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MOLECULAR MECHANISMS AND ALGORITHMIC REALITIES: A SOCIO-MEDICAL ANALYSIS OF GLP-1 RECEPTOR AGONISTS IN THE CONTEXT OF EATING DISORDER SPECTRUM

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ABSTRACT

Background: While Glucagon-like peptide-1 receptor agonists (GLP-1 RAs) are established treatments for obesity, their role in eating disorders (EDs) is a rapidly evolving frontier. This review evaluates the clinical efficacy, neurobiological mechanisms, and safety of GLP-1 RAs across binge eating disorder (BED), bulimia nervosa (BN), and anorexia nervosa (AN), while critically examining the socioeconomic and technological drivers of their recent proliferation.
Methods: A systematic literature search was conducted across PubMed, Scopus, Web of Science, and the Cochrane Library in December 2025. Inclusion criteria focused on clinical trials, mechanistic studies, and pharmacovigilance reports addressing behavioral and metabolic outcomes of GLP-1 RAs.
Results: Current evidence identifies BED as the primary clinical indication; meta-analyses demonstrate significant weight loss (-3.81 kg), BMI reduction (-1.48 kg/m²), and improved Binge Eating Scale scores (-8.14 points). These effects are driven by hypothalamic satiety regulation and the dampening of dopamine-mediated motivational "wanting" (incentive salience) for palatable foods. For BN, data remain exploratory. Conversely, GLP-1 RAs are discouraged in AN due to the high risk of reinforcing restrictive pathology and severe undernutrition. The review also identifies risks associated with direct-to-consumer telemedicine and social media trends like "Ozempic face", which may bypass psychiatric screening.
Conclusions: GLP-1 RAs offer significant therapeutic potential for BED with comorbid obesity but require a multidisciplinary framework to mitigate risks of unmonitored prescribing and weight stigma. Future research must prioritize long-term, disorder-specific safety data.

KEYWORDS

GLP-1 Receptor Agonists, Binge Eating Disorder, Bulimia Nervosa, Anorexia Nervosa, Incentive Salience

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1. Introduction

Eating disorders (EDs) represent one of the most significant and complex public health challenges in modern society, characterized by increasing prevalence and severe psychiatric and medical comorbidities. According to the World Health Organization (2021), in 2021, eating disorders affected approximately 16 million people, including nearly 3.4 million children and adolescents. These conditions involve abnormal eating behaviors and preoccupations with food, weight, and shape, often leading to significant distress and impairment in functioning.

The primary types of eating disorders include:

- Anorexia Nervosa (AN): Characterized by severe food intake restriction, an intense fear of weight gain, and a distorted body image, often resulting in dangerously low body weight (Krug et al., 2025).
- Bulimia Nervosa (BN): Involves recurrent episodes of binge eating followed by inappropriate compensatory behaviors such as self-induced vomiting or laxative misuse (Aoun et al., 2024).
- Binge Eating Disorder (BED): The most prevalent eating disorder, defined by recurrent episodes of uncontrolled overeating without regular compensatory behaviors, frequently leading to obesity and significant psychological distress (Guerdjikova et al., 2017; Kessler et al., 2013).

A major hurdle in management is the limited availability of effective pharmacotherapy. Currently, the only FDA-approved medications are lisdexamfetamine for moderate-to-severe BED and fluoxetine for BN, both of which can have significant side effects or potential for abuse (McElroy et al., 2015; Allison et al., 2023). Consequently, there is growing interest in GLP-1 receptor agonists (GLP-1 RAs), such as liraglutide and semaglutide, which have transformed the treatment of obesity and type 2 diabetes by promoting satiety and reducing energy intake (van Bloemendaal et al., 2014, Müller et al., 2019).

Neurobiologically, GLP-1 RAs influence appetite through distinct gut-brain circuits, acting on the hypothalamus to regulate homeostatic feeding and on the mesolimbic reward system - including the ventral tegmental area (VTA) and nucleus accumbens (NAc) - to dampen the dopamine-mediated "wanting" (incentive salience) of highly palatable foods (Alhadeff et al., 2012; van Bloemendaal et al., 2014; Berridge & Robinson, 2016).

However, the implementation of GLP-1 RAs transcends clinical narratives, evolving into a complex socio-technological phenomenon. The rise of asynchronous direct-to-consumer (DTC) telemedicine platforms has altered the patient-provider relationship, often bypassing synchronous clinical gatekeeping vital for detecting subtle signs of restrictive or purging behaviors (Hoffman, 2020). This "frictionless" access introduces a risk where individuals with undiagnosed AN or BN may obtain medications that actively promote their pathology through extreme appetite suppression (Krug et al., 2025).

Furthermore, social media platforms like TikTok have transformed GLP-1 RAs into cultural symbols of rapid transformation. The viral "Ozempic face" phenomenon - describing the distorted facial contours resulting from rapid weight loss - has consumed digital discourse, potentially intensifying weight stigma and glamorizing extreme thinness (Carboni et al., 2024; Raiter et al., 2023). Such narratives can act as social contagions, triggering disordered eating in vulnerable populations. Ethical concerns also arise from the high cost and subscription-based models of these therapies, risking a two-tiered healthcare system where marginalized groups are excluded from metabolic and psychological care (Krug et al., 2025).

2. Methodology

2.1 Study Design and Rationale

To address the convergence of neurobiological innovation and evolving psychosocial risks, this study employs an integrative narrative synthesis. Given that the therapeutic application of GLP-1 receptor agonists (GLP-1 RAs) in eating disorders is a rapidly shifting frontier, a purely quantitative meta-analysis would be insufficient to capture the multidimensionality of the current evidence. While clinical trials for Binge Eating Disorder (BED) provide robust metabolic data, the evidence for Bulimia Nervosa (BN) and Anorexia Nervosa (AN) remains largely exploratory or mechanistic.

Consequently, this narrative framework was selected to bridge the gap between molecular neurobiology - specifically the functional dissociation between homeostatic satiety and hedonic reward processing - and the sociotechnical phenomenon of direct-to-consumer access and digital discourse. This approach allows for a rigorous synthesis of high-impact Phase 3 trials alongside pilot RCTs and pharmacovigilance reports, accommodating the inherent heterogeneity in outcome measures (e.g., transitioning from interview-based EDE scores to self-reported BES metrics). By utilizing a biopsychosocial and equity-based framework, this review moves beyond clinical efficacy to critically evaluate how medication-driven weight loss interacts with weight stigma and healthcare accessibility.

2.2 Search Strategy and Information Sources

A systematic search of four primary electronic bibliographic databases - PubMed/MEDLINE, Scopus, Web of Science, and the Cochrane Library - was conducted in December 2025. These sources were supplemented by manual searches of Google Scholar and cross-referencing to ensure a comprehensive reach across medical, pharmacological, and psychological literature. The search utilized a predefined combination of Boolean operators and Medical Subject Headings (MeSH), including terms such as "GLP-1 receptor agonists", "semaglutide", "liraglutide", "tirzepatide" and "dulaglutide" paired with "binge eating disorder", "bulimia nervosa" and "anorexia nervosa". Focus was placed on high-impact literature published between 2012 and 2025 to reflect the most recent clinical and pharmacological advancements.

2.3 Eligibility Criteria: Inclusion and Exclusion

To maintain academic rigor, specific eligibility criteria were established:

- Inclusion Criteria: Primary research including Phase III trials for metabolic outcomes and pilot RCTs targeting ED populations; large-scale pharmacovigilance analyses; and systematic reviews or meta-analyses. Peer-reviewed mechanistic studies focusing on the central nervous system (CNS), reward-related signaling, and the gut-brain axis were also included.
- Exclusion Criteria: Studies focusing solely on glycemic control in Type 2 Diabetes without reporting appetite or weight outcomes were excluded. Additionally, trials with exclusionary psychiatric comorbidities, such as severe depression (PHQ-9 $\geq$ 15) or recent suicidality (C-SSRS score of 4 or 5), were excluded to ensure the synthesis reflects clearly defined clinical populations.

2.4 Study Selection and Data Extraction

The selection process followed a multi-stage screening protocol. Initially, titles and abstracts were independently screened for relevance, followed by a full-text review of potentially eligible studies. "Backward snowballing" of reference lists was employed to identify landmark papers and neurobiological mechanism studies. Data were extracted using a standardized template capturing study design (e.g., Phase 3 trials like SURPASS-2 or pilot RCTs), population demographics (ED diagnosis, BMI $\geq$ 27 kg/m^2), pharmacological intervention (molecule type, dose, titration protocol), and primary outcomes such as binge frequency and weight change.

2.5 Quality Assessment and Narrative Synthesis

Methodological quality was assessed using tools appropriate for the study design, including the Cochrane Risk of Bias (RoB) tool for clinical trials and the STROBE checklist for observational studies. The synthesis linked clinical outcomes to underlying neurobiological mechanisms, such as the activation of GLP-1 receptors in the mesolimbic reward system. This interpretative approach aimed to explain behavioral shifts - like the reduction in "wanting" (incentive salience) versus "liking" - based on known GLP-1 RA pharmacodynamics in the brain. Evidence was stratified by hierarchy, giving primary weight to RCTs while using case reports and mechanistic extrapolations to inform discussions on Bulimia Nervosa and Anorexia Nervosa where large-scale data are currently lacking.

3. Results

3.1 Mechanistic Basis of GLP-1 RA Action

GLP-1 receptor agonists exert their effects on eating behavior through multiple physiological and neurobiological pathways, acting as key regulators of systemic energy homeostasis (van Bloemendaal et al., 2014; Müller et al., 2019). Centrally, GLP-1 RAs activate receptors in the hypothalamus and brainstem, modulating appetite control and enhancing satiety signaling (van Bloemendaal et al., 2014). Experimental neuroimaging studies indicate that GLP-1 RAs reduce activity in reward-related areas, such as the ventral tegmental area and nucleus accumbens, thereby attenuating food-related reward and motivation (Alhadeff et al., 2012).

This modulation of the mesolimbic pathway is crucial; it dampens the dopamine release associated with the consumption of highly palatable foods, effectively reducing the salience and motivational drive (the "wanting") that often triggers uncontrolled binge episodes in BED and BN (Alhadeff et al., 2012; Berridge & Robinson, 2016). The resultant neurochemical environment shifts the focus from hedonic craving to homeostatic control (van Bloemendaal et al., 2014). Furthermore, activation of GLP-1 receptors in the nucleus of the solitary tract (NTS) integrates satiety signals from the gastrointestinal tract with central regulation, contributing to improved impulse control and reduced compulsion related to food seeking (Alhadeff et al., 2012).

Peripherally, GLP-1 RAs slow gastric emptying, prolonging postprandial fullness and reducing caloric intake (van Bloemendaal et al., 2014). Clinical pharmacology studies demonstrate decreased meal size and enhanced satiety, with dual GIP/GLP-1 agonists like tirzepatide showing superior weight loss and glycemic control compared to selective GLP-1 RAs (Frías et al., 2021). Central to the efficacy of GLP-1 RAs in disordered eating is the functional dissociation between homeostatic satiety and hedonic reward processing (Berridge & Robinson, 2016).

While traditional weight-loss interventions often fail due to the biological drive to compensate for caloric deficit (the "starvation response"), GLP-1 RAs modulate the neurochemical environment to favor homeostatic control (van Bloemendaal et al., 2014). By activating receptors in the hypothalamus, these agents reinforce the signals of "biological fullness", while simultaneously decreasing the excitatory output of the mesolimbic dopamine system (Alhadeff et al., 2012). This dual action effectively raises the threshold for food-related reward; it does not necessarily diminish the pleasure of eating (hedonic "liking"), but significantly reduces the incentive salience or the compulsive "wanting" that drives binge episodes (Berridge & Robinson, 2016). In the context of the gut-brain axis, this creates a stabilized feedback loop: peripheral signals of gastric distension are integrated with central reductions in reward-seeking, providing a neurobiological "buffer" that allows patients to engage more effectively with cognitive-behavioral strategies (van Bloemendaal et al., 2014; Alhadeff et al., 2012).

3.2 GLP-1 RAs in Binge Eating Disorder (BED): Analysis of Key Clinical Trials

The application of GLP-1 receptor agonists (RAs) in the treatment of Binge Eating Disorder (BED) represents a growing area of clinical inquiry, as these agents are known to stimulate satiety and influence brain regions involved in the regulation of feeding behavior (van Bloemendaal et al., 2014). While Cognitive Behavior Therapy (CBT) is considered the most effective treatment for BED, it typically does not produce significant weight loss (Allison et al., 2023). Currently, lisdexamfetamine is the only FDA-approved medication for BED, having shown mean reductions of 4.1 binge episodes per week in randomized trials (McElroy et al., 2015). However, its potential for abuse has prompted the investigation of non-stimulant alternatives like liraglutide (Allison et al., 2023).

3.2.1 The Liraglutide Pilot Study: Design and Methodology

A significant investigation in this domain is the 17-week randomized, double-blind, placebo-controlled pilot trial conducted by Allison and colleagues (Allison et al., 2023), which evaluated the efficacy and safety of liraglutide 3.0 mg in adults with BED and a BMI ≥ 27 kg/m^2. The study followed a standard titration protocol: participants initiated treatment at 0.6 mg per day, with weekly increments of 0.6 mg until the target dose of 3.0 mg was reached (Allison et al., 2023). This gradual titration is critical for mitigating gastrointestinal adverse events, such as nausea and diarrhea, which are common with GLP-1 RA therapy (Allison et al., 2023; van Bloemendaal et al., 2014). Although the study was limited by a small sample size due to a medication dispensing error, it remains a key reference for using interview-based measures rather than self-report scales to assess binge frequency (Allison et al., 2023).

3.2.2 Efficacy Outcomes: Behavioral and Metabolic

The results of the pilot study by Allison et al. (Allison et al., 2023) provided mixed evidence regarding behavioral outcomes. The primary endpoint was the change in the number of objective binge episodes (OBEs) per week. While the liraglutide group demonstrated a reduction of 4.0 episodes compared to 2.5 in the placebo group, this difference was not statistically significant ($p=0.37$) (Allison et al., 2023). Similarly, the percentage of participants achieving remission (44% in the liraglutide group vs. 36% in the placebo group) did not differ significantly (Allison et al., 2023).

However, the metabolic outcomes were compelling; participants in the liraglutide arm achieved a significantly greater weight loss of 5.2% compared to only 0.9% in the placebo group ($p=0.005$) (Allison et al., 2023). This weight reduction is clinically meaningful and suggests that liraglutide may be a promising approach for weight management in patients with BED and obesity (Allison et al., 2023). Centrally, these effects are likely mediated by GLP-1 receptors in the mesolimbic reward system, which can decrease the intake of highly-palatable foods by attenuating reward-related signaling (Alhadeff et al., 2012). By modulating these pathways, GLP-1 RAs may reduce the compulsive “wanting” or incentive salience associated with binge episodes without necessarily diminishing the hedonic “liking” of food (Berridge & Robinson, 2016).

3.2.3 Safety and Tolerability Profile in the BED Population

Safety analysis from the pilot trial by Allison and colleagues (Allison et al., 2023) aligns with the known profile of GLP-1 RAs. Adverse events were experienced by 89.5% of participants in the liraglutide group compared to 64.7% in the placebo group (Allison et al., 2023). The most frequently reported adverse events were gastrointestinal, specifically diarrhea (26%) and nausea (26%), followed by tachycardia, dry mouth, and indigestion (Allison et al., 2023). While these effects were generally managed through the 5-week dose-escalation phase, two serious adverse events occurred in the liraglutide group: a cholecystectomy and a hospitalization due to severe vomiting (Allison et al., 2023).

In the context of eating disorders, gastrointestinal side effects theoretically pose a psychological risk for triggering maladaptive compensatory behaviors in patients with a history of purging; however, this trial excluded individuals with current anorexia nervosa or bulimia nervosa (Allison et al., 2023). Importantly, there were no significant differences between the treatment and placebo groups regarding psychiatric outcomes, including depressive symptoms measured by the PHQ-9 or overall quality of life (Allison et al., 2023). Suicidality was monitored at each visit using the Columbia-Suicide Severity Rating Scale (C-SSRS), and no significant psychiatric signals were observed among participants, though the 17-week duration of the study limits conclusions regarding long-term neuropsychiatric safety (Allison et al., 2023).

3.2.4 Broader Evidence and Systematic Reviews

Supporting the initial pharmacological inquiries into BED, smaller pilot studies and post-hoc analyses have increasingly contributed to the evidence base. For instance, liraglutide 3.0 mg has demonstrated a mean reduction of 4.0 binge episodes per week, a figure numerically similar to the 4.1-episode reduction observed in trials for lisdexamfetamine (McElroy et al., 2015; Allison et al., 2023). While pilot studies may struggle to reach statistical significance in binge reduction due to small sample sizes or strong placebo responses, the clinical magnitude of these changes is considered meaningful (Allison et al., 2023; Jacobs-Pilipski et al., 2007).

The chronic nature of BED - often co-occurring with mood and anxiety disorders and carrying a high risk for obesity-related complications - raises critical questions regarding treatment duration (Guerdjikova et al., 2017; Kessler et al., 2013). Because GLP-1 RAs are currently approved for chronic weight management, researchers are exploring whether their use in BED should be a short-term intervention to break the cycle of bingeing or a long-term strategy similar to the management of type 2 diabetes (Allison et al., 2023; van Bloemendaal et al., 2014).

Recent systematic reviews, such as the one by Aoun and colleagues (2024), synthesize available evidence to suggest that GLP-1 RAs (specifically liraglutide and dulaglutide) significantly reduce binge eating frequency and have a favorable psychiatric side effect profile compared to existing options (Aoun et al., 2024). Unlike lisdexamfetamine, which is a stimulant that poses a potential for abuse - a significant concern given the elevated rates of substance use disorders in the BED population - GLP-1 RAs lack addictive potential (McElroy et al., 2015; Allison et al., 2023). Furthermore, they do not carry the same sympathomimetic risks, making them a potentially safer alternative for older adults or those with cardiovascular comorbidities who require both weight and binge-eating management (Allison et al., 2023; Aoun et al., 2024).

3.2.5 Limitations of Current Evidence

Despite these promising results, several critical gaps remain in the literature that limit the broad application of current findings. Firstly, the demographic composition of these trials is often narrow; for instance, in the pilot study by Allison and colleagues, participants were predominantly female (63%) and White (59.3%) (Allison et al., 2023). This fails to fully capture the diverse reality of BED, a disorder where the female-to-male ratio is more balanced than in other eating disorders and which significantly affects various racial groups (Guerdjikova et al., 2017). Additionally, because recruitment was limited to individuals with a BMI $\geq$ 27 kg/m², the results may not apply to the broader population of patients with BED who fall within lower weight categories (Allison et al., 2023).

Secondly, the stringent exclusion of patients with “complex” psychiatric histories limits our understanding of the drug’s efficacy in real-world clinical populations. Trials have excluded participants with moderate-to-severe depressive symptoms (PHQ-9 score $\geq$ 15) or any history of suicidal ideation or behavior in the previous five years (Allison et al., 2023). Given that BED is highly comorbid with mood and anxiety disorders, the safety and effectiveness of GLP-1 RAs in patients with these common psychiatric conditions remain largely unknown (Guerdjikova et al., 2017; Kessler et al., 2013).

Finally, the “real-world” effectiveness of these agents may differ significantly from the results seen in clinical trials. Research protocols, such as the 17-week trial by Allison et al., involve intensive monitoring with assessments every two weeks, which likely contributes to the high placebo response (36% remission rate) often observed in BED research (Allison et al., 2023; Jacobs-Pilipski et al., 2007). Outside of such highly supportive structures, where “food noise” and compulsive behaviors are closely monitored, patients may face different challenges, especially considering that established treatments like CBT are already noted for being “expensive, time-intensive, and not available in many regions” (Allison et al., 2023).

3.3 GLP-1 RAs in Bulimia Nervosa (BN)

The evidence regarding the use of GLP-1 receptor agonists (RAs) in Bulimia Nervosa (BN) remains sparse, and comprehensive assessments are still needed to understand their full clinical impact (Jebeile et al., 2025). Mechanistically, GLP-1 RAs could potentially mitigate binge episodes by enhancing satiety signaling and influencing brain regions involved in appetite regulation (van Bloemendaal et al., 2014). Experimental studies have shown that GLP-1 receptor activation in the mesolimbic reward system reduces the intake of highly palatable foods and decreases reward-related motivation (Alhadeff et al., 2012).

Recent systematic reviews suggest that specific GLP-1 RAs, such as liraglutide and dulaglutide, may reduce binge eating frequency and associated comorbidities (Aoun et al., 2024). However, BN is characterized not only by dysregulated appetite but also by complex compensatory behaviors, such as purging, which are used to prevent weight gain (Allison et al., 2023). As such, pharmacological modulation of satiety may only

partially address the underlying psychopathology of BN. While early findings are promising for binge reduction, large-scale, placebo-controlled trials are necessary to establish the efficacy and safety of these agents in this specific population (Aoun et al., 2024).

3.4 GLP-1 RAs in Anorexia Nervosa (AN)

Evidence for the use of GLP-1 RAs in restrictive eating disorders like Anorexia Nervosa (AN) remains extremely limited (Krug et al., 2025). Mechanistically, the primary effects of GLP-1 RAs - stimulating satiety and reducing food intake - pose a significant physiological danger to individuals with AN, as these actions directly lead to weight loss and can exacerbate severe undernutrition (van Bloemendaal et al., 2014; Alhadeff et al., 2012).

Furthermore, clinical concerns have been raised that the appetite-suppressing effects of these medications could reinforce rigid control or perfectionistic traits, thereby strengthening the restrictive patterns central to AN (Krug et al., 2025). Because GLP-1 receptor activation significantly reduces reward sensitivity for food and caloric intake, these agents are currently considered problematic for this population (Alhadeff et al., 2012). Consequently, the long-term psychological and physical safety of GLP-1 RAs in restrictive eating disorders is unknown, and their use is generally avoided due to the high risk of harm (Krug et al., 2025).

3.5 Safety and Adverse Events

Adverse events associated with GLP-1 RA therapy are well-documented across clinical populations and are primarily gastrointestinal in nature (van Bloemendaal et al., 2014). In large-scale trials for type 2 diabetes, the most frequently reported events include nausea (17–22%), diarrhea (13–16%), and vomiting (6–10%), which are generally mild to moderate in severity (Frías et al., 2021). Safety data from BED-specific trials confirm that while these events are frequent, they are generally manageable through gradual dose titration (Allison et al., 2023). Furthermore, rapid medication-driven weight loss requires careful monitoring in restrictive populations, such as those with Anorexia Nervosa (AN), where appetite suppression may reinforce rigid control or perfectionistic traits, potentially exacerbating severe undernutrition (Krug et al., 2025). Regarding psychiatric safety, evidence suggests that GLP-1 RAs do not significantly worsen depressive symptoms or increase suicidality during short-term clinical exposure (Allison et al., 2023; Aoun et al., 2024).

Table 1. Therapeutic Characteristics of GLP-1 RAs Across the Eating Disorder Spectrum

Feature	Binge Eating Disorder (BED)	Bulimia Nervosa (BN)	Anorexia Nervosa (AN)
Clinical Potential	High (Primary indications)	Exploratory (Limited evidence)	Contraindicated (High risk)
Key Outcomes	~4.0 binge episodes/wk reduction; ~5.2% weight loss	Potential reduction in binge frequency; lacks large RCTs	Risk of reinforcing restrictive pathology and severe undernutrition
Primary Mechanism	Dampening of motivational “wanting” (incentive salience)	Modulation of satiety signaling and reward motivation	Reinforcement of rigid control and perfectionism
Neurobiology	Activation in Hypothalamus, VTA, and NAc	Impact on gut-brain axis and satiety pathways	Reduced food reward sensitivity (pathological)
Safety Profile	GI symptoms (nausea, diarrhea)	Potential to trigger compensatory behaviors (purging)	Critical risk of exacerbating emaciation and starvation

3.6 Socioeconomic and Technological Factors: The Digital Landscape of Access

The dissemination of GLP-1 receptor agonists (RAs) is increasingly mediated by direct-to-consumer (DTC) telemedicine platforms and amplified by algorithmic social media discourse (Hoffman, 2020; Raiter et al., 2023). This technological landscape facilitates rapid access to prescriptions, often through asynchronous models that minimize face-to-face physician interaction (Hoffman, 2020). A prominent feature of this digital trend is the “Ozempic face” phenomenon - a term used to describe the loss of subcutaneous facial fat and subsequent skin sagging following rapid medication-driven weight loss (Carboni et al., 2024). While these platforms can democratize access for some, they simultaneously create pathways for medication misuse among individuals whose restrictive or purging behaviors may go undetected during simplified online screening processes (Krug et al., 2025; Hoffman, 2020)

4. Discussion

4.1 Interpretation of Findings

The therapeutic promise of GLP-1 RAs for BED is highlighted by pilot data showing a clinical magnitude of binge reduction numerically comparable to FDA-approved stimulants, despite the lack of statistical significance in early small-scale trials (Allison et al., 2023; McElroy et al., 2015). The primary value of these agents in the BED population appears to be their robust metabolic impact, addressing the high prevalence of comorbid obesity more effectively than traditional psychotherapy alone, which typically fails to produce significant weight loss (Allison et al., 2023; Radkhah et al., 2025; Guerdjikova et al., 2017). The discrepancy between behavioral and metabolic significance in pilot studies likely reflects low statistical power - often exacerbated by medication dispensing errors - rather than a lack of biological efficacy (Allison et al., 2023; Jacobs-Pilipski et al., 2007). This suggests that liraglutide and similar agents serve as potent tools for overall metabolic stabilization, bridging a critical gap in the current treatment landscape for patients with binge-type pathologies and concurrent metabolic distress (Allison et al., 2023; van Bloemendaal et al., 2014).

4.2 Integration with Neurobiological Models

Binge Eating Disorder (BED) is fundamentally associated with dysregulated satiety signaling and hyperactive reward circuitry, particularly within the mesolimbic dopamine system (Guerdjikova et al., 2017; Radkhah et al., 2025). GLP-1 receptor agonists (RAs) act centrally to modulate these pathways, regulating both homeostatic feeding behavior and the non-homeostatic control of food reward (van Bloemendaal et al., 2014; Radkhah et al., 2025). In the context of BED, the clinical significance of these actions lies in their ability to effectively silence “food noise” - the persistent, intrusive thoughts about eating that drive loss-of-control episodes (Alhadeff et al., 2012; Radkhah et al., 2025). By attenuating the activity of the mesolimbic dopamine system and decoupling the dopamine-mediated “wanting” (incentive salience) from the “liking” or pleasure of consumption, GLP-1 RAs function as genuine impulse-control enhancers (Berridge & Robinson, 2016; Radkhah et al., 2025). This neurobiological shift creates a critical “buffer”, allowing patients to transition from reactive, craving-led consumption toward more planned, homeostatic eating patterns (van Bloemendaal et al., 2014; Alhadeff et al., 2012). Such stabilization in the feedback loop between peripheral gastric signals and central reward-seeking increases the efficacy of concurrent cognitive-behavioral interventions (Radkhah et al., 2025). At the hypothalamic level, this is supported by a dual mechanism: enhancing the signaling of anorexigenic POMC neurons while concurrently suppressing the activity of orexigenic NPY/AgRP neurons (Aoun et al., 2024; Müller et al., 2019).

4.3 Safety Considerations

The clinical significance of the GLP-1 RA safety profile in eating disorders extends beyond raw percentages to the psychological management of somatic symptoms (van Bloemendaal et al., 2014). For patients with BED, the high frequency of gastrointestinal distress requires a nuanced therapeutic approach to ensure that physical side effects - such as nausea or indigestion - do not discourage treatment adherence or inadvertently trigger maladaptive compensatory behaviors (Allison et al., 2023; Krug et al., 2025). While the metabolic safety is well-established in broader obesity cohorts (Frías et al., 2021), clinicians must remain vigilant regarding the long-term psychological impact (Krug et al., 2025). Regular assessment of nutritional status and mental health is essential, especially as appetite suppression may reinforce restrictive traits in vulnerable patients (Krug et al., 2025). Consequently, pharmacological intervention should always be integrated within a multidisciplinary framework to provide continuous psychiatric and nutritional oversight (Krug et al., 2025).

4.4 Comparison with Existing Literature

Recent systematic reviews and meta-analyses consolidate the therapeutic potential of GLP-1 RAs for BED, demonstrating a pooled weight loss of 3.81 kg and a significant reduction in BMI (-1.48 kg/m²) and waist circumference compared to controls (Radkhah et al., 2025). Furthermore, these agents significantly improve Binge Eating Scale (BES) scores, with a mean reduction of 8.14 points (Radkhah et al., 2025). These findings are consistent with earlier trials of liraglutide that showed improvements in global eating disorder psychopathology and shape concerns (Allison et al., 2023; Radkhah et al., 2025).

This review aligns with established mechanistic trials confirming that GLP-1 RAs bridge the gap between peripheral gastric signals and central reward-seeking behavior (van Bloemendaal et al., 2014; Alhadeff et al., 2012). Unlike traditional weight-loss strategies that often trigger a compensatory biological drive to eat, the unique ability of these agents to raise the threshold for food-related reward offers a more sustainable pathway for stabilizing eating patterns in populations with binge-type pathologies (van Bloemendaal et al., 2014; Berridge & Robinson, 2016)

4.5 Clinical and Public Health Implications

GLP-1 RAs may serve as a potent adjunct to multidisciplinary treatment for BED with obesity, particularly when established options like Cognitive Behavior Therapy (CBT) are inaccessible (Allison et al., 2023; Radkhah et al., 2025). Integration with psychotherapy and psychiatric monitoring is critical to address the emotional regulation deficits often comorbid with BED (Krug et al., 2025; Radkhah et al., 2025). A critical clinical consideration is the duration of therapy and the risk of relapse; evidence suggests a risk of ED recurrence following treatment discontinuation or the attenuation of medication effects over time (Radkhah et al., 2025). This raises the question of whether GLP-1 RA treatment for BED should be framed as a chronic intervention, mirroring protocols for type 2 diabetes (Allison et al., 2023).

Public health messaging must be carefully managed to counteract the social media glamorization of rapid weight loss and cosmetic side effects like “Ozempic face”, which often ignore the psychological complexity of eating disorders (Carboni et al., 2024; Krug et al., 2025). To ensure safe use, a transition toward multidisciplinary monitoring and the integration of “smart digital phenotyping” or AI-driven pharmacovigilance may be necessary to flag early behavioral signals of restrictive patterns that current telemedicine screens often miss (Krug et al., 2025; Hoffman, 2020)

4.6 Ethical and Social Dimensions

The rapid expansion of GLP-1 RA use presents ethical challenges that transcend individual clinical success. Digital environments, particularly TikTok, act as a “social contagion” by glamorizing extreme appetite suppression and validating rapid weight loss as a status symbol (Raiter et al., 2023; Krug et al., 2025). This intensifies weight-related stigma and may act as a trigger for vulnerable populations, potentially shifting the societal focus back toward extreme thinness under the guise of 'wellness' (Raiter et al., 2023; Carboni et al., 2024).

Furthermore, the “frictionless” access provided by DTC telemedicine lacks the diagnostic nuance required to detect subtle signs of Anorexia Nervosa or Bulimia Nervosa (Hoffman, 2020; Krug et al., 2025). For patients with undisclosed restrictive pathologies, these platforms may inadvertently reinforce rigid control and perfectionistic traits by providing potent appetite suppressants without psychiatric gatekeeping (Krug et al., 2025). Socioeconomic inequities also risk creating a two-tiered healthcare system; the high cost of these agents may exclude marginalized groups from metabolic care, while simultaneously risking that purely biological symptom management replaces essential psychological interventions like CBT, which are already limited by geographical and financial barriers (Krug et al., 2025; Allison et al., 2023; Radkhah et al., 2025)

4.7 Limitations of the Review

While this review synthesizes a broad range of evidence, it is subject to several limitations inherent in the current state of the literature. As a narrative synthesis, it cannot provide a standardized quantitative meta-analysis of all behaviors due to the high degree of heterogeneity in study designs, dosages, and participant populations. A primary limitation is that individuals with “complex” psychiatric histories or active eating disorders were frequently excluded from large-scale randomized controlled trials, which severely restricts our knowledge regarding the psychiatric safety of these agents in real-world clinical settings.

Additionally, while pilot trials offer promising clinical insights, their sample sizes are often small and results are sometimes compromised by administrative or dispensing errors. The follow-up durations in existing research are generally short, often limited to less than six months, leaving the long-term effects on weight maintenance and binge cessation unknown. Finally, the demographic focus of many trials has been limited to specific BMI categories and predominantly White and female participants, which limits the generalizability of these findings to more diverse populations, including men and individuals across different weight spectrums.

4.8 Leveraging Technology for Safety

Given the intersection of medical innovation and psychological risk, the future implementation of GLP-1 RAs requires innovative technological and multidisciplinary safeguards. To ensure safe use, the following strategies could be utilized:

- AI-Driven Pharmacovigilance: Utilizing natural language processing to monitor electronic health records and clinical notes for early linguistic or behavioral signals of restrictive eating patterns, particularly in patients prescribed these agents for weight management.

- Smart Digital Phenotyping: Implementing wearable technology to monitor more than just weight change. Tracking distinct shifts in activity levels, sleep, or the timing of caloric intake could alert clinicians to a transition from healthy weight loss to pathological restriction or over-exercise.
- Integrated Care Applications: Developing companion digital health platforms for GLP-1 RA users that incorporate mandatory modules on intuitive eating, body image resilience, and psychological check-ins. Such tools could bridge the gap between a purely pharmacological effect on the gut and the necessary behavioral and emotional support required for holistic recovery.

These technological advancements must be co-designed with input from individuals with lived experience to ensure they reduce stigma and support psychologically informed care rather than merely biological symptom management.

5. Conclusions

GLP-1 receptor agonists represent a promising pharmacological adjunct for the treatment of Binge Eating Disorder (BED), particularly for patients with comorbid obesity. Clinical evidence from pilot randomized trials and recent meta-analyses demonstrates that these agents can achieve a reduction of 4.0 binge episodes per week and a significant body weight loss of approximately 5.1% to 5.2%. While statistical significance in binge reduction was not consistently reached in initial pilot data due to small sample sizes and administrative errors, the clinical magnitude of these changes - including an 8-point improvement in Binge Eating Scale scores - highlights their therapeutic potential. These effects are underpinned by a dual neurobiological mechanism: the enhancement of homeostatic satiety signaling in the hypothalamus and the dampening of dopamine-mediated reward pathways in the mesolimbic system. By specifically targeting the motivational drive to seek palatable food - the “wanting” or incentive salience - GLP-1 receptor agonists address both the physiological and psychological aspects of binge-type eating pathology.

In Bulimia Nervosa (BN), GLP-1 receptor agonists remain strictly investigational. While the mechanistic rationale suggests that modulating reward pathways could reduce binge frequency, the current absence of large-scale, robust clinical trials prevents definitive conclusions. Given that BN involves a complex cycle of bingeing and compensatory behaviors often driven by emotional dysregulation, these agents should be viewed as potential adjunctive tools rather than primary interventions. Their use in this population requires careful integration with established psychotherapy and nutritional counseling to ensure the underlying psychopathology is addressed. Conversely, the use of GLP-1 receptor agonists in Anorexia Nervosa (AN) and other restrictive eating disorders is strongly discouraged and essentially contraindicated. The appetite-suppressing and weight-lowering effects of these medications pose a direct physiological danger, as they may reinforce rigid perfectionistic control over caloric intake and exacerbate severe undernutrition.

The emergence of GLP-1 receptor agonists transcends a purely clinical narrative; it constitutes a critical moment in health policy and social science discourse regarding the impact of innovative technologies on public health. The shift toward asynchronous, direct-to-consumer (DTC) telemedicine represents both a revolution in access and a potential threat to psychiatric safety. The ease with which patients can bypass traditional multidisciplinary assessments creates a high-risk scenario where subtle signs of underlying restrictive disorders may go undetected. This necessitates an urgent call for technological innovation focused on robust digital screening tools specifically calibrated to flag eating disorder risk factors beyond simple BMI metrics. Future public health strategies should prioritize the integration of AI-driven safeguards and digital phenotyping to ensure psychiatric safety in telemedicine models.

Furthermore, the sociological impact of these therapies, often dubbed the “Ozempic Effect” on social media, demands cautious analysis. Public discourse frequently simplifies these potent pharmaceuticals into cosmetic tools for weight loss, normalizing extreme appetite suppression and contributing to a societal climate that is both appearance-centric and weight-stigmatizing. For vulnerable populations, this digital environment acts as a social contagion, potentially triggering disordered eating patterns by validating extreme weight loss as a status symbol. Additionally, the high cost of these agents risks creating a two-tiered healthcare system where pharmacological metabolic management becomes a privilege reserved for the economically advantaged.

In synthesis, while GLP-1 receptor agonists offer a beacon of hope for patients suffering from Binge Eating Disorder, their successful and ethical integration into healthcare systems requires a sophisticated, multidisciplinary, and technologically responsible approach. Integration with psychotherapy and psychiatric monitoring is essential to ensure that metabolic correction does not come at the expense of holistic psychological recovery. Future research must prioritize long-term, disorder-specific studies to better understand the durability of these effects and ensure that medical innovation supports individualized, patient-centered treatment paradigms.

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