Sports Science Exchange (2026) Vol. 42, No. 274, 1- 8

NUTRITIONAL AND PHYSICAL ACTIVITY OPTIMIZATION FOR GLP-1 BASED THERAPIES

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KEY POINTS

- The discovery of nutrient-stimulated incretin hormones including glucagon-like peptide 1 (GLP-1) and gastric inhibitory polypeptide (also known as glucose-dependent insulinotropic polypeptide, GIP) has influenced the treatment of disorders associated with energy balance and systemic metabolism (i.e. obesity and diabetes) with approval of incretin-based therapies (i.e., GLP-1 and dual GLP-1/GIP receptor agonists) representing a major breakthrough for weight loss therapy. The mechanism in which these medications are effective appears to be multi-factorial, including reduced appetite and increased satiety at the onset of therapy.
- Despite the clinical implications of incretin-based therapy for weight loss, these therapies are not without unintended consequences. Most notably, as energy intake often declines substantially, individuals may be at risk of inadequate intake of key nutrients such as protein, fiber, and micronutrients, which can contribute to loss of lean mass, and potentially increase the risk of sarcopenia and malnutrition.
- Due to reduced energy intake, along with side effects of the medications such as nausea and vomiting, incretin-based therapies should implement a diet including nutrient-dense and fiber-rich foods such as vegetables, fruits, and whole grains to prevent micronutrient deficiencies, along with appropriate fluid intake to minimize the risk of dehydration, a frequent adverse event contributing to serious outcomes in incretin-based therapy users.
- Physical activity (i.e., exercise training) should be progressively increased in this population to meet public health guidelines, which consist of 150 minutes per week of moderate to vigorous activity, with at least 2 days per week focused on whole-body resistance training. Progressive resistance exercise, along with adequate protein intake, may preserve or attenuate the loss of fat-free/skeletal muscle mass during incretin-based therapy.
- Shifting from medication-led weight loss to a lifestyle-sustained health approach by focusing on key lifestyle pillars (nutrient-dense and high protein diet, proper hydration, and structured resistance exercise training) is critical for continued health support for individuals taking incretin-based therapies.

INTRODUCTION

Body weight regulation is a complex process dependent on various factors including genetics and lifestyle. While lifestyle-based interventions based on diet and physical activity remain a cornerstone for body weight regulation, additional interventions, including medically focused therapies, could be used to effectively achieve desired health outcomes. This is particularly important in individuals with obesity, in which >5% weight loss reduces the risk of obesity-related complications, with greater benefits as weight loss increases (Almandoz et al., 2024). Incretins are a group of nutrient-stimulated hormones released by the gut in response to food. These hormones, including glucagon-like peptide 1 (GLP-1) and gastric inhibitory polypeptide (GIP, also known as glucose-dependent insulinotropic polypeptide), have influenced the treatment of disorders associated with energy balance and systemic metabolism (i.e., obesity and diabetes). The approval of GLP-1 receptor agonists (GLP-1 RA), that were initially recommended for diabetes treatment, now represents a major breakthrough for weight loss therapy. The mechanism in which these medications are effective appears to be multi-factorial, including reduced appetite and increased satiety at the onset of therapy.

While most pharmacological interventions yield <10% weight loss, some GLP-1 receptor agonists, such as semaglutide, have been shown to achieve significantly higher (>15%) loss of body mass (Wilding et al., 2021). Incretin-based therapies are approved for weight management in obese (Body Mass Index (BMI) ≥ 30 kg/m^2) and overweight (BMI ≥ 27 kg/m^2) individuals in the presence of at least one weight related comorbidity (Gigliotti et al., 2025). However, these medications have demonstrated multiple additional benefits beyond weight loss and glucose management, such as improvements in cardiovascular risk factors (e.g., blood pressure and lipids), inflammation, obstructive sleep apnea, metabolic dysfunction-associated steatotic liver disease (MASLD) and metabolic dysfunction-associated steatohepatitis (MASH), and symptoms related to heart failure with preserved ejection fraction (American Diabetes Association Professional Practice Committee for Diabetes, 2026) and additional therapeutic effects continue to be investigated.

Despite the clinical implications of incretin-based therapies for weight loss, these therapies are not without unintended consequences that may have negative implications. Most notably, as energy intake often declines substantially, individuals may be at risk of inadequate intake

of key nutrients such as protein, fiber, and micronutrients, which can contribute to loss of lean mass, and potentially increase the risk of sarcopenia and malnutrition (Mogna-Peláez & Guasch-Ferré, 2026). Additionally, fluid losses due to gastrointestinal symptoms and reduced fluid intake may increase the risk of dehydration in this population (Al-Najim et al., 2025). Collectively, these findings suggest that adjunct lifestyle therapies, such as dietary and physical activity recommendations are needed for those undergoing incretin-based therapies to optimize health outcomes (Kushner et al., 2026; World Health Organization, 2025). Unfortunately, people using incretin-based therapies usually do not receive dietary or physical guidance. The aim of this Sports Science Exchange (SSE) article is to provide an overview of the physiological impact of incretin-based therapies beyond weight loss. The article provides potential lifestyle recommendations that may combat the unintended consequences provoked by these medications. Further, as the number of individuals using incretin-based therapies rises along with the advent of newer medications, gaps in the literature that are needed to provide scientifically backed lifestyle recommendations for this population are discussed.

INCRETIN–BASED THERAPIES: THE GOOD AND THE BAD

Type of Medications and Mode of Action

The most common incretin-based therapy are GLP-1 RAs, which include early versions such as liraglutide and newer generation semaglutide. These synthetic peptides or small molecules activate the GLP-1 receptor, leading to glucose-dependent insulin secretion, suppression of glucagon release, delayed gastric emptying and appetite suppression mediated through modulation of hypothalamic pathways. Along with single molecule agonists, dual GLP-1/GIP RAs, such as tirzepatide, generate a synergistic effect to improve weight loss. Specifically, GLP-1 reduces caloric intake and improves glycemic regulation, while GIP enhances anorexigenic effects, and favorably modulates adipocyte metabolism to promote greater fat mass reduction (Cesaro et al., 2026). Currently, tirzepatide is available as weekly injection, while semaglutide is available as weekly injection and daily oral pill. Another oral Food and Drug Administration (FDA)-approved therapy in the United States is orforglipron, a once-daily small-molecule, non-peptide GLP-1 RA. Future innovation is expected to focus on dose intensification of established agents, the development of next-generation long-acting molecules, and agents combining multiple incretins (Khachaturov & Goulis, 2026).

Reduced Energy Intake

The most common causes for weight loss during incretin-based therapy are alterations in caloric consumption, with clinical and observational studies showing a marked reduction (~30–50%) in energy intake (Mogna-Peláez & Guasch-Ferré, 2026). In a 6-week trial, tirzepatide reduced energy intake by 525 kcal compared with placebo at week 3 and by 686 kcal at week 6, with reductions being non-selective across carbohydrate, protein, and fat intake (Martin et al., 2025). A dose-response effect of semaglutide on total energy intake is also apparent (~24% reduction at 1.0 mg for 12 weeks and ~35% reduction at 2.4 mg for 20 weeks) (Blundell et al., 2017; Friedrichsen et al., 2021). A more recent trial reported that semaglutide (2.4 mg) users consumed 30.1% fewer calories at week 20 compared with placebo. Interestingly, energy intake gradually increased after week 20 (25.4% fewer calories at week 40 and 21.9% at week 60), which coincided with attenuations in appetite-related indices (e.g., hunger, satiety). Nevertheless, the semaglutide group maintained a calorie deficit relative to placebo through week 60, supporting maintenance of weight loss (Tronieri et al., 2026).

Despite evidence of reduced energy intake, data remains limited on the actual amount of energy consumed by incretin-based therapy users, which will vary depending on the medication phase. For example, a retrospective cohort study, with a mean treatment duration of ~16 weeks, reported an average intake of 753 kcal/day (Korus et al., 2026), while a cross-sectional study, in which 39.1% of the participants used the medication for more than one year, reported a daily intake of 1,748 kcal (Johnson et al., 2025c) Both studies included different types of medications and did not specify doses. Thus, differences in study populations, methodologies, treatment duration, and medication type and dose should be considered when interpreting these findings. Beyond energy intake, the quality of diet of this population is usually poor, as a secondary analysis reported that individuals using incretin-based therapy had a "Healthy Eating Index" score of 54, indicating poor overall diet quality, and similar to the adult population average (~57) (Johnson et al., 2025a).

Body Composition

Changes in body mass are dictated by the gain/loss of fat mass and fat-free/lean mass, which consists of skeletal muscle, internal organs, and connective tissue (Janssen et al., 2023). Weight loss is driven by the loss of both fat and fat-free mass, with skeletal muscle being the main component in the loss of fat-free mass (Christoffersen et al., 2022). Wilding et al, (2021) reported that ~40% of total weight loss during semaglutide treatment was attributed to the loss in fat-free mass. An analysis of 17 studies revealed that incretin-based therapies elicit a loss of lean body mass in the range of 20-50%, which is equivalent to other weight loss therapies, including pharmacological sodium/glucose transport protein inhibitors (SGLT2i), diet-induced, and bariatric surgery. In a clinical trial, tirzepatide induced a ~6 kg loss in fat-free mass after 12-18 months of treatment. In context, this equated to ~10% of fat-free mass, which is equivalent to the loss of muscle mass seen with a decade of aging (Alcazar et al., 2020; Grosicki et al., 2022, 2024).

While the significance of the loss of fat-free and/or skeletal muscle mass in this population is not clear, this loss can potentially lead to severe outcomes in various populations. Of note, age is associated with the progressive loss in muscle mass, also known as sarcopenia, which can lead to decreased physical function and increased prevalence of frailty. In older adults (>60 years), incretin-based therapies increased markers of neuromuscular decline and increased risk of developing sarcopenia (Langer, et al., 2026b; Prokopidis, 2026; Qaisar et al., 2026; Ren et al., 2025). Additionally, a loss of fat-free mass can have

severe consequences for weight maintenance in this population. While not an uncommon characteristic of weight loss interventions, weight regain following cessation of pharmacotherapy occurs faster than after stopping behavioral weight management programs (by 0.3 kg monthly), independent of initial weight loss (West et al., 2026). The loss of fat-free mass with a consequent increase in fat mass can potentially increase the prevalence of sarcopenic obesity, a condition characterized by greater fat mass and lower muscle mass that is associated with an increased risk of cardiovascular disease, insulin resistance, and mortality (Miller & Wolfe, 2008). While several potential mechanisms have been suggested (i.e., lower total energy expenditure, decreased mitochondrial efficiency) (Choi et al., 2025; Christoffersen et al., 2022; Wang et al., 2025), it is unclear why incretin-based therapies accelerate weight regain following treatment cessation.

The changes in fat-free mass, presumably skeletal muscle, have led to an increased interest in preserving muscle during incretin-based therapies (Grosicki et al., 2024). However, recent reports suggest that incretin-based therapies may not accelerate the loss of skeletal muscle mass (Langer et al., 2026a; Tinsley & Heymsfield, 2024; Wu et al., 2026). While the absolute changes align with other weight loss interventions (i.e., very low-calorie diet and gastric bypass surgery) (Langer et al., 2026a; Tinsley & Heymsfield, 2024), the higher loss in fat-free mass appears to be attributed not solely to muscle but to other organ systems, such as the liver and bones (Karasawa et al., 2025; Langer et al., 2026a). Future research is needed to understand how skeletal muscle responds to incretin-based therapies, and whether these changes result in functional and/or metabolic maladaptations.

Despite not accelerating the loss of skeletal muscle, preserving or attenuating tissue loss appears to have a beneficial effect in incretin-based therapy users. In recent clinical trials, inhibition of myostatin, a negative inhibitor of skeletal muscle mass, has been shown to attenuate or preserve fat-free mass in subjects undergoing incretin-based therapies including semaglutide and tirzepatide (Heymsfield et al., 2026; Pratley et al., 2026). Along with the positive effects on fat-free mass, inhibition of myostatin signaling appears to impact fat mass. Specifically, in those prescribed semaglutide with bimagrumab, an antibody that targets activin II receptors to reduce myostatin signaling within tissue, led to a greater loss in total body fat mass than semaglutide alone (Heymsfield et al., 2026). Together, these studies highlight the important role of preserving skeletal muscle mass during incretin-based therapies.

NUTRITIONAL CONSIDERATIONS

Protein

Protein is essential in the human diet because it supplies amino acids needed for numerous physiological functions, including muscle mass maintenance, digestion, absorption, and transport of dietary nutrients (Kanter et al., 2026; Wu, 2016). Protein recommendations for individuals using incretin-based therapies are largely extrapolated from broader weight loss and sarcopenia literature, rather than based on direct evidence (Mogna-Peláez & Guasch-Ferré, 2026). During active weight loss, a protein intake of 1.2–1.6 g/kg/day is generally proposed to help preserve muscle mass (Mozaffarian et al., 2025). A cross-sectional study conducted in individuals that used incretin-based therapies for at least one month reported that protein intake (18.5% of total calories) was within the Acceptable Macronutrient Distribution Range (AMDR; 10-35%). However, absolute protein intake (77.3 g) fell along the lower end of the proposed targets of 1.2–2.0 g/kg/day for muscle mass preservation (calculated needs: 74–169 g/d). In addition, although 75% of participants reported eating more protein since starting medication, only 43% consumed at least 1.2 g/kg of protein, 10% consumed at least 1.6 g/kg, and 5% consumed at least 2.0 g/kg, calculated based on adjusted body mass (Johnson et al., 2025c).

While a relative amount of protein (1.2-1.6 g/kg/day) has been suggested as a starting point for this population, recent evidence shows that other variables (i.e., treatment stage, age, actual, ideal or adjusted body weight, etc.) should be considered for those taking incretin-based therapies. Sievenpiper et al. (2025) suggest that, during the weight loss phase, a protein intake of 1.2–1.5 g/kg actual body weight/day or equivalent to 25–30 % of energy on a 1600 kcal/day diet is recommended; while ≥0.8 g protein/kg/day is suggested during the weight maintenance phaseClick or tap here to enter text.. Populations with higher requirements such as older adults, those at risk of sarcopenia, or on high level of physical activity may require at least 1.0–1.2 g/kg/day (Sievenpiper et al., 2025), and up to 1.6–2.0 g/kg/day (Mogna-Peláez & Guasch-Ferré, 2026). Others suggest ensuring a minimum intake of 60 g of protein daily (Gigliotti et al., 2025). Further, while there is no consensus, Sievenpiper et al. (2025) recommend using actual body weight to estimate protein requirements, whereas others suggest using ideal or adjusted body weight as well as lean/fat-free mass to calculate protein requirements as actual body weight may overestimate requirements (Mozaffarian et al., 2025).

Along with total amounts of high-quality protein, even distribution across the day is recommended to help achieve optimal protein intake during weight loss or maintenance and maximize protein synthesis (Gigliotti et al., 2025; Kanter et al., 2026). Animal proteins are generally considered to be of higher quality than plant-based proteins because they are easily digested and readily provide all the essential amino acids (EAAs). In contrast, plant-based proteins often have lower levels of, or are lacking some EAAs such as lysine, methionine, and cysteine. However, combining different plant proteins can often improve the overall amino acid profiles (Kanter et al., 2026). Lean meats, poultry, plant proteins (e.g., beans, peas, lentils, whole grains), seafood, eggs, low-fat dairy, nuts, and seeds are preferred as protein sources, while red and processed meats should be limited (Gigliotti et al., 2025; Mozaffarian et al., 2025; Sievenpiper et al., 2025). Researchers have proposed distributing protein as ~0.3–0.4 g/kg of adjusted body weight containing ~2.5–3.0 g leucine per meal for most adults to preserve lean mass (Arslan, 2026). As appetite may be reduced, it may be useful to consume protein first during meals, prioritize lower-volume, nutrient-dense protein foods, and, if needed, consider supplementation with protein powders or use meal-replacement products, such as shakes,

bars, or other formulated foods, typically containing 15–25 g protein per serving (Almandoz et al., 2024; Gigliotti et al., 2025; Mozaffarian et al., 2025).

Nutrient Adequacy

Micronutrients

It is relevant to consider that individuals living with obesity tend to have micronutrient deficiencies, including B vitamins; vitamins A, C, and D; and minerals such as iron, zinc, and magnesium (Koceva et al., 2026). Key mechanistic drivers include poor diet quality, sequestration of fat-soluble vitamins in adipose tissue, inflammation, and altered metabolism, among others (Koceva et al., 2026). These deficiencies could be exacerbated among individuals using incretin-based therapies, where micronutrient deficiencies are also common and may be related to reduced energy intake, poor diet quality, gastrointestinal side effects, or interactions with other medications (Koceva et al., 2026).

Several recent studies have identified nutrient deficiencies in those undergoing incretin-based therapy. A retrospective observational cohort study using real-world claims data from ~461,000 patients found that 12.7% of patients developed a nutritional deficiency by 6 months, increasing to 22.4% by 12 months after medication initiation (Scott Butsch et al., 2025). Vitamin D was the most common deficiency observed, affecting 7.5% of individuals at 6 months, and 13.6% at 12 months. This finding is critical, as vitamin D deficiency is associated with impaired bone mineralization (osteomalacia), muscle weakness and pain, and hypocalcemia (Almandoz et al., 2024). Moreover, Scott Butsch et al., (2025) found that iron deficiency anemia is the most clinically relevant outcome. In support of this, one study reported that 10 weeks of semaglutide treatment was associated with a 13% median reduction in iron absorption compared to pretreatment levels (Melis et al., 2025). In another cross-sectional study (Johnson et al., 2025c), participants using incretin-based therapies completed a 3-day food record. Results confirmed insufficient intakes below the Dietary Reference Intake (DRI) for vitamin D and iron, as previously discussed, as well as calcium, choline, magnesium, potassium, and vitamins A, C, and E. The authors also reported adequate intakes, relative to the DRI reference values, for B-vitamins, copper, phosphorus, selenium, and zinc, while sodium intake exceeded the DRI. The noted insufficiencies were possibly related to low intake of essential food groups such as fruits, vegetables, grains, and dairy. However, evidence remains limited to observational studies, and randomized controlled trials with longer follow-up are needed to confirm these findings.

Overall, suggested key nutrients to monitor include vitamin D, vitamin B12, magnesium, iron, calcium, potassium and zinc (Simancas-Racines et al., 2026); and researchers emphasize dietary optimization rather than universal supplementation (Koceva et al., 2026). Mozaffarian et al. (2025) recommend prioritizing nutrient-dense, minimally processed foods to maintain vitamin and mineral intake, and suggest ongoing monitoring of diet. A recent expert consensus statement also recommends biochemical screening to monitor deficiencies, supplementation when intake is very low, and involvement of a dietitian during treatment (Sievenpiper et al., 2025). Management of gastrointestinal side effects is also relevant as it may compromise nutrient intake or absorption. For instance, to manage nausea it is recommended to eat smaller, more frequent meals slowly, stopping when full, limiting high-fat and spicy foods, maintaining adequate hydration, and moderating consumption of carbonated beverages (Gigliotti et al., 2025).

Fiber

Johnson et al. (2025c) reported a fiber intake below the DRI at 14.5 g/day in participants using incretin-based therapies. This nutrient supports gut and cardiometabolic health, promotes satiety, and may help manage common gastrointestinal symptoms. For constipation, physical activity and a fiber-rich diet that includes vegetables, fruits, and whole grains are recommended, along with appropriate hydration. Fiber and hydration are also important to help manage diarrhea, along with avoiding sugar alcohol and limiting intake of coffee, dairy, alcohol, and carbonated beverages (Gigliotti et al., 2025). Recommended fiber intake is ≥ 25 g/day for women, ≥ 30 g/day for men, and ≥ 35 g/day for individuals with diabetes, with emphasis on both soluble and insoluble fiber sources and consideration of fiber supplementation (e.g., psyllium) when needed (Sievenpiper et al., 2025). Fiber should be gradually increased and accompanied by adequate hydration to avoid gastrointestinal issues; a high fiber intake could cause flatulence or bloating. More research is needed on fiber supplements in this population, specifically side effect management, fiber type, and dosage (Johnson, 2026; Simancas-Racines et al., 2026).

Hydration

A pharmacovigilance study reported that dehydration occurred within the first weeks of incretin-based therapies and was the most frequent adverse event contributing to serious outcomes (He et al., 2024). Dehydration during incretin-based therapy may be due to reduced consumption of water-rich foods such as fruits and vegetables (Fitch et al., 2025), early satiety, and gastrointestinal side effects such as nausea, vomiting, and diarrhea, potentially resulting in dehydration and electrolyte disturbances (Al-Najim et al., 2025). Consequences of dehydration include a reduced estimated glomerular filtration rate and increased creatinine and potassium levels, which may have a negative impact on renal function (Fitch et al., 2025). Other symptoms such as hypotension, tachycardia, or dizziness may also be present (Almandoz et al., 2024). Thus, individuals undergoing incretin-based therapy should be advised to monitor hydration status (Simancas-Racines et al., 2026).

Due to the increased risk of dehydration, hydration remains a cornerstone in those undergoing incretin-based therapy. Sievenpiper et al. (2025) recommend tailoring hydration guidance to individual needs, although general guidance is 2–4 L/day or ~35 mL/kg body mass/day of water or non-sugary beverages. Recommended fluids include low-calorie beverages (e.g., tea, coffee), nutrient-dense beverages (e.g., low-fat milk, soy milk), and oral rehydration solutions when needed (Almandoz et al., 2024; Simancas-Racines et al., 2026). Alcohol and high caffeine intake should be limited because of potential diuretic

effects at high intakes (Almandoz et al., 2024). In addition, alcohol intake may increase nausea or gastrointestinal reflux (Mozaffarian et al., 2025).

Despite its importance, few studies have examined the impact of incretin-based therapies on fluid intake and thirst. Winzeler et al. (2020) reported a trend for reduced fluid intake and lower 24 h urine output during dulaglutide (a GLP-1 RA) treatment compared with placebo. Other hydration-related outcomes such as thirst perception may not be altered by incretin-based therapy (Blundell et al., 2017; Tronieri et al., 2026; Winzeler et al., 2020). However, Tronieri et al. (2026) showed that 5.6% of participants treated with semaglutide reported thirst as a side effect compared with 2.1% in the placebo group. More research is required to understand the fluid intake habits of incretin-based therapy users.

Dietary Supplements

Although a healthy diet including the relevant nutrients mentioned above is preferred, dietary supplements should be considered when this cannot be achieved solely from foods. A recent narrative review (Johnson et al., 2025b) proposed several dietary supplements that may help address unintended consequences associated with incretin-based therapies. They classified vitamin D, protein supplements, fiber, and omega 3 fatty acids as having strong evidence, while multivitamins, vitamins, minerals; whey protein, creatine, antioxidants, anti-inflammatory compounds, and probiotics were classified as having moderate evidence. Other supplements such as beta-hydroxy-beta-methylbutyrate (HMB; to stimulate muscle growth), and thermogenic agents (to increase energy expenditure and fat oxidation) were classified as having emerging evidence. More research in individuals using these medications is needed.

PHYSICAL ACTIVITY

Physical activity remains a critical adjunct to incretin-based therapy as it addresses physiological and functional domains that medications alone do not fully target. Physical activity interventions, such as exercise, can stimulate various physiological benefits beyond solely weight management, including insulin sensitivity, cardiovascular fitness, and metabolic flexibility. While data is still emerging, the combination of incretin-based therapies with exercise has highlighted several benefits. Notably, in liraglutide-treated subjects, exercise resulted in greater weight and total fat loss compared to incretin-based therapy alone (Lundgren et al., 2021). In a post-treatment follow-up, the authors observed that those undergoing liraglutide and exercise combination therapy had reduced body weight and body-fat percentage than those that underwent liraglutide therapy alone (Jensen et al., 2024a). Along with changes in body composition, emerging data has also shown that exercise combined with semaglutide treatment can improve markers of cardiometabolic health. This includes increased cardiorespiratory fitness, insulin sensitivity, and physical function, as well as decreased HOMA-IR and waist circumference in subjects undertaking combined exercise and liraglutide intervention (Lundgren et al., 2021). Additionally, Ingersen and colleagues (2023) showed that β-cell function improved to a greater extent in subjects undergoing 12 weeks of combined aerobic exercise training and semaglutide treatment than either approach alone. Collectively, these findings reveal that exercise is an important adjunct intervention to those undergoing incretin-based therapy.

Recently, resistance exercise training has been highlighted as an important adjunct intervention in those undergoing incretin-based therapy. Resistance exercise training is an effective intervention to enhance skeletal muscle mass, as well as combat muscle loss, resulting in improved strength and physical function (Lim et al., 2022; Mcleod et al., 2024; Schoenfeld et al., 2017). While there are no clinical studies that have examined the efficacy of a resistance-exercise based program on health outcomes with incretin-based therapy, previous work in interventions that promote energy deficits suggest resistance exercise training can mitigate the loss in fat-free mass (Hector et al., 2018; Sardeli et al., 2018; Villareal et al., 2017). Beyond muscle mass, resistance exercise training can also positively impact bone mineral density. This is critical as studies have shown that some (Cortes et al., 2025; Hernández-Martínez et al., 2022; Jensen., 2024a) but not all (Beavers et al., 2025; Mok et al., 2023), weight loss therapies, including incretin-based therapies, can reduce bone mineral density. Jensen et al. (2024b) have shown that combined exercise with liraglutide treatment preserved bone mineral density in adults with obesity, compared to liraglutide alone, which reduced spine and hip bone mineral density.

Currently, there are no physical activity guidelines specifically for those undergoing incretin-based therapies. In general, Jakicic et al. recommend that physical activity should be progressively increased in this population to meet public health guidelines, which consist of 150 min/week of moderate to vigorous activity, with 2 days per week focused on whole-body resistance training (Jakicic et al., 2024). However, if individuals are unable to complete 150 min of supervised exercise per week, a stepwise approach with shared and measurable goals agreed on by both the individual and the health care professional, such as a structured exercise plan or at least 4000–6000 steps/day is recommended (Sievenpiper et al., 2025). Furthermore, while higher physical activity levels (200-300 min/week) are suggested for those with overweight/obesity (Donnelly et al., 2009), it is unclear whether increased physical activity levels beyond public health guidelines will help those undergoing incretin-based therapy.

LIMITATIONS AND FUTURE RESEARCH

This SSE article has described the potential nutritional and physical activity interventions for users of incretin-based therapies. However, there are remaining gaps in the literature to provide clear lifestyle intervention guidelines for these individuals. First, despite a few recent reports of the nutritional habits of incretin-based therapy users (Johnson et al., 2025c), the precise nutritional deficiencies within this population are unclear. Additionally, nutritional recommendations are mostly extrapolated from other literature in individuals with obesity or sarcopenia. Further, while emerging evidence suggests an integral role of exercise during therapy, the proper prescription for exercise (i.e., mode, frequency, intensity, etc.) is unknown. For both nutrition and

exercise interventions, randomized controlled trials are needed to better define lifestyle prescriptions for these individuals.

While the efficacy of lifestyle interventions within a controlled laboratory setting are clear, their effectiveness under real-world, unsupervised conditions is less known. Under real-world, unsupervised conditions to support weight management, adherence to the exercise prescriptions ranged from 10-84% in overweight or obese subjects (Lieberman et al., 2026). These findings extend to those who have undergone other types of weight loss interventions, as previous research has shown that bariatric surgery patients accrue less than the recommended 150 min/week of physical activity that is required for metabolic benefits (Coen & Goodpaster, 2015). While the data are still emerging, it is critical to understand how physical activity habits are altered in those undergoing incretin-based therapies.

PRACTICAL APPLICATIONS

- Incretin-based therapies should be paired with behavioral and lifestyle modifications delivered through a multidisciplinary care model that integrates nutrition, physical activity, and clinical monitoring to help mitigate unintended consequences such as nutrient deficiencies, dehydration, gastrointestinal side effects, and lean mass loss, among others.
- Set individualized dietary targets that account for reduced appetite and lower energy intake, with emphasis on adequate protein, fiber, and micronutrient intake, and hydration.
- While set physical activity guidelines have not been identified, incretin-based therapy users should undertake at least 150 minutes per week of moderate to vigorous activity. Resistance exercise training (at least 2 days per week), in conjunction with adequate intake (1.2-1.6 g/kg/day) of high-quality protein, may help preserve or attenuate the loss of muscle mass observed in various weight-loss interventions, including incretin-based therapy.

SUMMARY

The discovery of incretin-based therapies has influenced the treatment of disorders associated with energy balance and systemic metabolism (i.e., obesity and diabetes) representing a major breakthrough for weight loss therapy. The mechanism in which these medications are effective appears to be multi-factorial, including reduced appetite and increased satiety at the onset of therapy. However, due to potential unintended consequences of these therapies (macro- and micro-nutrient deficiencies, dehydration, loss of fat-free/skeletal muscle mass), adjunct therapies are needed in this population. Shifting from medication-led weight loss to a lifestyle-sustained health approach by focusing on key lifestyle pillars (nutrient-dense, high protein diet, adequate fiber, proper hydration, and structured resistance exercise training) is critical for continued health support for individuals undergoing incretin-based therapies.

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REFERENCES

Alcazar, J., P. Aagaard, B. Haddock, R.S. Kamper, S.K. Hansen, E. Prescott, L.M. Alegre, U. Frandsen, and C. Suetta (2020). Age- and sex-specific changes in lower-limb muscle power throughout the lifespan. J. Gerontol. Series A Biol. Sci. Med. Sci. 75:1369–1378.

Almandoz, J.P., T.A. Wadden, C. Tewksbury, C.M. Apovian, A. Fitch, J.D. Ard, Z. Li, J. Richards, W.S. Butsch, I. Jouravskaya, K.S. Vanderman, and L.M. Neff (2024). Nutritional considerations with antiobesity medications. Obesity 32:1613–1631.

Al-Najim, W., A. Raposo, M.N. BinMowyna, and C.W. le Roux (2025). Unintended consequences of obesity pharmacotherapy: A nutritional approach to ensuring better patient outcomes. Nutrients 17:1934.

American Diabetes Association Professional Practice Committee for Diabetes (2026). Obesity and weight management for the prevention and treatment of diabetes: Standards of care in diabetes—2026. Diab. Care 49(Suppl. 1):S166–-S182.

Arslan, S. (2026). Medical nutrition in the glucagon-like peptide-1 (GLP-1) era: Protein strategies, micronutrient monitoring, and lean mass preservation. Clin. Nutr. ESPEN 73:103305.

Beavers, K.M., T.M. Cortes, C.M. Foy, L. Dinkla, F. Reyes San Martin, J.D. Ard, M.C. Serra, and D.P. Beavers (2025). GLP1Ra-based therapies and DXA-acquired musculoskeletal health outcomes: A focused meta-analysis of placebo-controlled trials. Obesity 33:225–237.

Blundell, J., G. Finlayson, M. Axelsen, A. Flint, C. Gibbons, T. Kvist, and J.B. Hjerpsted (2017). Effects of once-weekly semaglutide on appetite, energy intake, control of eating, food preference and body weight in subjects with obesity. Diab. Obes. Metab. 19:1242–1251.

Cesaro, A., V. Acerbo, and P. Calabrò (2026). Tirzepatide and semaglutide: different twins? Eur. Heart J. 28(Suppl. 5):v57–v61.

Choi, R.H., T. Karasawa, C.A. Meza, J.A. Maschek, A.M. Manuel, L.S. Nikolova, K.H. Fisher-Wellman, J.E. Cox, A. Chaix, and K. Funai (2025). Semaglutide-induced weight loss improves mitochondrial energy efficiency in skeletal muscle. Obesity 33:974–985.

Christoffersen, B.Ø., G. Sanchez-Delgado, L.M. John, D.H. Ryan, K. Raun, and E. Ravussin (2022). Beyond appetite regulation: Targeting energy expenditure, fat oxidation, and lean mass preservation for sustainable weight loss. Obesity 30:841–857.

Coen, P.M., & B.H. Goodpaster (2015). A role for exercise after bariatric surgery? Diab. Obes. Metab. 18:16.

Cortes, T.M., K. Chae, C.M. Foy, D.K. Houston, and K.M. Beavers (2025). The impact of lifestyle-based weight loss in older adults with obesity on muscle and bone health: A balancing act. Obesity 33(Suppl 1):22–40.

Donnelly, J.E., S.N. Blair, J.M. Jakicic, M.M. Manore, J.W. Rankin, and B.K. Smith (2009). American College of Sports Medicine Position Stand. Appropriate physical activity intervention strategies for weight loss and prevention of weight regain for adults. Med. Sci. Sports Exerc. 41:459–471.

Fitch, A., L. Gigliotti, and H.E. Bays (2025). Application of nutrition interventions with GLP-1 based therapies: A narrative review of the challenges and solutions. Obes. Pillars 16:100205.

Friedrichsen, M., A. Breitschaft, S. Tadayon, A. Wizert, and D. Skovgaard (2021). The effect of semaglutide 2.4 mg once weekly on energy intake, appetite, control of eating, and gastric emptying in adults with obesity. Diab. Obes. Metab. 23:754–762.

Gigliotti, L., H. Warshaw, A. Evert, C. Dawkins, J. Schwartz, C. Susie, R. Kushner, S. Subramanian, D. Handu, and M. Rozga (2025). Incretin-based therapies and lifestyle interventions: The evolving role of registered dietitian nutritionists in obesity care. J. Acad. Nutr. Dietet. 125:408–421.

Grosicki, G.J., C.S. Zepeda, and C.W. Sundberg (2022). Single muscle fibre contractile function with ageing. J. Physiol. 600:5005–5026.

Grosicki, G.J., N.V. Dhurandhar, J.L. Unick, S.M. Arent, J.G. Thomas, H. Lofton, M.C. Shepherd, J. Kiel, C. Coleman, and S.S. Jonnalagadda (2024). Sculpting success: The importance of diet and physical activity to support skeletal muscle health during weight loss with new generation anti-obesity medications. Curr. Develop. Nutr. 8:11.

He, L., Q. Li, Y. Yang, J. Li, W. Luo, Y. Huang, and X. Zhong (2024). Pharmacovigilance study of GLP-1 receptor agonists for metabolic and nutritional adverse events. Front. Pharmacol. 15:1416985.

Hector, A.J., C. McGlory, F. Damas, N. Mazara, S.K. Baker, and S.M. Phillips (2018). Pronounced energy restriction with elevated protein intake results in no change in proteolysis and reductions in skeletal muscle protein synthesis that are mitigated by resistance exercise. FASEB J. 32:265–275.

Hernández-Martínez, A., L. Veras, G. Boppre, G., A. Soriano-Maldonado, J. Oliveira, F. Diniz-Sousa, and H. Fonseca (2022). Changes in volumetric bone mineral density and bone quality after Roux-en-Y gastric bypass: A meta-analysis with meta-regression. Obes. Rev. 23:8.

Heymsfield, S.B., L.J. Aronne, P. Montgomery, L.B. Klickstein, L.A. Coleman, K. Dole, L. Mindeholm, S. Spruill, X. Li, K.M. Attie, K. M., and BELIEVE trial investigators. (2026). Bimagrumab plus semaglutide alone or in combination for the treatment of obesity: A randomized phase 2 trial. Nat. Med. 32:869-882.

Ingersen, A., M. Schmücker, C. Alexandersen, B. Graungaard, T. Thorngreen, J. Borch, J.J. Holst, J.W. Helge, and F. Dela (2023). Effects of aerobic training and semaglutide treatment on pancreatic β-cell secretory function in patients with type 2 diabetes. J. Clin. Endocrinol. Metab. 108:2798–2811.

Jakicic, J.M., R.J. Rogers, and C.M. Apovian (2024). Contemporary treatments for obesity: Physical activity in the context of antiobesity medications. Translat. J. Am. Coll. Sports Med. 9:2.

Janssen, T.A.H., D.W. Van Every, and S.M. Phillips (2023). The impact and utility of very low-calorie diets: the role of exercise and protein in preserving skeletal muscle mass. Curr. Opin. Clin. Nutr. Metab. Care 26:521–527.

Jensen, S.B.K., M.B. Blond, R.M. Sandsdal, L.M. Olsen, C.R. Juhl, J.R. Lundgren, C. Janus, B.M. Stallknecht, J.J. Holst, S. Madsbad, and S.S. Torekov (2024a). Healthy weight loss maintenance with exercise, GLP-1 receptor agonist, or both combined followed by one year without treatment: A post-treatment analysis of a randomised placebo-controlled trial. EClin. Med. 69:102475.

Jensen, S.B.K., V. Sørensen, R.M. Sandsdal, E.W. Lehmann, J.R. Lundgren, C.R. Juhl, C. Janus, T. Ternhamar, B.M. Stallknecht, J.J. Holst, N.R. Jørgensen, J.E.B. Jensen, S. Madsbad, and S.S. Torekov (2024b). Bone health after exercise alone, GLP-1 receptor agonist treatment, or combination treatment: A secondary analysis of a randomized clinical trial. JAMA Network Open, 7:e2416775.

Johnson, B., M. Milstead, M., L. Green, R. Kreider, and R. Jones (2025a). Diet quality and nutrient distribution while using glucagon-like-peptide-1 receptor agonist: A secondary cross-sectional analysis. Obes. Pillars 16:100195.

Johnson, B., M. Milstead, R. Kreider, and R. Jones (2025b). Dietary supplement considerations during glucagon-like Peptide-1 receptor agonist treatment: A narrative review. Obes. Pillars 16:100209.

Johnson, B., M. Milstead, O. Thomas, T. McGlasson, L. Green, R. Kreider, and R. Jones (2025c). Investigating nutrient intake during use of glucagon-like peptide-1 receptor agonist: A cross-sectional study. Front. Nutr. 12:1566498.

Johnson, B. (2026). Fiber supplementation during and after glucagon-like peptide-1 receptor agonists treatment: A perspective on clinical benefits. J. Nutr. 156:4.

Kanter, M.M., S. Aaron, S.N. Austad, A.W. Brown, N.A. Burd, W.W. Campbell, M. Cope, T.A. Davis, J.W. Erdman, M. Freitas, K. Hicks-Roof, S. Klein, W. Kriengsinyos, D.W. Lamming, D.K. Layman, H.J. Leidy, K.C. Maki, R.D. Mattes, S. McNeill, D.R. Moore, S.M. Pasiakos, S.M. Phillips, D. Raubenheimer, W. Reinhardt, N.R. Rodriguez, S. Simpson, H.H. Stein, L.J.C. van Loon, C.M. Weaver, and D.B. Allison (2026). Examining widely held propositions on human dietary protein needs and benefits: A critical review of the science that shapes both the data and our understanding of an essential macronutrient. Crit. Rev. Food Sci. Nutr. 81:35.

Karasawa, T., R.H. Choi, C.A. Meza, S. Rout, M.J. Drummond, A. Chaix, and K. Funai (2025). Unexpected effects of semaglutide on skeletal muscle mass and force-generating capacity in mice. Cell Metab. 37:1619–1620.

Khachaturov, M., and D.G. Goulis (2026). Beyond GLP-1 monotherapy: Novel multi-agonists, amylin analogues, and combination strategies in obesity and type 2 diabetes. Diab. Obes. Metab. 28:5586-5589.

Koceva, A., A. Janež, T. Pečko, and M. Jensterle (2026). Micronutrient deficiencies in the era of second-generation incretin-based therapies for obesity. Nutrients 18:677.

Korus, S., D. Cembrowska-Lech, K. Kłoda, and E. Stachowska (2026). Dietary intake patterns and nutritional adequacy among adults with overweight or obesity treated with GLP-1 or dual GIP/GLP-1 receptor agonists- preliminary study. J. Translat. Med. 24:532

Kushner, R.F., A.M. Chao, and T.A. Wadden (2026). Lifestyle modification and incretin-based therapy for obesity. JAMA 335:2160-2161.

Langer, H.T., N.K. Gilmore, C.M.T. Hayden, J. Roux, B. Bariohay, T. Rouquet, M. Awada, J. Marcotorchino, L. Bournot, E. Nunn, P.M. Titchenell, D. Liskiewicz, T.D. Müller, O. Anyiam, P.J. Atherton, I. Idris, A. Hentschel, A. Roos, N. Haritonow, K. Norman, U. Müller-Werdan, and K. Baar (2026a). Weight loss with GLP-1 medicines does not result in a disproportionate loss of muscle mass or function in obese mice and humans. Cell Rep. Med. 7:102665.

Langer, H.T., A. S. Joshi, U. Müller-Werdan, and K. Norman (2026b). Causes of sarcopenia and frailty in people taking GLP1RAs. Nat. Rev. Endocrinol. 22:383-384.

Lieberman, D.E., D.H. Aslan, and S.B. Heymsfield (2026). The conundrum of exercise for weight management in the GLP-1 receptor agonist era. JAMA 335:1841-1843.

Lim, C., E.A. Nunes, B.S. Currier, J.C. McLeod, A.C.Q. Thomas, and S.M. Phillips (2022). An evidence-based narrative review of mechanisms of resistance exercise-induced human skeletal muscle hypertrophy. Med. Sci. Sports Exerc. 54:1546–1559.

Lundgren, J.R., C. Janus, S.B.K. Jensen, C.R. Juhl, L.M. Olsen, R.M. Christensen, M.S. Svane, T. Bandholm, K.N. Bojsen-Møller, M.B. Blond, J.-E. B. Jensen, B.M. Stallknecht, J.J. Holst, S. Madsbad, and S.S. Torekov (2021). Healthy weight loss maintenance with exercise, liraglutide, or both combined. N. Engl. J. Med. 384:1719–1730.

Martin, C.K., O.T. Carmichael, S. Carnell, R.V. Considine, D.A. Kareken, U. Dydak, R.D. Mattes, D. Scott, S. Shcherbinin, H. Nishiyama, A. Knights, S. Urva, L. Biernat, E. Pratt, A. Haupt, M. Mintun, D. Otero Svaldi, Z. Milicevic, and T. Coskun (2025). Tirzepatide on ingestive behavior in adults with overweight or obesity: a randomized 6-week phase 1 trial. Nat. Med. 31:3141–3150.

Mcleod, J.C., B.S. Currier, C.V. Lowisz, and S.M. Phillips (2024). The influence of resistance exercise training prescription variables on skeletal muscle mass, strength, and physical function in healthy adults: An umbrella review. J. Sport Health Sci. 13:47–60.

Melis, P., M. Lucijanic, B. Kranjcec, M. Cigrovski Berkovic, and S. Marusic. (2025). The effect of semaglutide on intestinal iron absorption in patients with type 2 diabetes mellitus—A pilot study. Diab. Obes. Metab. 27:3542–3545.

Miller, S.L., and R.R. Wolfe (2008). The danger of weight loss in the elderly. J. Nutr. Health Aging, 12:487–491.

Mogna-Peláez, P., and M. Guasch-Ferré (2026). Avoiding malnutrition in the era of glucagon-like peptide-1 medications: Emerging evidence and opportunities for integrated nutrition care. J. Nutr. 156:101684.

Mok, J., M.O. Adeleke, A. Brown, C.G. Magee, C. Firman, C. Makahamadze, F.C. Jassil, P Marvasti, A. Carnemolla, K. Devalia, N. Fakih, M. Elkalaawy, A. Pucci, A. Jenkinson, M. Adamo, R.Z. Omar, R.L. Batterham, and J. Makaronidis (2023). Safety and efficacy of liraglutide, 3.0 mg, once daily vs placebo in patients with poor weight loss following metabolic surgery: The BARI-OPTIMISE randomized clinical trial. JAMA Surg. 158:1003–1011.

Mozaffarian, D., M. Agarwal, M. Aggarwal, L. Alexander, C.M. Apovian, S. Bindlish, J. Bonnet, W.S. Butsch, S. Christensen, E. Gianos, M. Gulati, A. Gupta, D. Horn, R.M. Kane, J. Saluja, D. Sannidhi, S. Fatima Cody, and E.A. Callahan (2025). Nutritional priorities to support glp-1 therapy for obesity: a joint advisory from the american college of lifestyle medicine, the american society for nutrition, the obesity medicine association, and the obesity society. Am. J. Lifestyle Med. Online ahead of print. PMID: 40452753.

Pratley, R.E., D.S. Denham, R. Trivedi, E. Watkins, L. Connery, J. Barnes, D. Yu, J. Hong, C. Simard, K. Umans, L. Liu, G.S. Tirucherai, and J.L. Marantz (2026). Apitegromab for lean mass preservation during tirzepatide-induced weight loss: a randomized, double-blind, placebo-controlled phase 2 trial. Nat. Med. Online Ahead of Print. PMID: 42260100.

Prokopidis, K. (2026). Glucagon-like peptide-1 receptor agonists and muscle strength changes in older adults: Risks beyond muscle mass reductions. Br. J. Pharmacol. Online Ahead of Print. PMID: 41577337.

Qaisar, R., I.U. Khan, A. Rehman, M.S. ur, Iqbal, F. Ahmad, and A. Karim (2026). Semaglutide use is associated with neuromuscular junction degradation in older adults with type II diabetes mellitus. Br. J. Clin. Pharmacol. 92:149–161.

Ren, Q., L. Zhi, and H. Liu (2025). Semaglutide therapy and accelerated sarcopenia in older adults with type 2 diabetes: A 24-month retrospective cohort study. Drug Design

Develop. Ther. 19:5645–5652.

Sardeli, A.V., T.R. Komatsu, M.A. Mori, A.F. Gáspari, and M.P.T. Chacon-Mikahil (2018). Resistance training prevents muscle loss induced by caloric restriction in obese elderly individuals: A systematic review and meta-analysis. Nutrients 10:423.

Sargeant, J.A., J. Henson, J.A. King, T. Yates, K. Khunti, and M.J. Davies (2019). A review of the effects of glucagon-like peptide-1 receptor agonists and sodium-glucose cotransporter 2 inhibitors on lean body mass in humans. Endocrinol. Metab. 34:247–262.

Schoenfeld, B.J., D. Ogborn, and J.W. Krieger (2017). Dose-response relationship between weekly resistance training volume and increases in muscle mass: A systematic review and meta-analysis. J. Sports Sci. 35:1073–1082.

Scott Butsch, W., S. Sulo, A.T. Chang, J.A. Kim, K.W. Kerr, D.R. Williams, R. Hegazi, T. Panchalingam, S. Goates, and S.B Heymsfield (2025). Nutritional deficiencies and muscle loss in adults with type 2 diabetes using GLP-1 receptor agonists: A retrospective observational study. Obes. Pillars 15:100186.

Sievenpiper, J.L., J. Ard, M. Blüher, W. Chen, J.B. Dixon, A. Fitch, L. Gigliotti, K. Khunti, A. Lecube, M.E.J. Lean, B. Mittendorfer, A.F.H. Pfeiffer, D.H. Ryan, T. Vilsbøll, and L.F. Van Gaal (2025). Nutritional and lifestyle supportive care recommendations for management of obesity with GLP-1 - based therapies: An expert consensus statement using a modified Delphi approach. Obes. Pillars 17:100228.

Simancas-Racines, D., M. Campuzano-Donoso, G. Rossetti, L. Cobellis, V. Pilone, F. Fascì-Spurio, D. Jima Gavilanes, C. Soria, C. Reytor-González, and L. Schiavo (2026). Micronutrient risk with GLP-1 receptor and dual incretin agonists in obesity: Mechanistic pathways, clinical signals, and a monitoring framework. Obes. Pillars 19:100920.

Tinsley, G.M., and S.B. Heymsfield (2024). Fundamental body composition principles provide context for fat-free and skeletal muscle loss with GLP-1 RA treatments. J. Endocrine Soc. 8:11.

Tronieri, J.S., K.C. Allison, K. DeRouen, A. Amaro, K.G. Collins, A. Roy, S. Watts, T. Ananna, and T.A. Wadden (2026). Short- and long-term effects of semaglutide 2.4 mg on energy intake, appetite, and food reward: a 60-week, double-blind randomized controlled trial. Am. J. Clin. Nutr. Online Ahead of Print. PMID: 42323166

Villareal, D.T., L. Aguirre, A.B. Gurney, D.L. Waters, D.R. Sinacore, E. Colombo, R. Armamento-Villareal, and C. Qualls (2017). Aerobic or resistance exercise, or both, in dieting obese older adults. New Engl. J. Med. 376:1943–1955.

Wang, D., A. Djalalvandi, C.T. Saed, K.M. Morrison, and G.R. Steinberg (2025). Can muscle avert GLP1R weight plateau and regain? Cell Rep. Med. 6:102308.

West, S., J. Scragg, P. Aveyard, J.L. Oke, L. Willis, S.J.P. Haffner, H. Knight, D. Wang, S. Morrow, L. Heath, S.A. Jebb, and D.A. Koutoukidis (2026). Weight regain after cessation of medication for weight management: systematic review and meta-analysis. Br. Med. J. 392:e085304.

Wilding, J.P.H., R.L. Batterham, S. Calanna, M. Davies, L.F. Van Gaal, I. Lingvay, B.M. McGowan, J. Rosenstock, M.T.D. Tran, T.A. Wadden, S. Wharton, K. Yokote, N. Zeuthen, and R.F. Kushner (2021). Once-weekly semaglutide in adults with overweight or obesity. N. Engl. J. Med. 384:989–1002.

Winzeler, B., I. da Conceição, J. Refardt, C.O. Sailer, G. Dutilh, and M. Christ-Crain (2020). Effects of glucagon-like peptide-1 receptor agonists on fluid intake in healthy volunteers. Endocrine 70:292–298.

World Health Organization. (2025). WHO guideline on the use of glucagon-like peptide-1 (GLP-1) therapies for the treatment of obesity in adults. (Licence: CC BY-NC-SA 3.0 IGO.).

Wu, G. (2016). Dietary protein intake and human health. Food Funct. 7:1251–1265.

Wu, J.S., L.Y. Lin, J.W. Sun, W.T. Yu, F.S. Yuliani, and S.H. Lin (2026). Musculoskeletal outcomes of glucagon-like peptide-1 receptor agonists versus other antiobesity agents in nondiabetic adults. Obesity Online Ahead of Print. PMID: 42225263.