



Why Protecting Muscle Matters More Than the Number on the Scale

Executive summary

The most important question in weight management is not simply “**How much weight was lost?**” but “**What kind of weight was lost?**” A clinically useful weight-loss programme should reduce excess fat while preserving as much lean tissue, strength, metabolic function, and day-to-day physical capacity as possible. That matters because skeletal muscle is a major determinant of insulin sensitivity, functional independence, physical performance, and healthy ageing. When lean tissue falls too quickly, the result can be a lower-quality weight loss outcome, even if the number on the scale looks impressive. [1]

In Ireland, obesity remains a major public-health issue. The CSO’s Irish Health Survey for 2025 reported that **21.5% of people were living with obesity**, and the Department of Health has separately described **56% of adults in Ireland as living with overweight or obesity**. The highest obesity rates in the CSO survey were seen in adults aged 55 to 64 years. An Irish endocrine health-economics analysis presented in 2026 estimated the **direct healthcare cost of overweight and obesity at about €1.1 billion in 2025**, or approximately **3.2% of the healthcare budget**. [2]

GLP-1–based medications, including **semaglutide** and **tirzepatide**, are evidence-based and clinically important tools. European Medicines Agency materials state that semaglutide helps regulate appetite by increasing fullness and reducing hunger and cravings, while tirzepatide acts through combined GIP and GLP-1 pathways, reducing appetite, energy intake, and food cravings. In pivotal studies summarised by the EMA and JAMA, semaglutide and tirzepatide delivered substantial average weight loss when paired with diet, physical activity, and counselling. [3]

The current evidence does **not** support portraying GLP-1 medicines as “bad” for patients. What it does show is that body composition must be monitored carefully. In a 2026 meta-analysis of obesity-dose GLP-1 receptor agonists, absolute lean mass decreased, but lean mass as a **proportion** of total body weight improved because fat loss predominated.

Another 2026 synthesis found that GLP-1 therapy generally produces meaningful weight loss largely through fat reduction, with relative preservation of lean tissue, but modest lean-mass declines can occur especially during larger or faster losses. [4]

That is why the best current clinical message is this: **Weight Loss Medications Are The Tool. The Programme Creates Lasting Results.** Medication can reduce appetite, lower the intensity of food noise, and make change easier to start. But long-term health outcomes depend on what happens alongside the prescription: protein adequacy, resistance exercise, sleep, psychological support, self-monitoring, relapse planning, and ongoing follow-up. The HSE model of care and the latest EASO framework both support long-term, patient-centred, integrated obesity management rather than one-off or purely short-term treatment. [5]

The real clinical goal is not the lightest possible body. It is a healthier body with less excess fat, preserved muscle, better function, and habits that can last. [6]

Key statistics

Indicator	Latest evidence	Why it matters
Adults in Ireland living with obesity	21.5% in the 2025 Irish Health Survey. [7]	Obesity is now common enough that weight-management services need to be routine, not exceptional.
Adults in Ireland living with overweight or obesity	56% in the Department of Health consultation summary for a new national obesity strategy. [8]	The clinical challenge in Ireland is population-scale.
Age group with highest obesity prevalence	Ages 55–64 , with 30.6% of females and 29.3% of males classified as obese in the CSO survey. [9]	This is also an age range when muscle preservation becomes increasingly important.
Estimated direct healthcare cost of overweight and obesity in Ireland	About €1.1 billion in 2025 , or 3.2% of the healthcare budget , in an Irish 2026 endocrine abstract. [10]	The burden is clinical, functional, and economic.
Mean weight loss with semaglutide 2.4 mg	Around 15% at 68 weeks in the EMA summary of pivotal studies. [11]	Confirms semaglutide as an effective treatment tool for appropriate patients.
Mean weight loss with tirzepatide 15 mg	22.5% at 72 weeks in SURMOUNT-1 as summarized in EMA product information. [12]	Confirms tirzepatide’s high efficacy for total body-weight reduction.
Share of weight loss attributable to fat-free mass in pooled	14.9% for diet with or without exercise overall, 22.3% for diet without exercise, 7.7% for diet with	Exercise materially improves the quality of

Indicator	Latest evidence	Why it matters
analyses	exercise, and 33.3% for incretin therapies. [13]	weight loss.
Weight regain after discontinuing semaglutide	About two-thirds of prior weight loss regained within one year in the STEP 1 extension. [14]	Stopping medication without a maintenance strategy is risky.
Average rate of weight regain after stopping weight-loss medication	Around 0.4 kg per month in a 2026 BMJ systematic review and meta-analysis. [15]	Obesity treatment should be planned as chronic care, not a short episode.

These figures frame the central clinical point of this report: **good weight loss is not just about how much weight falls off; it is about whether fat loss outpaces muscle loss, whether strength and metabolic health improve, and whether the patient can maintain the gains.** [16]

Ireland's clinical context

Ireland now has strong national surveillance for obesity, but less complete routine surveillance for muscle health across the whole adult lifespan. The best obesity snapshot comes from the CSO's Irish Health Survey, while much of Ireland's functional and ageing-related muscle-health understanding comes from Irish ageing research such as TILDA, a large nationally representative study of older adults. In practical terms, that means Irish clinicians can describe obesity at population level with confidence, while muscle-health risk often needs to be inferred from age, function, body composition, frailty, disease burden, and lifestyle patterns rather than from a single national sarcopenia dashboard. [17]

That matters because obesity and low muscle health often coexist. The modern clinical problem is not only excess body fat, but also **poor body composition**: too much fat mass, too little high-quality lean tissue, and too little strength or physical reserve. Reviews on sarcopenic obesity link this combination with worse cardiometabolic risk, functional decline, and higher mortality risk, especially in older adults. [18]

The HSE's updated obesity model of care is therefore important. It frames obesity management as **patient-centred, integrated care** with referral pathways, self-management support, scheduled follow-up, and multidisciplinary input across primary, community, and hospital settings. The document also stresses non-stigmatising communication and regular anthropometric assessment in primary care. That approach fits especially well with the challenge of muscle preservation, because muscle cannot be protected by a prescription alone; it usually requires coordinated nutrition, movement, behaviour support, and monitoring over time. [19]

For Irish GPs and referrers, the practical implication is straightforward: **weight-management success should be evaluated using more than BMI and scale weight.**

Waist circumference, strength, symptoms of fatigue, physical performance, dietary adequacy, and where possible body composition all add value. This is especially true for older adults, people with frailty, people with previous weight cycling, and patients with diabetes, cardiovascular disease, sleep apnoea, osteoarthritis, or limited mobility. [20]

Practical takeaway: In the Irish setting, a modern obesity programme should treat adiposity as a chronic disease while also protecting function, mobility, and muscle-related health. [21]

Why muscle matters during weight loss

Skeletal muscle is not simply “extra tissue” carried on the body. It is a metabolically active organ that plays a central role in glucose disposal, insulin sensitivity, movement, physical resilience, and healthy ageing. Reviews in endocrinology and ageing research describe skeletal muscle insulin resistance as a major driver of metabolic dysfunction, while age-related declines in muscle quantity and quality are linked to reduced functional performance and independence. [22]

That is why the phrase “**lean mass loss**” needs careful interpretation. In body-composition studies, lean mass or fat-free mass is a proxy that includes more than skeletal muscle alone. It can include water, organs, and connective tissue, and definitions vary by method. Even so, when lean mass falls substantially during weight loss, it raises legitimate concerns because excessive lean-tissue loss can reduce resting energy expenditure, compromise physical performance, and increase vulnerability to sarcopenia over time. [23]

Weight loss by calorie restriction has always involved some fat-free-mass loss. The question is how much. A 2026 systematic review and meta-analysis comparing weight-loss modalities found that among adults achieving at least 10% total-body-weight loss, **diet with or without exercise** was associated with the smallest pooled decrease in fat-free mass, while **exercise markedly improved the proportion of weight lost as fat rather than fat-free tissue**. Specifically, the proportion of total weight loss attributable to fat-free mass was **22.3% with diet without exercise** but only **7.7% when diet was combined with exercise**. [13]

That finding is clinically powerful. It shows that muscle loss is **not inevitable at the same magnitude** across all weight-loss strategies. How weight is lost matters. Resistance training, adequate protein, and gradual habit-based behaviour change improve the odds that more of the reduction comes from fat mass rather than from metabolically useful tissue. [24]

The biology helps explain why. Whole-body muscle mass reflects the balance between **muscle protein synthesis** and **muscle protein breakdown**. During energy deficit, especially if protein intake is low or physical loading is inadequate, the balance can shift toward catabolism. That risk rises further with ageing, inactivity, illness, or pre-existing frailty. [25]

For patients, the plain-English meaning is simple: **if appetite falls sharply and activity drops at the same time, some of the lost weight may come from the very tissue that helps keep metabolism, strength, and independence intact.** [26]

Practical takeaway: The best weight loss is not the fastest possible weight loss. It is the **highest-quality** weight loss meaning fat loss with preserved strength, function, and lean tissue. [27]

What the latest GLP-1 evidence shows

Wegovy and Mounjaro are both evidence-based anti-obesity medicines, but they are not identical. According to the EMA, **Wegovy** contains semaglutide, a GLP-1 receptor agonist that regulates appetite by increasing fullness and reducing hunger, food intake, and cravings. The EMA summary of pivotal trials reports average weight loss of about **15% after 68 weeks** in a large adult study, with substantially more patients achieving at least 5% weight loss than on placebo. [28]

Mounjaro contains tirzepatide, which the EMA describes as a long-acting **dual GIP and GLP-1 receptor agonist**. The product information states that tirzepatide reduces energy intake and appetite by increasing satiety and fullness and decreasing hunger and cravings. In the SURMOUNT-1 trial summarized in the EMA documentation, the 15 mg dose produced a **22.5% mean reduction in body weight at 72 weeks**, with nearly **63%** of participants achieving at least 20% weight loss, compared with **1.3%** on placebo. [29]

Both medications therefore deserve to be understood as **effective clinical tools**, not as shortcuts or gimmicks. They can reduce appetite enough to make dietary adherence easier, improve the starting conditions for behaviour change, and support clinically meaningful fat loss. The HSE and EASO frameworks are consistent with this interpretation, treating obesity pharmacotherapy as part of long-term personalised care rather than as a stand-alone event. [30]

The most common adverse effects are mainly gastrointestinal. For semaglutide, the EMA lists nausea, vomiting, diarrhoea, constipation, abdominal pain, and headache among the most common side effects. For tirzepatide, the EMA product information similarly highlights nausea, vomiting, and diarrhoea, and notes that gastrointestinal reactions were mostly mild or moderate and tended to decrease over time after the dose-escalation phase. [31]

The key body-composition question is more nuanced. A 2026 International Journal of Obesity meta-analysis of obesity-dose GLP-1 receptor agonists found that treatment **reduced absolute lean mass by about 1.74 kg, yet improved lean mass as a proportion of total body weight by 1.81%**, implying that fat loss remained the dominant component. A second 2026 review found that GLP-1 therapies generally reduce body weight and adiposity while often preserving lean tissue relatively better than headlines might suggest, although modest lean-mass reductions can still occur. [32]

That distinction matters. If a person loses a large amount of fat, their **percentage of body weight that is lean** may improve even if their absolute lean mass falls somewhat. For this reason, it is misleading to discuss “muscle loss” using only one metric. The clinically useful questions are:

How much lean tissue fell in absolute terms?

How much fat mass fell?

What happened to strength and function?

Was the patient eating and training in a muscle-protective way? [33]

The latest pooled analyses suggest that GLP-1–based weight loss often results in roughly **one-quarter to one-third** of total weight loss coming from fat-free mass, although estimates vary by study design, duration, body-composition method, and patient population. One recent review of 40 DXA-based reports put mean fat-free-mass loss at **29.1%** of total weight loss, while the 2026 modality comparison found **33.3%** for incretin-based therapies in pooled analyses. [34]

Emerging prospective evidence also suggests that the story is not uniformly negative. The SEMALEAN study, reported in 2025/2026 sources, found substantial fat-mass reduction during semaglutide therapy, with early lean-mass decline that later stabilised and accompanied improvements in handgrip strength. That supports a more balanced interpretation: **GLP-1 therapy can reduce lean mass, but under structured care it may still improve overall body composition and functional status.** [35]

Practical takeaway: GLP-1 medications should be presented as effective anti-obesity treatments. The clinical challenge is not whether to use them when indicated, but how to use them in a way that protects muscle and supports durable outcomes. [36]

What happens after stopping medication

The evidence on discontinuation is now strong enough to support a clear clinical warning: **stopping anti-obesity medication is a high-risk transition point** unless there is a structured maintenance plan. This is not because the medicines “failed.” It is because obesity is a chronic, biologically defended disease in which appetite, energy intake, and weight-regulation signals frequently shift toward regain after treatment stops. [37]

For semaglutide, the STEP 1 extension remains the best-known withdrawal dataset. One year after stopping semaglutide 2.4 mg, participants regained about **two-thirds of the weight they had previously lost**, and many cardiometabolic improvements moved back toward baseline. The extension authors concluded that the findings reinforce the chronicity of obesity and the need for ongoing treatment to maintain benefit. [14]

For tirzepatide, the SURMOUNT-4 randomized withdrawal trial showed the same broad pattern. After a 36-week open-label lead-in, those switched from tirzepatide to placebo **regained 14% body weight** during the following 52 weeks, whereas those who continued tirzepatide lost a further **5.5%**. In a 2026 JAMA Internal Medicine post hoc analysis, **82%** of participants who stopped tirzepatide regained at least **25% of their initial weight**

reduction within one year, and greater regain was associated with greater reversal of improvements in waist circumference, blood pressure, lipids, glycaemic measures, and insulin resistance. [38]

This is not just a trial phenomenon. A 2026 BMJ systematic review and meta-analysis of medication cessation found an average post-treatment regain of about **0.4 kg per month**, with regain occurring **faster after weight-management medication** than after behavioural weight-management programmes. The same paper projected that cardiometabolic markers would return to baseline within roughly **1.4 years** after cessation. [15]

The implication for practice is important. Patients should not be told, implicitly or explicitly, that medication is only useful if it can be stopped quickly and never revisited. That framing misunderstands both the disease and the evidence. At the same time, patients also should not be led to believe that medication alone is enough to protect long-term health after stopping. The research shows that **continuity of care, maintenance planning, and habit formation matter enormously**. [39]

A balanced interpretation is therefore the most defensible one: GLP-1 treatment creates a valuable therapeutic opportunity by lowering appetite and helping patients achieve clinically meaningful weight loss; however, **if the surrounding programme has not improved eating patterns, strength work, self-monitoring, and relapse management, regain is common**. [40]

Practical takeaway: A discontinuation plan should never mean “stop and hope.” It should mean active maintenance: follow-up, nutrition review, exercise support, appetite-regain planning, and rapid action if weight starts to drift upward. [37]

How to preserve muscle and build lasting results

The evidence on prevention is consistent across weight-loss modalities: **muscle preservation is most likely when medication, nutrition, exercise, and behaviour support are combined**. A 2026 review comparing diet, exercise, incretin therapies, and surgery concluded that strategies to preserve fat-free mass—especially exercise—should be included in all approaches to weight loss. [13]

Resistance training is central. Recent work on “high-quality weight loss” found that resistance training during calorie restriction maximised fat-mass reduction while preserving or even increasing fat-free mass, outperforming no exercise and aerobic exercise alone on body-composition quality. In other words, the **same number of kilograms lost** can represent very different clinical outcomes depending on whether resistance exercise is part of the plan. [41]

Protein adequacy also matters. A 2024 systematic review and meta-analysis of 47 studies found that increased protein intake significantly helped prevent muscle-mass decline in adults with overweight or obesity undergoing weight loss. A separate review focused on incretin-mimetic therapy concluded that high-quality protein and micronutrient adequacy,

sometimes supported by oral supplements, should be part of care when intake is reduced by appetite suppression. [42]

There is no single perfect protein prescription for every patient, but the pattern is clear enough for clinical practice: **very low intake is a problem**, and appetite suppression can make under-eating easier than patients realise. This is especially relevant for older adults, those with previous dieting, those with frailty risk, and patients who feel “full” too quickly on medication. [43]

Exercise and medication appear complementary rather than competitive. A 2026 review of GLP-1 receptor agonists, exercise, and their combination found that **combined GLP-1 therapy and structured exercise improved weight, insulin sensitivity, and selected lipid parameters more than monotherapy or placebo**. Likewise, a 2026 meta-analysis of GLP-1 and incretin therapies delivered within lifestyle interventions found clinically meaningful reductions in body weight when medications were embedded within broader lifestyle programmes. [44]

Behaviour change is what converts short-term physiological help into long-term self-management. A 2026 systematic review of behaviour-change techniques found that the techniques most consistently associated with better long-term outcomes included **goal setting, feedback, self-monitoring, instruction, demonstration, conserving mental resources, and incentives**. Digital self-monitoring has also been shown to support weight loss and behaviour improvement, especially when it reduces patient burden rather than increasing it. [45]

This is where mindset matters, but it should be described scientifically rather than vaguely. “Changing mindset” is not just positive thinking. In obesity care, it usually means: accepting that obesity is chronic rather than a personal failure; learning to respond to lapses without abandoning the whole effort; building routines that persist when motivation fluctuates; and shifting focus from punishment and restriction to consistent self-management. These ideas are consistent with the behaviour-change and relapse-prevention literature, and with modern obesity care that avoids stigma and supports patient autonomy. [46]

Practical takeaway: If medication reduces appetite, the programme must use that window wisely. The best use of that quieter appetite signal is to build protein sufficiency, resistance training, structured meals, self-monitoring, and relapse-resistant habits. [47]

Motivation Weight Management approach and practical guidance

A clinically credible obesity programme in Ireland should now be able to say, without exaggeration, that **medication and behavioural treatment are complementary**. That is the most evidence-aligned way to position a service such as Motivation Weight Management. Current evidence supports the view that anti-obesity medicines can create the physiological conditions that make change easier, while long-term success depends

on how well the programme protects muscle, improves routines, and supports maintenance. [48]

In that context, the strongest wording is not anti-medication and not medication-only. It is this:

Weight Loss Medications Are The Tool. The Programme Creates Lasting Results.

That framing is consistent with current evidence. Wegovy and Mounjaro can reduce appetite and support clinically meaningful fat loss, but high-quality outcomes require expert supervision, nutritional planning, exercise progression, behavioural coaching, and follow-up. The HSE model of care and the EASO 2026 framework both support precisely this kind of long-term, integrated, personalised treatment structure. [49]

A non-promotional, research-backed description of the Motivation philosophy would therefore read as follows:

Motivation Weight Management combines medically supervised GLP-1 treatment, where appropriate, with nutrition education, behaviour change support, a mindset-focused “mental weight” approach, and ongoing follow-up designed to help patients achieve sustainable weight loss while protecting overall health. This model aligns with the strongest current evidence because it recognises that obesity is chronic, medications are useful, and lasting results depend on the daily behaviours that continue when appetite, stress, and life circumstances change. [50]

For patients and referrers, the practical advice is straightforward. During treatment, monitor more than scale weight. Review appetite, nausea, meal frequency, protein intake, resistance exercise, sleep, bowel symptoms, and energy levels. Ask whether the patient is getting **stronger**, not only lighter. If weight is falling but diet quality, strength work, and follow-up are poor, the patient may be losing weight in a way that is less protective of long-term health. That is a clinical inference from the body-composition literature and a reasonable basis for programme design. [51]

For GPs and healthcare professionals, the referral message is equally clear: a worthwhile programme should offer medical assessment, medication suitability review, side-effect monitoring, nutrition support, activity guidance with a resistance component, behaviour-change tools, and a maintenance plan for either ongoing therapy or discontinuation. That reflects HSE and EASO best practice rather than marketing language. [52]

For publication as a lead magnet or GP resource, the most useful accompanying visuals would be these:

- an Ireland obesity snapshot using the latest CSO and Department of Health figures;
- a simple diagram showing **fat loss vs lean-tissue loss** during different weight-loss methods;

- a treatment pathway graphic showing **medication + nutrition + resistance exercise + behaviour support**;
- a withdrawal infographic showing why **maintenance planning after stopping medication** matters;
- a patient-friendly chart titled **“What to monitor besides the scale”**: protein, strength, waist, appetite, energy, habits. [53]

The core conclusion is therefore balanced and evidence-based: **GLP-1 medications are powerful and appropriate treatments for many patients with obesity. But the best long-term results come when medication is embedded in a medically supervised, behaviour-focused, muscle-protective programme of care.** [54]

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