Current Psychiatry Reports (2026) 28:78
https://doi.org/10.1007/s11920-026-01726-9

REVIEW

Miracle and/or Minefield? GLP-1 RA Drugs and Eating Disorders

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Received: 14 September 2026 / Accepted: 9 November 2026
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Abstract

Purpose of Review Glucagon-like peptide-1 receptor agonists (GLP-1 RAs) have transformed the treatment of obesity and type 2 diabetes (T2D), but their rapid uptake has outpaced understanding of their effects in individuals with eating disorders (EDs). GLP-1 RAs produce sustained effects on appetite, satiety, food reward, and energy intake, raising the possibility of markedly different consequences across ED presentations.

Recent Findings Preliminary studies suggest potential benefit for binge-eating symptoms, consistent with emerging evidence of overlap between binge eating and reward- and impulse-related pathways. Conversely, appetite suppression and attenuation of compensatory responses to negative energy balance could facilitate or mask restriction in individuals vulnerable to anorexia nervosa (AN) or atypical anorexia nervosa (AAN). Empirical evidence remains sparse.

Summary We review neurobiological and genetic evidence relevant to these potentially divergent effects, summarize the clinical literature, and highlight implications for screening and monitoring. Determining who may benefit, who may be harmed, and how to identify risk before treatment should be priorities for future research.

Keywords Eating disorders · GLP-1 RAs · Restriction · Binge eating · Relapse · Disordered eating

Introduction

Glucagon-like peptide 1 receptor agonists (GLP-1 RAs) are not a new therapeutic class: the first agent, exenatide, was approved by the U.S. Food and Drug Administration in 2005 for type 2 diabetes (T2D). Subsequent approvals of liraglutide (2010 for T2D; 2014 for chronic weight management) and dulaglutide (2014 for T2D) established the class as an effective tool in glycemic control, with weight loss recognized as a consistent but modest effect in the context of use for T2D. These earlier agents also had daily or early extended-release dosing and narrow uptake outside of T2D populations. Consequently, GLP-1 RAs remained fairly ringfenced for over a decade.

That is until the advent of longer-acting, more potent, once-weekly agents, particularly semaglutide in 2017, which combined greater weight-loss efficacy with less frequent dosing and broader indications. Semaglutide approval for chronic weight management in 2021 marked a key inflection point, after which GLP-1 RAs transitioned from niche T2D therapies to widely used metabolic agents with expanding applications across obesity and associated health conditions.

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Published online: 17 November 2026

This acceleration is reflected in research output, prescribing volume, and public search interest from 2022 onward [1–4]. Clinical and research applications have expanded to cardiovascular disease, chronic kidney disease, neurodegenerative conditions, osteoarthritis, and inflammatory pathways—an expansion described as reflecting "unprecedented broad efficacy" of this drug class [5]. Widespread coverage across traditional and social media amplified enthusiasm, infiltrating the general public with speed reminiscent of Viagra, statins, and Covid vaccines [6–8].

Countering this enthusiasm is a debate over their safety and efficacy in people with eating disorders (EDs)—a population that was excluded from clinical development trials. Commentaries have warned that these medications "may be too effective for unmonitored weight loss," patient accounts describe their experience as "doctor-approved anorexia," patients recovered from EDs report relapsing, and scholarly reports and popular media describe new onset cases [9–14]. Complicating matters, a smaller but important body of research is reporting reduced binge frequency and improved eating disorder-related psychopathology in individuals with binge-eating disorder (BED) who receive GLP-1 RAs [15–18].

This poses an intriguing dilemma for the ED field. The same medications that may be harmful for some individuals with EDs, hold promise for helping others. To inform this paradox, we explore neurobiological and genetic mechanisms that may help understand why they may be harmful for some and beneficial for others, through the lens of shared biology of binge-type EDs with substance use disorders (SUDs). We then summarize the current (and rapidly expanding) literature and discuss best practices and consensus recommendations for the use of GLP-1 RAs in individuals with EDs.

Mechanisms of Action of GLP-1 RAs

Glucagon-like peptide-1 (GLP-1) is an endogenous peptide produced from proglucagon and released predominantly by intestinal enteroendocrine cells after nutrient ingestion. An incretin hormone, GLP-1 enhances glucose-dependent insulin secretion, restrains glucagon secretion in a glucose-dependent manner, slows gastric emptying, and engages neural pathways that regulate satiety and appetite [19]. Foundational work in the 1980s identified the truncated peptide GLP-1 [7–37] as a biologically active form capable of stimulating insulin release in the presence of glucose [20]. Subsequently, the GLP-1 [7–36] amide was shown to act as a physiologic incretin in humans [21]. Studies in the 1990s demonstrated the therapeutic potential of GLP-1 in T2D [22]. Together, these discoveries laid the foundation for the development of GLP-1 RAs as therapeutic agents.

GLP-1 receptors are distributed across peripheral tissues and central and peripheral neural circuits, including hindbrain, hypothalamic, vagal, and reward-related pathways. Although the appetite and weight loss effects of these drugs are mediated through interacting central and peripheral pathways, appetite regulation is complex and biologically redundant [19]. Within the hypothalamus, GLP-1 signaling engages opposing neuronal populations in the arcuate nucleus that balance hunger and fullness: proopiomelanocortin (POMC) neurons, which promote satiety, release α-melanocyte-stimulating hormone (α-MSH), which activates the melanocortin-4 receptor (MC4R), reducing hunger and food intake. Agouti-related peptide (AgRP) neurons act in opposition, releasing AgRP (an endogenous antagonist of MC4R), thereby promoting hunger and food-seeking behavior. Preclinical studies indicate that some GLP-1 RAs can activate POMC neurons and inhibit NPY/AgRP neurons, shifting this circuit toward satiety [23–25]. Other areas of the brain, including the area postrema, which is a key mediator of nausea and vomiting, also express GLP-1 receptors and are implicated in the common gastrointestinal side effects seen with GLP-1 RAs [26].

Pharmacologic Effects and Duration of Action

Like endogenous GLP-1, GLP-1 RAs increase fullness and satiety, decrease hunger [27, 28], can cause nausea and slow gastric emptying [19], thereby contributing to reduced energy intake and weight loss. The subjective experience is reported as feeling sated with less food, staying full longer, and experiencing reduced food craving and food-seeking behavior. Unlike endogenous GLP-1, which exhibits a brief post-prandial elevation and rapid decay [29], GLP-1 RAs produce pharmacologic receptor activation that can persist for days, reflecting their substantially longer half-lives [29]. Rather than gently amplifying the body's natural signals like a ripple in a pond, they function more like a high tide, producing greater and more persistent activation of pathways that regulate appetite, satiety, and energy balance. The magnitude and duration of these effects contribute to their impact on eating behavior and weight regulation.

These effects are evident in clinical trials. In a randomized crossover study of adults with obesity, 12 weeks of once-weekly semaglutide reduced total ad libitum energy intake by ~24%, with participants losing an average of 5.0 kg. Participants reported less hunger, fewer food cravings, and improved perceived control of eating relative to their 12-week placebo period, during which participants gained about 1.0 kg [27]. A 60-week trial found reduced

food preoccupation at 20 weeks and diminished responsiveness to the rewarding value of food through 40 weeks, while reductions in energy intake persisted throughout the trial [30]. Some of these experiences overlap with the popular term "food noise" which has been used to describe constant hunger and cravings, preoccupation with food, lack of control over eating, and desire for highly palatable foods [31].

Reward Circuitry and Overlap with Substance Use Disorder

Emerging pre-clinical and clinical studies suggest that GLP-1 RAs may reduce some substance use and substance-seeking behaviors [32–35], potentially through effects on brain systems involved in reward, motivation, and craving. GLP-1 receptors are distributed across multiple brain regions implicated in reward and reinforcement. Preclinical studies indicate that GLP-1 receptor activation can modulate mesolimbic dopaminergic signaling and reduce the reinforcing effects of addictive substances [36]. GLP-1 signaling also modulates glutamatergic and GABAergic neurotransmission, and in preclinical models, dampens cue-induced craving and relapse-like behaviors, and alters reward-related signaling through gut-brain pathways [36]. Collectively, these findings suggest that GLP-1 RAs may reduce the motivational salience of reinforcing stimuli including addictive substances and highly rewarding foods.

Research on SUDs may provide insights into the potential effects of GLP-1 RAs on binge-eating phenotypes because these conditions share some features of altered reward processing, impulsivity, and compulsive behavior. Neuroimaging studies in BED have identified alterations in reward-related circuitry and connectivity with prefrontal regions involved in cognitive control [37–39]. Recent genomic findings provide complementary evidence for this overlap. In the Termorshuizen et al. [40] genome-wide association study (GWAS), binge-eating behavior (BE-BROAD) showed stronger genetic correlations than anorexia nervosa (AN) with attention deficit/hyperactivity disorder, problem alcohol use, heavy smoking, and general risk tolerance, whereas AN showed a stronger genetic correlation with obsessive compuslive disorder. These findings suggest that binge eating and AN have partially distinct genetic relationships with behavioral disinhibition and compulsivity, respectively. Rare variants in GLP-1-related genes have also been reported in bulimia nervosa (BN) and BED [41], although the generalizability of these findings to the broader population of individuals with BN and BED remains to be established.

These converging observations provide a rationale for investigating whether GLP-1 RAs might preferentially affect binge-eating symptoms. Consistent with this possibility, preliminary studies suggest that GLP-1 RAs may reduce binge-eating frequency or severity (see below) [16, 18, 42].

A second mechanism relevant to both the potential benefits and risks of GLP-1 RAs in EDs is their effect on the usual response to negative energy balance (NEB). Under usual physiological conditions, NEB elicits compensatory responses that promote restoration of energy stores, including increased hunger and food motivation and reduced energy expenditure. The psychological consequences of prolonged NEB were demonstrated strikingly in the Minnesota semi-starvation study, in which participants developed intense preoccupation with food, changes in mood and behavior, and episodes of overeating during the post-experimental renourishment period [43]. GLP-1 RAs alter these compensatory responses designed to sustain life: individuals can remain in NEB and lose weight while experiencing reduced hunger, food cravings, and food preoccupation. Thus, GLP-1 RAs may attenuate many of the compensatory signals that ordinarily defend against weight loss and that make dieting so challenging. Critically, the ability of GLP-1 RAs to attenuate NEB-associated hunger and food reward may have very different implications across eating-disorder phenotypes, and herein lies the complexity with the use of these medications in individuals with eating disorders. For those with binge-eating symptoms, the reduction of hunger, craving, food reward, and "food noise" could potentially reduce key drivers of binge episodes. GLP-1 RAs have been associated with reduced preference for highly palatable, energy-dense foods [27, 28], and preliminary clinical studies suggest reductions in binge-eating symptoms [16, 18, 42]. These effects are consistent with, although do not confirm, the reward- and impulse-related mechanisms described above.

The same effects could be problematic in restrictive EDs. In AN and AAN, reducing hunger, food reward, and motivation to eat could facilitate continued caloric restriction and make NEB easier to maintain. This is particularly concerning because NEB itself produces physiological and psychological adaptations that can perpetuate illness and contribute substantially to the medical morbidity of AN and AAN [44–46].

These differential effects must also be considered in the context of symptom and diagnostic crossover among EDs [47–50]. Individuals may experience restriction, binge eating, and compensatory behaviors at different points in the course of illness. Thus, a medication that reduces binge-eating symptoms at one stage could potentially facilitate problematic restriction at another. This may be particularly relevant for individuals with underlying liability to restrictive EDs, although whether GLP-1 RA-induced NEB can precipitate or exacerbate AN in genetically susceptible individuals remains an important research question.

Empirical Evidence in Eating Disorders

Prior to the development of GLP-1 RA medications, researchers investigated the role of endogenous GLP-1 in people with EDs. Findings vary by diagnosis. Studies of AN have reported both elevated and reduced endogenous GLP-1 relative to controls [51–54]. BN studies more consistently suggest attenuated GLP-1 secretion [55–57], whereas BED studies have yielded mixed findings [58, 59]. Thus, endogenous GLP-1 findings do not support a consistent pattern of dysregulation across EDs. Research on GLP-1 RAs in individuals with EDs is sparse and studies in BED have yielded mixed findings (Table 1). A case report of a dramatic decrease in self-report binge-eating symptoms that reversed upon discontinuation [60]. Two studies reported greater reductions in binge-eating symptoms with GLP-1 RAs than comparison medications [16, 18], whereas a small randomized controlled trial and a post hoc analysis of a bipolar disorder trial found no significant advantage of liraglutide over placebo [15, 17]. Evidence from non-ED samples provides additional information about eating behavior (Table 2): GLP-1 RA treatment has been associated with increased dietary restraint or restriction [62, 63], reduced hunger [61, 68], and reductions in food craving, preoccupation, and hedonic responses to food [30, 61, 68], although findings are not uniform and some effects attenuate over time or following discontinuation [64, 67]. Baseline emotional eating, eating in response to external stimuli, and restrained eating may moderate the effects of liraglutide on brain responses to food cues [65]. Qualitative studies similarly describe smaller portions, earlier fullness, and greater perceived control over eating [64, 66, 67].

No controlled studies have evaluated GLP-1 RA medications in individuals with current or past restrictive EDs. Available evidence is limited to case reports describing misuse, relapse, or emergence of AN/AAN during GLP-1 RA treatment (Table 3) [69–72]. While case reports do not establish causality or estimate risk at a population level, these reports raise concern that GLP-1 RAs could exacerbate restrictive eating in vulnerable individuals.

Table 1 Empirical studies in populations with binge-eating disorder

Author, Year	Drug	Design	Population	Key Findings
Allison et al., 2023[15]	Liraglutide 3.0 mg	Pilot RCT	Adults with BMI ≥ 27 kg/m^2 and BED	Both groups reduced objective binge episodes with no statistically significant difference. The study was significantly limited by a pharmacy dispensing error.
Da Porto et al., 2020 [16]	Dulaglutide	Open label, pilot RCT	Adults with type 2 diabetes, HbA1c 7.5-9.0%, and BED taking metformin	Dulaglutide led to significantly greater reductions in Binge Eating Scale (BES) scores compared to gliclazide ($p < 0.001$).
Richards et al., 2023 [18]	Semaglutide	Retrospective cohort comparing semaglutide alone, semaglutide + OAOM, OAOM alone	Adult patients of an obesity medicine and bariatric surgery clinic with likely moderate to severe BED based on BES scores	Semaglutide monotherapy achieved significantly greater reductions in BES scores compared to traditional anti-obesity medications alone ($p < 0.01$). Adding topiramate or lisdexamfetamine to semaglutide did not significantly change BES score reduction compared to semaglutide alone.
Gong & Wentworth 2024 [60]	Semaglutide	Case Report	27-year-old woman with BMI > 30 kg/m² and type 1 diabetes, comorbid anxiety, and BED	BES score dropped from 39 (severe) to 3 (little/no binge eating) after 4 weeks of semaglutide; improvement in glucose control compared with similar insulin adherence and a lower dose. Binge eating recurred (BES score increased to 41), accompanied by worsened glucose control during a 1-month lapse in access to semaglutide. Subsequent 8 months of sporadic semaglutide supply/intermittent dosing still remitted binge eating symptoms (BES was 4 at 11-month follow-up). Authors report semaglutide did not exacerbate disordered eating in this patient.
Guerdjikova et al., 2025 [17]	Liraglutide 3.0 mg	Post hoc analysis of 40-wk placebo-controlled RCT in bipolar disorder	Adults with obesity/overweight and stable bipolar disorder, (analysis stratified for BED status)	Participants with BED had higher binge-eating severity (BES), disinhibited eating and hunger than those without BED. Among participants with BED ($n = 17$), liraglutide and placebo produced similar reductions in binge-eating severity; there was no significant difference between drug and placebo groups.

OAOM other anti-obesity medication, lisdexamfetamine and topiramate

RCT randomized controlled trial

BED binge-eating disorder

BMI body mass index

BES Binge Eating Scale

Table 2 Empirical studies of GLP-1 RA medications with eating behavior secondary outcomes

Author, Year	Drug	Design	Population	Key Findings
Gatta-Cherifi et al., 2024 [61]	Exenatide	Double-blind, multicenter, placebo-controlled RCT	Adults with craniopharyngioma-related obesity	Exenatide reduced self-reported hunger more than placebo over 26 weeks. No significant differences were observed in loss of control over eating or cognitive restraint between groups.
Koide et al., 2025 [62]	Liraglutide, dulaglutide, semaglutide	Multicenter prospective observational, 12 months	Adults with T2D starting a GLP-1 RA	Eating driven by external cues (sight/smell of food) dropped and stayed lower through 12 months. Eating driven by emotions dropped at 3 months but returned to baseline by 12 months. Conscious food restriction increased at 3 months but also returned to baseline by 12 months.
Behary et al., 2023 [63]	GLP-1 + oxyntomodulin + PYY infusion	Secondary analysis of RCT, with gastric-bypass comparator group	Adults with obesity and prediabetes/T2D	Four weeks of hormone infusion increased deliberate food restriction to a similar degree as gastric bypass surgery. It did not change food preferences.
Coppin et al., 2023 [64]	Liraglutide 3.0 mg	Double-blind, placebo-controlled RCT, 16 weeks, with fMRI	Adults with obesity, no diabetes	Despite significant weight loss, liraglutide did not reduce how much participants liked eating a high-calorie food, either by self-report or by brain activity. Weight loss occurred without a measurable drop in food liking.
Tronieri et al., 2026 [30]	Semaglutide 2.4 mg	Double-blind RCT, 60 weeks	Adults with overweight/obesity	Semaglutide reduced food preoccupation and responsiveness to food cues more than placebo at 20 weeks; these differences mostly became non-significant by 40–60 weeks even though semaglutide's effect on actual calories eaten persisted.
van Ruiten et al., 2022 [65]	Liraglutide	Secondary analysis of randomized crossover trial (liraglutide vs. insulin glargine, 12 weeks each), with fMRI	Adults with obesity BMI ≥ 26 kg/m^2 and T2D	Participants who ate more in response to emotions or external food cues at baseline showed less change in brain reward-region response to liraglutide. Participants with more deliberate food restriction at baseline showed more change in brain response. Baseline eating style predicted how much the drug affected the response to food in the brain.
Goetz et al., 2025 [66]	Retatrutide	Qualitative exit interviews, phase 2 trial	Adults with obesity/overweight exiting phase 2 RCT	Participants described eating less often, eating smaller portions, feeling hungry less often, feeling full sooner, and feeling more in control of their eating, compared to before the trial.
Kiyosue et al., 2026 [67]	Tirzepatide	Qualitative exit interviews (SURMOUNT-J)	Adults with obesity, trial completers	During treatment, most participants reported appetite loss (75.9%) and conscious changes in eating habits (72.4%), including increased attention to food choices/portions; a minority (10.3%) reported loss of joy in eating. After discontinuation, 65.5% reported a return of poor appetite control, though 51.7% maintained some eating-behavior changes (e.g., reduced desire for greasy foods).
Martin et al., 2025 [68]	Tirzepatide vs. liraglutide vs. placebo	Randomized, partially blinded phase 1 RCT, 6 weeks, with fMRI	Adults without diabetes, BMI 27–50 kg/m^2	At 3 weeks, tirzepatide reduced food cravings, the tendency to overeat, and brain response to food images, compared to placebo.

RCT randomized controlled trial

fMRI functional magnetic resonance imaging

T2D type 2 diabetes

BMI body mass index

Uncertain Effects of GLP-1 RA Discontinuation

The use of GLP-1 RAs in individuals with EDs is further complicated by the effects of medication discontinuation. Whether improvements in binge-eating symptoms or food preoccupation persist after GLP-1 RA discontinuation is unknown. Evidence from obesity trials demonstrates substantial weight regain after discontinuation, but comparable data on eating behaviors and cognitions are lacking.

A recent review and meta-regression pooling six placebo-controlled clinical trials reported progressive weight regain after GLP-1 RA cessation. Participants regained ~ 60% of their treatment-related weight loss by one year after discontinuation, with regain projected to plateau at approximately 75% of the weight lost [73]. In the two dedicated drug withdrawal trials, participants who were switched to placebo regained approximately 7% and 14% of body weight over the following year [74, 75].

Real-world data are more heterogeneous. Discontinuation is common: 46–65% of individuals discontinue treatment

Table 3 Case studies of GLP-1 RA use in restrictive eating disorders (AN/AAN)

Author, Year	Drug	Population	Key Findings
Guerdjikova et al., 2024 [70]	Semaglutide	Adult woman with history of AN and current AAN diagnosis at the time of prescription	The patient intentionally misused semaglutide by taking more than prescribed and shortening dosing intervals, losing > 40 lbs over several months.
Liekens, Kaïret, & Elst, 2026 [71]	Semaglutide	Adolescent girl, no prior ED diagnosis, prescribed by general practitioner for weight concerns	The patient was diagnosed with atypical AN during hospitalization with bradycardia and pericardial effusion. She continued losing weight after discontinuation.
Guerdjikova et al., 2025 [69]	Tirzepatide	Adult woman with obesity (BMI 37), T2D, and history of restrictive AN (no prior ED treatment) at time of prescription	Rapid weight loss over 14 months on tirzepatide use was associated with relapse of AN symptoms. Even after physician discontinued the prescription, patient continued to use saved doses.
Saito et al., 2026 [72]	Semaglutide, then tirzepatide	Patient with severe obesity and T2D, post sleeve gastrectomy	The patient developed atypical AN following sequential postoperative treatment with semaglutide and tirzepatide.

AN anorexia nervosa

T2D type 2 diabetes

within the first year [76, 77]. Unlike the controlled withdrawal trials, discontinuation in observational studies may be unplanned (typically related to cost, medication availability, or side effects) and often followed by reinitiation or transition to another treatment. Consequently, estimates of post-discontinuation weight regain in real-world studies vary considerably from little or no average weight change at one year in large EHR cohorts [76] to substantial regain in smaller cohorts, with one observational study reporting that 49% of patients who had previously lost weight exceeded their pre-treatment weight within one year of discontinuation [78]. Largely unstudied are the effects of discontinuation and reinitiation on hunger, satiety, food preoccupation, binge-eating, and other eating behaviors. For individuals with EDs, this evidence gap is particularly concerning because the consequences of discontinuation may extend beyond weight regain to changes in hunger, food preoccupation, binge eating, restriction, and compensatory behaviors.

Clinical Implications

Given the rapid uptake of GLP-1 RAs, clinicians should proactively screen for current and past EDs before initiating treatment and monitor for emerging or worsening symptoms during treatment and following discontinuation. In a nationally representative survey of 1309 individuals, those who screened positive for EDs (especially women) were more likely to have considered taking GLP-1 RAs for weight loss and attempted to access the medications than those who screened negative for EDs [79]. A modified Delphi study recommended brief ED screening before initiation, risk-stratified monitoring, and referral to an ED specialist when screening is positive or concerning symptoms emerge. The panel recommended that GLP-1 RAs generally be avoided in individuals with active AN, AAN, or BN with marked dietary restraint and/or significant weight suppression, with caution in those with a history of these disorders [80]

The intended effects of GLP-1 RAs may complicate clinicians' ability to recognize emerging or recurrent restrictive EDs. Experience with bariatric surgery may provide a useful precedent. Clinically recommended changes in eating behavior following bariatric surgery can mimic some features of disordered eating, complicating recognition of emerging restrictive pathology [81, 82]. With GLP-1 RAs, reduced appetite and food intake are expected pharmacological effects but could also facilitate or mask pathological restriction in vulnerable individuals [10, 83]. Distinguishing therapeutic appetite suppression from emerging ED pathology is an important clinical challenge and although guidance for clinicians is emerging [80], validated approaches for doing so are lacking. Clinicians should therefore monitor not only weight and nutritional status but also changes in ED cognitions and behaviors by tracking within-person change on items from brief validated measures such as the Eating Disorder Examination Questionnaire – Short [84].

Patients may access these medications outside of their usual healthcare system through online providers, medical spas, and other unregulated sources [10, 85, 86]. A 2024 survey indicated that approximately one in six users obtained GLP-1 RAs from an online provider and approximately one in ten from a medical spa [86]. Similarly, in the nationally representative survey, those who screened positive for an ED were significantly more likely to have attempted to access these medications through a telehealth pharmacy than those who screened negative [79]. Clinicians should therefore ask patients directly about GLP-1 RA use rather than assuming that absence of a prescription in the medical record indicates nonuse. They should also inquire about the source of the medication, particularly given concerns about the quality and composition of non-FDA-approved compounded preparations [87, 88].

Conclusion and Future Directions

The available evidence suggests potentially different risk–benefit profiles of GLP-1 RAs across ED presentations, but the evidence remains insufficient to quantify either benefit or harm. Preliminary studies suggest possible benefit for binge-eating symptoms, whereas case reports raise concern about restrictive ED onset, relapse, and misuse. Whether GLP-1 RAs precipitate new EDs or exacerbate underlying vulnerability, and which individuals are at greatest risk, remain unknown. Large longitudinal studies are needed to estimate incidence, identify moderators of risk, and distinguish temporal association from causation. Of particular concern, GLP-1 RA use among individuals with EDs may exceed that in the general population [89], underscoring the critical need for active pharmacovigilance in this population and inclusion of EDs in post-marketing adverse outcome reporting.

Future studies should move beyond weight outcomes to assess ED-specific cognitions and behaviors, including restriction, binge eating, compensatory behaviors, food preoccupation, body image, and medication misuse, during treatment and following discontinuation. Studies should determine whether effects differ by ED diagnosis, symptom profile, prior ED history, and genetic liability, and should include individuals with EDs who were largely excluded from preapproval trials. Surveillance cannot rely exclusively on routine healthcare records because some GLP-1 RA use occurs through online and other alternative channels; linkage of EHR and registry data with population-based surveys and prospective cohorts would enable capture of exposure and outcomes more completely.

GLP-1 RAs may ultimately prove beneficial for some individuals with EDs and harmful for others. Determining who benefits, who is vulnerable to harm, and how those individuals can be identified before treatment will require moving beyond weight as the dominant outcome and systematically measuring ED psychopathology throughout treatment and after discontinuation.

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    - A consensus document based on modified Delphi methodology yielding critical recommendations for the use of GLP-1 RA medications in individuals with current or past EDs as well as recommendations for post-approval pharmacovigilance for unanticipated emergence or recurrence of EDs associated with GLP-1 RA use and discontinuation.
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    - A thoughtful clinical commentary detailing both the potential benefits and risks of GLP-1 RAs for individuals with EDs and a clear call for those involved with women's and mental health clinicians and researchers, pharmaceutical companies, investors, and policymakers to "leverage wins in the development of GLP-1 RAs to help find solutions for preventing and treating AN."

Author Contributions R.P and L.T. drafted initial outlines of the manuscript. A.M.assisted with literature review and tables. C.B., R.P, L.T. and A.B. iteratively wrote the manuscript text. All authors reviewed the manuscript.

Funding Open access funding provided by Karolinska Institute.

Data Availability No datasets were generated or analysed during the current study.

Declarations

Competing interests CMB receives royalties from Pearson Education, Inc. The other authors have nothing to disclose. Dr. Bulik is also the Founding Director of the Karolinska Institutet Centre for Eating Disorders Innovation (cynthia.bulik@ki.se).

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