other), phase of development, and study status (ongoing, completed, or terminated). Age group classifications are defined by ClinicalTrials.gov, as child (birth–17 years), adult (18–64 years), and older adult (65 years and older).

The analysis included trials from Phase I through to Phase IV, with a focus on completed studies to evaluate efficacy, as well as ongoing studies to highlight promising future treatments. Descriptive statistics were used to summarize the data. [Figure 1](#) shows the filtration process of the included studies. Statistical analyses were performed to identify trends in therapeutic targets and to assess the success rates of different approaches.

Results

A total of 209 interventional clinical trials focused on Alcohol Use Disorder (AUD) have been completed and their results reported on ClinicalTrials.gov. All were interventional in nature, and the majority targeted adult populations, as shown in [Table 2](#).

The vast majority of studies focused on adults and older adults, with a limited inclusion of children, consistent with AUD prevalence. A summary of age distribution in AUD clinical trials is presented in [Table 3](#).

Pharmacological interventions in AUD trials targeted a range of neurobiological systems implicated in alcohol use and dependence. The cholinergic system, particularly via nicotinic receptors (eg, varenicline), was the most frequently studied target ($n = 8$). This was followed by the opioid system (eg, naltrexone) and combined GABAergic/glutamatergic systems (eg, topiramate), each represented in 5 trials. Additional studies targeted the serotonergic system and GABAergic system independently, highlighting the diversity of neurochemical pathways explored in AUD pharmacotherapy development. The pharmacological targets studied in AUD trials are outlined in [Table 4](#).

Pharmacological and behavioral interventions were the most frequently tested therapeutic strategies. Placebos were used in nearly one-third of trials, and device-based interventions were comparatively rare. An overview of the types of therapeutic interventions investigated is provided in [Table 5](#).

Among the 209 AUD trials, Phase 2 was the most common ($n = 77$), followed by Phase 1 ($n = 27$), and Phase 4 ($n = 19$). Phase 3 trials were less frequent ($n = 15$), and early-phase studies accounted for just 4 trials. Notably, 88 studies did not report a specific phase, but the total count of clinical trial phases was still 229. This reflects the fact that some AUD studies are registered under multiple phases (eg, Phase 1 and Phase 2), resulting in more phase entries than the 209 unique trials analyzed. The distribution of clinical trial phases for AUD is provided in [Table 6](#).

Most trials were modest in size, enrolling fewer than 200 participants. Very few trials exceeded 500 enrollees, indicating a need for larger confirmatory studies in AUD. The distribution of participant numbers in AUD trials is shown in [Table 7](#).

Common outcome measures in AUD clinical trials primarily focused on behavioral and physiological indicators of alcohol use. Craving scores were frequently assessed using visual analog scales or standardized questionnaires. Percentage of drinking days and abstinence duration were typically recorded through self-report methods such as the

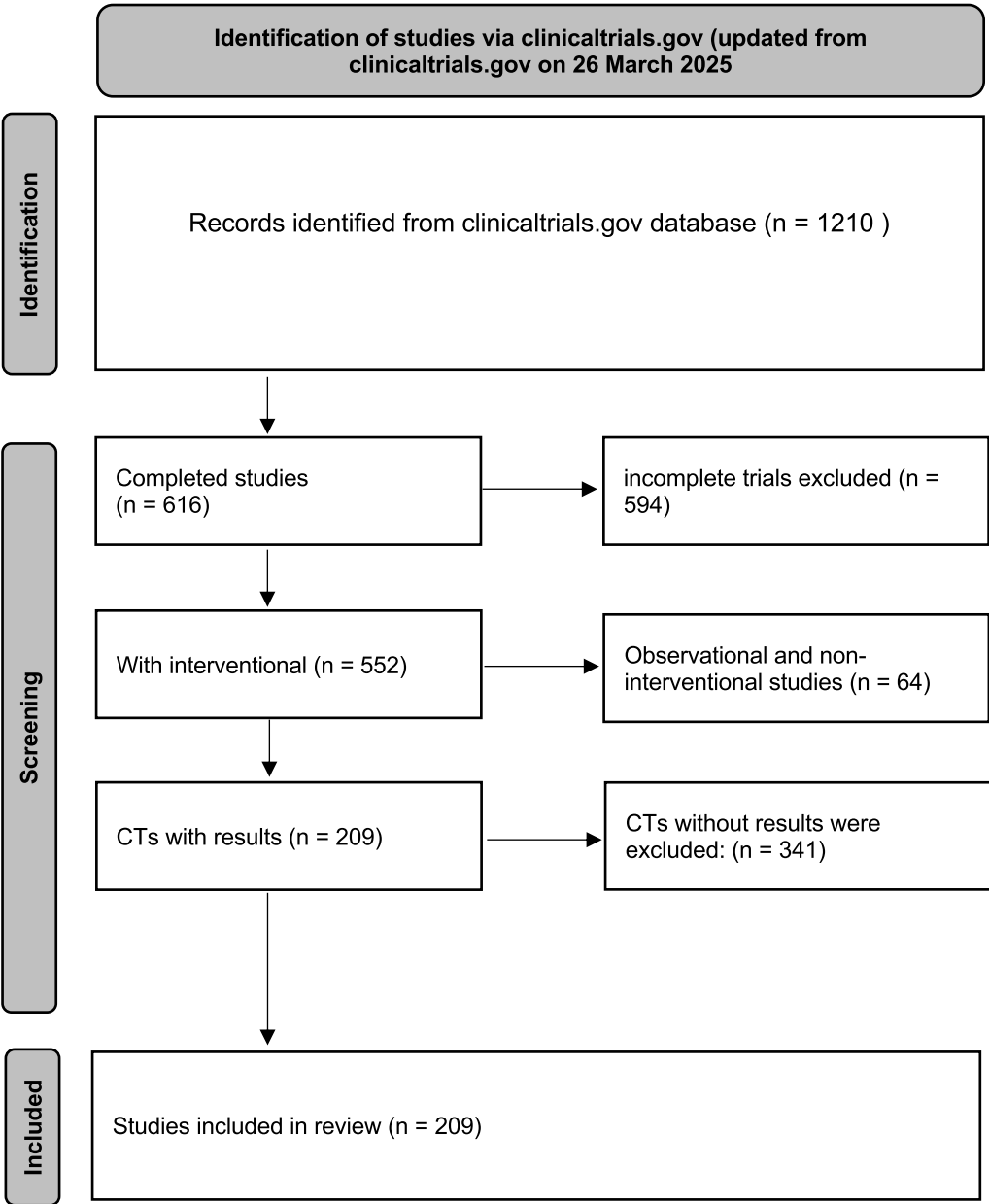


Figure 1 Prisma chart of clinical trials used for AUD.

Timeline Followback (TLFB). Relapse rates, often defined as time to first drink or binge, were another key outcome. Some studies also included liver function tests to evaluate physiological recovery. [Table 8](#) provides a brief description of the common trial outcome measures.

Table 2 Overview of Clinical Trials for AUD

Metric	Value
Total Studies	209
Study Type	Interventional (Clinical Trial)
Status Filter	Completed
Studies with Results Posted	Yes
Condition	Alcohol Use Disorder

Table 3 Age Distribution in AUD Clinical Trials

Age Group	Number of Trials
Adult, Older Adult	157
Adult only	37
Child, Adult	7
Child, Adult, Older Adult	7
Child only	1

Table 4 Pharmacological Target Systems Investigated in AUD Clinical Trials

Pharmacological Target System	Number of Trials
Other/Unclassified	170
Cholinergic (Nicotinic Receptor)	8
Opioid System	6
GABAergic/Glutamatergic System	5
GABAergic System	4
Serotonergic System	4
GABA-B Receptor	3
Alcohol Metabolism (Aversive Agent)	2
Glutamatergic System	2
Dopaminergic/Serotonergic System	2
Neuroimmune (PDE4 Inhibition)	1
Glutamatergic System (NMDA Antagonist)	1
Neurosteroid (GABAergic Modulation)	1

Table 5 Overview of the Types of Therapeutic Interventions Investigated with Number of Clinical Trials

Therapeutic Target	Count
Pharmacological	117
Behavioral	95
Device-Based	9
Other	7
Nutritional	1

Table 6 Distribution of Clinical Trial Phases for AUD

Trial Phase	Frequency (Est.)
Early Phase	4
Phase 1	27
Phase 2	77
Phase 3	15
Phase 4	19
Not Specified	88

Table 7 Distribution of Participant Numbers in AUD Trials

Participant Range	Number of Trials
<50	71
50–99	57
100–199	42
200–499	22
500–999	11
>1000	6

Table 8 Outcome Categories

Common Outcome Measures	Description
Craving Scores	Visual analog scale, questionnaires
Percent Drinking Days	TLFB or self-report
Relapse Rate	Time to first drink or binge
Liver Function Tests	For physiological improvement
Abstinence Duration	Often measured in weeks/months

Several pharmacologic agents were evaluated for their potential to reduce cravings, prevent relapse, or modulate underlying neurobiology in AUD. Table 9 provides a summary of commonly studied medications, their pharmacological classes, and the rationale for their inclusion in AUD clinical trials.

Box 1 highlights the behavioral interventions most frequently employed in phase 4 clinical trials for AUD, including stepped care management, medical management counseling, and cognitive behavioral therapy (CBT), among others. These strategies emphasize structured and scalable approaches suitable for real-world implementation. Their repeated use reflects their practical value in enhancing treatment engagement, medication adherence, and long-term recovery outcomes. The table provides concise descriptions to assist in understanding how each strategy contributes to AUD care.

Table 9 List of Medications Used in AUD Trials

Medication	Pharmacological Class	Rationale for Use in AUD
Naltrexone	Opioid antagonist	Reduces rewarding effects of alcohol by blocking opioid receptors. FDA-approved for AUD.
Acamprosate	NMDA receptor modulator	Restores glutamate balance to reduce cravings. FDA-approved for AUD.
Disulfiram	Aldehyde dehydrogenase inhibitor	Causes unpleasant reaction with alcohol; used as deterrent. FDA-approved.
Varenicline	Partial nicotinic receptor agonist	Modulates dopamine release, reduces craving and consumption.
Gabapentin	GABA analog	Reduces alcohol cravings, withdrawal symptoms, and anxiety. Off-label use in AUD.
Topiramate	Anticonvulsant	Modulates GABA and glutamate to reduce cravings and reward.
Ondansetron	5-HT ₃ antagonist	Targets early-onset AUD; modulates serotonin and dopamine.
Quetiapine	Atypical antipsychotic	Reduces anxiety, sleep disturbances, and cravings.
Baclofen	GABA-B receptor agonist	Reduces craving and withdrawal; especially in liver-impaired patients.
Aripiprazole	Atypical antipsychotic	Modulates dopamine; studied for relapse prevention.
Ivermectin	Antiparasitic, potential GABA modulator	Explored for neurological impact on craving and withdrawal.
Mifepristone	Glucocorticoid receptor antagonist	Targets stress systems implicated in alcohol dependence.
Pregnenolone	Neurosteroid	Modulates GABAergic activity; affects stress and craving.
Apremilast	PDE4 Inhibitor	Reduces alcohol intake through neuroinflammatory modulation.
Ketamine	NMDA receptor antagonist	Studied for rapid craving reduction and neuroplasticity enhancement.
ASP8062	GABA-B receptor positive allosteric modulator	Aimed at reducing craving and relapse.

Box 1 Summary of the Most Common Behavioral Treatment Strategies Used in Clinical Trials for Alcohol Use Disorder (AUD)

Strategy and Description
Stepped Care Management: Tiered treatment that escalates in intensity based on patient response.
Medical Management Counseling: Structured sessions to support medication adherence and monitor drinking.
Cognitive Behavioral Therapy (CBT/CBT4CBT): Focus on coping skills, craving control, and relapse prevention; available in digital formats.
Motivational Interviewing (MI): Client-centered counseling to resolve ambivalence and enhance motivation.
Family-Involvement Therapy: Involves family in treatment to improve support and accountability.
Trauma-Informed Approaches (eg, Seeking Safety, COPE): Designed for individuals with co-occurring trauma and AUD; integrates PTSD and addiction treatment.

Discussion

The findings of this study provide a comprehensive overview of the current pharmacological and behavioral therapeutic interventions for AUD, based on clinical trials registered on ClinicalTrials.gov up to March 2025. The dominance of pharmacological interventions targeting classical neurotransmitter systems, particularly GABAergic, glutamatergic, cholinergic, and opioid pathways, reflects the continued reliance on well-established neurobiological models of addiction. These clinical trends are largely consistent with insights from recent preclinical research, which emphasize the role of dysregulated neurotransmission, neuroinflammation, and stress circuitry in the development and persistence of AUD. For example, The GABAergic system has long been recognized for its role in alcohol's sedative and anxiolytic effects.^{68,69}

Several clinical trials in the dataset explored agents acting on GABA_B receptors (eg, baclofen and ASP8062) and neurosteroids with GABA-modulating properties (eg, pregnenolone).^{70–73} These align with preclinical studies showing that enhancing GABA_B signaling can reduce alcohol consumption, mitigate withdrawal symptoms, and decrease relapse likelihood by restoring the inhibitory tone in key brain circuits such as the amygdala and prefrontal cortex.⁶⁸ Likewise, the use of topiramate and gabapentin in trials reflects an interest in dual-action medications that modulate both GABAergic and glutamatergic systems to rebalance neural excitability disrupted by chronic alcohol exposure.^{74–76} The glutamatergic system's involvement in relapse and craving is further supported by clinical interest in both acamprosate and ketamine. Acamprosate, which is already FDA-approved, helps stabilize glutamate levels during abstinence.^{77,78} In contrast, ketamine is still under investigation but shows promise in preclinical and early human studies for rapidly reducing cravings and enhancing neuroplasticity.^{79–81} These agents are directly linked to mechanisms described in recent translational research that focuses on reversing synaptic alterations and the excitotoxicity driven by alcohol dependence.

Another significant therapeutic target highlighted in both the clinical and preclinical domains is the opioid system. Naltrexone, one of the most frequently studied agents in the dataset, continues to be central to reducing alcohol reward through opioid receptor antagonism. The consistency of naltrexone's application in clinical trials underscores its robust evidence base and supports the concept, reinforced by animal models, that modulating endogenous opioid signaling is critical for managing alcohol-seeking behavior.^{82,83}

Interestingly, the review also reveals increasing clinical interest in the cholinergic system, particularly through trials involving varenicline.^{84–86} Acting on nicotinic acetylcholine receptors (nAChRs), varenicline is believed to reduce alcohol consumption by modulating dopamine release in mesolimbic reward pathways. This trend is consistent with preclinical studies suggesting that the cholinergic system plays a more significant role in AUD than previously appreciated, particularly through its influence on impulsivity and reward valuation.^{87,88}

Furthermore, the analysis identifies an emerging emphasis on novel therapeutic targets, especially neuroimmune and stress-related targets. Apremilast and mifepristone are being evaluated for their potential to reduce alcohol intake via modulation of neuroinflammatory pathways and hypothalamic pituitary adrenal axis activity.^{89–92} These targets mirror the evolving preclinical understanding that AUD is not solely a disorder of reward circuitry but also involves systemic inflammation, stress dysregulation, and neuroendocrine disruption. For instance, increased cytokine levels and immune activation have been observed in individuals with AUD, and animal studies have shown that reducing neuroinflammation can attenuate alcohol-seeking behavior.^{93–95} Moreover, neuromodulation techniques such as repetitive transcranial magnetic stimulation represent a promising area for further studies.^{96–98} Preclinical findings support the modulation of prefrontal-striatal circuits as a means of reducing impulsivity and compulsivity, which are core behavioral components of addiction.^{99–102} Combining such techniques with pharmacological or behavioral treatments could provide synergistic benefits, and future trials may increasingly adopt these multimodal strategies.

Behavioral interventions remain integral, with almost 82 trials in the dataset incorporating psychological therapies. Their frequent combination with pharmacological agents underscores the real-world recognition that AUD is best treated through a biopsychosocial model.^{103–105} CBT, motivational interviewing, and contingency management are known to address cognitive-behavioral triggers and support long-term abstinence, particularly when combined with medications that reduce physiological craving or enhance mood regulation.^{106–108}

Despite these promising directions, several challenges persist. The majority of studies enrolled fewer than 200 participants, limiting statistical power and generalizability. Additionally, a large proportion of trials were early or mid-phase, with fewer progressing to Phase 3 validation. The heterogeneity in study design, outcome measures (eg, craving scores and abstinence duration), and target populations (eg, limited inclusion of adolescents or older adults) also hampers direct comparison and meta-analysis. These gaps highlight the need for harmonized, large-scale trials using standardized endpoints and stratified recruitment criteria.

Conclusion

The clinical trial landscape for AUD highlights a strong emphasis on targeting dysregulated neurotransmitter systems, including GABAergic, glutamatergic, dopaminergic, serotonergic, cholinergic, and opioid pathways. These neurochemical disruptions underlie key aspects of addiction behavior such as craving, relapse, and withdrawal, supporting the

continued development of pharmacological interventions. Emerging therapies focused on neuroinflammation, stress-response systems, and neuromodulation also showed promise as novel strategies. Many interventions have demonstrated modest efficacy in reducing cravings and promoting abstinence; however, most trials are limited by small sample sizes and early-phase designs. To advance the field, there is a pressing need for large-scale, rigorous clinical trials that adopt standardized outcome measures. Combining pharmacological, behavioral, and neuromodulatory approaches may offer the most effective path toward sustained recovery in individuals with AUD.

Future Directions and Recommendations

Future research on AUD should prioritize large, multicenter trials with standardized outcome measures to improve comparability and evidence synthesis. Greater inclusion of underrepresented populations such as adolescents, older adults, and those with comorbidities is essential to enhance generalizability. Promising therapies targeting neuroimmune and neuromodulatory pathways should be evaluated in long-term, well-powered studies. Combining pharmacological, behavioral, and neuromodulatory approaches may offer enhanced treatment efficacy. Incorporating real-world data and digital tools could further optimize intervention delivery and patient outcomes.

Disclosure

The author reports no conflicts of interest in this work.

References

1. Alcohol Use Disorder (AUD) in the United States: Age Groups and Demographic Characteristics. National Institute on Alcohol Abuse and Alcoholism (NIAAA). Available from: <https://www.niaaa.nih.gov/alcohols-effects-health/alcohol-topics/alcohol-facts-and-statistics/alcohol-use-disorder-aud-united-states-age-groups-and-demographic-characteristics>. Accessed March 17, 2025.
2. Huang H, Shen H, Ning K, et al. Quality of life and its correlates in alcohol use disorder patients with and without depression in China. *Front Psychiatry*. 2021;11:627338. doi:10.3389/FPSYT.2020.627338/BIBTEX
3. Dayal P, Kaloiya GS. Quality of life in alcohol use disorder: exploration of predictive factors in a cross-sectional study. *Psych Int*. 2024;5:101–120. doi:10.3390/PSYCHIATRYINT5010008
4. Stickel F, Datz C, Hampe J, Bataller R. Pathophysiology and management of alcoholic liver disease: update 2016. *Gut Liver*. 2017;11:173. doi:10.5009/GNL16477
5. Kim Y, Hack LM, Ahn ES, Kim J. Practical outpatient pharmacotherapy for alcohol use disorder. *Drugs Context*. 2018;7:212308. doi:10.7573/DIC.212308
6. Nehring SM, Chen RJ, Freeman AM. Alcohol use disorder. *Instant Wisdom for Gps: Pearls From All the Specialties: Second Edition*. 2024;1–8.
7. Cook S, Saxena S, Mathur R, et al. Diabetes complications in people with alcohol use disorder and type 2 diabetes. *BJGP Open*. 2025. doi:10.3399/BJGPO.2024.0133
8. Wackernah RC, Minnick MJ, Clapp P. Alcohol use disorder: pathophysiology, effects, and pharmacologic options for treatment. *Subst Abuse Rehabil*. 2014;5(1). doi:10.2147/SAR.S37907
9. Sung C, Chung CH, Lin FH, et al. Risk of cardiovascular disease in patients with alcohol use disorder: a population-based retrospective cohort study. *PLoS One*. 2022;17:e0276690. doi:10.1371/JOURNAL.PONE.0276690
10. Zhou H, Vasilio V. Alcohol use and use disorder and cancer risk: perspective on causal inference. *Complex Psych*. 2022;8(1–2):9. doi:10.1159/000526407
11. Yokoyama A, Omori T. Genetic polymorphisms of alcohol and aldehyde dehydrogenases and risk for esophageal and head and neck cancers. *Alcohol*. 2005;35:175–185. doi:10.1016/j.alcohol.2005.04.003
12. Lai JH, Han S, Park C, Avanceña ALV. Association between alcohol use disorder and hospital readmission rates and outcomes in cancer survivors: a population cohort study. *J Natl Compr Canc Netw*. 2024;22. doi:10.6004/JNCCN.2023.7076.
13. Bouchery EE, Harwood HJ, Sacks JJ, Simon CJ, Brewer RD. Economic costs of excessive alcohol consumption in the U.S. 2006. *Am J Prev Med*. 2011;41:516–524. doi:10.1016/j.amepre.2011.06.045
14. Thavorncharoensap M, Teerawattananon Y, Yothasamut J, Lertpitakpong C, Chaikledkaew U. The economic impact of alcohol consumption: a systematic review. *Subst Abuse Treat Prev Policy*. 2009;4:1–11. doi:10.1186/1747-597X-4-20/TABLES/7
15. Costin BN, Miles MF. Molecular and neurologic responses to chronic alcohol use. *Handb Clin Neurol*. 2014;125:157–171. doi:10.1016/B978-0-444-62619-6.00010-0
16. Mason BJ. Emerging pharmacotherapies for alcohol use disorder. *Neuropharmacology*. 2017;122:244–253. doi:10.1016/J.NEUROPHARM.2017.04.032
17. Seo D, Sinha R. Neuroplasticity and predictors of alcohol recovery. *Alcohol Res*. 2015;37:143.
18. Rossi DJ, Richardson BD. The cerebellar GABAAR system as a potential target for treating alcohol use disorder. *Handb Exp Pharmacol*. 2018;248:113–156. doi:10.1007/164_2018_109/FIGURES/3
19. Hartwell EE, Kranzler HR. Pharmacogenetics of alcohol use disorder treatments: an update. *Expert Opin Drug Metab Toxicol*. 2019;15:553–564. doi:10.1080/17425255.2019.1628218
20. Sachs BD, Dodson K. Serotonin deficiency and alcohol use disorders. *Addictive Substances Neurol Dis*. 2017;181–189. doi:10.1016/B978-0-12-805373-7.00018-9

21. Govindaiah G, Cox CL. Metabotropic glutamate receptors differentially regulate GABAergic inhibition in thalamus. *J Neurosci*. 2006;26:13443–13453. doi:10.1523/JNEUROSCI.3578-06.2006
22. Davis KM, Wu JY. Role of glutamatergic and GABAergic systems in alcoholism. *J Biomed Sci*. 2001;8:7–19. doi:10.1159/000054008
23. Rao PSS, Bell RL, Engleman EA, Sari Y. Targeting glutamate uptake to treat alcohol use disorders. *Front Neurosci*. 2015;9:104167. doi:10.3389/FNINS.2015.00144/XML/NLM
24. Engel JA, Jerlhag EA. Mechanisms along the mesolimbic dopamine system. *Prog Brain Res*. 2014;211:201–233. doi:10.1016/B978-0-444-63425-2.00009-X
25. Adinoff B. Neurobiologic processes in drug reward and addiction. *Harv Rev Psychiatry*. 2004;12:305. doi:10.1080/10673220490910844
26. Dahchour A, Ward RJ. Changes in serotonin neurotransmission as assayed by microdialysis after acute, intermittent or chronic ethanol administration and withdrawal. *Fundam Clin Pharmacol*. 2024;38:42–59. doi:10.1111/FCP.12949;PAGE:STRING:ARTICLE/CHAPTER
27. McBride WJ. Role of serotonin in brain reward and regulation of alcohol drinking behavior. *Handb Behav Neurosci*. 2010;21:399–414. doi:10.1016/S1569-7339(10)70092-8
28. Witkiewitz K, Litten RZ, Leggio L. Advances in the science and treatment of alcohol use disorder. *Sci Adv*. 2019;5. doi:10.1126/SCIADV.AAX4043/ASSET/24EE4B9D-EF9F-41B7-8230-EB6538155170/ASSETS/GRAPHIC/AAX4043-F1.JPEG
29. Seneviratne C, Johnson BA. Advances in medications and tailoring treatment for alcohol use disorder. *Alcohol Res*. 2015;37:15.
30. Medina KL, McQueeney T, Nagel BJ, Hanson KL, Schweinsburg AD, Tapert SF. Prefrontal cortex volumes in adolescents with alcohol use disorders: unique gender effects. *Alcohol Clin Exp Res*. 2008;32:386. doi:10.1111/J.1530-0277.2007.00602.X
31. Varodayan FP, Patel RR, Matzeu A, et al. The amygdala noradrenergic system is compromised with alcohol use disorder. *Biol Psychiatry*. 2022;91:1008–1018. doi:10.1016/J.BIOPSYCH.2022.02.006
32. Tomasi D, Wiers CE, Manza P, et al. Accelerated aging of the amygdala in alcohol use disorders: relevance to the dark side of addiction. *Cerebral Cortex*. 2021;31:3254. doi:10.1093/CERCOR/BHAB006
33. Melkumyan M, Randall PA, Silberman Y. Central amygdala neuroimmune signaling in alcohol use disorder. *Addiction Neurosci*. 2025;14:100194. doi:10.1016/J.ADDICN.2024.100194
34. Crane NA, Gorka SM, Weaver J, Langenecker SA, de Wit H, Luan Phan K. Preliminary evidence for disrupted nucleus accumbens reactivity and connectivity to reward in binge drinkers. *Alcohol Alcoholism*. 2017;52:647–654. doi:10.1093/ALCALC/AGX062
35. Bracht T, Soravia L, Moggi F, et al. The role of the orbitofrontal cortex and the nucleus accumbens for craving in alcohol use disorder. *Transl Psych*. 2021;11:1–10. doi:10.1038/s41398-021-01384-w
36. Ho AL, Salib AMN, Pendharkar AV, Sussman ES, Giardino WJ, Halpern CH. The nucleus accumbens and alcoholism: a target for deep brain stimulation. *Neurosurg Focus*. 2018;45. doi:10.3171/2018.5.FOCUS18157.
37. Yoon EJ, Choi JS, Kim H, et al. Altered hippocampal volume and functional connectivity in males with internet gaming disorder comparing to those with alcohol use disorder. *Sci Rep*. 2017;7:1–12. doi:10.1038/s41598-017-06057-7
38. Nickell CG, Thompson KR, Pauly JR, Nixon K. Recovery of hippocampal-dependent learning despite blunting reactive adult neurogenesis after alcohol dependence. *Brain Plast*. 2020;6:83. doi:10.3233/BPL-200108
39. Nagel BJ, Schweinsburg AD, Phan V, Tapert SF. Reduced hippocampal volume among adolescents with alcohol use disorders without psychiatric comorbidity. *Psychiatry Res*. 2005;139:181. doi:10.1016/J.PSYCHRESNS.2005.05.008
40. Ray LA, Meredith LR, Kiluk BD, Walthers J, Carroll KM, Magill M. Combined pharmacotherapy and cognitive behavioral therapy for adults with alcohol or substance use disorders: a systematic review and meta-analysis. *JAMA Network Open*. 2020;3:e208279–e208279. doi:10.1001/JAMANETWORKOPEN.2020.8279
41. Burnette EM, Nieto SJ, Grodin EN, et al. Novel agents for the pharmacological treatment of alcohol use disorder. *Drugs*. 2022;82:251–274. doi:10.1007/S40265-021-01670-3/TABLES/2
42. Ralevski E, Olivera-Figueroa LA, Petrakis I, Lening R, Olivera-Figueroa Ismene Petrakis A. PTSD and comorbid AUD: a review of pharmacological and alternative treatment options. *Subst Abuse Rehabil*. 2014. doi:10.2147/SAR.S37399
43. Agabio R, Composera A, Saulle R, Krupchanka D, Leggio L, Minozzi S. Combined pharmacological and psychosocial interventions for alcohol use disorder. *Cochrane Database Syst Rev*. 2023;2023:CD015673. doi:10.1002/14651858.CD015673
44. Antonelli M, Sestito L, Tarli C, Addolorato G. Perspectives on the pharmacological management of alcohol use disorder: are the approved medications effective? *Eur J Intern Med*. 2022;103:13–22. doi:10.1016/J.EJIM.2022.05.016
45. Anversa RG, Barron ML, Walker LC, Lawrence AJ. Emerging GPCR targets for AUD: insights from preclinical studies. *Curr Opin Neurobiol*. 2024;87:102896. doi:10.1016/J.CONB.2024.102896
46. Yaseen W, Mong J, Zipursky J. Sobering perspectives on the treatment of alcohol use disorder. *JAMA Network Open*. 2024;7:e243340–e243340. doi:10.1001/JAMANETWORKOPEN.2024.3340
47. McCrady BS, Epstein EE, Fokas KF. Treatment interventions for women with alcohol use disorder. *Alcohol Res*. 2020;40:08. doi:10.35946/ARCR.V40.2.08
48. Sawicka M, Tracy DK. Naltrexone efficacy in treating alcohol-use disorder in individuals with comorbid psychosis: a systematic review. *Ther Adv Psychopharmacol*. 2017;7:211–224. doi:10.1177/2045125317709975/ASSET/6A2BA138-279A-42FF-8401-218E6436092B/ASSETS/IMAGES/LARGE/10.1177_2045125317709975-FIG1.JPG
49. Avery J. Naltrexone and alcohol use. *Am J Psychiatry*. 2022;179:886–887. doi:10.1176/APPI.AJP.20220821
50. Lanz J, Biniarz-Harris N, Kuvaldina M, Jain S, Lewis K, Fallon BA. Disulfiram: mechanisms, applications, and challenges. *Antibiotics*. 2023;12:524. doi:10.3390/ANTIBIOTICS12030524/S1
51. Jørgensen CH, Pedersen B, Tønnesen H. The efficacy of disulfiram for the treatment of alcohol use disorder. *Alcohol Clin Exp Res*. 2011;35:1749–1758. doi:10.1111/J.1530-0277.2011.01523.X
52. Plosker GL. Acamprostate: a review of its use in alcohol dependence. *Drugs*. 2015;75:1255–1268. doi:10.1007/S40265-015-0423-9/FIGURES/1
53. McPheeters M, O'Connor EA, Riley S, et al. Pharmacotherapy for alcohol use disorder: a systematic review and meta-analysis. *JAMA*. 2023;330:1653–1665. doi:10.1001/JAMA.2023.19761
54. Scheen AJ. Glucagon-like peptide-1 receptor agonists and alcohol use disorders: an emerging unexpected beneficial effect. *Diabetes Obes Metab*. 2025. doi:10.1111/DOM.16453;CSUBTYPE:STRING:AHEAD
55. Donnelly A. Repurposing semaglutide to treat alcohol use disorder: drug repurposing. *Nature Mental Health*. 2025;3:274.

56. Chuong V, Farokhnia M, Khom S, et al. The glucagon-like peptide-1 (GLP-1) analogue semaglutide reduces alcohol drinking and modulates central GABA neurotransmission. *JCI Insight*. 2023;8:e170671. doi:10.1172/JCI.INSIGHT.170671
57. Quddos F, Hubshman Z, Tegge A, et al. Semaglutide and tirzepatide reduce alcohol consumption in individuals with obesity. *Sci Rep*. 2023;13:1–12.
58. Sessa B. Why MDMA therapy for alcohol use disorder? And why now? *Neuropharmacology*. 2018;142:83–88. doi:10.1016/J.NEUROPHARM.2017.11.004
59. Venugopal KL, Bedri NA. The therapeutic potential of psilocybin in alcohol use disorder recovery: a literature review. *Undergraduate Res Nat Clin Sci Technol J*. 2023;7:1–13. doi:10.26685/URNCT.420
60. O'Donnell KC, Mennenga SE, Owens LT, et al. Psilocybin for alcohol use disorder: rationale and design considerations for a randomized controlled trial. *Contemp Clin Trials*. 2022;123:106976. doi:10.1016/J.CCT.2022.106976
61. Srivastava AB, Wai JM. The future of alcohol use disorder treatment and research. In: *From Stigma to Support*. Springer. 2024:189–203.
62. Celik M, Gold MS, Fuehrlein B. A narrative review of current and emerging trends in the treatment of alcohol use disorder. *Brain Sci*. 2024;14:294. doi:10.3390/BRANS114030294
63. Klimas J, Field CA, Cullen W, et al. Psychosocial interventions to reduce alcohol consumption in concurrent problem alcohol and illicit drug users: Cochrane Review. *Syst Rev*. 2013;2:3. doi:10.1186/2046-4053-2-3/TABLES/5
64. Klimas J, Fairgrieve C, Tobin H, et al. Psychosocial interventions to reduce alcohol consumption in concurrent problem alcohol and illicit drug users. *Cochrane Database Syst Rev*. 2018;2018. doi:10.1002/14651858.CD009269.PUB4/MEDIA/CDSR/CD009269/IMAGE_N/CD009269-CMP-003-03.PNG
65. Kelly JF, Humphreys K, Ferri M. Alcoholics anonymous and other 12-step programs for alcohol use disorder. *Cochrane Database Syst Rev*. 2020;2020. doi:10.1002/14651858.CD012880.PUB2/MEDIA/CDSR/CD012880/IMAGE_N/CD012880-CMP-001.02.SVG.
66. Coriale G, De Rosa F, Battagliese G, et al. Motivational enhancement therapy versus cognitive behavioral therapy in a cohort of men and women with alcohol use disorder. *Biomed Rev*. 2019;30:125–135. doi:10.14748/BMR.V30.6393
67. Laaksonen E, Vuoristo-Myllys S, Koski-Jannes A, Alho H. Combining medical treatment and CBT in treating alcohol-dependent patients: effects on life quality and general well-being. *Alcohol Alcoholism*. 2013;48:687–693. doi:10.1093/ALCALC/AGT053
68. Koob GF. A role for GABA mechanisms in the motivational effects of alcohol. *Biochem Pharmacol*. 2004;68:1515–1525. doi:10.1016/J.BCP.2004.07.031
69. Liang J, Olsen RW. Alcohol use disorders and current pharmacological therapies: the role of GABAA receptors. *Acta Pharmacol Sin*. 2014;35:981–993. doi:10.1038/aps.2014.50
70. Gao H, Sinha R, Wemm S, Milivojevic V. Pregnenolone effects on parasympathetic response to stress and alcohol cue provocation in treatment-seeking individuals with alcohol use disorder. *Alcohol Clin Exp Res*. 2025;49. doi:10.1111/ACER.15529.
71. Milivojevic V, Sullivan L, Tiber J, et al. Pregnenolone effects on provoked alcohol craving, anxiety, HPA axis, and autonomic arousal in individuals with alcohol use disorder. *Psychopharmacology*. 2022;240:101. doi:10.1007/S00213-022-06278-3
72. Schacht JP, Ray LA, Miranda R, et al. Effects of a novel GABA-B positive allosteric modulator, ASP8062, on alcohol cue-elicited craving and naturalistic alcohol consumption in a multisite randomized, double-blind, placebo-controlled trial. *Alcohol Clin Exp Res*. 2024;48. doi:10.1111/ACER.15468
73. Farokhnia M, Schwandt ML, Lee MR, et al. Biobehavioral effects of baclofen in anxious alcohol-dependent individuals: a randomized, double-blind, placebo-controlled, laboratory study. *Transl Psychiatry*. 2017;7:e1108. doi:10.1038/TP.2017.71
74. Anton RF, Latham P, Voronin K, et al. Efficacy of gabapentin for the treatment of alcohol use disorder in patients with alcohol withdrawal symptoms: a randomized clinical trial. *JAMA Intern Med*. 2020;180:728–736. doi:10.1001/JAMAINTERNMED.2020.0249
75. Pennington DL, Bielenberg J, Lasher B, et al. A randomized pilot trial of topiramate for alcohol use disorder in veterans with traumatic brain injury: effects on alcohol use, cognition, and post-concussive symptoms. *Drug Alcohol Depend*. 2020;214:108149. doi:10.1016/J.DRUGALCDEP.2020.108149
76. Kranzler HR, Feinn R, Pond T, et al. Post-treatment effects of topiramate on alcohol-related outcomes: a combined analysis of two placebo-controlled trials. *Addict Biol*. 2022;27:e13130. doi:10.1111/ADB.13130;WGROU:STRING:PUBLICATON
77. Kalk NJ, Lingford-Hughes AR. The clinical pharmacology of acamprosate. *Br J Clin Pharmacol*. 2014;77:315. doi:10.1111/BCP.12070
78. Frye MA, Hinton DJ, Karpayak VM, et al. Anterior cingulate glutamate is reduced by acamprosate treatment in patients with alcohol dependence. *J Clin Psychopharmacol*. 2016;36:669. doi:10.1097/JCP.0000000000000590
79. Krystal JH, Kavalali ET, Monteggia LM. Ketamine and rapid antidepressant action: new treatments and novel synaptic signaling mechanisms. *Neuropsychopharmacology*. 2023;49:41. doi:10.1038/S41386-023-01629-W
80. Hilal F, Jeanblanc J, Naassila M. Intérêt et Mécanismes d'action de La Kétamine Dans Le Traitement de l'addiction à l'alcool – revue Des Études Cliniques et Précliniques. *Biol Aujourd'hui*. 2023;217:161–182. doi:10.1051/JBIO/2023028
81. Wu H, Savalia NK, Kwan AC. Ketamine for a boost of neural plasticity: how, but also when? *Biol Psychiatry*. 2021;89:1030. doi:10.1016/J.BIOPSYCH.2021.03.014
82. Santos GM, Ikeda J, Coffin P, et al. Targeted oral naltrexone for mild to moderate alcohol use disorder among sexual and gender minority men: a randomized trial. *Am J Psychiatry*. 2022;179:915. doi:10.1176/APPI.AJP.20220335
83. Simpson TL, Achtmeyer C, Batten L, et al. Naltrexone augmented with prazosin for alcohol use disorder: results from a randomized controlled proof-of-concept trial. *Alcohol Alcoholism*. 2024;59. doi:10.1093/ALCALC/AGAE062.
84. Litten RZ, Ryan ML, Fertig JB, et al. A double-blind, placebo-controlled trial assessing the efficacy of varenicline tartrate for alcohol dependence. *J Addict Med*. 2013;7:277. doi:10.1097/ADM.0B013E31829623F4
85. Phimarn W, Sakhancord R, Paitoon P, Saramunee K, Sungthong B. Efficacy of varenicline in the treatment of alcohol dependence: an updated meta-analysis and meta-regression. *Int J Environ Res Public Health*. 2023;20:4091. doi:10.3390/IJERPH20054091/S1
86. Bold KW, Zweben A, Fucito LM, et al. Longitudinal findings from a randomized clinical trial of varenicline for alcohol use disorder with comorbid cigarette smoking. *Alcohol Clin Exp Res*. 2019;43:937. doi:10.1111/ACER.13994
87. Peltier MKR, Sofuoglu M. Pharmacological cognitive enhancers. *Cogn Addiction*. 2020;303–320. doi:10.1016/B978-0-12-815298-0.00023-X
88. Söderpalm B, Danielsson K, de Bejczy A, Ademark L, Ericson M. Combined administration of varenicline and bupropion produces additive effects on accumbal dopamine and abolishes the alcohol deprivation effect in rats. *Addict Biol*. 2020;25. doi:10.1111/ADB.12807.

89. Grigsby KB, Mangieri RA, Roberts AJ, et al. Preclinical and clinical evidence for suppression of alcohol intake by apremilast. *J Clin Invest.* **2023**;133:e159103. doi:10.1172/JCI159103
90. Orio L, Alen F, Pavón FJ, Serrano A, García-Bueno BO. Neuroinflammation, and alcohol abuse. *Front Mol Neurosci.* **2019**;11:427528. doi:10.3389/FNMOL.2018.00490/XML/NLM
91. Sharrett-Field L, Butler TR, Berry JN, Reynolds AR, Prendergast MA. Mifepristone pretreatment reduces ethanol withdrawal severity in vivo. *Alcohol Clin Exp Res.* **2013**;37:1417. doi:10.1111/ACER.12093
92. Vozella V, Borgonetti V, Cruz B, et al. Apremilast reduces co-occurring alcohol drinking and mechanical allodynia and regulates central amygdala GABAergic transmission. *JCI Insight.* **2025**;10. doi:10.1172/JCI.INSIGHT.189732
93. Li Z, Vidjro OE, Guo G, et al. NLRP3 deficiency decreases alcohol intake controlling anxiety-like behavior via modification of glutamatergic transmission in corticostriatal circuits. *J Neuroinflammation.* **2022**;19:1–18. doi:10.1186/S12974-022-02666-W/FIGURES/8
94. Kelley KW, Dantzer R. Alcoholism and inflammation: neuroimmunology of behavioral and mood disorders. *Brain Behav Immun.* **2010**;25:S13. doi:10.1016/J.BBI.2010.12.013
95. Erickson EK, Grantham EK, Warden AS, Harris RA. Neuroimmune signaling in alcohol use disorder. *Pharmacol Biochem Behav.* **2018**;177:34. doi:10.1016/J.PBB.2018.12.007
96. Petit B, Soudry-Faure A, Jeanjean L, et al. Efficacy of repetitive transcranial magnetic stimulation (RTMS) for reducing consumption in patients with alcohol use disorders (ALCOSTIM): study protocol for a randomized controlled trial. *Trials.* **2022**;23:1–10. doi:10.1186/S13063-021-05940-Z/FIGURES/2
97. Belgers M, Van Eijndhoven P, Markus W, Schene AH, Schellekens A. RTMS reduces craving and alcohol use in patients with alcohol use disorder: results of a randomized, sham-controlled clinical trial. *J Clin Med.* **2022**;11:951. doi:10.3390/JCM11040951/S1
98. Jansen JM, Van Den Heuvel OA, Van Der Werf YD, et al. The effect of high-frequency repetitive transcranial magnetic stimulation on emotion processing, reappraisal, and craving in alcohol use disorder patients and healthy controls: a functional magnetic resonance imaging study. *Front Psychiatry.* **2019**;10:440397. doi:10.3389/FPSYT.2019.00272/BIBTEX
99. Morein-Zamir S, Robbins TW. Fronto-striatal circuits in response-inhibition: relevance to addiction. *Brain Res.* **2015**;1628:117. doi:10.1016/J.BRAINRES.2014.09.012
100. McCalley DM, Kaur N, Wolf JP, et al. Medial prefrontal cortex theta burst stimulation improves treatment outcomes in alcohol use disorder: a double-blind, sham-controlled neuroimaging study. *Biol Psych Global Open Sci.* **2023**;3:301–310. doi:10.1016/J.BPSGOS.2022.03.002
101. Jaramillo AA, Randall PA, Stewart S, Fortino B, Van Voorhies K, Besheer J. Functional role for cortical-striatal circuitry in modulating alcohol self-administration. *Neuropharmacology.* **2018**;130:42–53. doi:10.1016/J.NEUROPHARM.2017.11.035
102. Blaine SK, Wemm S, Fogelman N, et al. Association of prefrontal-striatal functional pathology with alcohol abstinence days at treatment initiation and heavy drinking after treatment initiation. *Am J Psychiatry.* **2020**;177:1048–1059. doi:10.1176/APPI.AJP.2020.19070703
103. Hallihan H, Srimoragot M, Ma J, et al. Integrated behavioral interventions for adults with alcohol use disorder: a systematic review. *Drug Alcohol Depend.* **2024**;263:111406. doi:10.1016/J.DRUGALCDEP.2024.111406
104. Flanagan JC, Jones JL, Jarnecke AM, Back SE. Behavioral treatments for alcohol use disorder and post-traumatic stress disorder. *Alcohol Res.* **2018**;39:181.
105. Megherbi-Moulay O, Igier V, Julian B, Franchitto N, Sordes F. Alcohol use in older adults: a systematic review of biopsychosocial factors, screening tools, and treatment options. *Int J Ment Health Addict.* **2022**. doi:10.1007/S11469-022-00974-Z
106. Magill M, Ray L, Kiluk B, et al. A meta-analysis of cognitive-behavioral therapy for alcohol or other drug use disorders: treatment efficacy by contrast condition. *J Consult Clin Psychol.* **2019**;87:1093–1105. doi:10.1037/CCP0000447
107. Ray LA, Bujarski S, Grodin E, et al. State-of-the-art behavioral and pharmacological treatments for alcohol use disorder. *Am J Drug Alcohol Abuse.* **2019**;45:124–140. doi:10.1080/00952990.2018.1528265;PAGE:STRING:ARTICLE/CHAPTER
108. McHugh RK, Hearon BA, Otto MW. Cognitive-behavioral therapy for substance use disorders. *Psychiatr Clin North Am.* **2010**;33:511. doi:10.1016/J.PSC.2010.04.012

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