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Review Article

Modulating Reward and Satiety: Tirzepatide's Neural and Behavioral Pathways of Appetite Regulation

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Abstract

Tirzepatide, a dual GIP/GLP-1 receptor agonist, has shown remarkable efficacy in promoting weight loss and reducing appetite. While its metabolic effects are well established, the central mechanisms driving its anorectic properties remain less understood. This manuscript explores how tirzepatide influences neural circuits involved in reward, motivation, and satiety, with particular attention to mesolimbic and hypothalamic pathways. Neuroimaging and behavioral data suggest that tirzepatide alters food-related salience and decision-making, leading to sustained changes in eating behavior. These effects involve both dopaminergic modulation and enhanced cognitive control. By integrating findings from neuroscience and behavioral science, this work proposes a model in which tirzepatide reshapes appetite regulation through adaptive changes in brain function. The implications for long-term weight management and individualized treatment strategies are discussed

Keywords: Tirzepatide, Appetite Regulation, Receptors, Glucagon-Like Peptide-1, Glucose-Dependent Insulinotropic Polypeptide, Obesity / drug therapy.

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Introduction

The advent of dual agonists targeting glucagon-like peptide-1 (GLP-1) and glucose-dependent insulinotropic polypeptide (GIP) receptors has redefined the therapeutic landscape of obesity and metabolic disease. Tirzepatide, a first-in-class GIP/GLP-1 receptor agonist, has demonstrated unprecedented efficacy in reducing body weight, with effects surpassing those of earlier incretin-based therapies (Jastreboff et al., 2022). While its metabolic benefits are well-characterized, the mechanisms underlying its profound impact on appetite regulation remain incompletely understood.

Emerging evidence suggests that tirzepatide's anorectic effects extend beyond peripheral satiety signals, engaging central neural circuits implicated in reward, motivation, and behavioral control (Min et al., 2023). Neuroimaging studies have begun to reveal altered activity in mesolimbic pathways following administration, pointing to a possible recalibration of hedonic drive and food-related salience (Kullmann et al., 2023). These findings raise critical questions about how tirzepatide modulates both homeostatic and non-homeostatic feeding behavior, and whether its effects are sustained through adaptive changes in neural plasticity. Moreover, behavioral analyses indicate that tirzepatide may influence decision-making processes related to food intake, including delay discounting and effort-based choices, suggesting a broader cognitive impact on appetite regulation (Müller et al., 2022). Understanding these neural and behavioral pathways is essential for optimizing therapeutic strategies and anticipating long-term outcomes in weight management and relapse prevention.

Methods

A structured literature review was conducted to identify peer-reviewed studies published between 2019 and 2025

on tirzepatide's effects on appetite regulation, weight loss, neural mechanisms, and psychosocial outcomes. Searches were performed in PubMed, Web of Science, Google Scholar, and ClinicalTrials.gov using the following MeSH terms and their combinations: "Tirzepatide," "Appetite Regulation," "Receptors, Glucagon-Like Peptide-1," "Glucose-Dependent Insulinotropic Polypeptide," "Obesity/drug therapy," and "Neuroimaging." Reference lists of key articles were also screened to capture additional sources.

Eligible studies included randomized controlled trials, controlled feeding studies, neuroimaging experiments, behavioral analyses, and pharmacovigilance reports. Both efficacy and safety outcomes were considered, with attention to metabolic, neurological, and psychosocial effects. Data were synthesized thematically to highlight clinical results, behavioral adaptations, neural mechanisms, and adverse effects, ensuring integration of evidence across biological, cognitive, and social domains.

Comparative Efficacy and Safety of Tirzepatide vs. Semaglutide

At each of the doses examined, tirzepatide was superior to semaglutide and induced more weight loss. The most frequent adverse events were gastrointestinal, mild to moderate, and comprised of nausea (17-22% in tirzepatide compared with 18% in semaglutide), diarrhea (13-16% compared with 12%), and vomiting (6-10% vs. 8%). Hypoglycemia (<54 mg/dL blood glucose) happened infrequently (0.6%, 0.2%, and 1.% in 5 mg, 10 mg, and 15 mg, respectively) and in 0.4% in semaglutide. Serious adverse events happened in 5-7% of tirzepatide recipients and 3% of semaglutide recipients (Frías et al., 2021).

Subjects who were treated with tirzepatide were more successful at weight loss (≥5%; hazard ratio [HR], 1.76; 95% CI, 1.68, 1.84; ≥10%; HR, 2.54; 95% CI, 2.37, 2.73; and ≥15%; HR, 3.24; 95% CI, 2.91, 3.61). Weight changes during treatment were larger with tirzepatide at 3 months (difference, −2.4%; 95% CI −2.5% to −2.2%), 6 months (difference, −4.3%; 95% CI, −4.7% to −4.0%), and 12 months (difference, −6.9%; 95% CI, −7.9% to −5.8%). Gastrointestinal adverse event rates were comparable between the groups (Rodriguez et al., 2024).

Impact of Tirzepatide on Appetite Regulation and Energy Consumption

Male and female adults (n=114) without diabetes, with a body mass index range of 27-50 kg/m2, were randomly assigned to receive blinded tirzepatide, once a week, to examine the impact of tirzepatide on energy consumption. The primary outcome was the variation from baseline to week 3 in energy consumption during lunch with tirzepatide compared to placebo. Other outcomes evaluated self-reported eating habits and blood oxygenation level dependent fMRI using images of food. Tirzepatide decreased energy consumption compared to placebo at week 3 (-524.6 kcal, 95% CI -648.1 to -401.0), $p < 0.0001$. Compared to the placebo, tirzepatide reduced overall appetite, food cravings, inclination to overeat, perceived hunger, and sensitivity to environmental foods, yet did not affect voluntary dietary intake restriction. At week 3, tirzepatide did not produce a statistically significant change in response to the combined category of highly palatable food cues (high fat/high sugar and high fat/high carbohydrate) compared with placebo. A selective reduction in neural activation was observed for high-fat/high-sugar food images, suggesting that tirzepatide may attenuate brain responses to particularly energy-dense food high in sugar (Martin et al., 2025).

GLP-1 as Hormone and Neurotransmitter: Distinct Mechanisms of Action

The current data suggest the presence of two separate GLP-1 systems within our body. Gut-released GLP-1 is a hormone and acts on receptors outside the BBB, presumably primarily in the gastrointestinal tract, in order to fulfill its role as an incretin and to activate vagal afferent neurons in order to inform the brain about the state of digestion of a meal. These vagal afferent neurons are among those that generate an electrical impulse to the CNS that initiates satiation and/or satiety pathways. However, they are distinct from the pathways initiated by brain-derived GLP-1, which is secreted locally in a more neurotransmitter- or neuromodulator-like fashion. Thus, GLP-1 is a hormone and a neurotransmitter, yet the gut-derived GLP-1 cannot be a neurotransmitter because it cannot permeate the GLP-1 receptors of the CNS. Systemically given GLP-1 receptor agonists thus mimic the postprandial effect of native gut-derived GLP-1 and also permeate the GLP-1 receptors in the non-BBB-covered portion of the CNS (Trapp & Brierley, 2022).

Central GIP–GLP-1 Signaling and Energy Homeostasis

The GLP1 system has long been associated with appetite regulation. GLP1R is expressed in the hypothalamus, brainstem, and mesolimbic regions. (Kanoski 2016; Jones et al. 2025). GLP1R activation lowers food intake by regulating homeostatic neurons in the arcuate nucleus and by blunting activities related to reward pathways in regions such as the nucleus accumbens and ventral tegmental area (Jones et al., 2025). GIPR expression in the brain has been inaccessible, but recent RNAscope analyses have identified GIPR in non-identical and overlapping hypothalamic cell populations. This indicates that GIP signaling may

supplement GLP1 pathways through synergistic activation in the same neuron or integrated signals from distinct neuronal populations. Acute stimulation of hypothalamic GIPR neurons in rodents has been shown to suppress food intake potently. Co-stimulation with GLP-1R does not have simple additive effects, suggesting that synergy between the two systems may occur with chronic, rather than acute, stimulation. One hypothesis is that sustained GIPR activity enhances GLP-1–induced anorexia via receptor sensitivity adaptations or by modulating blood–brain barrier (BBB) permeability to circulating peptides (Geisler et al., 2022). Such mechanisms might partly explain why tirzepatide produces greater long-term weight loss than GLP-1RAs alone, despite equivalent short-term anorectic actions.

RNAscope analyses showed that GIPR and GLP1R localize in distinct, overlapping hypothalamic cell populations. The metabolic benefit of dual GIP and GLP1R activation could either result from synergistic action within the same cells or from the convergence across different neuronal populations. The absence of an apparent additive effect of GLP1R activation on the acute anorexigenic response to GIPR may be required to enhance GLP1-mediated anorexigenic action. Identifying GIPR expression in non-neuronal populations also provides an area for consideration of the possibility that GIP signaling could modulate central access of GLP1 or other hormones in the circulatory system. These findings reveal the central hypothalamic GIP signaling axis in rodents and humans and establish central GIP signaling as a pathway to energy homeostasis (Adriaenssens et al., 2019).

Besides the hypothalamus, incretin signals communicate with vagal afferents and brainstem nuclei. GLP1 from the intestine activates vagal afferents to relay messages of meals to the nucleus tractus solitarius (NTS), in which second-order neurons relay messages of gastric distension, nutrients, and hormonal composition (Zheng et al., 2024). Tirzepatide, via GLPR activation, likely engages these pathways, causing earlier satiation and reduced meal size. Whether GIPR participates in vagal signaling is less well established. However, rodent data suggest that peripheral GIP may act on BBB-associated cells, thus altering penetration into the CNS of GLP1 and other hormones (Geisler et al., 2022). If confirmed, this would represent an atypical modulatory function of GIPR distinct from the direct mediation of satiety via GLP1.

Discussion

GLP-1RAs are shown to be incredibly effective in weight loss by increasing peripheral and central satiety and decreasing craving and impulsive feeding by modulating reward systems. Functional imaging also points to the suppressed mesolimbic activity and changed hypothalamic-prefrontal regulation, allowing healthier food preferences and effortless healthier food choices. These advantages are, however, offset by the neurological and psychosocial risks such as mood disorders, uncommon encephalitic syndromes, and cerebrovascular complications. The following discussion will discuss these two outcomes, considering therapeutic potential versus some adverse outcomes. With the increase in the prescription and usage of GLP1 medication, analyzing its long-term systemic effects plays an important role. Exploring the links between metabolic syndrome and its treatment, neuropsychiatry is crucial as it impacts patients' lives.

The application of glucagon-like peptide-1 receptor agonists (GLP-1RAs) has been linked with substantial weight loss and unique behavioral and neurocognitive changes that transform eating habits. Rodriguez et al. surveyed patients taking GLP-1RAs and found that most respondents reported a significant decrease in impulsive snacking, mitigated response to food cravings, and a general reduction in the tendency to overeat (Rodriguez et al., 2024). These results indicate that GLP-1RAs act on central and peripheral mediators of appetite control, which have cumulative effects on eating behavior (Jones et al., 2025).

Peripheral and Central Mechanisms of Appetite Control

Mediating mechanisms of peripheral effects are delayed gastric emptying and increased postprandial satiety, which play a role in lowering the intensity of instantaneous hunger sensation. Patients report feeling fuller after meals, less frequent, and less insistent (Rodriguez at al., 2024). The central effects are the dampening of the mesolimbic system and its dopamine signaling (Kanoski et al., 2016). GLP-1RAs directly decrease the impulse to eat highly palatable foods impulsively by inhibiting dopaminergic anticipation of reward and decreasing the desire to consume food high in fat and sugar.

Neuroimaging Evidence and Reward Circuit Modulation

These changes are also explained by functional neuroimaging. The human fMRI experiments always show the reduction of blood-oxygen-level-dependent (BOLD) activation in the brain's reward-related parts when exposed to palatable food signals. This reduced neural activity is especially pronounced in hyperpalatable food containing high sugar and fat levels, which manifests itself in the suppressed dopamine signaling and regulation of hypothalamic circuits. The GLP-1 receptors in the brainstem have been found to directly suppress mesolimbic dopamine release directly, hence suppressing hedonic anticipation. It leads to dual attenuation of the wanting

(anticipatory motivation) and liking (hedonic evaluation) that minimizes overeating (Martin et al., 2025).

Behavioral Adaptations and Eating Habits

The weight loss is usually reported to happen with startling ease, most of the time without an overtly motivating effort towards the intake limitation. Tirzepetide reorganizes the appetite and reward systems in which behavior is supported by diminished cue-induced eating internal drive. In fact, this (that food seems less appetizing) is also reported by many patients, accompanied by a decrease in insular activation (interoceptive awareness) and changes in prefrontal activity (inhibitory control and value-based decision making) (Lauren A Jones et al., 2025).

Adverse Neurological and Psychiatric Effects

Other effects, which include nausea, loss of taste perception, and emergence of mild aversions, can also add to the behavioral adaptation by making foods rich in calories less attractive (Mishra R et al., 2023) (Ghusn W et al., 2024). Notably, GLP-1RAs have been effective in refractory cue-induced eating, suppressing conditioned food response, and abating hedonic overconsumption. This has been especially applicable to those who are likely to develop addictive eating habits and, therefore, therapeutic in changing the established patterns of dysregulated consumption (Lauren A Jones et al., 2025).

GLP-1RAs transform pre-ingestive and post-ingestive interactions within the system. Anticipatory hunger and cue-stimulated food seeking are dulled, and postprandial satiety is greatly exaggerated. The coordination of the hypothalamic regulation, the limbic modulation of the process, and the prefrontal control network creates the novel balance in homeostatic and hedonic feeding systems (Kim KS et al., 2025). Additionally, the body's energy intake changes correlate with the overall metabolic changes, such as fluid and systemic energy regulation changes.

Overall, GLP-1RAs have far-reaching effects on appetite and eating behavior by influencing the gastrointestinal tract's physiological and neurocognitive levels. Their ability to inhibit reward-directed consumption while increasing satiety and re-examine motivational activities underscores their special place in obesity management. In addition to causing weight loss, they provide an alternative paradigm of treating the neurobehavioral causes of overeating, and their therapeutic model is where decreased consumption is not due to external restraint but rather an intrinsic reprocessing of the hunger and reward pathways (Martin et al., 2025).

The discussed therapeutic intervention has demonstrated high efficacy in achieving significant weight loss. It has become a promising choice when it comes to the management of obesity and the comorbidities associated with it. Although such an advantage is present, there has been a growing body of literature showing a variety of neurological and psychosocial risks, which should be carefully observed. The two-sidedness of its high efficacy and the occurrence of adverse events that may be devastating highlight the necessity of a multidisciplinary and balanced approach to its clinical practice.

Systematically, the manifestations of the patients often include early neurological symptoms of dizziness, headache, and cognitive fatigue. Though these manifestations may be viewed as mild and transient, their persistence may affect the functionality of everyday life and lead to the decline of cognitive efficiency. The net effect of the symptoms poses critical concerns about the consequences of the neurocognitive performance in the long run, especially in patients who have to undergo a long treatment period (Mengmeng Huang et al., 2025).

In addition to these general effects on the system, there are more serious psychiatric complications. Depression, increased levels of anxiety, and occasionally suicidal ideation have been reported. They have significant power over mood, stress responses, emotional stability, and neuropsychiatric regulation. Even a small group of patients with the presence of suicidality represents the seriousness of these risks and the need for constant psychological evaluation during the treatment period (Bezin, Julien et al., 2024).

Severe neurological complications increase the risk factors and profile. Cases of immune-mediated neurotoxicity have been of concern since only a few cases have reported the onset of autoimmune encephalitis (Abdi A et al., 2025). Also, cerebrovascular events, such as ischemic strokes, neurodegenerative presentations, and intracerebral hemorrhages, have been reported to be associated with the condition in clinical reports (Lin H et al., 2025). Although causality is yet to be determined, the magnitude of such events is such that they are clinically important and warrant increased vigilance in vulnerable populations.

Psychosocial Implications of Weight Loss

The psychosocial impact of the intervention is also complicated. Although weight loss tends to trigger positive external reinforcement and social approval, it can be ironic and create new avenues of mental pressure. Patients often report an increase in anxiety levels, emotional numbing, and the inability to get used to the changed social identities in the aftermath of the alterations of body images. The self-focus of outer attention on the physical change, on the one hand, may strengthen the discord of the individual. On the other hand, it may cause stress and abnormal psychosocial operations. This paradox draws attention to

the fact that it is necessary to pay attention to the biological and psychosocial consequences of treatment (Pierret ACS, 2025).

Balancing Therapeutic Promise and Risk

Tirzepatide has emerged as one of the most effective pharmacologic tools for obesity management, achieving weight reductions that exceed earlier incretin-based therapies. By simultaneously targeting peripheral satiety mechanisms and central reward pathways, it reconfigures the biology of eating in ways that feel effortless to many patients. This transformative efficacy, however, cannot be disentangled from its capacity to influence neural circuits that regulate mood, cognition, and motivation. Headache, dizziness, and cognitive fatigue illustrate a spectrum of neurological effects that may at first appear tolerable, yet raise concerns about cumulative burden during long-term therapy. More concerning are psychiatric sequelae such as depression, anxiety, and suicidality, which highlight that tirzepatide is not solely a metabolic drug but also a neuromodulator capable of reshaping affective states.

The psychosocial consequences of such profound weight loss add another layer of complexity. For some patients, the benefits of improved body image and external validation coexist with heightened anxiety, emotional blunting, or difficulties reconciling a transformed physical identity with prior self-perceptions. This paradox, in which clinical success introduces new psychosocial vulnerabilities, highlights the need for clinicians to anticipate not only biological outcomes but also the lived experiences of patients navigating rapid change. Tirzepatide's clinical narrative therefore extends well beyond caloric reduction, touching domains of mental health, social adaptation, and identity reconstruction that can either reinforce or destabilize its therapeutic benefits.

The path forward lies in embedding tirzepatide within a framework of integrative care. Informed consent should move beyond weight loss statistics to acknowledge potential neurological and psychiatric complications, while follow-up must involve interdisciplinary monitoring across primary care, neurology, and psychiatry. Research must refine patient selection criteria and identify biomarkers of vulnerability to adverse effects, allowing benefits to be maximized while minimizing harm. Only through such vigilance can tirzepatide fulfill its promise as a therapy that reshapes appetite and behavior without replicating the unintended harms of past weight loss interventions.

Conclusion

Tirzepatide exemplifies a new generation of incretin-based therapies that extend beyond metabolic regulation to reshape appetite, reward, and satiety circuits. Clinical trials, behavioral studies, and neuroimaging evidence demonstrate that its effects are mediated through peripheral satiety mechanisms and central modulation of mesolimbic and hypothalamic pathways. These changes translate into sustained weight loss and altered food-related decision-making that often requires little conscious effort from patients. At the same time, reports of neurological, psychiatric, and psychosocial risks highlight the importance of careful monitoring, informed consent, and multidisciplinary care. Future research must clarify the mechanisms driving both therapeutic and adverse outcomes, refine patient selection, and develop strategies to maximize benefit while mitigating risk. In doing so, tirzepatide may help define a broader paradigm for treating obesity that integrates biology, behavior, and brain function.

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