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GLP-1-Based Therapies and Relative Energy Deficiency in Sport Among Physically Active Individuals: Plausible Pathways, Evidence Gaps, and Monitoring Considerations

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Abstract

Background: GLP-1 receptor agonists and related incretin-based therapies reduce appetite, energy intake, and body mass. Their growing use and discussion in sport raise concern about possible interactions with low energy availability, although a direct relationship with Relative Energy Deficiency in Sport (REDs) has not been established.

Aim: To evaluate how GLP-1-based therapies may intersect with REDs-relevant pathways in physically active individuals while separating established clinical effects, athlete evidence, and untested inference.

Materials and Methods: A structured narrative review used three PubMed/MEDLINE search streams addressing GLP-1 therapies and sport, appetite/body composition/exercise pathways,

and REDs or low energy availability. Fifty-five full texts were assessed and the evidence was synthesized thematically.

Results: Trials conducted mainly in non-athletic populations show reduced hunger, cravings, and laboratory-measured intake; gastrointestinal effects may further limit food tolerance. Weight loss includes variable reductions in lean-mass indices, but these measures do not establish loss of contractile muscle, strength, or performance. Athlete studies separately associate problematic low energy availability with reproductive, bone, endocrine, injury, and selected performance outcomes. No prospective study has tested progression from GLP-1 exposure to problematic low energy availability and REDs in athletes.

Conclusions: GLP-1-based therapy should be considered a potential modifying factor within the REDs framework, not a proven cause. Current evidence supports individualized monitoring of treatment indication, weight trajectory, dietary adequacy, gastrointestinal tolerance, training, recovery, body composition, function, reproductive and bone health, and eating behavior. Athlete-specific prospective studies are required.

Keywords: GLP-1 receptor agonists, relative energy deficiency in sport, low energy availability, exercise; weight loss

1. Introduction

Glucagon-like peptide-1 receptor agonists and related incretin-based therapies are established treatments for obesity and type 2 diabetes. Randomized studies in adults with overweight or obesity show reductions in hunger, food cravings, and laboratory-measured ad libitum intake during semaglutide or tirzepatide treatment [1–4]. These findings arise mainly from non-athletic populations, and laboratory meal intake is not equivalent to habitual intake or energy availability in active individuals.

Sport relevance is nevertheless emerging. Online bodybuilding communities describe off-label use, self-experimentation, cycling, stacking with other substances, and acquisition outside medical pathways [5], while potential use for weight-making in combat sports has been identified as an unresolved issue [6]. These reports establish relevance to selected sport and physique settings but do not quantify prevalence or clinical consequences.

The concern is not treatment-related weight loss itself, which may improve metabolic health, mobility, and exercise participation when medically indicated. It arises when appetite suppression, altered satiation, gastrointestinal intolerance, or rapid weight loss makes it difficult to match intake to exercise expenditure and physiological requirements, particularly during high training volumes, restrictive eating, repeated weight-making, or pressure for leanness. REDs is a multifactorial syndrome associated with problematic low energy availability (LEA), not every transient reduction in intake [7]. Reduced appetite during treatment should therefore not be equated with LEA, and LEA should not automatically be equated with REDs. The clinical effect of an energy mismatch depends on its magnitude, duration, frequency, within-day distribution, training context, dietary quality, and individual susceptibility [8].

The proposed GLP-1–REDs relationship remains inferential. Clinical studies provide evidence on appetite, intake, gastrointestinal tolerability, weight loss, body composition, and selected functional outcomes; athlete studies separately describe associations between problematic LEA and REDs-related health and performance indicators. Direct prospective studies of energy availability, REDs indicators, bone or reproductive health, injury, recovery, and athletic performance in GLP-1-treated athletes are lacking. Recent reviews have addressed tirzepatide in sport [9], GLP-1 receptor agonists in recreational athletes [10], and obesity-related body composition and performance [11]. The present review instead uses the contemporary IOC REDs framework, distinguishes adaptable from problematic LEA, and explicitly separates direct evidence from the untested inferential bridge between these literatures.

Accordingly, this review examines appetite and energy intake, gastrointestinal tolerability, weight loss, lean-mass indices, exercise and function, sport-specific vulnerability, and practical monitoring in physically active GLP-1 users.

2. Review methods

A structured narrative review was conducted using PubMed/MEDLINE, with searches finalized on 22 July 2026 and a targeted novelty update on 25 July 2026. Search concepts covered GLP-1-based therapies, athletes and physical activity, appetite and energy intake, gastrointestinal effects, body composition, muscle and physical function, and REDs/low energy availability. Eligible publications included primary clinical and athlete studies, systematic reviews, consensus statements, and methodological papers relevant to the review question. Animal-only studies, unrelated or redundant publications, and unavailable full texts for detailed claims were excluded. Titles and abstracts were screened for relevance, followed by assessment of 55 available full texts; 51 were retained, of which 37 were cited, while 3 journal-specific reviews

informed the novelty assessment. Evidence was synthesized narratively as direct GLP-1 clinical evidence, athlete/REDs evidence, and contextual or methodological evidence.

3. REDs and Problematic Low Energy Availability

3.1. Conceptual Framework

Energy availability is the dietary energy remaining for physiological functions after exercise energy expenditure has been subtracted and is conventionally expressed relative to fat-free mass. It differs from total energy balance; stable body mass or a normal body mass index does not exclude inadequate availability. The 2023 IOC framework treats LEA as a continuum [7]. Adaptable LEA is short term and associated with minimal or rapidly reversible effects, whereas problematic LEA is more severe, prolonged, recurrent, or maladaptive. Its consequences vary with the magnitude, duration, frequency, origin, within-day distribution, training context, and individual susceptibility [8].

REDs is a possible clinical outcome of problematic LEA, not a synonym for reduced intake. It may affect reproductive, bone, metabolic, hematological, cardiovascular, gastrointestinal, immune, psychological, and performance-related domains, although evidence strength differs across outcomes [7]. Reproductive and bone outcomes have a comparatively developed evidence base, whereas several neurological, psychological, and performance domains rely more heavily on observational or indirect data. Problematic LEA may arise from intentional restriction, disordered eating, inadvertent under-fueling, high exercise expenditure, repeated weight cycling, restrictive diets, food insecurity, malabsorption, or disease [12]. Pharmacological appetite suppression could contribute to inadequate intake in some users, but it has not been established as an independent cause of REDs.

3.2. Identification and Athlete Evidence

REDs cannot be diagnosed from a single questionnaire, biomarker, body-composition value, or calculated estimate of energy availability. Free-living energy intake and exercise expenditure are difficult to measure accurately, and error in either component can substantially distort an energy-availability calculation. The IOC REDs Clinical Assessment Tool version 2 (CAT2) combines screening, weighted primary and secondary indicators, and physician-led diagnosis with risk stratification [13]. Differential diagnosis is essential because fatigue, endocrine changes, gastrointestinal symptoms, mood disturbance, recurrent injury, or impaired training response have multiple causes. The Low Energy Availability in Females Questionnaire may

assist screening in populations similar to its validation sample but is not a universal diagnostic tool [14].

Athlete studies report associations between low-EA surrogates and reproductive, bone, metabolic, hematological, psychological, gastrointestinal, and performance outcomes in women [15], with similar clusters described in men and poor performance of low body mass index as an isolated marker [16]. Higher CAT2 severity has been associated with multiple REDs indicators and greater prospective risk of self-reported bone stress injury [17]. Systematic review evidence also links LEA/REDs risk with impaired bone health, illness, injury, and selected performance outcomes, but definitions, thresholds, screening instruments, and study designs are heterogeneous and mostly observational [18]. These limitations preclude a simple exposure-outcome model. None of these studies evaluated GLP-1 exposure; their role here is to identify health and performance domains that require prospective study in physically active users.

4. How GLP-1-Based Therapies May Intersect With REDs-Relevant Pathways

4.1. Appetite and Measured Energy Intake

Appetite and intake suppression is the best documented GLP-1-related pathway relevant to the proposed REDs model. In adults with obesity, semaglutide 1.0 mg reduced laboratory meal intake by 24% after 12 weeks [1], and semaglutide 2.4 mg reduced ad libitum lunch intake by 35% after 20 weeks [2]; oral semaglutide produced effects in the same direction [19]. In a 60-week trial, semaglutide was associated with approximately 292, 240, and 270 kcal lower lunch intake at weeks 20, 40, and 60, respectively, even when some subjective appetite differences were no longer consistent [4]. Tirzepatide reduced lunch intake by an estimated 525 kcal versus placebo at week 3 and improved hunger, cravings, susceptibility to overeating, and responsiveness to food cues [3]; similar reductions in appetite and buffet-meal intake were reported in adults with type 2 diabetes [20]. These trials consistently demonstrate a pharmacological effect on ingestive behavior, but the magnitude and persistence of free-living compensation remain uncertain.

These trials demonstrate pharmacological effects on appetite and controlled meal intake, not LEA. They did not measure habitual intake across training and rest days, exercise expenditure, nutrient timing, carbohydrate availability, recovery nutrition, calculated energy availability, or REDs outcomes. A lower laboratory meal intake may be offset by later intake, dietary planning, or reduced expenditure; conversely, it may underestimate risk when appetite suppression

persists during high training demand. Athlete-specific interpretation therefore requires direct measurement rather than extrapolation from a single meal test.

4.2. Satiation, Gastric Emptying, and Gastrointestinal Tolerability

Early satiation, nausea, vomiting, constipation, diarrhea, and altered gastric emptying may reduce tolerated meal size or disrupt planned intake before, during, or after exercise. Gastric-emptying findings are heterogeneous: liraglutide delayed solid emptying and reduced maximum tolerated meal volume in a small obesity trial [21], whereas semaglutide studies using paracetamol absorption did not show significant delay at 20 weeks [2,19]. A secondary analysis suggested that gastric emptying explained only a small proportion of appetite variation [22], so it should not be presented as the sole mechanism of intake reduction. Gastrointestinal symptoms remain common and evidence for optimal dietary management is limited [23]. Their practical importance is likely to depend on dose escalation, symptom severity, treatment duration, meal composition, training schedule, and the ability to use alternative food or fluid strategies. No study directly tested their effect on athlete fueling or recovery.

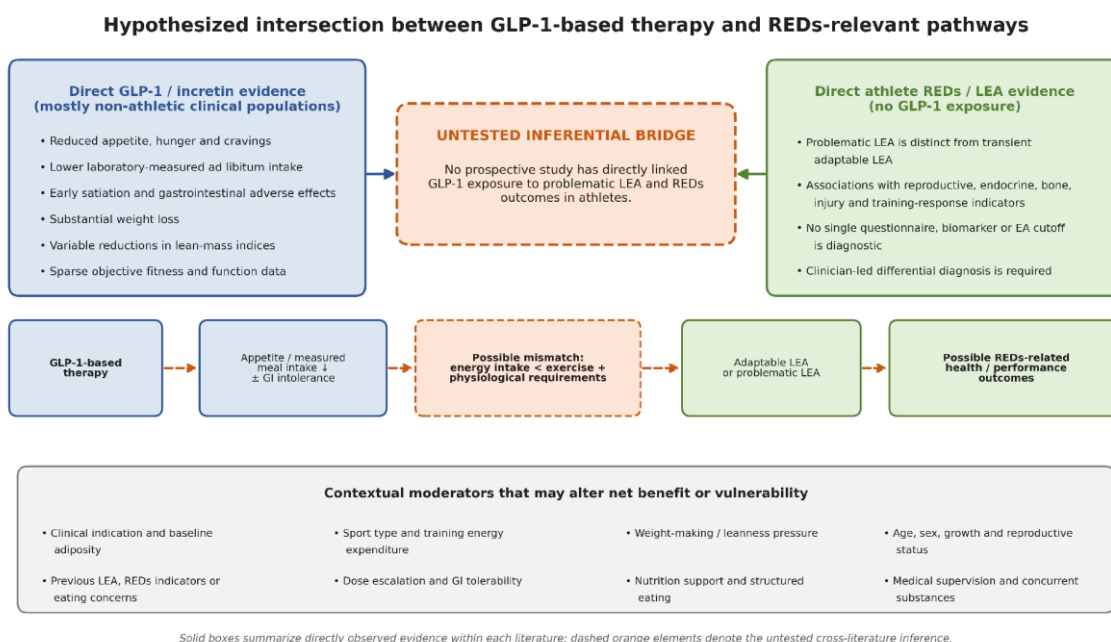
4.3. Weight Loss and Dietary Adequacy

Weight loss is an intended therapeutic effect and is not evidence of REDs. Concern increases when its rate or extent is inconsistent with the clinical indication, growth, nutritional intake, or training demands, or when treatment is used for further loss in an already lean athlete. In 4,726 participants from SURMOUNT-1–4, adverse events potentially indicating overt macronutrient malnutrition were uncommon: abnormal weight loss, underweight, and hypoalbuminemia were each reported in no more than 0.06% of tirzepatide-treated participants, and vitamin-deficiency-related events were similar to placebo [24]. These findings are important counterevidence against assuming that clinically overt malnutrition is frequent during supervised treatment in adults with overweight or obesity. However, the trials were not designed to detect subtle dietary inadequacy, carbohydrate unavailability around exercise, micronutrient insufficiency, REDs indicators, or inadequate intake relative to high training expenditure. Adverse-event terminology may miss gradual or subclinical compromise, and trial monitoring differs from unsupervised sport use. The evidence therefore supports neither an assumption of frequent malnutrition nor reassurance that sport-specific risk is absent.

The proposed sequence remains inferential: treatment can reduce appetite and measured intake, gastrointestinal effects may further constrain intake, and substantial weight loss may reflect sustained energy deficit. Progression to problematic LEA depends on actual intake, exercise

expenditure, physiological requirements, duration, and susceptibility (Figure 1).

Figure 1. Hypothesized intersection between GLP-1-based therapy and REDs-relevant pathways.



Direct clinical GLP-1/incretin evidence demonstrates effects on appetite, measured meal intake, gastrointestinal tolerability, weight loss, and variable lean-mass indices, mainly in non-athletic populations. Direct athlete REDs/LEA evidence identifies affected domains and assessment principles but does not include GLP-1 exposure. The cross-literature sequence from treatment to problematic LEA and REDs outcomes remains untested.

Abbreviations: EA, energy availability; GI, gastrointestinal; LEA, low energy availability; REDs, Relative Energy Deficiency in Sport.

4.4. Lean-Mass and Muscle-Related Indices

Weight loss during GLP-1-based therapy is predominantly fat loss but often includes reductions in lean-mass or muscle-related indices. Fat-free mass, DXA-derived lean soft tissue, bioimpedance estimates, and CT- or MRI-derived muscle measures are not interchangeable. DXA compartments include non-contractile tissue, bioimpedance depends strongly on hydration assumptions, and regional anatomical measures do not independently establish whole-body strength, power, or function. Changes in glycogen, water, organ mass, and connective tissue may also contribute to apparent lean-mass loss.

A meta-analysis of 36 randomized trials included 21 GLP-1 receptor agonist and 15 sodium-glucose cotransporter-2 inhibitor trials. In the GLP-1 subgroup, the pooled mean difference in lean body mass was -1.51 kg (95% CI, -2.00 to -1.01); in the combined analysis, lean mass accounted for 28% (95% CI, 22%-34%) of total weight loss [25]. Other syntheses reported smaller reductions in muscle-related measures than in fat mass [26] and estimated lean-mass proportions of 35.2% with semaglutide, 25.4% with tirzepatide, 26.8% with liraglutide, and 26.2% with lifestyle intervention [27]. These heterogeneous estimates are not fixed drug-specific rates or percentages of contractile muscle loss.

Among 35 randomized studies reviewed by Batsis et al., the median proportion of weight loss attributed to muscle-related indices was 28.3% (interquartile range, 15.9%-39.9%); heterogeneity precluded pooling and none reported objective physical-function outcomes [28]. In EMBRAZE, apitegromab added to tirzepatide preserved approximately 1.9 kg more DXA-measured lean mass, but exploratory function did not clearly differ [29]. These findings illustrate why preservation of a measured compartment cannot be assumed to improve strength, exercise capacity, athletic performance, or protection from REDs. Lean-mass change becomes clinically relevant when accompanied by inadequate intake, rapid unintended loss, declining strength, impaired recovery, endocrine disturbance, injury, or worsening training response. No study has linked GLP-1-related lean-mass changes to REDs severity, bone stress injury, cardiorespiratory fitness, or sport performance.

A reduction in a lean-mass index is therefore not sufficient to diagnose REDs or infer clinically meaningful muscle loss. Interpretation should integrate the rate and purpose of weight loss, dietary intake, gastrointestinal tolerance, hydration, training load, strength or functional change, recovery, endocrine and reproductive findings, bone health, and injury. This is especially important because the available body-composition trials were conducted largely in non-athletic populations and were not designed around sport-specific performance or REDs outcomes.

5. Exercise, Physical Function, and Performance

5.1. Exercise Co-Interventions and Muscle Preservation

Exercise and GLP-1-based pharmacotherapy are complementary rather than interchangeable. In 33 sedentary adults with type 2 diabetes, combined liraglutide and supervised aerobic/resistance exercise improved weight and glycemia more than exercise with placebo, while estimated maximum oxygen uptake increased similarly in both groups [30]. A network meta-analysis of nine trials involving 1,009 adults with overweight or obesity ranked GLP-1 therapy plus structured exercise favorably for weight and fat mass, but lean-mass effects were not adequately analyzable [31]; a broader synthesis likewise found sparse and heterogeneous evidence for lean mass, regional body composition, fitness, and function [32]. These findings support exercise for established health benefits but do not show that it prevents REDs or pharmacotherapy-associated loss of contractile tissue in athletes.

Resistance exercise is a rational strategy to support strength and function during weight loss, but it has not been shown to prevent GLP-1-associated muscle loss or REDs. The LEAN-PREP trial will test resistance exercise and protein supplementation during semaglutide or tirzepatide treatment, but it is a protocol without efficacy results and is not an athlete REDs study [33].

5.2. Function, Fitness, Performance, and Energy Availability

Self-reported function, habitual activity, exercise capacity, cardiorespiratory fitness, and athletic performance are distinct. Weight loss may improve mobility or symptoms without increasing absolute aerobic capacity, while sport performance additionally depends on technique, economy, pacing, power, recovery, and competition context. A review of 14 randomized pharmacotherapy trials found a modest improvement in self-reported function (standardized mean difference, 0.27), but none reported habitual activity and only one measured cardiorespiratory fitness objectively [34]. STRIDE showed improved maximum walking distance with semaglutide in 792 older adults with type 2 diabetes and symptomatic peripheral artery disease (treatment ratio, 1.13) [35]. This demonstrates improved disease-limited walking, not athletic endurance, and should not be generalized to healthy trained populations.

Exercise may support fitness and function but also raises energy expenditure. If appetite suppression or gastrointestinal intolerance limits compensatory intake, increasing training without nutritional adjustment could widen the energy mismatch. This interaction is plausible within the REDs model but has not been demonstrated experimentally. Interpretation should distinguish absolute from body-mass-normalized fitness because weight loss can improve relative values without increasing absolute oxygen uptake. Declining strength, inability to

complete expected training, prolonged recovery, recurrent illness or injury, or deterioration in objective performance should prompt broader assessment rather than automatic attribution to medication or training alone.

6. Sport-Specific Vulnerability

Potential vulnerability is context dependent. A patient with obesity receiving supervised treatment differs from a lean athlete using medication to reach a lower weight category or alter appearance. No sport or demographic characteristic is a validated predictor of GLP-1-associated problematic LEA or REDs. Higher-concern contexts are those in which appetite suppression or rapid weight loss overlaps with chronic restriction, repeated weight cycling, high training expenditure, adolescent growth, previous REDs indicators, disordered eating, or limited medical and nutritional supervision.

6.1. Weight-Category, Combat, Aesthetic, and Physique Sports

Weight-category athletes may combine chronic restriction with acute weight-making, and possible GLP-1 use for this purpose remains an unresolved issue [6]. Appetite suppression does not ensure adequate carbohydrate, hydration, recovery nutrition, or lean-tissue preservation, and gastrointestinal symptoms could complicate post-weigh-in refueling. Female weight-category athletes also require attention to reproductive and bone health, although existing guidance does not evaluate pharmacotherapy [36].

In aesthetic and physique sports, pressure for low body fat may promote restrictive eating despite the absence of a universal body-fat threshold for performance [37]. Online bodybuilding discussions describe off-label GLP-1 use, cycling, stacking, illicit sourcing, and attempts to offset perceived muscle loss [5]. These data establish an emerging context but cannot estimate prevalence or determine health and performance outcomes.

6.2. Endurance and Adolescent Athletes

Endurance athletes may have high, variable energy expenditure and may underfuel unintentionally. In 50 competitive male cyclists, low-EA classification was associated with lower lumbar spine bone mineral density, and chronic low-EA classification with lower testosterone and underperformance relative to training load [38]. No study has examined free-living energy availability, glycogen restoration, bone stress injury, or endurance performance during GLP-1 treatment.

Adolescents require additional caution because training coincides with growth, maturation, and bone accrual. In 13 male adolescent endurance athletes, fitness and strength improved despite poor baseline bone health or failure to achieve expected bone accrual [39], illustrating that performance gains do not guarantee physiological adequacy. Direct evidence on growth, puberty, reproductive maturation, nutrition, and sport performance in young GLP-1 users is absent.

6.3. Previous REDs Indicators, Eating Concerns, and Supervision

Previous reproductive dysfunction, low bone mineral density, bone stress injury, endocrine suppression, recurrent restrictive dieting, or unexplained deterioration in training response warrants careful assessment before further weight loss. These findings are not specific to REDs and require differential diagnosis. Relationships between problematic LEA, disordered eating, mood, fatigue, and exercise-related conflict may be bidirectional [40]. GLP-1 therapy has not been shown to cause eating disorders in athletes, but appetite suppression, rapid weight change, or fear of weight regain could reinforce restriction in vulnerable individuals and complicate treatment decisions.

Benefit-risk assessment depends strongly on indication. Medically supervised treatment for obesity, type 2 diabetes, or obesity-related functional limitation may improve health and participation. Unsupervised use, illicit sourcing, self-directed dose changes, and polysubstance use remove clinical safeguards and create additional uncertainty about product identity, dose, interactions, and adverse effects.

These contexts should guide monitoring intensity rather than create categorical contraindications. Endurance participation, weight-category sport, adolescence, or previous REDs-related findings do not independently prove that treatment is inappropriate; they indicate that the margin for nutritional error may be smaller and that treatment goals, training demands, and recovery should be reviewed together. Direct comparative data across sports, competitive levels, and indications are not available.

7. Clinical and Sport-Practice Implications

7.1. Risk-Based Assessment

The absence of direct evidence justifies neither routine alarm nor uncritical reassurance. The following approach is pragmatic and unvalidated. Assessment should consider indication, age and growth, reproductive status, weight history, sport and competition demands, training load, intended rate and purpose of weight loss, previous REDs indicators, gastrointestinal tolerance,

eating behavior, psychological health, and medical/nutritional supervision. No isolated weight, body mass index, questionnaire, laboratory value, or body-composition result diagnoses REDs. The principal monitoring domains are summarized in Table 1.

Table 1. Proposed pragmatic monitoring framework for physically active individuals using GLP-1-based therapies.

Monitoring domain	Baseline / longitudinal assessment	Potential warning pattern	Suggested response	Required caveat
Clinical indication and prescribing context	Indication, treatment goal, dose escalation, concurrent drugs/substances, supervision, previous weight-loss attempts.	Use without a clear indication; illicit sourcing; self-directed escalation; high-risk polysubstance use.	Reassess indication, product source, interactions, treatment goal, and need for specialist input.	Medication exposure alone does not diagnose REDs or justify automatic discontinuation.
Weight trajectory, growth, and body-composition context	Recent weight history, planned rate/extent of loss, repeated weight cycling; growth and puberty in adolescents; method-consistent body composition when justified.	Loss faster or greater than intended; continued loss after goal; loss in an already lean individual; impaired growth.	Review dose, target, intake and training; consider modifying the weight-loss plan and broader clinical assessment.	No universal rate of weight loss or body fat threshold has been validated for GLP-1-treated athletes.

Monitoring domain	Baseline / longitudinal assessment	Potential warning pattern	Suggested response	Required caveat
Dietary adequacy and GI tolerance	Meal pattern, tolerated portion size, carbohydrate around exercise, protein distribution, recovery intake, fluids and electrolytes, dietary variety, and nausea, vomiting, diarrhoea, or constipation.	Repeated missed meals; inability to fuel or recover; persistent vomiting; dehydration; progressive restriction.	Dietitian and medical review; symptom management; structured eating and tolerated nutrient-dense options; urgent assessment when oral intake is compromised.	Appetite suppression or GI symptoms do not independently prove LEA.
Training load, recovery, and performance	Training volume/intensity, recent load changes, session completion, perceived exertion, recovery, sleep, illness, injury, objective sport-specific output where feasible.	Persistent decline in output or strength; prolonged recovery; recurrent illness/injury; inability to complete expected training.	Assess intake-expenditure mismatch, training load, medication effects, and alternative medical explanations; modify training when clinically indicated.	A single poor session or subjective symptom is non-specific.

Monitoring domain	Baseline / longitudinal assessment	Potential warning pattern	Suggested response	Required caveat
Body composition, muscle, and function	Consistent DXA/BIA/CT/MRI method when clinically justified, paired with strength or functional measures.	Rapid lean-index decline accompanied by reduced strength/function, fatigue, or poor recovery.	Evaluate hydration/method effects, dietary adequacy, resistance exercise, and clinical causes; track function rather than body composition alone.	Lean mass is not synonymous with contractile muscle, sarcopenia, or performance.
Reproductive, endocrine, and bone health	Menstrual/reproductive history, libido/endocrine symptoms, prior low BMD or bone stress injury; targeted labs/imaging based on presentation.	New reproductive disturbance, bone pain/stress injury, or a cluster of endocrine abnormalities.	Clinician-led differential diagnosis, REDs-oriented assessment, and treatment/training review.	Hormonal contraception may mask menstrual change; no single biomarker confirms REDs.

Monitoring domain	Baseline / longitudinal assessment	Potential warning pattern	Suggested response	Required caveat
Psychological health and eating behavior	Dietary rigidity, fear of regain, body dissatisfaction, binge-restrict cycles, compulsive exercise, mood, anxiety, concealment of medication use.	Escalating restriction, distress after eating, psychological deterioration, or conflict compromising health decisions.	Mental-health and sports-dietetic assessment; address eating pathology and sport pressures; coordinate communication.	Relationships may be bidirectional; GLP-1 therapy has not been shown to cause eating disorders in athletes.
Multidisciplinary escalation	Integrate findings across prescribing clinician, sports medicine, dietetics, mental health, and training staff with consent.	Multiple persistent indicators, an orange- or red-level CAT2-type presentation, or a severe gastrointestinal, medical, or psychological concern.	Reassess medication, weight-loss, training and competition plans; restrict participation only according to clinical severity and established principles.	The proposed framework is evidence-informed and not prospectively validated for GLP-1 users.

Abbreviations: BIA, bioelectrical impedance analysis; BMD, bone mineral density; CAT2, Clinical Assessment Tool version 2; CT, computed tomography; DXA, dual-energy X-ray absorptiometry; GI, gastrointestinal; LEA, low energy availability; MRI, magnetic resonance imaging; REDs, Relative Energy Deficiency in Sport.

Before or early in treatment, clinicians should document treatment goals, dose escalation, concurrent medication or substance use, recent or repeated weight loss, and the relationship between body mass and sport participation. In adolescents, longitudinal growth and pubertal development are more informative than an isolated weight. A REDs-oriented history should

address reproductive or endocrine symptoms, bone health, injury, fatigue, recovery, recurrent illness, gastrointestinal symptoms, sleep, mood, restrictive eating, and previous problematic LEA. Body composition is not required routinely; when clinically justified, it should be measured with a consistent method under standardized conditions and interpreted with intake, strength, function, and clinical findings rather than used to impose arbitrary body-fat targets. The IOC REDs CAT2 may structure clinician-led assessment when findings cluster, but it has not been validated in medication-treated populations [13]. LEAF-Q may assist screening in selected female athletes; its original threshold of at least 8 showed 78% sensitivity and 90% specificity, but these estimates should not be generalized across sexes, ages, sports, hormonal states, para athletes, or GLP-1 users [14]. Male-specific questionnaire screening is less established, so clinical history, examination, differential diagnosis, and contextual interpretation remain central [12,13].

7.2. Longitudinal Monitoring and Coordination

Monitoring should emphasize change from baseline and intensify during dose escalation, rapid weight change, increased training, competition preparation, or recurrent gastrointestinal symptoms. Key domains include the rate and purpose of weight loss; meal pattern, appetite, and tolerance; carbohydrate around demanding sessions; protein distribution and recovery nutrition; hydration and dietary variety; training volume and intensity; ability to complete planned work; perceived exertion at a familiar workload; recovery, strength, and sport-specific output; injury and illness; sleep and fatigue; reproductive and bone symptoms; and psychological response. Objective measures should be used where feasible, but an isolated decline does not diagnose REDs. Laboratory testing should follow the clinical presentation and differential diagnosis rather than a universal REDs panel, because normal results do not exclude problematic LEA and abnormal values may have alternative causes.

During appetite suppression, structured eating may be more reliable than hunger alone. Smaller, frequent, well-tolerated energy-dense meals and prioritization of carbohydrate, protein, fluids, and electrolytes around key sessions may be practical, but they have not been shown to prevent REDs in GLP-1-treated athletes. Resistance exercise may support function but cannot compensate for inadequate energy intake. Medication, nutrition, training, and recovery should therefore be coordinated, particularly when gastrointestinal symptoms or competition schedules limit food intake.

Psychological monitoring should also consider increasing dietary rigidity, fear of weight regain, body dissatisfaction, concealment of medication use, compulsive exercise, or conflict between

medical and sport goals. These findings should not be attributed automatically to treatment, but they may alter intake, adherence, training decisions, and recovery. Reproductive changes, bone pain, suspected bone stress injury, or persistent endocrine symptoms require clinical evaluation rather than reliance on screening scores alone.

7.3. Reassessment and Multidisciplinary Care

No validated GLP-1-specific threshold mandates stopping medication, training, or weight loss. Reassessment is warranted when concerns accumulate or persist, including unintended or excessive loss, inability to maintain food or fluid intake, recurrent vomiting, progressive restriction or disordered eating, reproductive disturbance, low bone density or bone stress injury, declining strength or objective performance, prolonged fatigue, recurrent illness, psychological deterioration, concerning clinical abnormalities, unsupervised dose changes, illicit sourcing, or high-risk polysubstance use.

Reassessment should revisit treatment indication and goals, dose and tolerability, nutritional intake, training and competition demands, psychological factors, and alternative medical explanations. Depending on the pattern and severity of findings, the weight-loss target, dose-escalation schedule, training plan, competition strategy, or dietary support may require modification. Pharmacotherapy decisions remain with the treating clinician and should balance demonstrated treatment benefits against emerging concerns. Care may require coordination among the prescriber, a clinician experienced in REDs, a sports dietitian, mental health professionals, and exercise or rehabilitation specialists. Communication should avoid stigmatizing medically indicated obesity treatment while not normalizing unsupervised pharmacological weight loss in already lean athletes.

8. Research Priorities

Prospective studies should recruit physically active individuals before or at treatment initiation and follow them through dose escalation, maintenance, intensified training or competition, and discontinuation where feasible. Cohorts should distinguish recreationally active patients treated for a clinical indication, competitive athletes with overweight or obesity, lean athletes seeking additional loss, weight-category or aesthetic-sport participants, adolescents, older adults, and supervised versus unsupervised use. Baseline assessment before substantial appetite suppression or weight loss is essential for interpreting subsequent change.

Research should determine whether appetite suppression produces inadequate free-living intake relative to exercise and physiological requirements. Repeated dietary assessment should be

integrated with training load, exercise expenditure, resting energy expenditure, weight trajectory, and within-day meal and training patterns rather than relying on a single calculated energy-availability value. Nutritional outcomes should include carbohydrate availability, protein intake and distribution, recovery nutrition, hydration, dietary variety, micronutrient adequacy, and the relationship between gastrointestinal symptoms and missed meals. Health outcomes should include reproductive and endocrine function, bone density and turnover, bone stress injury, hematological and gastrointestinal health, sleep, fatigue, mood, eating behavior, injury, and recovery. Body-composition measures should be method-specific and paired with strength, power, objective function, cardiorespiratory fitness, habitual activity, and sport-specific performance.

Randomized trials should evaluate resistance and aerobic exercise, protein and carbohydrate strategies, and combined interventions during treatment. Outcomes should determine whether interventions preserve health and performance rather than merely altering a lean-mass estimate. Particular priorities include weight-category, combat, physique, aesthetic, endurance, and adolescent settings; reasons and patterns of use; supervision and concurrent substances; post-weight-in recovery; and sex-specific outcomes. The pragmatic monitoring framework in Table 1 also requires prospective evaluation for feasibility, sensitivity to meaningful change, specificity in the presence of medication-related symptoms, and applicability across sports, ages, sexes, and competitive levels.

9. Limitations

The central limitation is the absence of prospective studies jointly evaluating GLP-1 exposure, energy availability, and REDs outcomes in athletes. Clinical evidence comes mainly from adults with overweight, obesity, type 2 diabetes, or disease-related functional limitation and may not generalize to competitive athletes with different adiposity, training status, dietary requirements, and treatment goals. Laboratory meals do not measure habitual intake or energy availability; gastrointestinal findings vary by agent, dose, duration, and method; and body-composition studies use heterogeneous definitions, rarely pair tissue estimates with strength or function, and do not assess REDs. Athlete studies are mostly observational and rely on surrogate indicators or self-report, while sport-specific GLP-1 exposure data are limited to qualitative reports and commentary that cannot establish prevalence or consequences.

This structured narrative review searched PubMed/MEDLINE but did not use a dual-reviewer systematic process, protocol registration, or formal risk-of-bias assessment; studies indexed elsewhere, conference material, unpublished data, or unavailable full texts may have been

missed. The corpus included different incretin therapies, doses, indications, and populations, so class-level conclusions require caution. Evidence weighting involved reviewer judgment, and sponsorship was not formally analyzed. Finally, the proposed clinical framework is unvalidated and should not be treated as a diagnostic algorithm, contraindication framework, medication-stopping rule, or substitute for individualized clinical judgment.

10. Conclusions

GLP-1-based therapies affect appetite, measured intake, satiation, gastrointestinal tolerance, weight, and lean-mass indices. In physically active individuals, these effects may make it harder to match intake to exercise demands when combined with high training volume, restrictive eating, weight-making, pressure for leanness, or previous REDs-related concerns. Lean-mass changes do not independently establish loss of contractile muscle, strength, function, or performance.

This biological and clinical plausibility is not evidence of causation. No prospective study has shown progression from GLP-1 exposure to problematic LEA and REDs-related health or performance outcomes in athletes, and most evidence comes from separate non-athletic clinical and athlete REDs literature. Current evidence supports individualized, risk-based monitoring rather than universal restriction or false reassurance. Medically indicated treatment should not be stigmatized, whereas unsupervised use, rapid loss, or further weight reduction in already lean athletes warrants particular caution. GLP-1-based therapy should therefore be viewed as a potential modifying factor within the REDs framework pending athlete-specific prospective research

Disclosure

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