macklin METHOD

THE MACKLIN METHOD · MEDICATION

Rx

Obesity medication can be part of treatment.

Earlier modules describe excess weight as a real, genetically influenced, brain-centred, progressive, chronic condition. Behavioural treatment remains foundational. Obesity medications are now an important second treatment pillar, and bariatric surgery remains an important third pillar.

Why medication?

Earlier modules have described overweight and obesity as a real, genetically influenced, brain-centred, progressive, chronic disease. They have also shown that effective treatment is not limited to willpower or lifestyle advice alone.

Behavioural treatment remains a foundational treatment pillar. Obesity medications are now an important second pillar, and bariatric surgery remains an important third pillar. **Diet and exercise are not pillars of treatment in this framework — they are outcomes of treatment, as developed in the module on diet, exercise, and calories.** The focus of this page is obesity medication.

Medical therapy has advanced substantially. Newer incretin-based therapies have produced greater average weight loss and broader health benefits than earlier medications. Semaglutide has also shown cardiovascular benefit in selected populations. At the same time, bariatric surgery remains a highly effective treatment option, especially for severe obesity.

> *Modern obesity care should present medication and surgery as complementary tools within chronic disease treatment, not as competing ideologies.*

What medications do

Obesity medications work in different ways. Some, such as orlistat, work mainly in the gut. Others act more directly on the biology of appetite, hunger, fullness, and food reward. This includes naltrexone/bupropion, liraglutide 3.0 mg, semaglutide 2.4 mg, and tirzepatide.

CURRENT CANADIAN OPTIONS

Liraglutide, naltrexone/bupropion, orlistat, semaglutide, tirzepatide, and setmelanotide for specific rare genetic obesities.

Liraglutide 3.0 mg and semaglutide 2.4 mg are GLP-1-based medications that increase fullness and reduce hunger. Tirzepatide works through both GIP and GLP-1 pathways and also increases fullness while decreasing hunger and calorie intake. In practical terms, these medications help quiet the biological pressures that defend against weight loss and push appetite upward.

The trials and labels describe these effects as increased satiety and decreased hunger. Current thinking, though, places the effect less on the stomach and more on wanting: the medications create less interest in food. This is the diminution of wanting from the Wanting module — and it's often what people are really describing when they say they feel full sooner.

The central role of **WANTING** has been established in the Wanting module. Wanting drives overeating and can make weight management much harder. Naltrexone/bupropion acts on appetite and food reward pathways, and newer incretin-based medications such as semaglutide and tirzepatide also appear to reduce hunger, food preoccupation, and cravings in many people.

The key message is that obesity medications can influence the brain systems involved in fullness, hunger, and food reward. This is why medication can be a powerful addition to behavioural treatment: it can help reduce the biological and psychological pull toward overeating while you continue to build skills, routines, and restraint.

A useful reframe: these are not weight loss medications. They are **manage-the-symptom-between-you-and-your-best-weight medications.** The symptom is wanting, and the medication treats it directly. Weight loss follows because you can now live at a calorie intake your body will let you sustain — landing at a best weight that would otherwise be defended by rising appetite and a slowing metabolic rate.

Outcomes

Clinical trials are conducted to determine the safety, tolerability, and effectiveness of obesity medications. Previously, expected outcomes with medication were more modest. Earlier medications helped many people achieve modest-to-moderate weight loss. Newer incretin-based medications have moved average outcomes into a new range and have reshaped expectations for what medical obesity treatment can achieve.

STEP 1

Semaglutide 2.4 mg

Semaglutide 2.4 mg combined with lifestyle intervention produced an average weight loss of about 14.9% at 68 weeks.

SURMOUNT-1

Tirzepatide

Tirzepatide produced average weight loss ranging from about 15.0% to 20.9% at 72 weeks, depending on dose.

Dose-by-dose averages

The STEP 1 and SURMOUNT-1 figures above are for each trial's maintenance dose — but you'll spend months on the lower doses first, and those have their own averages. For semaglutide: about 4–5% at 0.5 mg, 8% at 1.0 mg, 11–12% at 1.7 mg, and around 15% at the 2.4 mg maintenance dose. For tirzepatide: about 15% at 5 mg, 19–20% at 10 mg, and 21% at 15 mg.

These are real-world numbers — they already fold in missed doses, stressful stretches, and other medications that can nudge appetite or weight up. So imperfect use doesn't put you below average; it puts you inside the group the average describes. If you know your highest-ever weight, these percentages give a rough sense of where a dose might bring you, measured down from that peak — the weight your body defended hardest. Treat it as orientation, not a target: where you land is set by your biology.

Not every person responds to every medication in the same way. Some people notice an early and powerful reduction in appetite, cravings, and food noise, while others have a smaller response or find a given medication difficult to tolerate. A limited response to one medication does not mean that another medication will not work well for the same person.

Treatment with obesity medications is also associated with improvements in weight-related health conditions such as blood pressure, blood sugar, cholesterol, physical function, and quality of life.

Who should consider medication?

Many people living with overweight or obesity have internalized shame, blame, and the false idea that this condition is simply a matter of willpower. A more accurate and medically grounded view is that obesity is a complex, chronic, biologically regulated disease that is influenced by genetics, physiology, environment, and life experience.

In that framework, medication is not a shortcut or a failure. It is one evidence-based treatment option for a real chronic disease. The decision to use medication remains yours, and the role of the clinician is to explain the potential benefits, risks, alternatives, and appropriateness of treatment for your situation.

Obesity medications work best when they are part of comprehensive long-term care. That care may include behavioural treatment, nutrition support, physical activity, sleep optimization, and psychological support where appropriate.

INDICATIONS

In Canadian obesity care, pharmacotherapy should generally be considered for adults with a BMI of 30 kg/m² or above, or a BMI of 27 kg/m² or above

CONTRAINDICATIONS

Each medication has its own contraindications, cautions, and monitoring requirements. These must be reviewed carefully with the prescribing clinician.

in the presence of at least one adiposity-related complication.

Examples include hypertension, type 2 diabetes, dyslipidemia, obstructive sleep apnea, osteoarthritis, or other clinically important weight-related health problems.

The safest and most appropriate choice depends on the medication, medical history, concurrent medications, treatment goals, preferences, affordability, and access.

When within treatment?

Behavioural treatment remains a foundational pillar of obesity care, but modern obesity management is best approached as chronic disease care rather than a step-by-step test of willpower. Medication can be considered at different points depending on health status, treatment goals, response to behavioural care, and preference.

Option 1

Begin with behavioural treatment alone and do not use obesity medication at this time. This may be reasonable if health goals are being met, complications are limited, or the person prefers to begin without medication.

Option 2

Start medication at the same time as behavioural treatment begins. This can be reasonable when earlier improvement in health, appetite control, weight-related complications, or quality of life is important.

Option 3

Add medication after behavioural treatment has begun if progress is limited, weight-related complications remain active, or appetite biology continues to make change very difficult.

Option 4

Add medication when behavioural treatment alone has brought someone to their **BEST WEIGHT**, but health, function, or quality of life are still not where they need to be.

The timing of medication initiation should be discussed with a qualified clinician and decided through shared decision-making. Once started, medication is generally titrated as needed and tolerated, continued long term if helpful, and reassessed over time.

How to initiate medication

Initiating obesity medication begins with a careful discussion of how the medication works, what benefits are being targeted, what side effects or contraindications need to be reviewed, and whether treatment fits the patient's goals, preferences, and circumstances.

For many modern medications, treatment starts at a lower dose and is increased gradually to improve tolerability. For example, semaglutide is typically escalated stepwise to a maintenance dose, and tirzepatide is also started at a low weekly dose and increased gradually as needed and tolerated.

Because obesity is a chronic disease, medication should usually be framed as part of long-term treatment rather than a short-term fix. The same logic is familiar from other chronic conditions: blood pressure medication manages hypertension rather than curing it, diabetes medication manages diabetes, thyroid replacement supplies what the thyroid no longer makes. Stop the treatment and the condition returns. Obesity medication works the same way — when it's stopped, the brain's defence against fat loss reasserts itself, appetite rises, and weight regain often follows. When effective and tolerated, pharmacotherapy is generally continued long term to help maintain benefits and reduce the risk of weight regain or regression of health improvements.

Follow-up should focus on more than the number on the scale. Assessment should include appetite response, hunger and craving control, tolerability, adherence, weight or waist-related progress if the patient wishes to track it, and improvement in obesity-related health complications, physical function, and quality of life.

Appetite during dose escalation, and after

Starting medication drops your appetite sharply, and you lose weight. Appetite then slowly rises — until your next dose increase brings it back down. The pattern repeats through the schedule. On slower schedules, many people feel this rhythm directly: appetite creeping up in the days before the next increase, then settling. On the faster schedules in the product monograph, the increases may come too quickly to notice the rise in between. While you're still moving up through the doses, your intake may keep dropping, or at least hold steady.

Once the increases stop — at the maintenance dose, or wherever you stop — appetite begins a slow, steady climb, because your body is sensing the loss of fat. Intake rises until it matches your energy needs, which are now lower than before. That is the plateau on medication: typically around twelve to fifteen months, and stretching longer with newer medications — some still in development may not plateau until past 100 weeks. The Expectations module covers this in depth.

Through the whole arc, your effort stays constant. The gap between what your brain asks for and what you eat is about the same in the first weeks as at the plateau. What changes is appetite and intake rising together — which is why weight loss slows — not your effort.

Owning your dose, and the appetite signal

You live this rhythm from the inside, and that gives you real authority over it. In this method you're invited to become — always with your clinician's agreement — the owner of your dose. Not by adjusting the medication yourself: the prescriber still decides. Owning your dose means arriving able to describe, in very specific terms, the reasons for a dose change.

The signal to listen for is a subtle rise in appetite — your brain fighting back as you lose fat. A building pull toward food: subconscious, hard to put your finger on, but there. You are the one who feels it, so you're the best judge of when it's present. It shows up first in your high-risk times, because that's where the conditioning lives and where your brain's defence is most active — exactly the rise the medication exists to defend against. (Mapping your high-risk times is covered in the Wanting module.)

The Wanting module describes the scale of wanting — the range that runs from high interest in food down to indifference. When the medication is working, you feel wanting turned down: less interested, not thinking about it, the food noise quiet. What gets called fullness — full sooner, satisfied with less — is usually lowered wanting, and it means the medication is doing its job. The thing to watch for is wanting turning back up — food becoming easier to notice again, especially in your high-risk times. Describing where your wanting sits, in your own words, is exactly what you bring to your prescriber.

The logic runs both ways. A strengthening appetite in your high-risk times is a reason to consider moving up — a higher dose is the tool for a stronger defence, and on average higher doses do more. No signal, or barely any, is a reason not to: a quiet appetite is evidence your current dose is still holding the line.

Where could medication be prescribed?

Obesity medication should no longer be viewed as care available only through a narrow group of specialists. Obesity treatment can and should increasingly be delivered within routine evidence-based medical care, including primary care.

Some people may still benefit from referral to clinicians or programs with additional obesity expertise, especially when treatment is complex, when multiple health conditions are involved, or when access and coverage are difficult to navigate.

Weight bias and outdated eat-less, move-more thinking still create barriers, so some people may still need to advocate for evidence-based care.

SAFETY NOTE

Use only authorized, prescribed medications. Avoid unauthorized, counterfeit, or misleadingly marketed GLP-1 products sold online or through unofficial channels.