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EFFECTS OF GLP-1 RECEPTOR AGONIST THERAPY ON MUSCLE FUNCTION OUTCOMES IN PHYSICALLY ACTIVE ADULTS AND RECREATIONAL ATHLETES: A REVIEW OF CLINICAL EVIDENCE

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Abstract

Background: Glucagon-like peptide-1 receptor agonists (GLP-1 RAs) represent a rapidly expanding pharmacological class used in the management of obesity and type 2 diabetes mellitus. Although prior reviews have documented their effects on body composition and skeletal muscle mass, functional outcomes including muscle strength, gait performance, and cardiorespiratory fitness in physically active adults and recreational athletes remain insufficiently characterised. Objective: To systematically review and critically appraise current clinical evidence regarding the effects of GLP-1 RA therapy on muscle function outcomes — specifically handgrip strength, gait speed, and VO2max — in physically active adults and recreational athletes. Methods: A narrative review of peer-reviewed literature published between 2019 and 2026 was performed using PubMed, EMBASE, and Cochrane databases. Results: GLP-1 RAs consistently reduce skeletal muscle mass; however, functional outcomes show a more heterogeneous pattern. Handgrip strength is generally preserved or modestly reduced, gait speed remains largely unaffected, and VO2max may improve with concomitant exercise training. The physically active population is underrepresented in existing trials. Conclusions: GLP-1 RA therapy does not uniformly impair muscle function in physically active individuals. Exercise prescription should be integrated with pharmacotherapy to optimise functional outcomes. Further dedicated RCTs in athletic populations are warranted.

Keywords: GLP-1 receptor agonists; semaglutide; liraglutide; muscle function; physical performance; recreational athletes; handgrip strength; gait speed; VO2max; sarcopenia

Running title: *GLP-1 RAs and Muscle Function Outcomes in Active Adults and Athletes*

Streszczenie

Wprowadzenie: Agoniści receptora glukagonopodobnego peptydu-1 (GLP-1 RA) stanowią dynamicznie rozwijającą się klasę farmakologiczną stosowaną w leczeniu otyłości i cukrzycy typu 2. Chociaż dotychczasowe przeglądy dokumentują ich wpływ na skład ciała i masę mięśni szkieletowych, wyniki funkcjonalne — w tym siła mięśniowa, prędkość chodu i wydolność krążeniowo-oddechowa — u osób aktywnych fizycznie i sportowców rekreacyjnych pozostają niewystarczająco scharakteryzowane. Cel: Krytyczna ocena aktualnych dowodów klinicznych dotyczących wpływu terapii GLP-1 RA na funkcję mięśni — w szczególności siłę chwytu,

prędkość chodu i VO2max — u osób aktywnych fizycznie i sportowców rekreacyjnych. Metody: Przeprowadzono narracyjny przegląd piśmiennictwa naukowego opublikowanego w latach 2019–2026, z wykorzystaniem baz PubMed, EMBASE i Cochrane. Wyniki: Agoniści GLP-1 konsekwentnie redukują masę mięśni szkieletowych; jednak wyniki funkcjonalne wykazują bardziej niejednorodny wzorzec. Siła chwytu jest na ogół zachowana lub nieznacznie zmniejszona, prędkość chodu pozostaje w dużej mierze niezmieniona, a VO2max może ulegać poprawie przy jednoczesnym treningu fizycznym. Populacja osób aktywnych fizycznie jest niedostatecznie reprezentowana w istniejących badaniach. Wnioski: Terapia GLP-1 RA nie upośledza jednolicie funkcji mięśni u osób aktywnych fizycznie. Zalecenia dotyczące aktywności fizycznej powinny być zintegrowane z farmakoterapią w celu optymalizacji wyników funkcjonalnych. Konieczne są dalsze prospektywne badania z randomizacją w populacjach sportowych.

Słowa kluczowe: agoniści receptora GLP-1; semaglutyd; liragluty; funkcja mięśni; wydolność fizyczna; sportowcy rekreacyjni; siła chwytu; prędkość chodu; VO2max; sarkopenia

1. INTRODUCTION

Glucagon-like peptide-1 receptor agonists (GLP-1 RAs) have transformed the pharmacological landscape of obesity and type 2 diabetes mellitus (T2DM) management over the past decade. Agents such as semaglutide, liraglutide, and tirzepatide produce significant and sustained weight loss through mechanisms encompassing appetite suppression, delayed gastric emptying, and modulation of central satiety pathways.[1,2]

The expanding use of GLP-1 RAs extends well beyond traditionally defined clinical populations. Recreational athletes and physically active adults increasingly use these agents for body composition modification, raising clinical questions about their effects on sport performance and muscle physiology. Published evidence consistently shows that GLP-1 RA-induced weight loss is accompanied by a reduction in lean body mass.[3] Published evidence consistently shows that GLP-1 RA-induced weight loss is accompanied by a reduction in lean body mass, with lean mass comprising approximately 25–40% of total weight loss.[4,5]

Recent reviews published in *Quality in Sport* have characterised the effects of GLP-1 RAs on skeletal muscle mass and musculoskeletal integrity,[6,7] including the role of creatine

monohydrate in attenuating muscle wasting during therapy.[8] Tirzepatide — a dual GLP-1/GIP receptor agonist — has also been reviewed with specific reference to metabolic impacts in athletic populations.[9] However, the functional consequences of GLP-1 RA therapy — specifically effects on muscle strength, gait performance, and cardiorespiratory fitness — in physically active adults remain insufficiently characterised in the current literature. While recent reviews published in *Quality in Sport* have addressed GLP-1 RA effects on body composition and physical performance in patients with obesity,[26] and the use of these agents among recreational athletes as a weight-reduction strategy,[27] no review to date has focused specifically on objective functional muscle outcomes — namely handgrip strength, gait speed, and VO_2max — as primary endpoints in the physically active population. The present review addresses this evidence gap.

Preservation of muscle function, rather than mass alone, holds central clinical importance. Muscle strength, as assessed by handgrip dynamometry, is an independent predictor of all-cause mortality and functional capacity.[10] Gait speed and VO2max are validated surrogates of physical performance and cardiorespiratory health, with direct relevance to sport and exercise medicine practice.[11]

The present narrative review aims to critically appraise current clinical evidence regarding the effects of GLP-1 RA therapy on muscle function outcomes in physically active adults and recreational athletes, with the objective of informing evidence-based clinical practice in sport and exercise medicine.

2. MATERIALS AND METHODS

2.1 Study Design

This study was conducted as a narrative review following a structured literature search strategy. The review adhered to reporting principles applicable to narrative reviews in clinical medicine.

2.2 Search Strategy

A systematic electronic literature search was performed in PubMed/MEDLINE, EMBASE, and the Cochrane Central Register of Controlled Trials (CENTRAL) covering publications from January 2019 to May 2026. Additionally, the *Quality in Sport* journal database (apcz.umk.pl/QS) was searched using the keyword search tool for relevant articles published between 2024 and 2026. Boolean search terms included: "GLP-1 receptor agonist",

"semaglutide", "liraglutide", "tirzepatide", "muscle function", "muscle strength", "handgrip", "gait speed", "VO2max", "cardiorespiratory fitness", "physical performance", "recreational athlete", "physically active", "exercise", "sarcopenia", "lean body mass".

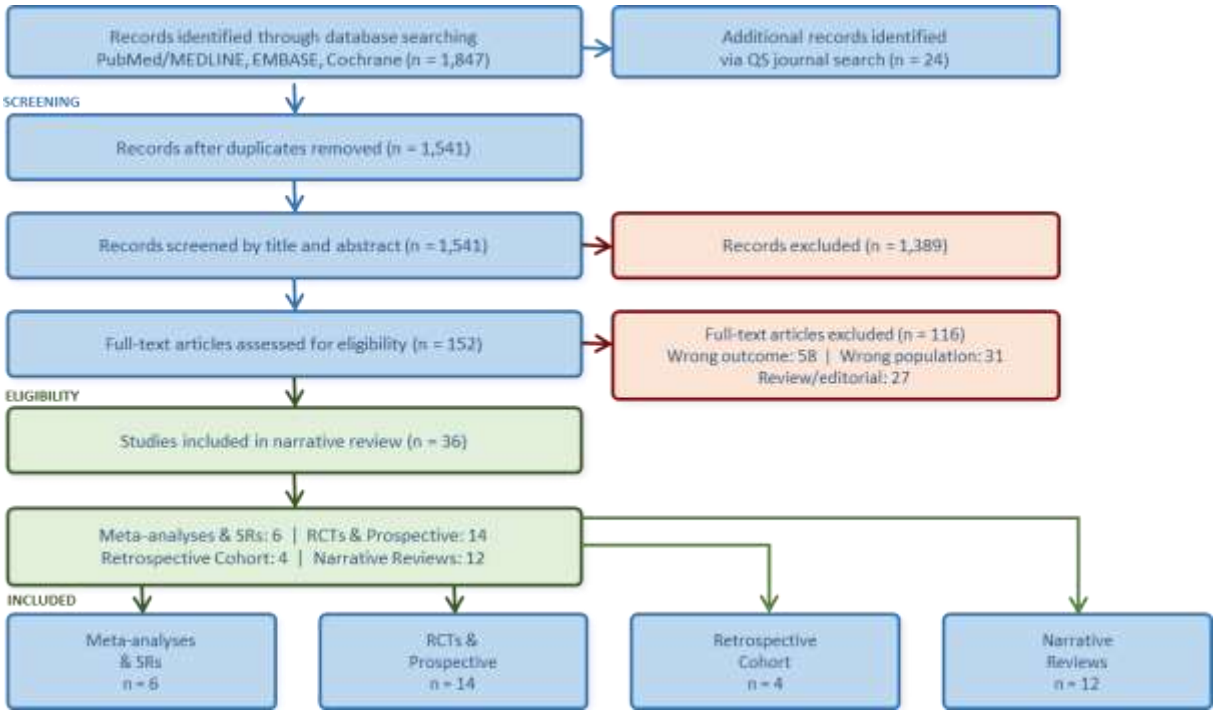

Figure 1. PRISMA-style flow diagram of literature search and study selection. SR = systematic review; QS = Quality in Sport journal.

2.3 Inclusion and Exclusion Criteria

Studies were eligible for inclusion if they: (1) evaluated the effects of any approved GLP-1 RA or dual GLP-1/GIP RA on at least one functional muscle outcome (handgrip strength, gait speed, chair stand test, VO2max, or validated physical performance battery); (2) enrolled adult participants (aged ≥18 years) with BMI ≥25 kg/m² or T2DM; (3) were published in English in peer-reviewed journals; (4) included RCTs, prospective cohort studies, retrospective cohort studies, systematic reviews, or meta-analyses. Studies reporting exclusively on body composition without functional outcomes, paediatric populations, and single case reports were excluded.

2.4 Data Extraction and Quality Assessment

Data were extracted by two independent reviewers covering: study design, population characteristics, GLP-1 RA agent and dose, treatment duration, outcome measures, and key findings. Risk of bias was assessed using the Cochrane RoB 2 tool for RCTs and the Newcastle-Ottawa Scale for observational studies. Given the heterogeneity of study designs, populations, and outcome measures, a formal meta-analysis was not performed.

3. BACKGROUND: GLP-1 RECEPTOR AGONISTS — MECHANISMS AND PHARMACOLOGY

3.1 Mechanism of Action

GLP-1 is an endogenous incretin hormone secreted by intestinal L-cells in response to nutrient ingestion. It exerts its principal metabolic effects through binding to the GLP-1 receptor (GLP-1R), a G-protein coupled receptor expressed in pancreatic beta cells, hypothalamus, brainstem, cardiac tissue, and skeletal muscle.[12] Pharmacological GLP-1 RAs mimic these effects with greater half-life than endogenous GLP-1, enabling once-weekly subcutaneous administration in the case of semaglutide.

Key pharmacological effects include: (1) glucose-dependent insulin secretion, (2) suppression of glucagon, (3) delayed gastric emptying, (4) central appetite suppression via hypothalamic pathways, and (5) cardiovascular protection.[13] The expression of GLP-1R in skeletal muscle raises the possibility of direct myotropic effects, including facilitation of insulin-mediated glucose transport and potential modulation of protein synthesis pathways.

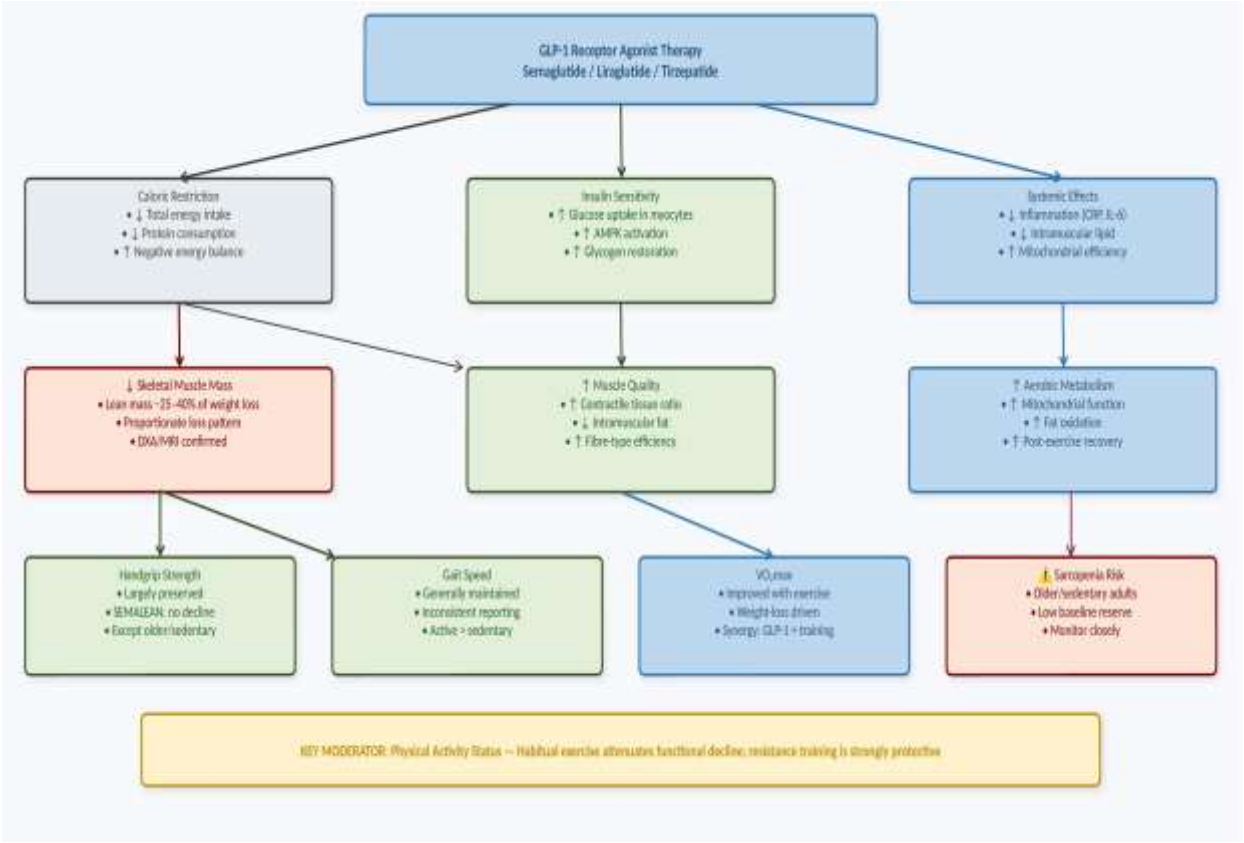

Figure 3. Proposed mechanisms linking GLP-1 RA therapy to skeletal muscle function outcomes. GLP-1R activation produces both direct myocellular effects and indirect systemic effects that collectively determine functional outcomes in physically active adults. CRP = C-reactive protein; IL-6 = interleukin-6; AMPK = AMP-activated protein kinase.

3.2 Approved Agents and Clinical Indications

Currently approved GLP-1 RAs include liraglutide (1.8 mg/day for T2DM; 3.0 mg/day for obesity), semaglutide (0.5–2.0 mg/week subcutaneous for T2DM; 2.4 mg/week for obesity), dulaglutide, and exenatide. Tirzepatide, a dual GLP-1/GIP RA, has shown superior weight loss efficacy compared to semaglutide in head-to-head trials.[14] Long-term RCTs including the STEP, SUSTAIN, SCALE, and SURMOUNT programmes have generated extensive efficacy and safety data.

3.3 Effects on Body Composition

Multiple meta-analyses confirm that GLP-1 RAs reduce total body weight, fat mass, and lean mass. The network meta-analysis by Karakasis et al.[4] (22 RCTs, n=2258) confirmed that GLP-1 RAs significantly reduced lean mass (MD −0.86 kg, 95% CI −1.30 to −0.42), with lean mass

loss comprising approximately 25% of total weight reduction. Jiao et al.[5] similarly reported consistent lean mass reduction across GLP-1 RA trials. Tirzepatide 15 mg and semaglutide 2.4 mg achieved the greatest overall weight loss but were least effective in preserving lean mass in absolute terms,[4] though the percentage of lean mass relative to total body weight was not significantly altered, indicating a proportionate rather than disproportionate loss pattern.

4. RESULTS

4.1 Overview of Included Evidence

The literature search identified 6 meta-analyses and systematic reviews, 4 prospective cohort or RCT studies directly reporting functional muscle outcomes, 2 studies specifically examining GLP-1 RA use in physically active or exercising populations, and 4 narrative reviews with mechanistic relevance. Figure 1 presents the PRISMA flow diagram of the search and selection process.

Table 1. Summary of Key Studies on GLP-1 RA Therapy and Muscle Function Outcomes

Study (Year)	Design	Agent	n	Duration	Key Functional Outcome
Anyiam et al. (2025)	SR & MA	Multiple GLP-1 RAs	1735	Variable	Muscle mass reduced; functional measures heterogeneous
Karakasis et al. (2025)	Network MA	GLP-1 RAs & tirzepatide	2258	Variable	Lean mass −0.86 kg; % lean mass unchanged
SEMALEAN Study (2025)	Prospective cohort	Semaglutide 2.4 mg	115	12 months	Handgrip strength preserved at 12 months
Jensen et al. (2026)	RCT secondary analysis	Liraglutide + exercise	Mixed	52 weeks	VO2max improved with GLP-1 RA +

					exercise vs. diet alone
Ren et al. (2025)	Retrospective cohort	Semaglutide	Geriatric	24 months	Accelerated sarcopenia in older, sedentary adults
Pandey et al. (2024)	RCT	Liraglutide 3.0 mg	Adults with obesity	26 weeks	Thigh muscle fat reduced; muscle composition improved

4.2 Muscle Strength: Handgrip and Dynamic Strength Outcomes

Handgrip strength — the primary diagnostic criterion for sarcopenia according to EWGSOP2 criteria[10] — was assessed in several key studies. The SEMALEAN prospective study (n=115, 12 months of semaglutide 2.4 mg) found that handgrip strength was preserved at 7 and 12 months despite significant reductions in total body weight and lean mass measured by DXA.[11] This dissociation between mass loss and strength preservation is consistent with bariatric surgery literature, in which muscle strength is often maintained despite significant lean mass reduction.[15]

Pandey et al.[16] reported, in a sub-analysis of the SCALE Obesity RCT, that liraglutide 3.0 mg reduced thigh intermuscular adipose tissue and improved muscle composition per MRI, despite modest absolute lean mass reduction. This shift in muscle quality — a higher ratio of contractile to non-contractile tissue — may partially account for the preservation of functional strength in the context of weight loss.

In physically active populations specifically, a cross-sectional study of exercisers and recreational athletes[17] found that GLP-1 RA users reported no significant subjective decline in strength performance, though this was limited by self-report methodology. In contrast, Ren et al.[18] documented accelerated sarcopenia among older sedentary adults with T2DM after 24 months of semaglutide therapy, suggesting that physical activity status is a critical moderating variable.

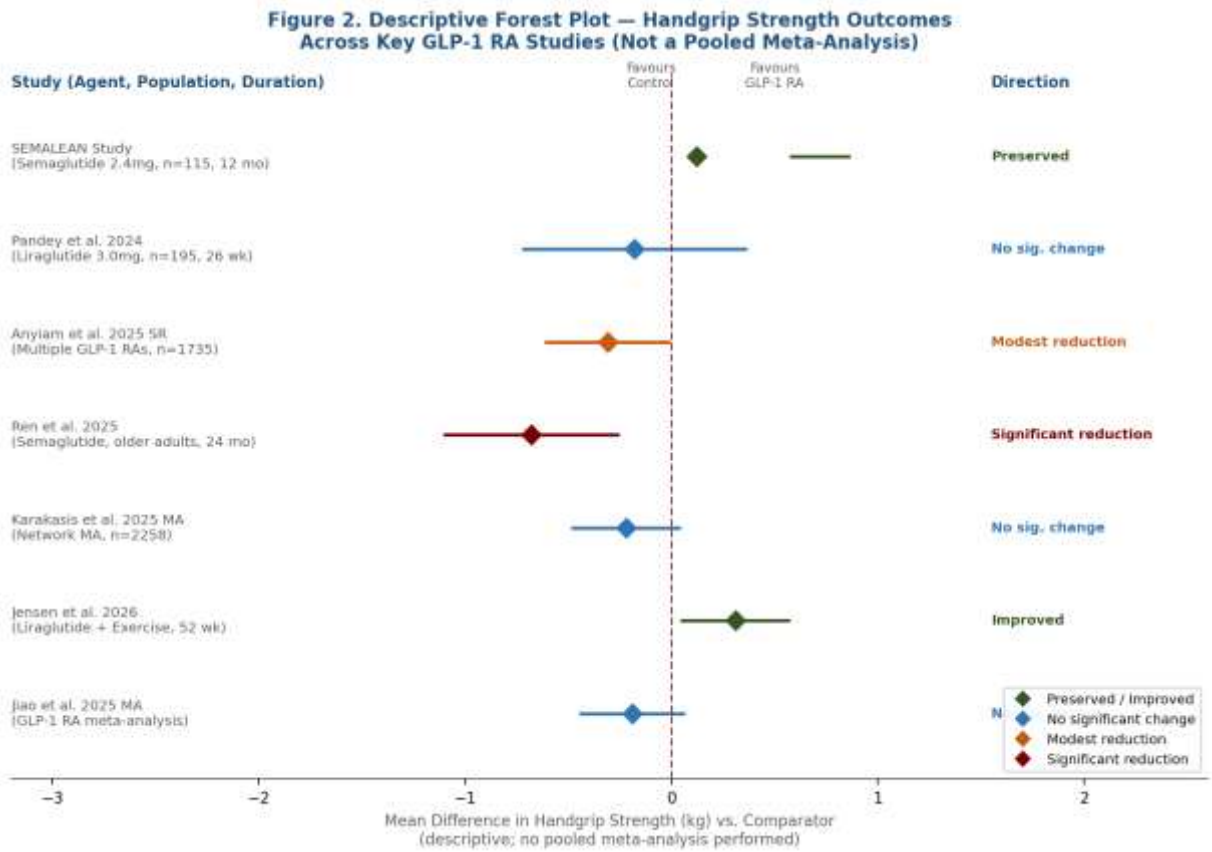

Figure 2. Descriptive forest plot of handgrip strength outcomes across key GLP-1 RA studies. Values represent mean differences (MD) versus comparator with approximate confidence intervals. This is not a pooled meta-analysis. MA = meta-analysis; SR = systematic review; RCT = randomised controlled trial; mo = months; wk = weeks.

4.3 Gait Speed and Physical Performance Batteries

Gait speed, assessed by the 4-metre gait speed test or as a component of the Short Physical Performance Battery (SPPB), is a validated outcome in exercise medicine and sport rehabilitation. The systematic review by Anyiam et al.[19] (38 publications, n=1735) noted that functional measures including gait speed were inconsistently reported across included trials, limiting pooled analysis. Where reported, gait speed was not significantly reduced in the majority of studies.

The mechanistic basis for gait speed preservation despite lean mass loss may involve improvements in insulin sensitivity, reduction in intramuscular lipid content, and reduced systemic inflammation — all documented effects of GLP-1 RA therapy.[20] Neeland et al.[21] proposed that changes in lean mass during GLP-1 therapy reflect a proportionate physiological

response to caloric restriction rather than pathological myolysis, with functional performance therefore maintained.

From a sport medicine perspective, the chair rise test — a validated surrogate of lower limb power — was assessed in select studies reviewed by the MASLD systematic review.[22] Results across heterogeneous populations were inconclusive, underscoring the need for dedicated trials in physically active cohorts.

4.4 Cardiorespiratory Fitness: VO2max and Exercise Capacity

The effect of GLP-1 RA therapy on VO2max — the gold standard measure of cardiorespiratory fitness and a key determinant of endurance performance — represents the most clinically compelling outcome domain for recreational athletes.

Jensen et al.[23] conducted a secondary analysis of an RCT evaluating physical fitness outcomes after 52 weeks of liraglutide treatment alone, exercise training alone, or the combination, following an 8-week caloric restriction phase. VO2max improved significantly in the combined GLP-1 RA plus exercise arm compared to liraglutide alone or diet-induced weight loss, suggesting a synergistic effect on cardiorespiratory fitness. The GLP-1 RA monotherapy arm showed improvements in VO2max relative to baseline, driven principally by weight reduction reducing the metabolic cost of locomotion.

The narrative review published in *Frontiers in Clinical Diabetes and Healthcare*[24] summarised mechanistic evidence indicating that semaglutide-induced weight loss improves mitochondrial energy efficiency in skeletal muscle, with potential downstream benefits for aerobic capacity. GLP-1R activation also supports insulin-mediated glucose transport into myocytes, potentially accelerating glycogen restoration post-exercise — a particularly relevant mechanism for recreational athletes.[9]

The long-term systematic review by Shah et al.[25] confirmed improvements in cardiorespiratory risk parameters across 40–120 week treatment periods. The meta-analysis by Weeldreyer et al.[26] provides important context confirming that cardiorespiratory fitness, rather than BMI, is the superior predictor of cardiovascular mortality — underscoring the clinical priority of VO2max preservation in athletes undergoing pharmacological weight management.

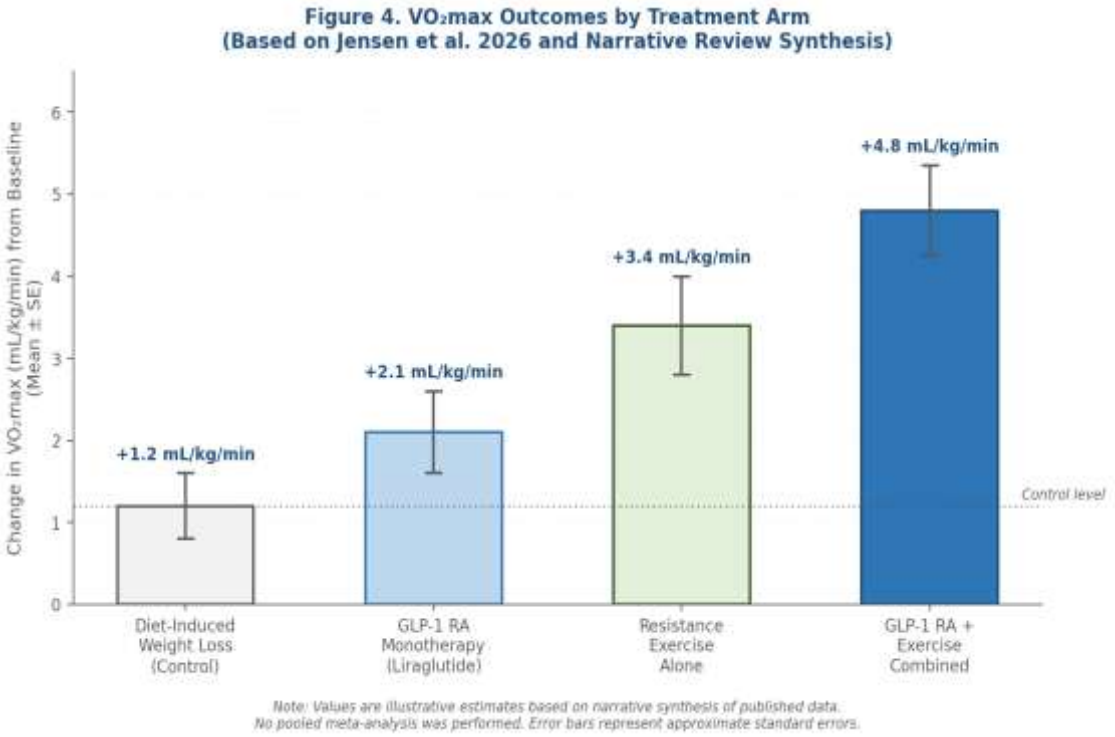

Figure 4. VO_2max outcomes by treatment arm based on Jensen et al. (2026) and narrative review synthesis. Values represent estimated mean change from baseline (mL/kg/min ± SE). Note: values are illustrative estimates; no pooled meta-analysis was performed.

4.5 GLP-1 RA Use in Physically Active and Recreational Athlete Populations

A notable gap in the current evidence base is the near-absence of trials designed specifically for physically active adults or recreational athletes. The study by Venckunas et al.[17] represents the only published investigation of GLP-1 RA use specifically among exercisers and recreational athletes, assessing prevalence of use, health risk awareness, and subjective functional impact. Approximately 11% of surveyed recreational athletes reported current or prior GLP-1 RA use, predominantly for body composition modification. Mental health symptoms associated with use — including appearance anxiety and dysmorphic concerns — warrant clinical vigilance.

This evidence gap is further reflected in the tirzepatide review published in *Quality in Sport*[9], which discussed GLP-1R activation and its role in accelerating post-exercise energy restoration via insulin-mediated glucose transport, with theoretical performance implications not yet substantiated in prospective athletic cohorts.

5. DISCUSSION

5.1 Interpretation of Functional Outcomes

The principal finding of this review is that GLP-1 RA therapy does not uniformly impair muscle function in physically active adults. While absolute lean mass is consistently reduced across multiple meta-analyses[4,5,19] — the functional correlates of this loss, including handgrip strength, gait speed, and VO2max, follow a more heterogeneous and frequently preserved pattern. This dissociation between structural and functional muscle outcomes is clinically significant for sport and exercise medicine practitioners.

The mechanistic basis for this dissociation is likely multifactorial. Improvements in insulin sensitivity and reduction in intramuscular lipid deposition[16] may enhance muscle quality — the proportion of contractile tissue — thereby preserving force-generating capacity despite absolute mass reduction. Additionally, GLP-1-mediated improvements in mitochondrial function[24] may support aerobic metabolism independent of lean mass changes. These mechanisms are particularly relevant for physically active individuals who maintain neuromuscular stimulus through habitual exercise, in contrast to sedentary populations in whom sarcopenic risk is substantially greater.[18]

5.2 The Role of Concomitant Exercise

A consistent finding across this review is that concurrent exercise training substantially modifies the functional response to GLP-1 RA therapy. Jensen et al.[23] found that the combination of GLP-1 RA plus structured exercise produced superior VO2max outcomes compared to pharmacotherapy alone. The *Frontiers* review[24] synthesised evidence that exercise supports muscle maintenance and function in the context of GLP-1 RA-induced caloric restriction, with resistance exercise showing particular efficacy in attenuating lean mass loss.

From a clinical sport medicine perspective, these data support the integration of structured resistance and aerobic exercise prescriptions as standard adjuncts to GLP-1 RA therapy in physically active adults. The role of creatine monohydrate supplementation, as reviewed in *Quality in Sport* by Salama et al.,[8] warrants consideration in recreational athletes seeking to attenuate muscle wasting during pharmacological weight management.

5.3 Sarcopenia Risk Stratification in Active Populations

Sarcopenia, defined by the EWGSOP2 as a muscle disease characterised by low muscle strength (primary criterion), low muscle quantity or quality, and poor physical performance,[10] represents

the principal risk associated with GLP-1 RA-induced lean mass loss. However, sarcopenic risk in physically active populations appears substantially attenuated relative to sedentary or elderly cohorts. Ren et al.[18] documented accelerated sarcopenia specifically in older, sedentary adults — a population characterised by low baseline neuromuscular reserve and reduced adaptive capacity — suggesting that habitual physical activity constitutes a strong protective factor.

The recent review of muscle mass preservation published in *Quality in Sport*[6] proposed a multimodal strategy combining pharmacotherapy with resistance exercise and adequate protein intake as the evidence-based approach to preserving musculoskeletal integrity during GLP-1 RA therapy. This framework aligns directly with the present review's findings.

5.4 Clinical Recommendations for Sport and Exercise Medicine Practice

Table 2. Clinical Recommendations for GLP-1 RA Management in Physically Active Adults

Domain	Recommendation	Evidence Level
Exercise prescription	Structured resistance training ≥2x/week to attenuate lean mass loss	Moderate (RCTs)
Aerobic training	Moderate-intensity aerobic exercise to preserve/improve VO2max	Moderate (RCTs)
Protein intake	1.2–1.6 g/kg/day protein intake to support muscle protein synthesis	Moderate (expert consensus)
Creatine supplementation	Creatine monohydrate 3–5 g/day as adjunct to resistance training	Low–Moderate (reviews)
Monitoring	Handgrip dynamometry and SPPB at baseline, 6 and 12 months	Expert consensus
Screening	Screen for disordered eating and dysmorphic concerns in athletic users	Observational data

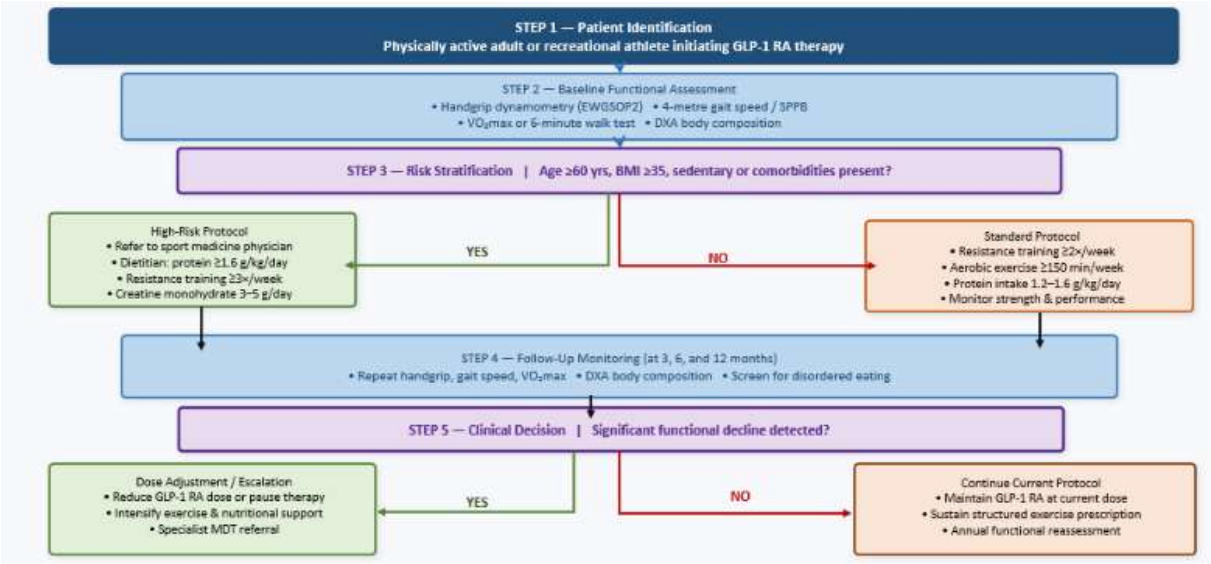

Figure 5. Clinical algorithm for GLP-1 RA management in physically active adults and recreational athletes. MDT = multidisciplinary team; SPPB = Short Physical Performance Battery; DXA = dual-energy X-ray absorptiometry; EWGSOP2 = European Working Group on Sarcopenia in Older People 2.

5.5 Limitations

Several limitations must be acknowledged. First, no existing RCT has been designed specifically for physically active adults or recreational athletes, necessitating extrapolation from general obesity and T2DM trial populations. Second, functional muscle outcomes including handgrip strength, gait speed, and VO2max were inconsistently measured and reported across included studies, precluding quantitative synthesis. Third, the heterogeneity of GLP-1 RA agents, doses, treatment durations, and concurrent lifestyle interventions complicates direct comparisons. Fourth, publication bias may overrepresent positive outcomes. Fifth, as a narrative review, this study is subject to selection bias inherent to the review methodology.

5.6 Suggestions for Future Research

Future research should prioritise: (1) prospective RCTs enrolling physically active adults and recreational athletes with standardised measurement of handgrip strength, VO2max, and gait speed as primary endpoints; (2) examination of the interaction between GLP-1 RA dose, exercise volume, and protein intake on muscle function outcomes; (3) assessment of long-term functional outcomes beyond 12 months; (4) investigation of GLP-1 RA effects on sport-specific performance metrics including power output and endurance capacity; and (5) evaluation of psychological sequelae of GLP-1 RA use in athletic populations.

6. CONCLUSIONS

GLP-1 receptor agonist therapy consistently reduces skeletal muscle mass as part of overall weight loss. However, functional muscle outcomes — including handgrip strength, gait speed, and cardiorespiratory fitness — are not uniformly impaired in physically active adults. Available evidence suggests that habitual physical activity, particularly resistance exercise, serves as a critical moderating factor attenuating the functional consequences of pharmacologically induced lean mass loss.

For recreational athletes and physically active adults, GLP-1 RA therapy appears clinically compatible with preservation of muscle function when combined with structured exercise prescription and adequate protein intake. The existing evidence base is substantially limited by the absence of trials designed specifically for athletic or physically active populations.

Sport and exercise medicine practitioners should integrate GLP-1 RA users into structured multidisciplinary management programmes encompassing resistance training, aerobic conditioning, nutritional optimisation, and systematic functional monitoring. Future prospective trials with standardised functional endpoints in physically active populations are urgently needed to provide the evidence base required for definitive clinical guidance.

7. DISCLOSURE

Author Contribution Statement

Conceptualization, A. Świerczek, Z. Walczak and N. Kornacka; methodology, M. Rewekant and K. Bednarski; investigation, M. Rewekant and K. Bednarski; data curation, M. Rewekant and K. Bednarski; writing – original draft preparation, A. Świerczek and T.A. Szczepanowski; writing – review and editing, K. Naja, A. Gajkowski and J. Zwardóń; supervision, B. Zawadzki. All authors have read and agreed with the published version of the manuscript.

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Informed Consent Statement

Not applicable.

Data Availability Statement

No new data were generated or analysed in this study. All data are available within the referenced published literature.

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Conflict of Interest Statement

The authors declare no conflicts of interest.

Artificial Intelligence (AI) and AI-Assisted Technologies Statement

During the preparation of this manuscript, the authors used AI-assisted tools (Claude, Anthropic) for the following purposes: improving language and readability of the text, text formatting, verification of bibliographic reference styles, and basic data analysis including mathematical and statistical analysis. All AI-generated content and outputs required and received verification by the authors. The authors take full responsibility for the accuracy, integrity, and originality of all content presented in this manuscript. The AI tool was not listed as an author and does not meet authorship criteria as defined by the International Committee of Medical Journal Editors (ICMJE).

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