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GLP-1 Receptor Agonists in Alcohol Use Disorder: Neurobiological Mechanisms and Therapeutic Potential

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Abstract

Alcohol use disorder (AUD) remains a significant global health challenge, characterized by compulsive alcohol intake, impaired control over consumption, and a high risk of relapse. Despite its widespread impact, pharmacological treatment options for AUD are limited and often insufficient. Glucagon-like peptide-1 receptor agonists (GLP-1RAs), originally developed for the treatment of type 2 diabetes and obesity, have recently gained attention for their potential to modulate reward-related behaviors and neurobiological pathways implicated in addiction. Preclinical studies in rodent models have demonstrated that agents such as dulaglutide, semaglutide, and liraglutide can reduce alcohol intake, reverse neurochemical disruptions in key brain regions, and alleviate anxiety and cognitive impairments associated with chronic alcohol exposure. These findings are supported by emerging clinical evidence, including randomized controlled trials and large-scale cohort studies, which suggest that GLP-1RAs—particularly semaglutide—may reduce alcohol consumption and alcohol-related hospitalizations, especially in individuals with coexisting metabolic conditions. Although further randomized trials are needed to establish causality and define optimal treatment populations, GLP-1RAs hold substantial promise as a novel pharmacological approach in the treatment of AUD.

Keywords: GLP-1 Receptor Agonists; Alcohol Use Disorder, Semaglutide, Liraglutide, Dulaglutide, Addiction, Pharmacotherapy, Neurobiology, Substance Use Disorder

Introduction

Alcohol use disorder (AUD) is a chronically relapsing condition characterized by compulsive alcohol consumption, impaired control over intake, and the emergence of a negative emotional state when alcohol is unavailable [1–3]. Stress and anxiety are recognized as key contributors to alcohol craving and intake, playing a significant role in both the development and maintenance of AUD [3,4]. The frequent co-occurrence of post-traumatic stress disorder (PTSD) further highlights the importance of stress-related vulnerability in the onset and persistence of this disorder [5,6]. AUD is also associated with neuroadaptive changes in brain regions involved in motivated behavior, emotional regulation, and executive functioning, including the midbrain, limbic system, prefrontal cortex, and amygdala [7].

Beyond its neurobiological underpinnings, AUD remains a major global public health issue. According to the World Health Organization's (WHO) Global Status Report on Alcohol and Health (2024), alcohol consumption was responsible for approximately 2.6 million deaths worldwide in 2019, accounting for 4.7% of all global fatalities [8]. Among individuals aged 20–39, alcohol was the leading contributor to premature mortality and disability, causing 13% of deaths in this age group [8].

In terms of prevalence, an estimated 400 million people globally—equivalent to 7% of the population aged 15 and older—suffer from AUDs, with approximately 209 million individuals (3.7% of adults) affected by alcohol dependence [8]. Despite this considerable burden, access to treatment remains limited [8]. WHO data collected via the SDG 3.5.1 monitoring system indicate that treatment services for substance use disorders, including AUD, remain underdeveloped in many regions [8]. Estimates from the United Nations Office on Drugs and Crime (UNODC) suggest that fewer than 20% of individuals with drug use disorders receive treatment, with a similar gap likely for alcohol-related conditions [8]. These findings underscore the urgent need to improve the accessibility, availability, and affordability of treatment for individuals affected by AUD worldwide [8].

Aim

This narrative review aims to critically evaluate and synthesize current preclinical and clinical evidence regarding the potential role of glucagon-like peptide-1 receptor agonists (GLP-1RAs) in the treatment of AUD. By integrating findings from animal models and human

studies, the review explores the underlying neurobiological mechanisms through which GLP-1RAs may modulate alcohol-related behaviors, particularly those involving dopaminergic and GABAergic signaling. Special attention is given to the translational relevance of these findings, including their implications for relapse prevention, cognitive and emotional outcomes, and treatment efficacy in individuals with comorbid metabolic conditions. The objective is to provide a comprehensive and up-to-date resource for researchers and clinicians, highlighting the therapeutic promise of GLP-1RAs and identifying areas for future investigation in the context of addiction medicine.

Material and methods

Databases such as PubMed, MEDLINE, Google Scholar, and Europe PMC were searched using the following keywords: GLP-1 Receptor Agonists; Alcohol Use Disorder; Semaglutide; Liraglutide; Dulaglutide; Addiction; Pharmacotherapy; Neurobiology; Substance Use Disorder; Dopamine; GABA.

Studies published between 2007 and 2025 were considered. Both preclinical (animal-based) and clinical (human-based) studies were included to provide a balanced synthesis of mechanistic insights and translational relevance. Articles were selected based on scientific rigor, relevance to the role of GLP-1RAs in AUD, and clarity of outcomes. Reviews, randomized controlled trials, cohort studies, and mechanistic animal studies were prioritized. Publications with outdated data, insufficient methodological quality, or unrelated to GLP-1 signaling in the context of alcohol use were excluded.

In total, the review draws on 23 primary research articles and supporting background literature to critically evaluate the therapeutic potential and mechanisms of GLP-1RAs in AUD.

Glucagon-like Peptide-1: Its Role in Physiology and Potential in Addiction Treatment

Glucagon-like peptide-1 (GLP-1) is an incretin hormone that is synthesised in the intestinal L-cells through the differential processing of proglucagon [9,10].

It exerts a broad range of physiological effects, including neuroprotection, enhancement of cognitive function, cardiovascular protection, reduction of blood pressure, suppression of gastric acid secretion, promotion of lipolysis, and anti-inflammatory activity [11].

However, its most significant role lies in the regulation of glucose homeostasis [9,11].

GLP-1RAs, widely used in the treatment of type 2 diabetes and obesity, act by enhancing glucose-dependent insulin secretion, suppressing glucagon release, slowing gastric emptying, attenuating postprandial glucose excursions, and reducing caloric intake, which collectively promote weight loss and glycaemic control [12].

Beyond their established metabolic effects, growing evidence suggests that GLP-1 and its receptor agonists may have therapeutic potential in modulating reward-related behaviors and treating substance use disorders (SUD), including AUD [10]. Notably, GLP-1 is also synthesized in the nucleus tractus solitarius (NTS) of the brainstem and functions as a neurotransmitter across multiple brain regions. GLP-1 receptors are widely expressed in areas critically involved in reward processing and addiction, including the ventral tegmental area (VTA), nucleus accumbens (NAc), amygdala, and prefrontal cortex, suggesting a direct neuromodulatory role in the central regulation of addictive behaviors [10,13]. Moreover, studies in rodent models have demonstrated that several GLP-1RAs are capable of crossing the blood–brain barrier to some extent following systemic administration, enabling them to act on central GLP-1 receptors involved in reward-related pathways [10].

Mechanisms of Action of GLP-1 Receptor Agonists in Alcohol Use Disorder

GLP-1RAs, such as liraglutide and exendin-4, have been shown to modulate the neurocircuitry of addiction by acting on key brain regions involved in reward processing [14–16]. These compounds have been shown to attenuate alcohol-induced dopamine release in the nucleus NAc, a key structure within the mesolimbic reward pathway [14,16,17].

Moreover, GLP-1RAs reduce alcohol-induced hyperlocomotion and diminish the rewarding and conditioned properties of alcohol, including the formation of alcohol-associated memories, as observed in mCPP paradigms [16].

VTA represents another significant area of investigation as it constitutes the primary center for dopaminergic neurons in the mesolimbic dopamine system [16]. Given the VTA's heterogeneous composition, its various sub-regions may differentially influence alcohol-mediated behaviors [16].

Additionally, the laterodorsal tegmental area (LDTg), which projects to the VTA, plays a regulatory role in its dopaminergic activity [16].

Beyond these classical reward areas, other brain regions—including the lateral hypothalamus, hippocampus, lateral habenula, and nucleus of the NTS—have also been

identified as potential sites where GLP-1R signaling may modulate alcohol-related behaviors [16].

Collectively, these findings support a role for GLP-1RAs in dampening the reinforcing effects of alcohol through a distributed network of brain regions.

Another proposed mechanism through which GLP-1RAs, such as semaglutide, may influence alcohol-related behaviors involves modulation of GABAergic neurotransmission [18]. However, most of the observed effects have been reported in alcohol-naive animals, and data from alcohol-exposed models remain less consistent [18]. These findings suggest that modulation of GABAergic neurotransmission—particularly in regions such as the central amygdala (CeA) and infralimbic cortex (ILC)—may represent an additional pathway through which semaglutide exerts its effects on alcohol-related behaviors [18].

Preclinical Evidence for GLP-1 Receptor Agonists in AUD

A comprehensive study by Vallöf, Kalafateli, and Jerlhag (2020) investigated the effects of long-term administration of dulaglutide (0.1 mg/kg, s.c.) on voluntary ethanol consumption in male and female rats [19]. The treatment significantly reduced alcohol intake in both sexes; however, the effects were more pronounced and sustained in males [19]. In male rats, ethanol consumption was markedly decreased after both 5 and 9 weeks of treatment, and this reduction persisted even after treatment discontinuation [19]. In females, dulaglutide also reduced alcohol intake after 5 and 9 weeks of treatment, but the effect was less pronounced and did not persist following cessation [19].

Regarding water intake, dulaglutide induced a persistent increase in males, potentially reflecting a compensatory response to reduced ethanol consumption [19]. In contrast, female rats exhibited a temporary increase in water intake only after 5 weeks of treatment; this effect was not observed after 9 weeks and did not persist post-treatment, indicating a time-dependent and transient influence on fluid consumption [19].

The observed behavioral effects of dulaglutide were accompanied by distinct, sex-specific neurochemical alterations [19]. In male rats, nine weeks of dulaglutide treatment led to a marked reduction in dopaminergic neurotransmission in the amygdala, accompanied by decreased levels of serotonin (5-HT), its metabolite 5-HIAA, and noradrenaline [19]. Additional reductions in dopamine and DOPAC were observed in NAc, and dulaglutide also decreased DOPAC levels and dopamine turnover (DOPAC/dopamine ratio) in the prefrontal cortex—indicating a broad suppression of mesolimbic reward signaling pathways [19].

In contrast, neurochemical changes in females were more limited and region-specific. Dulaglutide reduced dopamine and DOPAC levels in the striatum and decreased DOPAC concentrations in the prefrontal cortex [19]. Additionally, an increase in noradrenaline levels was observed in the striatum, while serotonergic transmission remained unaffected across all examined brain regions [19].

Importantly, these neurochemical effects were reversible. In male rats, monoaminergic levels returned to baseline following a six-week washout period [19]. In females, most changes—particularly in the striatum—normalized after just one week of treatment discontinuation [19]. This sex-dependent difference in both the extent and persistence of neurotransmitter modulation may help explain the stronger and more lasting anti-addictive effects of dulaglutide observed in males compared to females [19].

A preclinical study conducted by Chuong et al. (2023) examined the effects of semaglutide—a long-acting GLP-1RA —on alcohol intake and central neurotransmission in rodent models [18]. Using a "drinking-in-the-dark" paradigm, the researchers demonstrated that semaglutide dose-dependently reduced binge-like alcohol consumption in both male and female C57BL/6J mice [18]. Similarly, in rats, semaglutide significantly attenuated both binge-like and dependence-induced ethanol intake, indicating robust efficacy across models and sexes [18].

Notably, semaglutide also reduced the intake of non-alcoholic palatable solutions, including sucrose, saccharin, and water, suggesting that its effects may extend beyond alcohol to general consummatory behavior [18]. This broad suppression might reflect reduced appetite, lowered motivational drive for rewarding stimuli, altered thirst regulation, or metabolic effects unrelated to substance-specific reward [18].

At the neurophysiological level, semaglutide increased the frequency of spontaneous inhibitory postsynaptic currents (sIPSCs) in neurons of the CeA and ILC in alcohol-naive rats, indicating an enhancement of GABAergic neurotransmission in brain regions involved in emotional regulation and executive control [18]. Interestingly, this effect was not observed in alcohol-dependent animals, potentially reflecting neuroadaptive changes associated with chronic ethanol exposure [18].

Taken together, these findings highlight the potential of semaglutide to attenuate alcohol consumption through modulation of inhibitory signaling in key brain regions, supporting its candidacy as a novel pharmacotherapy for AUD [18].

A recent preclinical study by Liu et al. (2024) investigated the therapeutic potential of liraglutide, a GLP-1RA, in a mouse model of alcohol dependence [20]. Male C57BL/6J mice were exposed to chronic intermittent ethanol (CIE) using a two-bottle choice paradigm, followed by a period of alcohol withdrawal [20]. Liraglutide administration (50 nmol/kg, i.p.) during the withdrawal phase significantly reduced both alcohol consumption and preference during relapse-like re-drinking [20]. In addition to its effects on intake, liraglutide alleviated anxiety-like behaviors, as evidenced by increased time spent in the center of the open field and in the open arms of the elevated plus maze [20]. It also improved cognitive function by reducing escape latency and enhancing spatial memory performance in the Morris Water Maze [20].

On a neurobiological level, liraglutide reversed alcohol-induced reductions in dendritic spine density in the medial prefrontal cortex (mPFC) and hippocampus (HP), as revealed by Golgi staining [20]. Moreover, it restored the expression of key synaptic proteins—p-GluA1, vGluT1, synaptophysin, and PSD-95—in both brain regions, which had been diminished by chronic alcohol exposure [20]. These results suggest that liraglutide mitigates alcohol-induced structural and functional synaptic impairments, potentially supporting its efficacy in reducing relapse and improving emotional and cognitive outcomes in alcohol-dependent individuals [20].

Together, these preclinical findings strongly support the potential utility of GLP-1RAs—namely dulaglutide, semaglutide, and liraglutide—in attenuating alcohol consumption and reversing neurobehavioral impairments associated with alcohol dependence in rodent models [18–20].

Clinical Evidence for GLP-1 Receptor Agonists in AUD

In a recent phase 2 randomized clinical trial, Hendershot et al. (2025) investigated the effects of once-weekly semaglutide on alcohol use outcomes in non-treatment-seeking adults with AUD [21]. The study included participants aged 21–65 who met DSM-5 criteria for AUD and had reported at least two heavy drinking episodes in the past month [21]. Using a hybrid design involving outpatient and laboratory assessments, the researchers evaluated both subjective and objective alcohol-related outcomes over a 9-week dosing period followed by a post-treatment assessment [21]. Participants were randomly assigned to receive either semaglutide or placebo in a double-blind fashion [21]. The dosing regimen started at 0.25 mg per week for 4 weeks, then increased to 0.5 mg for another 4 weeks, with an optional increase to 1.0 mg in week 9 depending on tolerability [21].

The results demonstrated that semaglutide significantly reduced alcohol consumption during the laboratory alcohol self-administration task, with medium to large effect sizes observed for both total alcohol intake and peak breath alcohol concentration [21]. In addition, participants in the semaglutide group reported fewer heavy drinking days and lower alcohol craving scores compared to placebo [21]. Semaglutide also led to significant weight loss (average of approximately 5%) by the end of the trial [21]. These findings suggest that semaglutide may hold promise as a pharmacological treatment for AUD, particularly in reducing alcohol consumption and craving in non-treatment-seeking individuals [21].

A large-scale, register-based cohort study conducted by Lähteenvuo et al. (2025) evaluated the association between GLP-1RA use and alcohol-related hospitalizations in individuals diagnosed with AUD in Sweden [22]. Using a within-individual design, the study found that both semaglutide and liraglutide were significantly associated with a reduced risk of hospitalization due to AUD and other SUDs [22]. Specifically, semaglutide use was linked to the lowest risk of AUD-related hospitalization (adjusted hazard ratio [aHR] = 0.64, 95% CI: 0.50–0.83), followed by liraglutide (aHR = 0.72, 95% CI: 0.57–0.92) [22]. These reductions were greater than those observed with officially approved AUD medications such as naltrexone, disulfiram, and acamprosate (group aHR = 0.98, 95% CI: 0.96–1.00) [22].

Semaglutide and liraglutide were also associated with a decreased risk of somatic hospitalizations, but not with suicide attempts [22]. These effects may be especially relevant for individuals with co-occurring type 2 diabetes or obesity, given the established metabolic benefits of GLP-1RAs in this population [22].

These findings are consistent with preclinical evidence and underscore the potential of GLP-1RAs—particularly semaglutide—as promising candidates for repurposing in the treatment of AUD [22]. However, as this was an observational study, randomized clinical trials are needed to confirm causality [22].

In a randomized, placebo-controlled clinical trial conducted by Klausen et al. (2022), the effects of exenatide, administered once weekly, were investigated for the treatment of AUD [23]. The study aimed to assess the efficacy of exenatide in reducing alcohol consumption and improving brain function and behaviors related to addiction [23]. Participants were randomly assigned to receive either exenatide (2 mg once weekly) or a placebo for a duration of 26 weeks [23]. Exenatide was delivered via subcutaneous injection at a weekly dose of 2 mg, the same dose commonly used in the management of type 2 diabetes [23].

Brain activity was assessed using functional magnetic resonance imaging (fMRI) and positron emission tomography (PET) [23]. fMRI results showed a significant reduction in activity in the ventral striatum, a key brain region involved in reward processing, in the exenatide group compared to the placebo group [23]. However, PET scans did not reveal any significant differences between the two groups in terms of dopamine transporter availability, suggesting that exenatide might influence the brain's reward system through mechanisms independent of dopamine transporter activity [23].

Regarding alcohol consumption, both the exenatide and placebo groups exhibited a reduction in heavy drinking days and overall alcohol intake, but these changes were not statistically significant between the two groups [23]. Furthermore, the exenatide group showed modest reductions in body mass index (BMI) and HbA1c levels, although these changes were not correlated with the reduction in alcohol consumption [23]. A subgroup analysis revealed that exenatide was more effective in reducing heavy drinking days in participants with obesity (BMI > 30 kg/m²), with a 23.6 percentage point reduction compared to the placebo group. In contrast, participants with a BMI < 25 kg/m² in the exenatide group experienced an increase in heavy drinking days [23].

These results suggest that while exenatide did not significantly reduce alcohol consumption in the overall study population, it had some effects on brain activity in regions associated with addiction [23]. Notably, exenatide demonstrated greater efficacy in participants with obesity, which may point to a need for further research focusing on this subgroup to fully assess its potential benefits for treating AUD [23].

Conclusions

GLP-1RAs represent a promising therapeutic avenue for AUD, supported by compelling preclinical and emerging clinical evidence. Studies in animal models consistently demonstrate the efficacy of agents such as dulaglutide, semaglutide, and liraglutide in reducing alcohol consumption and reversing neurobehavioral deficits associated with chronic alcohol exposure. Early-phase clinical trials and large-scale observational data suggest that GLP-1RAs—particularly semaglutide—may also reduce alcohol intake and alcohol-related hospitalizations in humans, with especially promising outcomes among individuals with comorbid obesity or metabolic disorders. While these findings highlight the potential of GLP-1RAs for repurposing in AUD treatment, further randomized clinical trials are needed to confirm causality, determine

optimal dosing, and identify patient subgroups most likely to benefit from this pharmacological strategy.

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