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GLP-1 RECEPTOR AGONISTS AND ALCOHOL USE DISORDER: PROMISE FOR A NEW TREATMENT APPROACH

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ABSTRACT

The role of glucagon-like peptide-1 receptor agonists (GLP-1RAs) as effective glucose-lowering and nephroprotective agents in type 2 diabetes is well established. Their emergence as treatments for obesity is considerably more recent. In the short time since this indication was approved, these medications have attracted considerable attention, and their use in clinical practise has expanded rapidly. Amid this rapid expansion, clinicians have encountered growing anecdotal reports from patients taking GLP-1RAs describing unexpected improvements in substance use behaviours. Such reports are consistent with a growing body of preclinical evidence demonstrating that GLP-1RAs reduce intake of alcohol and other addictive substances. Whether preclinical observations can be replicated in human studies is currently being tested in clinical trials. One prevailing hypothesis suggests that glucagon-like peptide-1 (GLP-1) exerts its effects via central mechanisms, including, at least in part, modulation of dopamine transmission. Studies imply that activation of central glucagon-like peptide -1 receptors (GLP-1Rs) functionally regulates the mesolimbic reward pathway and reduces addiction-like behaviors in rodent models. This narrative review summarizes the existing evidence regarding the effects of GLP-1RAs on alcohol consumption. Relevant studies published between January 2015 and March 2026 were identified via literature search. Collectively, the evidence supporting GLP-1RAs for the treatment of AUD is promising yet inconsistent, with randomized clinical trials yielding mixed results for the primary outcome of reduced alcohol consumption. Although current findings do not justify changes to clinical practice, they point toward potential benefits of GLP-1RAs, extending beyond glycemic and weight control, particularly among patients with AUD.

KEYWORDS

Endocrinology, Addiction, Alcohol, Neurobiology, GLP-1

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1. Introduction

According to a WHO report, substance use disorder (SUD) was attributable to 3.2 million deaths in 2019, with alcohol contributing to 2.6 milion deaths and psychoactive substances contributing to 0.6 milion deaths. The report also states that around 400 million people are struggling with alcohol use disorder (AUD) (1). Despite the significant disease and death toll caused by AUD, few effective pharmacotherapies are approved, and their use remains low (2). Current AUD treatment relies on integrated behavioral and medication-based strategies. Cognitive behavioral therapy, motivational interviewing, and contingency management are proven psychotherapies [3], while pharmacological options include acamprosate and naltrexone (4). Given the variable efficacy and side effect profiles of these medications, there is a pressing need to discover and develop additional treatment options.

One emerging contender for such a treatment are GLP-1RAs, which are primarily used to treat type 2 diabetes and obesity. These agents work by replicating the action of the endogenous GLP-1 hormone, which is released following food intake and plays a key role in regulating hunger and body weight. These agents promote insulin secretion, thereby lowering blood glucose levels, and also act on the stomach to suppress its emptying and enhance postprandial satiety (5). GLP-1Rs are present not only in the gastrointestinal tract, but also in the brain's mesolimbic system, a pathway responsible for the neural circuits governing reward and motivation, which modulates satiety and influences the drive to consume food. The same neural region is also involved in the development and persistence of addictive behaviors, including chronic substance use, as it shares overlapping neurobiological mechanisms (6). A growing body of evidence from preclinical rodent studies suggests that GLP-1RAs influence these neural pathways, resulting in decreased consumption of opioids, nicotine, and alcohol in animal models of SUD (7)(8)(9)(10)(11)(12)(13). As a result of these promising findings, recent years have seen a rise in clinical trials.

2. Methodology

A literature search in PubMed, Scopus, Web of Science, and Google Scholar was performed to identify studies published between January 2015 and March 2026. Searches terms included: “GLP-1 receptor agonist”, “liraglutide”, “semaglutide”, “tirzepatide”, “dulaglutide”, “exenatide” combined with “addiction”, “alcohol”, “alcohol use disorder”, “alcohol craving”. Systematic reviews, cohort studies, and randomized controlled trials were included. The reference sections of earlier review articles on this subject were also examined to uncover any additional studies that might be relevant. Studies were excluded based on the following criteria: off-topic content, use of social media posts as a data source, or publication in a language other than English. The remaining articles were reviewed to assess their relevance.

2. GLP-1 Physiology

GLP-1 belongs to the incretin family of hormones and is secreted in the gut by both the pancreas and intestinal L-cells following nutrient intake. Its actions include boosting insulin release from the pancreas, suppressing glucagon secretion, decreasing appetite, and delaying gastric emptying (5). By interacting with receptors situated on vagal afferent endings, GLP-1 amplifies signaling directed to the brainstem. The resulting activation of the nucleus of the solitary tract (NTS) lowers efferent vagal tone to the stomach (14)(15). The outcome is diminished gastric smooth-muscle activity, a delay in nutrient absorption, and a lesser elevation of blood glucose levels after a meal (16).

Beyond its peripheral sources, GLP-1 is generated within the central nervous system and its receptor-mediated signaling is critical for providing neuroprotective benefits (17). Its effects span multiple domains: it boosts cognitive functions in the hippocampus, dampens inflammatory responses and cell death, influences reward processing, encourages the birth of new neurons, and restricts feeding (17)(18). Within the brain, GLP-1 is produced in the NTS (Table 1.)and distributed via neuronal projections to areas that regulate homeostatic and hedonic feeding behaviours (19)(20). In both rodent and non-human primate models, hypothalamic regions, most notably the arcuate nucleus (ArcN), paraventricular nucleus (PVN), and lateral hypothalamus (LH) (Table 1.) exhibit dense expression of GLP-1Rs (19)(20)(21). Studies in rats have shown that direct activation of GLP-1Rs in the LH and PVN results in a significant reduction in the amount of food consumed (22)(23). Available evidence suggests that Exendin-4, a GLP-RA, suppresses overall metabolism and counteracts ghrelin-driven increases in metabolic drive by activating GLP-1R pathways in the ArcN and PVN (24)(25).

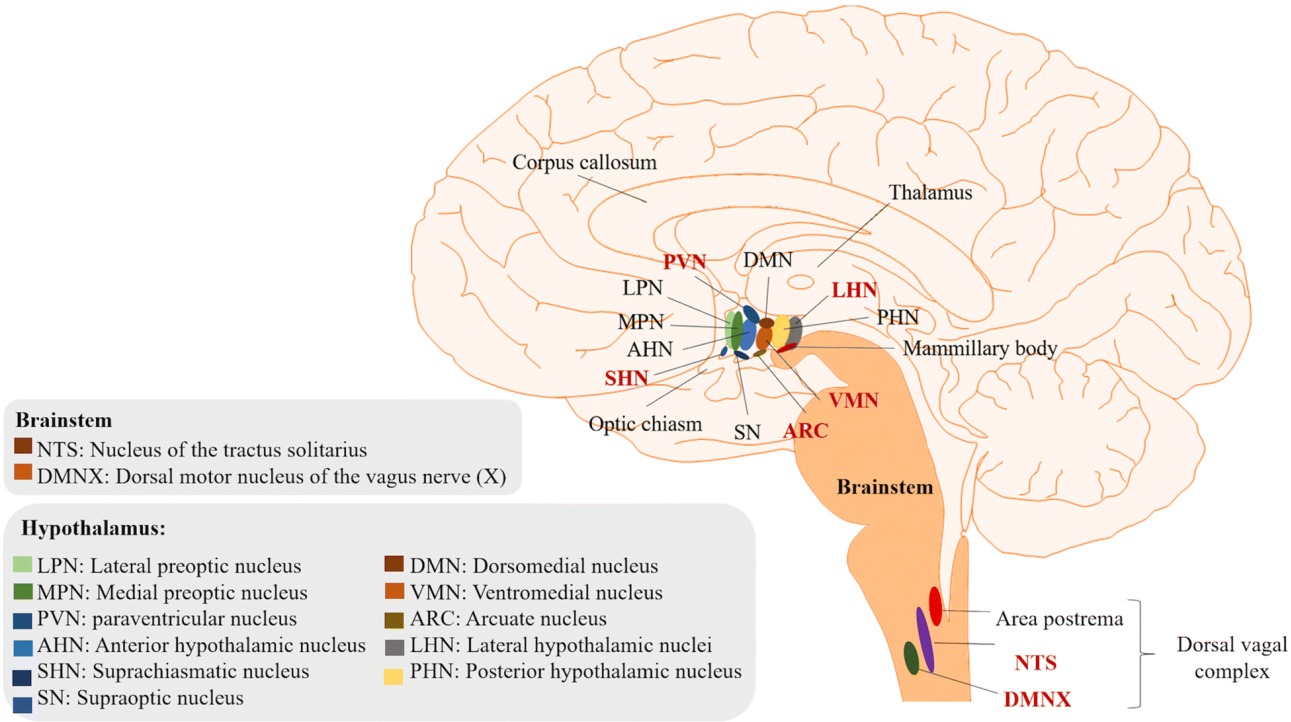

Fig. 1. *Anatomical Locations of Selected Hypothalamic and Brainstem Nuclei*

Note: Illustration depicting the regional anatomy of the hypothalamus and brainstem. Of the structures shown, the nucleus of the solitary tract (NTS), arcuate nucleus (ArcN), paraventricular nucleus (PVN), and lateral hypothalamus (LH) are established sites of GLP-1 receptor expression and signaling.

"Key hypothalamic nuclei and other areas involved in glucose homeostasis" by Amparo Güemes and Pantelis Georgiou. (2018). Review of the role of the nervous system in glucose homoeostasis and future perspectives towards the management of diabetes. *Bioelectron Med* **4**, 9 (2018). https://doi.org/10.1186/s42234-018-0009-4. Licensed under CC BY 4.0. To view a copy of this license, visit https://creativecommons.org/licenses/

3. Clinical Studies

In 2011, a scientific meeting abstract first described the potential of GLP-1RA to reduce alcohol intake in humans. This finding came from a cross-sectional review of patients with Type 2 diabetes following 3 months of treatment of liraglutide (26)(27).

In a subsequent study, genetic variation in the GLP-1R was found to be associated with AUD diagnosis in two cohorts of patients with AUD. Furthermore, the study showed a positive correlation between breath alcohol levels after intravenous alcohol self-administration and genetic variations in GLP-1R (28).

The first randomized clinical trial (RCT) was conducted by Klausen et al. (2022). A total of 127 patients were enrolled. Once-weekly exanetide combined with cognitive-behavioral therapy (CBT) did not result in a significant reduction in heavy drinking days compared with placebo plus CBT. However, a significant reduction in overall alcohol consumption and heavy drinking days was observed among patients with a body mass index (BMI) > 30 kg/m^2. Notably, the number of drinking days increased by 27.5% for patients with a BMI < 25 kg/m^2. Functional MRI and single-photon emission computed tomography (SPECT) brain scans were also performed in a subgroup. After 26 weeks of treatment, analyses revealed a significant reduction in cue-induced activity in the ventral striatum, dorsal striatum, and putamen in the exenatide group compared with the placebo group (29).

Another RCT, conducted by Probst et al. (2023), examined the effectiveness of dulaglutide as a therapy for smoking cessation and alcohol consumption. Among 255 participants, 151 consumed alcohol at baseline. After 12 weeks, patients receiving dulaglutide drank an estimated 29% less alcohol than the placebo group. However, in the subgroup of heavy drinkers, there was no significant difference observed between the placebo and the dulaglutide group (30).

Hendershot et al. performed a RCT with 48 participants, using low-dose semaglutide administered over 9 weeks. They found no significant difference between the placebo and treatment groups in average consumption per calendar day or in the number of drinking days. However, there was a significant reduction in drinks per drinking days, consumption during a post-treatment laboratory self-administration task, and the peak breath alcohol concentration (BrAC) (31).

A nationwide retrospective cohort study by Wium-Andersen et al. was performed using data on the entire Danish population from nationwide registers. Researchers compared the number of alcohol-related events (AREs) between a group of patients receiving GLP-1RAs and a group of patients receiving dipeptidyl peptidase-4 (DPP-4) inhibitors. Overall, the study consisted of 87.676 patients. The following AREs were included: hospital contacts with the main diagnosis of AUD; treatment for alcoholism; and purchases of medications for alcohol withdrawal syndrome or alcohol dependence. They observed a lower risk of AREs in the GLP-1RA group compared to the DPP-4 inhibitor group, with a 90-day hazard ratio (HR) of 0.46 and one year of treatment of 0.62 (32).

Retrospective cohort study by Wang et al. used patient electronic health records from the TrineX Research US Collaborative Network, which at the time of analysis contained 105.3 million patients. Researchers examined the associations between receipt of at least one prescription for semaglutide and the incidence or recurrence of AUD. Overall, after applying inclusion criteria and propensity-score matching, the study consisted of 86,270 patients, further divided into six cohorts. They were classified by obesity status, diagnosis of T2DM, and prior diagnosis of AUD. Control cohorts were taking other AUD medications, such as naltrexone and topiramate, as well as other non-GLP-1RA antidiabetic medications. The study showed significantly lower risk of recurrent AUD diagnosis in cohorts of patients with obesity and prior history of AUD who received semaglutide compared with those receiving naltrexone and topiramate at month 12 of observation (HR, 0.44). Moreover, in cohorts of people with T2DM, the group taking GLP-1RAs had significantly lower risk of both incident AUD and recurrent AUD (HR, 0.56 and HR: 0.61, respectively)

compared with non-GLP-1RA medications at month 12 of observation. Notably, in patients with T2DM, lower risks were observed in both patients with and without obesity (33).

Another retrospective cohort study by Qeadan et al. aimed to examine a similar association. They analyzed 136 American health systems in search of a correlation between GLP-1RAs and the incidence of alcohol intoxication and opioid overdose. Patients with a history of Opioid Use Disorder (OUD) (503,747 patients) and patients with a history of AUD (817,309 patients) were assessed for the presence of any prescription of a GLP-1RA in the electronic health records (8,103 in the OUD group and 5,621 in the AUD group). The researchers reported that among the cohort with AUD, patients with a GLP-1RA prescription had a 50% lower rate of incident alcohol intoxication compared to patients without such a prescription. The rate of alcohol intoxication per 10,000 patients among those without a GLP-1RA prescription was 280.85 at month 0 and declined to 201.47 at month 24 (a 28.26% decrease). By contrast, the GLP-1RA group had a much lower baseline rate (82.09) and decreased further to approximately 62.35 at month 24 (an approximate 24% decrease) (34).

Lähteenvuo et al. used Swedish nationwide electronic registries to examine the risk of hospitalization among patients with AUD taking GLP-1RAs, comparing periods of active GLP-1RA use with periods of nonuse within the same individuals. The study included 4321 semaglutide users, 2509 liraglutide users, and 1118 dulaglutide users. Semaglutide use was associated with the lowest risk of AUD hospitalization (adjusted hazard ratio [aHR], 0.64), followed by liraglutide (aHR, 0.72). Moreover, both medications were associated with a lower overall risk of all somatic hospitalizations (aHR, 0.78 for semaglutide and aHR, 0.79 for liraglutide). The dulaglutide group showed less favorable results, the risk of AUD hospitalization was only 4% lower (aHR, 0.96). Dulaglutide was also associated with a significantly higher risk of suicide (aHR, 1.38) (35).

In a retrospective cohort study based on electronic health records from the U.S. Department of Veterans Affairs, Farokhnia et al. compared Alcohol Use Disorder Identification Test-Consumption (AUDIT-C) scores across three groups: GLP-1RA recipients, DPP-4 inhibitor recipients, and unexposed individuals. They found that patients taking GLP-1RAs experienced a greater reduction in AUDIT-C scores compared with both unexposed individuals (difference-in-differences [DiD], 0.09) and DPP-4 inhibitor recipients (DiD, 0.11). The reductions in drinking were more pronounced among subgroups with higher baseline alcohol use. Among individuals with baseline AUD, GLP-1RAs use was associated with a greater reduction in AUDIT-C scores compared with unexposed individuals (DiD, 0.51) and compared with DPP-4 inhibitor recipients (DiD, 0.65). Similarly, among individuals with baseline hazardous drinking, GLP-1RA recipients showed a greater reduction than unexposed individuals (DiD, 1.38) and DPP-4 inhibitor recipients (DiD, 1.00) (36).

In a separate cohort study of 262 patients with obesity by O'Farrell et al., treatment with GLP-1RAs over 6 months led to a marked decrease in alcohol consumption. On average, weekly alcohol intake fell by 7.1 units, and a modest positive association was observed between the magnitude of alcohol reduction and the degree of weight loss. Baseline drinking levels averaged 11.8 ± 1.0 units/week (mean ± SEM), falling to 4.3 ± 0.5 units/week after intervention. Among those with higher baseline consumption (≥ 11 units/week), intake declined from 23.2 ± 1.8 units/week to 7.8 ± 0.9 units/week, a 68% decrease (37).

Additionally, Quddos et al. performed a small study consisting of 20 participants, measuring BrAC after alcohol consumption in patients taking GLP-1RAs. They detected a lower BrAC in the GLP-1RA group at 10, 15, and 20 minutes after consumption, and no significant difference between groups from 35 to 60 minutes after consumption. Moreover, cumulative BrAC, calculated as the area under the curve (AUC), was significantly lower in the GLP-1RA group. Furthermore, alcohol consumption increased the appetite score in the control group, but not in the GLP-1RA group (38).

4. Discussion

The use of GLP-1RAs as a treatment for AUD has yielded promising results, although these findings have been inconsistent, particularly among RCTs. While all trials completed to date have demonstrated positive effects on specific aspects of AUD-related behavior, results regarding the primary endpoint of reduced total alcohol intake have been mixed. In only one trial was treatment with a GLP-1RA (dulaglutide) associated with a reduction in overall alcohol consumption (30). Neither exenatide nor semaglutide produced a significant reduction in their respective primary endpoints of heavy drinking days or total drinks per calendar day (29)(31). In the exenatide trial, a reduction in alcohol consumption was observed only in a subgroup of patients with a BMI > 30 kg/m^2. Notably, however, exenatide increased the number of drinking days among patients with a BMI < 25 kg/m^2 (29). Subsequent studies did not replicate these findings, though it is important to note that the later trials comprised almost exclusively patients with elevated BMI. Whether baseline BMI modifies

treatment response remains unclear, as subsequent trials enrolled few participants with a normal BMI. The semaglutide RCT consisted of 27 patients with a BMI > 30 kg/m^2, 20 patients with a BMI between 25 and 30 kg/m^2, and only one patient with a BMI < 25 kg/m^2 (31). Similarly, the dulaglutide RCT consisted largely of patients with a BMI > 29.9 kg/m^2 (90.8% of participants) with only six patients having a BMI < 25 kg/m^2 (<4% of participants) (30). Consequently, the existing RCT evidence therefore leans heavily toward patients with overweight or obesity, a population in which GLP-1RAs may modulate the reward system differently compared with lean individuals.

Observational studies show more consistent, protective effects against alcohol-related events, alcohol-related hospitalizations and overall alcohol intake. Still, some caution is warranted. Unmeasured factors like socioeconomic status may still influence the observed associations, even when methods such as propensity-score matching are applied.

5. Conclusions

Although these findings do not support altering clinical practise at this time, they signal a possibility, that GLP-RAs may exert favourable effects beyond weight and glycemic control, especially among patients with AUD. For the time being, underutilized agents such as naltrexone and acamprostate should retain their position as first-line therapy. Future trials should emphasize stratification according to BMI, gender, alcohol use severity, and comorbidities to clarify which individuals are most responsive.

Conflicts of Interest: No conflicts of interest to declare.

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