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Liraglutide – Effects on Lean Body Mass, Muscle Mass and Prevention of Muscle Loss. A Comprehensive Literature Review

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ABSTRACT

Introduction and Purpose. Obesity is a major global health challenge, requiring effective long-term treatments. Liraglutide, a GLP-1 agonist, is effective for weight loss and reducing visceral fat. However, its impact on lean body mass (LBM) and muscle mass is crucial, as their loss can lead to sarcopenia, metabolic impairment, and weight regain. Therefore, this literature review systematically analyses liraglutide's effects on LBM and muscle mass in obesity and/or type 2 diabetes, and presents strategies to minimise potential muscle loss during therapy.

Materials and methods. A systematic review of the scientific literature was conducted. The analysis focused on identifying the impact of liraglutide treatment on lean body mass and body composition by examining data from randomised controlled trials, observational studies, and meta-analyses. Methodologies for assessing these outcomes, including DXA, MRI, and BIA, were evaluated to ensure a robust synthesis of the available evidence. Literature available in the PubMed and Google Scholar databases was searched using keywords.

Results. The findings from clinical trials are not definitive. While several studies report a statistically significant loss of lean body mass (LBM), it remains proportionally smaller than fat loss and may constitute up to 40% of total weight reduction. Conversely, other studies—including the most recent ones—indicate that liraglutide primarily reduces adipose tissue. This effect preserves LBM and can enhance muscle quality by reducing intramuscular fat. The observed absolute decrease in LBM appears strongly correlated with total weight loss, implying it is a consequence of weight reduction itself rather than a specific drug-induced catabolic effect. Preliminary data also suggest liraglutide may have a direct anabolic and protective effect on skeletal muscle via modulation of signalling pathways. To mitigate muscle loss, pharmacotherapy should be combined with non-pharmacological interventions, such as a high-

protein diet (1.2–1.6 g/kg bw/day) and regular resistance training. Combination therapies including anabolic agents also represent a promising approach.
Keywords: liraglutide, GLP-1 receptor agonist, muscle mass loss, lean body mass, body composition

Introduction

Obesity has become one of the most pressing global challenges in public health. The condition is defined as a body mass index (BMI) of at least 30 kg/m²[1]. Over the past 50 years, there has been a continuous global increase in the incidence of obesity (with cases now exceeding 1 billion) and diabetes (with cases approaching 1 billion). The problem is pervasive, manifesting in all geographical regions, though its prevalence varies significantly from one locale to another (for instance, 4.5% obesity in Japan versus >50% in certain Pacific countries)[2]. The increasing prevalence of overweight and obesity poses a significant challenge to global health systems. In 2019, these conditions resulted in 5 million deaths and 160 million DALYs (Disability-Adjusted Life Years)[3]. Obesity is defined as a chronic, recurrent and progressive disease with complex underlying causes (genetic, environmental, socio-economic) and is not a consequence of individual choices. In order to effectively improve the health and well-being of patients, treatment must be based on long-term, complex strategies[4]. Lifestyle modification constitutes the foundation of treatment; however, its efficacy is constrained by the body's robust biological defence mechanisms (e.g. hormonal changes, metabolic slowdown) that counteract the maintenance of a negative energy balance. It is evident that even moderate weight loss (5–15%) can yield clinically significant benefits, including the prevention of the progression of complications and the enhancement of quality of life[5]. Recent studies have shown an increased tendency for the utilisation of pharmacotherapy in conjunction with bariatric surgery to facilitate weight reduction and ensure sustained outcomes[6].

GLP-1 receptor agonists, which were originally developed for the treatment of type 2 diabetes, are now considered to be among the most effective and promising pharmacological options for the treatment of obesity[7,8]. Liraglutide, a pioneering GLP-1 analogue, has garnered significant attention for its documented cardiovascular advantages. Numerous studies have demonstrated its efficacy in reducing the risk of cardiovascular death, heart attack and stroke in patients with type 2 diabetes and elevated cardiovascular risk[9]. In the case of liraglutide, studies report that it has a lesser effect on lean body mass (LBM) loss compared to newer drugs such as semaglutide and tirzpatide, while still achieving significant weight loss[10,11]. Liraglutide, a drug utilised in the treatment of type 2 diabetes and obesity, exerts a multifaceted effect on the human body. It has been demonstrated to suppress appetite, enhance glycaemic control, reduce blood pressure and promote weight reduction. It has been demonstrated that, in the absence of concomitant dietary recommendations, administration of liraglutide results in a weight reduction of 4–6 kg in patients diagnosed with type 2 diabetes. In combination with adequate dietary counsel for individuals without diabetes, liraglutide facilitates the maintenance of an average weight reduction of 9–10 kg for a period of 1–2 years[12]. Liraglutide exerts its effects both peripherally and centrally (via GLP-1 receptors in the brain), impacting appetite control and energy expenditure[13].

Liraglutide has been demonstrated to have a significant and beneficial effect on body composition. The therapeutic effect of the intervention results in significant, clinically relevant weight loss, which is predominantly due to a reduction in fat mass (FM)[14]. It is crucial to note that the therapeutic intervention resulted in a substantial decrease in visceral adipose tissue (VAT) ranging from 12.5% to 23%, thereby substantiating its notable efficacy in mitigating this pivotal cardiometabolic risk factor[15]. A plethora of studies, including LEAD 2, have documented a consistent reduction in both total fat mass (e.g., -0.7 to -2.4 kg) and VAT[16]. This ability to modify body composition, and in particular to target dangerous visceral adipose tissue, provides a rational justification for the use of liraglutide in the treatment of obesity and the reduction of associated complications[15]. Liraglutide has been demonstrated to be efficacious in the reduction of both total body weight and regional adipose tissue. A 16-week study was conducted in which a dose of 3 mg/day was administered, resulting in a substantial decline in total body weight of 5.8 kg and a decrease in total, trunk and limb fat[17]. Analogous outcomes were observed subsequent to 24 weeks of treatment with a dose of 1.8 mg/day, where the patients' body weight diminished from 81.1 kg to 75.5 kg (Δ= –5.6 kg; $P<0.01$), and BMI decreased by 1.9 kg/m²[18]. In addition, it has been confirmed that liraglutide causes a significant reduction in absolute fat mass in all analysed body regions, as measured by DXA

(dual-energy X-ray absorptiometry)[18]. Liraglutide, administered at a dose of 3.0 mg/day, resulted in a statistically significant (p=0.009) reduction in intramuscular adipose tissue (IMAT) when compared to the placebo. The mean percentage decrease in thigh muscle fat was -2.87% in the liraglutide-treated group, while a minimal change (+0.05%) was observed in the placebo group. This reduction was consistent across all patient subgroups analysed, regardless of age, race, ethnicity or BMI category. This finding is of significance as excess intramuscular fat has been demonstrated to be a risk factor for cardiovascular disease, insulin resistance and impaired muscle function[19].

The weight loss induced by GLP-1 agonists (including liraglutide) and dual GLP-1/GIP agonists has been shown to be comparable to the effects of bariatric surgery. However, concerns have been raised regarding the potential consequences of muscle loss. A plethora of clinical studies have observed a considerable variation in the proportion of lean body mass in total weight loss, ranging from 40-60% to approximately 15% or less[20]. Despite the efficacy of GLP-1 agonists in reducing fat mass, studies demonstrate that up to 40% of total weight loss may originate from fat-free mass (FFM). It is imperative to differentiate FFM from muscle mass (SM), as FFM encompasses additional components[21]. The most common adverse effects of liraglutide include nausea, diarrhoea, vomiting, constipation, abdominal pain, tachycardia, hypotension, indigestion, and injection site reactions such as itching, erythema, and rash[9,22]. Further investigation is required to ascertain the impact of liraglutide on fat-free mass (FFM) and lean body mass (LBM). In the extant literature, these terms are frequently employed in a mutually exclusive manner, despite the fact that LBM exclusively refers to fat-free soft tissue, whereas FFM is defined as the sum of LBM and bone mass[23]. The phenomenon of undesirable LBM loss is of particular relevance in the context of obesity treatment. A comprehensive analysis of the repercussions of bariatric surgery has revealed that a loss of 8 kg of LBM (representing 21% of the overall weight reduction) can engender deleterious long-term consequences for functional fitness, metabolism, and the probability of weight regain[24]. This has been demonstrated to result in the development of sarcopenic obesity, a condition characterised by the convergence of detrimental effects arising from low muscle mass and high fat mass[24]. Liraglutide has been demonstrated to exacerbate sarcopenia, a condition characterised by a decline in muscle mass and strength, which has been associated with an elevated risk of falls, cardiovascular disease and mortality. In older individuals with low muscle strength, sarcopenia has been shown to be associated with a 50% increased risk of all-cause mortality and an 81% increased risk of cardiovascular events at the lowest muscle mass[10,25–27]. In the case of incretin-based therapies, greater weight loss has been shown to correlate with

improved metabolic parameters (HbA1c, lipids, blood pressure)[28], however the impact of muscle mass loss on long-term cardiovascular risk remains unclear. Consequently, it is imperative that weight reduction strategies prioritise fat loss while minimising muscle mass reduction.

The effect of liraglutide on lean body mass

The impact of liraglutide on lean body mass constitutes a pivotal concern in the therapeutic management of obesity and type 2 diabetes. A plethora of studies have been conducted on this topic, yet the results remain inconclusive. The majority of these studies have reported a significant decrease in body weight and improvement in body composition, primarily attributed to a reduction in adipose tissue. The findings of liraglutide therapy exhibited variability across studies, with some studies reporting a significant decrease in lean body mass, while others observed stability or a minimal decrease that was deemed to be clinically insignificant.

The findings of several clinical studies demonstrate that liraglutide therapy results in a reduction of both lean body mass and fat mass. In a study of obese individuals, 20 weeks of treatment with 3.0 mg liraglutide resulted in a 2.0% (~1.1 kg) loss of LBM with a simultaneous 15.4% (~6.8 kg) reduction in fat mass[14]. In another population of overweight/obese patients with type 1 diabetes, 26 weeks of treatment with liraglutide (1.8 mg) was associated with a loss of 4.7% (~2.5 kg) of LBM[29]. The loss of lean body mass encompassed both muscle mass and other lean tissue (e.g. water, bone), as confirmed by DXA. Meta-analyses indicate an absolute decrease in LBM of -2.5 kg ($P<0.001$) under the influence of liraglutide, with no significant change in the placebo group, with the ratio of fat mass loss to LBM being approximately 1.8:1.0. It has been determined that muscle loss can account for up to 36% of total weight loss, thereby underscoring its clinical significance[29]. This effect is statistically significant and is also observed when the drug is used in isolation, without intensive lifestyle intervention (change: -0.8 ± 1.5 kg; $p=0.007$). This is in contrast to calorie restriction-based intervention, which may allow LBM to be preserved[30]. As demonstrated by the preceding studies, whilst liraglutide therapy has been shown to be effective in reducing adipose tissue, there is a possibility that it may also be associated with a loss of lean body mass. This is a significant side effect that must be considered within the framework of therapeutic strategies[10].

In contrast to the findings of the aforementioned studies, the majority of recent literature suggests that liraglutide has the capacity to preserve lean body mass or muscle mass, and may even exert a beneficial effect on body composition. In a study evaluating the use of liraglutide 3.0 mg over a period of 20 weeks, a significant 15.4% reduction in FM was observed, while

LBM loss was only 2.0%, and the difference in LBM loss compared to placebo was not statistically significant (p=0.61)[14]. This finding indicates that the drug does not result in a greater loss of LBM beyond that observed with lifestyle modifications alone. In a further comparative study, the median loss in body mass (LBM) in the liraglutide group (-1.3 kg) did not differ significantly from that in the placebo group (-0.65 kg), confirming that weight loss is primarily derived from adipose tissue[17]. These observations are corroborated by studies in which no statistically significant reduction in LBM was observed after three months of treatment with 3 mg liraglutide compared to baseline[31], and in a short-term study with a dose of 0.6 mg, a trend towards an increase was even observed[32]. Detailed regional analyses employing the DXA method indicate that any LBM losses are minimal and may only concern minor, statistically significant changes in the android (-0.11 kg) and gynoid (-0.19 kg) regions, without resulting in a substantial reduction in total muscle mass, trunk mass or limb mass[18]. It is important to note that imaging studies, including magnetic resonance imaging (MRI), have documented a significant reduction in visceral fat with no changes in musculature[33]. In a separate study, liraglutide (1.2 mg/day) induced a loss of both fat mass (FM) and lean body mass (LBM), with the loss of FM being predominant (~75% of the total weight reduction). The increase in natriuretic peptides (ANP/BNP) in response to liraglutide has been observed to correlate with weight and fat reduction, thus suggesting a novel mechanism of action for the drug[34]. Despite the fact that a prolonged, 10-month observation in real-world conditions demonstrated an absolute decrease in LBM of 2.7 kg, which accounted for 23% of total weight loss, its percentage share in body composition exhibited a significant increase from 49.3% to 51.8%, attributable to a proportionally greater reduction in fat[35]. This ratio of fat to muscle loss is favourable and has also been observed in other population studies[19,36]. It is important to note that the absolute loss of LBM during liraglutide therapy is strongly correlated with total weight loss ($p<0.0001$), indicating that the decrease in LBM is mainly due to the weight reduction itself, and not to a specific adverse effect of the drug, which does not cause a disproportionately greater loss of LBM compared to lifestyle intervention alone (insignificant difference, p=0.06)[37,38]. The scientific data cited confirm that liraglutide causes a beneficial change in body composition, consisting of a predominant reduction in adipose tissue – especially dangerous visceral fat – while effectively preserving lean body mass (LBM). This is a significant metabolic advantage over traditional low-calorie diets and some other weight loss interventions[33,39].

The effect of liraglutide on muscle mass and muscle function

Liraglutide exerts a multifaceted effect on muscle tissue, which is contingent upon the metabolic context and the method of assessment employed. Advanced MRI measurements revealed a substantial decrease in absolute muscle volume (-0.29 L). However, following normalisation for gender and body weight (muscle volume z-score), the discrepancy compared to the placebo became non-significant, suggesting a loss that is proportional to the overall weight reduction[19]. However, the beneficial effect on muscle quality, consisting in the reduction of harmful fat infiltration and improvement of muscle composition, is crucial[19]. These observations are corroborated by DXA studies, which demonstrated that treatment did not result in a substantial decrease in skeletal muscle mass (SMM) or skeletal muscle mass index (SMI)[40,41]. In addition, in a population of elderly patients with type 2 diabetes on a normocaloric diet, liraglutide not only preserved but potentially stabilised LBM, suggesting a protective or even anabolic effect on skeletal muscle[42]. Furthermore, a single study has confirmed that liraglutide treatment results in significant improvement in glycaemic control and body composition through reduction in body weight and fat mass, while preserving both muscle mass and muscle function (measured by handgrip strength). This is a key advantage in obese patients with type 2 diabetes at risk of sarcopenia[43].

It is evident that liraglutide therapy, akin to other interventions that result in expeditious weight reduction, is associated with a diminution of LBM. This phenomenon poses a substantial clinical conundrum. Within the domain of obesity treatment, LBM loss in response to GLP-1 analogues can contribute up to approximately 10% of the total weight reduction, and its magnitude (approximately 6 kg) is occasionally analogous to the loss resulting from over a decade of ageing or intensive chemoradiotherapy[10]. This has serious consequences, as skeletal muscle is a key metabolic organ that determines basal metabolic rate; its loss reduces energy expenditure, promoting weight regain after discontinuation of therapy. Furthermore, the evidence suggests that reduced muscle mass is an independent risk factor for sarcopenia, frailty syndrome and, consequently, falls, disability and higher overall and cardiovascular mortality[10]. Conversely, mounting preclinical evidence signifies that liraglutide may exert a direct, protective and anabolic effect on skeletal muscle. A plethora of studies utilising both cell and animal models have revealed that the pharmaceutical compound, by binding to the GLP-1 receptor present in myoblasts and myocytes, activates cAMP-dependent signalling cascades, including the PKA/CREB and PI3K/Akt pathways[44]. This activation has been shown to inhibit the expression of key ubiquitin-proteasome system proteins involved in proteolysis, such as atrogin-1 (MAFbx) and MuRF-1, while increasing the expression of myogenic factors such

as MyoD and myogenin[44–46]. It is noteworthy that the SIRT1 protein is regarded as the pivotal mediator of these advantageous effects, and its activation by liraglutide is imperative for the suppression of muscle wasting and the promotion of muscle differentiation[46]. Liraglutide has been demonstrated to enhance muscle insulin sensitivity through the augmentation of GLUT4 transporter translocation and glucose uptake. In addition, it has been observed to exert a protective effect on mitochondria by stimulating their biogenesis via PGC-1α and enhancing oxidative capacity[42,45]. It is evident that additional mechanisms exist, which include improved muscle microcirculation, thus enhancing the delivery of nutrients and oxygen[41,47]. Furthermore, there is a reduction of lipotoxicity and inflammation in muscles by lowering intramuscular lipid levels and pro-inflammatory cytokines such as TNF-α and IL-6[45]. In various animal models of atrophy of different etiologies (induced by denervation, glucocorticoids, mechanical damage or ovariectomy), liraglutide has been demonstrated to not only prevent muscle mass and strength loss, but also accelerate regeneration by increasing the cross-sectional area of fibres and restoring motor function[44]. This finding suggests the potential of liraglutide to have therapeutic benefits in cases of obesity, including conditions of pathological muscle wasting. In clinical trials involving humans, the observations are inconclusive, but some data indicate that liraglutide may promote beneficial body composition changes, where fat loss predominates over muscle loss[41,48]. It has been hypothesised that this phenomenon may be attributable to the selective activation of fatty acid oxidation and thermogenesis in adipocytes, without the concomitant activation of catabolic adrenergic pathways, which serves to protect muscle proteins from degradation[33]. Moreover, glucagon suppression by liraglutide has the potential to restrict systemic catabolism[33].

Methods for minimising muscle loss during treatment with liraglutide

The effect of liraglutide on LBM, including muscle mass, remains unclear. Nevertheless, a substantial body of scientific research has emerged that underscores the necessity to prevent and counteract muscle mass loss. The following are the currently suggested methods for achieving this goal during treatment with liraglutide.

In order to prevent muscle mass loss during treatment with liraglutide, the concept of combination therapy with anabolic drugs is a promising direction of research aimed at balancing beneficial fat reduction with the preservation of LBM[23,49]. This strategy may be based on monoclonal antibodies that target the myostatin and activin pathway, such as bimagrumab (which blocks the activin receptor type II, ActRII), trevogrumab (which is an anti-myostatin antibody) and garetosmab (which is an anti-activin A antibody). In preclinical studies, these antibodies have been shown to improve LBM when used in combination with other GLP-1

agonists (e.g. semaglutide)[16,21]. It is also promising to consider combining liraglutide with selective androgen receptor modulators (SARMs) or targeted nutritional support, e.g. β-hydroxy-β-methylbutyrate (HMB)[23]. In selected cases, such as obesity accompanied by growth hormone deficiency, the addition of growth hormone (GH) may be considered. However, its use requires caution due to potential side effects, including insulin resistance and oedema[16,49]. In order to prevent muscle mass loss during treatment with liraglutide in cases of confirmed hypogonadism, it appears to be important to consider testosterone supplementation, which can be carried out under medical supervision[39]. In men with testosterone levels below 350 ng/dL and no contraindications, a long-acting ester, testosterone undecanoate, was utilised for this purpose at a dose of 1000 mg administered intramuscularly every 90 days[13]. In women with confirmed testosterone deficiency (concentration <14 ng/dL), a topical testosterone cream at a dose of 2 mg per day was used[13]. The authors posit that such replacement therapy in conditions of deficiency not only promotes fat loss, but may also help to preserve lean muscle mass, potentially complementing treatment with a GLP-1 receptor agonist[13].

Non-pharmacological lifestyle interventions, with particular emphasis on proper nutrition and physical activity, play a fundamental role in preventing muscle mass loss during liraglutide therapy. A fundamental nutritional aspect is the assurance of adequate protein intake in the diet, with recommended levels ranging from 1.2 to 1.6 grams per kilogram of body weight per day. This is imperative to facilitate muscle protein synthesis in conditions of a calorie deficit[50–53]. Research has demonstrated that augmenting protein intake during weight reduction regimens may directly correlate with enhanced preservation of lean body mass, encompassing muscle mass[30]. The fundamental and most evidence-based physical intervention is regular resistance training, recommended at a frequency of 2–3 sessions per week, at 50–80% of maximum weight, involving multi-joint exercises for large muscle groups[10,22,48,50,51]. Meta-analyses have confirmed that supervised resistance training lasting more than 10 weeks can increase muscle mass or reduce its loss by approximately 1 kilogram during calorie restriction interventions[10]. Studies such as S-LiTE and the work of Lundgren et al. have shown that combining liraglutide with an exercise programme (especially one combining resistance and aerobic training) results in a more favourable weight loss profile, with greater fat reduction and better preservation or even a slight increase in LBM, compared to drug monotherapy[22,49,50]. A comprehensive, multidisciplinary approach combining pharmacotherapy with personalised dietary counselling (e.g. based on programmes such as LEARN), behavioural counselling and regular monitoring of body composition using precise

methods [DXA, BIA (Bioelectrical Impedance Analysis)] is considered an integral and key element of effective therapy, minimising the risk of sarcopenia[13,14,17,23,35,54]. For older people or those at high risk of sarcopenia, the recommendations are extended to include vitamin D and calcium supplementation, ensuring adequate hydration and avoiding excessive calorie restriction[52,54,55]. It is important to note that the use of pharmacotherapy in isolation, without the concurrent implementation of these interventions, may be associated with an increased risk of adverse muscle loss[30,37,49,52].

The discontinuation of liraglutide therapy has been demonstrated to be associated with adverse changes in body composition and an increased risk of sarcopenia. As demonstrated in the relevant animal studies, the repeated cycles of 'switching on' and 'switching off' the drug result in complete regeneration of fat mass, whilst only partial recovery of lean body mass occurs. This results in an increase in the fat-to-muscle ratio[56]. In human subjects, the cessation of drug administration typically results in weight regain, which is primarily adipose tissue (approximately 6.3 kg of fat vs. 2.5 kg of lean mass), potentially exacerbating the imbalance and promoting sarcopenic obesity[57]. The fundamental preventive strategy, therefore, is to avoid discontinuing therapy and to combine it with lifestyle interventions, primarily regular resistance training and adequate protein intake in the diet, in order to minimise muscle loss and unfavourable fat gain[57].

Conclusion

Obesity, a major public health concern, necessitates multifaceted and prolonged therapeutic interventions that extend beyond mere lifestyle modification. GLP-1 receptor agonists, including liraglutide, have transformed the pharmacological management of this condition, resulting in substantial weight reduction and documented metabolic and cardiovascular advantages. The primary mechanism of action of liraglutide is the reduction of fat mass, with a particularly beneficial effect on visceral adipose tissue, which is key in terms of metabolic risk. The impact of the pharmaceutical compound on lean body mass (LBM) and muscle mass remains the subject of ongoing research and analysis. There are reports of a statistically significant, albeit proportionally smaller than fat loss, decrease in LBM. However, recent data suggest that liraglutide results in a favourable body composition change, characterised by predominantly fat reduction and minimal muscle mass loss, which is proportional to overall weight loss and occasionally statistically insignificant in comparison to placebo. Notably, the efficacy of the drug is characterised by its ability to reduce adverse fat infiltration, thereby enhancing muscle quality.

The primary conclusion derived from the analysis is that liraglutide is an effective tool for reducing body weight and fat tissue. However, its optimal and safe use requires awareness of its potential impact on muscle mass. The primary challenge is not the potential minor loss of LBM during therapy, but rather the risk of adverse alterations in body composition following discontinuation, when adipose tissue is primarily restored, which can result in sarcopenic obesity. Consequently, the utilisation of liraglutide pharmacotherapy as a monotherapy is not recommended. It is imperative that this approach is complemented by non-pharmacological interventions, chiefly a diet that ensures sufficient protein intake (1.2-1.6 g/kg bw/day) and regular resistance training, both of which are pivotal in preserving and augmenting muscle mass.

The outlook for the future focuses on the further personalisation and optimisation of treatment. It is imperative that research is conducted to develop standardised, evidence-based protocols combining liraglutide with a precisely tailored nutrition and exercise plan. A promising direction in this research is the development of combination therapies that combine GLP-1 agonists with anabolic or anti-catabolic substances, such as antibodies that block the myostatin/activin pathway (e.g. bimagrumab) or selective androgen receptor modulators (SARMs). In clinical practice, it will be necessary to implement routine monitoring of body composition using precise methods (such as DXA or BIA), which will allow for early detection of adverse trends and modification of therapy. Long-term studies should evaluate the impact of muscle-sparing strategies on distant endpoints, such as functional fitness, cardiovascular risk and the maintenance of weight loss effects. In summary, the future of obesity and diabetes treatment with liraglutide lies in integrated, multidisciplinary care models, where the drug acts as a catalyst for lasting change, focused not only on weight loss but on achieving and maintaining an optimal, healthy body composition.

Disclosure

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