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NARRATIVE REVIEW

The Potential of Glucagon-like Peptide-1 Receptor Agonists in Addictive Disorders

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ABSTRACT

Addictive disorders are often difficult to treat and show high relapse rates. Particularly, alcohol use disorder presents a major treatment challenge leading to severe physical and mental health impacts. So far, pharmacological treatment options are limited. Agonists of the glucagon-like peptide-1 receptor are anti-hyperglycemic and weight-reducing drugs that have anti-inflammatory properties and can modulate brain reward pathways. Mounting evidence suggests that glucagon-like peptide-1 receptor agonists may have the potential to treat addictive disorders. Preclinical research demonstrated that glucagon-like peptide-1 receptor agonists influence addiction primarily by reducing dopaminergic activation in brain reward pathways. The aim of this review is to give an insight into the neurobiological mechanisms by which glucagon-like peptide-1 receptor agonists influence addiction and to evaluate their effectiveness across different substance use disorders and behavioral addictions in humans.

Keywords: Addictive disorders, glucagon-like peptide-1 receptor agonists, alcohol use disorder, brain reward pathways, behavioral addictions.

1. The burden of addictive disorders

Substance addiction is characterized by a recurrent desire to continue taking a psychoactive substance despite negative consequences[1]. Alcohol use disorder (AUD) is the most prevalent substance use disorder (SUD) worldwide, with higher rates in high-income countries[2-4]. The most common drug use disorders are cannabis dependence and opioid dependence, while amphetamine dependence and cocaine dependence are less common[2]. To the best of our knowledge, SUDs, particularly AUD, are often difficult to treat, have a high relapse risk, lead to severe physical and mental health impacts, and contribute essentially to the burden of global disease[5,6]. There is a very high prevalence of alcohol-associated liver disease (ALD) and alcohol-associated cirrhosis in individuals with AUD[7]. Up to 50% of people with ALD are also suffering from obesity[8]. Over the last 20 years there was a 14.66% increase in AUD, a 38.68% increase in ALD, and a 94.12% increase in alcohol-attributable primary liver cancer prevalence worldwide[9]. AUD is strongly associated with other mental illnesses, particularly with mood disorders[10-12]. Their co-occurrence as dual disorders may affect their course, severity and treatment outcomes[10]. There is evidence that AUD is associated with a higher mortality and suicide risk[13,14]. A recent meta-analysis indicated that alcohol use is associated with a 94% increase in the risk of death by suicide[15].

To date, only a few medications have been approved to treat AUD, the NMDA-modulator acamprosate, the acetaldehyde dehydrogenase inhibitor disulfiram, and the opioid antagonists naltrexone and nalmefene. Medications to treat AUD in combination with behavioral treatment have shown a modest effect in reducing alcohol consumption and improving abstinence rates[16]. Medication for AUD is often prescribed with caution and still underutilized because of adverse events, no long-term efficacy, malcompliance, and high withdrawal rates[16,17]. Therefore, there is a high need for a better understanding of the neurobiological mechanisms involved in AUD and further investigation of new treatment targets for the development of novel pharmacotherapies to treat AUD safely and effectively.

Mounting evidence suggests that agonists of the glucagon-like peptide-1 receptor (GLP-1RAs) may have a great potential to treat addictive disorders by targeting brain reward pathways[8,18]. The aim of this review is to give an insight into the neurobiological mechanism of action of GLP-1RAs in addictive disorders and to evaluate their effectiveness across different substances and addictive behaviors.

Table 1: Common substance and behavioral addictions

Common Substance Addictions	Common Behavioral Addictions
Alcohol	Gambling
Nicotine	Food
Cannabis	Work
Opioids	Sex
Stimulants (cocaine, amphetamine)	Gaming
Hallucinogens	Internet
Sedative and Tranquilizer	Social media
Caffeine	Shopping

2. Neurobiological mechanisms by which glucagon-like peptide-1 receptor agonists influence addiction

To the best of our knowledge, SUDs are characterized by a dysregulation in dopaminergic signaling within the mesolimbic reward system, leading to compulsive drug use despite negative consequences[19,20].

GLP-1RAs are an innovative class of antidiabetic agents that have also shown great effectiveness in reducing body weight and cardiovascular risk. Due to their anti-inflammatory and antioxidative properties, GLP-1RAs may provide great therapeutic potential with broad clinical implications. The most commonly used GLP-1RAs are exenatide, dulaglutide, liraglutide, and semaglutide, differing in their molecular structures

and half-lives. Tirzepatide is the newest GLP-1RA with an additional agonism on the glucose-dependent insulinotropic polypeptide receptor.

GLP-1 is an incretin hormone that is synthesized in the intestine and central nervous system (CNS) and stimulates postprandial insulin secretion. GLP-1RAs have the same mechanism of action. They are able to cross the blood-brain barrier (BBB) and exert a direct effect on brain function. Neurons with GLP-1 receptors are distributed along addiction-relevant brain areas, such as the prefrontal cortex, the ventral tegmental area, the nucleus accumbens, and hypothalamic subnuclei[21,22]. Their activation has an impact on glutamatergic, dopaminergic, and GABAergic neurotransmission[22]. Shared neural mechanisms between the food reward system and AUD pathways are well established[23,24]. There is evidence from animal models that GLP-1RAs have a modulating effect on the release of hunger- and reward-related neurotransmitters in the striatum and hypothalamus[25]. A recent animal study showed that semaglutide can bind to the nucleus accumbens, decreasing alcohol-induced dopamine increase in alcohol-drinking rats[26]. Thus, GLP-1RAs may influence addiction primarily by reducing dopaminergic activation in brain reward pathways, thereby attenuating the pleasurable effects and cravings for psychoactive substances[22,26,27].

It is well established that GLP-1RAs have anti-inflammatory properties[28-30]. The treatment with GLP-1RAs leads to a reduction of microglial activation, resulting in a decrease of the production of pro-inflammatory cytokines[31]. Through anti-inflammatory effects GLP-1RAs can reduce neuroinflammation and oxidative stress and help to restore brain function disrupted by chronic substance abuse, especially in reward pathways[32]. As GLP-1RAs have an impact on the hypothalamic-pituitary-adrenal (HPA)-axis, they can reduce cravings for addictive substances and stress-induced relapses[33].

3. The impact of glucagon-like peptide-1 receptor agonists on substance use disorders

A recent double-blind randomized controlled trial (RCT) that investigated the effect of exenatide once weekly in combination with standard cognitive-behavioral therapy on AUD showed that the treatment with exenatide for 26 weeks did not lead to a significant reduction of heavy drinking days compared to placebo[34]. However, in a subgroup of patients with obesity (BMI ≥ 30 kg/m^2) a significant reduction in heavy drinking days ($p = 0.034$) and total alcohol intake was detected. In addition, a significant attenuation of fMRI alcohol cue reactivity in the ventral striatum and septal area, which represent important brain areas for reward and addiction, was found in patients treated with exenatide[34]. A double-blind RCT showed that after 12 weeks of treatment, participants receiving dulaglutide consumed 29% less alcohol than participants on placebo[35]. The study population was rather obese (BMI ≥ 30 kg/m^2) and the major part of participants suffered from mild or moderate AUD. The subgroup of heavy drinkers was too small to provide valuable results[35]. A very recent double-blind RCT revealed that 9 weeks of low-dose semaglutide treatment significantly reduced the amount of daily drinks and weekly alcohol craving[36]. However, the study examined a small sample during a short treatment duration, and AUD severity was mild to moderate[36]. Several other studies indicated that GLP-1RAs may lead to a reduction of alcohol consumption[37-39], cannabis use[40], nicotine consumption[41,42], opioid use[43], and cocaine use[44]. The RCT by Yammine et al. (2021) showed that exenatide led to a significantly greater abstinence rate, reduction of craving and withdrawal symptoms, and lower post-cessation weight than placebo in persons suffering from nicotine dependence. Another recent RCT indicated that after treatment with dulaglutide, craving for smoking declined without a significant difference to placebo[42]. Dulaglutide significantly decreased post-cessation weight compared to placebo[42]. A recent retrospective cohort study revealed that semaglutide was associated with a significantly lower risk for incident cannabis use disorder diagnosis (CUD) in patients with no prior history of CUD and a significantly lower risk for recurrent CUD in patients with a prior history of CUD compared to non-GLP-1RA anti-diabetes medications in patients with type 2 diabetes and/or obesity for a 12-month follow-up period[40]. A recent retrospective cohort study indicated that patients with GIP/GLP-1RA prescriptions had significantly lower rates of opioid overdose in persons with opioid use disorder[43]. A case series showed that the treatment

with 2 mg exenatide for 6 weeks was safe and led to decreased cocaine craving and positive affect in 3 patients with cocaine use disorder[44]. These retrospective findings are very promising and suggest the conduction of larger clinical trials to investigate the effects of GLP-1RAs in patients with cannabis, cocaine, and opioid use disorders.

4. The effect of glucagon-like peptide-1 receptor agonists in behavioral addictions

Behavioral addiction is characterized by a persistent urge to engage in a behavior that produces natural reward[1]. Behavioral addictions include pathological gambling, food addiction, pathological gaming, excessive shopping, and work and sex addiction[1] (see figure 2). Addictive behaviors can be just as disruptive as substance addictions, causing cravings, withdrawal, and affecting daily functioning, quality of life, relationships, and self-esteem.

There is evidence that GLP-1RAs are showing promise in treating not just substance addictions but also behavioral addictions by targeting shared neurobiological mechanisms involved in reward and motivation[45]. GLP-1RAs target the mesolimbic dopamine system, reducing dopamine release in reward centers, thereby decreasing motivation, cravings, and impulses for addictive behaviors[22,45]. Moreover, GLP-1RAs have strong effects on central satiety by activating receptors in the gut and in the brain, especially in the hypothalamus and the brainstem, reducing appetite, increasing satiety, dampening cravings, leading to reduced food intake and weight loss[46].

A recent retrospective study explored the effects of GLP-1RAs on substance and behavioral addictions by analyzing data from various social platforms, including 5859 threads and comments about GLP-1RAs[45]. Regarding behavioral addictions, 21.35% of comments reported a compulsive shopping cessation[45]. During the treatment with GLP-1RAs, an increase in sexual drive and libido was observed in several users[45]. A recent observational study showed that after 4 months of treatment with semaglutide, the prevalence of food addiction decreased from 57.5% to 4.2% ($p < 0.001$) suggesting that semaglutide is an effective medication to improve food addiction in people with obesity[47]. There is evidence that patients receiving semaglutide showed a significant improvement in binge eating disorder compared to patients treated with other anti-obesity medications[48].

Therefore, GLP-1RAs offer a novel approach to addiction, showing potential for both SUDs and behavioral addictions. There is a need for larger clinical trials to better understand their way of action and to investigate their long-term safety and efficacy in addiction treatment.

Table 2: Clinical studies on the use of glucagon-like peptide-1 receptor agonists in patients with addictive disorders

Study	Study Design	Sample size	Addictive disorder	GLP-1RA/ Control	Outcomes
Angarita et al., 2021[49]	RCT	13	Cocaine use disorder	Exenatide 5mcg once 3 hours before cocaine self-admini-stration / Placebo	No significant difference in cocaine infusions, self-reported euphoria, and cocaine craving.
Arillotta et al., 2024[45]	Retrospective study from social media using a mixed-methods approach	5859 threads and comments were extracted	Alcohol, caffeine, nicotine, cannabis, psychostimu - lants, shopping, sex drive/libido	GLP-1RAs (not specified)	29.75% of alcohol-related, 22.22% of caffeine- related, 23.08% of nicotine-related comments stated cessation of the substance following GLP-1RAs use. Limited results for cannabis, cocaine and psychostimulants. 21.35% of comments reported compulsive shopping

Study	Study Design	Sample size	Addictive disorder	GLP-1RA/ Control	Outcomes
					cessation. Libido increased in some cases.
Hendershot et al., 2025[36]	RCT	24/24	AUD	Semaglutide 0.25-1mg weekly, 9 weeks / Placebo	Semaglutide significantly reduced alcohol consumption during a post-treatment laboratory self-administration task **p=0.01**, drinks per drinking day **p=0.04**, alcohol craving **p=0.01**, and heavy drinking over time **p=0.04**.
Klausen et al., 2022[34]	RCT	62/65	AUD	Exenatide 2mg weekly, 26 weeks / Placebo	No significant difference in AUDIT-scores, heavy drinking days, and suicidal behavior; Significant reduction in heavy drinking days only in patients with BMI ≥ 30 kg/m^2 **p=0.034**
Lengsfeld et al., 2023[42]	RCT	127/128	Nicotine dependence	Dulaglutide 1.5mg weekly, 12 weeks / Placebo	Craving for smoking declined without a significant difference to placebo.
Nicolau et al., 2024[47]	Prospective observational study	113	Food addiction	Semaglutide weekly, 4 months	Prevalence of food addiction diminished from 57.5% to 4.2% **p<0.001.**
Probst et al., 2023[35]	RCT	76/75	Nicotine dependence ; AUD	Dulaglutide 1.5mg weekly, 12 weeks / Placebo	Dulaglutide led to a significant reduction of alcohol intake **p=0.04.**
Qeadan et al., 2025[43]	Retrospective cohort study	503747 with OUD 817309 with AUD	OUD and AUD	GIP/GLP-1RA prescription	Patients with GIP/GLP-1RA prescriptions had significantly lower rates of opioid overdose and alcohol intoxication compared to those without.
Quddos et al., 2023[38]	Retrospective cohort study	56/50/47	Current alcohol drinker	Semaglutide 1mg weekly, tirzepatide 7.5mg weekly, ≥ 30 days/ no medication	Significantly lower self-reported alcohol intake, drinks per drinking episode, binge drinking odds, AUDIT-scores in the semaglutide or tirzepatide group compared to control group.
Richards et al., 2023[37]	Retrospective case series	6	AUD	Semaglutide weekly	Significant reduction in AUD symptoms based on AUDIT score **p<0.001.**
Wang et al., 2024[40]	Retrospective cohort study of electronic health records	85.223 with obesity	CUD	Semaglutide once weekly / non-GLP-1RA anti-obesity medications	Semaglutide was associated with a significantly lower risk for incident CUD diagnosis in patients with no prior history, and a significantly lower risk for recurrent CUD diagnosis in patients with a prior history for a 12-months follow-up period.

Study	Study Design	Sample size	Addictive disorder	GLP-1RA/ Control	Outcomes
Wang et al., 2024[40]	Retrospective cohort study of electronic health records	596.045 with type 2 diabetes	CUD	Semaglutide once weekly / non GLP-1RA anti-diabetes medications	Semaglutide was associated with a significantly lower risk for incident CUD disorder diagnosis in patients with no prior history, and a significantly lower risk for recurrent CUD diagnosis in patients with a prior history for a 12-months follow-up period.
Yammine et al., 2021[41]	RCT	41/41	Nicotine dependence	Exenatide 2mg weekly, 6 weeks + NRT / Placebo + NRT	Exenatide led to a significantly higher abstinence rate, reduction of craving and withdrawal, and lower post-cessation weight.
Yammine et al., 2023[44]	Retrospective case series	3	Cocaine use disorder	Exenatide 2mg weekly, 6 weeks	Treatment was safe, feasible and led to decreased cocaine craving and positive affect.

Legend: AUD: alcohol use disorder; CUD: cannabis use disorder; GIPR: glucose-dependent insulinotropic polypeptide-1 receptor; GLP-1RA: glucagon-like peptide-1 receptor agonist; NRT: nicotine replacement therapy; OUD: opioid use disorder; RCT: randomized controlled trial.

5. Conclusion

Growing evidence suggests that GLP-1RAs are promising addiction treatments by influencing crucial interconnected neurobiological mechanisms contributing to addiction, such as brain reward pathways, the HPA-axis, and neuroinflammation[22,32,33].

Preclinical research demonstrated that GLP-1RAs influence addiction primarily by reducing dopaminergic activation in brain reward pathways, thereby reducing the pleasurable effects, substance-seeking behavior, and cravings for various psychoactive substances[22,26,27]. Mounting clinical literature suggests that GLP-1RAs may have the potential to reduce the consumption and craving for addictive substances in humans, particularly for alcohol and nicotine[18,38,42,50,51]. There is evidence from RCTs that GLP-1RAs led to a greater reduction of alcohol consumption and craving than placebo, especially in individuals suffering from obesity[34-36]. As yet, the off-label use of GLP-1RAs to treat AUD is not justified. It is still not known whether GLP-1RAs are safe and effective for treating more severe AUD and whether the reduction in alcohol consumption will persist in the long-term.

There is a need for more data on long-term outcomes of GLP-1RAs therapy targeting treatment challenges, such as drug tolerability and comorbid mental illnesses and effects on different substances and behavioral addictions. Large-scale and long-term RCTs are required to establish the safety, efficacy, and optimal dosing strategies for GLP-1RAs in addiction treatment.

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References:

1. Zou Z, Wang X, Wang H, et al. Definition of Substance and Non-substance Addiction. *Adv Exp Med Biol.* 2025;1495:3-25.

2. Alcohol GBD, Drug Use C. The global burden of disease attributable to alcohol and drug use in 195 countries and territories, 1990-2016: a systematic analysis for the Global Burden of Disease Study 2016. *Lancet Psychiatry.* 2018;5(12):987-1012.

3. Nehring SM, Chen RJ, Freeman AM, Doerr C. Alcohol Use Disorder: Screening, Evaluation, and Management (Nursing). In: *StatPearls.* Treasure Island (FL) ineligible companies. Disclosure: Richard Chen declares no relevant financial relationships with ineligible companies. Disclosure: Andrew Freeman declares no relevant financial relationships with ineligible companies. Disclosure: Chaddie Doerr declares no relevant financial relationships with ineligible companies.2025.

4. Moubchiri CE, Chahboune M, Guennouni M, Hilali A. The prevalence of tobacco, alcohol, stimulant, khat, and cannabis use among school-going students in African and Arab countries: a systematic review and meta-analysis. *Osong Public Health Res Perspect.* 2025;16(1):28-41.

5. Rehm J, Gmel GE, Sr., Gmel G, et al. The relationship between different dimensions of alcohol use and the burden of disease-an update. *Addiction.* 2017;112(6):968-1001.

6. Peacock A, Leung J, Larney S, et al. Global statistics on alcohol, tobacco and illicit drug use: 2017 status report. *Addiction.* 2018;113(10):1905-1926.

7. Amonker S, Houshmand A, Hinkson A, Rowe I, Parker R. Prevalence of alcohol-associated liver disease: a systematic review and meta-analysis. *Hepatol Commun.* 2023;7(5).

8. Subhani M, Dhanda A, King JA, et al. Association between glucagon-like peptide-1 receptor agonists use and change in alcohol consumption: a systematic review. *EClinicalMedicine.* 2024;78:102920.

9. Danpanichkul P, Diaz LA, Suparan K, et al. Global epidemiology of alcohol-related liver disease, liver cancer, and alcohol use disorder, 2000-2021. *Clin Mol Hepatol.* 2025;31(2):525-547.

10. Lai HM, Cleary M, Sitharthan T, Hunt GE. Prevalence of comorbid substance use, anxiety and mood disorders in epidemiological surveys, 1990-2014: A systematic review and meta-analysis. *Drug Alcohol Depend.* 2015;154:1-13.

11. Hunt GE, Malhi GS, Cleary M, Lai HM, Sitharthan T. Prevalence of comorbid bipolar and substance use disorders in clinical settings, 1990-2015: Systematic review and meta-analysis. *J Affect Disord.* 2016;206:331-349.

12. Hunt GE, Malhi GS, Lai HMX, Cleary M. Prevalence of comorbid substance use in major depressive disorder in community and clinical settings, 1990-2019: Systematic review and meta-analysis. *J Affect Disord.* 2020;266:288-304.

13. Poorolajal J, Haghtalab T, Farhadi M, Darvishi N. Substance use disorder and risk of suicidal ideation, suicide attempt and suicide death: a meta-analysis. *J Public Health (Oxf).* 2016;38(3):e282-e291.

14. Carr T, Kilian C, Llamosas-Falcon L, et al. The risk relationships between alcohol consumption, alcohol use disorder and alcohol use disorder mortality: A systematic review and meta-analysis. *Addiction.* 2024;119(7):1174-1187.

15. Isaacs JY, Smith MM, Sherry SB, Seno M, Moore ML, Stewart SH. Alcohol use and death by suicide: A meta-analysis of 33 studies. *Suicide Life Threat Behav.* 2022;52(4):600-614.

16. Mason BJ, Heyser CJ. Alcohol Use Disorder: The Role of Medication in Recovery. *Alcohol Res.* 2021;41(1):07.

17. Kotake K, Hosokawa T, Tanaka M, et al. Efficacy and safety of alcohol reduction pharmacotherapy according to treatment duration in patients with alcohol dependence or alcohol use disorder: A systematic review and network meta-analysis. *Addiction.* 2024;119(5):815-832.

18. Martinelli S, Mazzotta A, Longaroni M, Petrucciani N. Potential role of glucagon-like peptide-1 (GLP-1) receptor agonists in substance use disorder: A systematic review of randomized trials. *Drug Alcohol Depend.* 2024;264:112424.

19. Koob GF, Volkow ND. Neurobiology of addiction: a neurocircuitry analysis. *Lancet Psychiatry.* 2016;3(8):760-773.

20. Volkow ND, Michaelides M, Baler R. The Neuroscience of Drug Reward and Addiction. *Physiol Rev.* 2019;99(4):2115-2140.

21. van Bloemendaal L, RG IJ, Ten Kulve JS, et al. GLP-1 receptor activation modulates appetite- and reward-related brain areas in humans. *Diabetes.* 2014;63(12):4186-4196.

22. Amorim Moreira Alves G, Teranishi M, Teixeira de Castro Goncalves Ortega AC, James F, Perera Molligoda Arachchige AS. Mechanisms of GLP-1 in Modulating Craving and Addiction: Neurobiological and Translational Insights. *Med Sci (Basel).* 2025;13(3).

23. Blumenthal DM, Gold MS. Neurobiology of food addiction. *Curr Opin Clin Nutr Metab Care.* 2010;13(4):359-365.

24. Shirazi RH, Dickson SL, Skibicka KP. Gut peptide GLP-1 and its analogue, Exendin-4, decrease alcohol intake and reward. *PLoS One.* 2013;8(4):e61965.

25. Woodward ORM, Gribble FM, Reimann F, Lewis JE. Gut peptide regulation of food intake - evidence for the modulation of hedonic feeding. *J Physiol.* 2022;600(5):1053-1078.

26. Aranas C, Edvardsson CE, Shevchouk OT, et al. Semaglutide reduces alcohol intake and relapse-like drinking in male and female rats. *EBioMedicine.* 2023;93:104642.

27. Marquez-Meneses JD, Olaya-Bonilla SA, Barrera-Carreno S, et al. GLP-1 Analogues in the Neurobiology of Addiction: Translational Insights and Therapeutic Perspectives. *Int J Mol Sci.* 2025;26(11).

28. Savchenko LG, Digtiar NI, Selikhova LG, et al. Liraglutide exerts an anti-inflammatory action in obese patients with type 2 diabetes. *Rom J Intern Med.* 2019;57(3):233-240.

29. Bendotti G, Montefusco L, Lunati ME, et al. The anti-inflammatory and immunological properties of GLP-1 Receptor Agonists. *Pharmacol Res.* 2022;182:106320.

30. Diz-Chaves Y, Mastoor Z, Spuch C, Gonzalez-Matias LC, Mallo F. Anti-Inflammatory Effects of GLP-1 Receptor Activation in the Brain in Neurodegenerative Diseases. *Int J Mol Sci.* 2022;23(17).

31. Moaket OS, Obaid SE, Obaid FE, et al. GLP-1 and the Degenerating Brain: Exploring Mechanistic Insights and Therapeutic Potential. *Int J Mol Sci.* 2025;26(21).

32. Cui C, Shurtleff D, Harris RA. Neuroimmune mechanisms of alcohol and drug addiction. *Int Rev Neurobiol.* 2014;118:1-12.

33. Nikbakhtzadeh M, Ranjbar H, Moradbeygi K, et al. Cross-talk between the HPA axis and addiction-related regions in stressful situations. *Heliyon.* 2023;9(4):e15525.

34. Klausen MK, Jensen ME, Moller M, et al. Exenatide once weekly for alcohol use disorder investigated in a randomized, placebo-controlled clinical trial. *JCI Insight.* 2022;7(19).

35. Probst L, Monnerat S, Vogt DR, et al. Effects of dulaglutide on alcohol consumption during smoking cessation. *JCI Insight.* 2023;8(22).

36. Hendershot CS, Bremmer MP, Paladino MB, et al. Once-Weekly Semaglutide in Adults With Alcohol Use Disorder: A Randomized Clinical Trial. *JAMA Psychiatry.* 2025;82(4):395-405.

37. Richards JR, Dorand MF, Royal K, Mnajjed L, Paszkowiak M, Simmons WK. Significant Decrease in Alcohol Use Disorder Symptoms Secondary to Semaglutide Therapy for Weight Loss: A Case Series. *J Clin Psychiatry.* 2023;85(1).

38. Quddos F, Hubshman Z, Tegge A, et al. Semaglutide and Tirzepatide reduce alcohol consumption in individuals with obesity. *Sci Rep.* 2023;13(1):20998.

39. Wang W, Volkow ND, Berger NA, Davis PB, Kaelber DC, Xu R. Associations of semaglutide with incidence and recurrence of alcohol use disorder in real-world population. *Nat Commun.* 2024;15(1):4548.

40. Wang W, Volkow ND, Berger NA, Davis PB, Kaelber DC, Xu R. Association of semaglutide with reduced incidence and relapse of cannabis use disorder in real-world populations: a retrospective cohort study. *Mol Psychiatry.* 2024;29(8):2587-2598.

41. Yammine L, Green CE, Kosten TR, et al. Exenatide Adjunct to Nicotine Patch Facilitates Smoking Cessation and May Reduce Post-Cessation Weight Gain: A Pilot Randomized Controlled Trial. *Nicotine Tob Res.* 2021;23(10):1682-1690.

42. Lengsfeld S, Burkard T, Meienberg A, et al. Effect of dulaglutide in promoting abstinence during smoking cessation: a single-centre, randomized, double-blind, placebo-controlled, parallel group trial. *EClinicalMedicine.* 2023;57:101865.

43. Qeadan F, McCunn A, Tingey B. The association between glucose-dependent insulinotropic polypeptide and/or glucagon-like peptide-1 receptor agonist prescriptions and substance-related outcomes in patients with opioid and alcohol use disorders: A real-world data analysis. *Addiction.* 2025;120(2):236-250.

44. Yammine L, Balderas JC, Weaver MF, Schmitz JM. Feasibility of Exenatide, a GLP-1R Agonist, for Treating Cocaine Use Disorder: A Case Series Study. *J Addict Med.* 2023;17(4):481-484.

45. Arillotta D, Floresta G, Papanti Pelletier GD, et al. Exploring the Potential Impact of GLP-1 Receptor Agonists on Substance Use, Compulsive Behavior, and Libido: Insights from Social Media Using a Mixed-Methods Approach. *Brain Sci.* 2024;14(6).

46. Moiz A, Filion KB, Tsoukas MA, Yu OH, Peters TM, Eisenberg MJ. Mechanisms of GLP-1 Receptor Agonist-Induced Weight Loss: A Review of Central and Peripheral Pathways in Appetite and Energy Regulation. *Am J Med.* 2025;138(6):934-940.

47. Nicolau J, Tamayo MI, Sanchis P, et al. Short-term effects of semaglutide among patients with obesity with and without food addiction: an observational study. *J Addict Dis.* 2024;42(4):535-543.

48. Richards J, Bang N, Ratliff EL, et al. Successful treatment of binge eating disorder with the GLP-1 agonist semaglutide: A retrospective cohort study. *Obes Pillars.* 2023;7:100080.

49. Angarita GA, Matuskey D, Pittman B, et al. Testing the effects of the GLP-1 receptor agonist exenatide on cocaine self-administration and subjective responses in humans with cocaine use disorder. *Drug Alcohol Depend.* 2021;221:108614.

50. O'Keefe JH, Franco WG, O'Keefe EL. Anti-consumption agents: Tirzepatide and semaglutide for treating obesity-related diseases and addictions, and improving life expectancy. *Prog Cardiovasc Dis.* 2025;89:102-112.

51. Zheng YJ, Soegiharto C, Au HCT, et al. A systematic review on the role of glucagon-like peptide-1 receptor agonists on alcohol-related behaviors: potential therapeutic strategy for alcohol use disorder. *Acta Neuropsychiatr.* 2025;37:e51.

