
The Chronicles of
Semawin
WIN
Semaglutide
Not Magic. Metabolic Science

Disclaimer

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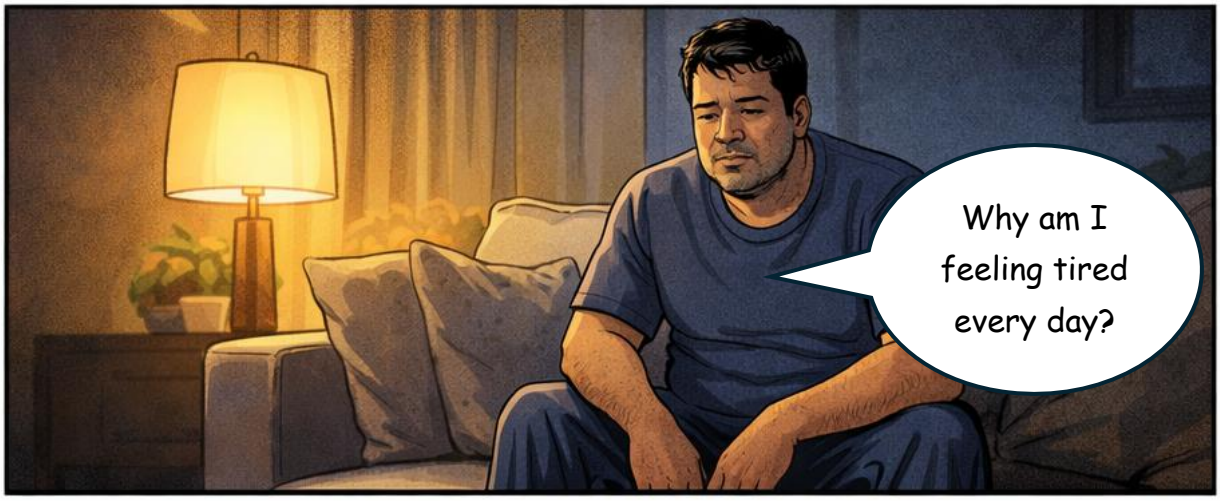
Team Medical Affairs,

Intas Pharmaceuticals Ltd.

Some Wishes are not spoken loudly…

they are whispered in fear!




Magic Pen???
Let me rub it?
Your wish is my command.
WIN
You're...real?
What's your wish?
WIN


I don't want money…
WIN
I just want a healthy and long life.
WIN
Long Life is not a wish. It's a metabolic decision.
And your first enemy …is **OBESITY**.
WIN

What is obesity, and why is it considered a disease?

Obesity is a complex, chronic medical disease defined by abnormal or excessive fat accumulation that poses risks to health, typically classified as a Body Mass Index (BMI) of 30 or higher.

It is not merely a consequence of lifestyle choices but results from complex interactions among genetic predisposition, neurohormonal regulation, environmental influences, and metabolic adaptations. Dysregulation of appetite control mechanisms, energy expenditure, and adipose tissue biology contributes to persistent weight gain and difficulty in maintaining weight loss.

Obesity is associated with systemic inflammation, insulin resistance, and alterations in endocrine function, which collectively increase the risk of multiple comorbidities, including type 2 diabetes, cardiovascular disease, chronic kidney disease, non-alcoholic fatty liver disease, and certain cancers. Recognizing obesity as a disease is essential because it shifts the focus from blame-based approaches to evidence-based medical management.

Prevalence of generalized obesity as well as of abdominal obesity were reported to be 20% and 23.7%, respectively, in India by the INDIAB- ICMR investigators in 2015. Various studies report obesity prevalence ranging from 40% to 60% across different populations in India.

Obesity indicators used to define obesity

Parameter studied	Criteria for overweight/obesity
Body mass index	≥23 kg/m^2 (over weight); ≥25 kg/m^2 (obesity)
Waist circumference	>90 cm in men and >80 cm in women
Waist-hip ratio	0.9 in men and 0.8 in women
Body fat percentage*	>25% in men and >30% in women

* As measured by dual-energy X-ray absorptiometry (DXA)


But I thought, obesity is a sign of being rich!
WIN
Fat cells are toxic factories. Obesity is a chronic disease. Obesity Drives Diabetes, Hypertension, CV disease, Fatty liver and CKD.
Treating Obesity means protecting Organs!

Why is it important to treat obesity actively?

Active treatment of obesity is crucial because it is a major driver of cardiometabolic morbidity and mortality. Excess adiposity contributes to hemodynamic stress, atherogenesis, insulin resistance, and organ dysfunction.

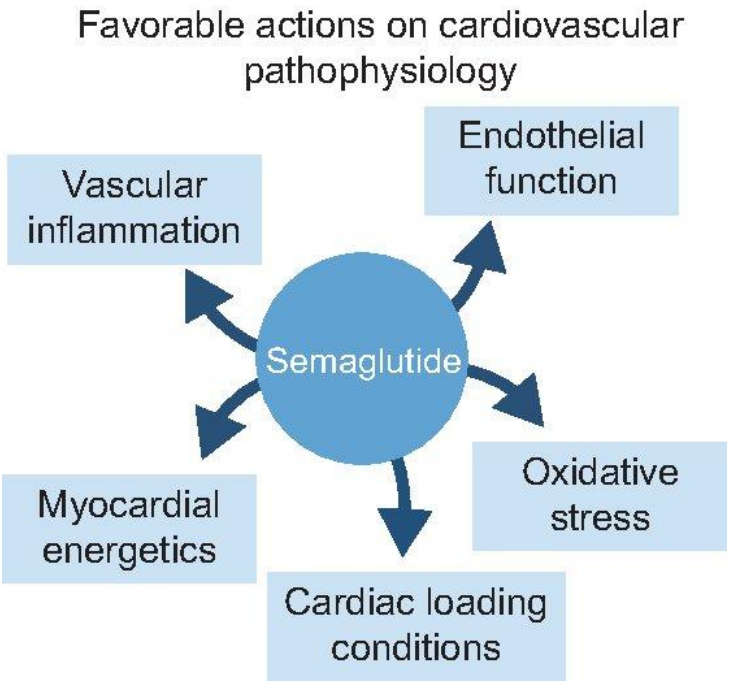
Untreated obesity leads to progressive deterioration of metabolic health, increasing the likelihood of developing complications such as heart failure, myocardial infarction, stroke, and renal impairment.

Furthermore, obesity negatively impacts quality of life, physical functioning, and psychological well-being.

Early and sustained intervention can reduce disease burden, improve metabolic parameters, and decrease long-term healthcare costs. Treating obesity is therefore not solely about weight reduction but about preventing downstream complications and improving overall health outcomes.

Semaglutide provides significant organ protection in individuals with obesity by reducing cardiovascular risks, improving kidney function, and potentially enhancing liver health, often independent of diabetes status. Trials demonstrate it reduces major adverse cardiovascular events (MACE) and preserves kidney function by reducing inflammation and albuminuria. The

SELECT trial showed that 2.4 mg of semaglutide reduced the risk of death from cardiovascular causes, nonfatal heart attacks, and nonfatal strokes by 20% in adults with obesity and established cardiovascular disease.



In addition to weight loss, semaglutide favorably influences several biological pathways that contribute to cardiovascular disease. These include improvements in endothelial function, reductions in vascular inflammation and oxidative stress, enhancement of myocardial energetics, and favorable modulation of cardiac workload


WIN
But I have tried! Diet,
Walking, Skipping dinner.
0:30
WIN
Weight Monster keeps
returning.

Why is lifestyle modification alone often insufficient for many patients?

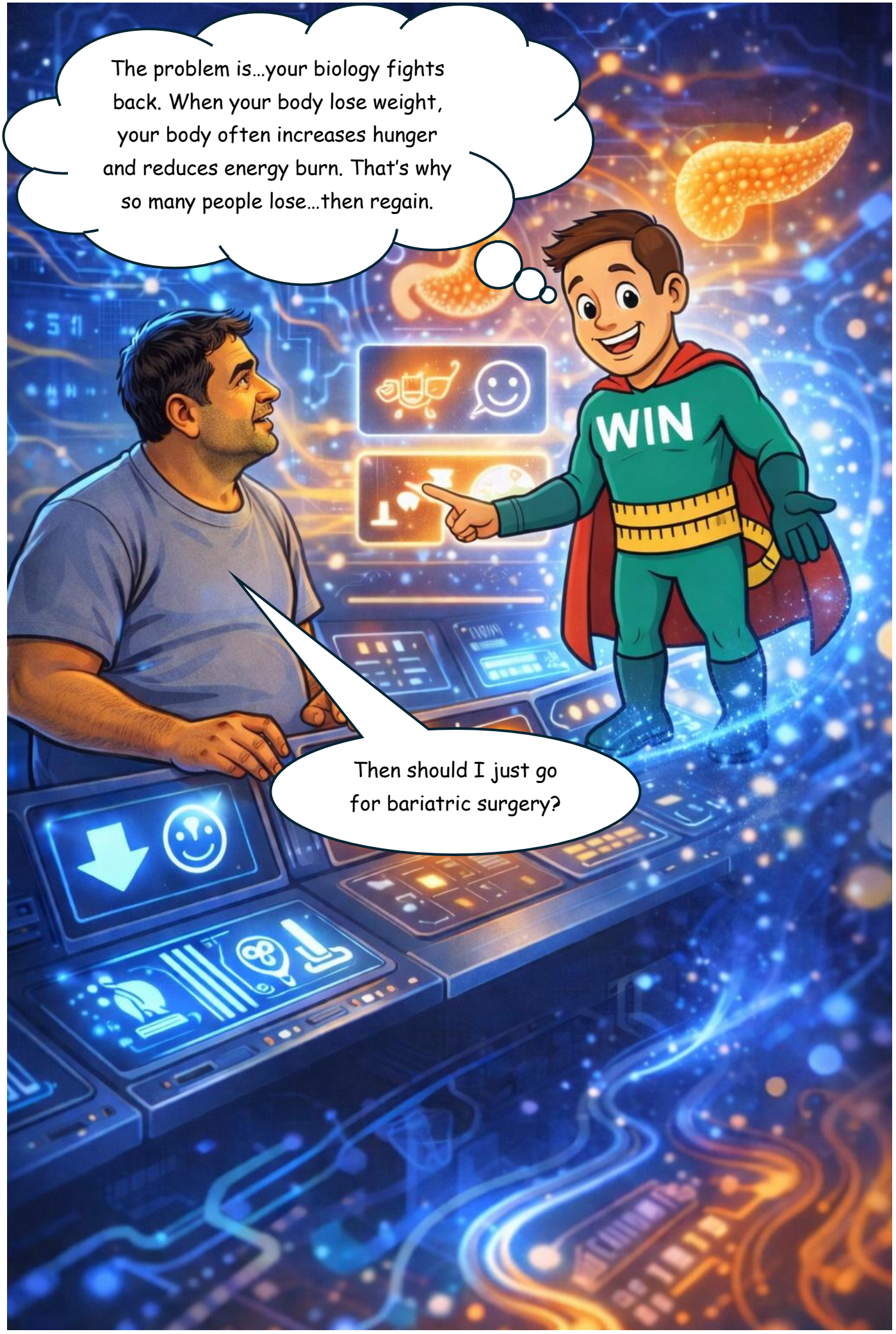
Lifestyle interventions, including dietary modification and physical activity, are foundational components of obesity management; however, they are often insufficient as standalone strategies in many individuals. Large studies including RCTs, on average, show 4–6% weight loss

This is primarily due to biological adaptations that occur during weight loss, including reductions in resting metabolic rate and compensatory increases in hunger mediated by hormonal changes such as decreased leptin and increased ghrelin levels. When we lose weight, we lose fat and muscle. The more muscle we lose, the lower our metabolic rate – the amount of energy the body must burn to achieve homeostasis. These physiological responses favor weight regain and make sustained weight loss challenging.

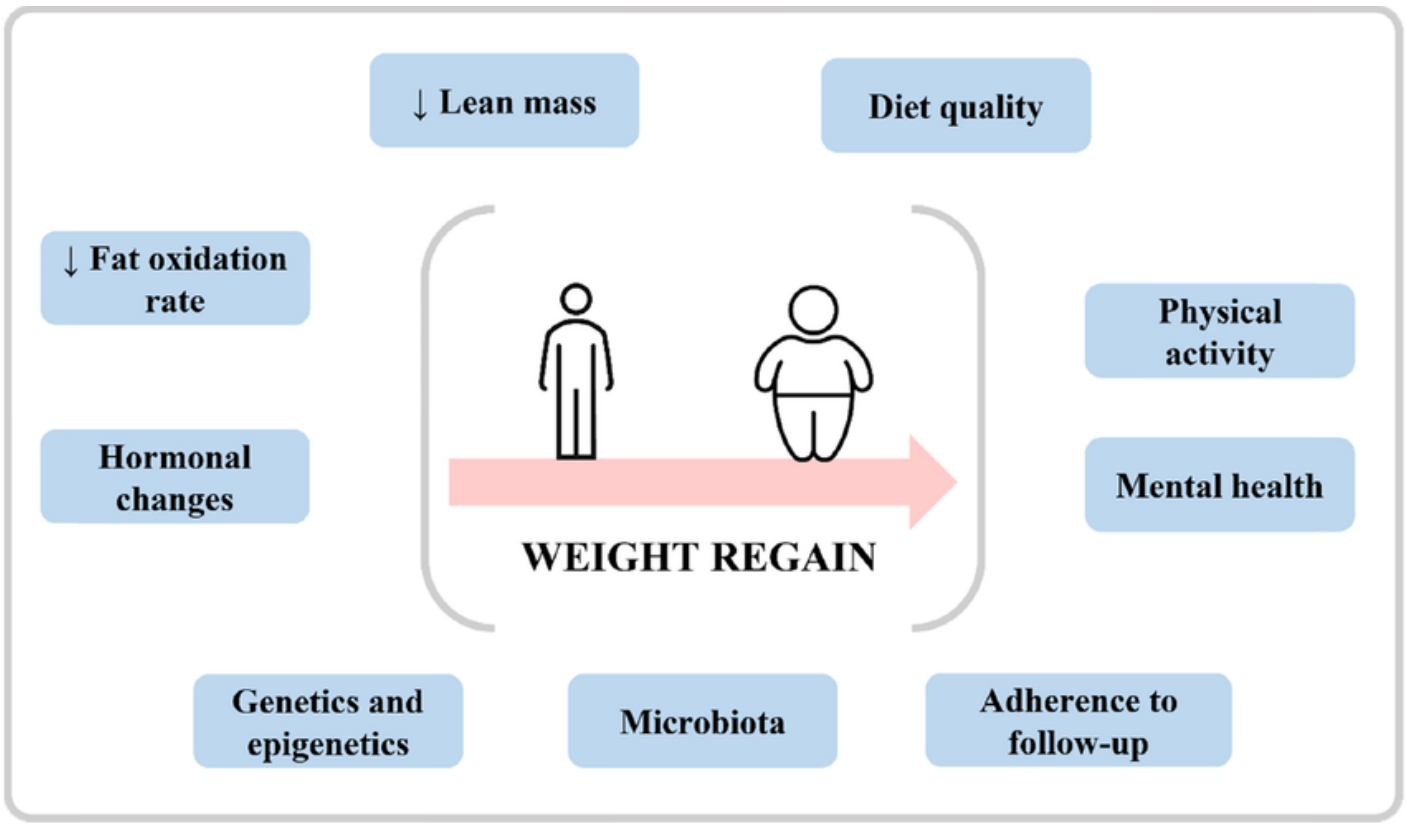
Although calorie restriction can produce short-term metabolic improvements, including improved insulin resistance, it does not modify the underlying neuroendocrine drivers of adipocyte mass regulation; consequently, exclusive reliance on calorie-restriction–centred models may delay the introduction of mechanism-based treatments and allow obesity-related complications to recur or progress over time.

Additionally, genetic susceptibility and environmental factors further limit the effectiveness of lifestyle interventions alone. As a result, many patients require adjunctive pharmacotherapy to achieve and maintain clinically meaningful weight loss. Integrating medication with lifestyle changes addresses both behavioral and biological components of obesity.

Some studies suggest it takes 5-10 years for the body to achieve a new homeostasis with respect to body weight. Effective options are available that can help people achieve and maintain long-term weight wellness – including an ever-growing list of anti-obesity medications (AOMs) that have been proven to support long-term weight maintenance.


The problem is…your biology fights back. When your body lose weight, your body often increases hunger and reduces energy burn. That's why so many people lose…then regain.
WIN
Then should I just go for bariatric surgery?

Factors potentially involved in the weight regain



It is a physiological reality that weight regain can occur across **all weight-loss modalities**, including intensive diet and lifestyle interventions, bariatric surgery, and GLP-1 receptor agonists (RAs). This is primarily due to the body's compensatory metabolic mechanisms that attempt to return to a previous "set point."

However, **Semaglutide** and other GLP-1 RAs are increasingly viewed as the preferred long-term therapeutic option for the following reasons:

- **Ease of Administration:** Unlike daily oral regimens or rigorous lifestyle tracking, the once-weekly subcutaneous injection offers a high level of convenience that supports long-term adherence.
- **Chronic Disease Management Model:** Obesity is a chronic, relapsing condition. Much like hypertension or dyslipidemia, it requires ongoing treatment. GLP-1 RAs provide a sustainable pharmacological bridge to maintain weight loss that lifestyle changes alone often cannot sustain.
- **Sustainability & Tolerability:** Once a patient has successfully titrated to a maintenance dose and side effects (such as nausea) have plateaued, long-term continuation is generally well-tolerated.

Superiority Over Surgery for Some: While bariatric surgery is effective, it is invasive and carries surgical risks. GLP-1 RAs offer a high-efficacy, non-surgical alternative that can be adjusted or maintained over the lifespan of the patient.


Surgery is powerful. But not without surgical complications. **Surgery is not the first or only option for every patient**. Some patients are unwilling, unsuitable or not yet ready.
WIN
Bariatric surgery is indicated for individuals with a BMI 35 kg/m², regardless of comorbidities, or a BMI 30 kg/m² with metabolic disease (e.g., type 2 diabetes, hypertension)
So, what next?
Not every battle needs an operation. Some need metabolic strategy. Go for GLP1 RA.
But Why GLP1 RA (Semaglutide)?

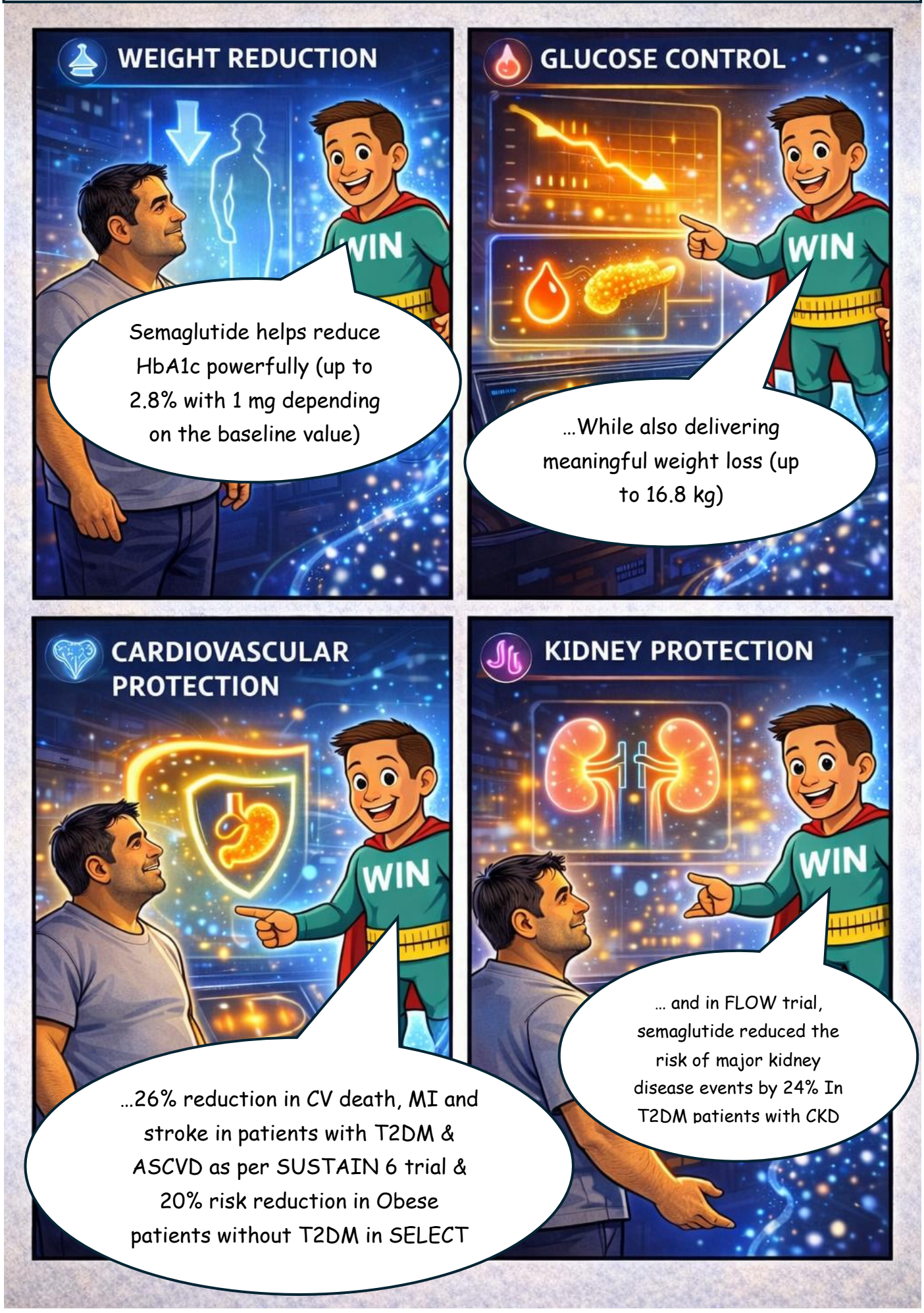
Why is bariatric surgery not considered an early treatment option in obesity?

Bariatric surgery, or weight-loss surgery, comprises procedures that alter the digestive system—typically reducing stomach size and/or limiting nutrient absorption.
Bariatric surgery is an effective and evidence-based intervention for obesity, particularly in individuals with severe obesity or those with significant obesity-related comorbidities who have not achieved adequate outcomes with medical therapy. However, it is generally not considered an early-line treatment for most patients due to its invasive nature, requirement for lifelong nutritional monitoring, and potential for perioperative and long-term complications.

Current clinical practice emphasizes a stepwise approach to obesity management, beginning with lifestyle modification and progressing to pharmacotherapy when necessary, before considering surgical options.

Advances in medical therapy, particularly with agents such as GLP-1 receptor agonists, have demonstrated substantial efficacy in weight reduction and metabolic improvement, thereby offering a less invasive alternative that can be initiated earlier in the disease course.
Additionally, bariatric surgery requires careful patient selection, psychological readiness, and access to specialized multidisciplinary care, which may not be appropriate or necessary for all individuals with obesity. Therefore, while bariatric surgery remains a powerful therapeutic option, it is typically reserved for patients with more advanced disease or those who do not achieve adequate benefit from non-surgical interventions, ensuring that treatment is individualized and proportionate to disease severity.

Semaglutide is indicated for chronic weight management in adults with a BMI of 30 kg/m² or higher (obesity), or 27 kg/m² or higher (overweight) with at least one weight-related comorbidity



What benefits can be expected from semaglutide therapy?

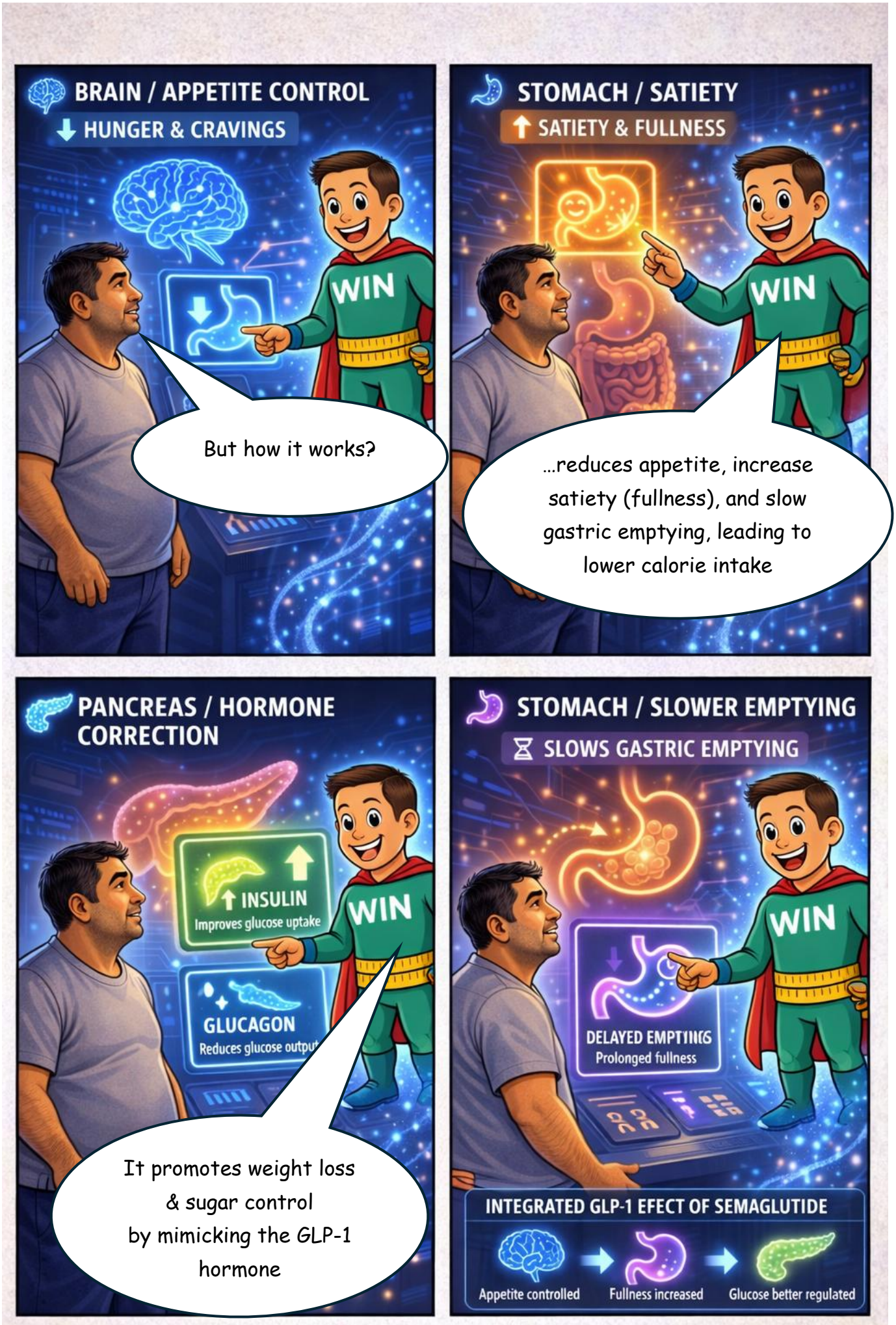
Across the SUSTAIN phase 3 program, once-weekly injectable semaglutide 1.0 mg produced mean HbA1c reductions of ~1.5–1.8% with substantial weight loss over ~30–56 weeks, clearly exceeding the glycemic and weight effects of DPP-4 inhibitors (~0.6–0.8% HbA1c, weight-neutral) and SGLT2 inhibitors (~0.5–1.0% HbA1c, ~2–3 kg weight loss), and avoiding the weight gain and hypoglycemia seen with sulfonylureas. In a subanalysis, semaglutide resulted in a **2.8% reduction in HbA1c from baseline** in patients with baseline HbA1c >9%.

These data position semaglutide as a single agent capable of equitably achieving targets for both glycemia and body weight, in contrast to current first-line drugs, which typically optimize one domain at the expense of the other.

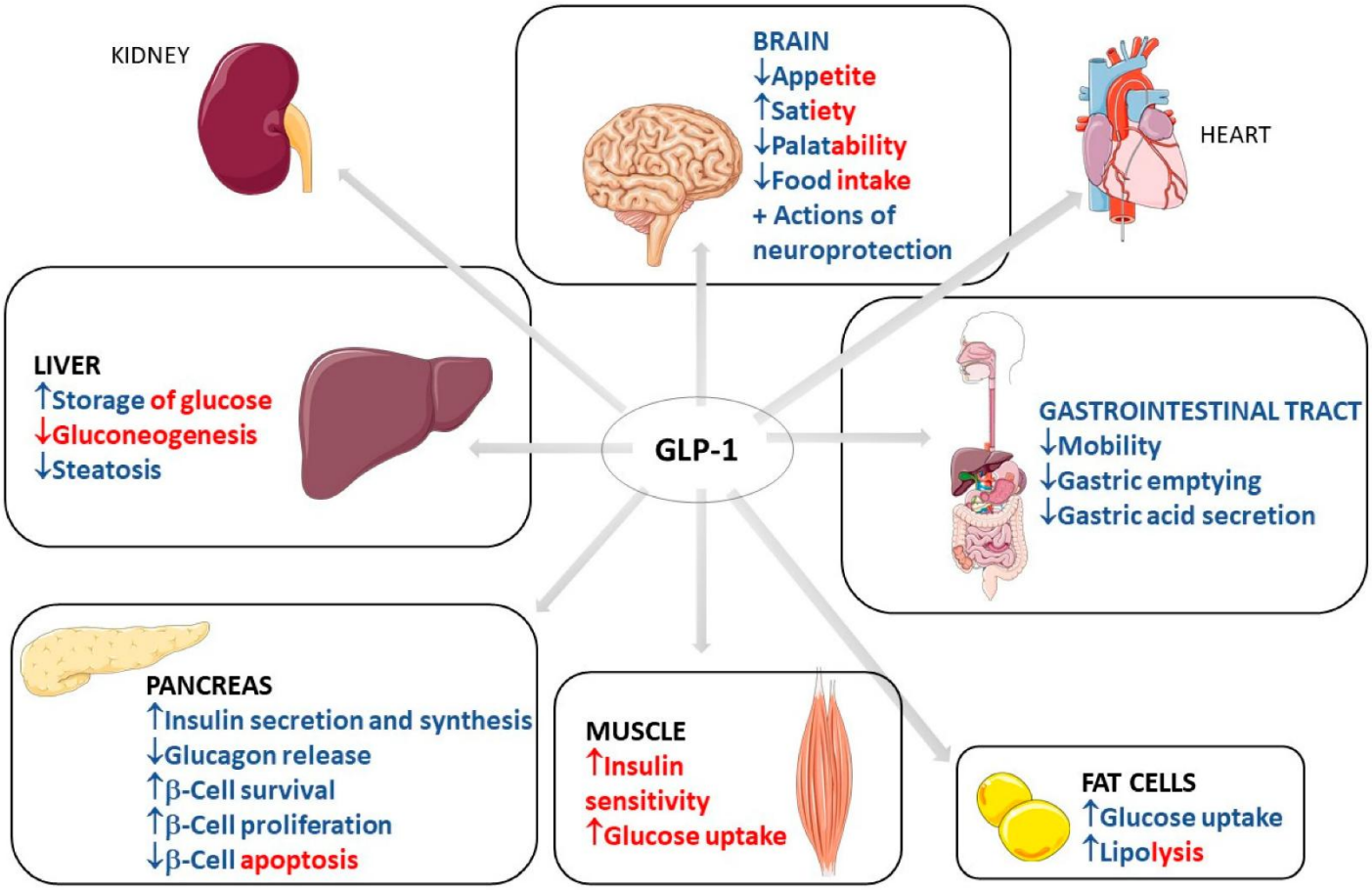
Further, data from the SUSTAIN 6 cardiovascular outcomes trial shows a 26% reduction of 3-point major adverse cardiovascular events in people with established ASCVD or at high risk of ASCVD. The FLOW trial, a dedicated renal outcomes trial with semaglutide, showed a significant 24% risk reduction of renal endpoints. Also, the STRIDE trial has shown a 13% functional improvement in peripheral arterial disease (PAD) outcomes with semaglutide.

Contemporary guidelines position semaglutide ahead of other molecules: ADA/EASD and AACE recommend GLP-1 RAs with proven CV benefit (including semaglutide) as preferred early therapy in people with type 2 diabetes and established ASCVD, high CV risk or obesity, independent of metformin use; ADA standards 2026 also position GLP-1 RAs as the 4th pillar of CKD management (based on FLOW results).

RSSDI guidance similarly highlights GLP-1 RAs in Indians living with obesity/overweight and type 2 diabetes mellitus (T2DM) with added cardiorenal risk. Thus, semaglutide aligns closely with the "ideal" first-line diabesity agent with high glycemic control efficacy, significant weight loss, low hypoglycemia risk, and outcome benefits.


BRAIN / APPETITE CONTROL
HUNGER & CRAVINGS
WIN
But how it works?
STOMACH / SATIETY
SATIETY & FULLNESS
WIN
…reduces appetite, increase satiety (fullness), and slow gastric emptying, leading to lower calorie intake
PANCREAS / HORMONE CORRECTION
INSULIN
Improves glucose uptake
GLUCAGON
Reduces glucose output
WIN
It promotes weight loss & sugar control by mimicking the GLP-1 hormone
STOMACH / SLOWER EMPTYING
SLOWS GASTRIC EMPTYING
DELAYED EMPTYING
Prolonged fullness
WIN
INTEGRATED GLP-1 EFECT OF SEMAGLUTIDE
Appetite controlled
Fullness increased
Glucose better regulated

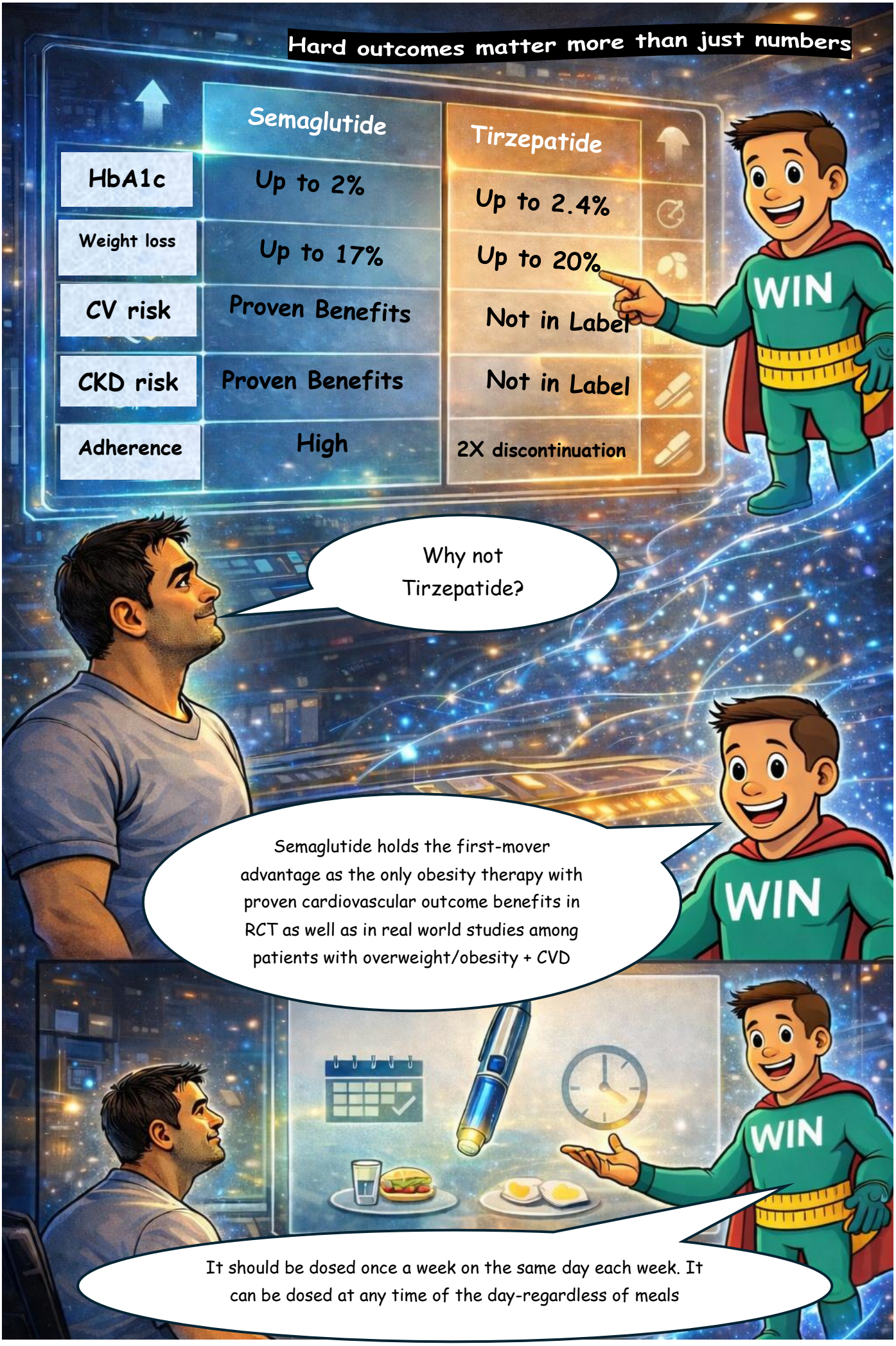
Most important traditional organ targets for GLP-1 and its actions on each target



GLP-1RAs have been shown to increase insulin and decrease glucagon secretion in a glucose-dependent manner, resulting in reduced blood glucose levels. GLP-1RAs also improve multiple pathophysiological defects in T2D beyond glycaemic control and suppress appetite leading to weight loss.

In the central nervous system, particularly within the hypothalamic nuclei responsible for appetite regulation, GLP-1R activation by semaglutide influences neuronal pathways associated with hunger and satiety. This results in reduced appetite, lower caloric intake, and enhanced feelings of fullness, making it effective not only for glycemic regulation but also for weight management. Neuroimaging studies have demonstrated that semaglutide decreases activity in brain regions associated with food craving and reward, providing a mechanistic basis for its appetite-suppressing effects.

Additionally, semaglutide exerts favorable cardiovascular effects, which are thought to result from improvements in endothelial function, reduced inflammation, and decreased oxidative stress. Clinical outcome trials have shown that treatment with semaglutide significantly reduces major adverse cardiovascular events (MACE), along with beneficial changes in body weight, lipid parameters, and systolic blood pressure.


Hard outcomes matter more than just numbers
Semaglutide
Tirzepatide
HbA1c
Up to 2%
Up to 2.4%
Weight loss
Up to 17%
Up to 20%
CV risk
Proven Benefits
Not in Label
CKD risk
Proven Benefits
Not in Label
Adherence
High
2X discontinuation
WIN
Why not Tirzepatide?
Semaglutide holds the first-mover advantage as the only obesity therapy with proven cardiovascular outcome benefits in RCT as well as in real world studies among patients with overweight/obesity + CVD
WIN
WIN
It should be dosed once a week on the same day each week. It can be dosed at any time of the day-regardless of meals

Why not tirzepatide when choosing therapy for obesity and cardiometabolic risk management?

Tirzepatide is an effective incretin-based therapy with strong efficacy in weight reduction; however, current clinical decision-making extends beyond the magnitude of weight loss alone. Contemporary obesity management guidelines emphasize achieving sufficient weight reduction to improve overall health outcomes rather than pursuing the highest percentage of weight loss. This reflects a paradigm shift toward complication-centric care, where the primary goal is reduction in cardiometabolic risk, improvement in quality of life, and prevention or management of obesity-related comorbidities.

In this context, **semaglutide** currently holds a distinct advantage due to its **robust and mature evidence base**. Large randomized controlled trials and real-world studies have demonstrated that **semaglutide significantly reduces major adverse cardiovascular events, including myocardial infarction, stroke, and cardiovascular mortality, even in patients with overweight or obesity without type 2 diabetes**. This positions semaglutide as the only therapy in its class with proven cardiovascular outcome benefits across both randomized and real-world settings in this population. **In contrast, definitive cardiovascular outcome data for tirzepatide in obesity populations are still awaited, with ongoing trials expected to provide clarity in the future.**

Tirzepatide, being a relatively newer agent, has comparatively limited long-term real-world data, which is an important consideration in chronic disease management where durability of effect and safety are critical.

In several obesity-related conditions such as cardiovascular disease, heart failure, chronic kidney disease, and osteoarthritis, semaglutide has demonstrated clinically relevant benefits and is positioned favorably within guideline frameworks. While tirzepatide is also considered a promising option, its positioning in some of these conditions is still evolving as more outcome data become available.

While tirzepatide offers significant weight loss, semaglutide presently provides a more comprehensive and evidence-supported profile, particularly in terms of cardiovascular outcomes, guideline alignment, and long-term clinical experience. Treatment decisions should remain individualized, considering patient characteristics, clinical goals, comorbidities, and the strength of available evidence, ensuring that therapy selection is both scientifically grounded and patient-centric.


Glycemic Control
Weight Loss
WIN
Can I use it if I am having renal impairment?
Yes, it can be safely used in patients with Renal or Hepatic impairment.
CAN BE USED
Kidney
Liver shrinking
WIN
USE WITH CAUTION
Type 1 diabetes / DKA
Pregnancy & Lactation

What are the considerations for using semaglutide in patients with renal and hepatic impairment?

Patients with renal impairment:

No dose adjustment is required for patients with mild or moderate renal impairment.

Experience with the use of semaglutide in patients with severe renal impairment is limited.

Semaglutide is not recommended for use in patients with severe renal impairment (eGFR <30 mL/min/1.73m2) including patients with end-stage renal disease.

Patients with hepatic impairment:

No dose adjustment is required for patients with mild or moderate hepatic impairment.

Experience with the use of semaglutide in patients with severe hepatic impairment is limited.

Semaglutide is not recommended for use in patients with severe hepatic impairment and should be used cautiously in patients with mild or moderate hepatic impairment.

Contraindications:

Hypersensitivity to the active substance or to any of the excipients.

A personal or family history of medullary thyroid carcinoma (MTC) or in patients with Multiple Endocrine Neoplasia syndrome type 2 (MEN 2)

Pregnant Women:

Studies in animals have shown reproductive toxicity. There are limited data from the use of semaglutide in pregnant women. Therefore, semaglutide should not be used during pregnancy. If a patient wishes to become pregnant, or pregnancy occurs, semaglutide should be discontinued. Semaglutide should be discontinued at least 2 months before a planned pregnancy due to the long half-life.

Lactating Women:

In lactating rats, semaglutide was excreted in milk. A risk to a breast-fed child cannot be excluded. Semaglutide should not be used during breast-feeding.


What about Safety?
WIN
Most common adverse effects are gastrointestinal. Mostly mild. Temporary.
WIN
It can be mitigated by few easy steps. "Eat Smaller Meal"
WIN
Stop eating when full or when not hungry. Avoid high fat, fried and spicy foods. Avoid carbonated and alcoholic beverages.
WIN
Drink plenty of water and eat high fibre foods.

What are the common side effects of semaglutide, and how can they be managed?

The most commonly reported adverse effects of semaglutide are gastrointestinal in nature, including nausea, vomiting, and a sensation of fullness. These effects are generally mild to moderate, transient, and tend to diminish over time as the body adapts to the medication.

Gradual dose escalation, consuming smaller meals, and avoiding high-fat foods can help mitigate these symptoms. Most patients are able to continue therapy with appropriate counseling and supportive measures. Severe adverse events are uncommon when the medication is used appropriately.

Patient Counselling

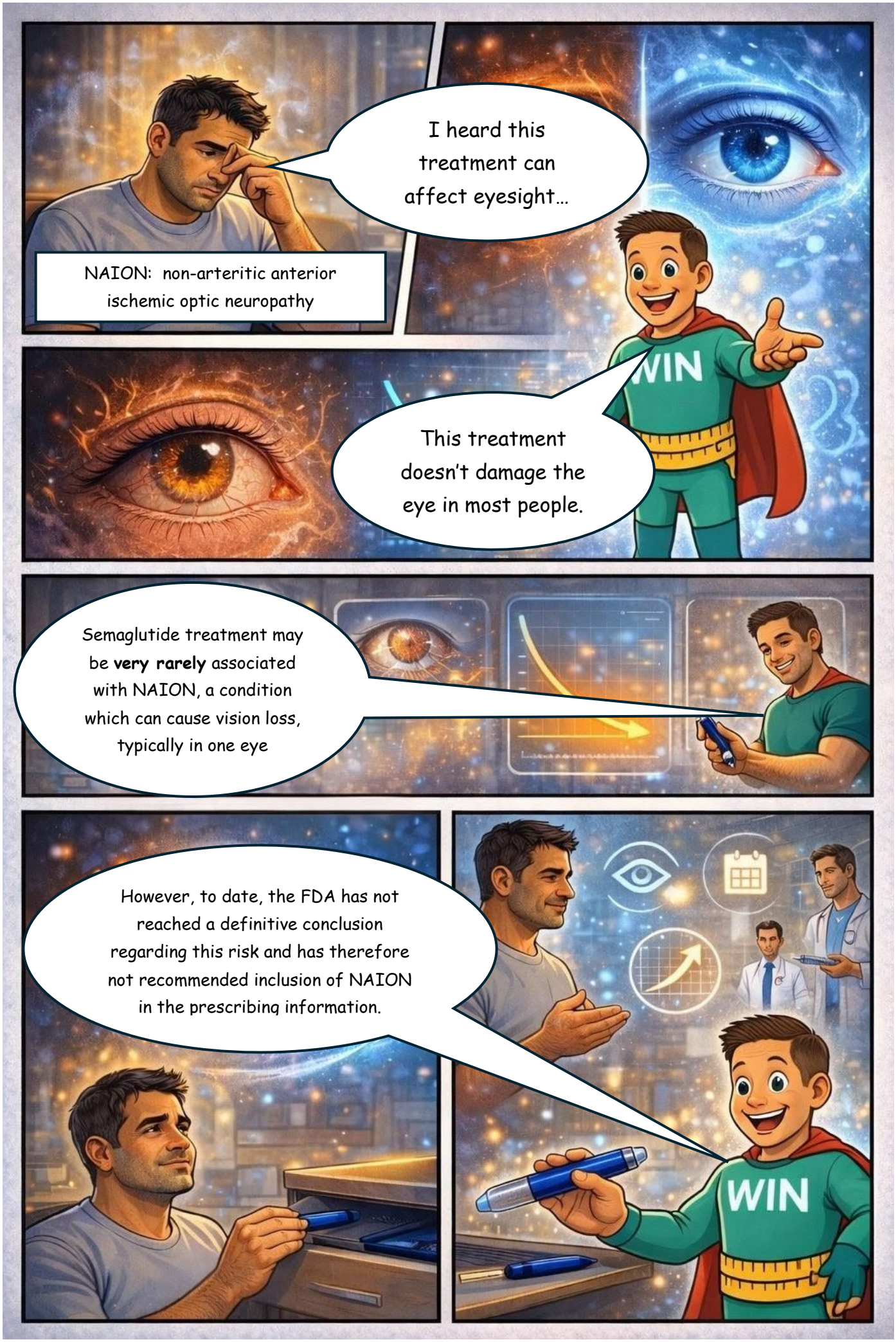
- Patients should be proactively informed about the possibility of gastrointestinal and other adverse events prior to therapy initiation.
- Communication should adopt a reassuring and non-threatening tone to improve acceptance and adherence.
- It is important to emphasize that most adverse events are mild, transient, and self-limiting, particularly during the dose-escalation phase.
- Patients should be reassured that these events are manageable with simple supportive measures and rarely necessitate discontinuation.

Supportive Dietary Measures

- Traditional remedies such as ginger, liquorice, cloves, and fennel seeds may help alleviate nausea.
- Cold-based options, including ice cubes or ice candies, can provide symptomatic relief.
- Suggest intake of non-carbonated fluids (e.g., buttermilk, kokum juice) in small quantities to maintain hydration while minimizing discomfort.

Corrective (Symptomatic) Management:

- For nausea and vomiting, consider antiemetics
- Dyspeptic symptoms may be managed with proton pump inhibitors
- For diarrhea, prebiotics and probiotics may be beneficial.
- Constipation may be addressed with prokinetic agents or selective agents such as prucalopride when appropriate.


I heard this treatment can affect eyesight…
NAION: non-arteritic anterior ischemic optic neuropathy
WIN
This treatment doesn't damage the eye in most people.
Semaglutide treatment may be **very rarely** associated with NAION, a condition which can cause vision loss, typically in one eye
However, to date, the FDA has not reached a definitive conclusion regarding this risk and has therefore not recommended inclusion of NAION in the prescribing information.
WIN

Semaglutide and vision loss: A new concern for NAION risk?

Non-arteritic anterior ischemic optic neuropathy (NAION) is a leading cause of vision loss in adults and the second most common optic neuropathy after glaucoma. It typically presents as sudden, painless, monocular vision loss accompanied by optic disc edema. The vision loss is generally irreversible, and there is currently no effective treatment available.

Semaglutide treatment may be very rarely associated with NAION, a condition which can cause vision loss, typically in one eye, potentially affecting up to 1 in 10,000 users. In contrast, some studies have found no significant association. Abbass, et al. conducted a large, matched cohort study using TriNetX, evaluating over 120,000 patients with type 2 diabetes and 58,000 overweight patients prescribed semaglutide. Across all groups, they found no increased five-year risk of NAION or ischemic optic neuropathy. Similarly, Klonoff, et al. conducted seven real-world cohort analyses across institutional and national datasets; while early signals were observed, they disappeared after adjusting for comorbidities and healthcare utilisation.

Should clinicians be concerned? The short answer is not yet but stay alert. While some large-scale studies suggest a delayed yet modest increase in risk of NAION among semaglutide users, these findings remain observational and must be carefully weighed against semaglutide's clear benefits in reducing cardiovascular and renal morbidity and mortality.

Closer monitoring may be warranted in patients with known risk factors – such as prior NAION or poorly controlled vascular disease. If patients experience a sudden loss of vision or rapidly worsening eyesight during treatment with semaglutide, they should contact their doctor without delay. If NAION is confirmed, treatment with semaglutide should be stopped.


Okay! What if I lose muscle instead fat? Will I become weak?
WIN
WIN
Weight loss can include small changes in muscle…most of the weight you lose is fat. That's the primary goal! So, it's not major muscle loss.
WIN
WIN
Muscle loss is more likely if: you don't eat enough protein, stay inactive or lose weight too quickly
WIN
You can also consult a dietician to create a structured diet plan to mitigate risk.

Does semaglutide cause muscle loss, and how clinically significant is it?

Weight reduction achieved with semaglutide is primarily driven by a decrease in adipose tissue, although a modest reduction in lean body mass, including skeletal muscle, can occur as part of overall weight loss. **This phenomenon is not unique to semaglutide and is observed with most weight-loss interventions, including lifestyle modification and bariatric surgery**.

Changes in body composition during weight loss reflect a physiological adaptation in which both fat mass and lean mass are reduced, but clinical trial data consistently demonstrate that the majority of weight loss with semaglutide is attributable to fat reduction rather than muscle loss.The extent of lean mass reduction is influenced by multiple factors, including baseline nutritional status, rate of weight loss, physical activity levels, and protein intake.

Rapid and unstructured weight loss, particularly in the absence of adequate dietary protein and resistance exercise, may increase the proportion of lean mass loss. However, when semaglutide therapy is combined with appropriate lifestyle measures, including sufficient protein consumption and regular resistance training, preservation of skeletal muscle mass can be optimized.

Importantly, current evidence does not suggest that semaglutide causes disproportionate or clinically harmful muscle loss when used appropriately. In the SEMALEAN study, patients treated with semaglutide 2.4 mg achieved substantial weight loss of approximately 10% at 7 months and 13% at 12 months, with a marked reduction in fat mass of up to 18%, confirming that fat loss is the dominant component of weight reduction. Although a modest decline in lean mass was observed initially, this reduction stabilized over time and did not translate into functional impairment. Importantly, muscle function, assessed using handgrip strength, improved significantly during treatment, demonstrating that muscle quality and performance were preserved or even enhanced despite weight loss.

With semaglutide, patients often experience enhanced mobility, improved insulin sensitivity, and better overall physical performance as excess adiposity is reduced. From a clinical perspective, the goal is not only weight reduction but also improvement in the ratio of fat mass to lean mass, which contributes to better long-term health outcomes.

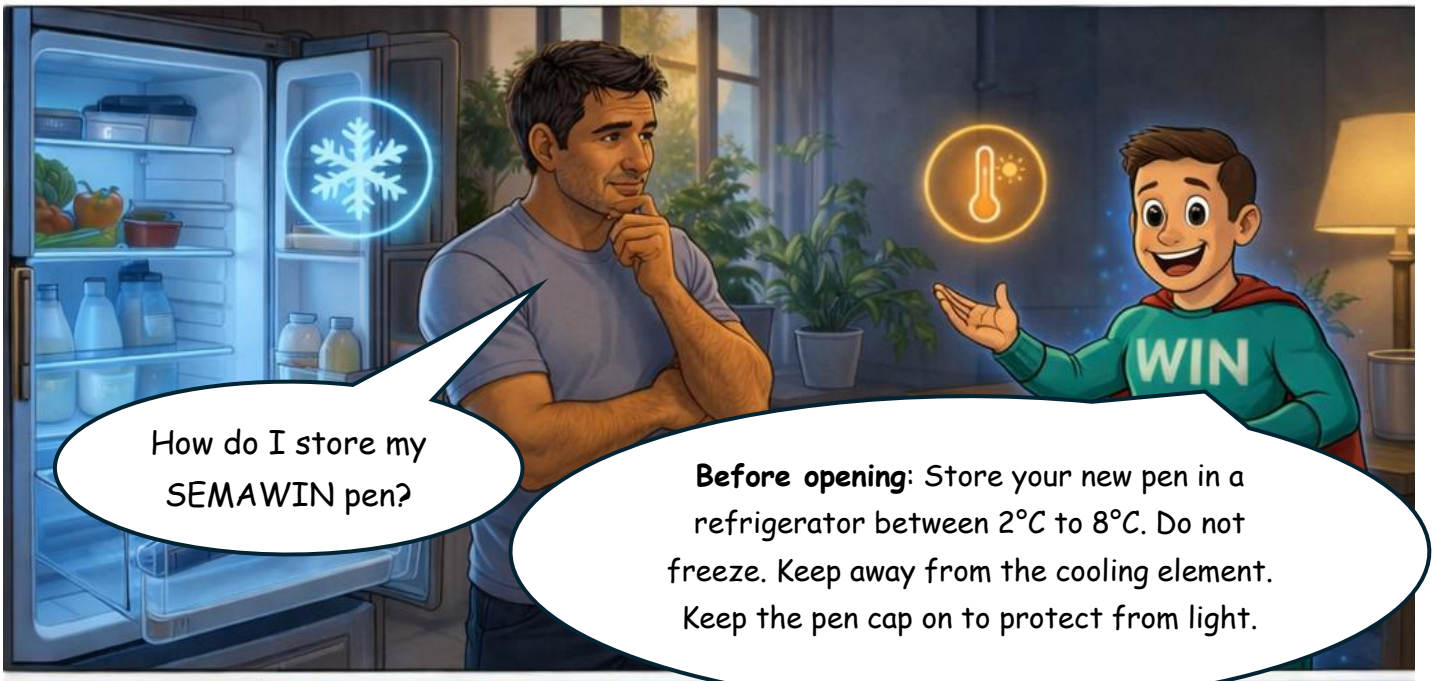
In summary, while a small degree of lean mass reduction may occur during weight loss with semaglutide, it is generally limited and manageable. With appropriate nutritional support and incorporation of resistance exercise, muscle mass can be preserved effectively, ensuring that weight loss is both healthy and sustainable.


Okay! So how should I take this? Hopefully, this pen isn't a complicated torture device!
WIN
Relax. Simple Technique. Smart Consistency. This is what this pen is going to offer!
Scan QR code for More Details
Read the Instruction carefully before using your SEMAWIN pen. Also, check video on how to use SEMAWIN pen.
Hand Washing
Soap & Water
Ask your doctor
Do not share your pen
New needle for each inj.

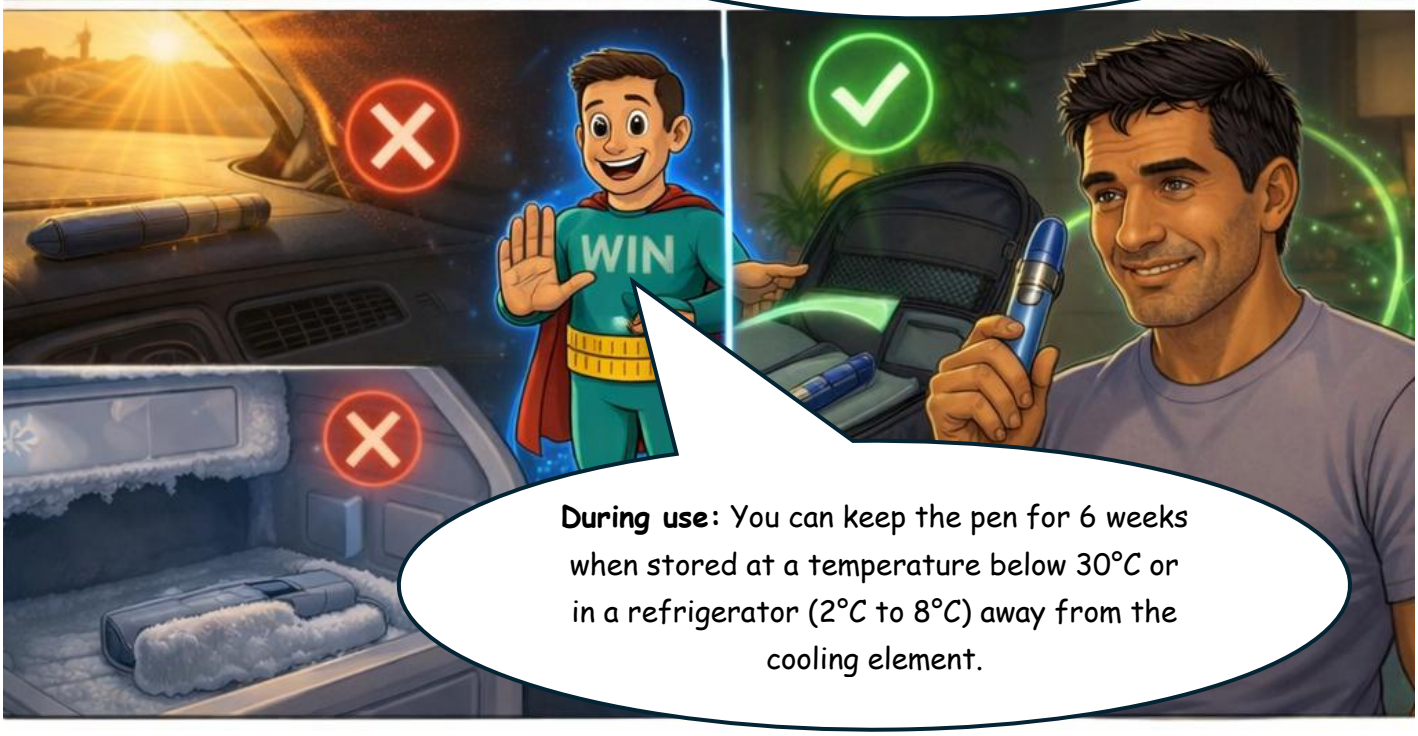
(In a whisper) Let me ask my favorite question?

No Adjustment is required in dose of Metformin, Pioglitazone and SGLT2i when GLP1 RA used concomitant. However, you need to reduce the dose of Insulin and Sulfonylurea. You can not use DPP4 inhibitor together with Semaglutide!


What if I forget the one weekly dose?
WIN
If a dose is missed for fewer than 5 days…
WIN
WIN
WIN
…Administer the missed dose immediately and inject next dose as usual
If 5 days or more…Skip the missed dose. Inject Next dose as usual
WIN


How do I store my SEMAWIN pen?
WIN
Before opening: Store your new pen in a refrigerator between 2°C to 8°C. Do not freeze. Keep away from the cooling element. Keep the pen cap on to protect from light.


WIN
When you travel: Carry in cabin baggage or a handbag and avoid freezing. Avoid storing in a car or glove box as heat may lower its potency!


WIN
During use: You can keep the pen for 6 weeks when stored at a temperature below 30°C or in a refrigerator (2°C to 8°C) away from the cooling element.

You're welcome! Next time, don't rub the pen—just write me at mithilesh_nayak@intaspharma.com

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