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Semaglutide and Other GLP-1 Receptor Agonists as Potential Therapies for Addiction – Mechanisms, Evidence, and Clinical Implications

Katarzyna Wajda[1] https://orcid.org/0009-0008-6631-8923
Wiktoria Mika[2] https://orcid.org/0009-0007-6853-5342
Justyna Słowik[2] https://orcid.org/0009-0006-9219-8377
Izabela Sieradzka[2] https://orcid.org/0009-0004-3849-9844

[1]University of Rzeszów al. Tadeusza Rejtana 16C, 35-310 Rzeszów, Poland
[2]Provincial Clinical Hospital No. 2 St. Jadwiga the Queen in Rzeszów, Poland

Abstract

Objective: The aim of this article is to review and analyse the current state of knowledge on the potential of glucagon-like peptide-1 receptor agonists (GLP-1RAs), particularly semaglutide, in the treatment of substance use disorders (SUD). The analysis focuses on evaluating their effects on reducing craving and substance intake, as well as their potential as novel therapeutic interventions targeting neurobiological mechanisms of addiction.

Material and methods: A systematic literature review was conducted using PubMed, Scopus, and Google Scholar. Preclinical rodent studies and clinical/observational human studies evaluating the effects of GLP-1 receptor agonists, including semaglutide, on substance intake, craving, and relapse were included. Studies assessing impacts on dopaminergic pathways, the HPA axis, and neuroinflammation were also considered.

Results: Preclinical and clinical evidence indicates that GLP-1 receptor agonists, including semaglutide, reduce alcohol and drug intake, limit binge-like behaviors, and decrease relapse risk. Mechanisms likely involve modulation of dopaminergic pathways, regulation of the HPA axis, and reduction of neuroinflammatory processes. Clinical studies show decreased substance

craving, lower consumption, and reduced relapse, with a favorable safety and tolerability profile. Observational real-world data further support effectiveness across different patient populations and substances.

Conclusions: GLP-1 receptor agonists, represent a promising novel pharmacotherapeutic approach for AUD and potentially other SUDs. Despite encouraging results, larger, multi-center trials with longer follow-up are necessary to establish definitive efficacy, safety, and optimal dosing strategies.

Keywords: Semaglutide; GLP-1 receptor agonists; Substance use disorders; Alcohol use disorder; Novel pharmacotherapies.

1.Introduction

Addiction remains a chronic, relapsing disorder that poses a significant public health challenge worldwide. Alcohol use disorder (AUD) is among the most prevalent forms of addiction. Globally, approximately 7% of individuals aged 15 years and older – around 400 million people– struggle with AUD, of whom 3.7% (209 million) meet the criteria for alcohol dependence (WHO data). In Poland, according to EZOP II (Comprehensive Study on the Health Status of the Population and Its Determinants), around 583,000 individuals meet the criteria for alcohol dependence, representing approximately 1.5% of the total population, or nearly 2% of adults. When including those who drink harmfully but are not necessarily dependent, the total number of adults with alcohol-related disorders may reach 11% [21, 16].

Despite decades of research, available pharmacotherapies for alcohol use disorders demonstrate limited efficacy, with high relapse rates and unsatisfactory long-term outcomes. This

underscores the urgent need for novel interventions targeting the neurobiological mechanisms of addiction [13].

In recent years, growing attention has focused on the gut–brain axis and the role of glucagon-like peptide-1 (GLP-1), an incretin hormone primarily recognised for its role in glycaemic control in patients with diabetes and in the management of obesity. Beyond its metabolic functions, GLP-1 exhibits neuromodulatory effects via receptors located in brain regions responsible for reward processing, including the ventral tegmental area (VTA), nucleus accumbens (NAc), and prefrontal cortex [1].

Preclinical studies indicate that GLP-1 receptor activation attenuates dopamine release induced by psychoactive substances, reduces reward reinforcement, and diminishes behaviours resembling substance craving [4]. GLP-1 receptor agonists (GLP-1RAs), such as exenatide, liraglutide, and particularly semaglutide, appear to represent a promising avenue for addiction therapy. In animal models, these agents consistently reduce the intake of alcohol, nicotine, opioids, and psychostimulants. Observational data in humans, particularly among patients treated for obesity or diabetes, suggest decreased consumption of alcohol and other substances in individuals receiving GLP-1RAs. Early clinical studies further corroborate these findings—semaglutide significantly reduced alcohol intake and craving in patients with AUD [15].

In summary, current evidence indicates that GLP-1RAs constitute a novel and biologically rational therapeutic strategy for addiction treatment. Nevertheless, the translation of research findings into clinical practice remains at an early stage. Questions remain regarding the long-term efficacy of these therapies, their tolerability, potential comorbid psychiatric disorders, and ethical considerations associated with their use. This article reviews the mechanisms of action, available preclinical and clinical evidence, and clinical implications of GLP-1RA use, with particular emphasis on semaglutide as a potential breakthrough in the treatment of AUD and other substance use disorders.

2.Materials and Methods

The present literature review was conducted to evaluate the potential of GLP-1 receptor agonists, including semaglutide, as a treatment for addiction. Literature searches were performed in three major databases: PubMed, Scopus, and Google Scholar. Publications from 2003 to 2025 were considered, allowing the inclusion of both early studies on the mechanisms of incretin-based drugs and the most recent clinical reports from 2024–2025. The search strategy combined terms related to metabolic pharmacology with concepts from addiction psychiatry. English-language search terms included, among others, “GLP-1 receptor agonists,” “Semaglutide,” “Exenatide,” and “Liraglutide,” which were combined using the logical operator AND with addiction-related terms such as “Substance use disorders,” “Alcohol use disorder,” “Addiction,” and “Craving.”

A variety of study types were included to provide a comprehensive overview. Preclinical experiments in rodent models (mice and rats) assessing the effects of GLP-1 agonists on substance intake, binge-like behaviors, and relapse risk were considered. Clinical studies, both

randomized and observational, evaluating the efficacy of these drugs in reducing craving, limiting consumption, and preventing relapse were also included. Particular attention was given to their effects on dopaminergic pathways, HPA axis function, and neuroinflammatory processes.

Publications focusing solely on metabolic effects in the treatment of diabetes or obesity, without reference to substance-related behaviors, were excluded. Case reports and studies not available in full text in English or Polish were also omitted.

Due to the heterogeneity of study models and types of substances, a formal meta-analysis was not performed. Instead, a narrative synthesis of the results was applied, allowing coherent integration of preclinical evidence with clinical observations.

3.Neurobiological Mechanisms

GLP-1 receptor agonists (GLP-1RAs) influence multiple central nervous system mechanisms that play a key role in the pathophysiology of addiction. A primary mechanism involves modulation of the mesolimbic dopamine system through activation of GLP-1 receptors in the ventral tegmental area (VTA) and the nucleus accumbens (NAc). This leads to reduced dopamine release in response to rewarding stimuli, including alcohol and drugs of abuse [6, 3]. The effect translates into a reduction of subjective craving, decreased reward sensitivity, and lower impulsivity, as observed in animal studies [17, 13].

Another important aspect is the interaction of GLP-1 with the hypothalamic–pituitary–adrenal (HPA) axis. GLP-1 has been shown to enhance HPA axis activity and increase corticosterone release, potentially modulating stress responses and thereby influencing relapse vulnerability [10].

Additionally, GLP-1RAs exhibit anti-inflammatory effects in the central nervous system, reducing microglial activation and pro-inflammatory cytokine levels, which may support the restoration of homeostasis in neuronal circuits disrupted by chronic substance use [19]. Synaptic-level studies also suggest that GLP-1RAs can improve synaptic plasticity in brain regions involved in reward control and impulse regulation, potentially facilitating the recovery of adaptive neural pathways [13].

Compared with currently used addiction pharmacotherapies—such as naltrexone, acamprosate, or bupropion—which primarily act through opioid receptor blockade, glutamatergic modulation, or monoaminergic systems, GLP-1RAs represent a novel approach, integrating metabolic, dopaminergic, and neuroimmune effects [19, 12].

4.Preclinical Evidence

Over the past decade, numerous experiments using animal models have provided strong support for the hypothesis that GLP-1 receptor agonists (GLP-1RAs) modulate addictive behaviours. Studies in rats and mice have shown that exogenous stimulation of the GLP-1 receptor with exendin-4 (Ex-4), liraglutide, or semaglutide significantly reduces voluntary alcohol intake,

both in two-bottle choice paradigms and intravenous self-administration models [3, 10, 7]. Observed effects included reductions in total ethanol consumption, decreased episodes of binge drinking, and prolonged time to relapse in alcohol-seeking behaviour following periods of abstinence.

Thomsen et al. demonstrated that Ex-4 administration in a model of alcohol relapse prevented the reinstatement of seeking behaviours, indicating an effect on motivational mechanisms and context-dependent reward memory [19]. Similar effects were observed in cocaine studies, where self-administration models showed reduced lever presses in operant chambers and decreased cocaine-induced locomotor activity [17]. Additionally, GLP-1RAs modulated dopamine levels in the nucleus accumbens, reducing dopamine peaks following substance exposure, suggesting a direct influence on the reward circuitry.

Some studies also examined the effects of GLP-1RAs on other compulsive behaviours, such as excessive consumption of high-calorie foods, further confirming their ability to regulate reward-driven behaviour. Collectively, preclinical data indicate that GLP-1RAs act on both metabolic and neurobiological levels, offering a multidimensional approach to treating substance use disorders – Table 1.

Table 1. Selected preclinical studies of GLP-1RAs in addiction. [6, 3, 17, 10, 19]

Authors	Species/Model	Drug	Findings
Egecioglu et al., 2013[3]	Rats, two-bottle preference	Exendin-4	↓ total alcohol intake, ↓ ethanol preference
Liu et al., 2024 [14]	Mice, two-bottle preference	Liraglutide	↓ alcohol consumption, ↓ alcohol preference, ↓ relapse drinking
Thomsen et al., 2017[19]	Mice, reinstatement	Exendin-4	Prevented relapse after alcohol priming
Sørensen et al., 2016[17]	Rats, IV self-administration	Exendin-4	↓ lever presses, ↓ ethanol intake
Hernandez et al., 2023[6]	Mice, cocaine SA	Semaglutide	↓ motivation, ↓ locomotor activity, ↓ dopamine peak in NAc

5. Clinical Studies and Emerging Data

Clinical studies investigating the use of GLP-1 receptor agonists (GLP-1RAs) in the treatment of addiction are still at an early stage, but the results are promising. The potential effects of GLP-1RAs on reducing alcohol consumption in humans were first reported in a conference abstract in 2011, in a cross-sectional review of patients with type 2 diabetes treated with liraglutide for three months [8].

In a randomised, double-blind, placebo-controlled trial, patients with diagnosed alcohol use disorder (AUD) received once-weekly exenatide for 26 weeks. This study observed a reduction in reward system reactivity measured by fMRI and a decrease in heavy drinking days in the

subgroup of patients with obesity [11]. A pilot study published in 2024 showed that semaglutide significantly reduced subjective alcohol craving and alcohol consumption during controlled self-administration sessions [2].

Hendershot et al. (2025) provided preliminary prospective evidence that low-dose semaglutide can reduce alcohol craving and affect certain drinking outcomes, supporting the need for larger clinical trials to assess the efficacy of GLP-1RAs in AUD treatment. The study included a small sample of 48 participants, 71% of whom were women, randomly assigned to treatment groups. Low-dose semaglutide reduced alcohol consumption during a laboratory self-administration task, showing medium-to-large effects for grams of alcohol consumed and peak breath alcohol concentration. Treatment did not affect the average number of drinks per day or total drinking days in the calendar, but significantly reduced drinks on drinking days and weekly alcohol craving. Semaglutide treatment also predicted greater reductions in heavy drinking episodes over time compared with placebo. A significant treatment-time interaction indicated that semaglutide also predicted a relative reduction in daily cigarette consumption in current smokers [5].

Wang et al. (2024) conducted a retrospective cohort study including 83,825 patients with obesity, comparing semaglutide with other anti-obesity medications. Results showed that GLP-1RA use was associated with a 50–56% lower risk of incident and recurrent AUD over a 12-month follow-up. Similar findings were replicated in a study population of 598,803 patients with type 2 diabetes. These data provide evidence of the potential benefits of semaglutide in AUD in real-world populations [20].

Supplementary data from observational and registry analyses indicate that patients receiving GLP-1RAs, particularly semaglutide, have a lower risk of AUD relapse and fewer alcohol-related hospitalisations compared with patients treated with other anti-obesity or anti-diabetic medications [20,18]. Overall, clinical and observational evidence suggests that GLP-1RAs may represent a promising therapeutic option for AUD, but larger, multicentre trials with extended follow-up are needed to confirm the efficacy and safety of this drug class – Table 2.

Table 2. Selected clinical studies and observational data on GLP-1RAs in addiction.

Study	Design	Population	Intervention	Duration	Outcomes
Klausen et al., 2022[12]	RCT, placebo-controlled	AUD, n≈127	Exenatide once weekly vs placebo	26 weeks	↓ fMRI cue reactivity in reward regions; ↓ heavy drinking days in BMI>30 subgroup
Hendershot et al., 2025[5]	RCT, pilot	AUD, n=48	Semaglutide 2.4 mg s.c. once weekly vs placebo	12 weeks	Significant ↓ craving; ↓ alcohol consumption in controlled self-administration session
Wang et al.,	Retrospective	>80,000 patients	Semaglutide vs other anti-	12 months	↓ risk of incident and recurrent AUD (~50%)

2024[20]	cohort (EHR)	with obesity	obesity drugs		
National registries, 2024–2025[18]	Case crossover / cohorts	Hundreds of thousands	Semaglutide/liraglutide	Various	Associated with ↓ AUD/SUD-related hospitalisations

6. Semaglutide in Focus

Semaglutide, a long-acting GLP-1 receptor agonist, offers several pharmacokinetic advantages over earlier drugs in this class. Its extended half-life allows once-weekly administration, which may improve adherence to treatment. It also demonstrates strong penetration into the central nervous system, potentially enhancing its modulatory effects on brain reward circuits.

Preliminary preclinical and clinical studies suggest that semaglutide may reduce drug craving and decrease substance use, particularly in alcohol use disorder. This effect appears to occur through attenuating responses to cues (e.g., the sight of alcohol) and modulating dopaminergic signalling.

However, challenges remain, including tolerability issues (e.g., nausea and other gastrointestinal side effects), the presence of co-occurring psychiatric disorders in patients with addiction, and significant ethical implications. These include the widespread off-label use of the drug, which raises questions about safety and liability. Furthermore, the high cost of therapy may lead to widening inequalities in healthcare access, limiting the availability of the treatment for patients with a lower socioeconomic status. Further large-scale clinical trials are necessary to fully evaluate semaglutide's efficacy and safety profile in addiction treatment [20].

7. Clinical and Ethical Implications

Although GLP-1 receptor agonists, such as semaglutide, represent a novel and promising avenue for addiction treatment, their path to widespread clinical use remains long. The primary challenge is the lack of large, well-designed human clinical trials that definitively confirm their efficacy and safety.

Furthermore, a comprehensive understanding of the mechanisms underlying their effects in the context of addiction still requires further investigation, as their action may vary depending on the specific substance of abuse and even between individual patients. Unresolved questions also remain regarding sex-based differences in treatment response and potential genetic variants that could influence therapeutic outcomes.

Despite these challenges, the potential future approval of oral formulations of GLP-1 agonists could substantially improve patient adherence to treatment, which is critical in managing chronic conditions such as addiction. Overall, GLP-1 agonists offer a promising new direction, but intensive research is required to fully realise their clinical potential [11].

8. Conclusions

GLP-1 receptor agonists, including semaglutide, represent a promising class of medications for the treatment of substance use disorders. Preclinical studies have demonstrated that stimulation of GLP-1 receptors in reward-related brain regions, such as the ventral tegmental area and nucleus accumbens, reduces the intake of alcohol and other psychoactive substances and suppresses relapse-like seeking behaviours. These effects are mediated through modulation of the mesolimbic dopaminergic system, influence on the hypothalamic–pituitary–adrenal (HPA) axis, reduction of neuroinflammation, and enhancement of synaptic plasticity.

Although clinical evidence remains limited, available data suggest that GLP-1 receptor agonists may reduce substance craving and decrease alcohol consumption, as shown in both pilot randomised trials and observational studies. Semaglutide stands out due to its favourable pharmacokinetic properties, including a long half-life, once-weekly dosing, and strong penetration into the central nervous system, which may enhance its effectiveness in modulating addictive behaviours.

Challenges remain regarding drug tolerability, the presence of coexisting psychiatric disorders, and use beyond approved indications. There is an urgent need for larger, multicentre clinical trials with extended follow-up and objective endpoints to definitively evaluate the efficacy, safety, and optimal treatment regimen of GLP-1 receptor agonists in addiction therapy.

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