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+15878858911
editorial-office@sciformat.ca

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GLP-1 RECEPTOR AGONISTS AND ALCOHOL USE DISORDER: A NARRATIVE REVIEW OF THERAPEUTIC POTENTIAL AND WELL-BEING OUTCOMES

Nina Polek *(Corresponding Author, Email: nina.polek00@gmail.com)*
Cardinal Stefan Wyszynski University in Warsaw, Warsaw
ORCID ID: 0009-0000-3233-5866

Małgorzata Sikorska
Nowodworskie Centrum Medyczne (Nowy Dwór Medical Center), ul. Miodowa 2, 05-100 Nowy Dwór Mazowiecki
ORCID ID: 0009-0006-3768-7338

Patrycja Mularczyk
Nowodworskie Centrum Medyczne (Nowy Dwór Medical Center), ul. Miodowa 2, 05-100 Nowy Dwór Mazowiecki
ORCID ID: 0009-0000-1733-7185

Wojciech Łysoniewski
Szpital Specjalistyczny im. F. Ceynowy w Wejherowie, ul. dr. A. Jagalskiego 10, 84–200 Wejherowo
ORCID ID: 0009-0006-4850-6379

Artur Marcysiak
Mazowiecki Szpital Wojewódzki im. św. Jana Pawła II w Siedlcach, ul. Poniatowskiego 26, 08-110 Siedlce
ORCID ID: 0009-0002-8801-3836

Tomasz Mruzek
UCK WUM Centralny Szpital Kliniczny, ul. Banacha 1a, 02-097 Warszawa
ORCID ID: 0009-0002-3800-0172

Magdalena Kiełbasiewicz
Nowodworskie Centrum Medyczne (Nowy Dwór Medical Center), ul. Miodowa 2, 05-100 Nowy Dwór Mazowiecki
ORCID ID: 0009-0003-7055-0518

Karolina Zawadzka
Nowy Dwór Medical Center, ul. Miodowa 2, 05-100 Nowy Dwór Mazowiecki
ORCID ID: 0009-0005-8304-4993

ABSTRACT

Alcohol use disorder (AUD) remains a major global health challenge associated with substantial morbidity, mortality, and impaired well-being, while currently approved pharmacotherapies offer only modest and heterogeneous benefits. This narrative review examines the therapeutic potential of glucagon-like peptide-1 receptor agonists (GLP-1RAs) in AUD, with particular attention to their neurobiological rationale, emerging clinical relevance, and broader implications for health and well-being. A structured narrative search of PubMed and Google Scholar, supplemented by reference screening, identified thirty-one relevant publications spanning preclinical studies, human observational analyses, randomized controlled trials, Mendelian randomization research, and review papers. Across preclinical models, GLP-1 receptor activation consistently reduced alcohol intake, reward-related responding, and relapse-like behaviour, supporting a mechanistic role for GLP-1 signalling in the modulation of mesolimbic reward pathways, gut–brain communication, and the interface between metabolic and motivational processes. Early human studies provide a promising but still inconclusive translational signal, with some trials reporting reductions in craving and selected drinking outcomes, while pooled randomized evidence remains limited and heterogeneous. Observational findings further suggest associations between GLP-1RA use and reduced alcohol-related events, although causal inference remains restricted. Overall, GLP-1RAs represent a biologically plausible and clinically promising, but still investigational, approach to AUD. Their potential to integrate metabolic and neurobehavioral dimensions of care may be especially relevant for improving long-term health outcomes, although larger and more rigorous trials are needed to define their efficacy, safety, and place in treatment.

KEYWORDS

Alcohol Use Disorder, GLP-1 Receptor Agonists, Addiction Pharmacotherapy, Health and Well-Being, Metabolic And Mental Health

CITATION

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1. Introduction

Alcohol use disorder (AUD) remains a major global health concern, responsible for approximately three million deaths annually and accounting for about five percent of global disability-adjusted life years (Organisation mondiale de la santé, 2018). Despite this substantial and persistent burden, and significant advances in understanding the neurobiological mechanisms underlying addiction, alcohol-related harm continues to remain high worldwide (Heilig et al., 2021). Currently approved pharmacotherapies—naltrexone, acamprosate, and disulfiram—demonstrate modest but clinically meaningful efficacy in reducing alcohol consumption and return to drinking (McPheeters et al. 2023), with treatment responses varying across individuals. Considering this, developing innovative pharmacological treatments that directly target the central neural mechanisms of craving and reward remains a critical scientific and public health priority.

A growing body of research suggests a potential new avenue in addiction treatment, arising from a previously unexpected therapeutic class: glucagon-like peptide-1 receptor agonists (GLP-1RAs). Originally designed to enhance glycemic control and support weight reduction in metabolic disease (Ussher & Drucker, 2023), these incretin-based agents have demonstrated broader neurobiological effects influencing motivation, craving, and reward. GLP-1 receptors are widely distributed across mesolimbic reward circuits, including projections from the nucleus of the solitary tract to the ventral tegmental area and nucleus accumbens, where they modulate dopaminergic signalling (Jerlhag, 2020). Converging preclinical and early clinical findings suggest that GLP-1 receptor activation attenuates alcohol reward, reduces intake, and suppresses motivational and relapse-related behaviours (Jerlhag, 2023).

Beyond these central effects, peripheral gut-brain and hypothalamic pathways contribute to the regulation of appetite, stress, and interoceptive signalling (Martinelli et al., 2024). Collectively, these

convergent mechanisms suggest that GLP-1RAs influence both metabolic and motivational systems, potentially restoring balance between homeostatic and hedonic regulation and offering a promising neuropharmacological target for alcohol use disorder.

Given the convergence of clinical observations and translational neuroscience, GLP-1RAs represent a biologically plausible in pharmacotherapy of alcohol use disorder, warranting further investigation. Their well-characterized safety in metabolic disease, availability of long-acting formulations, and additional metabolic and cardiovascular benefits make GLP-1 receptor agonists particularly attractive candidates for individuals with co-occurring metabolic and substance use disorders—though dedicated trials are still needed to confirm safety and efficacy in metabolically healthy SUD populations (Jerlhag, 2023).

This article synthesizes evidence from preclinical and clinical studies, evaluates the proposed neurobiological mechanisms linking GLP-1 signalling to alcohol consumption, and discusses the translational implications of repurposing GLP-1RAs as potential anti-craving agents in the treatment of AUD.

2. Objectives and Scope of the Review

This narrative review aims to integrate the selected body of evidence describing the interaction between the GLP-1 signalling pathway and alcohol-related behaviours, with a focus on the therapeutic potential of GLP-1 receptor agonists (GLP-1RAs) in alcohol use disorder (AUD). Drawing upon thirty-one eligible publications—including preclinical experiments, human observational and interventional studies, and meta-analyses—the review seeks to:

- Summarize preclinical mechanisms by which GLP-1R activation influences alcohol consumption, reinforcement, and relapse through modulation of mesolimbic, hypothalamic, and stress-related circuits.
- Evaluate translational and clinical evidence from randomized trials, cohort studies, and genetic analyses assessing the association between GLP-1RA treatment and reduced drinking behaviour or relapse risk.
- Integrate findings from recent reviews and meta-analyses to define the overall strength and consistency of existing evidence.
- Identify knowledge gaps and research priorities required to establish GLP-1RAs as a validated treatment modality for AUD.

The scope of this review is intentionally broad, encompassing both basic neuroscience and clinical pharmacotherapy, to provide a coherent overview of how incretin-based signalling intersects with the neurobiology of addiction. Emphasis is placed on mechanistic convergence, translational relevance, and the identification of research priorities for future randomized controlled trials aimed at determining efficacy, optimal dosing, and long-term safety in individuals with alcohol use disorder.

3. Methods and Search Strategy

A comprehensive literature search was conducted to identify studies examining the overlap between metabolic regulation and addiction and assessing GLP-1 receptor agonists as novel therapeutic strategies acting on these common pathways. The search covered PubMed and Google Scholar using combinations of the following keywords: “GLP-1”AND “alcohol” OR “use disorder.” Reference lists of all relevant papers and prior reviews, as well as additional sources were hand-searched to include additional eligible citations.

Because this work aimed to produce a narrative synthesis, no formal systematic search strategy, no statistical pooling or standardized risk-of-bias scoring was conducted. Seventy-one full-text articles were assessed: preclinical experimental studies, human observational analyses, randomized controlled trials, Mendelian-randomization research, and review papers. Case reports, editorials, conference abstracts, and studies unrelated to alcohol, but rather focusing on other addictions such as opioid or nicotine were excluded. Among the final selection, thirty-one studies met inclusion criteria and were used to provide a synthesis of current evidence.

For each study, key information such as study design, population characteristics, intervention type, and principal findings was extracted and synthesized. Evidence was grouped into thematic domains, including preclinical mechanisms, translational findings, and clinical evidence, to facilitate integration across levels of analysis.

4. Biological Rationale for Targeting GLP-1 System in Addiction

To organize this paper, evidence from the 41 included studies was grouped into subsections that facilitate tracing the path from mechanistic understanding to clinical—and ultimately human—translation.

4.1. Preclinical and Mechanistic Evidence

Experimental studies in rodents consistently demonstrate that activation of the GLP-1 receptor attenuates alcohol-related reward behaviours. Across multiple paradigms—including voluntary alcohol intake, conditioned place preference, and operant self-administration—GLP-1 receptor agonists such as exenatide, liraglutide, and semaglutide have been shown to reduce alcohol consumption and reward-related responding (Chuong et al., 2023). More recent work using semaglutide further demonstrates reduced alcohol intake and attenuation of alcohol-induced dopamine release within the nucleus accumbens, reinforcing the role of mesolimbic GLP-1 signalling in reward modulation (Aranäs et al., 2023).

Pharmacological and genetic evidence indicates that GLP-1 receptor signalling plays a key role in regulating reward-related behaviours. Neuroanatomical studies show that GLP-1 receptors are expressed across mesolimbic and hindbrain regions—including the nucleus accumbens, ventral tegmental area, prefrontal cortex, hypothalamus, and nucleus tractus solitarius—where they influence dopaminergic, glutamatergic, and GABAergic neurotransmission (Jerlhag, 2023; Martinelli et al., 2024; Alves et al., 2025). GLP-1 neurons originating in the nucleus tractus solitarius project to these regions, linking homeostatic and reward-related signalling. At the circuit level, GLP-1 receptor activation can suppress drug-seeking behaviour through pathways involving GABAergic modulation of the ventral tegmental area (Hernandez et al., 2021). In addition, GLP-1 signalling integrates peripheral metabolic input via gut–brain pathways, further connecting physiological state with motivated behaviour. Together, these findings provide a coherent neurobiological basis for targeting GLP-1 receptors in alcohol use disorder (Leggio et al., 2025).

Together, preclinical evidence supports a strong mechanistic rationale for targeting GLP-1Rs in alcohol addiction. By recalibrating dopaminergic, limbic, and metabolic circuits, GLP-1RAs appear to normalize the dysregulated interplay between homeostatic and hedonic control—a process central to both addiction and obesity (Jerlhag, 2023; Martinelli et al., 2024). This integrated neurobiological action positions GLP-1–based therapies as promising candidates for translational development in alcohol use disorder.

Functional neuroimaging studies in humans provide preliminary support for these mechanisms, showing that GLP-1 receptor agonist treatment reduces alcohol cue reactivity in the ventral striatum and related reward-associated regions (Klausen et al., 2022).

4.2. Translational and Clinical Evidence

Evidence from human studies increasingly reflects preclinical findings suggesting that glucagon-like peptide-1 receptor agonists (GLP-1RAs) influence alcohol-related reward processes. Translational research indicates that GLP-1 signalling modulates mesolimbic circuitry and reduces responsiveness to drug-related cues, supporting a central neurobiological mechanism (Leggio et al., 2025). In the first randomized, double-blind, placebo-controlled clinical trial evaluating a GLP-1 receptor agonist in individuals with alcohol use disorder (AUD), once-weekly exenatide (2 mg) did not significantly reduce heavy drinking days compared with placebo, although it significantly attenuated alcohol cue reactivity in reward-related brain regions and altered dopamine transporter availability (Klausen et al., 2023). Exploratory analyses further demonstrated reductions in alcohol intake among individuals with obesity, suggesting potential phenotype-specific treatment effects. Complementing these findings, large-scale observational data indicate that GLP-1RAs are not associated with increased neuropsychiatric risk and may be linked to reduced substance use behaviours, supporting broader effects on reward-related pathways (De Giorgi et al., 2024). Together, these findings suggest that GLP-1RAs exert measurable neurobehavioral effects in humans, although consistent reductions in alcohol consumption have yet to be established.

Concomitantly, neurochemical imaging findings indicating reduced dopamine transporter availability in striatal regions following GLP-1RA treatment suggest modulation of dopaminergic signalling. This observation is consistent with longstanding models implicating striatal dopamine in reward, motivation, and reinforcement processes central to addiction (Nutt et al., 2015). Rather than uniformly increasing or decreasing dopamine activity, contemporary perspectives emphasize dysregulated dopaminergic function—particularly within mesolimbic circuits—as a key feature of substance use disorders. In this context, GLP-1RA–induced changes in reward-related neural activity may reflect modulation of incentive salience and cue-driven behaviour, processes known to be disrupted in alcohol use disorder.

Importantly, these neural effects were not fully explained by changes in weight or metabolic parameters, suggesting the involvement of central mechanisms rather than solely indirect metabolic effects. From a clinical perspective, these findings support the hypothesis that GLP-1RAs may influence alcohol-related motivation through modulation of reward-related neural circuitry.

Subsequent clinical investigations using newer glucagon-like peptide-1 receptor agonists (GLP-1RAs) have provided additional, albeit preliminary, support for their effects on alcohol-related behaviours. In a recent randomized, laboratory-based trial of low-dose semaglutide, participants exhibited reduced alcohol consumption during a controlled self-administration paradigm, with medium to large effect sizes observed for both grams of alcohol consumed and peak breath alcohol concentration (Hendershot et al., 2025). Semaglutide treatment was also associated with significant reductions in weekly alcohol craving and drinks per drinking day, as well as greater reductions in heavy drinking over time relative to placebo, although no significant effect was observed for overall drinking frequency.

In addition to these behavioural effects, participants in the semaglutide group experienced clinically meaningful weight loss, consistent with the drug's established metabolic profile. While these findings do not yet demonstrate consistent reductions in alcohol consumption across all measures, they provide converging evidence that GLP-1RAs can influence both reward-related behaviour and metabolic regulation.

A complementary clinical investigation using dulaglutide reported similar trends, with participants exhibiting a significant reduction in weekly alcohol consumption compared with placebo (Probst et al., 2023). However, subgroup analyses indicated no significant effect among individuals classified as heavy drinkers, suggesting potential heterogeneity in treatment response. This pattern aligns with broader observations that individuals with higher baseline substance use may respond differently to pharmacological interventions, although the neurobiological mechanisms underlying this variability remain to be elucidated.

Clinically, these data suggest that semaglutide and dulaglutide may attenuate alcohol-related motivation through modulation of overlapping metabolic and reward pathways, producing effects that are both behaviourally and physiologically relevant. Although these pilot studies remain limited by modest sample sizes and short durations, their consistency with mechanistic evidence and emerging observational findings—such as reports of reduced alcohol craving and intake among individuals using GLP-1 receptor agonists for metabolic indications (Quddos et al., 2023)—supports a coherent, though still preliminary, translational pattern.

Above-mentioned clinical and mechanistic findings from these early trials support the concept that GLP-1 receptor activation reduces alcohol consumption by recalibrating the balance between homeostatic and hedonic signalling, offering a promising therapeutic avenue for individuals with AUD, particularly those with comorbid metabolic dysregulation.

Real-world and registry-based data further support these clinical observations at the population level. In large retrospective cohort analyses of patients with established alcohol or opioid use disorders, GLP-1 receptor agonist prescriptions have been associated with significantly lower rates of alcohol intoxication and related adverse outcomes compared to non-use (Qeadan et al., 2025). Across these datasets, adjusted incidence rate ratios and complementary time-to-event analyses consistently favoured GLP-1RA exposure, with effects remaining robust after controlling for demographic, metabolic, and psychiatric comorbidities. However, given the observational and retrospective nature of these studies, these findings should be interpreted as associative rather than causal, while nonetheless providing important population-level support for a potential role of GLP-1 signalling in modulating alcohol-related behaviours.

In summary, evidence from systematic review and meta-analysis indicates a divergence across study designs rather than a uniformly consistent effect. In pooled randomized controlled trials, glucagon-like peptide-1 receptor agonists (GLP-1RAs) were associated with small but non-significant reductions in alcohol consumption, drinks per drinking day, and craving (standardized mean differences approximately −0.14 to −0.24), with substantial heterogeneity across outcomes (Sinha & Ghosal, 2026). In contrast, large-scale observational analyses demonstrated a consistent association between GLP-1RA use and reduced alcohol-related events (hazard ratio ≈ 0.64), including alcohol use disorder incidence, recurrence, and alcohol-related hospitalizations. To sum up, these findings suggest a promising but as of now inconclusive translational signal, with stronger effects observed in real-world datasets than in controlled clinical trials.

Although the aggregate clinical signal remains preliminary, the coherence of findings across randomized trials, pharmacologic extension studies, population cohorts, and genetic analyses constitutes a cohesive translational framework. Altogether, these data indicate that GLP-1RAs may represent a mechanistically novel, metabolically complementary, and psychologically integrative therapeutic option for AUD—warranting rigorous evaluation in large-scale Phase III investigations that examine both drinking outcomes and broader well-being endpoints.

4.3. Neurobiological Mechanisms Underlying Anti-Craving Effects

Engagement of the GLP-1 receptor (GLP-1R) appears to recalibrate reward valuation rather than merely suppress consummatory behaviour. Mechanistically, GLP-1R stimulation attenuates the reinforcing properties of alcohol by reducing alcohol-induced dopamine release within mesolimbic pathways, particularly in the nucleus accumbens, a key region mediating reward encoding and motivational salience (Jerlhag, 2023). These effects are mediated through projections from GLP-1–producing neurons in the nucleus tractus solitarius to reward-related regions including the ventral tegmental area and nucleus accumbens, linking homeostatic and hedonic regulation. Preclinical studies using microdialysis demonstrate that GLP-1 receptor agonists attenuate alcohol- and drug-induced dopamine release within the nucleus accumbens. While GLP-1 receptors in the ventral tegmental area contribute to these effects, current evidence suggests that modulation of dopaminergic activity is mediated indirectly through local circuit mechanisms, including GABAergic signalling, rather than through directly demonstrated reductions in dopamine neuron firing (Hernandez et al., 2018; Jerlhag, 2023).

At the circuit level, functional neuroimaging studies in humans provide preliminary support for these mechanisms, demonstrating that GLP-1 receptor agonist administration reduces alcohol cue reactivity within key reward-related regions, particularly the striatum (Klausen et al., 2022). These findings are consistent with broader evidence implicating fronto-striatal networks in the regulation of motivation, valuation, and goal-directed behaviour. While reductions in neural cue reactivity may relate to decreases in subjective craving observed in some studies, the relationship between these neural changes and behavioural outcomes remains incompletely characterized. Together, these data suggest that GLP-1RAs may modulate fronto-striatal circuitry involved in alcohol motivation, although further research is required to clarify the extent of cortical involvement and clinical significance.

Beyond central networks, peripheral GLP-1 signalling may contribute indirectly to the modulation of alcohol-related motivation. Activation of GLP-1 receptors on vagal afferents and intestinal enteroendocrine cells engages gut–brain signalling pathways that project to the nucleus tractus solitarius (NTS) and hypothalamus, supporting the integration of metabolic and neural regulation (Martinelli et al., 2024). Through these pathways, GLP-1 receptor agonists may influence reward-related processes alongside their established effects on satiety and energy balance. This gut–brain axis provides a plausible mechanism linking metabolic state and alcohol-related behaviour, consistent with observations that some clinical effects of GLP-1RAs may vary across patient subgroups, although the extent to which metabolic status directly modulates treatment response remains to be established (Probst et al., 2023).

4.4. Clinical and Therapeutic Implications

The emerging evidence suggests that GLP-1 receptor agonists (GLP-1RAs) could represent a promising new direction in the pharmacologic treatment of alcohol use disorder (AUD). Their unique capacity to modulate both metabolic homeostasis and motivational drive offers a therapeutic duality that few current AUD medications provide. In populations where AUD frequently converges with obesity, type 2 diabetes, and non-alcoholic fatty liver disease, GLP-1RAs present a rare opportunity to treat *both* the metabolic and neurobehavioral manifestations of disease using a single, mechanistically coherent intervention [Leggio et al., 2024; Martinelli et al., 2024].

A medication class with established infrastructure, known tolerability, and high public familiarity may accelerate the clinical adoption curve—a long-standing bottleneck in addiction therapeutics. Clinically, these agents are attractive not only because of their well-established cardiometabolic safety profile, but also because they are already widely prescribed across multiple medical disciplines. Their existing use in the management of diabetes and obesity may facilitate translational adoption in addiction settings, particularly given the familiarity of clinicians with their administration and monitoring. This familiarity could help address the historically low uptake of approved pharmacotherapies for alcohol use disorder, although this potential remains to be demonstrated in practice (Leggio et al., 2023).

Thus, GLP-1RAs embody a “whole-person” pharmacology that simultaneously addresses brain, body, and behaviour.

Important uncertainties remain. Optimal dosing for neurobehavioral benefit, the duration of therapy required to sustain reductions in alcohol use and craving, and the extent to which treatment response varies across metabolic or genetic subgroups all require systematic investigation (Leggio et al., 2023; Jerlhag, 2023). Furthermore, while preliminary signals are encouraging, the field must progress from opportunistic secondary analyses and small-scale or open-label studies to adequately powered, placebo-controlled trials using standardized clinical endpoints such as percent days abstinent, time to relapse, and craving trajectories. Such

trials will be essential to determine whether GLP-1 receptor agonists can translate their mechanistic promise into meaningful real-world recovery outcomes.

If validated, the public health implications could be substantial. Alcohol use disorder (AUD) remains one of the most burdensome yet undertreated psychiatric conditions worldwide, with fewer than 10% of affected individuals receiving evidence-based pharmacotherapy (World Health Organization, 2023). A repurposed, metabolically beneficial treatment such as a GLP-1 receptor agonist could help expand access to care by leveraging existing prescribing pathways in primary care and metabolic medicine. This may offer an opportunity to bridge the gap between general medical and addiction-focused services. In this context, advancing these agents from metabolic settings into addiction-focused protocols is not only a scientific possibility but also a public health opportunity—one that could align the prevention of cardiometabolic disease with recovery from addiction within a single therapeutic framework.

4.5. Limitations of Current Evidence and Research Directions

Despite rapidly growing interest, the current evidence supporting glucagon-like peptide-1 receptor agonists (GLP-1RAs) for alcohol use disorder (AUD) remains preliminary and methodologically constrained. Much of the literature consists of preclinical studies, small or short-term clinical trials, and retrospective or register-based analyses, with several trials still ongoing (Klausen et al., 2025; Petrie et al., 2025). Moreover, a substantial proportion of human data derives from populations treated primarily for metabolic conditions such as obesity or type 2 diabetes rather than individuals with primary AUD.

As a result, confounding by metabolic improvement—notably weight loss, improved insulin sensitivity, or enhanced liver function—cannot yet be excluded. GLP-1 receptor agonists exert substantial metabolic effects that may indirectly influence alcohol consumption, and alternative explanations such as treatment-related side effects or behavioural changes have also been proposed (Subhani et al., 2024; Sinha & Ghosal, 2025). Establishing whether GLP-1RAs reduce alcohol craving independently of these metabolic changes therefore remains a central research question.

Another limitation concerns dose selection and pharmacokinetics. The optimal exposure required to influence central nervous system (CNS) pathways involved in reward and craving may differ from doses established for glycaemic control or weight management. Experimental evidence indicates that incretin receptor agonists vary in their ability to access the brain, and that central exposure is not solely determined by peripheral dose (Rhea et al., 2024). Moreover, the mechanisms by which GLP-1 receptor agonists influence central pathways remain incompletely understood and are subject to ongoing debate. Current evidence suggests that these agents may act through multiple routes, including limited penetration across the blood–brain barrier, access via circumventricular organs, or indirect signalling via vagal afferents, rather than uniform passive diffusion into the brain (Feng & Jin, 2025).

As a result, the relationship between systemic exposure and central effects is likely complex and non-linear, and the optimal dosing required to modulate reward processing and craving may differ from that established for metabolic indications. Higher systemic exposure is, however, associated with an increased likelihood of adverse effects—most commonly gastrointestinal symptoms—which are consistently reported across clinical studies (Martinelli et al., 2024). While it is biologically plausible that higher doses aimed at enhancing central effects could exacerbate such adverse events, direct evidence linking CNS-targeted dosing strategies to increased risk remains limited. Furthermore, although GLP-1 receptor agonists are associated with a low intrinsic risk of hypoglycaemia when used without concomitant insulin or insulin secretagogues, their safety profile in metabolically healthy, normal-weight individuals has not been systematically established.

Given the limited clinical experience with GLP-1 receptor agonists in euglycemic populations, long-term safety in this subgroup remains uncertain. Theoretically, chronic overstimulation of the GLP-1 pathway could lead to excessive appetite suppression, unintended weight loss, or dysregulated energy balance; however, such outcomes have not been consistently observed in human studies. Until dedicated trials evaluate efficacy and safety specifically in metabolically healthy individuals, cautious use and close clinical monitoring are warranted.

Furthermore, existing clinical studies of GLP-1 receptor agonists in AUD are typically of short duration, with randomized trials ranging from approximately 9 weeks to 26 weeks of treatment (Hendershot et al., 2025; Farokhnia et al., 2025). The overall evidence base remains limited and heterogeneous, comprising a small number of randomized trials alongside observational and preclinical studies (Bernstein & Schacht, 2025). As a result, there is a lack of robust long-term follow-up data, particularly beyond treatment discontinuation. Consequently, it remains unclear whether reductions in alcohol craving and consumption persist after cessation

of therapy, and optimal treatment duration has yet to be established (Bernstein & Schacht, 2025). These limitations are especially important given the chronic, relapsing nature of AUD, which requires interventions capable of sustaining remission and preventing relapse over extended periods.

Evidence on drug–drug interactions involving GLP-1 receptor agonists in individuals with alcohol use disorder (AUD) is scarce. Indirect insights can be drawn from a recent systematic review of GLP-1RA interactions with oral medications, which found that these agents slow gastric emptying, leading to delayed and reduced peak plasma concentrations (Calvarysky et al., 2024). Although these effects are generally not clinically significant, they may be relevant for medications with a narrow therapeutic index. Overall, evidence remains limited in complex, polypharmacy populations such as individuals with AUD, highlighting the need for further pharmacovigilance and dedicated interaction studies.

Finally, several key research priorities emerge, including large multicentred randomized controlled trials evaluating multiple GLP-1RAs and dual incretin agonists across standard AUD outcomes, alongside mechanistic studies using functional imaging and multimodal biomarkers to distinguish central from peripheral effects. Further work is needed to identify moderators of treatment response, including sex-specific, metabolic, and genetic factors, as well as to establish safety and tolerability in normal-weight, non-diabetic populations. In addition, combination strategies integrating GLP-1RAs with established pharmacological and psychotherapeutic approaches warrant systematic evaluation.

Until these gaps are addressed, GLP-1RAs should be considered experimental but promising adjunctive treatments rather than replacements for established first-line therapies. While findings across preclinical and early clinical studies are encouraging, addressing these limitations will be essential to define their long-term efficacy, safety, and role in the treatment of alcohol use disorder.

5. Patient Stratification and Precision Treatment Approaches

An emerging theme across studies of GLP-1 receptor agonists (GLP-1RAs) in alcohol use disorder (AUD) is the potential importance of patient stratification. Evidence from both preclinical and clinical research suggests that treatment effects vary across individuals, reflecting underlying metabolic and neurobiological differences. For example, real-world data indicate that GLP-1RA–associated reductions in alcohol-related outcomes are observed across patient subgroups, including those with comorbid metabolic conditions (Qeadan et al., 2025), while preclinical studies demonstrate variability in response by sex and treatment duration (Kalafateli et al., 2020).

In parallel, genetic and causal inference studies provide further support for heterogeneity in treatment response. Mendelian randomization analyses show that genetically proxied GLP-1 receptor signalling is associated with reduced binge drinking and problematic alcohol use, suggesting a mechanistic link between GLP-1 pathways and alcohol-related behaviours (Reitz et al., 2025). Together, these findings support a precision medicine framework in which metabolic state, biological variability, and genetic factors may influence therapeutic response (Klausen et al., 2025). Stratifying patients along these dimensions could improve treatment targeting and efficacy, although this approach remains to be tested in prospective clinical trials.

6. Standard-of-Care Pharmacotherapy in Alcohol Use Disorder

6.1 Current Standard

Current pharmacotherapies for alcohol use disorder (AUD) remain limited in both efficacy and real-world implementation. The primary approved agents—naltrexone, acamprosate, and disulfiram—form the foundation of treatment but demonstrate modest and variable effects across clinical settings (Maisel et al., 2013; McPheeters et al., 2023). Naltrexone, an opioid receptor antagonist, reduces alcohol-related reward and craving, whereas acamprosate, a glutamatergic modulator, supports abstinence by stabilizing excitatory–inhibitory balance (Maisel et al., 2013). Disulfiram, an aldehyde dehydrogenase inhibitor, operates via aversive conditioning but is limited by adherence and tolerability concerns. Although these medications provide statistically significant improvements over placebo, their overall clinical impact is moderate, and many patients do not achieve sustained remission (Maisel et al., 2013; McPheeters et al., 2023). Relapse rates remain high, with approximately two-thirds of individuals returning to alcohol use within six months of treatment, reflecting the chronic and multifactorial nature of AUD rather than any single treatment limitation (Nguyen et al., 2020). Treatment response is also heterogeneous across individuals and contexts. Together, these challenges highlight the need for novel therapeutics that extend beyond traditional neurotransmitter targets to address the broader metabolic and motivational dimensions of addiction.

6.2 Mechanistic Contrast

The predominantly neurochemical and behavioural selectivity of naltrexone, acamprosate, and disulfiram reflects a traditional neurotransmission-based framework of addiction treatment. Naltrexone reduces alcohol-related reward through μ-opioid receptor blockade, indirectly modulating reinforcement processes, while acamprosate modulates glutamatergic (NMDA) and GABAergic signalling to restore neurobiological balance disrupted by chronic alcohol exposure (Kalk et al., 2014; Ooteman et al., 2007).

In contrast to conventional pharmacotherapies that primarily target discrete neurotransmitter systems, GLP-1 receptor agonists (GLP-1RAs) operate through broader integrative mechanisms linking metabolic and reward-related processes. By engaging both peripheral and central pathways, these agents extend beyond a purely neurotransmission-based model and instead influence the interaction between physiological state and motivational behaviour. This systems-level mode of action distinguishes GLP-1RAs from existing treatments and supports a reconceptualization of addiction as a disorder.

6.3 Clinical Comparison

Emerging clinical studies have begun to evaluate the effects of GLP-1 receptor agonists (GLP-1RAs) in substance use disorders. Agents such as liraglutide and semaglutide have shown reductions in alcohol craving in early trials (Bernstein & Schacht, 2025). Importantly, observed effects vary considerably. Some studies—most notably a recent semaglutide trial—report reductions in craving and selected drinking outcomes, with effect sizes that appear notable in the context of typically modest effects observed with established treatments such as naltrexone, although direct comparisons are not available, while others demonstrate modest or nonsignificant results. This variability likely reflects methodological limitations, including small sample sizes, short study durations, and differences in study populations and compounds. Emerging evidence also suggests that treatment response may differ across substances, reflecting differences in the underlying neurocircuitry engaged by specific drugs (Alves et al., 2025). In addition, long-acting formulations administered once weekly may offer an adherence advantage over daily oral pharmacotherapies, representing a clinically relevant consideration in real-world settings.

6.4 Key Limitations

Despite these advantages, several constraints temper enthusiasm for the widespread adoption of GLP-1RAs in addiction medicine. The lack of head-to-head randomized controlled trials comparing GLP-1RAs with established treatments such as naltrexone, acamprosate, or disulfiram limits understanding of their relative efficacy. Many studies are small, short-term, or use open-label or crossover designs, making it difficult to assess durability or relapse prevention. High cost and limited access—particularly outside metabolic indications—may further constrain implementation, especially in publicly funded health systems where existing AUD medications are inexpensive generics. Long-term safety in populations with psychiatric comorbidities also remains insufficiently characterized.

6.5 Health-economic and access considerations

Cost and accessibility remain important and underexplored limitations of GLP-1 receptor agonists, particularly for newer agents such as semaglutide and tirzepatide. At the same time, substance use disorders impose substantial societal and economic burdens, including healthcare costs, lost productivity, and increased morbidity and mortality (Rosiejka et al., 2026). In this context, the introduction of high-cost pharmacotherapies raises critical questions regarding real-world feasibility, scalability, and equitable access.

Compared with currently approved treatments for alcohol use disorder (AUD), which are widely available as low-cost generics, GLP-1 receptor agonists are substantially more expensive and typically restricted to indications such as type 2 diabetes or obesity. This creates structural barriers to access, particularly in publicly funded healthcare systems and among socioeconomically disadvantaged populations, who are disproportionately affected by substance use disorders (Rosiejka et al., 2026). Moreover, as clinical efficacy in AUD has not yet been clearly established and randomized controlled trials are still required to demonstrate safety and efficacy (Leggio et al., 2023), the cost-effectiveness of these agents in this context remains uncertain.

From a health-economic perspective, future research should evaluate whether the potential benefits of GLP-1 receptor agonists—such as reductions in alcohol intake, relapse, and alcohol-related complications—translate into meaningful long-term savings for healthcare systems. This is particularly relevant given the chronic and relapsing nature of addiction and the limited effectiveness of existing pharmacotherapies. Until

such data are available, the high cost, restricted reimbursement, and uncertain cost-effectiveness of GLP-1-based therapies may significantly limit their implementation in routine addiction care, despite their promising mechanistic and therapeutic profile.

7. Discussion

The accumulated evidence across preclinical, translational, and early clinical studies supports a coherent biological rationale for the use of glucagon-like peptide-1 receptor agonists (GLP-1RAs) in alcohol use disorder (AUD). Experimental models consistently demonstrate that GLP-1 receptor activation modulates reward-related behaviour, reducing alcohol intake, motivation, and relapse-like responding through effects on mesolimbic and stress-related pathways. These findings provide a robust mechanistic foundation and have guided the initial translation into human research.

Early clinical studies offer preliminary support for these effects, suggesting that GLP-1RAs may reduce alcohol consumption and craving in some individuals. However, results remain heterogeneous, with variability across compounds, study designs, and patient populations. Notably, stronger responses have often been observed in individuals with co-occurring metabolic dysfunction, raising the possibility that metabolic state may influence treatment response. This observation aligns with the proposed mechanism of GLP-1RAs as integrators of metabolic and reward signalling, although the extent to which these effects are independent of weight loss or other metabolic improvements remains unclear.

Beyond individual trials, observational and genetic studies provide converging, though indirect, support for a link between GLP-1 signalling and alcohol-related outcomes. Together with preclinical findings, this cross-level consistency strengthens the biological plausibility of GLP-1–based interventions. At the same time, the current evidence base is limited, and causal inferences remain constrained by the small number of randomized controlled trials and their methodological heterogeneity.

From a mechanistic perspective, GLP-1RAs differ from currently approved AUD medications by acting across both central and peripheral systems. Rather than targeting a single neurotransmitter pathway, these agents appear to modulate reward processing, stress reactivity, and interoceptive signalling in parallel. This broader mode of action may offer theoretical advantages, particularly in addressing the complex interplay between metabolic state, motivation, and substance use. However, whether this translates into clinically meaningful improvements over existing treatments has not yet been established.

Several practical considerations further influence their potential role in addiction care. Long-acting formulations may support adherence by reducing the burden of daily dosing, and their established use in metabolic medicine may facilitate integration into general healthcare settings. At the same time, substantial barriers remain. GLP-1RAs are considerably more expensive than current AUD medications and are often restricted to metabolic indications, limiting accessibility. In the absence of clear evidence of clinical efficacy in AUD, their cost-effectiveness in this context cannot yet be determined.

Important limitations of the current literature must also be considered. Most studies are small, of short duration, and frequently include participants without primary AUD diagnoses. The durability of treatment effects after discontinuation is unknown, and optimal dosing for central nervous system engagement has not been established. In addition, potential interactions with existing pharmacotherapies and long-term safety in metabolically healthy populations remain insufficiently characterized.

However, further research is required to determine whether this mechanistic promise translates into sustained clinical benefit. Large, well-designed randomized controlled trials—alongside studies addressing patient stratification, long-term outcomes, and real-world implementation—will be essential to define their place in addiction treatment.

8. Conclusions

The body of evidence reviewed here supports glucagon-like peptide-1 receptor agonists (GLP-1RAs) as a mechanistically compelling and clinically promising therapeutic avenue for alcohol use disorder (AUD). Preclinical and early clinical findings converge in suggesting that GLP-1 receptor activation modulates key neural and behavioural processes underlying alcohol use, including reward signalling, craving, and relapse-related mechanisms.

Clinically, GLP-1RAs are of particular interest because they bridge metabolic regulation and addiction medicine. Their combined effects on reward processing and metabolic health may be especially relevant in individuals with co-occurring metabolic dysfunction, where preliminary studies suggest enhanced

responsiveness. Observational and genetic data further support a potential relationship between GLP-1 signalling and reduced alcohol-related risk.

However, current clinical evidence remains limited. Most studies are small, short-term, and often conducted in populations without primary AUD diagnoses. Key questions regarding long-term efficacy, optimal dosing, safety in metabolically healthy individuals, and cost-effectiveness remain unresolved. Addressing these gaps will require well-powered randomized controlled trials and careful evaluation of patient subgroups.

Overall, GLP-1 receptor agonists represent a novel and biologically grounded approach that extends beyond traditional neurotransmitter-based treatments. While further evidence is needed to establish their clinical role, their ability to integrate metabolic and motivational pathways highlights a promising direction for future research and may contribute to more comprehensive models of addiction treatment.

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