RUNNING ON EMPTY

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**Why Obesity is Not a Problem of Excess Weight—
It's a Brain Convinced Fuel Is Running Out.**

A New Framework for Understanding Obesity—and Why
Understanding This Changes Everything

SANDRA CORTINA, MD, MPH, DABOM

Running on Empty

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To anyone living with obesity who has been searching for answers, questioning themselves, and wondering why it has always felt so hard. May these pages replace confusion with understanding.

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The views expressed in this book are those of the author and are intended to help readers better understand obesity as a chronic disease and the principles of evidence-based medical weight management, including the use of GLP-1 and other anti-obesity medications. Reading this book does not establish a physician–patient relationship with the author, nor should it be used to diagnose or treat any medical condition or to start, stop, or change any medication without consulting a qualified healthcare professional.

Individual circumstances vary, and treatment decisions should always be made in partnership with your own healthcare provider. References to medications, products, or treatment approaches are provided for educational purposes only and should not be interpreted as endorsements or guarantees of specific outcomes. While this book aims to challenge common misconceptions surrounding obesity and weight management, no treatment can guarantee a particular amount of weight loss or health outcome. The author and publisher disclaim responsibility for any loss, injury, or damages arising from the use or misuse of the information contained in this book.

PREFACE

If you are reading this book, chances are you've spent years trying to understand your weight—and recently, you've realized the story is changing.

Perhaps you are wondering why glucagon-like peptide-1 (GLP-1) medications like semaglutide or tirzepatide suddenly seem to be everywhere. Maybe you've started one yourself and found that the experience wasn't what you expected. Or perhaps you've watched someone close to you begin treatment and realized that everything you thought you understood about weight loss no longer seems to fit.

For many people, the GLP-1 era has brought something unexpected: confusion.

Confusion about why obesity is suddenly being treated with medication after decades of people being told to simply eat less and exercise more. Confusion about what these medications are supposed to do, and how they manage obesity. Confusion about what we're supposed to experience while taking them, only to be met with endless and often conflicting advice online.

For many people, starting a GLP-1 medication brings hope, but it also brings new questions. What should hunger feel like now? Is it normal to still enjoy food? Why has my weight stopped changing? Has the medication stopped working? If you've ever wondered these things, you're not alone. They're some of the most common questions I hear in my practice, and one of the reasons I wrote this book.

The problem isn't that we are asking the wrong questions. The problem is that we are trying to answer new questions using an old way of thinking.

The modern understanding of obesity – and what these medications are supposed to do – has never been fully explained to most people. Not because anyone is trying to keep it from you, but because the science is complex and it has evolved remarkably quickly. Many healthcare professionals completed their training before obesity medicine became the rapidly advancing field it is today, and public understanding has lagged even further behind. In that gap, social media has stepped in, often relying on the only framework it knows: diet culture.

For decades, most conversations about obesity focused on what people were doing: what they ate, how much they exercised, and whether they had enough motivation to stay on track. Far less attention was paid to what the body itself was doing to regulate weight.

As science advanced, so did our understanding.

Over the past several decades, researchers have transformed our understanding of obesity. Today, we recognize it as a chronic disease in which the brain plays a central role in regulating body fat, fullness, metabolism, and energy balance. This system is shaped by the complex interaction between our genes and our environment. Rather than simply storing excess energy, body fat is continuously monitored by the brain as an energy reserve. In obesity, the brain begins to underestimate how much energy is actually available. As a result, it activates biological mechanisms that increase hunger, conserve energy, and defend a higher amount of body fat than is healthy.

This new understanding changed how we thought about obesity, but it didn't immediately change how we treated it.

For years, obesity medicine was filled with frustration. Most medications produced only modest, sometimes short-lived results, and several promising drugs were withdrawn because of safety concerns. For many people with significant obesity, bariatric surgery – a hormonal surgery, by way of bypassing part of

the small intestine or reducing stomach size that can both increase and decrease fullness and hunger hormones respectively – remained the only treatment that consistently produced meaningful and lasting weight loss.

The arrival of GLP-1 medications marked a turning point.

For the first time, we have medications that directly target the biology involved in regulating body weight. For clinicians, they have transformed what is possible in obesity care. For patients, they have introduced an entirely new experience with food, one that many people have never been taught how to understand.

Eating begins to feel different. Fullness comes sooner. Satisfaction lasts longer. The constant mental negotiation around food often becomes quieter. Those changes naturally lead to new questions. *Should I still feel hungry? Is it normal to think about food? Has the medication stopped working?*

These questions don't mean something is wrong. They mean we're finally asking better questions about obesity. This book is about answering those questions and understanding the biology behind them.

As an internal medicine physician specializing in obesity medicine, I have cared for thousands of people living with obesity. As an internal medicine physician specializing in obesity medicine, I've come to appreciate just how common these questions are. They arise because our understanding of obesity has changed dramatically in a relatively short time, while much of the conversation around it has not.

Today, obesity specialists practice within a modern scientific framework, but much of the public conversation about obesity is still shaped by decades of diet culture. At the same time, GLP-1 medications have rapidly expanded beyond obesity specialty clinics, making it difficult for patients to receive consistent education about what obesity is, how these medications work, and what to expect from treatment. In the absence of

clear guidance, many people turn to social media, where simple messages often spread more quickly than accurate ones. It's no surprise that so many people are left feeling overwhelmed and confused.

This book is my attempt to bring clarity to that confusion.

It is not a diet book. It is not a list of food rules. And it is not a promise of rapid weight loss.

It is a guide to understanding the biology of body-weight regulation and how it influences nearly every aspect of living with obesity, including hunger, weight plateaus, weight regain, and the changes many people experience after starting treatment.

The title, *Running on Empty*, reflects one of the central ideas you'll encounter throughout these pages. In obesity, the brain is perceiving less available energy than actually stored, much like a fuel gauge that reads closer to empty than the tank really is. When that internal fuel gauge approaches "empty," the brain responds exactly as it was designed to: It increases hunger, conserves energy, and defends body weight.

Understanding that biology changes more than the way we think about obesity. It changes the way we think about ourselves.

My hope is that, as you read this book, confusion gives way to clarity. Not because obesity is simple – it isn't – but because understanding the biology helps experiences that once felt frustrating, mysterious, or even shameful finally make sense.

May these pages leave you with a deeper understanding of not only obesity, but also of your own body, with greater clarity, greater compassion, and far fewer unanswered questions.

AUTHOR'S NOTE

Transparency matters. I believe readers should understand any professional relationships that could reasonably be perceived as influencing the perspectives presented in this book.

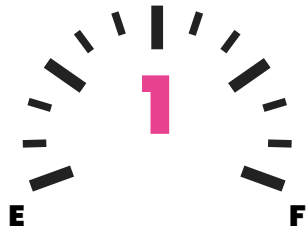
I have received honoraria for speaking engagements from Bausch Health and Novo Nordisk. I have also served on advisory boards and speakers' bureaus for Bausch Health and Novo Nordisk.

The opinions expressed throughout this book are my own and are based on my clinical experience, interpretation of the scientific evidence, and commitment to evidence-based obesity medicine. I have not received funding from any pharmaceutical company for the writing of this book, and no company reviewed, edited, or approved its contents before publication.

PART I

UNDERSTANDING OBESITY

These Are the Conversations About Obesity You Never Got



CHAPTER

WHY OBESITY NEEDED A DIFFERENT ANSWER

YOU PROBABLY NEVER IMAGINED that managing your weight would require medical treatment. Most people don't. But obesity needed a different answer than another diet.

Perhaps you resisted medical treatment for years – whether that was medication, bariatric surgery, or both. Maybe you were skeptical. Maybe you felt treatment was a last resort. Or perhaps you had simply grown tired of hoping after so many disappointments.

For many people, the availability of highly effective obesity medications has dramatically expanded access to treatment. More importantly, it offered a possibility that had become difficult to imagine after years of trying – that this time might be different.

When you finally started treatment, you might have also felt something you had not felt in a long time: hope. Perhaps the constant negotiations about cravings became quiet and less exhausting. But underneath that hope was a quieter voice asking: *Why can't I do this on my own? What is wrong with me? What if this stops working?* These questions are so common that

they have become part of the experience itself. They are also built on one of the greatest misunderstandings about obesity. If any of that feels familiar, I want you to pause for a moment and hear this clearly:

There is nothing wrong with you, and you did not fail your body.

An Outdated "Weight Loss" Map

What's been missing isn't discipline or a lack of effort. It's an explanation of how your biology regulates weight and why you're susceptible to this regulation becoming disrupted over time. For decades, most people have navigated obesity using a weight-loss map built on a simple premise: That body weight was simply a matter of "calories in, calories out," and that the number on the scale reflected your personal choices, discipline, and effort. The scale became the measure of whether treatment was working. A lower number signalled "success." A plateau or regain meant you were told to be more disciplined, more consistent, or more in control. The explanation never seemed to change: The answer was always more discipline, more effort, and more sacrifice. But the old map was built from diet culture logic. It placed personal choices at the centre of the story while leaving out much of what actually governs body weight in the first place. What rarely entered the discussion was the possibility that body weight is governed by a biological system that does not perfectly obey math or discipline.

SIDEBAR

Meet Sarah

Sarah is a patient who started working with me two years ago. Throughout this book, I'll share Sarah's story because many of her experiences are common among people living with obesity. During our first consultation, she walked me through her experiences with weight management and dieting over the years.

Sarah had been on a diet for as long as she could remember. Like many people, it happened in cycles. Starting, stopping, then restarting again. Each time with a new diet plan and determination to be more vigilant than the last, as well as a quiet hope that it would be the last one she could stick to for the rest of her life.

Initially, they all worked. She had lost over forty pounds on three different attempts. On all of those occasions, she felt like she had control – in the beginning. But eventually, something always changed. A holiday season. A stressful work month. A family death. Life would reenter the picture. What had once felt manageable slowly became rigid, exhausting, and eventually impossible to sustain. The mental effort it took to manage her weight on top of everything else eventually became too much. Then the weight would creep back. Every. Single. Time.

Despite years of repeated dieting, Sarah never viewed those experiences as evidence that dieting was an ineffective way to manage her weight. Instead, Sarah blamed herself and never the diet, because the diet seemed to work at first, at least in the beginning. Instead, she had convinced herself that if she had only been more disciplined, the diet would have continued to work.

The last three to five years had been different. Something was slowly eroding her belief system. Glucagon-like peptide-1

(GLP-1) medications were making headlines as breakthrough treatments. The Kardashians were using them. Apparently, so was everyone at the Oscars. Then, in a single week before her family doctor referred her to see me, she learned that her friend, her boss, and her neighbour were all taking a GLP-1 medication.

At the same time, she couldn't ignore how much space dieting occupied in her mind. The constant thinking about food. Planning around meals. Negotiating choices in social settings. The quiet calculation of macros or calories (depending on the diet she was on) running in the background all day. She was tired.

Sarah wanted relief from the mental effort of managing her weight just as much as she wanted to lose weight. So when more and more people around her started using GLP-1 medications with visible success, she began to feel hopeful. But almost immediately, another question followed: *Is this something I will have to rely on for the rest of my life?*

The question of whether we'll have to "rely on medication" doesn't emerge out of nowhere. It is shaped by years of exposure to diet culture. Our expectations about treatment have often followed a temporary model: start, stop, "succeed," or "fail." A typical black-and-white framework. Needing treatment for the long term? That felt like failure.

But what if the expectation of finite treatment from diet culture *is* the problem? What if the very reason this has felt so hard for Sarah for so long is because she has been trying to manage a biology that was never meant to be managed consciously in the first place?

That's hard to accept when we're constantly being sold stories of people who supposedly lost significant amounts of weight through diet and exercise alone.

The Truth About Diet and Exercise

It is important to acknowledge that some people do lose a significant amount of weight through changes in diet and physical activity alone, and some are able to maintain that weight loss long term. These stories are real and should not be dismissed. In fact, research suggests that approximately 20 percent of people who lose at least 10 percent of their body weight maintain that loss for at least one year. Studies of long-term weight-loss maintainers also show that many continue to follow highly structured eating patterns and engage in about an hour of physical activity on most days of the week.

What is often overlooked, however, is the magnitude of change that usually accompanies these success stories. Significant weight loss rarely comes from a single small adjustment. More often, it reflects a substantial transformation in daily habits. Imagine someone who spends most of the day gaming, eats fast food several times a week, rarely exercises, and drinks sugary beverages every day. If that person begins exercising regularly, dramatically improves the quality of their diet, reduces highly processed foods, and becomes more physically active overall, meaningful weight loss is entirely possible. The greater the change in behaviour, the greater the potential effect on body weight.

The challenge is that many people seeking obesity treatment are not starting from that place. They are already trying. They may be preparing most of their meals at home, walking regularly, limiting restaurant food, and making thoughtful food choices. Their lives do not contain obvious sources of processed food and drink waiting to be removed. For these individuals, being told to "eat healthier" or to eliminate a weekly milkshake is unlikely to produce the kind of weight loss they are hoping for.

This is where many people begin to blame themselves. They look at someone who lost a large amount of weight through lifestyle changes and conclude they must not be trying hard enough. But that comparison is often unfair. The question is not whether lifestyle changes can work. Clearly, they can. The more important question is whether there is enough room left in your current lifestyle to create the degree of change needed to meaningfully alter your body's weight regulation system. For a few people, the answer is yes. For most people – especially those who have already spent years making healthy choices – the answer is no. Their obesity persists because their disease is defending their current weight, a concept we'll explore shortly.

Perhaps you already understand that obesity is a chronic disease and far more complex than most people realise. But understanding something intellectually does not always make living with it any easier. You may still feel frustrated that you need medication. You may still wonder why this feels harder for you than it does for other people. And if you've already started treatment, you may feel confused when your experience doesn't match what you expected. Maybe your weight loss has slowed. Maybe your hunger has become stronger. Maybe you've reached a plateau and are wondering if you're doing something wrong.

The truth is that these experiences are usually not signs that treatment is not working. They are signs that obesity is a complex disease, one that behaves very differently from the simple "calories in, calories out" story many of us were taught.

Why Diet Culture Still Shapes How We Think About Obesity

Even when we understand that obesity is a complex, chronic disease, it can be difficult to let go of the beliefs we learned from decades of weight-loss messaging. For years, we were

taught that weight loss should be temporary, yet predictable. If you were doing the right things, the scale should keep moving down. If weight loss slowed, plateaued, or reversed, something must have gone wrong. We were taught that success came from consistency, discipline, and effort, and that long-term treatment shouldn't be necessary if we were doing things "properly."

These ideas have become so familiar that they still feel like common sense. They continue to appear in conversations with friends, weight-loss programs, social media, and sometimes even in healthcare. We're still told that weight loss should be linear and that hard work should always be rewarded on the scale. We have simply shifted from obsessing over calories to obsessing over protein, often under the banner of "optimizing health." When diet culture goes unchallenged, it reinforces old beliefs about weight that never served us.

Sarah is the everyday person who followed that advice. She tried diets. She lost weight. She regained it. Then she tried again. The cycle repeated. Looking back, the repeated cycles of weight loss and regain might have raised questions about the framework itself. It's like following a GPS map that repeatedly takes you to the wrong destination. At some point, we would start blaming Apple or Google, but we would never blame ourselves, the driver. This is rarely the case for obesity. Instead, like many people, Sarah concluded that the problem was *her*. Partly because effective obesity treatments were limited, and partly because the prevailing narrative offered few explanations beyond personal responsibility.

Nostalgia Is Not a Strategy

For decades, the dominant explanations for obesity were rarely questioned. Then came the GLP-1 era, forcing many of us to reconsider what we thought we knew about obesity and how it should be treated.

Medicine evolves when new treatments reveal that our understanding of a disease was incomplete. Before effective treatments for stomach ulcers became available, ulcers were often attributed to stress, personality, diet, or lifestyle. Patients were told to avoid certain foods, reduce stress, and change their habits, yet many continued to experience recurrent ulcers and serious complications.

The discovery of *Helicobacter pylori* (a stomach bacteria) and the development of effective antibiotic treatment transformed the field. We no longer tell patients that changing their personality or reducing stress is the treatment for stomach ulcers.

Obesity is beginning a similar transformation. The introduction of GLP-1 medications is forcing us to rethink explanations that once seemed like common sense.

The GLP-1 Era: What Incretin Therapy Revealed About Obesity

Geologists divide Earth's history into eras – periods marked by events that permanently changed life on our planet. The asteroid that ended the age of the dinosaurs is one example. If obesity medicine had its own geologic time scale, we are standing at one of those boundaries right now, watching a new era begin.

The biological basis of obesity has been misunderstood for decades, and we're still only beginning to understand it. Researchers identified many of the key hormonal systems involved in body-weight regulation only about twenty years ago. Effective obesity medications have existed for little more than a decade, and it has been only within the past five years that medications have produced weight loss approaching that seen with bariatric surgery.

We do have an early, evolving collection of therapies now centred around GLP-1 hormone signalling. GLP-1 is one member of a larger family of hormones called incretins – hor-

mones your gut releases after you eat to help tell your brain and pancreas that food has arrived. Incretin-like therapies are not a cure for obesity but rather a key input needed to sustain long-term weight management in an otherwise complex, chronic disease. Helping the brain regulate its long-term fuel supply is an essential part of treating obesity. No matter who you are, helping the brain regulate its long-term fuel supply is always part of treating obesity. That is true 100 percent of the time. All effective anti-obesity medications, including GLP-1 medications, work by enhancing the biological signals that tell your brain how much fuel is available in ways that dieting alone cannot. In short, these medications strengthen fullness signals, creating room for weight loss even as the brain continues to monitor and defend its energy stores. As a result, fullness increases, cravings often decrease, and eating becomes easier to regulate. Together, these therapies form the foundation of modern obesity treatment.

SIDEBAR

"GLP-1s": Quick Note About the Term

The media often uses "GLP-1s" as shorthand for today's obesity medications. Strictly speaking, GLP-1 is only part of the story. Many newer medications combine GLP-1 with other hormones, and future therapies may work through entirely different biological pathways. Throughout this book, I'll use the term "GLP-1 medications" because it's the name most readers are already familiar with. But these medications represent something much bigger. They are the beginning of a new generation of treatments that work by influencing the biological systems that regulate body weight. The medications themselves will continue to evolve, but the principle behind them is unlikely to change.

GLP-1 medications work, in part, because they interact with this appetite regulatory system. They increase fullness by enhancing satiety signalling in the brain and by slowing stomach emptying. For many people, the result feels almost startling. Profoundly fuller. Fewer intrusive thoughts about food. A sense that the volume has finally been turned down, and that quiet can feel life-changing.

At first glance, it may seem as though these medications have finally solved obesity. But it is important to understand what is actually happening. These medications do not cure or reverse obesity. They help manage obesity. They strengthen signalling pathways that were already there. They help rebalance parts of a system that have become misregulated. They are powerful medications that work within your biology, not in place of it. That may help explain why the early experience can feel dramatic, while the later experience often becomes more nuanced. To understand why, we first need to understand what obesity is and how the brain makes sense of the body's energy reserves.

Obesity: A Brain Convinced Fuel Is Running Low

The word *obesity* originates from the Latin *obesus*, and translates to "having eaten oneself fat." By modern standards, it is a terrible definition—one that is judgmental, simplistic, and stigmatizing. Yet buried within that outdated description is an important clue. I'm not defending the definition, but it points us towards significant information. It recognizes obesity as not simply about the presence of fat tissue. It is about a biological system that regulates eating behaviour.

Fundamentally, body fat is regulated through a communication system between three main players: the brain, fat tissue, and the gut (Figure 1.1). Together, they constantly exchange signals about nutrient availability and energy storage. Their

collective job is to maintain energy balance and ensure the body has enough fuel to survive.

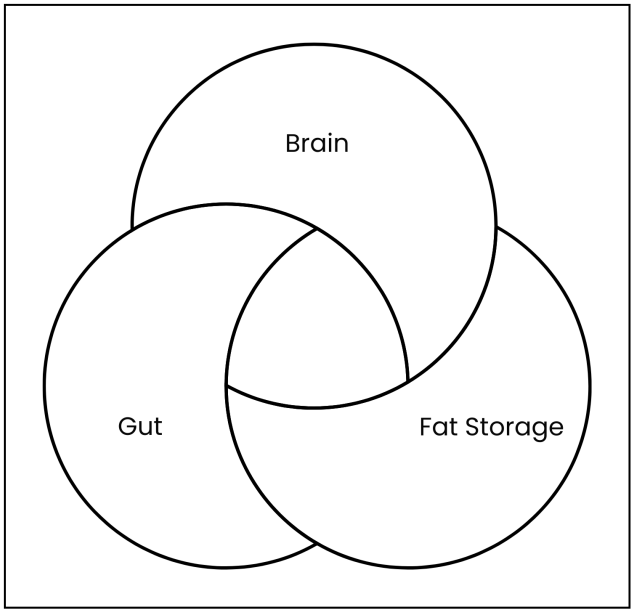


Figure 1.1. The Weight Regulation System. Weight is regulated through a complex communication system involving the brain, fat storage, and gut.

Which raises an important question: If someone is carrying abundant energy stores in the form of fat tissue, why does the brain still trigger appetite signals such as early fullness waning, food cravings, and constant thoughts about food? Aren't these systems supposed to be talking to each other?

This is the central paradox of obesity. The body has plenty of stored energy, yet the brain behaves as though fuel is running low. It's as if communication between the brain and fat tissue has become uncoupled. As a result, the brain defends an inappropriately high amount of body fat. In obesity, the core problem is not a lack of stored energy; it is that the brain has become convinced that fuel is running out.

Weight Loss Turns on the Brain's Starvation Alarm

At first, this sounds backwards. If obesity is a disease in which the brain behaves as though fuel is running low, why would losing weight activate the body's starvation response? The answer is that this apparent contradiction is actually one of obesity's defining features. In obesity, the biological systems that regulate body fat defend against weight loss far more aggressively than they defend against weight gain. As a result, the body protects an amount of fat that can ultimately harm health.

To understand why this happens, it helps to look at one of the brain's most important biological messengers: a hormone called leptin. Produced by fat tissue, leptin tells the brain how much energy is stored. This surprises many people because fat tissue isn't just where the body stores energy; it's also one of the body's largest hormone-producing organs. Leptin is often described as a long-term fullness signal, but a more accurate way to think about it is as the brain's anti-starvation signal. As fat mass increases, leptin levels rise. In theory, this should tell the brain that energy stores are plentiful, increasing feelings of fullness.

However, people living with obesity have *very high* leptin levels. If leptin simply worked as a "fullness" signal, very few people would develop obesity. Instead, many people continue to experience blunted satiety, cravings, and food preoccupation despite having both abundant energy stores and high leptin levels.

These observations led researchers to propose the idea of leptin resistance – the idea that the brain stops responding normally to leptin's signal, even when leptin levels are high. Today, many researchers believe it is more helpful to think of leptin differently. Rather than acting primarily as a fullness

signal, leptin appears to function mainly as an *anti-starvation signal*.

A more useful way to think about communication between the brain and fat tissue is this: The brain mainly pays attention to leptin when levels fall. Rising leptin levels are largely ignored. Falling leptin levels are not.

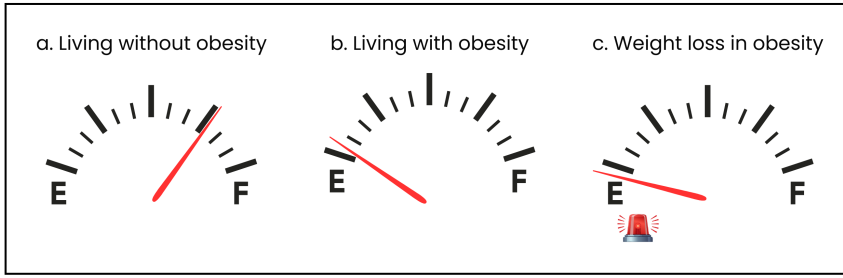


Figure 1.2. The Brain's Fuel Gauge in Obesity. The brain continuously monitors available energy stored in the body, much like a fuel gauge: (a) In people without obesity, the brain perceives adequate energy reserves and does not activate a starvation response until energy stores become substantially depleted; (b) in people living with obesity, the brain interprets the same amount of stored energy as less available, shifting the fuel gauge closer to "empty"; and (c) as weight is lost, the brain reaches its internal "low-fuel" threshold sooner, activating a biologically programmed starvation response characterised by increased hunger, reduced energy expenditure, increased food reward, and other adaptations that defend body weight. This response reflects how the brain regulates perceived energy availability – not the actual amount of body fat.

Whenever we lose weight, our fat stores shrink. As they do, leptin levels fall. Because leptin acts primarily as an anti-starvation signal, the brain interprets that drop as a sign that fuel is running low. It responds by activating a biologically programmed starvation response. Fullness decreases. Hunger becomes stronger. Energy expenditure falls. The drive to regain the lost weight intensifies.

This is why maintaining weight loss after dieting is so difficult. Weight loss activates the very starvation response that

obesity is already primed to defend against – even when the body still has plenty of stored energy.

This process can be difficult to picture, so it helps to think of your brain as using an internal fuel gauge. In a healthy system, that gauge accurately reflects how much stored energy is available. In obesity, however, the gauge becomes miscalibrated and reads lower than it should. As weight is lost, the needle falls even further, eventually crossing the brain's starvation threshold and triggering the powerful biological defenses that increase hunger, reduce energy expenditure, and make maintaining weight loss so difficult. Figure 1.2 illustrates how this fuel gauge changes before obesity, during obesity, and after weight loss.

How We Learned to Work Around the Starvation Response

Weight loss is not the hard part. Defending weight loss against the brain's anti-starvation response is the hard part. That raises an obvious question: What if we could prevent the brain from switching into starvation mode during weight loss? For a time, researchers thought they had the answer. After all, leptin is the body's primary anti-starvation hormone. If weight loss lowers leptin levels and triggers the brain's starvation response, wouldn't replacing leptin solve the problem?

Well, we tried that, and it turned out to be a bit of a doozie.

Researchers gave people with obesity extra leptin, expecting it would tell the brain that energy stores were plentiful, strengthen feelings of fullness, and produce meaningful weight loss. Instead, very little happened. People living with obesity already have high leptin levels because leptin is produced by fat tissue. Giving even more leptin was like shouting through a megaphone at someone who had already stopped listening.

To make matters worse, some participants developed antibodies – proteins the immune system makes to recognize and

attach things it sees as foreign – against the medication, making the treatment even less effective. What began as one of the most promising ideas in obesity medicine ended as a disappointing dead end for obesity treatment.

Dead ends can be informative. If people with obesity already had plenty of leptin, perhaps the problem wasn't that there was too little leptin – or even that leptin itself was broken. In fact, what if I told you there is nothing wrong with leptin in obesity? Leptin isn't designed to protect us from obesity. It's designed to protect us from starvation. By that measure, it is working remarkably *well*. That realisation would send obesity research in an entirely new direction.

The Problem Was Never Weight Loss; It Was the Starvation Response That Followed

For decades, obesity research focused on a simple question: How do we help people lose weight? But over time, we learned that weight loss was not the hardest part. Many people could lose weight for a period of time. The real challenge was keeping it off. Every time body weight falls, the brain responds as if food has become scarce: Weight loss triggers a biological starvation response. This shifted the focus of obesity research. The goal was no longer simply to help people lose weight. It became finding ways to prevent the brain from interpreting weight loss as a threat to survival – a response driven in large part by falling leptin levels.

One of leptin's jobs is communicating with the brain's fullness circuits, deep in a region called the hypothalamus – a small area near the base of your brain that acts like a control centre for hunger, body temperature, and other automatic functions. This is partly why leptin has often been mislabelled as a "fullness hormone," even though its primary role is protecting against starvation during weight loss. But leptin's job description doesn't end at the hypothalamus. We'll come back

to that in a moment. For now, here's the question that mattered most to obesity researchers: if leptin talks to the brain's fullness circuits, could they reach those same circuits through a different pathway? Instead of targeting leptin directly, perhaps they could work around the leptin system by using incretin hormones.

There was one problem. Natural incretin hormones were never designed to provide long-lasting signals. They're released after a meal and disappear within minutes. But what if we could create a version that lasted for days instead of minutes – perhaps even an entire week? Could that provide the kind of sustained signalling needed to help work around the brain's starvation response?

That idea became the foundation of modern obesity medicine. Researchers found that long-acting GLP-1 medications reach many of the same brain cells leptin normally reaches – a cluster of neurons in the hypothalamus called the arcuate nucleus. Once there, GLP-1 medications turn on the same fullness circuits leptin is supposed to turn on, without needing leptin to deliver the message. In other words, these medications found a workaround for the fullness message that leptin can no longer deliver once its levels fall. They step in to tell the brain's appetite centre that fuel is available, even when leptin levels are low.

This is exactly why obesity needed a different solution.

We needed an answer to the drop in appetite-related signalling that occurs with weight loss. We needed to stop the brain's appetite centre from interpreting weight loss as a sign that fuel was running out. Only then could weight loss become more tolerable and sustainable – more in line with the standards we expect from treatments for other chronic diseases.

But "workaround" is an important word, and an honest one. GLP-1 medications don't replace leptin. They step in for one part of its message. And it turns out leptin's message goes to more places than the appetite centre alone.

Medical treatments should be tolerable. Dieting alone asks people to live with a biologically programmed starvation response. Modern obesity medications offer a different approach. Instead of asking people to lose weight while feeling hungry, they help people lose weight while feeling fuller.

How We Manage the Fuel Gauge

At this point, you may be wondering: *If obesity is a disease of a brain convinced that fuel is running low, how do we fix that?* The answer can be frustrating at first because it challenges many of the ideas we've been taught about weight loss. But it also offers something far more valuable: an explanation.

We do not currently know how to permanently "fix" the lower fuel gauge. If we did, obesity medicine would look very different. I recognize that this can feel discouraging or disappointing. Most of us have been taught to think of weight loss as a temporary phase with a finish line, so that expectation is understandable. The reality, however, is that obesity behaves much more like other chronic diseases, where long-term management is part of treatment.

A brain convinced that fuel is running low lies at the heart of what makes obesity a chronic disease. As a result, it actively defends a higher level of body fat. While we cannot permanently correct this underlying misregulation, medications and bariatric surgery can shift the brain's fuel gauge in our favour. By working around leptin's fullness message and strengthening the brain's own fullness signals, these treatments help reassure the appetite centre that fuel remains available, reducing the pressure to defend excess energy stores.

A Workaround, Not a Replacement

Here is where it's worth being honest about what we do and don't yet know.

Leptin's job isn't only to talk to the brain's appetite centre. When leptin falls during weight loss, it also quietly turns down other systems in the body, including how much energy you burn at rest and how actively your thyroid gland works. Researchers have shown this directly: Giving weight-reduced volunteers just enough leptin to bring their levels back to where they were before they lost weight reverses the drop in energy burned at rest and restores thyroid hormone levels, without changing what anyone eats. In other words, falling leptin doesn't just turn up hunger. It also quietly turns down the body's metabolic engine.

This is the part of leptin's job that GLP-1 medications have not been clearly shown to replace. Even though these medications help the brain's appetite centre feel reassured that fuel is available, current research does not show that they reliably prevent the drop in resting energy expenditure that comes with weight loss. That's an important distinction, and an honest one: we have found a workaround for what falling leptin does to appetite. We have not yet found one for what falling leptin does to metabolism.

If you've ever felt like your progress slowed over time, even while staying consistent with your medication, this may be part of why. It isn't a sign that the medication has "stopped working," and it isn't a reflection of your effort or willpower. It's a reminder that these medications solve one piece of a much larger biological puzzle – a real and current gap in obesity medicine, not a personal shortfall.

That gap is one of the reasons obesity research keeps moving forward. Finding a workaround for leptin's fullness message was a major breakthrough. Finding one for its effect on metabolism may be the next one.

SIDEBAR

Fullness vs. Hunger: A Quick Note on the Terms

Before we move on, it is worth paying close attention to the following word: fullness.

Many people assume obesity treatments work by eliminating hunger. They do not. In fact, no effective or ethical obesity treatment is trying to remove hunger altogether. Hunger is a normal biological signal. It is an essential part of survival. Without feeling hungry at appropriate times, we would forget to eat and eventually become malnourished.

Instead, effective obesity treatments work by enhancing fullness. They help the brain receive a clearer message that fuel is available, reducing the need to prepare for starvation. This distinction between hunger and fullness is *incredibly* important, and we will return to it throughout the rest of the book.

Once you understand that obesity is not simply a problem of excess weight but a problem of how the brain regulates and defends energy stores, many common assumptions begin to fall apart. The language of diets, finish lines, and quick fixes starts to feel less useful. In its place emerges a different way of understanding obesity, one that is more consistent with both the science and the lived experience of people managing the disease.

A Fuel Gauge Framework for Understanding Obesity

The purpose of this book is not simply to explain the biology of weight regulation. It is to offer a different way of understanding obesity – one that more accurately reflects how the disease behaves and makes that biology easier to understand.

Many of the frustrations people experience come from trying to navigate obesity with a map that was never designed for the terrain. When obesity is viewed through the lens of chronic disease rather than weight loss, experiences that once felt confusing, discouraging, or deeply personal begin to make sense.

This book is an invitation to leave behind diet culture's expectations that no longer serve you and adopt a framework grounded in biology rather than personal responsibility. Throughout the chapters ahead, we'll use a new framework to make sense of the common experiences that accompany medically managed weight loss. Many of the experiences that once felt confusing, discouraging, or deeply personal begin to make more sense when we stop viewing obesity as a problem of willpower and start seeing it as a disorder of energy regulation. To understand why, it helps to think of obesity as a malfunctioning fuel gauge.

When the Brain Starts Asking for More Fuel

If obesity is a disease in which the brain becomes convinced that fuel is running low, the next question is obvious: How did the brain fall out of balance in the first place?

Obesity develops when genetic susceptibility meets layered environmental pressures. These pressures can include weight-promoting medications, disordered eating, poor sleep, easy access to ultra-processed foods (packaged foods made mostly from refined ingredients and additives rather than whole foods), or repeated cycles of weight loss and regain. Over time, they interact in ways that signal the brain to store more energy without generating the normal fullness signals that would reassure it that enough fuel is available. As a result, the fuel gauge needle remains closer to empty. Because genetic susceptibility differs from person to person, so does the threshold at which the brain begins defending a higher fuel reserve. For some

people, relatively small pressures are enough to keep the gauge reading low despite abundant energy stores. For others, it takes much more.

At its core, the brain is doing exactly what it believes it needs to do: asking for more fuel. It does this by encouraging the body to store more energy while keeping the fuel gauge needle near empty. From the brain's perspective, this is a protective response. It is trying to build a larger fuel reserve that can be drawn upon during a future crisis. The problem is that the anticipated catastrophe never arrives, yet the brain continues to behave as though it is just around the corner.

Obesity emerges when the brain begins defending a larger fuel reserve. As these pressures build, the fuel gauge shifts closer to "empty," signalling the need to store more energy even when the body already has abundant fuel available. Over time, this new setting can become the brain's default, allowing it to defend higher levels of body fat long after the original pressures have disappeared.

Why We Must Target the Fuel Gauge, Not the Fuel Tank

Think of your body's weight-regulation system as the fuel gauge in a car. In obesity, the problem isn't the size of the fuel tank; it's the accuracy of the gauge reading it.

Now, imagine driving a car with a faulty gas gauge. The tank is full, but the gauge keeps hovering near empty. Every time you look down at the dashboard, it warns you that you're running out of fuel. You know the tank is full. The problem is that the system responsible for measuring the fuel isn't reporting accurately.

What is likely to happen?

You would probably still stop at gas stations more often. You would think about fuel more frequently. You might even feel anxious about running out. Not because the car lacks energy, but because the gauge keeps telling you that you soon will.

This is remarkably similar to what happens in obesity.

The body contains abundant stored energy in the form of body fat, yet the brain behaves as though energy reserves are running low. Hunger becomes more frequent. More food is needed to feel full. Thoughts about food become more persistent. The biological drive to acquire fuel remains activated despite abundant energy already being available.

You may have already done many of the things you were told would solve the problem. You became more selective about the fuel entering the tank and worked to reduce the forces pushing the system toward storing more fuel. Yet despite all that effort, the needle still hovers near empty – not because you didn't try hard enough, but because the problem lies with the fuel gauge itself.

Improving these environmental pressures – such as eating fewer ultra-processed foods or getting better sleep – still matters. These changes reduce some of the pressures that encourage the brain to store more energy. But they do not correct the faulty fuel gauge itself. The brain continues to signal that more fuel is needed, and when weight loss begins, the starvation response is activated. That is why obesity is not simply a disease of behaviour.

How Medical Treatments Target the Fuel Gauge

A common question I hear is this: If medication amplifies fullness, reduces cravings, and makes weight management feel easier, will the brain eventually learn to do this on its own?

It's a reasonable question, and one many people hope has a different answer. Unfortunately, the brain's weight-regulation system doesn't appear to work that way. It doesn't respond to reasoning, determination, or repeated experience. We can't train it to behave differently. We wouldn't expect someone to take blood pressure medication for a year and then expect their

body to permanently remember how to regulate blood pressure once the medication is stopped.

Like many medical treatments, current obesity therapies work by recalibrating biology. In obesity, they recalibrate how much weight your brain is comfortable defending. They enhance fullness and help the brain perceive its fuel reserves differently. As the gauge moves farther from empty, fullness lasts longer, food occupies less mental space, and the biological pressure to refuel begins to ease. When the dashboard starts displaying a more accurate reading, the driver naturally stops pulling into gas stations so often because the system is no longer signalling that fuel is running low.

Recalibration may sound intrusive, but it is actually reassuring. It means we understand where the problem lies. We are no longer chasing every possible external pressure or blaming ourselves for a lack of discipline. Instead, we are targeting the signalling system responsible for regulating the interpretation of fuel availability. The goal is to recalibrate the level of fuel reserves the brain defends, allowing the body to settle at a lower weight.

Obesity is far more complex than any analogy can fully capture. Even so, the fuel gauge provides a useful framework for understanding what obesity is, how it behaves, and how current treatments fit into that picture. It also helps us understand why obesity must be managed as a chronic disease.

Why Obesity Must Be Managed Like a Chronic Disease

Once obesity is understood as a chronic disease, my hope is that the conversations we have about it begin to change as well. It is completely understandable to ask "How do I lose another twenty pounds?" After all, that's the question most of us have been taught to ask. But as our understanding of obesity deepens, a different question becomes more valuable: "How do I

manage this disease in a way that supports my health and quality of life for years to come?"

This may sound like a subtle distinction, but it fundamentally changes how treatment is approached and experienced. It marks the difference between diet culture and medicine. Diet culture is organized around weight loss at all costs. Medicine is organized around the long-term management of disease and the improvement of health. Although these perspectives sometimes overlap, they are built on very different assumptions and often lead to very different expectations.

For decades, body weight has been treated as the primary measure of success. Progress was defined by how much weight was lost, how quickly it happened, and whether it could be maintained indefinitely. When weight loss slowed or weight was regained, the explanation usually rested on the individual's perceived shortcomings. This framework only "makes sense" when obesity is viewed as a problem of excess body weight. It is much harder to defend when obesity is recognized as a chronic biological disease.

Consider how medicine evaluates other chronic diseases. We do not consider asthma treatment a failure because symptoms return when therapy is withdrawn. We do not expect chronic diseases to disappear simply because they have been successfully managed for a finite period of time. Instead, treatment is evaluated according to disease control, symptom burden, functional improvement, health outcomes, and long-term sustainability.

Obesity should be approached no differently.

Once obesity treatment is viewed as a way to recalibrate the level of body weight the brain defends, weight becomes one outcome among many rather than the sole objective. If the number on the scale has a role in chronic disease management, it is to provide clinical feedback to your healthcare provider. It helps us evaluate how effectively treatment is modifying your

physiology. Weight still matters, but it is no longer the only measure of success. Equally important are questions such as these: Is food occupying less mental space? Is the treatment sustainable over years rather than weeks?

These questions are central to chronic disease management because they focus on the disease itself rather than the numbers alone.

This distinction also helps explain why the GLP-1 era has generated so much confusion. While these medications are relatively new, the way many people think about weight loss is not. They are often viewed through the same weight-loss lens that has shaped dieting conversations for decades. As a result, people naturally ask these questions: How much weight have I lost? Why has the scale stopped moving? When can I stop treatment? These are understandable questions, but they arise from a weight-loss mindset rather than a chronic disease mindset.

The Hidden Cost of Weight-Loss Thinking

The shift from weight-loss thinking to chronic disease management may be the greatest challenge facing obesity medicine. Until that shift occurs, it stands in the way of fully understanding obesity with compassion, providing care that is both evidence-based and ethical, and redefining what successful treatment truly looks like.

In fact, I would argue that "successful" may not be the right word at all.

Successful implies a finish line. Treatment has been completed, the problem has been cured, and a permanent state has been achieved. This is the language of *transformation* – the same language that has shaped weight-loss narratives and dominated conversations about obesity for decades. Today, transformation is simply being rebranded to sell subscriptions, programs, and products tied to GLP-1 treatments.

The use of transformational language in obesity care may seem like an odd thing to focus on, but it helps explain many experiences that leave people feeling frustrated, confused, or as though they have done something wrong. Once obesity is understood as a chronic disease that misreads energy stores, the question is no longer whether someone has succeeded or failed; it is whether the disease is being effectively managed.

So, why do we continue answering the weight-loss industry's call to chase a final definition of success?

Why the Confusion Persists

As GLP-1 medications have entered mainstream practice, they have collided with two powerful and deeply entrenched forces: diet culture and weight bias. Here's the fundamental problem: Neither recognizes obesity as a chronic disease. Instead, both depend on the public believing that excess weight can be managed simply by subscribing to their messaging, services, or products.

Diet culture is not simply a collection of weight-loss products or nutrition rules. It is an ideological framework for interpreting excess body weight – what it represents, what causes it, and how it should be addressed. At its core, it divides outcomes into two categories: success and failure. If weight decreases, the individual is praised for discipline, motivation, and commitment. If weight returns or does not decrease as expected, the individual is assumed to have lost focus, made poor choices, or simply not tried hard enough. The biology never changed; only the explanation did. The reality that body weight is regulated by powerful biological systems is rarely part of the discussion because it complicates the simpler story often told by people who have never been trained in obesity biology.

Unfortunately, the prescribing of GLP-1 medications within a medical model did not automatically displace this older way

of thinking. In many ways, diet culture has simply adapted to a new set of expectations around weight loss *with* medications. Instead of counting points, there are "GLP-1 point programs." The language sounds more medical, but the underlying assumptions remain remarkably familiar.

The clearest evidence is online. Scroll through social media and you'll find physicians, dietitians, and health influencers who support obesity medications while still focusing on maximizing weight loss, accelerating results, "breaking through plateaus," avoiding adaptation, or eventually discontinuing treatment. Some sell protocols for "optimizing" outcomes. Others promote supplements, protein products, fasting strategies, body composition programs, or metabolic "boosters" designed to enhance weight loss.

The persistent obsession with optimization reveals a deeper tension in how obesity treatment is discussed. If GLP-1 medications are helping manage a chronic disease, why does so much of the conversation remain focused on pursuing ever-greater weight loss and continual optimization, even at the expense of quality of life? The pursuit of perfection creates a new cognitive load: more rules, more monitoring, more self-surveillance, and more sacrifice. Although many of these messages are presented as medical guidance, they reveal a deeper allegiance to a diet-centric framework. The weight bias is both subtle and obvious: The medication is never enough; more optimization is always required.

Medicine and chronic disease management ask a different question: not "How do I lose more weight?" but "How do I manage this disease more effectively over time?" One question is rooted in weight loss. The other is rooted in chronic disease management.

This distinction matters because social media has increasingly become many people's primary source of health information – a space that rewards content reinforcing weight-loss nar-

ratives. The patient who loses one hundred pounds becomes the featured "success" story. Dramatic before-and-after photographs become clickbait. Physicians whose patients achieve extraordinary results gain attention and credibility. Meanwhile, the far more common experiences of obesity management – weight loss slowing, stabilizing, partially returning, or requiring ongoing treatment – receive far less attention. People whose experiences do not match these extraordinary expectations gradually disappear from view. Perhaps they stop attending follow-up appointments, leave treatment programs, or simply move on. What remains are the exceptional outcomes, creating the illusion that they represent the typical experience.

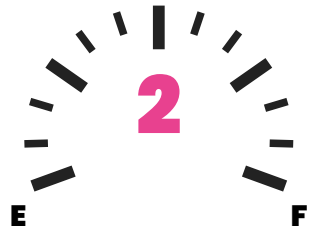
The arrival of GLP-1 medications should have challenged many of these assumptions. Rather than prompting a shift toward chronic disease management, however, these treatments have largely been evaluated using the same measures that have long defined weight-loss culture: how much weight was lost, how quickly it happened, and whether treatment can eventually be discontinued. Until obesity is understood first as a chronic disease and only secondarily as a weight-loss concern, we will continue to ask the wrong questions, celebrate the wrong outcomes, and misunderstand what "successful" treatment actually looks like. We will continue to focus on endpoints instead of management, and transformation instead of regulation. The future of compassionate, ethical obesity care depends on our willingness to move beyond the assumptions of diet culture and adopt a framework grounded in biology, medicine, and long-term disease management.

A better framework begins with a simple idea: Obesity is a disease of a brain convinced that fuel is running low. Once we understand that, many of the assumptions that have tradition-

ally shaped conversations about obesity begin to change. From there, two important questions naturally follow:

1. How do we determine when this process has crossed the threshold into disease?
2. What causes the brain's internal fuel gauge to become miscalibrated in the first place?

The next two chapters explore these questions by examining how obesity is defined and diagnosed, followed by the environmental pressures most capable of pushing the fuel gauge toward empty.



CHAPTER

WHAT ARE WE CALLING OBESITY?

IN MEDICINE, DISEASES ARE typically defined by identifying the underlying biological abnormality and demonstrating that it causes harm or increases the risk of harm. Obesity presents a unique challenge because its underlying biological abnormality cannot be measured in routine clinical practice. I believe the following definition best captures obesity's underlying biological abnormality:

> *Obesity is primarily a chronic disease of a brain that is convinced fuel is running low. As a result, it defends a larger energy reserve. Paradoxically, weight loss pushes the fuel gauge toward empty, triggering a starvation response that mounts a biological defense to replenish the weight that was lost.*

Instead, we rely on indirect markers such as BMI, waist circumference, and waist-to-height ratio. These measures help identify the consequence of the disease – namely excess body fat (adiposity) – but they do not directly capture the brain's persistent perception that fuel is running low.

Another reason obesity has been so difficult for patients and the public to understand is that it does not fit neatly into the

way we typically think about disease. When we have heart disease, we know the root of the disease can be narrowed to the heart. When we have thyroid disease, we can point to the thyroid gland. But where exactly does obesity live? The answer is not nearly as straightforward, and that complexity has contributed to decades of confusion about what obesity is – and where it lives.

SIDEBAR

The Disease Without a Single Address

Eight weeks after starting treatment, Sarah returned for a follow-up appointment. She had lost weight, her cravings were more manageable, and many of the changes we had hoped to see were beginning to happen. Yet something still didn't sit right with her.

"I've been wondering," she said. "Should we recheck my thyroid?"

We had, in fact, already checked it.

"What about cortisol? I've heard stress can cause weight gain."

We had assessed that too.

A pause.

"Maybe it's my hormones. Should we check my estrogen levels?"

The question wasn't really about thyroid function, cortisol, or estrogen. Sarah was searching for something deeper: the reason. The root cause. The one thing that would finally explain why weight had been such a struggle for so many years.

I understood the impulse completely.

When we have heart disease, we investigate the heart. When we have asthma, we order lung tests. Even when the causes

are complex, there is usually an identifiable organ, tissue, or system where the disease lives. Patients often sense this intuitively. If obesity is a disease, where exactly is the disease? Is it the thyroid? The hormones? The stomach? The metabolism?

But obesity feels different.

The question seems simple, yet it exposes one of the central challenges in understanding obesity. Unlike diseases that can be localized to a single organ or biological abnormality, obesity emerges from a complex regulatory system distributed throughout the entire body, but largely between our brain, gut, and adipose tissue (adipose tissue is the medical term for fat tissue, which is made up mostly of fat cells that store energy for the body). That's three different organs involved! That complexity has contributed to decades of confusion about where and what obesity is – and where and what it is not.

Where Is Obesity Located?

"*Where is the disease?*" reflects a misled expectation that obesity should be a localized problem, or a single organ that we can point to on a scan or confirm with a blood test. But obesity is not a disease of a single *organ*. Rather, it is a disease of a *system* largely centred on the brain that is defending a higher fat mass. Once we understand this, we can begin to appreciate the complexity at play and stop exhaustively searching for the "broken part." We can stop blaming the thyroid, cortisol levels, or whatever organ influencers have caused momentum around online. We gain more clarity when we start recognizing that obesity is a complex disorder that is defending a higher weight state, even when that state becomes metabolically or physically harmful.

These defense mechanisms operate within an integrated regulatory network involving the brain, gut, and adipose tissue. Over time, that network shifts the body's internal fuel gauge inappropriately toward "empty," despite adequate energy stores. This becomes the brain's new default setting. The faulty fuel gauge is the underlying mechanism; body size is simply its visible consequence. Unfortunately, body size became synonymous with obesity, and body mass index (BMI) came to define what obesity was.

We Outgrew the Ruler

BMI had been the standard measure for years. Over time, however, obesity, body size, and health risk became increasingly conflated. This created a fundamental dilemma: Is obesity simply the state of living in a larger body, or is a larger body the consequence of an underlying disease?

This is more than a semantic question. How we answer it determines where we place the disease and, ultimately, how we understand, diagnose, and treat obesity. Yet this distinction remained largely unnoticed until researchers began unraveling the biology of obesity in the mid-1990s. As that science advanced, the problem became increasingly apparent: The underlying *mechanism* was being overshadowed by its *measurement*. BMI could describe body size, but it could not explain the disease itself.

BMI measures size, not biology. And even then, not particularly well, I would argue. Its origins trace back to measurements collected from nineteenth-century Belgian men – not a population intended to diagnose disease in today's diverse population. So, the next time you enter your height and weight into a BMI calculator, ask yourself: "Am I a nineteenth-century Belgian man?" Probably not. So don't take the number too seriously, and certainly don't let it define you. It should never be used on its own to diagnose overweight or obesity, regardless

of how insurance companies choose to define those terms. BMI remains a useful screening tool because it is quick, inexpensive, and easy to measure. But screening is not the same as diagnosis.

What distinguishes obesity, however, is not body size. Obesity is a state of excess adiposity in which the brain resists weight loss and actively defends a higher fat mass despite the resulting metabolic, physical, or mental health burden. That's the definition I use, within clinical reason. It isn't perfect, but few things in obesity medicine are. That's also what makes obesity medicine beautiful. When diagnostic criteria aren't rigid or binary, there is more room for nuance, clinical reasoning, and stronger therapeutic relationships with the people sitting across from us.

The Academic Debate Over Rulers

There is currently considerable debate about how obesity should be diagnosed. In 2025, an international commission published in *The Lancet Diabetes & Endocrinology* proposed a new clinical definition of obesity that combines BMI with measures of body fat and organ impairment. The goal is to distinguish between "clinical obesity," in which excess adiposity is causing health impairment, and "preclinical obesity," in which excess adiposity is present without measurable impairment. Accordingly, the commission recommends incorporating additional measures such as waist circumference, waist-to-hip ratio, and body composition into assessments.

Obesity medicine specialists, however, have not embraced this definition with open arms. In fact, there is considerable debate within our field. We know we are still arguing about the wrong variables. Whether we rely on BMI alone or add waist measurements, organ dysfunction, or expensive body composition scans, we are still trying to infer the presence of a faulty fuel gauge from static measurements. None of these

approaches helps us better identify who is most likely to benefit from treatment, which is the fundamental purpose of diagnosing obesity.

The purpose of diagnosis is to identify *who* will benefit from treatment.

For now, we largely rely on the eligibility criteria used in pharmaceutical trials to answer that question. Those trials established the product monograph, which you can think of as a medication's official "rulebook." It serves as guidance for physicians and pharmacists on how to prescribe a medication safely and effectively. Perhaps you've already been told that you don't "qualify" for a GLP-1 because your BMI is too low or because you don't have enough obesity-related health conditions. Those eligibility criteria were originally designed to determine who could enter a clinical trial, not necessarily who is most likely to benefit from treatment in everyday practice. In other words, body size became the gateway for selecting research participants, and those same criteria continue to shape who receives treatment today.

Body Composition Is the New BMI

As criticism of BMI has grown, a new measurement has begun taking its place: body composition. Today, it is increasingly common to hear that obesity should be diagnosed using body-fat percentage rather than BMI. Some clinicians recommend DEXA scans or, more commonly, bioelectrical impedance analysis (BIA) – a method that sends a tiny, harmless electrical current through your body to estimate how much of you is fat versus muscle to more accurately diagnose obesity or monitor treatment response. At first glance, the argument sounds convincing. BMI measures body size, whereas body-fat percentage measures fat directly. Surely that must be better.

But before replacing one yardstick with another, we should ask a more important question: What problem are we actually trying to solve?

The same criticisms levelled at BMI also apply to body-fat percentage thresholds, and in some ways, they may be even *less* helpful for diagnosis. Body-fat percentage also tells us nothing about where fat is stored. As we'll discuss later, where fat is stored is often far more important than how much fat is stored.

How can body-fat percentage thresholds be less helpful than BMI? One reason is that many body composition methods were developed and validated in relatively narrow populations – for example, otherwise healthy young White men – that do not reflect the diversity of people or health risks encountered in everyday obesity practice.

We know that different ethnic groups have different relationships between body-fat percentage and metabolic risk. Some Asian populations, for example, develop metabolic complications at lower levels of body fat than populations of European descent. Ethnic-specific BMI thresholds are available for reference. Equivalent body-fat percentage thresholds have never fully solved this problem. Attempts to develop ethnic-specific body-fat cutoffs have largely been derived from the corresponding BMI cutoffs. As you can appreciate, a surrogate measure cannot be more valid than the standard from which it was derived.

Many critics reject BMI because a single cutoff cannot apply equally to everyone. Replacing it with body-fat percentage thresholds recreates the same problem while introducing new sources of uncertainty. Body-fat percentage may appear biologically more precise, but there are currently no validated thresholds that correspond to disease risk across different ages, sexes, and ethnic groups. The Obesity Medicine Association acknowledges this, noting that its body-fat percentage classifications are based on a combination of scientific evidence and

expert clinical opinion, and that these thresholds may change as additional evidence becomes available.

We changed the ruler, but not the problem.

All static measures share the same conceptual limitation. They attempt to draw a hard diagnostic line across a biological continuum: above this number, obesity; below it, not obesity. But where exactly should that line be drawn? Surprisingly, no one really agrees.

A common source of confusion is that there is no World Health Organization (WHO) body-fat percentage definition of obesity comparable to the WHO BMI classification. Instead, body-fat percentage reference ranges emerged from a mixture of fitness classifications, expert opinion, population averages, and attempts to translate BMI categories into body-fat categories. The most commonly quoted cutoffs are greater than 25 percent body fat in men and greater than 35 percent in women. These thresholds, however, were not developed to identify the precise biological point at which obesity begins causing harm.

Body composition analysis has become increasingly embedded in clinical practice, yet its appearance of scientific certainty often exceeds the evidence supporting it. In some respects, body-fat percentage creates an even greater illusion of precision than BMI. The limitations of BMI as a population measure are widely recognized. Body-fat percentage, by contrast, feels more sophisticated, and a number reported to one decimal place can create the impression of greater accuracy than actually exists. If you are pursuing body composition analysis because you believe it will finally reveal the precise nature of your obesity, you may be searching for precision where precision does not exist.

A more precise measurement of adiposity is still only a measurement of adiposity. The central question remains unanswered: Does this person have a chronic disease that is

causing harm or increasing the risk of harm, and would treatment meaningfully reduce that risk?

If predicting cardiometabolic risk is the goal, waist-to-height ratio may be one of the most useful body measurements. Waist-to-height ratio compares your waist size with your height; a ratio of 0.5 or higher generally signals a higher risk of weight-related health complications. Studies often show it outperforms BMI in predicting diabetes, hypertension, and cardiovascular disease because it reflects abdominal fat relative to height. This is why some experts recommend keeping your waist circumference to less than half your height. Yet it also has limitations: It estimates risk, not disease. It cannot determine whether a person has obesity, explain why excess adiposity developed, or identify the biological mechanisms that maintain a higher body weight.

This does not mean body composition analysis is useless. These measurements can provide valuable information, such as tracking changes in fat mass and lean mass (muscle, bone, and other non-fat tissues) over time. But that is very different from suggesting that everyone needs a body composition scan to determine whether they have obesity or whether treatment is improving their health.

If this discussion of BMI and body composition is starting to feel a little technical, I want to reassure you that the field is moving in a more practical, patient-centred direction. The Canadian Adult Obesity Clinical Practice Guideline has been a global leader in this shift, placing less emphasis on BMI and more emphasis on clinical judgment rather than an ever-growing collection of measuring tools. The definition of obesity should be grounded in physiology and clinical science. As clinicians and researchers, we need to take ownership of how this disease is conceptualized. Unfortunately, we're still debating the ruler.

SIDEBAR

The Illusion of Precision

Sarah came to her appointment to discuss how she was responding to a GLP-1 medication. Before we even began talking about her progress, she had a request.

"I think I should get a DEXA scan first."

I asked her why.

"My friend sent me a video on Instagram," she explained. "The doctor said everyone taking a GLP-1 should get a baseline DEXA scan. He said you need to know your body-fat percentage and muscle mass first. Otherwise, you're basically treating obesity blindly."

Sarah wasn't being unreasonable. In fact, she was doing what many patients do: trying to make sense of an overwhelming amount of information. She wanted to be informed. She wanted to do things correctly. Most of all, she wanted certainty.

The video seemed convincing. It showed colourful bodycomposition reports, precise percentages, and detailed measurements of fat and lean mass. Everything looked scientific. More importantly, it appeared to answer a question that many people living with obesity quietly carry:

What is actually wrong with me?

If the scan could reveal exactly how much fat she had, perhaps it could finally explain why her body behaved the way it did.

I explained that a DEXA scan could certainly estimate how much fat and lean tissue she had. It could even estimate where some of that fat was distributed. But it could not determine whether she had obesity any more accurately than a careful clinical assessment. It could not explain why her body defended its current weight, why previous weight-

loss attempts had been difficult to sustain, or whether she would respond to treatment.

Sarah looked puzzled.

"Then why are so many people recommending it?"

It was a fair question.

The growing enthusiasm for DEXA reflects a broader desire to find a better number – one that can finally explain who does and does not have obesity. That desire is understandable, but it risks keeping the conversation focused on measurement rather than disease. As we'll see, even sophisticated body composition scans have important limitations that make them less definitive than they first appear.

DEXA: More Data, Same Blind Spot

Not all body fat is the same. Some fat is stored beneath the skin (subcutaneous fat), while some surrounds the internal organs (visceral fat). Advanced imaging techniques such as CT and MRI can distinguish these fat compartments directly, while DEXA provides useful estimates. This distinction matters because visceral fat is more strongly associated with many of the health complications linked to obesity.

Proposals have been made to incorporate body composition DEXA scans into clinical practice to support the diagnosis of obesity. A DEXA scan is a type of low-dose X-ray that measures how much of your body is made up of bone, fat, and muscle.

The intention, again, is understandable. Greater precision may help us estimate risk more accurately and reduce misclassification.

But the more practical question is: Would adopting more complex diagnostic operations improve health outcomes?

Unless greater diagnostic complexity meaningfully changes the answer to who is most likely to benefit from treatment, it

risks becoming another measurement without meaningful clinical impact.

In many cases, the diagnosis of obesity is not ambiguous. A patient with significant excess adiposity and type 2 diabetes does not require a DEXA scan to determine whether treatment is likely to help. The disease has already declared itself. The metabolic consequences are present, and treatment is likely to improve health. In patients with clear physical, metabolic, or functional complications, the diagnosis is rarely subtle enough to justify more complex testing. The regulatory system is misregulated, and medical treatment is appropriate.

So who does the debate over better diagnostics actually matter for? Patients whose excess adiposity is less obvious or whose metabolic markers remain within normal ranges. In these situations, more sophisticated measures such as waist-to-height ratio or imaging-based body composition analysis may provide additional descriptive information. But they do not fundamentally change the most important clinical question: Will treatment improve this person's health?

Whether excess adiposity is contributing to metabolic, physical, or mental health impairment can usually be determined through a careful clinical assessment that includes a patient's history, physical examination, and laboratory findings. If the answer is yes in any of these domains, treatment is medically indicated. Additional data from a DEXA scan rarely changes that conclusion, even in people with lower BMI values.

If the answer is no across all three domains, treatment becomes an elective decision. Excess weight can be approached as a risk factor, with management guided by the patient's values and willingness to reduce future risk. If someone has a high tolerance for that risk or places little value on reducing it, there may be no medical indication for treatment. Instead, the conversation becomes one of risk tolerance, preferences, and shared decision-making.

Diagnosis Is About Treatment, Not Measurement

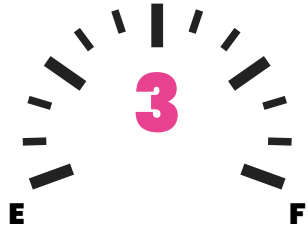
The purpose of diagnosing obesity is not simply to apply a label. It is to identify who stands to benefit from treatment.

This is the pivot most people miss. That does not mean waist measurements or DEXA scans are useless. They provide helpful information about body composition and can be useful – albeit relatively cost-ineffective – for monitoring changes over time. But at the diagnostic stage, they rarely change medical recommendations in a meaningful way.

Medicine is not about chasing increasingly intricate definitions. It is about improving metabolic, physical, and mental health outcomes. In obesity medicine, the most important question is not "How precisely can we measure excess fat?" It is "Will treating excess adiposity meaningfully improve health?"

At this point, I hope I've convinced you that despite the limitations of BMI, body composition measurements are not inherently better, and you probably don't need a DEXA scan. Obesity is a clinical diagnosis. A clinical diagnosis is not based on a single measurement. It is the determination that a disease is present and that treatment is likely to benefit the person sitting in front of us.

If you're still with me, you're probably asking, "Okay, but how did I develop obesity in the first place?" The answer begins long before weight gain becomes visible. It starts with genetic susceptibility and repeated exposure to biological and environmental pressures. Over time, these pressures gradually shift the body's fuel gauge toward "empty," causing the body to store and defend more fat. Understanding the most important pressures that shift the fuel gauge is where we turn next.



CHAPTER

STRESS CAUSES STORAGE

OBESITY DOES NOT APPEAR overnight, as you now know. Nor does it develop because of a single meal, a single stressful year, or one moment of weakness around chocolate. It develops gradually, as our biology responds to repeated pressures from the environment. What begins as a series of subtle stressors eventually accumulates enough force to shift the body's fuel gauge toward "empty," creating a new defended state. This process unfolds over years. In that sense, obesity is best understood as a chronic adaptation to repeated environmental pressures. Storage is the result.

Storage Is a Stress Response

When most people think about stress, they think about emotional stress: work deadlines, financial pressures, family conflict, major life events. But biologically, stress is anything that disrupts the body's internal balance and requires it to adapt. Some stressors are obvious. Others are so subtle that we barely notice them, yet they accumulate over time.

These stressors are different for everyone because they arise from our unique interactions with the environment. For some people, poor sleep, chronic inflammation, illness, disordered

eating patterns, or weight-promoting medications create ongoing physiological stress. Highly processed foods often fall into this category as well. While they may not feel stressful in the moment – in fact, they're designed to be pleasurable – they can create internal conditions that the body experiences as instability rather than balance.

Processed foods are associated with low-grade inflammation and metabolic strain, which can shift the body toward energy conservation. Rapid spikes and drops in blood sugar may trigger swings in hunger and activate stress hormones that increase appetite. At the same time, highly engineered, hyperpalatable foods can overstimulate the brain's drive to keep eating, reinforcing cycles of craving and overconsumption. The result is a physiological environment that signals instability rather than balance. The body interprets these conditions as a form of energy uncertainty and responds by defending a higher fat mass as a safeguard against future stress.

Even when exposed to the same stressors, not everyone's body responds the same way. How we respond is influenced, in part, by our genetics. You've probably noticed this in real life: Some people can eat more without gaining much weight, while others gain weight more easily under the same conditions. Genetics help determine how each person's body regulates weight and largely explain why some people are more susceptible to obesity than others. Before examining the environmental and biological stressors that influence weight, we first need to understand the genetic foundation on which they act.

The Blueprint: Genetics and Biological Vulnerability

Every body begins with a genetic blueprint.

Long before calorie counting, gym memberships, or medications entered the picture, your genes were shaping how your weight-regulation system was wired. They influence how sensitive you are to hunger, how quickly you feel full, and how

rewarding food feels to your brain. They also influence how efficiently your body stores energy and how strongly your metabolism adapts when your weight changes.

Twin studies, which compare identical twins raised in similar or different environments, have shown that body weight is highly heritable. Roughly 60 percent to 70 percent of the variation in body weight is explained by genetics. These are not single-gene mutations that "cause" obesity in a simple, light-switch manner. Rather, weight regulation is shaped by hundreds of genetic variants, each making a small contribution – more like a complex mixing board with many dials than a single switch that can be flipped on or off.

Genome-wide association studies (large studies that scan the DNA of thousands of people to find genetic patterns linked to a trait) have identified hundreds of these genetic variants. Most are expressed in the *brain*, particularly in regions involved in appetite regulation and food reward. In other words, many of these genes influence *feeding behaviour*. This can affect how easily we're distracted by food, how intensely we crave highly palatable foods, and how strong our drive is to seek food when it's available.

Polygenic Obesity

The most common form of obesity, known as polygenic obesity, is not caused by a single obesity gene. Instead, it reflects the combined effects of hundreds – and likely thousands – of common genetic variants, each contributing a very small amount to a person's susceptibility. Individually, these variants have almost no effect. Together, they can substantially influence how the brain regulates body weight.

From an evolutionary perspective, these traits made sense. For most of human history, food was scarce or unpredictable. Survival favoured individuals who were highly responsive to food cues, efficient at storing energy, and motivated to seek

food when it became available. Traits that helped our ancestors survive periods of scarcity increased the likelihood that they would reach reproductive age and pass on their genes. This idea is commonly known as the "thrifty gene" hypothesis. It proposes that genes promoting efficient energy storage and food-seeking behaviours were advantageous during times of famine.

In today's environment, where food is abundant, constantly available, and engineered for palatability, these same genes increase our vulnerability to weight gain and obesity. We have caveman genes in a spaceman's world. One of the clearest examples of this genetic vulnerability is binge-eating disorder, a condition in which the biological systems that regulate food reward and fullness become dysregulated in ways that promote overeating.

When Self-Control Is Genetic

Some of the most powerful drivers of weight are wired into the brain long before willpower ever enters the picture. Conditions that impair regulation of eating or executive functioning make this impossible to ignore. Regulation of eating is the brain's ability to guide when we start eating, how much we eat, and when we are able to stop. Executive functioning is the system that lets you pause, override impulses, and stay aligned with long-term goals. When these systems are not functioning optimally, it can become much harder to stop, reflect, and act in ways that align with our long-term intentions.

Binge-Eating Disorder

Consider binge-eating disorder (BED), the most common eating disorder, affecting approximately 1 percent to 2 percent of the population. BED is fundamentally a disorder of food-reward regulation. The brain's "wanting" system is turned up, creating a powerful drive toward food, while the brain's "pause and

reconsider" system is less effective. Under stress, this imbalance often becomes even stronger. Food feels unusually compelling, and once eating begins, stopping can be difficult. In many ways, BED reflects a mismatch between a powerful drive to seek food and weaker signals that bring eating to an end.

In a food environment engineered for constant stimulation, that biology is amplified. Left unrecognized and untreated, BED predictably drives weight gain.

Attention-Deficit/Hyperactivity Disorder

BED is not the only condition that challenges the idea that eating is simply a matter of willpower. Attention-deficit/hyperactivity disorder (ADHD) offers an important example of impaired executive functioning. ADHD is one of the most heritable conditions in medicine, with genetic influences comparable to those of height. It arises from differences in the brain's dopamine networks (the brain's reward and motivation circuits, which also help drive the urge to seek out food), particularly within the prefrontal cortex – the region responsible for planning, self-regulation, and impulse control. In adults, ADHD rarely resembles the stereotype of a child bouncing off the walls. More often, it presents as emotional reactivity, inconsistency, distractibility, and chronic difficulty following through, even when the intention is genuine.

Like BED, ADHD is often mistaken for a problem of motivation or self-discipline. It is not. ADHD is fundamentally a disorder of executive functioning – the brain's ability to plan, organize, prioritize, regulate emotions, resist impulses, and translate intentions into action. Executive functions are the brain's management system. As previously mentioned, they help us pause before acting, follow through on plans, and stay aligned with our long-term goals. When these systems are less efficient, everyday life can become incredibly frustrating. You know exactly what you're "supposed" to do, yet following

through remains unexpectedly difficult. The problem is not a lack of knowledge; you already know what to do. The problem is the brain's reduced ability to translate intention into action.

This matters enormously in obesity medicine. Long-term weight management depends on executive functioning at many levels: planning meals, grocery shopping, cooking at home, delaying gratification, maintaining routines, resisting impulses around hyper-palatable foods, recovering from setbacks, and navigating an environment designed to capture our attention. When these systems are less efficient, everyday tasks become substantially more difficult because they rely on brain functions that are working harder than most people realise.

Viewed through the lens of weight management, these challenges are often misunderstood and mislabelled as "poor adherence," "emotional eating," or a "lack of discipline." What we're often seeing, however, is a brain with less effective braking systems trying to navigate an environment engineered to reward immediate action. Highly palatable foods, constant food cues, and instant gratification all widen the gap between intention and behaviour. A person may recognize they're not physically hungry, know that eating the extra piece of cake doesn't align with their goals, and still find themselves reaching for it almost automatically.

Many traits associated with ADHD, such as rapid environmental scanning, novelty seeking, and quick responsiveness, would likely have been advantageous in environments where detecting change improved survival. The difficulty is that today's world presents an endless stream of highly rewarding distractions, with ultra-processed foods among the most powerful. A brain that evolved to notice *everything* must now compete with an environment specifically designed to capture its attention.

The clinical implications for obesity care are difficult to ignore. Untreated ADHD is consistently associated with higher

rates of obesity, whereas appropriate treatment substantially reduces that excess risk. This mirrors what we see in BED. In both conditions, weight gain is driven less by a lack of knowledge than by differences in how the brain regulates reward, impulses, and behaviour within a food environment that continually challenges those systems.

My hope is that you're beginning to notice a subtle pattern: The genes most strongly associated with obesity don't primarily influence metabolism; they influence feeding behaviour. Patients often tell me they believe they inherited a "slow metabolism." It's an understandable assumption. However, research consistently shows that the strongest genetic influences in polygenic obesity affect appetite, fullness, cravings, and food reward, not metabolism. When these predispositions coexist with conditions that impair executive functioning and behavioural regulation, such as ADHD or BED, the cumulative effect can make weight regulation substantially more difficult.

By now, we've been talking almost entirely about one question: How much fat does your body defend? Genetics has shown us that much of the answer resides in the brain. These inherited differences shape how strongly the brain responds to signals of hunger, fullness, and reward and, ultimately, how strongly it defends its energy stores.

But there's a second question that's just as important: Where does your body choose to store that fat? It turns out these questions are related, but not identical. The genes that influence fat distribution are largely expressed within adipose tissue itself. In other words, one set of biological processes helps determine how much fat we store, while another helps determine where we store it.

This matters because not all body fat behaves the same. Two people can carry the same amount of body fat yet have very different health risks depending on where that fat is stored. A more complete understanding of obesity requires answering

two fundamental questions: How much fat is the body defending, and where is it being stored?

When Location Is Genetic

Genes don't just influence how much fat your body defends; they also influence where that fat is stored. Some people naturally store more fat around their hips and thighs, while others store it primarily around the abdomen and upper body. Researchers have identified roughly twenty genes involved in fat distribution. These genes are expressed and act directly within adipose tissue.

This is also why "spot reduction" never really worked – not with the ThighMaster in the 1990s, and not today with endless abdominal exercises. We can strengthen specific muscles, but we can't tell our bodies where to lose fat. Short of liposuction, fat loss follows the biological blueprint established by our genes. But the real importance of where fat is stored has very little to do with how it looks and everything to do with how it behaves.

First, fat is not simply a passive storage depot. It is a living, hormonally active organ, and where it is stored influences how it functions. Subcutaneous fat – the fat beneath the skin – is relatively stable. It serves as the body's primary energy reservoir, safely storing excess fuel with relatively little disruption to normal metabolism.

Visceral fat is different. Stored around the abdominal organs, it is far more metabolically active and hormonally responsive. It releases inflammatory signals, interferes with insulin function, and is strongly associated with fatty liver disease (a buildup of fat in the liver that can affect how well it works), type 2 diabetes, cardiovascular disease, and other metabolic complications. This difference explains why two people can have the same body weight – or even the same BMI

– yet very different metabolic health. The difference lies in where their fat is stored.

To understand why fat location matters – and whether you're at increased metabolic risk – it helps to think of each of us as having a unique, genetically influenced safe fat-storage capacity. Each of us has a unique capacity to safely store excess energy within subcutaneous fat. That capacity is influenced by genetics, life stage, and hormonal factors such as puberty. As long as excess energy can be stored safely within subcutaneous fat, the body can often buffer it without immediate metabolic consequences.

But these fat cells are not infinitely expandable.

Once that storage capacity is exceeded, excess energy has to go somewhere. Fat begins accumulating in places that were never designed to store it, including around the abdominal organs and within the liver. This process is known as lipotoxicity. Fat in these locations behaves differently. It promotes chronic low-grade inflammation, disrupts insulin signalling, alters blood sugar regulation, and increases the risk of metabolic disease.

This helps explain why not everyone living with obesity develops type 2 diabetes, while some people develop type 2 diabetes despite carrying only a modest amount of excess weight. The difference lies in how much fat their bodies can safely store before that storage system becomes overwhelmed. In other words, the difference is their *absolute safe fat-storage capacity*.

SIDEBAR

Why Liposuction Sucks for Obesity

Think of your body's safe fat-storage capacity as a series of storage closets. Some people have more room than others. Subcutaneous fat—the fat just beneath the skin—is your main storage closet. Within it, fat cells act like shelves, storing excess energy safely and expanding the closet's capacity as needed.

Each person is born with a different number of closets and shelves. Some people have spacious walk-in closets with plenty of shelving. Others have smaller closets with far less storage space. These differences are largely determined by genetics.

As long as your closets have room, excess energy can be stored safely with little disruption. You may gain weight as the closets gradually fill, yet your metabolism remains relatively stable because the storage system is still functioning as intended.

But our closets are not infinite. When the shelves become full and the doors can no longer close, the overflow has to go somewhere.

Imagine those extra belongings spilling into the hallway, the kitchen, and other places never designed for storage. That's visceral fat. This is lipotoxicity. The problem isn't simply that there's more stuff; it's that your safe storage capacity has been exceeded.

Now, imagine that instead of reducing the amount of stuff entering the house, we start removing the closets.

Liposuction removes subcutaneous fat cells, or, in this analogy, the closets and shelves. The room may look cleaner afterward, but the amount of storage space has been reduced. That may reduce the body's capacity to safely store excess energy beneath the skin without addressing the

underlying biology driving fat storage. Removing storage space doesn't eliminate the drive to store energy; it reduces the body's capacity to store it safely.

This is why liposuction may change our appearance, but it does so at the expense of reducing safe fat-storage capacity. Keep in mind that liposuction is generally limited to small, localized fat deposits, which by themselves do not meaningfully increase a person's overall obesity risk. While this is unlikely to cause obesity, it runs counter to the goals of obesity treatment. More importantly, it is not a treatment for obesity. The goal is not to remove the closets or tear out the shelves. The goal is to reduce the pressure within the storage system – particularly the overflow – so that fat can once again be stored safely, especially within subcutaneous fat rather than visceral fat, where metabolic risk is highest.

From an evolutionary perspective, visceral fat likely exists because it can provide a rapid source of energy when it's desperately needed. Stored triglycerides within visceral fat can be mobilized much more quickly than those stored in subcutaneous fat. Because visceral fat is designed to be mobilized rapidly, it is also lost more readily during weight loss. This means that even modest weight loss can produce disproportionately large reductions in the fat that carries the greatest metabolic risk. A 5 percent reduction in body weight is associated with roughly a 9 percent reduction in visceral fat, while a 16 percent reduction is associated with approximately a 30 percent reduction.

The Genetics That Came Before You

Another layer of obesity biology is epigenetics, which helps explain why the risk of obesity can begin long before we have any control over our lifestyle – even before we're born. Epigenetics influences how our genes are expressed, or how the

instructions within our genetic blueprint are read. Factors such as maternal obesity, gestational diabetes, prenatal and early-life nutrition, and childhood adversity can influence genes involved in appetite and energy regulation long before we make our first independent choices.

One of the best examples of epigenetics comes from the Dutch Hunger Winter of 1944–1945, when a severe famine left many people with almost nothing to eat. Years later, researchers discovered that babies developing in the womb during the famine were more likely to develop obesity and metabolic disease as adults.

What made this finding so remarkable was that researchers compared these individuals with their own brothers and sisters who were born before or after the famine. They had the same parents, grew up in the same home, and shared much of the same DNA. The greatest difference was the environment they experienced before birth.

It appears that the developing fetus interpreted the lack of food as a signal that the outside world would also be scarce. In response, the body adapted by becoming more efficient at conserving energy and storing fat – an advantage if food truly remained scarce. But when those children grew up in a world where food was plentiful, that same adaptation became a disadvantage, increasing their risk of obesity. The DNA itself never changed; what changed was how those genes were "read." That's epigenetics: changing the instructions, not the genetic blueprint.

If this discussion feels heavy, it's because it challenges a message many of us have heard our entire lives: that obesity is simply the result of poor choices. My purpose in walking you through genetics and epigenetics isn't to convince you that change is impossible. It's to show you that much of your obesity biology was established long before willpower ever entered the picture.

We don't blame ourselves for inheriting our height, eye colour, or shoe size because we understand those traits are biologically determined. Obesity feels different because it involves behaviours such as eating and physical activity, making it easy to assume that biology no longer matters. But as you've seen, the brain systems that regulate fullness, energy storage, food reward, and even aspects of executive functioning are themselves strongly influenced by genetics. Willpower cannot rewrite those systems any more than it can make someone six inches taller.

When you understand what you are actually up against, you can stop asking "What's wrong with me?" and start asking "Given the biology I inherited, what treatments and strategies are most likely to help me succeed?"

Why Your Fuel Gauge Reads Empty

Think back to the fuel gauge.

Your genetics determine how sensitive your fuel gauge is to falling fat stores. Some people inherit a gauge that remains comfortably near "full" until their fat stores become quite low. Others inherit a much more sensitive gauge, one that drifts toward "empty" much sooner and triggers stronger biological drives to eat and store energy.

Epigenetics can influence where that needle is calibrated before birth.

Then, throughout life, environmental stressors push that gauge toward "empty." Poor sleep, chronic stress, highly processed foods, certain medications, and conditions that impair executive functioning all increase the brain's perception that energy is becoming scarce. As these stressors accumulate, the pressure on the fuel gauge increases, pushing it progressively closer to "empty."

This also explains why obesity is rarely caused by a single factor. Instead, it develops as multiple biological and environ-

mental stressors gradually apply enough pressure to shift the fuel gauge toward "empty." Understanding this changes the question from "What caused my obesity?" to "What is pushing my fuel gauge toward empty, and how can I reduce those pressures?" That's where we turn next.

The Stressors That Push the Needle

We've spent the last several sections exploring the fuel gauge you're born with. But that gauge doesn't operate in isolation. Whether it begins signalling low fuel depends, in part, on the environment in which it interacts with. Some of us inherit a biology that makes us more susceptible to obesity, but without exposure to environmental stressors, we may never develop obesity at all.

Think about asthma. Imagine two people living in a city filled with cigarette smoke and air pollution. Everyone breathes the same air, but not everyone develops wheezing or struggles to breathe. The difference is that some lungs are genetically more sensitive to those environmental triggers. Now imagine moving one of those people to a clean mountain town. Their genetic predisposition hasn't disappeared, but without the trigger they may experience few symptoms – or perhaps never have been diagnosed with asthma at all.

Obesity works in much the same way. Whether – and how – we interact with environmental stressors reveals this underlying biology. The rest of this chapter explores the most important stressors that push the fuel gauge toward "empty." Each one applies pressure to the system. As those pressures accumulate, the brain becomes increasingly convinced that fuel is running low, even when the body's energy stores remain abundant.

Stressor: The Modern Food and Drink Environment

Imagine your brain's fuel gauge sitting comfortably in the middle. Now begin adding the pressures of modern life: ultra-

processed foods engineered for reward, constant access to calorie-dense foods and sugary drinks, increasingly sedentary routines, fragmented sleep, and chronic stress. Each of these nudges the gauge toward "empty." On their own, any one of these pressures may have only a modest effect. Together, however, they accumulate until the brain becomes increasingly convinced that fuel is running out, even when fat stores remain abundant.

When the brain believes fuel is scarce, it responds exactly as it was designed to. Hunger increases, fullness becomes harder to achieve, food becomes more rewarding, and energy expenditure (the amount of energy your body burns) decreases whenever possible. The body shifts toward storing rather than burning energy. The farther the fuel gauge drifts toward "empty," the more aggressively the brain defends its energy stores. Today's food environment continually pushes the fuel gauge toward a state that favours fat storage. Of all these pressures, perhaps none is more powerful than ultra-processed foods.

Food Without Limits

The modern food environment – particularly in North America – is unlike anything our biology evolved to navigate. Highly processed foods are available almost everywhere, at any time of day, and can arrive at our doorstep within minutes with just a few taps on a phone. Many of these foods are intentionally engineered to be exceptionally rewarding, encouraging us to eat beyond our natural feelings of hunger and fullness. As food processing has evolved into ultra-processed foods, our environment has become increasingly "spacier," while our biology has remained remarkably unchanged. We now live in a world of twenty-four-hour convenience stores stocked with ice cream, doughnuts, and sugary drinks, alongside delivery apps that provide instant access to nearly any meal we crave. Our envir-

onment is changing far more quickly than our biology can adapt, continually stressing biological systems that evolved for scarcity rather than abundance.

Why Am I Still Hungry? I Just Ate a Bag of Potato Chips!

Have you ever been really hungry, only to find that the only food nearby was a vending machine?

So you buy a bag of potato chips.

You look at the nutrition label. It says there are a few hundred calories. That should keep you going for a while, right?

But twenty minutes later, you're hungry again.

How is that possible?

Your body isn't counting the calories printed on the bag. It's responding to the signals that food sends to your brain – and those aren't the same thing.

Consider the difference between eating a baked potato and eating a bag of potato chips.

A whole potato still contains the water and fibre that nature packed into it. It takes time to chew, digest, and absorb. As it moves slowly through your digestive system, it stretches your stomach and reaches the lower part of your gut, where it stimulates the release of natural fullness hormones such as GLP-1, GIP, and PYY – other gut hormones that work alongside GLP-1 to help you feel full. Those hormones send a simple message to your brain:

"Food has arrived. You can stop looking for more."

The energy from that potato also enters your bloodstream gradually because it remains packaged with fibre, providing a steady supply of fuel over the next several hours.

Now compare that with potato chips. They're made from the same potato, but after processing, they're no longer the same food. Much of the fibre and water have been stripped away. In many ways, ultra-processed foods are partially predigested. Your body doesn't have to do as much work to break them

down, so they're absorbed much more quickly. Instead of a slow, steady stream of energy, your bloodstream receives a rapid surge.

Because these foods move through the digestive system so quickly, they spend less time reaching the lower gut, where the strongest fullness signals are generated. In other words, they deliver energy faster than they deliver fullness. That's one reason a bag of potato chips can leave you wanting more food, even though you've already eaten hundreds of calories.

Here's a surprising fact: It usually takes four or five potatoes to make one bag of potato chips. Now imagine trying to eat four or five whole baked potatoes. Most people couldn't do it. The potatoes would fill your stomach, take time to digest, and gradually stimulate your fullness hormones. Long before you reached the fifth potato, your brain would tell you to stop.

The chips don't activate those same brakes nearly as effectively. The more a food is processed, the easier it becomes to eat large amounts before your brain has time to recognize that you've had enough.

Then there's another layer to the story: design. Not all processed foods are the same. Processed foods are foods that have been altered from their natural form to make them safer, last longer, or easier to prepare, such as frozen vegetables, canned beans, plain yogurt, or whole-grain bread. Ultra-processed foods are different. They are industrially manufactured products made largely from refined ingredients and additives that are designed to be highly rewarding, convenient, and easy to overeat. Think of foods like potato chips, candy, soft drinks, instant noodles, and many packaged snacks.

With ultra-processed foods, food scientists combine sugar, refined starches, fat, salt, texture, and flavourings to create foods that are exceptionally rewarding. The longer you keep reaching into the bag, the more successful the product is. For people whose brains are genetically more sensitive to food

rewards, this environment becomes even more challenging. It's like turning up the volume on an already loud speaker. For some people, that creates cravings that feel overwhelming because these foods were designed to stimulate the brain's reward system far more intensely than nature ever intended.

A helpful comparison is nicotine. Tiny amounts of nicotine naturally occur in vegetables such as potatoes and eggplant. Yet no one becomes addicted to potatoes. Why? Because the nicotine is present in such small amounts and is absorbed very slowly. Nicotine only becomes highly addictive after it has been extracted, purified, concentrated, and delivered rapidly to the brain. The molecule didn't change. The processing did.

When natural protections are stripped away and ingredients are refined, concentrated, and engineered for rapid absorption, they stimulate the brain's reward system much more intensely.

Sugary Drinks Are Fat Drinks

One of the biggest changes in our modern food environment isn't just what we eat; it's what we drink.

Sugary drinks include regular soft drinks, sweetened coffees, energy drinks, sports drinks, sweet teas, slushes, fruit punches, and even many fruit juices. Although these drinks seem very different, they all have one thing in common: They deliver large amounts of sugar rapidly.

Before going further, it helps to understand what we mean by "sugar." Sugar is a general term for sweet-tasting carbohydrates. Two of the most important are glucose and fructose. In table sugar (sucrose), they're present in roughly equal amounts – about half glucose and half fructose. Glucose is the body's primary fuel and can be used by your muscles, brain, and almost every other cell for energy. Fructose, on the other hand, is handled very differently. It's processed primarily by the liver.

Sugary drinks contain far more sugar than most people realise. Even fruit juice, which is often viewed as a healthy choice, can act more like a sugar delivery system than a piece of fruit. The next time you're at the grocery store, pick up a bottle of apple or pomegranate juice and look at the nutrition label. The amount of sugar may surprise you.

Why are sugary drinks more harmful than they seem?

To understand this, it helps to picture your liver as a fat factory. Sugary drinks – including soft drinks and fruit juice – deliver sugar directly to this factory. In contrast, when you eat a whole piece of fruit, you're also eating fibre, water, and the fruit's natural structure. Those components slow digestion, so sugar enters your bloodstream gradually. It's like a series of small delivery trucks arriving at the factory throughout the day.

But when fruit or sugarcane is processed into juice, soft drinks, or other sweetened beverages, most or all of that fibre is removed. Without that natural brake, sugar is absorbed much more quickly. Instead of several small deliveries, it's like a fleet of trucks arriving at the factory all at once.

Remember, about half of that sugar is fructose. Unlike glucose, which nearly every cell in your body can use immediately for energy, fructose goes straight to the liver. Once it arrives, it stays there to be processed. When small amounts arrive gradually – as they do in whole fruit – the liver handles them without difficulty. But when large amounts of isolated fructose arrive quickly and repeatedly, the liver becomes overwhelmed.

The liver burns some of the fructose, stores some of it, and when large amounts arrive quickly – as they do in sugary drinks – it begins converting the excess into fat. In other words, drinking large amounts of isolated fructose is like sending a steady stream of raw materials directly to a fat factory. Without any natural slowdowns, the production line keeps moving, converting the excess into fat because that's the liver's default

response when more raw material arrives than it can immediately process.

This doesn't mean whole fruit is unhealthy. Quite the opposite. Whole fruit comes packaged with fibre and water that naturally slow digestion and trigger fullness, making it remarkably difficult to consume large amounts of sugar in a short period of time. The problem isn't the fruit; it's what processing did to it.

Alcohol Is the Queen of Fat Drinks

Here's something else that rarely gets talked about: Alcohol follows a very similar path to fructose. And I'm not talking about the sugar in cocktails; I'm talking about the alcohol itself.

The active ingredient in alcoholic drinks is *ethanol*. When ethanol reaches the liver, it jumps to the front of the line because the liver treats it as a priority. While the liver is busy processing alcohol, much of its normal work – including burning fat – is temporarily put on hold.

Different substance. Same fat factory.

This is why our modern food environment is so challenging. It isn't simply about willpower or making better choices. It's about living in a world that constantly delivers highly concentrated sources of sugar and alcohol in forms our biology was never designed to handle. A brain that evolved to respond strongly to rare opportunities for fuel is now surrounded by them every day. The result is a biological system that is continually pushed toward storing energy, even when we already have more than enough.

Stressor: Movement and Sleep Deprivation

An Environment That Removes Movement

Our bodies evolved in a world where movement wasn't optional. People walked long distances, gathered food, carried heavy loads, and hunted. You might assume that because our

ancestors were far more physically active, they burned many more calories than we do today.

Surprisingly, they didn't.

Research comparing modern hunter-gatherer populations with people living in Western countries has found that, despite being much more physically active, their total daily energy expenditure is remarkably similar.

How can that be? Because the body doesn't allow energy expenditure to increase without limit. Instead, it defends a relatively stable range so you don't burn through your fuel faster than you can replace it. When you become more active, you initially burn more calories. Over time, however, your body adapts. It may make you hungrier, encourage you to move less during the rest of the day, or adjust other energy-demanding processes. Together, these adaptations help prevent your total energy expenditure from continuing to climb. From an evolutionary perspective, this makes perfect sense. A body that could burn unlimited amounts of energy would struggle to survive when food became scarce.

This also explains why you often feel hungrier after a hard workout. Your body isn't trying to sabotage you. It's responding exactly as it was designed to. None of this means exercise is unimportant. Quite the opposite. Exercise is incredibly important – just not for the reason many of us were taught.

For most adults, maintaining good health and supporting weight regulation means aiming for roughly 150–200 minutes of moderate physical activity each week, or about 8,000–9,000 steps a day. The difference is that our ancestors accumulated this movement naturally. Today, many of us have to make time for it because modern life has engineered movement out of our daily routines. Cars, elevators, office jobs, streaming services, and food delivered to our door mean we can go through an entire day with very little physical activity.

Movement does far more than burn calories. It helps your body respond better to insulin, strengthens fullness signals, improves sleep, lowers stress, and supports many of the systems involved in weight regulation. Our bodies evolved to expect regular movement. When that movement disappears from everyday life, we lose one of the natural supports that helps keep our weight-regulation system functioning as it should.

Movement is just one of the natural supports your body relies on. Another is sleep.

A System Out of Sync

Like movement, sleep is one of the natural supports your body relies on to help regulate weight. When you're getting enough restful sleep, your brain is better able to balance the signals that control hunger, fullness, and energy use. But when sleep is cut short night after night, that balance begins to shift.

Research shows that adults who regularly sleep less than seven hours a night are more likely to develop obesity. In fact, for every hour below that amount, the risk increases by about 9 percent. That's because sleep helps keep the systems that regulate hunger and fullness functioning properly.

When you don't get enough sleep, your brain interprets it as a form of stress. Stress hormones rise, hunger signals become stronger, fullness signals become weaker, and your brain becomes more responsive to highly rewarding foods such as sweets and refined carbohydrates. In other words, your biology begins nudging you toward eating more at precisely the time your body is trying to conserve energy.

Sleep loss also encourages the body to store more fat, particularly around the abdominal organs as visceral fat. Over time, this can become a frustrating cycle. Poor sleep makes weight gain more likely, while excess weight can further disrupt sleep by increasing the risk of disorders such as obstructive sleep

apnea – a condition where breathing repeatedly stops and starts during sleep due to the airway becoming blocked, sometimes due to excess weight. Alcohol can also reduce sleep quality, even if it helps you fall asleep faster.

The good news is that this cycle can often be interrupted. Identifying and treating sleep disorders, reducing sleep disruptors, and making sleep a priority can help restore healthier hunger and fullness signals. In doing so, you give your body's weight-regulation system a better opportunity to function the way it was designed to.

Sleep isn't just about how long you sleep. It also matters when you sleep.

Your body runs on an internal twenty-four-hour clock called your *circadian rhythm*. As daylight fades, your brain releases a hormone called *melatonin*. Most people know melatonin as the hormone that helps us feel sleepy, but it also coordinates many of the body's overnight functions – including appetite regulation. As melatonin rises, your body gradually shifts away from eating and toward rest, repair, and recovery.

Modern life constantly disrupts this rhythm. Shift work, bright lights, late-night scrolling, and screens just inches from our faces can all delay the release of melatonin. As a result, your brain receives weaker signals that it's time to wind down. Hunger can linger longer, cravings become stronger, and your body stays in "fuel-up" mode when it should be focusing on rest and recovery.

Like processed foods and the loss of everyday movement, disrupted sleep is another modern pressure on the body's weight-regulation system. On its own, it may seem like a small factor. But weight regulation is shaped by the accumulation of many pressures, not just one. As those pressures build, they push the brain's fuel gauge toward storing more energy than the body actually needs.

For some people, the disruption goes beyond simply getting too little sleep. Certain sleep disorders further interfere with the brain's ability to regulate hunger, fullness, and body weight. Two conditions deserve special attention because they illustrate just how closely our internal body clock is linked to weight regulation: night eating syndrome (NES) and delayed sleep phase syndrome (DSPS).

Night Eating Syndrome

NES is a condition in which the body's internal clock becomes out of sync with eating. It affects about 1.5 percent of the general population and up to 8 percent of people seeking treatment for obesity.

People with NES often have very little appetite in the morning. As the day goes on, hunger gradually builds, with most of their food intake occurring in the evening. Some people also wake during the night feeling the need to eat before they can fall back asleep.

It's a problem of biological timing. Normally, as night falls, melatonin rises and helps coordinate the signals that tell your body it's time to rest rather than eat. In NES, that timing system doesn't work as well. Hunger stays switched on when it should be winding down, so eating shifts into the very part of the day when the body is meant for sleep, repair, and recovery rather than refueling. If this sounds like you, consider talking with your healthcare provider. They can help determine whether off-label treatment options may be appropriate for your situation.

Delayed Sleep Phase Syndrome

A similar pattern occurs in DSPS. In this condition, a person's internal body clock runs later than everyone else's. They may not feel sleepy until the early hours of the morning, no matter how hard they try to go to bed earlier. As a result, waking up for school or work can feel like climbing a hill every single day.

This is especially important for people with ADHD because up to 70 percent experience delayed sleep timing. When sleep is consistently shifted later, it can make the challenges of ADHD even greater by affecting focus, planning, impulse control, appetite regulation, and late-night eating.

When your internal body clock stays out of sync for long periods, the hormones that regulate hunger and fullness also become misaligned. Hunger signals remain switched on later into the evening, fullness signals become weaker, and eating shifts into a time when your body is naturally preparing to wind down, rest, and recover. If this sounds like you, consider talking with your healthcare provider. They can help determine whether melatonin supplementation might be a safe and appropriate option for you.

Stressor: Pressures Beyond Your Control

When Stress Rewrites Your Signals

Stress is another pressure that can quietly push the body's weight-regulation system in the wrong direction.

Our stress response evolved to help us survive short periods of danger. Imagine one of our ancestors escaping a predator. Their heart rate would race, stress hormones would surge, and, for a short time, hunger would disappear. Once the danger had passed, the body would settle, appetite would return, and life would continue as normal.

Modern stress is very different. Today's threats are rarely lions or bears. Instead, they're deadlines, financial worries, caregiving responsibilities, relationship challenges, social media, and the feeling of always being connected. Rather than coming in short bursts, these stressors can last for weeks, months, or even years. The body stays on alert far longer than it evolved to. When stress becomes chronic, the stress response stops being protective and begins interfering with the systems that regulate weight.

Chronic stress, however, can quietly promote weight gain by rewiring how we regulate comfort. Did you know that your brain's reward pathways also serve as stress-relief systems? When we eat highly palatable foods, endorphins – your brain's natural "feel good" chemicals – are released, creating a calming effect that helps us cope. During an immediate threat, cortisol is initially suppressed and appetite briefly shuts down. Think about going through a difficult breakup or receiving terrible news; you may lose your appetite for days. That's because, during an immediate threat, the body puts eating on hold. But as the threat begins to pass, hunger returns to help replenish the energy used to cope.

The challenge is that modern stress often doesn't end. As the brain repeatedly learns that highly rewarding foods provide temporary comfort, it strengthens that connection. Over time, eating can become less about physical hunger and more about relieving emotional stress. What once helped us survive short periods of danger can gradually become another pressure pushing the brain's weight-regulation system toward long-term weight gain.

Invisible Disruptors

Our environment also contains thousands of chemicals that our bodies interact with every day, often without us realising it. Some of these chemicals, called *endocrine disruptors*, are found in plastics, pesticides, and certain industrial materials. Researchers are studying how they may interfere with the hormones that regulate appetite, metabolism, and fat storage. This remains an evolving area of science, and there is still much we have to learn. But one thing is becoming increasingly clear: The environment surrounding us today is very different from the one our biology evolved in.

That doesn't mean these chemicals are a primary cause of obesity. Rather, they are another example of how many small

pressures can gradually accumulate over time. Like processed foods, poor sleep, chronic stress, and reduced movement, they may place additional strain on a weight-regulation system that is already working hard to stay balanced.

Sometimes we also see how powerful these biological signals are when they are suddenly removed. Smoking is a good example. Nicotine increases fullness and slightly increases the amount of energy the body burns each day. When someone quits smoking, those effects disappear. As the body adjusts, fullness decreases, and people gain an average of about five to seven pounds.

None of this means quitting smoking is the wrong decision. Quite the opposite. Quitting is one of the best things a person can do for their long-term health. It simply means that temporary changes in appetite and weight are common and expected. Healthcare providers can also help. For some people, medications such as bupropion, which is commonly prescribed to support smoking cessation, can reduce the amount of weight gained after quitting while also increasing the chances of becoming smoke-free. Ask your healthcare provider whether this option may be right for you.

When Medical Treatments Have Trade-Offs

Some prescription medications can also affect the body's weight-regulation system. They may increase hunger, change how the body uses energy, or make it easier to store fat. This can occur with certain antidepressants, antipsychotics, mood stabilizers, insulin, corticosteroids (steroid medications often used to calm inflammation, such as for asthma or arthritis), and some medications used to treat heart disease and high blood pressure.

It's important to remember that these medications are often essential – and sometimes lifesaving. The answer isn't to stop taking them. Instead, the goal is to understand how they affect

your body's weight-regulation system and manage those effects while continuing to treat the condition they were prescribed for.

Sometimes there are alternatives within the same medication class that are less likely to promote weight gain. For example, a healthcare provider may decide that switching from amitriptyline to nortriptyline, gabapentin to pregabalin, citalopram to escitalopram, or from beta-blockers such as propranolol, atenolol, or metoprolol to options such as carvedilol or labetalol is appropriate. These decisions are always individualized, but in some cases, thoughtful medication changes can reduce unintended weight gain without compromising treatment.

When there isn't a suitable alternative, we can often work around these medications. For many people, the benefits of treating the underlying medical condition far outweigh the downside of weight gain. In these situations, weight-management medications can help treat this side effect. It's similar to prescribing medication to protect the stomach when another necessary treatment causes acid reflux. The goal is to manage the side effects of weight gain while continuing the treatment that is improving your overall health.

All medication changes are context-dependent and must be considered within the context of your overall health and in consultation with your prescriber. Decisions about starting, stopping, or changing treatment should always be individualized and made in partnership with your prescribing clinician.

SIDEBAR

A Brief Note About Medical Decisions

Throughout this book, I'll discuss medications, behavioural strategies, and potential treatment approaches. While I'm a physician, your care is best guided by the healthcare provider who knows you and your medical history. Your health conditions, medical history, and medications are unique to you. Any decisions about starting, stopping, or adjusting medications – including those that may influence weight – should only be made in consultation with your healthcare provider.

If you're concerned that a medication you're taking may be contributing to weight gain, it's entirely reasonable to discuss it with your healthcare provider. In some cases, alternatives may exist. Whether they're appropriate for you depends on your individual circumstances. This book is designed to inform and empower you, not to replace personalized medical care.

Stressor: Life Stages and Hormones

It's easy to wonder whether hormones are the reason behind obesity. When your body feels different or your weight changes in ways you can't explain, hormones can seem like the missing piece of the puzzle.

You're not wrong to think about hormones. They play an important role in how your brain and body regulate weight. They influence how hungry or full you feel, where your body stores fat, and how strongly your body tries to hold on to its energy. But hormones don't cause obesity on their own. Instead, they influence how the body's weight-regulation system functions, usually in people who already have an underlying genetic susceptibility to obesity. In other words, hormones don't create the disease; they amplify a brain that is already more likely to perceive fuel as running low. This makes

the weight-regulation system even more sensitive to the pressures of today's environment. The point isn't to downplay the role of hormones but to recognize that they modify the disease rather than cause it. The underlying problem remains the same: a brain convinced that fuel is running low.

Think of hormones as another source of pressure on your brain's fuel gauge. They nudge the gauge toward "empty," but whether it moves – and by how much – still depends on your underlying genetics. Some people inherit a fuel gauge that is far more sensitive to these hormonal changes than others. When that genetic susceptibility is present, hormones can amplify the effect, especially when other biological and environmental pressures are already in place.

Before we continue, it's important to remember that hormones naturally change throughout life. The first major transition is puberty, when hormones reshape your body, influence where fat is stored, and guide many of the normal changes that occur as you grow. For many women, two additional hormonal transitions follow: pregnancy (if it occurs), and later, perimenopause and menopause. During each of these stages, weight regulation can become more challenging as the body adapts to a new hormonal environment. For people who are genetically susceptible to obesity, these hormonal shifts can make the brain's weight-regulation system more sensitive to storing and defending body fat. During perimenopause and menopause, declining estrogen also promotes a shift toward storing more fat viscerally, increasing the metabolic risks associated with obesity.

Pregnancy: When the Fuel Gauge Becomes More Protective

Pregnancy is one of the most remarkable things the human body can do. For nine months, it grows another human being. To make that possible, your brain naturally adjusts how it manages energy.

Using our fuel gauge analogy, pregnancy makes your brain more protective of its fuel reserves. Think of it like preparing for a long road trip with few gas stations. You'd want a larger safety margin before the tank gets low. During pregnancy, your brain does something similar. Because growing a baby requires extra energy, it encourages your body to store and protect a little more fuel.

This is completely normal. It's how a healthy body is designed to work.

The challenge is that this biology now exists in a world where highly processed, energy-dense foods are available almost everywhere. Research shows that women who gain more weight during pregnancy than recommended are more likely to retain some of that weight after delivery. Over time, especially across multiple pregnancies or alongside other life stressors, those small increases can gradually accumulate.

But pregnancy does *not* cause obesity. For people who inherit a more sensitive fuel gauge, it's simply another factor that nudges the brain toward storing energy. Add sleep deprivation, stress, and today's food environment, and the brain receives even stronger signals to protect its fuel reserves.

This also helps explain why postpartum weight isn't simply "baby weight" that someone failed to lose. Recovery from childbirth, caring for a newborn, and disrupted sleep all place additional demands on the body. From the brain's perspective, holding on to energy during this time is a protective response.

Seen through the lens of the fuel gauge, pregnancy is another example of how biology adapts to protect us. For some people, those adaptations gradually shift the level of stored energy the brain works to defend, making long-term weight regulation more challenging.

Perimenopause/Menopause

Perimenopause is another period when the biological systems that influence the level of body weight your brain defends can shift. As estrogen naturally fluctuates and declines, your body begins to store fat differently. Instead of storing most fat around the hips and thighs, it becomes more likely to store fat around the abdomen. Even if the number on the scale changes very little, this shift toward visceral fat increases the risk of conditions such as type 2 diabetes and heart disease.

Many people assume menopause itself causes weight gain. The reality is more nuanced. While declining estrogen promotes a shift toward storing more fat viscerally and may make the brain's weight-regulation system more protective of its energy stores, much of the gradual weight gain that occurs during midlife is related to the loss of muscle that accompanies aging in both women and men. As muscle mass declines, the body uses slightly less energy each day, making it easier to defend a higher level of stored energy.

Menopausal hormone therapy (MHT) is not a weight-loss treatment and does not prevent the gradual weight gain that often accompanies aging. Some studies suggest it may reduce the tendency to store fat around the abdomen, but the evidence is mixed and any effect appears to be modest. One of the most effective ways to respond is to incorporate regular resistance training, which helps preserve muscle as we age. We'll explore this in more detail later in the book.

Most importantly, this life transition is an opportunity to adjust our expectations with compassion. Just as we wouldn't expect our body to return to its prepubertal shape after puberty, we shouldn't expect it to return to its premenopausal state after menopause. These are normal stages of human biology. Your body has changed because it has adapted. Under-

standing these changes allows us to respond with knowledge, strategy, and self-compassion instead of self-criticism.

SIDEBAR

Menopausal Hormone Therapy: Clinical Myth vs. Reality

Myth:

You need menopausal hormone therapy (MHT) for GLP-1 medications, such as semaglutide or tirzepatide, to work after menopause.

Reality:

You don't. GLP-1 medications help people lose weight before menopause, during perimenopause, and after menopause. Research shows that tirzepatide remains highly effective regardless of menopausal status.

So Why Are People Talking About MHT?

Some recent studies have found that women taking MHT appeared to lose more weight while using a GLP-1 medication. That's an interesting finding, but it doesn't prove that MHT made the medication work better. Women who choose MHT often differ from those who do not in other important ways. They may have better access to healthcare, follow treatment plans more closely, or have healthier lifestyle habits. MHT can also improve menopausal symptoms, including sleep for some women, and better sleep can make weight management easier. Any of these factors could explain some – or even all – of the differences observed in these studies.

What Does the Evidence Tell Us?

The evidence we have today shows that GLP-1 medications remain effective regardless of estrogen levels. Menopause does not stop these medications from helping people lose weight. While it's possible that MHT may enhance the

response for some women, we don't yet have strong enough evidence to conclude that it does.

What Does This Mean for You?

If you're considering MHT, the decision should be based on treating menopausal symptoms, not on making your GLP-1 medication work better. GLP-1 medications target the brain's weight-regulation system, and those pathways continue to function throughout every stage of life.

Bottom Line:

Treat obesity directly. Treat menopause symptoms when appropriate. They are two different medical conditions, and each deserves the right treatment.

Polyendocrine Metabolic Syndrome

Some endocrine conditions can also place additional pressure on your body's fuel gauge. One of the most common is polyendocrine metabolic ovarian syndrome (PMOS), a hormonal condition affecting the ovaries that can disrupt periods, fertility, and how the body manages energy and blood sugar. If you have PMOS, you've probably wondered whether it's the reason you've struggled with your weight.

The answer is both *yes* and *no*.

PMOS does not directly cause obesity. Instead, certain forms of PMOS – particularly those associated with insulin resistance – make the body's weight-regulation system more likely to store and defend energy. In other words, PMOS can nudge the fuel gauge toward storing more fuel, but it isn't the fuel gauge itself.

It's also important to remember that PMOS doesn't look the same in everyone. Some people with PMOS live in larger bodies, while others have little or no difficulty managing their weight. That's because PMOS isn't a single disease. It includes

several different patterns of hormonal change, and some are much more closely linked to insulin resistance than others.

This is why it's helpful to think of PMOS and obesity as two separate conditions that often overlap. PMOS is a disorder of hormonal and ovarian signalling. Obesity is a disorder of energy regulation, in which the brain defends a higher level of stored energy. When they occur together, they can make each other worse, but they are not the same disease. Treating PMOS won't automatically treat obesity, just as treating obesity won't cure PMOS. Each condition deserves its own treatment plan.

Weight loss can improve many symptoms of PMOS, particularly in people with insulin resistance. Menstrual cycles may become more regular, fertility may improve, and metabolic health often improves. This doesn't mean obesity causes PMOS. Rather, reducing one of the biological pressures that was amplifying its effects allows the condition to function more normally.

You may be wondering, *What about type 2 diabetes? Doesn't insulin resistance cause obesity?* It seems like it should, but the biology usually works in the opposite direction.

Think back to the fuel gauge. When the brain continues to defend more and more stored energy over many years, the body's normal fat-storage system eventually reaches its limit. Once that happens, fat begins to spill into places it was never meant to be, such as the liver, pancreas, and muscles. As we discussed earlier, this is called lipotoxicity, and it interferes with the way insulin works.

Over time, insulin resistance develops, the pancreas struggles to keep up, and blood sugar begins to rise. In other words, type 2 diabetes is usually a consequence of the body being under prolonged pressure to store excess energy, not the other way around.

Once insulin resistance develops, it becomes another pressure on the fuel gauge, making weight regulation even more

challenging. That's why treating type 2 diabetes with weight loss is so important. But if we focus only on lowering blood sugar without addressing the underlying excess adiposity that is driving insulin resistance, we're treating one consequence of the problem rather than the problem itself.

The pattern should be starting to look familiar. Whether it's genetics, medications, pregnancy, menopause, PMOS, or type 2 diabetes, none of these conditions causes obesity on its own. Instead, each adds its own pressure to your body's fuel gauge. As those pressures accumulate, the more sensitive your brain becomes. It begins to interpret those signals as a warning that fuel may be running low, prompting it to defend and store more energy, even when your body already has more than enough.

Hypothyroidism

The thyroid is one of the most misunderstood parts of the weight conversation. Many people wonder, *Maybe it's my thyroid.* It's an understandable question. In fact, it's often the explanation people hope they've finally found.

The truth is a little more complicated. Thyroid hormones influence how your body uses energy. They help regulate metabolism, body temperature, and the way your body processes fats and carbohydrates. So yes, an underactive thyroid (hypothyroidism) can contribute to weight gain.

But probably not as much as you've been led to believe. Even when hypothyroidism is untreated, the average weight gain is usually only about five to ten pounds. That can certainly matter to the person experiencing it, but it doesn't explain obesity on its own.

Using our fuel gauge analogy, hypothyroidism is another pressure that can gently nudge the gauge toward "empty," encouraging the body to store more energy. But it doesn't fully

explain why the brain begins defending much larger amounts of stored energy.

Interestingly, research suggests that the relationship between obesity and hypothyroidism often runs in the opposite direction as well. Obesity is a state of chronic, low-grade inflammation, and over time that inflammation can affect many organs, including the thyroid. People living with obesity are at higher risk of developing autoimmune thyroid disease (a condition where the immune system mistakenly attaches the thyroid itself), such as Hashimoto's thyroiditis, along with several other autoimmune conditions. In some people, substantial weight loss may reduce the amount of thyroid hormone replacement they need, although many people with established hypothyroidism – particularly Hashimoto's disease – will continue to require lifelong treatment.

The relationship is nuanced. Sometimes hypothyroidism contributes to modest weight gain. In other cases, obesity itself places stress on the thyroid. The two conditions can coexist, but neither fully explains the other.

This is also why treating hypothyroidism doesn't usually lead to dramatic weight loss. Replacing thyroid hormone corrects a hormone deficiency, which is essential for your overall health. But it doesn't reset the brain's weight-regulation system.

Again, this pattern should be starting to feel familiar. Hypothyroidism is a disorder of thyroid hormone production. Obesity is a disorder of energy regulation. They can overlap, and each deserves treatment, but they are not the same disease.

So if you've ever hoped your thyroid was the missing piece, you weren't wrong to ask. It's absolutely worth checking because untreated hypothyroidism should be identified and treated. But if your goal is to understand obesity, the thyroid is usually just one small part of a much bigger story.

Low Testosterone

Testosterone is often described as the "missing piece" when it comes to weight gain in men—and sometimes in women after menopause. It can feel reassuring to think that if your testosterone is low, you've finally found the explanation. But, like many of the other hormones we've discussed, the relationship is more complicated.

Part of the confusion comes from how testosterone is measured. Some testosterone travels through the bloodstream bound to proteins, while some is "free" and available for your body to use. In people living with obesity, the amount of these binding proteins is often lower. That can make total testosterone appear low on a blood test, even though the amount of active testosterone may not be as reduced as it first appears.

Sleep also plays an important role. Many men living with obesity have obstructive sleep apnea, a condition that repeatedly interrupts breathing during sleep. Because much of the body's testosterone is produced while you sleep, poor-quality sleep can lower testosterone levels. In these cases, treating the sleep apnea often allows testosterone levels to recover toward normal.

Not everyone living with obesity needs to have their testosterone checked. The decision to test is based on symptoms of low testosterone, not body weight alone. One of the most helpful questions that your healthcare provider may ask men is whether you've noticed a change in your usual morning erections, as this can be an important clue that deserves further evaluation. If you don't have symptoms suggestive of low testosterone, testing is unlikely to explain why you're living with obesity.

One finding is remarkably consistent: Losing weight tends to increase testosterone levels. The reverse – using testosterone therapy to promote weight loss – is far less reliable. While

testosterone treatment can improve muscle mass, energy, and other symptoms in people who truly have low testosterone, it usually has only a modest effect on body weight because it doesn't address the brain systems that regulate how much energy your body wants to store. In fact, many people gain a small amount of weight after starting testosterone therapy because of increases in muscle mass and water retention.

So yes, low testosterone and obesity are connected. But the relationship is bidirectional. Obesity can contribute to lower testosterone, and low testosterone can make weight management somewhat more challenging. Neither condition fully explains the other, and low testosterone is not the root cause of obesity itself.

I hope you can appreciate that the same principle applies to all of the hormones we've discussed. Hormones and life stages can change how sensitive your body's weight-regulation system becomes, but they don't control the system itself. Your brain is still doing what it believes is its most important job: protecting you from running out of fuel. In today's environment, those protective systems are more easily triggered, making weight regulation feel harder, slower, and less predictable than it should.

Managing the Pressures Doesn't Reset the Fuel Gauge

By now, you've most likely noticed a pattern. We've talked about genetics, medications, sleep, stress, pregnancy, menopause, PMOS, thyroid disease, and many other factors. Each can place additional pressure on your body's fuel gauge, making your brain more likely to store and defend energy.

So it's natural to wonder: *If I fix all of those things, shouldn't the weight come off?*

I wish it were that simple.

Correcting these pressures is incredibly important. Improving your sleep, treating thyroid disease, changing weight-promoting medications when possible, and eating a healthier diet all reduce the signals telling your brain that fuel may be running low. Think of these changes as upgrading your body's fuel from one that burns inefficiently to one that burns cleaner, more efficiently, and with less waste. The engine runs better, and you're no longer adding unnecessary pressure to the fuel gauge.

But managing these pressures doesn't necessarily reset the fuel gauge. Some people do experience significant weight loss, but many others do not. If you've worked hard to address these factors and you're still carrying excess body fat, that doesn't mean you've failed. It means your fuel gauge is still sitting too close to "empty." Your brain is still convinced it needs to protect its fuel reserves, even though those reserves are already more than enough.

That is obesity.

That doesn't mean addressing these other factors was pointless. Quite the opposite. Every pressure you manage makes it easier to prevent future weight gain and improves your overall health. You're reducing the forces that keep pushing the fuel gauge toward "empty."

But if the fuel gauge remains stuck near "empty," your brain will continue to defend your current weight.

And if these pressures remain unaddressed, no treatment – not even medication or bariatric surgery – can fully compensate for them. Medications can help make room for weight loss on the fuel gauge, but they cannot completely overcome a system that is constantly receiving falsely low-fuel signals. Otherwise, we're asking today's treatments to do far more than they were designed to do: improve sleep, repair our relationship with food, reduce stress, *and* make room for weight loss all at the same time.

This is where I hope this book begins to fill in some of the missing pieces. My goal is to help you understand the most significant pressures that influence your body's weight-regulation system, not to overwhelm you but to bring clarity, reduce confusion, and validate what you've experienced while trying to manage your weight.

Just as importantly, I hope this knowledge helps you have more informed conversations with your healthcare provider. Many of these factors can be identified, treated, or managed, and recognizing them is an important step toward building a treatment plan that works with your biology rather than against it.

Some pressures can be addressed on your own, such as reducing sugary drinks or cutting back on screen time. Others may require medical support, such as treating BED, evaluating and managing obstructive sleep apnea, or getting help reducing alcohol consumption. (By the way, there are medications that can help with that too.)

Once we've reduced the pressures pushing your internal fuel gauge toward "empty," many people will still need treatment for obesity itself. This is where modern obesity therapies come in—to help recalibrate the level of stored energy the brain works to defend.

Earlier in this book, I wrote that restoring the brain's long-term energy regulation is an essential part of treating obesity. That is true 100 percent of the time. Every effective obesity treatment – including GLP-1 medications and bariatric surgery – works by recalibrating the level of body weight the brain defends. They strengthen the biological signals that tell your brain how much fuel is available, something dieting alone cannot do.

All current obesity treatments work by recalibrating how much body weight the brain defends, allowing the fuel gauge to temporarily move farther from "empty" and closer to "full." Not

all the way to "full"; how far the gauge moves depends on the treatment and its intensity, which we'll explore in the next chapter. The important point is that every effective obesity treatment, whether medication or bariatric surgery, works by moving the fuel gauge away from "empty." They do this by strengthening the biological signals of fullness and satiety, giving the brain a larger margin to use its stored energy before it tries to activate a starvation response.

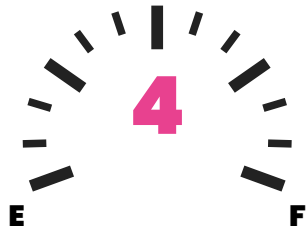
Obesity is, in many ways, a disease of an inappropriately hungry brain. If it weren't, why would people with abundant energy stores continue to experience frequent hunger, reduced fullness, and cravings? The problem is that the brain perceives an energy shortage when none exists. Communication between energy stores and appetite becomes uncoupled. The body has more than enough energy in reserve, yet the brain continues to behave as though it is starving.

And that's why so many people find themselves saying, "I'm doing everything right, but nothing is happening."

They're not imagining it.

The next step isn't to try harder. It's to change the position of the fuel gauge itself.

To lose weight without constantly triggering the body's starvation response, we need to move the fuel gauge farther away from "empty." We need to create a buffer – a margin on the fuel gauge – that allows your body to safely use its stored energy without your brain immediately sounding the alarm. That's exactly what modern obesity treatments are designed to do, and that's where we're going next. In the next chapter, we'll explore how modern obesity treatments work. Along the way, we'll answer many of the most common questions people have about these therapies and what they can realistically expect.



CHAPTER

CREATING ROOM FOR WEIGHT LOSS

BY NOW, YOU'VE SEEN how many different factors can push your brain's fuel gauge toward "empty." Poor sleep, ultra-processed foods, weight cycling (repeatedly losing and regaining weight, sometimes called "yo-yo dieting"), certain medications, hormonal changes, and conditions like ADHD or BED can all add pressure to the system. Imagine each of these as another finger pressing down on the fuel gauge (Figure 4.1). The more fingers pressing down, the closer the gauge moves toward "empty" – even while your body continues storing fat.

Now, imagine you begin removing those, one by one. You improve your sleep. You treat sleep apnea. You eat fewer ultra-processed foods. You switch to more weight-neutral medications when possible. You address BED or ADHD. As each pressure is reduced, the force pushing your fuel gauge toward "empty" may start to ease. For some, the fuel gauge can move from almost empty to perhaps one-quarter of a tank. That creates room for the body to draw on its stored energy, allowing weight loss to occur. This is why addressing these factors matters; they can make a meaningful difference.

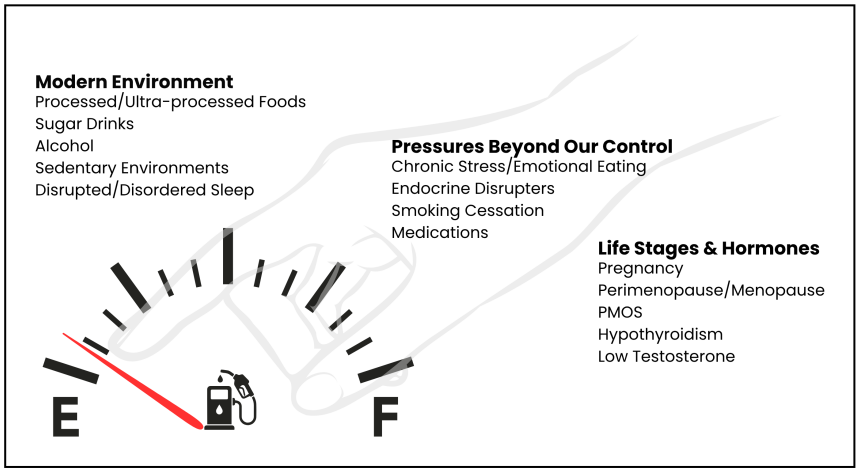


Figure 4.1. Factors Influencing the Brain's Perception of Energy Availability. Modern environmental factors, biological and medical influences, and life stages all affect how the brain perceives available energy and regulates hunger, fullness, energy expenditure, and body fat. These factors can shift the brain's internal "fuel gauge," making weight gain more likely or weight loss more difficult. Understanding these influences helps explain why obesity is a complex chronic disease rather than simply the result of personal choices.

But for many people, the story doesn't end there. As weight comes off, the fuel gauge gradually drifts back toward "empty." This happens because weight loss itself moves the fuel gauge in that direction. Remember, your brain isn't measuring how much body fat you have. It's responding to changes in your stored energy. Every pound of weight lost tells your brain that fuel reserves are shrinking, even when plenty of energy remains in reserve.

Eventually, after enough weight loss, the fuel gauge is once again hovering just above "empty." This is when many people notice their weight loss has stopped. It can feel as though their efforts have failed or the treatment has stopped working, but neither is true. What has actually happened is that they've used up all the room available before the brain once again began

defending their current level of stored energy. Even after reducing as many outside pressures as possible, the brain still believes that level of stored energy is worth protecting. This is obesity.

When Medical Treatment Is the Answer

This is where medical therapies change the equation. Modern obesity treatments work by increasing fullness and satiety. They strengthen the signals that tell your brain, "We have enough fuel." As those signals become stronger, the fuel gauge moves farther away from "empty," creating more room for your body to use its stored energy before it gradually drifts back toward "empty." By that point, your brain has adjusted to defending a lower level of stored energy.

A common misconception is that obesity treatments work by "forcing" your body to lose weight. They don't. Their job is to move the fuel gauge closer to "full." That's why the intended treatment experience is to feel full sooner during a meal and stay full longer afterward. The strength of those fullness signals helps determine how much energy your brain believes is available – and ultimately, what level of stored energy it is willing to defend.

With this in mind, you can think of medications and bariatric surgery as applying pressure in the opposite direction on the fuel gauge. If the fuel gauge moves from just above "empty" to one-quarter or even one-half of a tank, you've once again created room for your body to use its stored energy before it gradually drifts back toward "empty."

Not all treatments create the same amount of room. Bariatric surgery shifts the fuel gauge farther away from "empty" than any current medication, which is why it remains the most effective treatment for obesity. Medications also move the fuel gauge, but how far it moves depends on both the medication and the dose. In general, more effective medications and

higher doses create more room than less effective medications or lower doses.

That doesn't mean everyone responds the same way. Genetics still play an important role in determining how your fuel gauge responds. One person may achieve significant weight loss on a low dose of medication, while another may require the highest available dose to reach their best weight. Neither response is right or wrong. They simply reflect differences in how each person's brain regulates stored energy.

Think about treating high blood pressure. Two people can start the same medication at the same dose. One person's blood pressure reaches the target almost immediately, while the other needs a higher dose, or even two or three medications. We don't conclude that the medication "doesn't work" or that the second person's blood pressure is somehow their fault. We recognize that people respond differently because of differences in their biology. Obesity treatment is no different. Some people achieve their best weight with a low dose, while others require more intensive treatment.

No matter which treatment is used, everyone eventually reaches a weight-loss plateau. This often feels discouraging, but it's actually one of the most predictable parts of obesity treatment. Imagine your treatment has moved the fuel gauge from almost "empty" to one-quarter of a tank. As weight comes off, the fuel gauge gradually drifts back toward "empty." Eventually, it hovers just above "empty" again, and weight loss slows before eventually stopping. The plateau is simply telling us that the room created by that treatment has now been used.

If additional weight loss is appropriate, the solution is to create more room. Sometimes that means increasing the medication dose. Sometimes it means switching to a more effective medication. Sometimes it means combining medications. For some people, bariatric surgery creates the greatest shift in the fuel gauge. Each time we create more room, weight loss can

begin again until the fuel gauge gradually drifts back toward "empty." Although you can't see your fuel gauge moving, you can often feel the changes it creates. Understanding those changes can help you recognize what a normal treatment experience feels like.

Understanding Your Treatment Experience

This is also a good time to talk about the difference between *hunger* and *fullness*. These words are often used as if they mean the same thing, but they describe two very different biological signals.

One of the biggest misconceptions about GLP-1 medications is that they are "hunger or appetite suppressants." Current and future therapies can affect hunger pathways directly and indirectly, but their primary goal is to enhance the body's natural fullness signals, helping you feel full sooner during a meal and stay full longer afterward.

That distinction matters because hunger is not the enemy. Hunger is a normal, healthy survival signal. It reminds us to eat, nourish our bodies, and maintain enough energy to live. The goal of obesity treatment is not to eliminate hunger. It is to enhance the body's natural fullness signals, creating room for weight loss by telling the brain, "We have enough fuel."

At first glance, this may sound like semantics, but it isn't. Understanding the difference between hunger and fullness helps explain how these medications actually work and what the aim of treatment is. Rather than eliminating hunger, they strengthen feelings of fullness and satiety. By mimicking the body's natural fullness energy-regulating signals, they shift the fuel gauge away from "empty" and closer to "full." This tells the brain, "We have enough fuel," allowing it to defend a lower level of stored energy. That is what creates room for weight loss.

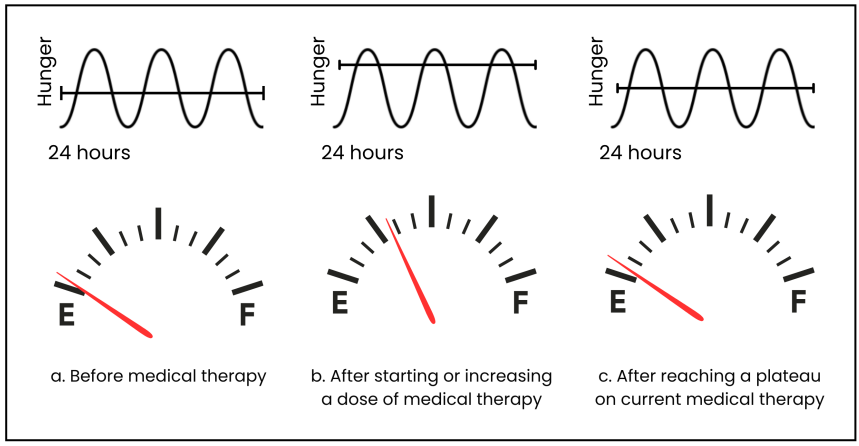


Figure 4.2. How Obesity Medications Create Room for Weight Loss. Obesity medications help recalibrate the level of stored energy the brain defends by temporarily shifting the fuel gauge away from "empty" and creating room for weight loss. This increases fullness – causing people to feel fuller faster while eating and longer after eating – while normal hunger fluctuations throughout the day continue. **(a)** Before treatment, the brain interprets the body's energy stores as low despite adequate energy reserves, placing the fuel gauge close to "empty" and defending a higher body weight. Hunger continues to rise and fall throughout the day, but baseline fullness is reduced, making it more difficult to stop eating. **(b)** After starting or increasing the dose of an obesity medication, the brain perceives more available energy, shifting the fuel gauge farther from "empty" and delaying activation of the biologically programmed starvation response during weight loss. Normal hunger fluctuations continue, but baseline fullness increases, allowing people to feel satisfied with less food and supporting weight loss. **(c)** As weight is lost, stored energy decreases and the fuel gauge gradually moves closer to its low-fuel threshold again. This reactivates the biologically programmed starvation response, slowing further weight loss and resulting in a weight-loss plateau. Continued treatment now helps the brain defend a lower body weight. At the same time, the increase in fullness partially wanes, although it generally remains greater than before treatment, allowing weight to stabilize at a lower weight.

Another common misconception is that obesity treatment should eliminate hunger altogether. That's simply not true. Hunger is a normal part of being human. Even with the most

effective medications available today, you should still become hungry before meals. In fact, if you never feel hungry, your treatment may actually be too strong.

The below illustrates what we hope to achieve with treatment (Figure 4.2). Look first at the wavy line across the top of each panel. It represents the natural rhythm of hunger over a twenty-four-hour day. Hunger rises before a meal, falls after you eat, and gradually returns a few hours later. Those waves are present before treatment, during treatment, and after a weight-loss plateau. In other words, the goal is not to flatten the curve or eliminate hunger. The natural rhythm of hunger should remain.

Now look at the corresponding fuel gauges below each graph.

In panel a, before treatment, the fuel gauge hovers just above "empty." Although your body may be carrying plenty of stored energy, your brain perceives less available energy than is actually stored. You still experience normal hunger throughout the day, but because the fuel gauge remains so close to "empty," your brain strongly defends your current level of stored energy. Trying to lose weight can feel like swimming against the current.

In panel B, treatment helps recalibrate the level of stored energy the brain defends by allowing the fuel gauge to move farther from "empty. Notice that the pattern of hunger above hasn't disappeared; it still rises before meals and settles after eating. What changes is your sense of fullness. You begin to feel satisfied sooner during a meal and stay satisfied longer afterward. Because the brain now perceives more available energy, it becomes willing to let go of stored energy, creating room for weight loss.

Over time, as weight is lost and approaches a plateau, something predictable happens. In panel c, the fuel gauge gradually drifts back toward "empty" until it once again hovers just above

the brain's low-fuel threshold. At this point, the brain resumes defending its current level of stored energy, causing weight loss to slow and eventually plateau. A weight-loss plateau is not the starvation response. Rather, it represents the point just before the starvation response – a state in which the brain is defending body weight to prevent perceived energy stores from falling any further. The biologically programmed starvation response is triggered only if the brain perceives available energy as falling below this threshold, making additional weight loss progressively more difficult.

This is why we focus on fullness, not hunger. Obesity medications do not eliminate hunger; they strengthen fullness signals. Hunger continues to rise before meals and settle after eating. What changes is that you feel full sooner during a meal and remain full longer afterward. Those stronger fullness signals tell the brain, "We have enough fuel," temporarily shifting the fuel gauge farther from "empty" and creating room for weight loss.

When you understand this, many of the experiences people describe during treatment begin to make sense. Feeling hungry before breakfast, lunch, or dinner is normal. Feeling satisfied after a smaller meal is expected. And when weight loss eventually plateaus and portions gradually begin to increase again, it does not mean your treatment has failed. More often, it means the fuel gauge has drifted back toward its low-fuel threshold and your brain has adapted to its new level of stored energy. If additional weight loss would still provide meaningful health benefits, it may be time to discuss with your healthcare provider whether creating more room for weight loss is appropriate.

Hopefully, you now have a clearer understanding of how modern obesity treatments work. Rather than forcing weight loss or eliminating hunger, they enhance your body's natural fullness signals and recalibrate the level of body weight your

brain works to defend. This temporarily shifts the fuel gauge farther from "empty," creating room for weight loss until the brain gradually adapts and weight stabilizes at a lower, biologically defended level.

Common Questions, Explained

With that foundation in place, the rest of this chapter answers many of the most common questions people have about their experience with GLP-1 medications. We'll explore those experiences through the lens of biology and the fuel gauge analogy, rather than the expectations created by weight-loss culture. Once you understand the biology, experiences that once felt confusing, frustrating, or even discouraging begin to make much more sense.

The Fear of Hunger Returning

By now, you know something many people don't: GLP-1 medications aren't designed to eliminate hunger; they're designed to strengthen fullness signals and help your brain recognize that enough fuel is available. Once you understand that, the first question becomes much easier to answer.

Why am I still hungry? Is my medication working?

Of all the questions people ask while taking a GLP-1 medication, this is probably the most common. If you're also asking it, you're certainly not alone. Many people begin treatment expecting hunger to disappear almost completely. They've heard stories about people who "forget to eat," "lose all interest" in food, or have to "remind themselves to eat." Others assume that if they still feel hungry before lunch or dinner, the medication isn't working.

That expectation is completely understandable, but it comes from decades of weight-loss culture. For years, we've been taught that hunger is the enemy. Successful dieting has been portrayed as learning to ignore hunger, fight through it, or

eliminate it altogether. We've celebrated eating as little as possible and often viewed the absence of hunger as proof that a diet was "working." It's no surprise that many people bring those same expectations to GLP-1 medications, assuming successful treatment means never feeling hungry again.

But as you now understand, that isn't what these medications are designed to do.

GLP-1 medications are not intended to create anorexia. When most people hear the word anorexia, they think of the eating disorder anorexia nervosa. In medicine, however, anorexia simply means a loss of appetite or a lack of desire to eat. The goal of GLP-1 medications is not to eliminate hunger, make food unappealing, or leave you feeling full all day. Although some people experience these effects when they first start treatment or after increasing their dose, this often means the treatment effect is stronger than necessary for that individual. If this describes your experience, it's worth discussing your current dose with your healthcare provider.

A helpful way to think about this is to return to our fuel gauge analogy. The goal is not to push the fuel gauge all the way to "full." The goal is simply to move it farther away from "empty." Why not all the way to "full"? Because hunger isn't a malfunction; it's one of the body's most important survival signals. It reminds us to eat enough to meet our nutritional needs, maintain muscle, support our daily activities, and respond when our bodies need more energy.

Figure 4.3 brings these ideas together. Before medical treatment, hunger rises and falls normally against a baseline level of fullness. Current obesity medications strengthen fullness signals and move the fuel gauge farther from "empty," while normal hunger continues before meals. However, online narratives may create the impression that treatment should make hunger almost disappear. Hunger may become less intense for

some people, but eliminating it, as shown in panel c, is not the goal.

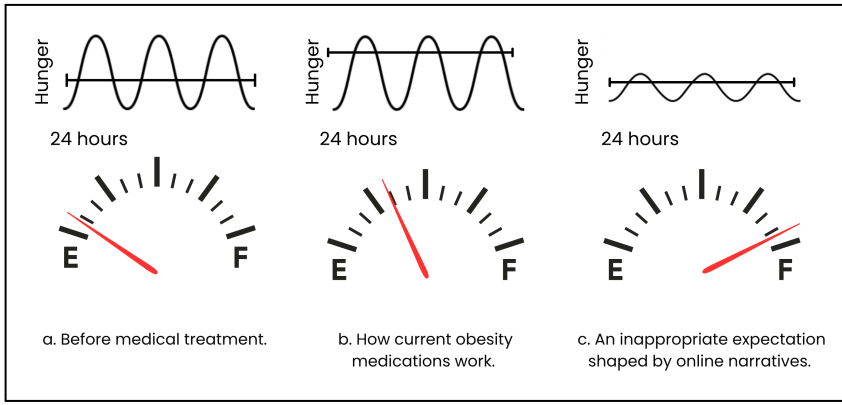


Figure 4.3. Understanding Hunger and Fullness During Obesity Treatment. (a) Before medical treatment, hunger rises and falls normally in the background of a baseline fullness level. **(b)** Current obesity medications strengthen fullness and move the fuel gauge farther from "empty," while normal hunger continues before meals. **(c)** Online narratives may inappropriately suggest that treatment should eliminate hunger. Hunger may lessen, but eliminating it, as depicted in this figure, is not the goal.

The problem in obesity has never been that hunger exists. The problem is that the brain underestimates how much energy is already stored. Even when abundant energy reserves are available, the brain behaves as though fuel is running low. It responds by encouraging us to seek and store more energy than we actually need. Diet culture has taught us to blame hunger. Biology teaches us that the real problem is the brain's inaccurate estimate of how much fuel is already available.

GLP-1 medications help correct that mismatch by strengthening fullness signals – the brain's way of recognizing that there is plenty of fuel in the tank. As treatment begins to work, most people notice that they feel full sooner during a meal and remain full longer afterward. Those stronger fullness signals tell the brain that more fuel is available than it previously

believed. As the fuel gauge gradually moves away from "empty," the brain becomes less protective of its stored energy. That's what creates room for weight loss.

This is also why understanding the difference between hunger and fullness is so important. Although we often use these words interchangeably, they describe two very different biological signals with very different jobs.

Hunger is your body's reminder that it's time to eat. It protects you from undernutrition and helps ensure you consume enough protein, vitamins, minerals, and energy to stay healthy. A successful treatment response should still include hunger appearing at appropriate times throughout the day, such as before breakfast, lunch, or dinner. Feeling hungry before a meal is not evidence that your medication has stopped working. It's evidence that your body's normal biology is doing exactly what it was designed to do.

Fullness serves a different purpose. Rather than telling you when to eat, it helps the brain estimate how much energy is already available. You can think of hunger as the reminder to refill the tank, while fullness is the fuel gauge itself. The stronger and more accurate the fullness signal, the more accurately the brain can estimate how much fuel remains. GLP-1 medications enhance these fullness signals, helping the brain adjust to defending a lower body weight.

Notice that the goal isn't to convince the brain that the tank is overflowing, either. That wouldn't be healthy. The goal is simply to help the brain recognize that there is far more fuel on board than it previously believed. Once the brain no longer thinks fuel is running dangerously low, it becomes willing to let go of some of its stored energy instead of defending it so fiercely.

This may be one of the most important expectations to understand before starting treatment. Many people spend months worrying that their medication isn't working simply

because they continue to feel hungry from time to time. In reality, successful obesity treatment isn't measured by the absence of hunger. It's measured by stronger fullness signals and a fuel gauge that more accurately reflects how much energy your body already has. Once you understand this biology, you can stop asking "Why am I still hungry?" and start asking a much better question: "Am I feeling full sooner and staying full longer?" That's the experience these medications were designed to create.

When the Next Injection Can't Come Soon Enough

Why am I hungrier before my next injection?

This is another very common question, and one that often catches people by surprise.

For the first few days after an injection, many people notice they feel full sooner during meals, stay full longer afterward, and spend less time thinking about food. Then, as they get closer to their next scheduled dose, something changes. Fullness doesn't feel quite as strong. Larger portions become a little easier to eat, and food becomes more noticeable again. If you've experienced this, it's easy to worry that something is wrong. Many people wonder whether their medication has stopped working, whether they've become "resistant" to it, or whether they need a higher dose. Some even worry that this change means they're about to regain all the weight they've lost. Those concerns are completely understandable, but in most cases, they aren't what's happening.

The explanation is actually much simpler: The amount of medication in your body naturally decreases throughout the week.

To understand why, it helps to understand one simple concept: a medication's half-life. A half-life is the time it takes for half of a medication to leave your body. Rather than disappearing all at once, the medication gradually declines over

time. You can think of it like a reservoir that slowly drains. That's why these medications need to be taken regularly. Each new dose refills the reservoir before it gets too low.

For example, tirzepatide has a half-life of about five days, while semaglutide has a half-life of about six days. By the time you're due for your next injection, there is naturally less medication circulating in your body than there was a day or two after your previous injection. As the medication level falls, the fullness signal naturally becomes a little less intense. That doesn't mean the medication has stopped working; it simply means there is less of it in your system than there was a few days earlier. That's exactly what we would expect.

Now, imagine stretching the time between injections from one week to an entire month. Some of the medications currently being developed may only need to be taken once a month. If a medication lasts an entire month, no one would expect fullness to feel exactly the same on day twenty-eight as it did on day two. A gradual change in fullness is simply part of how long-acting medications work. But it doesn't mean you're becoming resistant to treatment.

Our fuel gauge analogy also helps explain this. Imagine your medication has moved your fuel gauge from just above "empty" to somewhere closer to halfway. As the medication level gradually declines during the week, the fuel gauge may drift down slightly before your next injection. But drifting down a little isn't the same as returning to "empty." The fuel gauge is still much farther from "empty" than it was before treatment began.

This is why it's helpful to judge your treatment by the overall pattern, not by how you feel on any single day. A slight decrease in fullness before your next injection usually reflects the normal way the medication moves through your body – not medication resistance, treatment failure, or a sign that you're about to regain all the weight you've lost.

Understanding this can save you a great deal of unnecessary worry. Rather than asking "Why am I hungrier today?" a better question is "Overall, am I still feeling full sooner during meals and staying full longer than I did before treatment?" If the answer is yes, your medication is likely doing exactly what it was designed to do.

Why Do I Still Have Food Noise?

One of the reasons GLP-1 medications have captured so much attention is that many people describe something they had never experienced before: The food noise became quieter. The constant thoughts about food faded into the background. They could walk past a plate of cookies without feeling pulled toward them. For the first time, eating felt less like a battle.

But that isn't everyone's experience. Some people notice a dramatic reduction in food noise, while others experience little change despite responding well in other ways. This reminds us that the brain circuits involved in hunger, fullness, food reward, and cravings are remarkably complex. GLP-1 receptors (like small docking stations on cells that a hormone or medication attaches to in order to deliver its signal) are found throughout many of the brain regions that regulate eating, which is one reason these medications can reduce food noise. Yet obesity is influenced by hundreds of genes, most of which affect how the brain regulates feeding behaviour. Each person's biology is unique, so it isn't surprising that the experience of food noise varies from one individual to another.

This is also why GLP-1 medications are not the only effective treatments for obesity. Some medications target different parts of these same brain networks. For example, the combination of bupropion and naltrexone (Contrave®) works through brain pathways involved in satiety, food reward, and cravings. Like GLP-1 medications, its goal is to make eating less require less effort by improving the biological regulation of eating. For

some people, that means feeling fuller. For others, it means fewer cravings or quieter food noise. Different medications work through different pathways, but they all reinforce the same message: Food noise is one expression of the biological systems that regulate feeding behaviour.

How Long Will This Take?

One of the most common questions people ask me is "How long will it take?" It's a completely understandable question. We all like to know what to expect. But it also assumes there's a finish line waiting at the end. With obesity treatment, there isn't. Despite what diet culture has taught us, progress doesn't follow a calendar.

Another common question is "How much weight should I be losing each week?" The challenge is that neither the calendar nor the absolute number of pounds lost tells us very much. A twenty-pound weight loss means something very different for someone who starts at 180 pounds than for someone who starts at 350 pounds.

Weight loss also isn't perfectly predictable. Some weeks you'll lose more, some weeks less, and sometimes your weight may stay the same for a while before changing again. A plateau doesn't automatically mean your treatment has stopped working. It's simply part of how the body responds to weight loss.

Instead of focusing on how many pounds you've lost each week or month, obesity specialists focus on percent body weight loss without attaching it to a timeline. This allows us to compare progress fairly, regardless of where someone starts, and helps us determine whether treatment is improving health. As we discussed earlier in the book, weight loss rarely happens in a straight line, so adding a timeline doesn't make that information more meaningful.

As a general guide, many people lose about 5 percent of their body weight within the first three months, 10 percent by

six months, and 15 percent by one year. For someone who weighs 200 pounds, that would be about ten, twenty, and thirty pounds, respectively. These aren't deadlines or guarantees, and reaching them sooner or later doesn't mean you've succeeded or failed. They're simply average milestones that help us understand whether your treatment is working as expected and moving you toward better health.

Pressure to Lose Faster

Why isn't my weight loss happening faster?

It's completely understandable to want weight loss to happen faster. Many people have spent years – and sometimes decades – living with obesity before finally finding a treatment that works. When you've carried that burden for so long, it's only natural to want the weight to come off as quickly as possible.

Weight-loss culture has also taught us that faster is better. We celebrate dramatic before-and-after photos, rapid transformations, and stories of people losing extraordinary amounts of weight in just a few months. It's easy to look at those stories and wonder *Why isn't that happening to me?*

But your body isn't trying to win a race. Your body is trying to adapt safely to a changing energy supply.

Many people do lose weight more quickly during the first few weeks of treatment. That early progress can feel exciting and motivating, but it can also create expectations that are difficult to sustain. Part of that early weight loss isn't body fat alone. Your body also uses glycogen, a form of stored carbohydrate found in your muscles and liver. Glycogen acts like a sponge, holding on to water. As those glycogen stores are used, the water attached to them is released as well. This is why the number on the scale often drops quickly at first; you are losing both water and fat. Once those glycogen stores have been used up, weight loss naturally slows because a greater proportion of

the weight you lose now comes from body fat. This doesn't mean your medication has stopped working or that you've done anything wrong. It's a normal and expected part of how the body responds to weight loss.

It also helps to return to our fuel gauge analogy. As your body loses weight, the fuel gauge gradually drifts back toward "empty." The closer it gets to "empty," the more strongly your brain begins defending its remaining stored energy. Slower weight loss isn't a sign that something is wrong. Rather, it's evidence that your biology is responding exactly as we would expect.

At the same time, your healthcare provider is trying to achieve something much more important than simply making the number on the scale fall as quickly as possible. The goal is to lose fat while preserving as much lean body mass – including muscle – as possible.

Muscle is one of the most metabolically active tissues in your body. Even at rest, it requires energy to maintain. During weight loss, your brain is trying to conserve energy wherever it can. If given the opportunity, it won't just break down fat; it will also break down muscle because muscle is more metabolically expensive to maintain. Losing too much muscle can make long-term weight maintenance more difficult because muscle plays an important role in supporting metabolism, strength, and physical function.

That's why we don't judge success by how quickly the scale moves. We want as much of the weight you lose as possible to come from fat rather than muscle. For most people, losing about 1 percent of their body weight per week strikes a healthy balance between making meaningful progress and preserving the muscle needed for lifelong health and function.

If you find yourself wishing the weight would come off faster, it may be worth asking a different question: "Why does it need to happen faster?" Are you hoping to reach a certain

number on the scale? Are you comparing your progress with someone else's? Or are you simply tired of carrying the physical and emotional burden of obesity?

Those are all understandable feelings. But remember, the goal of treatment isn't to lose weight as quickly as possible. It's to improve your health in a way that your body can sustain. Sometimes the healthiest path forward isn't the fastest one. It's the one your biology can maintain for years to come.

Fear That Progress Is Slowing

Why has the weight loss slowed down?

This is one of the most common questions people ask during obesity treatment. Part of the reason is that weight-loss culture has taught many of us to expect weight loss to happen in a straight line. The story we're often told is simple: If treatment is working, the number on the scale should keep falling at the same steady pace. If it slows, stalls, or even fluctuates, something must be wrong. In the GLP-1 era, many people assume this means the medication has stopped working.

But nothing in physiology works in a straight line, and weight loss is no exception. As your body weight changes, so does the biology that regulates it.

One reason is that a smaller body simply needs less energy than a larger one. Imagine carrying a heavy backpack everywhere you go. The heavier the backpack, the more energy it takes to walk, climb stairs, or even stand. Now imagine taking the backpack off. Suddenly, your body doesn't have to work as hard. The same thing happens during weight loss. As you become lighter, your body naturally burns less energy because there is less body to move, maintain, and support.

Early in treatment, the difference between the energy your body needs and the energy you're eating can be quite large. You feel full sooner, your portions become smaller, and weight often comes off more quickly. Over time, as your body becomes

smaller, that gap naturally shrinks. Even if you're eating exactly the same way, weight loss usually slows because your body's energy needs have changed. But there's an even bigger reason weight loss slows.

Remember our fuel gauge analogy. As weight comes off, the fuel gauge gradually drifts back toward "empty." As it does, your brain becomes increasingly protective of its remaining stored energy. This is exactly what we would expect from a brain whose job is to prevent starvation. The closer the fuel gauge moves toward "empty," the harder the brain works to defend that stored energy.

When you put these two biological changes together – a smaller body needing less energy and a brain that is once again becoming more protective of its stored energy – it becomes much easier to understand why weight loss slows over time. Contrary to what many people naturally worry, this doesn't mean your medication has stopped working or that you've done anything wrong. In fact, it's evidence that your treatment is now working in a body that has changed since you started.

The real surprise would be if weight loss continued at the same rapid pace forever. That wouldn't be normal – or healthy. The goal of obesity treatment isn't to keep the scale falling as quickly as possible. It's to help your body settle at a lower weight that your brain is willing to defend. As you get closer to that point, weight loss should naturally slow before eventually reaching a plateau.

When the Early Momentum Fades

Why doesn't it work like it used to?

Many people remember exactly what treatment felt like when they first started. They felt full much sooner during meals. They stayed full longer afterward. Food occupied much less of their attention, and many describe those first few weeks as life-changing.

Then, a few months later, the experience begins to change. The feeling of fullness isn't quite as dramatic. Food starts taking up a little more mental space again. The medication simply doesn't feel as "powerful" as it once did. It's completely understandable to wonder *Has my medication stopped working?* The good news is that, in most cases, the answer is no.

Part of what you're experiencing is simply how the human brain works. Our brains are remarkably good at noticing contrast, but much less good at noticing what has become familiar. Think about putting on a watch for the first time. During the first few hours, you're constantly aware that it's on your wrist. You notice its weight, the feel of the strap, and every time it brushes against your skin. After a few days, however, you hardly notice it anymore. Your brain has simply adapted to what has become your new normal.

The same thing can happen with GLP-1 treatment. The first few weeks often feel dramatic because the change from your previous experience is so noticeable. That dramatic improvement becomes your new reference point. As time passes, your brain becomes accustomed to those stronger fullness signals, so they no longer feel as remarkable as they once did. But that's only part of the story. There's another reason this happens, and it takes us back to the biology of obesity.

Remember, your brain is designed to protect you from losing stored energy. Throughout most of human history, losing body fat could have meant starvation. Because of that, the brain evolved multiple overlapping systems that constantly monitor your stored energy and work together to defend it. When a GLP-1 medication strengthens fullness signals, the brain doesn't simply stop regulating energy balance. Instead, it continues doing the job it has always done: protecting you from starvation. As one biological signal becomes stronger, other pathways begin adjusting in an effort to slow further weight

loss. Together, these normal biological responses gradually cause the fuel gauge to drift back toward "empty."

This doesn't mean you're destined to regain the weight you've lost. In fact, you've probably noticed that your weight has stabilized at a lower level than where you started. That's exactly the goal. It simply means your brain is now defending a lower level of stored energy than it did before treatment.

So, if your treatment doesn't feel as dramatic as it did during those first few weeks, don't assume it has stopped working. Instead, ask yourself a different question: "Is my weight continuing to move in the right direction, or am I maintaining the progress I've already made?" Those are much better questions to ask than "Does the medication feel as strong as it used to?"

Can I Take a Break to "Restart" the Weight Loss?

Similarly, after a few months of treatment, as weight loss begins to slow, some people wonder whether taking a break from their medication might help "reset" their body. The hope is understandable because those first few weeks often feel so different. When that experience becomes less dramatic over time, it's natural to think *Maybe if I stop for a while and then restart, it'll work like it did the first time*.

It's a reasonable question, but our biology doesn't work that way. Rather than "resetting" your treatment, taking a prolonged break may actually make your response less predictable when you restart.

Remember what we learned in the last section. Part of what made those first few weeks feel so remarkable was the contrast. Your brain was experiencing a completely new set of fullness signals. Early weight loss also tends to include the loss of glycogen and the water stored alongside it, making the number on the scale fall more quickly than it will later in treatment. As time goes on, your body becomes smaller, your brain adapts to those stronger fullness signals, and weight loss naturally slows.

None of those biological changes are erased by stopping the medication.

Our fuel gauge analogy helps explain why. Taking a break doesn't move the fuel gauge farther from "empty"; it does the opposite. As the medication leaves your body, fullness signals weaken, the fuel gauge drifts back toward "empty," and your brain once again becomes more protective of its stored energy. Rather than creating a fresh start, a prolonged break restores the biology that promotes weight regain.

At the moment, there is no good evidence that planned "drug holidays" improve weight loss or reset the treatment effect. If anything, researchers are concerned that prolonged interruptions may have the opposite effect. Some people appear to experience a less dramatic response when they restart treatment, a phenomenon called *tachyphylaxis*, which simply means a reduced response after being re-exposed to a medication. We still need more research to understand how often this happens and why. Although there is no evidence that planned treatment breaks improve the medication's effectiveness, there are anecdotal reports suggesting that some people experience a less robust response when they restart.

If your weight loss has slowed, it doesn't mean you need to start over. This is one reason obesity medications are generally meant to be taken consistently. Just as we wouldn't stop a blood pressure medication every few months hoping it would work better when we restarted it, we don't usually recommend stopping obesity treatment simply to "reset" the body. Obesity is a chronic disease, and the biology that defends stored energy doesn't take a vacation.

Can I Dose Up Temporarily, Then Back Down?

Obesity medications are dose dependent. In general, higher doses support a lower defended level of stored energy. If you temporarily increase the dose and lose additional weight,

returning to the previous lower dose usually means returning to the weight that lower dose was capable of maintaining.

Think of it like temporarily increasing a blood pressure medication until your blood pressure reaches its target, then reducing it back to the original dose. If the lower dose wasn't enough before, there's little reason to expect it will suddenly be enough now. Obesity medications work much the same way because each dose creates a different amount of room on your fuel gauge. The higher the dose, the more room it creates for weight loss before your brain begins mounting a stronger starvation response. The dose you maintain is usually the dose that determines the weight you can maintain.

Rather than chasing the lowest possible number on the scale for a short time, the goal is to find the lowest effective dose that is both medically appropriate and financially sustainable for long-term treatment.

When Plateaus Start Creating Doubt

Why have I hit a plateau?

For some, a weight-loss plateau becomes the moment when confidence in treatment begins to fade. The scale stops moving, even though you're taking the same medication and following the same routine. It's easy to wonder whether you've done something wrong or whether the medication has simply stopped working.

A weight-loss plateau is generally defined as no meaningful weight loss for about twelve weeks despite continuing treatment. Many people see the number on the scale stay the same for a week or two and immediately conclude, "The medication isn't working anymore." That's completely understandable, but it usually isn't what a plateau means.

Remember our fuel gauge analogy. Earlier in the chapter, we learned that treatment works by moving the fuel gauge away from "empty," creating room for weight loss. As weight comes

off, however, the fuel gauge gradually drifts back toward "empty." Eventually, your brain settles into defending a new level of stored energy. When that happens, weight loss naturally slows before eventually stopping. In other words, a plateau often means your brain has reached a new equilibrium where it is willing to defend less stored energy than it did before treatment began. That's actually good news!

Remember, obesity is a chronic disease. Your brain will always defend some level of stored energy. The goal of treatment was never to stop your brain from defending it. The goal was to change how much stored energy it defends. A plateau is often a sign that your brain has settled into defending a lower level of stored energy than it did before treatment began.

This also helps explain an important principle about medications: Every medication has a ceiling effect. For any given dose, there is only so much benefit a medication can provide. Increasing the dose may create more room for weight loss, but only up to a point. Eventually, every medication reaches a limit where giving more no longer provides additional benefit. This isn't unique to obesity treatment. The same principle applies to medications used for high blood pressure, high cholesterol, depression, and many other chronic diseases.

A plateau often tells us that your body has reached the limit of what the current treatment can achieve at its present dose. Sometimes increasing the dose creates more room for additional weight loss. Sometimes another medication can help. Sometimes you've reached your best weight, where the healthiest decision is to focus on maintaining the progress you've already made. Whatever the reason, your brain is now defending a lower level of stored energy than it was before treatment began.

When Comparison Starts to Steal Your Confidence

Why am I losing less than other people?

This question often begins innocently enough. A friend starts the same medication. You join an online support group. Your social media feed fills with dramatic before-and-after photos. Before long, it becomes almost impossible not to compare. *They lost fifty pounds in six months. Why isn't that happening for me? We're taking the same medication. What am I doing wrong?*

The problem is that these comparisons come from the way weight-loss culture has taught us to think about obesity. For decades, weight loss has been treated like a competition: who lost the most, who lost the fastest, and who reached their goal first. Success became something we measured against other people instead of against improvements in our own health. In the age of social media, that comparison has only intensified.

Earlier in this book, we learned that obesity is a disease of energy regulation and that every person starts treatment with a different biology. We all have different genetics, different brains, different life experiences, different medications, different sleep patterns, different stress levels, and different medical conditions. Some people live with ADHD, binge-eating disorder, insulin resistance, or years of repeated dieting. Most importantly, each person's brain is defending a different level of stored energy. Why, then, would we expect everyone to respond to the same treatment in exactly the same way?

The truth is we shouldn't.

Consider high blood pressure. Two people may start the same medication, yet one reaches their target with a low dose while the other needs a higher dose, or even several medications together. We understand that their biology is different. Obesity deserves the same understanding.

Social media has made this comparison trap even harder to escape. We're no longer comparing ourselves with a few people we know. We're comparing ourselves to thousands of carefully selected success stories, often stripped of important context. We rarely know where someone started, how much weight they

needed to lose, what dose they're taking, what other medical conditions they have, how they're eating, how they're exercising, or what other treatments they're receiving. We're comparing one small chapter of someone else's story with the entire story of our own.

Once you understand obesity as a chronic disease, those comparisons begin to lose their meaning. We don't judge cancer treatment by how someone else's tumour responded. We don't decide whether an asthma inhaler is working by comparing ourselves with another person's lung function. People living with diabetes don't measure the success of their treatment by comparing their blood sugars with those of strangers on the internet. We recognize that every person's biology is different, so every person's response to treatment will be different. Obesity deserves exactly the same perspective.

Instead of asking "Why am I losing less than someone else?" try asking a different question: "Is my treatment helping me become healthier than I was before?" That's the comparison that matters. Your brain doesn't know – or care – how much weight someone else has lost. It responds only to your biology. Success isn't about keeping up with someone else's progress. It's about helping your brain defend a lower level of stored energy than it did before treatment began.

When Weight Loss Feels Too Slow

If you've ever thought *I'm doing everything right. Why is this taking so long?* or *Why does weight loss feel so slow?* you're not alone.

The simple answer is that fat loss is slow. Really slow.

Excess body fat is your body's long-term energy reserve. Throughout human history, those energy stores helped people survive famines, harsh winters, and long periods without enough food. Your body evolved to protect those reserves, not give them away quickly.

This is very different from glycogen, your body's short-term energy reserve. Glycogen is a form of stored carbohydrate found mainly in your liver and muscles. You can think of it as your body's chequing account. It's meant to be used and replenished regularly. Body fat, on the other hand, is your savings account. Your brain is much more willing to spend from your chequing account than from your savings account.

There's another important difference. As we discussed earlier, glycogen holds on to water almost like a sponge. When your body uses glycogen during the first few days or weeks of treatment, that stored water is released as well. That's why the scale often drops quickly at the beginning. It feels exciting, but much of that early change reflects water loss, not just body fat. Once those glycogen stores are depleted, your body relies more heavily on its fat stores. That's exactly what we want, but fat is released much more slowly. Your brain carefully regulates how quickly it allows those long-term energy reserves to be used because, from its perspective, it's protecting you from starvation.

This is one of the reasons weight loss often feels fast at first and slower later on. The early drop on the scale reflects a combination of water, glycogen, and fat loss. As treatment continues, a greater proportion of the weight you lose comes from body fat, and fat loss simply happens more slowly. Seen this way, slower weight loss isn't a sign that treatment has stopped working. It's the normal pace of losing the energy your brain is most determined to protect.

But slow weight loss doesn't continue forever. I often hear people say, "I've been taking this medication for a year – or even two years – so I thought I would have lost more weight by now." That disappointment is understandable, but the calendar doesn't determine how much weight your body will lose. Time gives a medication's dose a chance to work. Once you've been on the same dose long enough to see its effect – often three to

six months – and your weight has stabilized, another year on that dose doesn't guarantee that more weight will come off. Time is not a dose. The medication may still be doing important work by helping you maintain the weight you've already lost. If more weight loss is needed to improve your health, the question becomes whether your current dose – or your current treatment – is enough to help you get there safely.

Do I Need a Higher Dose?

It's easy to think that if some weight loss is good, then more weight loss must be even better. And if a higher dose helps people lose more weight, surely the highest dose must be the best choice.

Not necessarily.

Remember, the goal of treatment isn't to reach the highest possible dose. The goal is to find the lowest effective dose that helps your brain defend a lower level of stored energy while causing the fewest side effects. Increasing the dose simply strengthens the fullness signal. Some people need a stronger signal to move their fuel gauge farther from "empty." Others achieve excellent disease control with a much lower dose. Every person's biology is different, so every person's "right dose" will be different too.

This is no different from how we treat many other chronic diseases. Some people control their blood pressure with a low dose of one medication, while others need higher doses or several medications working together. We don't aim for the highest dose; we aim for the dose that works best for that individual.

For some people, increasing the dose feels reassuring. For others, it creates a different kind of worry.

"What if I increase my dose and it still isn't enough?"

"What if I run out of options?"

Those concerns are completely understandable. They come from thinking about treatment as though it were a limited resource – as though every dose increase is one less opportunity you'll have in the future. But increasing the dose isn't about using up future opportunities for weight loss. It's about matching today's treatment to today's biology. As your body changes, your treatment sometimes needs to change with it. That's exactly what your healthcare provider is trying to do. They aren't racing to the highest dose. They're carefully adjusting the intensity of treatment until they find the amount of support your brain needs to regulate stored energy effectively.

Sometimes that happens at a low dose. Sometimes it takes a higher dose. Sometimes another medication is added. None of those decisions mean you've failed or that you're "running out of options." They simply reflect the normal process of treating a chronic disease. The right dose isn't the highest dose: It's the dose that provides the best balance between meaningful disease control, side effects, cost, and long-term sustainability.

Will I Regain the Weight If I Stop?

Earlier in this book, we learned that obesity is a disorder of long-term energy regulation. The brain is convinced fuel is running low and actively defends a higher amount of stored energy by creating a stronger drive to eat than the body's actual energy stores justify. It's as though the fuel gauge is reading closer to "empty" than it really is. GLP-1 medications don't remove that underlying physiology. They work by strengthening your body's natural fullness signals, moving the fuel gauge farther away from "empty." As the brain begins to recognize that more energy is available than it previously believed, it becomes willing to defend a lower amount of stored energy.

The important point is that the medication doesn't cure the underlying disease; it helps manage it. By the time you've reached a weight-loss plateau, the fuel gauge has already drif-

ted back to hovering just above "empty." Stopping the medication doesn't suddenly push the fuel gauge lower because it's already there. Instead, the stronger fullness signals gradually fade, allowing the brain to begin restoring the level of stored energy it wants to defend. As weight is regained, the fuel gauge continues to hover just above "empty," but now it does so at a higher level of stored energy than it did during treatment. In other words, the biology of obesity hasn't suddenly returned; it was there all along. The medication was helping your brain defend a lower level of stored energy. Once that support is removed, your brain gradually returns to defending the higher level of stored energy it recognized before treatment began.

This shouldn't come as a surprise. We don't expect a blood pressure medication to keep lowering blood pressure after we stop taking it, and we don't expect an asthma inhaler to keep preventing symptoms once it's discontinued. Obesity treatment follows the same principle. The medication works while you're taking it.

The brain may not be the only part of the body that remembers its previous state. Weight loss makes fat cells smaller, but it may not completely reset them. Researchers found that fat tissue from people who had lost substantial weight retained a molecular "memory" of its previous state. In mice, this memory primed fat cells to absorb nutrients and grow more quickly when they were exposed to a high-fat diet again, contributing to faster weight regain. Although more research is needed to understand what this means in humans, these findings suggest that fat tissue may also remain vulnerable to weight regain after obesity treatment ends.

This is also exactly what we see in clinical trials.

In the STEP 4 trial, participants first lost weight with semaglutide before being randomly assigned to either continue the medication or switch to placebo. Those who continued treatment lost even more weight. Those who stopped treatment

regained a substantial portion of the weight they had already lost.

Researchers observed the same pattern in the SURMOUNT-4 trial with tirzepatide. Participants who continued treatment maintained – and often extended – their weight loss. Those who switched to placebo regained a significant amount of weight, even though they had responded well to treatment initially.

More recently, the ATTAIN-MAINTAIN trial demonstrated a similar principle. Even reducing treatment intensity resulted in greater weight regain than maintaining the treatment that produced the original weight loss.

These studies all tell the same story. Rather than showing that the medication "failed," weight regain after stopping treatment demonstrates something very different: The medication was successfully helping your brain defend a lower level of stored energy. When that treatment is removed, your brain gradually returns to defending the higher level of stored energy it recognized before treatment began.

Do GLP-1 Medications Work by Increasing Nausea?

Many people believe that GLP-1 medications work by making them feel nauseated. In reality, nausea isn't the goal of treatment. It's an extension of the same biological signals that create fullness when those signals become too strong. Remember, the job of these medications is to strengthen your body's natural fullness signals. They also slow the movement of food out of the stomach, helping you stay full longer. Together, these effects tell your brain that enough energy is available and that you don't need to keep eating.

The easiest way to understand nausea is to think of it as *fullness turned up too high*.

Imagine a dial. At first, you feel comfortably full. Turn the dial a little higher, and you feel stuffed. Turn it higher still, and you begin to feel nauseated. These aren't separate experiences;

they're different points along the same spectrum. Your body is simply telling you that your stomach has reached a level of fullness that is becoming uncomfortable.

This is why nausea is most common after increasing the dose, eating too quickly, eating large meals, or continuing to eat after you've already started feeling full. The medication hasn't suddenly stopped agreeing with you. Instead, it's a sign that your fullness signals have become stronger than your stomach is comfortable with.

One of the biggest misconceptions about GLP-1 medications is that they work by making people feel sick so they won't want to eat. That's not true. The goal of treatment isn't nausea. It's fullness. While nausea may reduce how much someone eats, it isn't the therapeutic effect we're trying to achieve, and it isn't necessary for successful treatment. The goal is to find the right balance: enough fullness to help your brain regulate stored energy while still allowing you to enjoy food, meet your nutritional needs, and feel well.

If nausea is making it difficult to eat, drink, or carry out your normal daily activities, it's a sign to speak with your healthcare provider. Treatment should help you regain control over your eating, not leave you feeling unwell.

Scientists also recognize that nausea is not the desired treatment effect. The same areas of the brain that regulate fullness are closely connected to areas involved in nausea, which is why the two can occur together. One of the most exciting goals of newer obesity medications is to strengthen fullness signals while reducing nausea. For example, some newer medications also activate the glucose-dependent insulinotropic polypeptide (GIP) receptor. Although GIP was first discovered for its role in blood sugar regulation, it also appears to reduce some of the nausea triggered by GLP-1 medications. Researchers are still learning exactly how this works, but the goal is clear: to help people feel comfortably full, not sick.

SIDEBAR

Does Where I Inject Affect Nausea?

Another common question is whether changing your injection site – from your abdomen to your thigh or upper arm – can reduce nausea. It's an understandable question because if your stomach feels nauseated, it seems logical that injecting farther away from your abdomen might help.

The medication is injected under the skin, but it doesn't stay there. It is absorbed into your bloodstream and carried throughout your body before eventually reaching your brain. That's important because the fullness and nausea signals are generated in the brain, not in the skin or the fat beneath your abdomen where the injection is given. Whether you inject into your abdomen, thigh, or upper arm, the medication ultimately reaches the same areas of your brain.

So why do some people swear that switching injection sites improved their nausea?

The most likely explanation is time. Nausea is usually most noticeable when you first start treatment or shortly after increasing the dose. As the days and weeks pass, your brain gradually adapts, and nausea often improves on its own. If someone changes their injection site around the same time their body is naturally adapting to the medication, it's easy to give credit to the new injection site when, in reality, time was probably the real reason for the improvement.

If you find one injection site more comfortable than another, there's absolutely nothing wrong with choosing the location you prefer. But based on what we know today, changing where you inject isn't expected to reduce nausea. The good news is that, for most people, nausea improves as the brain adapts to treatment, not because of where the injection is given, but because the brain is adjusting to those stronger fullness signals.

Is Medication Supposed to Make Me Stop Thinking About Food?

Few topics have generated more discussion in the GLP-1 era than *food noise*.

Many people describe a remarkable change after starting treatment. They find themselves thinking less about their next meal, feeling less distracted by cravings, or realising that food simply occupies less space in their minds. For some, it's one of the most life-changing parts of treatment. Because of that, a new expectation has emerged: If food noise disappears, the medication is working. If food noise comes back, it must have stopped working.

The reality is a little more complicated.

Food-related thoughts don't come from a single part of the brain. They can be influenced by hunger, fullness, reward, habits, emotions, stress, boredom, attention, and many other biological and psychological factors. For one person, food noise may be driven mostly by hunger. For another, it may be connected to emotional eating, ADHD, binge-eating disorder, or the rewarding nature of certain foods. Most people experience a combination of several of these influences at the same time.

GLP-1 medications can influence many of these systems. They strengthen fullness signals and reduce the urgency to eat. For some people, they also appear to affect parts of the brain involved in food reward and motivation, which can translate into fewer thoughts about food and less mental energy spent thinking about eating. Some people describe food noise as almost disappearing. But not everyone experiences this in the same way – or even at all. Some notice only a modest change. Others continue thinking about food regularly, even though they're losing weight and responding well to treatment in every other way. None of these experiences mean the medication is

working “better” or “worse.” They simply reflect the fact that food-related thoughts arise from many different brain systems, and those systems vary from one person to the next.

It’s also worth remembering that thinking about food isn’t a problem. Food is essential for life. We should notice hunger. We should plan meals. Cravings are a normal part of being human. We should enjoy eating with family and friends. The goal of treatment isn’t to stop thinking about food altogether. It’s to help food occupy the right amount of mental space – not too much and not too little.

In many ways, food noise follows the same principle we’ve seen throughout this chapter. The goal isn’t to eliminate a normal biological experience. Just as successful treatment isn’t defined by the absence of hunger, it also isn’t defined by the complete absence of thoughts about food. The goal is to restore balance.

Once you understand what obesity treatment is actually designed to accomplish, you’ll start to see the weight-loss world differently. The moment someone decides they want to lose weight, they step into an enormous marketplace built around that decision.



CHAPTER

WHY EVERYONE WANTS A PIECE OF YOUR OBESITY

SIDEBAR

Paying for Permission

Sarah returns for a follow-up appointment several months into treatment. Overall, she's doing well. Her fullness feels more manageable, food occupies less mental space, and she's beginning to trust her body again. But before we finish the visit, she pulls out her phone.

"I keep seeing these advertisements on social media," she says. "This one says I can get generic GLP-1 for $149 per month through an online pharmacy subscription. Is this where I should be getting my medication?"

The advertisement looks polished and professional. At first glance, the monthly fee seems reasonable. But as we look closer, an important detail emerges: the $149 isn't the cost of the medication. It's the cost of access to the medication. The patient still has to purchase the medication separately.

In other words, they're paying twice: once for the medication and once for permission to obtain it.

I pause and ask Sarah a question.

"Can I show you a simple test I use whenever I'm trying to decide whether something belongs to medicine or to weight-loss culture?"

She nods.

"Now, replace the word 'GLP-1' with 'asthma inhaler.'"

She laughs.

"Imagine the advertisement said, 'Join our exclusive asthma membership program for only $149 per month and gain access to the inhaler you need to breathe.'"

The absurdity is obvious immediately.

IMAGINE YOU HAD ASTHMA, high blood pressure, or diabetes, and someone told you that before you could access your medication, you first had to pay a monthly membership fee, subscribe to a coaching program, or purchase an accountability package. Most people would recognize that for what it is: charging people for access to healthcare.

Yet this kind of business model has become surprisingly common in obesity care, and many people no longer question it because weight-loss culture has taught us that this is simply the way weight loss works.

People have become accustomed to paying membership fees, coaching fees, subscription fees, accountability fees, program fees, and consultation packages simply to access treatment, because obesity has spent decades being viewed through the lens of weight loss rather than medicine.

SIDEBAR

The Asthma Test

Here's a simple way to recognize diet culture in the wild.

The next time you hear a message about obesity or GLP-1 medications, mentally replace the word *obesity* with *asthma* and *GLP-1* medication with *asthma inhaler*. Then ask yourself one simple question:

"Does this suddenly sound ridiculous?"

If the answer is yes, you're probably looking at diet culture rather than healthcare.

For example:

- A telehealth company advertises quick access to GLP-1 medications. Now imagine the advertisement promoted quick access to asthma inhalers instead.
- A supplement claims to "boost the effects of GLP-1 medications." Now imagine it claimed to "boost the effects of asthma inhalers."
- A social media advertisement promises discreet home delivery of weight-loss medication without the hassle of seeing a doctor. Now imagine the same message applied to asthma treatment.

Something changes when the disease changes.

Messages that seem perfectly normal in the context of obesity often sound strange, inappropriate, or even irresponsible when applied to another chronic disease. That difference is revealing because it suggests we still aren't viewing obesity through the same medical lens we use for other chronic diseases.

The Asthma Test won't answer every question, but it can quickly help you recognize when obesity is being treated as a commercial opportunity rather than a chronic disease.

Once obesity became one of the most common chronic diseases in society, an enormous marketplace grew around it. Billions of dollars are now made each year by selling the promise of weight loss. Some of these services provide real value. Others profit by convincing people they need far more than evidence-based medical care.

The better you understand obesity as a chronic disease – and the industries that profit from promising to solve it – the harder it becomes for others to convince you that the wrong solution is the right one.

The Business of Weight Loss

Obesity represents one of the largest healthcare markets in the world. Whenever millions of people are searching for answers, a marketplace naturally grows around that need.

That marketplace extends far beyond diets. It includes supplements, hormone testing, meal plans, coaching programs, memberships, online pharmacies, fitness challenges, wearable devices, protein products, accountability groups, and countless other services that promise to help people lose weight.

Most of us encounter this marketplace every day without even realising it. It appears in social media posts, podcast sponsorships, YouTube videos, email newsletters, influencer recommendations, online communities, and conversations with friends and family. One person says carbohydrates are the problem. Another blames hormones. Someone else points to insulin, inflammation, cortisol, gut health, toxins, or seed oils. The explanations may be different, but the message is almost always the same: The answer is just one purchase, one program, one test, or one subscription away.

If you've ever felt overwhelmed by all these competing messages, you're not alone. The problem isn't that there are too few opinions about obesity. It's that there are so many, and many

begin by trying to sell a solution before fully explaining the disease.

The result is that obesity has become one of the few chronic diseases where patients must constantly navigate the boundary between healthcare and marketing. The less obesity is understood as a chronic disease, the easier it becomes for that boundary to blur.

Not every supplement, coach, fitness professional, or nutrition program lacks value. Many people genuinely want to help, and some provide meaningful support. The question isn't whether a product or service has value. The question is whether it addresses the biology of obesity, or simply promises to solve it.

Fortunately, a few patterns appear again and again. Once you recognize them, it becomes much easier to separate evidence from advertising.

Trap #1: The Single Cause Trap

"It's your cortisol."
"It's insulin."
"It's your hormones."
"It's inflammation."

Obesity is a complex chronic disease. Be cautious whenever someone claims to have found the one cause or the one solution. When a disease is complex, simple explanations are often incomplete.

Trap #2: The Transformation Trap

"Become the new you."
"Finally fix yourself."
"This changes everything."

Healthcare is about improving health, not convincing you that you need to be fixed. Be cautious when the message focuses

more on dramatic transformation than on treating a chronic disease.

Trap #3: The Secret Knowledge Trap

"Doctors don't want you to know this."
"The missing piece."
"What nobody is talking about."

Medicine moves forward by sharing evidence. Be cautious when someone claims to have discovered a secret that everyone else has somehow overlooked, especially if the next step is buying a product, a test, or a subscription.

SIDEBAR

What About Peptides?

One of the fastest-growing trends in the obesity marketplace is the promotion of "peptides." They're often advertised as the secret doctors don't want you to know about or as treatments that "Big Pharma" can't profit from.

Be cautious of those claims.

The word *peptide* isn't a treatment; it's simply a type of molecule. In fact, many approved medications, including GLP-1 medications, are peptides. The issue isn't whether something is a peptide. The issue is whether you know exactly what you're injecting and whether it has been tested for safety, quality, and effectiveness.

With an approved medication, your healthcare provider knows exactly what you're receiving, how it was manufactured, how it should be stored, what dose is appropriate, what side effects to watch for, and whether it interacts with your other medications or medical conditions. We can also adjust treatment based on your kidney function, liver function, laboratory results, and overall health because these medications have been carefully studied.

With unregulated peptides, we simply don't have that information.

In some cases, it's impossible to independently confirm that a vial contains what the label claims. We may not know the true dose, the purity of the product, whether it was manufactured under sterile conditions, or whether it contains contaminants. Just as importantly, we often don't know how it interacts with other medications, whether it should be avoided in certain medical conditions, or how to adjust treatment safely if side effects occur.

That uncertainty matters because one of the first principles of medicine is simple: First, do no harm. Every treatment should have a known balance between its potential benefits and its risks. When we don't know exactly what someone is injecting, it's impossible to make that judgment with confidence.

Finally, be cautious when someone claims doctors are hiding peptides because pharmaceutical companies can't profit from them. If a peptide proved to be safe and effective, there would be every incentive to develop it into an approved medication. In fact, that's exactly how many modern medicines – including GLP-1 therapies – came to exist. Pharmaceutical companies routinely modify naturally occurring molecules to improve how they work, how long they last, or how safely they can be used. The barrier isn't whether a company can profit from a peptide. The barrier is demonstrating that it is safe, effective, and can be manufactured to a standard regulators can trust.

Curiosity is a good thing, but your safety should always come first. The better you understand obesity as a chronic disease – and the industries that profit from promising to solve it – the harder it becomes for someone to convince you that the wrong solution is the right one. Understanding the disease is one of the best ways to protect yourself because the obesity market-

place isn't built around biology; it's built around people searching for answers.

The Perfect Customer

The obesity marketplace exists because it reaches people at some of the most vulnerable moments of their lives.

By the time many people seek treatment, they've often spent years – sometimes decades – trying to solve the problem on their own. They've tried diets, exercise plans, meal replacements, supplements, coaching programs, and countless other approaches that sounded convincing at the time. This is a very human response to living with a chronic disease that has been misunderstood for far too long.

In business, the greatest opportunities often exist where people face problems that are common, deeply personal, and difficult to solve. Obesity unfortunately checks every one of those boxes. It affects millions of people, influences health in countless ways, and touches nearly every aspect of daily life. It's no surprise that an enormous marketplace has grown around the promise of solving it.

Most people and organizations genuinely want to help. Many treatments, programs, and healthcare professionals make meaningful differences in people's lives. But whenever there is a large market driven by hope, there will also be people selling certainty where science still has questions – or selling solutions that don't address the underlying biology of obesity. That's why knowledge is one of your best forms of protection. The more clearly you understand obesity, the harder it becomes for anyone to sell you a solution that doesn't match the science.

SIDEBAR

When Fear Becomes a Marketplace

One of the biggest conversations in the GLP-1 era has been about muscle loss. Interestingly, this wasn't a major public concern during decades of bariatric surgery, even though all forms of weight loss – including illness, surgery, dieting, and medications – result in some loss of lean body mass. Today, however, muscle loss has become headline news alongside GLP-1 medications.

Part of the reason is that the science is still evolving. Researchers have consistently shown that people lose some lean body mass along with fat during weight loss. The more important question, however, is whether those changes lead to meaningful problems such as reduced strength, poorer mobility, loss of independence, or worse long-term health. So far, this has not been shown to be the case for most people treated with modern obesity medications. But researchers continue to study these questions, and as the science evolves, so too does the conversation.

Another reason the discussion persists is that many different groups have an interest in it. Pharmaceutical companies are developing new medications designed to better preserve muscle. Body composition companies benefit when more people undergo scans. Supplement and nutrition companies promote protein products and "muscle-support" formulas. The fitness industry offers specialized exercise programs and equipment. Professional societies, researchers, regulators, and guideline committees continue to publish studies, hold workshops, and develop recommendations aimed at improving how muscle health is measured and protected. At the same time, people who are skeptical of medical treatments for obesity often use headlines about muscle loss to argue against these medications altogether. Together, these forces keep muscle preservation in the spotlight.

As a patient, it's helpful to keep your attention on the outcomes that matter most. A body composition scan is not the same as your health. What matters is whether you remain *strong, mobile, independent, and able to do the things that matter in your daily life.* Those are the outcomes that should guide both research and medical care. Clinicians and researchers should continue focusing on standardized, clinically meaningful measures of strength and function, not simply changes in lean body mass on a scan.

Resistance training, regular physical activity, adequate protein, and good nutrition are important parts of managing many chronic diseases, not just obesity. Yet notice how people taking these same medications for diabetes are rarely subjected to the same level of public scrutiny about muscle loss. The intense focus on muscle preservation in obesity reflects, at least in part, another form of weight bias: People living with obesity are often held to a different standard than those living with other chronic diseases.

But this also creates an opportunity for the marketplace. Be cautious when fear becomes the primary marketing message. If someone first convinces you that your medication is harming you and then immediately offers you the solution for sale, it's worth asking whether the recommendation is being driven by evidence – or by the marketplace.

Perhaps the most overlooked consequence of this marketplace is what it teaches people living with obesity about themselves. For decades, society has framed obesity as a problem of personal choice. As a result, many people have come to believe that managing obesity is their responsibility alone. They find themselves researching medications, comparing doses with strangers online, trying to interpret plateaus, troubleshooting

side effects, and deciding whether treatment is "working", often without enough guidance from a healthcare professional.

Think about that for a moment.

We don't expect people with asthma to adjust their inhalers by following social media influencers. We don't expect people with heart failure to troubleshoot their medications by reading online comment sections. We don't expect people with rheumatoid arthritis to compare themselves with strangers to decide whether their treatment is working.

Yet people living with obesity are expected to do all of these things every day, and many have come to believe that's simply what managing obesity requires.

I want you to understand this clearly: This is a form of weight bias.

When society treats obesity as a personal responsibility instead of a chronic disease, people are expected to become their own doctors, pharmacists, researchers, and coaches. They're asked to navigate one of the most complex areas of medicine largely on their own. That isn't how we treat other chronic diseases, and it shouldn't be how we treat obesity either.

Self-Management Is a Form of Weight Bias

One of the strangest expectations in obesity care is that people are often expected to manage this chronic disease largely on their own. Years ago, that often meant assigning points to foods and asking people to stay "within their points." Today, the expectation is more subtle. It often hides behind the convenience of GLP-1 medications. After all, in what other area of medicine would we expect someone to complete an online questionnaire, have prescription medication shipped to their door, and then manage a chronic disease largely on their own?

I've noticed an interesting shift in the GLP-1 era. As these medications have entered public awareness, media platforms,

and the obesity marketplace, people are increasingly expected to evaluate them as though they're both the patient and the doctor – comparing doses, weighing differences between medications, investigating microdosing (taking a smaller-than-prescribed dose, often without medical guidance), and calculating expected weight-loss outcomes.

On one hand, this level of engagement is encouraging. People living with obesity are asking questions, learning about their disease, and wanting to play an active role in their healthcare. That's a good thing. At the same time, it's important to remember that GLP-1 medications aren't vitamins, supplements, or over-the-counter products. They're prescription medications used to treat a chronic disease. Like all prescription medications, they have potential side effects, drug interactions, and contraindications – medical conditions or circumstances that may make one treatment unsafe or inappropriate for one person, even though it may be an excellent choice for someone else.

Choosing the right medication – and the right dose – is a decision that should be made in the context of your own health, not from general advice in a book, podcast, or social media post. Those decisions depend on your medical history, other medications, kidney and liver function, treatment goals, side effects, and many other factors that are unique to you. Think about it this way: We wouldn't expect someone with high blood pressure to choose their medication after reading a book. We wouldn't expect someone with an irregular heartbeat to adjust their heart medication based on advice from the internet. Learning about your treatment is important, but prescribing and adjusting medication should be guided by a healthcare professional who understands your individual circumstances. Obesity deserves that same standard of medical care.

My hope is that this book helps you understand the science behind obesity and the treatments we use, not so you can

manage a complex chronic disease on your own, but so you can have more informed conversations with your healthcare provider and make decisions together.

Treatment with GLP-1 medications should always begin with an individualized medical assessment and continue with ongoing follow-up. This isn't a book about secret prescribing strategies, hidden dosing tricks, or finding the "perfect" medication. As we've discussed throughout this chapter, those kinds of promises should always make you pause. Choosing the right treatment is a medical decision, not a consumer decision. It depends on your medical history, other health conditions, current medications, kidney and liver function, treatment goals, side effects, affordability, medication availability, and what you can realistically maintain over the long term. There is no single "best" medication, dose, or microdosing strategy that works for everyone. My goal is simply to help you understand why these treatments work, what you can realistically expect from them, and how to recognize information grounded in science rather than marketing.

For decades, we've been telling people to manage obesity on their own: eat less, move more, join this weight-loss program. I have a great deal of empathy for how we arrived here. For many years, our understanding of obesity was limited, and there were very few effective treatments to offer. But medicine has moved forward. We now understand far more about the biology of obesity, and we finally have therapies that can meaningfully treat it. Continuing to expect people to manage this disease on their own is no longer supported by what we know.

It isn't fair to place that responsibility entirely on your shoulders. Obesity deserves the same thoughtful, individualized medical care as any other chronic disease. That's also why this book won't tell you which medication to request, how to compare brands, or whether you're a candidate for treatment. It won't teach you how to adjust doses or manage side effects

on your own. Those are medical decisions and conversations that belong in the exam room, made together with a healthcare professional who understands your health, your goals, and the risks and benefits of treatment.

Perhaps nowhere has this expectation of self-management been more obvious than in conversations about *diet and exercise*.

People are constantly searching for the one strategy that will finally make weight loss stick. *What's the best diet? How many calories should I eat? Should I fast? Go low carb? Eat more protein?* The questions are understandable because we've spent decades being told that if we could just find the right eating plan or exercise routine, long-term success would follow.

Yet many people eventually find themselves asking a different question: *If I did everything I was supposed to do, why didn't it work?*

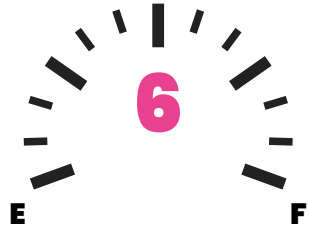
That frustration doesn't mean nutrition, physical activity, and sleep aren't important. They absolutely are. But they matter for reasons that are often very different from what weight-loss culture has taught us. Once we understand obesity as a chronic disease of energy regulation, their role becomes much clearer.

That's where we'll turn next.

PART II

SUPPORTING OBESITY TREATMENT

Why Nutrition, Exercise, and Sleep Matter – But Not for the Reasons You Think



CHAPTER

WHY YOUR DOCTOR NEVER PRESCRIBED YOU A DIET

YOUR OBESITY DOCTOR NEVER prescribed you a diet for the same reason people don't receive meal plans to treat depression.

But if there is one question patients expect obesity specialists to answer, it's this: What's the best diet to follow?

SIDEBAR

"Just Tell Me What to Eat"

Sarah sat down and pulled a notebook from her purse.

"I've been keeping a list," she said.

Over the years, she had tried low-fat, low-carb, keto, intermittent fasting, calorie counting, meal replacements, and more than a few plans she found online. Some worked for a while. Most didn't. A few worked surprisingly well – until the weight came back.

She opened the notebook.

"I don't need another explanation," she said. "Just tell me what to eat."

It's one of the most common requests in obesity medicine.

Some patients arrive expecting a meal plan. Not a medication prescription, but a food prescription. A definitive answer. A secret diet. The plan that finally works when all the others have failed.

The expectation makes sense. For decades, we've been taught that obesity is primarily a nutrition problem. If excess weight is caused by eating the wrong foods, then the solution must be finding the right ones. Nutrition certainly matters, but by the time many patients come to see me, they've spent years – sometimes decades – trying one diet after another. In many ways, they're the experts. They've tried more diets than I've ever heard of, and many could probably teach me more about dieting than I could teach them.

Then something unexpected happens in the obesity clinic.

The doctor doesn't hand Sarah another diet.

No keto plan. No low-fat plan. No daily calorie target. No special obesity meal plan. No forbidden foods. No list of foods she's allowed to eat or foods she isn't.

Instead, the conversation focuses on fullness, weight regulation, genetics, sleep, medications, and long-term disease management. At first, this can feel almost disappointing. After years of searching for the right diet, many patients are surprised to learn that obesity specialists aren't keeping a better diet a secret.

What Dieting Really Teaches Us

When I use the word *dieting*, I'm not talking about nutrition. I'm talking about an entire way of thinking about food. It's a framework built around rules, restrictions, and the promise of weight loss. Some diets eliminate entire food groups. Others tell you exactly when to eat, how much protein to consume, how many calories to count, or which foods you should never

touch. The details change from one diet to the next, but the message remains remarkably consistent: If you can just follow the rules closely enough, the weight will come off.

The problem isn't learning about healthy eating. The problem is confusing nutrition with dieting. Over time, dieting can gradually change our relationship with food.

Instead of seeing hunger as a normal biological signal, we begin to see it as something to ignore or overcome. Foods become "good" or "bad." Eating becomes a test of willpower instead of a normal part of being human. Over time, these behaviours become so common that we stop questioning them. Constantly thinking about food, feeling guilty after eating, fearing certain foods, skipping meals to make up for yesterday, or believing you've been "good" or "bad" because of what you ate can begin to feel normal. Yet outside the context of dieting, many of those same behaviours would raise concerns about disordered eating.

Dieting also changes the way we see each other. It quietly teaches us that body size reflects effort, discipline, or character rather than biology. It encourages bullying, discrimination, and stigma by reinforcing the idea that body size is simply a matter of personal responsibility. It also distracts us from the larger social forces that shape nutrition, including poverty, food insecurity, racism, sexism, and unequal access to healthcare. Together, these messages make weight bias seem normal.

Perhaps the biggest misunderstanding dieting has created is how we think about obesity treatment. We've been taught that there are two separate phases: *First you lose the weight, and then you maintain it*. The message is that weight loss is the real work, while maintenance is simply what comes afterward. But chronic diseases don't work that way.

We don't treat asthma until someone is breathing better and then declare treatment over. We don't separate high blood pres-

sure or diabetes into a "treatment phase" and a "maintenance phase." Treatment continues because the disease continues.

Obesity is no different. Weight loss isn't one phase of treatment followed by maintenance. The entire journey is disease management. Whether you're losing weight, maintaining it, adjusting medications, exercising, or making nutrition decisions, you're doing the same thing you've been doing all along: managing a chronic disease.

Dieting Still Can't Overcome Biology

Even if we set aside the problems of diet culture, dieting still runs into another obstacle: *biology*.

The problem isn't that diets can't produce weight loss. Most diets can produce modest weight loss in the short term. The problem is that your body doesn't simply watch the weight come off and do nothing. As you lose weight, your brain begins activating its anti-starvation defenses. Feelings of fullness become weaker, food becomes more rewarding, and your body begins defending its remaining stored energy.

That's why so many diets feel successful at first. But as the weight comes off, the biology we've discussed throughout this book becomes stronger. Eventually, many people regain some – often most – of the weight they worked so hard to lose. The restrictive diet was sold as the solution, but it was never capable of overcoming the predictable biological response to weight loss. Decades of research have shown this same pattern.

Intensive lifestyle programs – which combine nutrition counselling, physical activity, food tracking, and ongoing coaching – can produce meaningful weight loss, especially in the short term. The challenge is maintaining that weight loss over many years. On average, most dietary interventions struggle to sustain more than a 5 percent reduction in body weight over the long term.

That 5 percent number is important because even modest, sustained weight loss can meaningfully improve health. Blood sugar, blood pressure, fatty liver disease, sleep apnea, and many other obesity-related complications often begin to improve once someone loses and maintains about 5 percent of their body weight. That's why obesity specialists often use 5 percent as an important clinical milestone.

But it also highlights a larger problem.

If the biology of obesity naturally pushes people toward weight regain, then simply telling people to "eat less and move more" cannot, by itself, treat obesity. Nutrition and physical activity remain essential parts of treatment, but on their own they're often not enough to overcome a brain that's actively defending a higher level of stored energy.

An unresolved obesity epidemic will be the unintended consequence of relying solely on "diet and exercise" to treat obesity.

So, What Do Obesity Specialists Actually Recommend?

By now, you might be expecting me to reveal the eating plan that obesity specialists recommend.

There isn't one.

In fact, one of the biggest surprises in obesity medicine is that people living with obesity are generally given the *same nutrition advice as everyone else*.

In Canada, obesity specialists commonly recommend the principles outlined in Canada's Food Guide. In the United States, the recommendations are remarkably similar. Both encourage filling half your plate with vegetables and fruit, choosing whole grains more often, including a source of protein with meals, drinking water regularly, and preparing meals from minimally processed foods whenever possible.

Now, notice what's *not* on that list.

There are no forbidden foods. No mandatory fasting schedules. No rigid carbohydrate limits. No requirement to weigh every gram of food. No rigid protein target at every meal. No curated recipes. And there certainly isn't a special diet reserved for people living with obesity.

That's because *this is nutrition, not dieting.*

These recommendations are meant to be flexible. They can be adapted to different cultures, traditions, family meals, budgets, and personal preferences. Whether your family eats South Asian, Indigenous, African, Latin American, East Asian, Middle Eastern, European, or any other cuisine, the same basic principles apply. Healthy nutrition is about building eating patterns that are realistic enough to last a lifetime.

The same is true for foods we should eat less often. Public health organizations encourage everyone – not just people living with obesity – to limit sugary drinks, excessive alcohol, highly processed snack foods, desserts, and many packaged convenience foods. These foods are often engineered to be easy to eat, highly rewarding, and less filling, making it easier to consume more energy than our bodies need.

That last point is important.

These recommendations aren't unique to obesity because they aren't treating obesity directly. They're recommendations for good nutrition, and good nutrition benefits everyone. People living with obesity need the same healthy nutrition as everyone else, alongside treatments that address the biology of obesity itself.

Why Processed Foods Matter More in Obesity

People living with obesity don't need a perfect diet. They do, however, benefit from paying closer attention to ultra-processed foods. That's because the biology of obesity often makes people more susceptible to today's food environment than someone without obesity.

Many ultra-processed foods are designed to be inexpensive, convenient, highly rewarding, and easy to eat quickly. They often deliver a large amount of energy before your body's fullness signals have time to respond. Liquid calories are a perfect example. Specialty coffees, fruit juices, smoothies, alcohol, meal-replacement drinks, and protein shakes can all provide substantial calories in just a few minutes, often without creating the same lasting fullness as whole foods.

You might be surprised to hear me include protein shakes, powders, and protein bars in that list. As an obesity medicine specialist, I don't recommend them as a regular source of protein.

Throughout this book, I have emphasized the importance of eating whole, minimally processed foods. Promoting protein shakes and bars would contradict that principle, because they are, by definition, ultra-processed foods made from refined protein isolates, sweeteners, flavourings, stabilizers, and other ingredients designed to improve taste, texture, and shelf life.

Whole foods take time to chew, digest, and absorb, giving your brain time to recognize *fullness* – a principle at the heart of obesity treatment. Many highly processed foods, especially liquids, can be consumed very quickly, allowing a large amount of energy to enter the body before those fullness signals fully develop. That's one reason ultra-processed foods are consistently associated with higher energy intake in carefully controlled research studies.

Does that mean you can never have a protein shake or protein bar? Of course not. Nutrition isn't about perfection. There are times when convenience matters, and they may be the most practical option. But whenever possible, try to make whole foods your first choice.

Eggs, full-fat Greek yogurt, cottage cheese, fish, poultry, lean meats, tofu, beans, lentils, nuts, seeds, and dairy provide protein in the form your body evolved to eat. They also contain

fibre, healthy fats, and other nutrients within a natural food structure that slows eating and digestion, helping your brain recognize fullness.

Remember, the goal isn't to eliminate processed foods completely. The goal is to prioritize whole, minimally processed foods so they make up most of your diet. That's a pattern of eating that benefits everyone, but it's especially important for people living with obesity because of how the brain regulates fullness and long-term energy stores.

SIDEBAR

A Note About Whey Protein and Acne

If you've ever started drinking whey protein shakes and then noticed more acne, you're not imagining it. This is common enough that dermatologists and physicians routinely ask about whey protein when someone develops persistent or worsening acne.

Whey protein can trigger or worsen acne in some people. One reason is that whey stimulates the release of insulin and a hormone called *insulin-like growth factor 1 (IGF-1)*. Together, these hormones increase oil production in the skin and promote the growth of skin cells, making pores more likely to become blocked and increasing the risk of acne.

This doesn't happen to everyone, and it doesn't mean whey protein is "bad." It simply means that some people are more susceptible to its effects than others. Several studies have also found that acne often improves after whey protein is discontinued.

If you've developed acne after starting whey protein shakes or powders, one of the simplest things you can do is stop using them for about twelve weeks and see whether your skin improves. If it does, you've likely identified one contributor that you can avoid in the future.

The same principle applies to many foods marketed as "healthy." Granola, protein bars, protein cookies, energy bites, smoothie bowls, and many other "wellness" snacks often carry what nutrition experts call a health halo. Because they're labelled "organic," "high protein," "gluten-free," or "natural," it's easy to assume they're automatically healthier – or even weight-neutral.

That's why learning to read food labels can be so valuable. A registered dietitian can help you understand nutrition labels, plan balanced meals, adapt healthy eating to your culture and lifestyle, and build eating habits you can realistically maintain over the long term. Those are valuable skills that support lifelong health.

What a dietitian cannot do is provide a special diet that, on its own, treats the biology of obesity. If we expect nutrition alone to manage a chronic disease of energy regulation, we're simply repackaging diet culture in a different form and shifting that unrealistic expectation onto the shoulders of dietitians. The real value of dietetic care is helping you apply sound nutritional principles in a way that fits your life while supporting the medical treatment of obesity.

The Protein Trap

Spend enough time online and you'll quickly come across warnings that you're losing muscle, damaging your metabolism, becoming frail, or undoing all of your progress because you aren't eating enough protein. Almost every warning is followed by the same solution: protein shakes, protein bars, protein powders, high-protein snacks, or products marketed specifically to people taking GLP-1 medications.

Protein matters. There is no debate about that. Getting enough protein helps support muscle health, especially during weight loss and resistance training. The problem is what has

happened to the message. A sound nutritional recommendation has become a marketing strategy.

Protein is uniquely marketable. Unlike walking, resistance training, or getting enough sleep, protein can be packaged, branded, advertised, and delivered to your doorstep. As concerns about muscle loss have grown, so has the market for products promising to prevent it.

I regularly see people become frightened by online messaging about muscle loss. They start drinking several protein shakes a day, eating protein bars as meal replacements, and buying every "high-protein" product they can find – even though nothing else about their lifestyle has changed.

This reflects a common misunderstanding.

Protein provides the building blocks for muscle, but physical activity – especially resistance training – is what tells the body what to build. Without that stimulus, simply eating more protein doesn't magically create or preserve muscle. In many cases, it simply adds more energy than your body needs.

A helpful way to picture this is to imagine filling a bathtub. Your daily protein needs fill the tub, while your body's normal functions slowly drain it. Increased physical activity and resistance training opens the drain wider because your muscles need more protein to repair and grow. In that situation, increasing your protein intake makes perfect sense. But if the drain hasn't changed, pouring more water into the tub doesn't make it more useful; it simply spills over.

Your body doesn't have a storage tank for excess protein. Once your protein needs have been met, extra protein is broken down and used for energy. If that energy isn't needed, it's ultimately stored as body fat. In other words, eating far more protein than your body needs doesn't keep building muscle; it starts filling the wrong tank.

The good news is that most people don't need to obsess over protein. For many people, following a balanced eating pattern –

such as the principles outlined in Canada's Food Guide – and including protein-rich foods at regular meals is enough.

I encourage all my patients not to become fixated on hitting exact protein targets. Otherwise, we've simply replaced one form of diet culture with another, trading an obsession with counting calories for an obsession with counting grams of protein. That's still diet culture, just wearing different clothes.

People who regularly perform resistance training or other high levels of physical activity may benefit from a higher protein intake because protein requirements are not fixed. For most adults, approximately 0.8–1.0 g/kg/day (grams per kilogram of body weight per day) is sufficient to maintain muscle mass and support normal daily activities. Following the principles of Canada's Food Guide will easily meet those needs for most people. There is no need to be prescribed a strict daily protein target. When protein is treated as another number you must hit every day, it can start to look a lot like diet culture repackaged. That said, because I am often asked, people who regularly do high-intensity exercise or resistance training may benefit from a higher protein intake, often around 1.2–1.5 g/kg/day. Beyond about 2.0 g/kg/day, additional protein provides little added benefit for muscle building and may contribute to weight gain if it creates an overall energy surplus.

Protein is important, but protein marketing isn't the same as protein science. Before spending money on another shake, bar, or supplement, ask yourself whether you're meeting a genuine nutritional need or responding to a marketing message built around fear.



CHAPTER

FEAR-BASED FITNESS

WHEN OBESITY WAS TREATED as a problem of personal responsibility, exercise became one of the main duties expected of people living with it. That expectation became so deeply rooted in popular culture that entire television shows were built around it. Programs such as *The Biggest Loser* turned weight loss into a public test of discipline. The message was hard to miss: Exercise was the answer.

But something interesting happened along the way.

As obesity science evolved, shame-based explanations and treatments became much harder to defend. Researchers increasingly recognized that obesity is regulated by complex biological systems rather than a simple lack of discipline. At the same time, long-term follow-up of contestants from shows such as *The Biggest Loser* revealed another uncomfortable truth: Dramatic, publicly celebrated weight loss did not necessarily translate into lasting success. Many contestants regained a substantial amount of weight, while some continued to experience persistent biological adaptations that made maintaining weight loss more difficult. These findings raised important ethical questions about using shame, public scrutiny, and extreme weight-loss interventions as entertainment. As shame became

less acceptable, however, the obesity marketplace didn't disappear. It simply found a new emotion to sell: fear. Instead of making people feel guilty for not exercising enough, the message shifted toward making them afraid of what would happen if they didn't.

Today, the warnings just sound different. People are told they're losing muscle, weakening their bones, damaging their metabolism, becoming frail, or developing sarcopenia – a condition in which muscles lose strength and function, making everyday activities more difficult. Then come the solutions: protein powders, high-protein foods, GLP-1 fitness programs, body composition scans, supplements, and specialized fitness plans. The emotion has shifted from shame to fear, but the strategy remains remarkably similar: Create anxiety first, then sell the solution.

This is what makes conversations about physical activity during obesity treatment so complicated. Many of these concerns contain a kernel of truth. Muscle matters. Strength matters. Bone health matters. Physical activity is one of the most valuable tools we have to protect mobility, preserve independence, support mental health, and improve long-term health and quality of life. The challenge is learning to separate evidence-based advice from exaggerated messages designed to make people afraid.

Fear-based fitness advice still tends to treat exercise as though it's simply a matter of motivation, often ignoring the realities of people's lives. Chronic pain, disability, caregiving, shift work, financial constraints, fatigue, and a lack of safe places to be active can all make exercise far more difficult than a motivational slogan suggests.

This chapter isn't an argument against exercise. Quite the opposite. Exercise deserves to be valued for what it can actually do: build strength, protect mobility, preserve independence, support mental health, and help people keep doing the

things that matter most to them. My goal is to free exercise from the burden of being treated as a punishment for body size or a measure of whether someone deserves to lose weight. Exercise is far too important to be judged by what it does to the number on the scale.

Exercise Alone Produces Little Weight Loss

Exercise is remarkably good at many things, but producing significant weight loss isn't one of them. Ironically, our obsession with using exercise for weight loss has overshadowed the many health benefits that matter far more.

Now, imagine having the same conversation with someone being treated for type 2 diabetes. It would sound absurd. We don't tell people that successfully managing diabetes depends on calculating their basal metabolic rate (the amount of energy your body burns each day just to stay alive, even at rest), finding the perfect amount of cardio, or mastering resistance training with a personal trainer. Clinicians understand that exercise is an important part of chronic disease management, but it's rarely expected to be the focus of medical treatment.

The problem arises when exercise is presented as an all-or-nothing solution – as though it alone should be enough to treat obesity. That's simply another version of diet culture. We recommend physical activity because it improves health, preserves function, and works alongside medical treatment, not because it replaces it. Obesity deserves the same approach.

So why doesn't exercise produce more weight loss?

The answer takes us back to biology. Your body is designed to protect you from starvation.

Think about a lion in the wild. A lion's hunt is successful only about one out of every seven attempts. That means lions spend far more time chasing food than eating it. If exercise alone caused large amounts of weight loss, lions would gradually waste away between successful hunts. But they don't. Their

bodies have evolved ways to protect their energy stores when food is scarce.

Humans have many of those same survival systems.

When you become more physically active, your body doesn't simply keep burning more and more energy forever. It begins to adapt. Hunger often increases, making you want to eat more. Your body also becomes more efficient, using less energy for some everyday activities and at rest. You may even find yourself moving a little less without realising it because your body is trying to conserve energy – for example, by fidgeting less. Together, these adaptations help protect your long-term energy stores from being depleted.

That's why exercise alone usually produces only modest weight loss. But that doesn't mean exercise isn't working. It simply means we've been asking it to do a job it was never designed to do. Exercise wasn't designed to make us lose large amounts of weight. It evolved to help us survive while protecting the energy stores our bodies depended on.

Cardio Works Better Than Resistance Training, But That's Not the Point

Many weight-loss books, social media influencers, and fitness programs now present resistance training as the next breakthrough solution for obesity. Resistance training is one of the best things you can do for your health, but meaningful weight loss isn't one of its greatest strengths.

When researchers compare different forms of exercise, aerobic activity consistently produces more weight loss than resistance training. Activities such as walking, jogging, cycling, and swimming are generally more effective at reducing body weight than lifting weights alone. Yet even aerobic exercise produces only modest weight loss. As physical activity increases, the body adapts to defend against further weight loss, limiting the effect of exercise over time. As a result, even

intensive exercise programs typically produce only modest weight loss – often in the range of just two to twelve pounds.

This highlights an important distinction. Cardio produces more weight loss than resistance training, but neither is particularly effective as a treatment for obesity.

Resistance training remains valuable for other reasons. It helps preserve muscle mass, improve strength, maintain physical function, and support long-term health. These benefits are important, but they aren't the same as weight loss. The mistake is expecting resistance training to do something it was never designed to do. Its value isn't in how much weight it helps you lose. Its value is that it helps you live well at whatever weight you're currently managing.

Just as we wouldn't judge a blood pressure medication by how well it lowers cholesterol, we shouldn't judge resistance training by how much weight it causes someone to lose. Confusing these goals is one of the reasons so many conversations about exercise and obesity have become so misleading.

Weight Loss Lowers Metabolism

One reason resistance training is so strongly recommended during weight loss is the belief that it helps "protect your metabolism." When people lose weight, their metabolism usually slows. A smaller body simply needs less energy to keep itself alive than a larger one. That idea makes intuitive sense.

Some of that slowdown is related to muscle. Weight loss is often accompanied by some loss of lean body mass, including muscle, and muscle requires energy to maintain. Because resistance training helps preserve muscle, it's long been viewed as a way to help protect metabolism during weight loss.

The challenge is that metabolism slows for several reasons. A smaller body naturally requires less energy to move, breathe, and perform everyday functions. At the same time, weight loss triggers biological changes that make the body more efficient

with the energy it has. From an evolutionary perspective, this makes sense. If your brain believes food is becoming scarce, conserving energy improves your chances of survival.

This means that after losing weight, most people burn less energy for three reasons: they have a smaller body, they may have slightly less muscle, and their body has become more efficient at using energy. Resistance training can help address one of those factors by preserving muscle, but it cannot prevent the other biological adaptations that accompany weight loss.

This should not minimize the importance of resistance training.

Preserving muscle helps maintain strength, mobility, independence, and physical function as you age. Those are tremendous health benefits. But it's important to understand what resistance training can and cannot do. It helps preserve muscle, but it cannot prevent the normal metabolic adaptations that accompany weight loss or transform resistance training into a highly effective treatment for obesity.

Muscle Doesn't Burn as Many "Calories" as People Think

Muscle is metabolically active tissue, which is one reason resistance training is often promoted as a way to "boost your metabolism." There's some truth to that idea, but the effect is often much smaller than people expect.

Each kilogram (2.2 pounds) of skeletal muscle burns about 10–15 calories per day at rest. That means even gaining 5 kilograms (11 pounds) of muscle – a remarkable achievement for most people – would increase your resting energy expenditure by only about 50–75 calories per day.

Helpful? Absolutely.

Transformative? Not really.

It's also worth explaining why I'm using the word *calories* here. Throughout this book, I've focused on energy regulation

rather than calorie counting because obesity isn't simply the result of "too many calories in and not enough calories out." Obesity is a complex chronic disease governed by the brain and many interacting biological systems. I'm using calorie numbers here because that's how researchers measure metabolism and because it's a familiar way to describe its effects. But don't lose sight of the bigger picture: Body weight isn't controlled by a simple math equation.

The take-home message is simple: Building and preserving muscle is one of the best investments you can make in your long-term health. It improves strength, mobility, balance, physical function, and independence as you age. It also provides a modest increase in resting metabolism. Those are worthwhile benefits, but they aren't the reason resistance training matters most. Its greatest value isn't helping you lose weight. It's helping you stay strong, capable, and independent throughout your life.

Resistance Helps Protect Against Midlife Weight Gain

Earlier in the book, we discussed how midlife brings important hormonal changes for both women and men. Although these changes look different, they share something in common: They gradually make it easier to lose muscle and gain body fat.

For women, the years surrounding menopause are often accompanied by a shift toward storing more fat around the abdomen. For men, testosterone gradually declines with age, contributing to a slow loss of muscle and an increase in body fat over time. These changes are a normal part of aging, but they can make weight regulation feel more difficult than it did years earlier.

This is where resistance training offers additional value.

Unlike using resistance training as a weight-loss strategy, using it during midlife helps your body adapt to the natural

changes of aging. As we get older, we gradually lose muscle if we don't challenge it regularly. Resistance training tells the body, "This muscle is still needed. Keep it."

Some research also suggests that people who regularly perform resistance training gain less weight during midlife than those who remain inactive. It may help slow some of the gradual changes in body composition that naturally occur with aging. In other words, resistance training is often better at helping prevent future weight gain than producing large amounts of weight loss.

By midlife, one of the greatest benefits of resistance training may be something less obvious: helping you stay stronger, healthier, and more capable for decades to come. That's why I encourage people to think of resistance training less as a weight-loss strategy and more as an aging strategy. It isn't just an investment in today's health; it's an investment in the person you want to be twenty or thirty years from now. You're preserving strength, mobility, and independence that your future self will depend on.

SIDEBAR

Is Ozempic Eating My Muscle?

Sarah had been taking semaglutide – the medicine in Ozempic and Wegovy – for twelve months.

At our last appointment, she'd been content. She told me it was easier to get in and out of her car. Climbing the stairs no longer left her out of breath. Her knees hurt less. For the first time in years, she felt hopeful.

This visit felt different.

"I stopped taking it," she said quietly.

"What happened?" I asked.

"I kept seeing videos on Instagram about muscle loss. Everyone was saying these medications destroy your muscles and ruin your metabolism. I got scared. I didn't want to end up weak."

Sarah's concern is becoming increasingly common. Social media is filled with warnings about muscle loss on GLP-1 medications. The message is usually urgent: *Protect your muscle. Save your metabolism. Don't become frail.*

Like many things we've discussed throughout this book, the story contains a kernel of truth.

People do lose some muscle when they lose weight. They always have.

Whether weight loss happens through illness, dieting, increased physical activity, bariatric surgery, or medication, some loss of lean body mass is expected. This isn't unique to semaglutide, and it doesn't mean the medication is somehow "melting away" muscle.

The more important question is this: Do GLP-1 medications cause an unusually large amount of muscle loss compared with other ways of losing weight?

So far, the evidence suggests the answer is *no*.

Studies show that most of the weight lost with GLP-1 medications comes from body fat. Some lean tissue is lost as well, but that's an expected part of weight loss. More detailed MRI studies suggest that only a relatively small proportion of the total weight lost represents skeletal muscle—roughly 7 percent—with much of the remaining change reflecting reductions in body fat, glycogen, and the water stored alongside it.

Perhaps the most important finding, however, isn't what happens on a body composition scan. It's what happens in everyday life. People aren't returning to clinic unable to carry groceries, climb stairs, get out of a chair, or do the activities

that matter to them. In fact, many describe the opposite. As excess weight comes off, moving becomes easier. Joint pain improves. Walking feels less exhausting. Everyday life becomes more manageable.

Preserving muscle is an important part of healthy weight loss. Resistance training, regular physical activity, and eating enough protein all help support strength and physical function. But there is an important difference between protecting muscle and living in fear of losing it.

Sarah thought for a moment.

"So . . . you're saying I probably lost some muscle?"

"Yes," I said. "Just like almost everyone who loses weight."

"But the medication isn't destroying my muscles?"

"No."

The goal isn't to lose weight without losing any lean tissue. That wouldn't be biologically realistic. The goal is to improve your health, protect your strength, make everyday life easier, and minimize unnecessary muscle loss along the way.

Sarah restarted her medication. Our conversation gave her something social media couldn't: context. The story she'd been hearing was built on fear. The conversation we were having was grounded in physiology.

Body Composition Is Not the Goal

Because muscle is such an important tissue, it's completely understandable that people become concerned when they hear they may be losing it. The problem is when that concern is transformed into fear.

Notice how unusual the conversation becomes if we transfer this fear to people using weight-loss medications to treat type 2 diabetes. We don't tell people with diabetes that a treatment can only be considered successful if it lowers blood sugar

without changing body composition. We don't ask whether every gram of lean tissue was preserved before deciding whether their health has improved. Yet when these same medications are used to treat obesity, the conversation often shifts toward an expectation of perfection.

This is where body composition has become one of the newest frontiers of fear-based fitness. People are encouraged to monitor body-fat percentage, muscle mass, DEXA scans, and body composition reports as though these measurements alone determine whether treatment is succeeding or failing.

The reality is much more nuanced.

Body composition measurements have important limitations. Many of the body fat and muscle target ranges promoted today were never developed or validated as treatment goals for people living with obesity, let alone designed to determine whether obesity treatment is succeeding or failing. More importantly, these measurements don't tell us whether someone is actually stronger, moving more easily, or living a healthier life.

More body composition measurements do not always lead to better care. Devices can produce very different results, creating confusion, emotional distress, and an unhealthy focus on numbers that may not change treatment decisions. Unless a measurement is recommended in clinical guidelines and has a clear purpose, adding another scan may cause more harm than benefit.

Part of the issue is that fear captures attention. The message is no longer "You aren't trying hard enough." Instead, it's "You're losing muscle. You're damaging your metabolism. You're becoming frail." Then, almost inevitably, a solution appears: a body composition scan, a protein supplement, a specialized fitness program, or another product designed to solve the problem.

This is a good time to use the Asthma Test.

Imagine you're using an asthma inhaler and see an advertisement that says:

"Breathing better isn't enough. Book our advanced lung function assessment to measure exactly how each part of your lungs is performing."

Lung function can certainly be measured in great detail. Spirometry is a breathing test that measures how much air you can breathe in and out, and more specialized pulmonary function tests can measure different aspects of how your lungs work. These tests have an important role when there's a clinical reason to perform them. But if your breathing is improving, you're exercising more comfortably, you're waking up less at night, and you're no longer needing your rescue inhaler as often, most people wouldn't judge the success of their treatment by whether every number on a detailed lung function report was perfect.

This is not, however, how chronic disease management works. But it is how anxiety is often marketed.

The goal of obesity treatment has never been to produce a perfect body composition report. The goal is to improve health, preserve strength and mobility, reduce the complications of excess body fat, and help people live longer, healthier lives.

If we become fixated on body composition numbers, we risk losing sight of the outcome that matters most: Is this person stronger, healthier, and able to do more than they could before treatment? A body composition report can't answer that question.

The Same Exercise Prescription

People living with obesity are generally encouraged to follow the same physical activity guidelines as everyone else: aim for at least 150 minutes of moderate-intensity aerobic activity each week and include resistance training at least twice a week. Walking, cycling, swimming, hiking, dancing, resistance

bands, weight machines, and free weights all count. There isn't a secret exercise program for obesity.

Exercise improves health in countless ways. It strengthens your heart, builds muscle, protects your bones, improves mobility and balance, helps you sleep better, supports your mental health, lowers blood pressure, improves blood sugar control, and helps you stay independent as you get older. These benefits occur whether you lose a significant amount of weight or not.

That's why I encourage my patients not to overcomplicate exercise. You don't need the perfect workout routine, the ideal heart rate zone, the latest fitness gadget, or a body composition scan to know that being active is helping you. The best exercise program is usually the one you enjoy because you're far more likely to do it consistently. In the long run, consistency matters far more than perfection.

How to Safely Get Started

If exercise is already part of your routine, that's wonderful. Keep building on what you're already doing.

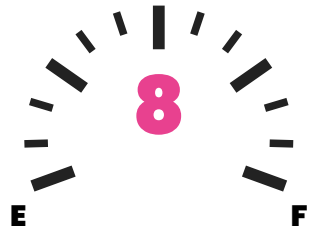
If becoming more active is a new goal, remember that you don't have to figure everything out on your own. Learning a few basic movements with good technique can make exercise feel less intimidating, build confidence, and reduce your risk of injury. For people who are otherwise healthy and don't have significant physical limitations, working with a qualified personal trainer can be a great way to get started.

Early on, many people aren't looking for exercise to be enjoyable; they're trying to overcome fear and uncertainty. Building confidence comes first; enjoyment often follows.

If you live with arthritis, heart disease, diabetes, chronic pain, or other medical conditions, it's often better to begin with a kinesiologist. Kinesiologists are healthcare professionals with specialized training in human movement, exercise physiology,

and designing safe, individualized exercise programs for people with medical conditions. They can help you find activities that fit your body, your health, and your goals so you can move with confidence.

Most importantly, don't feel like you have to do everything at once. The goal isn't to become an athlete overnight. It's to find ways to move that you enjoy, can do safely, and can imagine yourself still doing years from now.



CHAPTER

WHY SLEEP MATTERS MORE THAN YOU THINK

SLEEP IS ONE OF the most overlooked parts of successful obesity treatment. It rarely receives the same attention as nutrition or exercise, even though research consistently shows that insufficient or poor-quality sleep is associated with less weight loss and a greater likelihood of regaining weight over time.

Sleep doesn't just affect how tired you feel. It changes the way your brain regulates hunger, cravings, food choices, and energy use.

Why Sleep Matters (a Lot) for Weight Loss

Let's talk about one of the most overlooked parts of successful obesity treatment: *restorative sleep.*

By restorative sleep, I don't just mean how many hours you spend in bed. I mean waking up feeling rested. If you rarely feel refreshed in the morning, it's worth exploring why. Whether it's sleep apnea, shift work, chronic stress, pain, or another sleep disorder, addressing the underlying cause can make managing obesity much easier.

Getting enough restorative sleep won't cause weight loss on its own, but it creates the conditions that make weight loss more achievable. Poor-quality or insufficient sleep does the opposite. Research consistently shows that people who sleep less have a higher risk of weight gain and obesity. In fact, compared with sleeping about seven hours a night, each hour less of sleep has been associated with roughly a 9 percent higher risk of obesity.

The reason takes us back to your body's fuel gauge.

When you don't sleep well, your brain interprets it as a form of stress. From an evolutionary perspective, poor sleep may signal that conditions are uncertain or unsafe. A tired brain is much more likely to believe the fuel tank is running low, even when it isn't. In response, it becomes more protective of its energy stores. It increases the drive to eat, especially foods rich in sugar, fat, and energy, while making those foods harder to resist.

Part of this response involves changes in your hormones. A poor night's sleep activates the body's stress response, causing cortisol – a key stress hormone – to remain elevated when it would normally begin to fall. This is accompanied by an increase in ghrelin, the hormone that stimulates hunger, while making highly rewarding foods seem even more appealing. It's no surprise that after a restless night you often feel hungrier, snack more, and crave sugary or high-fat foods the next day.

Sleep loss also affects the way your brain makes decisions. Studies show that when we're sleep deprived, the brain becomes more responsive to the sight and smell of tempting foods, while the parts responsible for planning, self-control, and decision-making become less active. Meal planning feels harder. Cravings feel stronger. Resisting impulses takes more effort. All of this is simply how a tired brain responds when it believes the fuel tank is running low.

Even modest sleep loss can have measurable effects. Compared with sleeping eight to nine hours a night, sleeping only five to six hours is associated with higher ghrelin (hunger) levels. In other words, the less restorative sleep you get, the more your biology pushes you toward eating.

This is why I encourage patients to think of sleep as the third pillar of obesity support, alongside nutrition and physical activity. It isn't optional, and it isn't a luxury. For any Zelda fans, think of nutrition, physical activity, and sleep as the Triforce. When one piece is missing, the whole system becomes weaker. Sleep helps your brain regulate hunger, fullness, and energy balance, making it an essential part of successful obesity treatment.

How Much Sleep Is Ideal?

Research suggests there is a "sweet spot" for sleep when it comes to weight regulation. Adults who regularly sleep about seven to eight hours each night tend to have the lowest risk of obesity. Sleeping much less than that is associated with a higher body weight, but interestingly, sleeping much more is also linked with a higher body weight. Researchers describe this as a U-shaped relationship: Both too little and too much sleep are associated with poorer weight outcomes.

The strongest evidence, however, points to too little sleep as the bigger problem. As sleep duration drops below about seven hours per night, the risk of obesity rises in a stepwise fashion. The further sleep falls below this range, the harder it becomes for your brain to regulate hunger and maintain energy balance.

Studies have also shown that sleeping less than about six and a half hours a night causes people to eat more the following day. Even more concerning, the extra weight gained during periods of sleep restriction appears to be stored preferentially as visceral fat – the fat that surrounds the organs deep inside

the abdomen and is more strongly linked to diabetes, fatty liver disease, and heart disease.

One of the most surprising findings was that a few nights of "catch-up sleep" didn't immediately reverse this increase in visceral fat, at least not over the short period studied. This reminds us that consistently getting enough restorative sleep is far better than trying to make up for chronic sleep deprivation on weekends.

That's why identifying and treating sleep apnea is such an important part of obesity care. By restoring healthy sleep, treatment reduces one of the biological forces pushing the brain's fuel gauge toward "empty."

One of the most effective treatments for obstructive sleep apnea is continuous positive airway pressure (CPAP). A CPAP machine gently delivers air through a mask while you sleep, keeping the airway open so your brain can experience normal, restorative sleep again.

More recently, obesity treatment itself has also become part of sleep apnea treatment. In 2024, tirzepatide became the first medication approved to treat moderate-to-severe obstructive sleep apnea in adults with obesity, recognizing that treating the underlying obesity can also improve the sleep disorder. This represents an important shift in how we think about treating sleep apnea – by addressing one of its underlying causes.

Research suggests that people with obstructive sleep apnea who use CPAP while participating in a weight-loss program often lose more weight than similar patients who do not use CPAP. To be clear, using a CPAP machine does not cause weight loss on its own. Rather, by restoring healthy, restorative sleep, it helps reduce the biological pressures created by poor sleep that can work against obesity treatment.

Together, CPAP and obesity treatment address different parts of the same problem. CPAP keeps the airway open while you sleep, allowing your brain to recover. Obesity treatment

helps reduce the excess body fat that contributes to narrowing the airway in the first place. One treats the sleep disorder directly, while the other treats one of its major underlying causes. Rather than competing treatments, they are complementary. By improving sleep and treating obesity together, each makes the other more effective.

When Your Body Clock Is Off

Another way sleep affects weight regulation is through your circadian rhythm. Think of it as your body's internal twenty-four-hour clock. It helps coordinate when you feel awake, when you feel sleepy, when you're hungry, how your hormones are released, and how your body uses energy. This clock is synchronized by the natural cycle of light and darkness.

Your circadian rhythm is also one of the systems that helps calibrate your brain's fuel gauge. When that clock is disrupted, the gauge becomes less reliable, making hunger and energy regulation harder to manage.

Problems arise when your internal clock and your daily routine no longer match. Researchers call this circadian misalignment. It happens when you're awake, eating, or working while your brain is trying to prepare you for sleep. Shift work is one of the most common examples, but frequent jet lag, irregular sleep schedules, and staying up very late most nights can also disrupt your body's natural rhythm.

When your internal clock and your daily routine fall out of sync, your body experiences it as a form of stress. As we've discussed throughout this book, stress tends to push the brain's fuel gauge closer to "empty," making hunger stronger and long-term energy storage easier. This is one reason disrupted sleep and irregular sleep schedules are linked to a higher risk of weight gain and obesity.

One of the key hormones involved in this process is melatonin. As daylight fades in the evening, your brain begins

releasing melatonin. Many people think of melatonin as the "sleep hormone," but its role is much broader than simply making you feel sleepy. It signals to your body that nighttime has arrived, helping coordinate the many biological processes that occur while you sleep.

Under normal circumstances, your circadian system helps reduce hunger overnight and supports the normal rhythm of hormones involved in appetite regulation. This allows you to sleep instead of waking up to search for food. When your sleep schedule becomes irregular, those normal biological rhythms become disrupted, making it easier to feel hungry at the wrong times and harder for your brain to regulate long-term energy stores.

SIDEBAR

Special Note on Over-the-Counter Sleeping Aids

Many people who struggle with sleep eventually reach for an over-the-counter sleep aid. What many don't realise is that the active ingredient in many of these products – diphenhydramine – may quietly work against their weight goals when used regularly.

Diphenhydramine is an antihistamine commonly used in over-the-counter sleep aids because one of its side effects is drowsiness. But histamine also helps your brain recognize fullness. When histamine is blocked, some people feel less satisfied after eating.

Large population studies have found that people who regularly use antihistamines tend to have a higher body weight, larger waist circumference, and higher insulin levels than people who don't. These studies can't prove that antihistamines directly cause weight gain, but they do raise important questions about how long-term use may affect appetite regulation and metabolism.

The good news is that occasional use is unlikely to have a meaningful effect on your weight. The greater concern is relying on these medications night after night for months or years.

If you find yourself needing an over-the-counter sleep aid most nights, think of that as a signal rather than a solution. Instead of simply masking the problem, it's worth working with your healthcare provider to understand why you're not sleeping well. Whether the cause is sleep apnea, chronic stress, pain, shift work, or another sleep disorder, treating the underlying cause is much more likely to improve both your sleep and your ability to manage your weight.

When Your Brain Thinks It's Still Daytime

One of the simplest ways we sabotage our sleep is by staying glued to our phones late into the evening. Your brain relies on darkness to know it's time for bed. As the sun sets, it begins releasing melatonin, the hormone that signals to your body, "Night has arrived. It's time to slow down." Melatonin helps coordinate your body's overnight repair and prepares your brain for restorative sleep.

Bright light – especially the blue-enriched light from phones, tablets, computers, and televisions – interrupts that signal. When we're scrolling through social media, replying to emails, or watching videos late into the evening, we're essentially telling our brain that it's still daytime. Melatonin release is delayed, our internal body clock shifts later, and the natural sleepiness that should develop in the evening never fully arrives.

Our modern homes make this even harder.

For most of human history, homes became naturally dark after sunset. Today, many of us spend our evenings surrounded by bright ceiling lights, televisions, computer screens, kitchen

lights, and countless glowing LEDs. Then, the moment the final episode finishes, we switch off the lights, crawl into bed, and expect our brain to fall asleep on command. But our brains can't instantly switch from "It's daytime!" to "Time for sleep!" simply because the latest episode of *Grey's Anatomy* ended.

A growing body of research shows that people who regularly use smartphones and other screens before bed are more likely to gain weight and develop obesity. That's yet another way poor sleep nudges the brain's fuel gauge toward "empty," making it much harder to regulate hunger and food cravings the next day.

The good news is that small changes can make a big difference. Try dimming the lights and putting away bright screens one to two hours before bedtime whenever possible. Let your home gradually become darker as your body prepares for sleep. These simple habits help your brain release melatonin naturally, make it easier to fall asleep, and support the biological systems that regulate hunger, fullness, and energy balance.

Not all sleep alignment problems are caused by screens. Two other important examples are shift work, where work schedules conflict with the body's natural sleep-wake cycle, and night eating syndrome, a rare condition in which people consume a large proportion of their daily food in the evening or during the night. Both deserve proper medical attention because treating them can be an important part of improving both sleep and weight regulation.

Shift Work: When Your Body Clock Works Against You

If you work nights or rotating shifts, you're not imagining it if managing your weight feels harder.

Shift work asks your body to stay awake when its internal clock is trying to put you to sleep. Bright lights, overnight meals and snacks, fatigue, and an irregular sleep schedule all

work against your body's natural rhythm. Instead of allowing melatonin – the hormone that helps coordinate sleep – to rise naturally, your brain continues receiving signals that it's still daytime.

The result is a brain that pushes its fuel gauge closer to "empty." Hunger becomes stronger, while fatigue makes it harder to plan meals and make thoughtful food choices. Over weeks, months, and years, these small changes can make weight gain much more likely.

Then the challenge continues after your shift ends. Many people struggle to fall asleep after getting home because the sun is up, the world is awake, and their body clock is still out of sync. The result is less restorative sleep, more hunger the next day, and the cycle begins again.

If you work nights or shift work, here are a few practical strategies that may help.

1. Take a "distraction break."

Not every overnight craving is true physical hunger. When your body's internal clock is disrupted, it's common to crave food simply because your hunger signals are stronger than they should be. Before automatically reaching for a snack, ask yourself, "Am I physically hungry, or am I just tired and looking for something to do or perk me up?" Sometimes taking a short walk, chatting with a coworker, reading the news, or stepping outside for a few minutes is enough to let the craving pass.

2. Choose foods that help you stay full.

Late-night cravings often pull us toward highly processed snack foods because they're convenient and often accessible. Unfortunately, they're also less filling and make it easy to keep eating. When you do need to eat during your shift, try choosing meals or snacks that include whole

foods, fibre, and protein. These are more likely to keep you satisfied than chips, candy, or sugary drinks.

3. Help your brain know that bedtime is coming.

As your shift comes to an end, start preparing your brain for sleep. Reduce your exposure to bright light whenever possible. Switch your phone to night mode, dim screens, and consider wearing sunglasses on your commute home if it's already daylight. The goal is to send your brain the message that bedtime is approaching, even if the sun disagrees.

4. Limit caffeine toward the end of your shift.

Caffeine can be helpful early in a night shift, but drinking it too close to the end of your shift can make it much harder to fall asleep once you get home. Think of the last part of your shift as preparing for sleep rather than preparing to stay awake.

While you can't eliminate all the challenges that come with working nights, understanding how your body responds can help you make small changes that support better sleep, healthier appetite regulation, and better long-term health.

When Your Body Wants to Eat at Night

Another form of circadian misalignment that is particularly relevant to obesity is night eating syndrome (NES).

NES was first described in 1955 by psychiatrist Dr. Albert Stunkard. Today, it is recognized as an eating disorder in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5) under Other Specified Feeding or Eating Disorder (OSFED), although researchers are still working to better define its diagnostic criteria. It is more common than many people realise. About 1–2 percent of the general population is thought to have

NES, but among people seeking treatment for obesity, the number may be closer to one in twenty.

People with NES often notice a very different eating pattern from most others. They may have little or no appetite in the morning, eat a large proportion of their daily food in the evening, or wake during the night specifically to eat. Many also struggle to fall asleep, especially during periods of stress, and some describe feeling as though they can't fall asleep unless they've eaten something first.

Like many of the conditions we've discussed throughout this book, NES appears to involve changes in the brain's normal circadian rhythm. Researchers believe the biological signals that normally coordinate sleep, hunger, and fullness become disrupted, making the drive to eat stronger during the very time the body should be preparing for sleep. Not surprisingly, this can make weight management much more difficult, even when someone is trying very hard to lose weight.

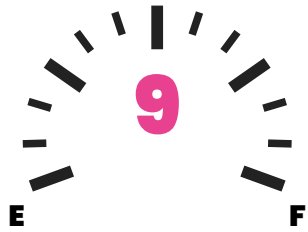
Throughout this section, we've explored nutrition, physical activity, and sleep, not as ways to overcome obesity through willpower but as ways to support the biological systems that regulate body weight. Together, these chapters remind us that obesity treatment isn't built around a single medication or a single habit. It's built around understanding your biology and working with it over time.

That brings us to one final question: Once we understand obesity as a chronic disease, how do we live well with it for the rest of our lives?

PART III

WEIGHT STEWARDSHIP

Shifting the Mindset from Weight Loss to Weight Stewardship



CHAPTER

HOPE THAT DOESN'T DEPEND ON FINISH LINES

UNDERSTANDING OBESITY IS ONLY part of the picture. Learning how to live well with it is the next challenge. If obesity is a chronic disease, it can't be treated like a temporary transformation project with a finish line. It calls for a different way of thinking, one that shifts the focus from losing weight to caring for your health over the long term. That is the mindset I call *weight stewardship*.

This is also one of the hardest ideas to accept: There may not be a finish line. At first, that can feel discouraging. Most of us have been taught to believe that if we just work hard enough, we'll eventually reach a point where the work is done – a place where we no longer have to think about our weight, take medication, or pay attention to the disease at all.

That hope has fueled countless diets, exercise programs, transformation challenges, and New Year's resolutions. We've been promised that if we can just make it to the finish line, everything will finally become easy.

But chronic diseases don't work that way. While that realisation can feel disappointing at first, it also opens the door to

something far more realistic – and ultimately, far more hopeful.

Finish Lines Create Fragile Hope

Finish lines are powerful because they give us hope. But the hope they offer can be surprisingly fragile. It depends on a specific future outcome: *I'll finally be okay once I reach my goal weight*.

As long as the number on the scale keeps moving, that hope feels secure. But when weight loss slows, a plateau occurs, weight returns, or treatment lasts longer than expected, that hope can begin to fall apart. Many of the frustrations people experience during obesity treatment aren't even about the weight itself. They're about grieving the future they believed was waiting for them – a future that never arrived.

To understand why finish lines have such a powerful hold on us, we really need to look at the culture surrounding weight. Weight-loss culture is built on the idea of transformation. Every story has a beginning and an ending. A *before* and an *after*. An old self and a new self. Success is portrayed as crossing a finish line and leaving your former self behind forever.

Because this message is everywhere, most of us never stop to question it.

Now let's consider how other chronic diseases are managed. We don't talk about "finishing" high blood pressure. We don't celebrate the completion of asthma. We don't think of diabetes as a temporary project that ends once someone's blood sugar reaches its target. The goal isn't to "complete" the disease. The goal is to manage it well. Obesity deserves the same understanding.

Losing weight doesn't make the disease disappear, just as lowering blood pressure doesn't cure hypertension, the medical term for high blood pressure. The biology that led your body to defend a higher weight is still there. Treatment may

change over time. The amount of support you need may change. Your weight may change. But the disease is still there, and it still needs care.

Finish lines are also appealing because our brains naturally create them. We often convince ourselves that reaching one milestone will finally bring lasting happiness, confidence, or peace. But once we get there, the goal usually moves. Someone might believe they'll feel secure once they save $10,000. Then the goal becomes $50,000. Then $100,000. Then $1 million. Each achievement is real, but the feeling of "enough" often fades because the brain quickly adapts and sets a new target.

The same thing can happen with weight.

A person may dream of reaching 220 pounds. Once they get there, the goal becomes 200. Then 180. Then 170. Before long, they're chasing a weight they haven't seen since they were a teenager. The finish line keeps moving because the belief underneath it hasn't changed: *I'll finally be happier on the other side of the next goal.*

But lasting hope doesn't come from chasing an ever-moving finish line. It comes from learning how to care for yourself wherever you are today. That's the heart of weight stewardship.

SIDEBAR

Sarah and the Moving Finish Line

When Sarah first came to see me, she was convinced everything would be different once she reached 200 pounds.

For years, that number had lived in her mind as the dividing line between the life she had and the life she wanted. If she could just get there, she believed she would finally feel comfortable in her body. She would stop worrying about her weight. She would stop thinking about food so much. She would finally feel like she had made it.

With treatment, Sarah gradually lost weight. Eventually, she reached 200 pounds.

But the feeling she had been waiting for never came.

Within weeks, the goal had changed.

Now she wanted to reach 180 pounds. As she got closer, her attention shifted again. Then it became 170. Before long, she found herself looking at photos from her twenties and wondering if she could get back to a weight she hadn't seen in decades.

One day, she stopped and asked herself a simple question:

If I wasn't happy at 200 pounds, what makes me so sure I'll be happy at 170?

That question began to change her perspective.

Sarah realised the problem had never been the number itself. It was the belief that peace, confidence, and happiness were waiting on the other side of the next milestone. Every time she crossed a finish line, a new one appeared.

What Sarah experienced is something many of us experience.

We place our hope in a future destination and then wonder why arriving there doesn't feel the way we imagined. That's because most people don't actually want a specific number on the scale. They want what they believe that number will give them.

If this sounds familiar, pause for a moment and ask yourself:

What do I believe will be different when I reach my goal weight?

What am I hoping to feel?

What am I hoping will become easier?

What am I hoping will finally stop?

For some people, the answer is confidence. For others, it's freedom from constantly thinking about their weight. Some hope for better health, more energy, easier movement, or greater independence. Others hope to feel more accepted, more attractive, or simply more comfortable in their own skin.

These hopes are deeply human.

The challenge is that we often tie all of them to a single number on the scale, believing they can only happen after we reach it.

But is that really true?

Can confidence only exist at one particular weight?

Can better health only begin after crossing a finish line?

Can life only be enjoyed after losing another ten pounds?

Sometimes the most important realisation is that many of the things we're searching for don't have to wait. They can begin long before we reach our next milestone.

Goals are meant to help us live out our values, not *become* our values.

When Sarah looked more closely at what she truly wanted, she realised her real goal was never 170 pounds. She wanted to feel healthier, have more energy, and feel more in control of her health. Losing weight was one way of moving toward those things, but it was never the destination itself. From that day forward, she approached treatment differently. She still had goals, but they no longer defined her hope. The number on the scale would no longer carry the weight of her self-worth or her future happiness.

This is where healthy goals can slowly become unhealthy. There is nothing wrong with wanting to lose weight or improve your health. The problem begins when a number on the scale starts to determine how you feel about yourself. Over time, it's

easy to slip into all-or-nothing thinking: A lower number means you've succeeded, while a higher number means you've failed. Food and weight begin to take up more and more space in your mind. Small changes on the scale can feel emotionally overwhelming, and slower progress can become a source of frustration, self-doubt, or anxiety. Without realising it, the scale stops being a tool that provides information and becomes a daily report card on your worth.

That doesn't serve you.

Instead of chasing finish lines, I encourage my patients to embrace weight stewardship. Weight stewardship is the ongoing practice of caring for your health and managing your weight over time, rather than trying to reach a finish line and be done. It recognizes that if obesity is a chronic disease, then learning how to live well with it is just as important as losing weight.

Weight stewardship offers a different kind of hope. Instead of placing your hope in one day being finished, it places it in becoming capable. The focus shifts from reaching a destination to building the knowledge, skills, habits, and support you need to care for yourself over the long term. That hope is more durable because it doesn't depend on reaching a particular number on the scale or becoming a different version of yourself. It comes from knowing that, whatever tomorrow brings, you have the ability to keep caring for your health.

Freedom from the Transformation Project

Understanding that obesity is a chronic disease also helps protect us from the "transformation" trap. Chronic diseases are complex and constantly changing. They are influenced by far more than willpower alone. Hunger, fullness, metabolism, genetics, sleep, stress, medications, your environment, and life's ups and downs all affect body weight. Expecting one finish line to permanently solve something this complex isn't

realistic. That's why obesity is managed over time, not completed like a transformation project. While that may sound discouraging at first, it offers something much more valuable: freedom from the exhausting belief that your life can't truly begin until you reach a certain weight.

This way of thinking also changes how we see the scale. Many people treat it like a report card on their effort, discipline, or worth. I don't. As an obesity medicine physician, I see your weight as a treatment measurement. Just as blood pressure helps me understand how well my treatment is working for someone with hypertension, your weight helps me understand how well your obesity treatment is working. Trust me when I tell you this: When I look at your weight, I'm not judging your effort; I'm evaluating the treatment. Your weight is simply information that helps me understand how well it's working and guides the medical decisions we make together.

Ultimately, weight stewardship asks us to place our hope somewhere more dependable. Instead of hoping that one day we'll finally be finished, we can hope to become more capable of managing the disease. Instead of waiting for a final destination, we can build confidence in our ability to navigate whatever comes next. That's the kind of hope that lasts because it isn't tied to reaching a finish line. It's built on knowing you can keep caring for your health, wherever the journey takes you.

From Weight Loss to Weight Stewardship

One of the most important shifts in obesity care is moving from a weight-loss mindset to a weight stewardship mindset. At first, those ideas may sound similar, but they lead to very different experiences. Weight loss focuses on reaching a finish line. Weight stewardship focuses on caring for your health over the long term. It offers a kind of hope that doesn't depend on one day being "finished."

Many people begin obesity treatment believing it will be a transformation project. There's a starting point, a goal weight, and the expectation that success means eventually crossing the finish line. That way of thinking can feel motivating, but as we've discussed, it's also fragile. As long as the weight keeps going down, everything feels on track. But when weight loss slows, a plateau occurs, weight returns, or treatment lasts longer than expected, it's easy to wonder *What am I doing wrong?* or *Have I failed?*

Weight stewardship offers a different perspective. To be a steward means to care for something over time. The goal isn't to reach a finish line and walk away. It's about becoming the kind of person who knows how to care for their health, no matter what challenges tomorrow brings.

The difference between these mindsets becomes clearer when placed side by side.

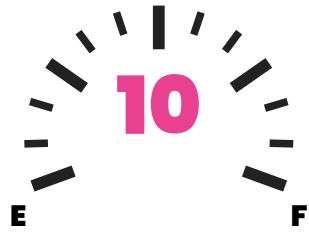
Weight-Loss Mindset	Weight Stewardship Mindset
Focused on reaching a goal weight	Focused on supporting long-term health and well-being
Sees treatment as something to finish	Sees treatment as ongoing care that evolves over time
Looks for a finish line	Focuses on learning, adapting, and caring for health over time
Plateaus feel like something has gone wrong	Plateaus are a normal part of managing a chronic disease
Weight regain feels like failure	Weight regain is information that helps guide the next step
Measures progress mainly by the number on the scale	Measures progress by health, function, consistency, and quality of life
Asks "When can I stop?"	Asks "How can I keep caring for my health?"
Hope comes from reaching a destination	Hope comes from knowing you can navigate whatever comes next

Table 9.1. The Weight-Loss Mindset Versus the Weight Stewardship Mindset. How each mindset shapes the way we think about goals, treatment, plateaus, weight regain, and progress.

Weight stewardship offers something much more freeing. Treatment changes are no longer seen as signs that something has gone wrong. They become a normal part of managing a chronic disease. Needing to adjust your treatment over time means you're responding to a body and a life that are always changing. Long-term management isn't the consequence of treatment. Long-term management is the treatment.

Once we begin thinking this way, the questions begin to change. Instead of asking "How much more weight can I lose?" we ask "What helps me stay in control of my health?" Instead of asking "When can I stop treatment?" we ask "What level of treatment helps me stay well?" These are the questions of weight stewardship.

This new mindset also changes the way we talk about obesity. Words like *cheating*, *falling off the wagon*, *being good*, *being bad*, or *starting over* come from the idea that weight loss is a temporary project. They don't fit a chronic disease that requires ongoing care. If we're going to think differently about obesity, we also need to speak differently about it. The words we choose shape how we think about ourselves. They can build self-compassion and resilience or quietly reinforce shame, guilt, and self-judgment.



CHAPTER

WHY WORDS MATTER

THE WORDS WE USE to talk about obesity shape much more than conversations. They shape what people expect treatment to accomplish, how they interpret their experiences, and whether they see themselves as "succeeding" or "failing." They also shape how other people respond to them. Many of the frustrations people experience during obesity treatment aren't caused by biology. They arise from the language of diet culture, which quietly creates unrealistic expectations.

If obesity is a chronic disease, then it deserves the same precision in language that we use for every other chronic disease. The words we choose today will help define the future culture of obesity treatment. Many of the words we use in obesity treatment carry hidden assumptions about what treatment is supposed to look like. Table 10.1 highlights some of the most common phrases, the expectations they quietly create, and how a weight stewardship mindset reframes them.

One of the most common phrases is *weight-loss journey*. The word *journey* makes it sound as though you're headed toward a final destination. But no one is handing you a boarding pass or taking you on a cruise. Obesity treatment isn't a journey with

an endpoint. It's the long-term management of a chronic disease. We don't talk about cholesterol journeys, asthma journeys, or blood pressure journeys for the same reason.

The same problem exists with the phrase *weight-loss story*. Stories have a beginning, a middle, and an end. They eventually reach a final chapter. But obesity treatment doesn't. Unless a publisher is signing you to write a book, your goal isn't to finish a story. It's to learn how to manage a chronic disease over time.

Perhaps no phrase creates more confusion than *goal weight*. Words like *goal weight* don't just describe treatment; they shape expectations. When the goal is defined as a single number, it's easy to overlook the meaningful improvements in health, function, and quality of life that happen along the way.

The truth is there is no biologically "correct" goal weight; just as there is no biologically "correct" goal height. Your genes play a major role in how your body stores fat, regulates hunger and fullness, and uses energy. Treatment can shrink fat cells and improve how your body functions, but it doesn't erase the biology that made weight gain possible in the first place. That's why the real goal isn't to reach one perfect number on the scale. It's to improve your health.

Language surrounding medication can also create misunderstandings. Medications are often described as tools. Crows use tools. Humans use treatments. We don't describe inhalers as tools. We don't describe blood pressure medications as tools. Calling obesity medications tools subtly suggests they're optional accessories rather than legitimate medical treatments.

You may also hear that medications only work if you're willing to "do the work." While often well-intentioned, this phrase can create unnecessary guilt. Studying requires work. Earning a paycheque requires work. Biology requires treatment. And managing *any* chronic disease requires ongoing effort along-

side that treatment. The purpose of treatment is not to eliminate effort. The purpose is to make the effort more effective.

Then there's the word *lifestyle*. It feels more appropriate for a home decorating magazine than a discussion about chronic disease. Obesity is often described as a lifestyle disease, yet we rarely apply the same label to heart disease, hypertension, or asthma. Lifestyle factors can influence many diseases, but that does not make them lifestyle diseases. Obesity is no exception.

Common Term or Phrase	Hidden Expectation	Weight Stewardship Perspective
Weight-loss journey	There is a destination waiting at the end.	Obesity is managed over time, not completed.
Weight-loss story	Every story has a final chapter.	There is no ending, only ongoing care.
Goal weight	There is one "right" number to reach.	The goal is better health, not a perfect weight.
Medication is a "tool."	Medication is optional.	Obesity medications are legitimate medical treatments, ranging from optional to medically necessary, depending on the individual.
Lifestyle disease	Obesity is mainly caused by personal choices.	Obesity is a chronic disease influenced by biology, environment, and behaviour.
Beat obesity • Cure obesity • Reverse obesity	Treatment eventually ends.	Health can improve, but long-term management continues.
Stay on track • Get back on course • Fell off the wagon	Falling off means you've failed.	Life happens. Treatment adapts and continues.

Common Term or Phrase	Hidden Expectation	Weight Stewardship Perspective
Weight maintenance	Treatment happens in phases.	There is no separate maintenance phase, only ongoing treatment.
Successful weight loss	There is a finish line—and failure if you don't reach it.	Stewardship means learning to care for your health over time.

Table 10.1. Common words or phrases from dieting culture that carry misleading expectations. Every expectation shapes how we experience treatment. Choosing language that reflects chronic disease management helps replace shame, unrealistic expectations, and finish-line thinking with stewardship, self-compassion, and long-term care.

Another common source of confusion is language about *curing*, *reversing*, or *beating* obesity. These words imply that treatment eventually ends. Obesity has no cure. Weight can improve. Health can improve. Symptoms can improve. Disease burden can improve. But the underlying biology that made weight gain possible doesn't disappear.

Even seemingly harmless phrases such as *staying on track* or *getting back on course* deserve scrutiny. Treatment isn't a race, and there is no track to fall off. No one is challenging you to a game of *Mario Kart*. Life happens. Stress increases. Schedules change. The question isn't whether you stayed perfectly on course. It's whether treatment continues despite life's normal disruptions.

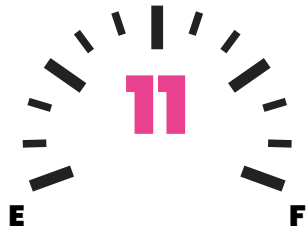
The phrase *weight maintenance* creates another problem because it implies that treatment occurs in phases. First comes weight loss. Then comes maintenance. In reality, there is only treatment. The same disease is being managed before, during, and after weight loss. The scale may behave differently, but the treatment itself hasn't fundamentally changed.

Finally, we should talk about the word *success*. Success sounds positive, but it carries a hidden assumption: that there is a finish line. If success exists, then failure must exist too.

Chronic disease management doesn't work that way. There is no graduation ceremony for asthma. There is no trophy for maintaining blood pressure. Obesity treatment isn't about success or failure. It's about stewardship.

That may be the most important language shift of all.

Stewardship recognizes that obesity requires ongoing attention, not a temporary transformation. It replaces fragile hope built around finish lines with a more lasting hope rooted in long-term care. It allows setbacks to become information instead of judgment. It makes room for biology, for humanity, and for life itself. Most importantly, it reminds us that the goal was never to become a different person. The goal has always been to become the healthiest version of yourself.



CHAPTER

THE COMPARISON TRAP AND WHY THOUGHTS MATTER

ONE COMMON EMOTION PEOPLE experience during obesity treatment is self-doubt. It often starts innocently. Someone in an online support group shares that they've lost fifty pounds. A friend reaches a lower weight while taking a smaller medication dose. A social media influencer posts dramatic before-and-after photos. Before long, instead of noticing your own progress, you find yourself wondering why your results don't look the same.

The comparison itself isn't the problem. It's the story your mind begins to tell you. Those thoughts can quietly shape how you feel about yourself, your treatment, and whether you believe you're making progress at all.

It is natural, by the way, to feel uncertain about your own progress and look to others for reassurance. That's part of being human. The problem is that someone else's experience tells you very little about your own. No two bodies respond to treatment in exactly the same way. Genetics, metabolism, medical conditions, medications, age, sex, sleep, stress, and many other factors all influence how much weight someone loses and how quickly they lose it. Comparing your results with someone

else's is like comparing two people climbing different mountains and wondering why they aren't reaching the top at the same speed. The comparison may feel meaningful, but it rarely provides information that actually helps you.

The same is true when people compare medication doses. It's easy to assume that needing a lower dose means you're doing better, while needing a higher dose means you've somehow failed. But medication doses aren't a competition. They're simply treatments used to help different people manage the same chronic disease. The dose that works well for one person may not be enough for someone else, just as another person may need a higher dose to achieve a similar effect. Neither is better or worse; they're simply different.

Social media often makes these comparisons even harder to avoid. Most people don't post average results. They post extraordinary ones. The stories that attract the most attention are usually the most dramatic. Over time, this can quietly distort your expectations. Exceptional results begin to seem normal, while perfectly normal results start to feel disappointing. But your treatment isn't meant to look like anyone else's. The only comparison that truly matters is whether your health is moving in the right direction compared with where you started.

SIDEBAR

Sarah and the Woman on the Internet

It had been eighteen months since Sarah began treatment, and she had lost nearly forty pounds – an 18 percent reduction in her body weight.

Her blood pressure had improved. She was sleeping better. She could walk farther without becoming short of breath. Her

knees hurt less. Her blood tests looked better than they had in years. By every medical measure, her treatment was working.

Yet when she came to her follow-up appointment, she looked discouraged.

When I asked what was wrong, she pulled out her phone.

She had joined an online support group for people taking the same medication. Earlier that week, someone had shared a photo celebrating a seventy-five-pound weight loss. Hundreds of comments followed. People congratulated her. Others shared stories of losing sixty, eighty, even one hundred pounds.

Sarah looked at the screen, then back at me.

"I've *only* lost forty."

Only.

It was remarkable how quickly that word appeared.

Just eighteen months earlier, losing forty pounds would have felt impossible. Now it somehow felt like it wasn't enough.

What had changed?

Not her health. Not her progress. Not her treatment.

Only what she was comparing it to.

The woman in the photo may have started at a different weight. She may have had different genes, different medical conditions, different medications, different eating habits, different stress levels, different sleep, and a completely different life.

But none of that mattered to Sarah in that moment. All she could see was that someone else's number was bigger than hers.

What Sarah experienced is something many people go through during obesity treatment. The moment we start

measuring our progress against someone else's, our own progress can suddenly feel smaller than it really is.

Comparisons are harmful because they can erase evidence.

The improvements we can see, feel, and measure in our own lives become harder to appreciate because we're focused on someone else's results.

The irony is that if Sarah had never seen that post, she probably would have walked into the appointment feeling proud of how far she'd come.

The only thing that had changed was her point of comparison.

That is the comparison trap.

How Comparison Steals Confidence

The comparison trap has real consequences.

The more we compare ourselves to other people, the easier it becomes to turn those comparisons inward. Confidence starts to fade. Instead of noticing how far we've come, we focus on how far we think we still have to go.

Over time, comparison stops being a harmless habit and starts affecting our mental health. When we repeatedly compare ourselves to others, it's easy to start believing that our weight says something about our value as a person. This is called internalized weight bias – when the negative messages society sends about body weight become the negative messages we tell ourselves. Research has linked internalized weight bias to higher levels of stress, anxiety, depression, and a lower quality of life.

Now, imagine if people with diabetes approached treatment this way. Imagine comparing blood sugar readings, medication doses, or cholesterol levels and deciding whether treatment

was working based on someone else's numbers. Most of us would immediately recognize how unhelpful that would be.

Yet this is exactly what many people experience during obesity treatment.

It's easy to feel like obesity treatment is a competition, but it isn't. If obesity treatment sometimes feels like the Weight Loss Olympics, you're not imagining it. For years, television shows like *The Biggest Loser* celebrated weight loss as a competition, teaching many of us that bigger losses meant greater success. But in reality, there are no medals for reaching a particular weight first or prizes for needing less medication. The purpose of treatment isn't to outperform other people. It's to improve your own health. The most useful comparison in obesity treatment is between where you were before treatment and where you are today.

For my fellow physicians reading this book, it's worth considering the unintended effects of using before-and-after photos to market treatment results. While these images celebrate one person's progress, they can also send the message that appearance is the most important measure of improvement. For some patients, they provide hope and motivation. For others, they trigger discouragement, self-criticism, or the feeling that they're falling behind. Even when well intentioned, these images may unintentionally reinforce the very comparison trap we're trying to help patients escape. For patients who are struggling, that pressure can feel isolating rather than encouraging.

The comparison trap doesn't end with comparing yourself to other people. It can also appear when you compare yourself to your own lowest weight.

In both cases, confidence becomes tied to a benchmark that may have little relevance to your current health or treatment. Comparing yourself to others can create self-doubt, while comparing yourself to your lowest weight can fuel fears of weight

regain and make every fluctuation feel like a sign that you're losing progress. Together, these comparisons can undermine confidence and become two of the most common psychological challenges in obesity treatment.



CHAPTER

THE WEIGHT STEWARDSHIP MINDSET

SOMETIMES ONE OF THE hardest parts of living with obesity isn't losing weight; it's wondering whether it will come back. For many people, that fear never completely disappears. Even after months or years of stable weight on treatment, there can still be a quiet worry in the background: *What if this stops working? What if I gain it all back?*

At the same time, it's important not to swing to the opposite extreme. Living in constant fear of weight regain isn't healthy, but neither is becoming complacent. This is where one of the biggest misunderstandings about obesity begins. Today's treatments – including GLP-1 medications and bariatric surgery – can be remarkably effective. They can help you lose weight, improve your health, and make it easier to keep that weight off. But they are not cures. Obesity remains a chronic disease that requires ongoing management, much like asthma, high blood pressure, or diabetes.

While this reality can sound discouraging at first, it doesn't have to be. In fact, accepting it is one of the most freeing mindset shifts you can make. This is where I'd like to introduce the idea of a *weight stewardship mindset*.

A weight stewardship mindset is about finding the middle ground between fear and complacency. It means recognizing that obesity is a chronic disease that deserves ongoing care without letting it take over your life. You don't need to worry about your weight every day, but you also shouldn't assume it will always take care of itself.

A *steward* is someone who looks after something valuable over time. They don't expect perfection. They don't panic every time something changes. They pay attention, adapt as life changes, and continue caring for what has been entrusted to them. A weight stewardship mindset means treating your body the same way. The goal isn't to reach a finish line where you never have to think about your weight again. It's to learn how to care for your weight through every season of life.

Even after your weight has stabilized on treatment, your body's fuel gauge settles into a new balance, with the needle still hovering above empty. That's why continuing to focus on the basics of nutrition, physical activity, and sleep remains important. They help support that new balance and make it easier to maintain your weight over time. It's also important to remember that current treatments can't erase your genetic tendency toward weight gain or make you immune to future stressors that promote weight gain.

Life continues while you're on treatment. For some of us, it brings new stressors that signal the body to store more energy. We might need a medication such as prednisone (a steroid used to treat inflammation) that promotes weight gain. We may become injured and no longer be able to exercise the way we used to. Our family might experience financial stress, making healthy food harder to afford. Stress, poor sleep, hormonal changes, and many of the other factors we discussed in Chapter 3 can all push your brain's fuel gauge closer to "empty" while continuing to overfill the fuel tank.

If this happens, it is not your fault. It also doesn't mean your treatment has stopped working. It simply means your body is responding to a new situation. That's exactly why a weight stewardship mindset matters. Just as you would adjust your plans if the weather suddenly changed, you can adjust your treatment plan when life changes too. Stewardship isn't about expecting your weight to stay the same forever. It's about learning how to care for your body through whatever life brings.

Before we talk about weight regain, we need to be clear about what it actually means. One of the biggest mistakes people make is assuming that every small change on the scale is a sign they're gaining weight back. It isn't. Your weight naturally moves up and down from day to day, even when your treatment is working exactly as it should. Learning to tell the difference between normal weight stabilization and true weight regain is one of the most important skills in developing a weight stewardship mindset.

The Difference Between Regain and Stabilization

Some weight regain should not automatically be seen as a sign that you're failing or that your treatment has stopped working. More often, it reflects the reality of living with a chronic disease in an environment that constantly challenges weight regulation. In fact, for many people, regaining up to about 5 percent of the lowest weight they reached is a normal part of long-term weight stabilization.

Why does this happen? As you lose weight, your brain doesn't know you're trying to improve your health. It simply detects that your body has less stored energy than before. In response, it gradually activates biological adaptations that slow further weight loss. From your brain's perspective, it's trying to protect you. As a result, many people regain a small amount of weight before settling into a new, stable balance. Instead of continuing to lose weight indefinitely, the body often stabilizes

at a weight slightly above its lowest point – commonly within about 5 percent of that lowest weight. This is often a sign that your body has reached a new equilibrium, not that your treatment has failed.

We become more attentive when weight continues to rise beyond that range. Regaining more than about 5 percent of your lowest weight – especially if it approaches 10 percent or more – is a signal to pause and take a closer look. It doesn't mean your treatment has failed. It simply means something may have changed. With a weight stewardship mindset, this is your cue to reconnect with your healthcare provider so you can work together to understand why and decide whether your treatment plan needs to be adjusted.

The Most Common Cause of Regain

One of the most common reasons people regain weight is simply because treatment is stopped. There are many reasons this happens. Insurance coverage may change. The cost may become too high. Side effects may make treatment difficult. Life circumstances can change. Sometimes people stop because they feel the medication has "stopped working," or because they believe they've reached a point where they no longer need it.

Whatever the reason, this isn't about blame. If you're thinking about stopping your medication, it's worth discussing it with your healthcare provider first. Together, you can explore why you want to stop, talk about what to expect, and decide whether another plan might better support your long-term health.

There is one more reason people stop treatment: Their healthcare provider advised them to. Sometimes this is absolutely the right decision. For example, if you're planning on becoming pregnant, current obesity medications are not recommended because we don't yet have enough evidence to

know if they are safe during pregnancy. There may also be other medical reasons why stopping treatment is the best choice for your health.

However, if you've been told to stop your medication simply because you've reached a "goal weight" or BMI target, I encourage you to ask more questions – and consider getting a second opinion from a healthcare provider with expertise in obesity medicine. Current scientific evidence and obesity treatment guidelines recommend that obesity medications be viewed as long-term treatments for a chronic disease, not short-term medications that are routinely stopped once a certain weight is reached. Reaching a healthier weight is wonderful, but it doesn't mean the disease has disappeared. If your treatment is being stopped without a clear medical reason, it's reasonable to ask how your obesity will be managed once the medication is gone. Current evidence consistently shows that most people regain weight after treatment is discontinued.

This is where the science is remarkably clear. The strongest evidence we have consistently shows that when obesity treatments are stopped, weight regain is common. This has been demonstrated repeatedly in large clinical trials, including STEP-4, SURMOUNT-4, and ATTAIN-MAINTAIN. Although the amount of weight regained varies from person to person, the overall message is remarkably consistent: Stopping treatment allows the biology of obesity to become active again. So far, no study has shown that people, on average, maintain all of their weight loss after treatment is discontinued.

When you think about obesity as a chronic disease, this makes sense. The treatment was helping manage the disease; it wasn't curing it. When treatment is removed, the biological processes that defend a higher body weight become active again, and the tendency to regain weight gradually returns.

SIDEBAR

Sarah and the Seven Pounds

Sarah had now lost just over forty-five pounds, or 20.5 percent of her body weight.

For the first time in years, she felt as though she had some control over her weight. Her energy was better. Her blood pressure had improved. She was sleeping more comfortably. Most importantly, she no longer felt trapped in the endless cycle of losing and regaining the same weight.

Then one morning, she stepped on the scale.

She was up three pounds.

A week later, it was up a total of five pounds.

Soon it was seven.

The number itself wasn't what upset her. It was what the number seemed to mean.

Almost immediately, the old thoughts returned.

It's happening again.

I knew this wouldn't last.

I'm ending up right back where I started.

By the time she came to see me, Sarah wasn't worried about seven pounds. She was worried about the next thirty-eight pounds because she was convinced she was heading right back to where she'd started.

What Sarah feared most wasn't seven pounds. It was what she believed those seven pounds meant. The scale had become an early warning system for failure instead of a source of information. But when we looked more closely, a very different picture emerged.

Over the previous several months, Sarah's work schedule had changed. She was sleeping less. Stress had increased. She

was eating more takeout because she had less time to prepare meals. Her treatment hadn't stopped working. Life had changed.

But even a seven-pound increase wasn't a verdict. It was simply information. The scale was doing exactly what it was supposed to do: alerting us that something had changed and that it was time to take a closer look.

We adjusted a few things. We reviewed her treatment plan. We addressed the factors that had changed. Most importantly, we stopped treating the scale as a judgment of her worth and started treating it as clinical information.

Sarah later told me the most important lesson she learned was how to respond when her weight increased. Weight stewardship taught her that regain isn't a verdict; it's information. And information helps us decide what to do next.

Weight Regain Is Information

If you're still taking your medication as prescribed and notice your weight starting to increase, don't assume your treatment has failed. The first step is to talk with your healthcare provider. Weight regain while you're continuing treatment deserves a thoughtful assessment because many different factors can influence body weight over time. The goal isn't to blame yourself. It's to understand what has changed.

It's also important to remember that your weight naturally goes up and down from day to day. A meal that's higher in carbohydrates or salt, changes in hydration, your menstrual cycle, constipation, travel, or even a hard workout can temporarily change the number on the scale. These short-term changes usually reflect shifts in water and glycogen (the form of carbohydrate your body stores for energy), not changes in body fat. Body fat is your body's long-term energy store. It takes time to gain and lose, so it doesn't appear or disappear overnight.

That's why small changes on the scale are usually nothing to worry about. Looking at long-term trends is much more helpful than reacting to every weigh-in. As we discussed earlier, regaining up to about 5 percent of the lowest weight you've reached is often part of normal long-term weight stabilization. Your body is simply settling into a new balance.

When weight continues to increase beyond that range, however, it's worth taking a closer look. Regaining more than about 5 percent from your lowest weight while you're still on treatment may be a sign that something has changed and that your treatment plan should be reassessed.

True weight regain is information. Maybe your sleep has worsened, stress has increased, you've started a new medication, an untreated medical condition has progressed, or your eating and physical activity patterns have changed. Sometimes there is more than one reason. And sometimes the increase on the scale isn't body fat at all. Conditions that cause your body to retain extra fluid—such as heart failure, kidney disease, liver disease, or lymphedema (swelling caused by fluid buildup when the body's lymphatic system can't drain properly) - can also increase the number on the scale.

SIDEBAR

Is It Muscle?

Although lifting weights can build muscle, the amount is usually modest and is unlikely to explain large changes on the scale. During weight loss, resistance training is more important for preserving the muscle you already have.

When you see weight regain this way, it becomes easier to respond instead of react. Rather than asking "What did I do wrong?" ask "What has changed, and what do we need to adjust next?" That's what a weight stewardship mindset looks like. It

means using changes in your weight to guide your next decision.

Weight regain and weight stabilization raise different questions. When weight is increasing, the question is "What has changed?" When weight has stabilized, the question becomes "Is my current treatment providing enough support?"

Sometimes, the problem isn't weight regain at all. It's that your weight has stabilized, and it feels like progress has stopped. This can feel just as frustrating as weight regain, but it calls for a different response. Just as some people with high blood pressure eventually need a higher dose or an additional medication to keep their blood pressure controlled, some people living with obesity need their treatment plan adjusted over time. That might mean increasing the dose of a medication, combining therapies, or, for some people, considering bariatric surgery. These changes don't mean you've failed. They simply reflect that chronic diseases sometimes require treatment to be adjusted as your needs change.

Matching Treatment to the Disease

For some people, a relatively low dose of medication is enough to keep their weight stable. For others, managing obesity over time may mean increasing the dose, combining medications, or considering bariatric surgery. In some cases, bariatric surgery may even be the most appropriate first treatment. None of these options mean you've failed. They simply reflect that chronic diseases often require different levels of treatment for different people and at different stages of life. Just as someone with high blood pressure may eventually need a higher dose or a second medication, obesity sometimes requires a more intensive treatment plan as the disease changes. Bariatric surgery isn't a failure of medication; it's simply another effective treatment that may be the right choice for some people.

In this situation, the question becomes "Is my current treatment providing enough support to help me reach my best weight?" If the answer is no, you and your healthcare provider can work together to determine how best to optimize your treatment. That might mean increasing the dose of your medication, adding another therapy, or considering bariatric surgery. Like many chronic diseases, obesity often requires treatment to be adjusted over time to match your body's needs.

This is also part of the weight stewardship mindset. The goal isn't to stay on the same treatment forever. It's to use the treatment – or combination of treatments – that best supports your health at each stage of your disease and your life.



CHAPTER

WHAT IS THE GOAL OF WEIGHT LOSS?

ONE OF THE BIGGEST misunderstandings about obesity treatment is believing that the goal is to reach a certain number on the scale.

It isn't.

Your weight is simply a measure of the force of gravity pulling your body toward the centre of Earth. It doesn't measure your health, your character, your effort, or your worth. It should never define who you are, let alone the goal of medical treatment. To that end, the goal of obesity treatment isn't to reach a "goal weight," a BMI below 25, or a perfect body composition. Those numbers can sometimes be useful tools, but they are not the destination. The real goal is better health.

That's why obesity specialists don't obsess over a single number on the scale. Instead, we focus on how much weight has been lost because decades of research have shown that different percentages of weight loss produce meaningful improvements in health. We know that many obesity-related complications begin to improve long before someone reaches a "normal" BMI.

Obesity Specialists Measure Progress Differently

The purpose of treatment is to reduce the health problems caused by excess body fat. Some of these problems are mechanical. Carrying extra weight places more stress on your joints, makes movement more difficult, contributes to sleep apnea, and can make everyday activities harder. Others are metabolic. Excess visceral fat – the fat stored around your internal organs – increases the risk of fatty liver disease, type 2 diabetes, high blood pressure, and abnormal cholesterol levels.

The encouraging news is that your body begins benefiting much earlier than most people realise. Weight loss doesn't reduce every type of body fat equally. Visceral fat is often one of the first to respond. For every 5 percent reduction in body weight, visceral fat decreases by roughly 9 percent. As weight loss continues, the benefits keep adding up. Losing about 15–16 percent of your body weight is associated with roughly a 30 percent reduction in visceral fat, along with substantial improvements in liver fat.

This is why relatively modest amounts of weight loss can have such a big impact on health. Losing 5–10 percent of your body weight often improves blood pressure, cholesterol, sleep apnea, inflammation, and joint pain. For people at risk of type 2 diabetes, losing about 10 percent of body weight can dramatically reduce the chance of developing diabetes. For some people who already have type 2 diabetes, losing around 15 percent of body weight can even lead to remission – meaning blood sugar returns to a health range without needing diabetes medication. Our knees especially benefit. Every pound of weight lost reduces the load across the knee joint by approximately four pounds with each step. Small changes on the scale can translate into meaningful improvements in daily function.

Notice what isn't on that list.

Nowhere does the research say you need to reach a certain number on the scale, achieve a BMI below 25, or reach a particular body-fat percentage before your health starts improving. Your body doesn't wait until you've crossed an imaginary finish line. It starts becoming healthier much sooner.

That's why obesity specialists focus on improving health – not pounds lost, BMI, or body composition. Instead, we monitor progress using evidence-based targets based on the percentage of body weight lost from where you started, a measurement called percent body weight loss (%BWL). It is calculated simply as:

% Body Weight Loss (% BWL) =
(Weight Lost ÷ Starting Weight) × 100

Percentages Matter More Than Pounds

Obesity specialists use percent body weight loss because research shows that different amounts of weight loss are associated with different health benefits. These are not rigid treatment targets, but evidence-based ranges that help estimate when a particular health condition may begin to improve – and when additional weight loss may provide further benefit.

Earlier in Chapter 11, we discussed how the only comparison that truly matters is whether your health is improving compared with where you started. That's why percent body weight loss is the best way to set initial treatment targets. The table below (Table 13.1) summarizes the ranges of weight loss that research has associated with meaningful improvements in many obesity-related health conditions.

Disease or Health Outcome†	Approximate Body Weight Loss Associated with Benefit (%)	What the Evidence Suggests
Elevated blood pressure / hypertension	5–10	Blood pressure often begins improving with about **5% weight loss**, with greater improvements as weight loss increases.
High triglycerides and other adverse lipid measures	5–10	Triglycerides often improve with about **5% weight loss**. Greater weight loss may provide additional benefit, although LDL cholesterol often requires separate treatment.
Prediabetes and prevention of type 2 diabetes	5–10	Losing **5–10%** of body weight can substantially reduce the risk of developing type 2 diabetes, with greater benefit as weight loss increases.
Type 2 diabetes – better glucose control	5–10	Blood sugar often improves with about **5% weight loss**, while greater losses usually lead to better glucose control and lower medication requirements.
Type 2 diabetes – remission	10–15 or more, with the probability rising as weight loss increases	Larger, sustained weight losses make remission increasingly likely, particularly when diabetes has been present for a shorter time. There is no single weight-loss threshold that guarantees remission.

† A few of the clinical terms in this table are explained in more detail elsewhere in the book. As a quick reference: MASH is fatty liver disease that has progressed to cause liver inflammation; fibrosis means scarring of the liver tissues; ejection fraction is a measurement of how well the heart pumps blood with each beat; LDL cholesterol is often called “bad cholesterol”; and triglycerides are a type of fat that travels in your blood.

Disease or Health Outcome[†]	Approximate Body Weight Loss Associated with Benefit (%)	What the Evidence Suggests
Metabolic dysfunction-associated steatotic liver disease – liver fat	3–5	Even relatively small amounts of weight loss can reduce liver fat. Larger losses are usually needed to improve liver inflammation or scarring.
MASH – liver inflammation	7–10	About **7–10%** weight loss increases the chance of resolving liver inflammation, although medications may also provide benefits beyond weight loss alone.
MASH with fibrosis	10 or more	Weight loss of **10% or more** offers the greatest chance of improving liver scarring, although not everyone responds the same way.
Obstructive sleep apnea	7–11 for meaningful improvement; often 15–20 or more for larger reductions	Weight loss often improves sleep apnea, with larger losses generally leading to greater reductions in disease severity. Some people will still need CPAP because body weight is only one contributor to sleep apnea.
Knee osteoarthritis pain and physical function	5–10, with greater benefit around 10–20	Even modest weight loss can improve knee pain, while larger losses generally provide greater improvements in pain, mobility, and physical function.
Mobility and physical function	5–10	Walking ability, mobility, and everyday function often improve within this range, particularly when combined with appropriate physical activity.
Cardiovascular risk factors	5–10	Blood pressure, blood sugar, triglycerides, and inflammation often begin improving with about **5% weight loss**, with additional benefits as weight loss increases.

Disease or Health Outcome†	Approximate Body Weight Loss Associated with Benefit (%)	What the Evidence Suggests
Established cardiovascular disease – reduction in major events	No validated weight-loss threshold	Certain obesity medications reduce cardiovascular events, but no specific percentage of weight loss has been shown to produce the same benefit regardless of how it is achieved.
Heart failure with preserved ejection fraction associated with obesity	No single validated threshold; benefits observed with losses around 10–20 in modern trials	Modern obesity treatments can substantially improve symptoms, physical function, and quality of life, but no single percentage of weight loss guarantees these benefits.

Table 13.1. Approximate percentages of body weight loss associated with improvements in common obesity-related health conditions. These ranges are based on current scientific evidence and are intended to guide treatment discussions, not define success or failure. Individual responses vary, and more weight loss is not always necessary or beneficial.*

Percent body weight loss is more than a way of measuring progress. It also helps guide medical decisions. For example, after about 10 percent weight loss, healthcare providers often reassess whether blood pressure medications can be safely reduced. This is one reason obesity specialists use percent body weight loss to monitor treatment.

Another advantage of tracking percent body weight loss is that it reminds us when other aspects of your medical care may

* These percentages represent approximate ranges associated with health improvements in clinical studies. Individual responses vary depending on the condition being treated, underlying biology, and the treatment used. More weight loss is not always necessary or appropriate. Treatment goals should always be individualized in discussion with your healthcare provider.

need to be reviewed. For example, after you've lost about 10 percent of your body weight, it's often a good time to reassess medications that may be affected by weight loss, such as thyroid replacement therapy (medication that replaces the thyroid hormone your body isn't making enough of), to make sure the dose still matches your body's needs.

Percent body weight loss also helps us understand how you're responding to treatment. Every obesity treatment – from medications to bariatric surgery – has an average amount of weight loss that people typically achieve. Although individual responses vary, comparing your progress with these expected averages helps determine whether your current treatment is providing enough benefit. If you're responding as expected, continuing your current treatment may be the best option. If your response is substantially lower than expected despite taking the treatment as prescribed, your healthcare provider may recommend optimizing your treatment by adjusting the dose, adding another therapy, or considering a more effective option. Once again, the goal isn't to chase a particular number on the scale. It's to make sure you're receiving the treatment that gives you the best opportunity to improve your health.

Percent Weight Loss Isn't the Whole Story

Percent body weight loss is useful. It's simple to calculate, and it helps guide treatment decisions. But it has a real limit: It can't capture everything that matters about your health.

Here's where the 5 percent and 10 percent numbers you've probably heard so often actually came from. They trace back to a small number of well-resourced studies from the 1990s and early 2000s, results that have been cited again and again in the decades since. That doesn't make them wrong, but it does mean the evidence behind these familiar targets is narrower than most people realize.

It also means the targets don't always predict what happens for real people. A recent review looked at thirty studies that set a specific percent-weight-loss target as their goal. The share of participants who actually reached that target ranged enormously – from under 6 percent of people in one nutrition-only study, up to 85 percent in a medication study. The same target that's "realistic" in one study can be almost unreachable in another, depending on the treatment, the population being studied, and how the study was run.

That review found something else worth knowing: Health sometimes improves even when someone doesn't hit their percent-weight-loss target, for reasons that have little to do with weight loss itself. In one study, participants' liver health and quality of life both improved even though many never reached the weight-loss goal the study had set.

None of this means percent body weight loss is useless. It's still one of the most practical tools we have. But the researchers behind that review reached the same conclusion many of us in obesity medicine have been reaching on our own: success should be measured by more than one number. It should include physical function, mental health, quality of life, fewer obesity-related complications, and – most importantly – what actually matters most to the person receiving care.

Percent weight loss can be part of the picture. It shouldn't be the whole picture.

SIDEBAR

Sarah and the Missing Twenty Pounds

Sarah was disappointed at her last follow-up appointment. She told me she had expected to lose more weight by now. She had been taking her medication consistently, yet after several months, she had only lost about forty-five pounds, or 20 percent of her body weight.

Only.

As we talked, I noticed something interesting.

Sarah was focusing entirely on the weight she hadn't lost.

She barely mentioned everything that had changed.

Her blood pressure had improved enough that we were discussing reducing one of her medications. Her liver enzymes had normalized. Her blood sugar had fallen out of the prediabetes range. She was sleeping better. Her knee pain had improved enough that she had recently gone on a family outing she would have avoided a year earlier.

Objectively, her health had improved in almost every way.

Yet Sarah still felt discouraged.

Finally, I asked her a simple question. "If you had lost no weight at all, but all of these health improvements still happened, would you feel the treatment had been worthwhile?"

She paused. Then she laughed. "Of course I would."

That was a turning point.

Like many people, Sarah had been taught that the purpose of treatment was to reach a certain number on the scale. She had never stopped to ask why that number mattered in the first place. The irony was that she had already achieved much of what she wanted. The scale simply hadn't caught up to the picture she had imagined in her mind.

The goal of treatment was never to lose the next twenty pounds. It was to improve Sarah's health. By that measure, her treatment was already working.

When More Weight Loss Will Help

If losing 5–15 percent of your body weight can improve your health, you might be wondering *What if I have much more weight to lose?*

The 5–15 percent range isn't meant to be everyone's final destination. Think of it as the point where health benefits often begin. It's the first milestone, not necessarily the last.

For many people, losing 5–15 percent of their body weight can improve blood sugar, blood pressure, cholesterol, fatty liver disease, sleep apnea, joint pain, fertility, and quality of life. Those are meaningful improvements that can happen long before someone reaches a particular clothing size or BMI.

But obesity isn't the same for everyone. Some people have modest excess weight with only a few health complications. Others have severe obesity with diabetes, advanced fatty liver disease, osteoarthritis (the most common type of arthritis, caused by the cushioning cartilage in a joint wearing down over time), heart disease, or sleep apnea. The amount of weight loss needed to improve these conditions varies from person to person and may be substantially greater.

Fortunately, decades of research can help guide us, much of it from bariatric surgery. Bariatric surgery remains the most effective treatment we have for obesity because it produces the greatest average long-term weight loss. Researchers have consistently found that the more weight people lose after surgery, the more likely they are to experience remission or meaningful improvement in obesity-related diseases.

For example, greater weight loss after bariatric surgery is associated with higher rates of type 2 diabetes remission. Similar patterns have been observed for high blood pressure, obstructive sleep apnea, fatty liver disease, and improvements in physical function and quality of life. Although no treatment guarantees that a disease will disappear, the overall pattern is

remarkably consistent: Greater weight loss often leads to greater health benefits.

Every treatment has limits, risks, and trade-offs. The goal is not to chase the biggest number on the scale. The goal is to achieve enough weight loss to meaningfully improve your health and quality of life while using the safest and most appropriate treatment for your situation.

This is why weight-loss goals should always be individualized and developed in partnership with your healthcare provider. Together, you'll consider questions such as:

- Which obesity-related conditions do you have?
- How severe are they?
- How much weight loss is likely to improve them?
- Which treatment is most likely to help you safely achieve that level of weight loss?

For some people, a 10 percent weight loss may be enough to accomplish their goals. Others may benefit from 20 percent, 25 percent, or even more. While current obesity medications have transformed treatment, they also have limits. On average, today's medications produce about 10–25 percent weight loss, depending on the medication, the dose, and the individual. Some people lose considerably more, while others lose less. Like everything else in medicine, these are averages, not guarantees.

When someone needs substantially greater weight loss – often more than 35 percent of their starting body weight – to adequately treat their obesity or its complications, medications alone may not be enough. In these situations, bariatric surgery remains the most effective treatment available and deserves to be part of the conversation. Modern bariatric surgery produces average weight losses of approximately 25–35 percent or more, depending on the procedure, while also offering the highest rates of remission for many obesity-related diseases.

But that doesn't mean surgery is right for everyone, and not everyone is comfortable with the idea of surgery. That's okay. Just as we match the right medication to the right patient in other areas of medicine, we aim to match the right obesity treatment to the amount of weight loss needed to improve health.

But it's important to remember that more weight loss isn't always better.

When Does More Weight Loss Stop Helping?

At some point, the benefits of losing more body fat become smaller, while the risks of continued weight loss may begin to grow. The challenge is that medicine doesn't yet have a single test that tells us exactly when that point has been reached. We have many tools to help determine who is likely to benefit from weight loss. We have far fewer tools to tell us when it's time to stop pursuing more.

BMI Can Offer a Lower Boundary

The simplest tool is body mass index (BMI). Although BMI is far from perfect, it can provide a useful warning that someone may be becoming too lean.

A BMI below 18.5 is the World Health Organization's definition of underweight. Below this threshold, the risks of osteoporosis (a condition where bones become thin and fragile and break more easily), fractures, malnutrition, impaired immunity, fertility problems, and early death increase. Weight-loss treatment should never allow someone to reach – or fall below – this level. In fact, waiting until someone reaches a BMI below 18.5 would be far too late to begin asking whether continued weight loss is still improving their health.

For some people, the lower boundary may be even higher. Canadian osteoporosis guidelines identify a BMI below 20 as a risk factor for osteoporosis and fragility fractures (broken

bones that happen from a fall or minor stress that wouldn't normally cause a break) in adults over fifty. For adults over sixty-five, several guidelines suggest that the healthiest BMI may be higher still – often between 22 and 27 – because lower BMIs are associated with greater frailty and a higher risk of death.

Taken together, these findings suggest that BMI is best viewed as a lower safety boundary rather than a treatment target. Instead of asking "How low can my BMI go?" a better question is "Am I approaching a BMI where further weight loss may begin to cause more harm than good?"

Of course, very few people receiving obesity treatment ever approach these lower BMI thresholds. BMI simply tells us when further weight loss is clearly becoming unsafe. The more difficult question is whether continued weight loss is still providing meaningful health benefits long before those thresholds are reached.

Body-Fat Percentage Does Not Give Us a Clear Stopping Point

Body-fat percentage may seem like the obvious answer. In theory, it should tell us when someone has lost enough fat and should stop losing weight. Unfortunately, the evidence isn't that clear.

There are no validated body-fat cutoffs that tell us:

- "Below this percentage, further weight loss begins to harm health."
- "Above this percentage, more weight loss is likely to improve health."
- "Achieving this percentage is associated with diabetes remission."

This remains true whether body fat is measured by DEXA, bioelectrical impedance analysis, or a Bod Pod. Most body-fat

thresholds are based on population averages, statistical definitions, or expert opinion. They were not developed by following people over time to identify the point at which further fat loss begins to increase the risk of fractures, weakness, disability, or early death. Body-fat percentage may provide useful information, but it cannot yet tell us when weight loss should stop.

Strength Matters More Than Lean Mass

I believe physical function will become one of the most useful tools for deciding when further weight loss is no longer improving health, especially as newer and more effective obesity treatments become available. During weight loss, some lean tissue is usually lost along with fat. That's why body composition scans can create alarm when they show a decline in lean mass. But the more important question isn't how much lean mass you've lost. It's whether you've become weaker or less able to function.

Healthcare providers can assess physical function using simple tests such as grip strength, walking speed, standing up from a chair five times, the Timed Up and Go test, or the Short Physical Performance Battery. These tests help predict the risk of falls, disability, hospitalization, and early death. In many cases, they tell us more about a person's health than the amount of lean tissue shown on a DEXA scan.

Studies of GLP-1 medications have generally found that people lose some lean mass while their physical function remains stable. This reminds us that muscle mass and muscle function are not the same thing. Someone who has lost lean mass but remains strong, mobile, and active is very different from someone who is becoming weaker, slower, or less steady.

Frailty Is the Most Important Warning Sign

Frailty is especially important in older adults, but it doesn't simply mean being thin. It describes a loss of physical reserve.

A frail person may have less strength, less energy, slower movement, and more difficulty recovering from illness or injury. Healthcare providers can assess frailty using tools such as the Clinical Frailty Scale, the Frailty Index, or the Fried Frailty Phenotype.

Frailty predicts hospitalization, loss of independence, the need for long-term care, and even early death more accurately than BMI alone. As people begin obesity treatment earlier in life and remain on treatment for decades, I believe assessing frailty will become an increasingly important part of obesity care.

Imagine someone who has successfully managed their weight with obesity treatment for twenty years and is now in their eighties. If they begin to develop frailty or are no longer eating enough to meet their nutritional needs, the goals of treatment may need to change. Instead of continuing to maintain a lower weight, it may be healthier to allow some weight regain toward the range generally recommended for older adults.

At that stage, the benefits of maintaining a lower weight must be carefully weighed against the risks of frailty, malnutrition, and loss of muscle and bone health. The priority may no longer be achieving the lowest possible weight but preserving strength, mobility, independence, and quality of life. The goal remains the same: to protect the health and function that person has worked so hard to achieve.

Waist Size May Tell Us Whether More Fat Loss Could Still Help

One common question I hear is: "Doctor, I feel so much better, but I'd still like to lose another ten pounds. Should I?"

It's a great question, and the answer isn't always simple.

For some people, losing another ten pounds may continue to improve their health. For others, it may provide very little

additional benefit and could even increase the risk of problems such as frailty or poor nutrition.

So how do we decide?

The answer may depend less on the number on the scale and more on where fat is stored. Fat around the organs, called visceral fat, is much more harmful than fat stored beneath the skin. This means that two people with the same BMI can have very different health risks – and may benefit differently from losing more weight.

This is where waist circumference and waist-to-height ratio can be helpful because, unlike BMI, they provide clues about where fat is stored. Although these measurements can't tell us exactly when to stop losing weight, they can help us judge whether additional fat loss is still likely to provide meaningful metabolic benefits.

For example, someone with a BMI of 24 and a waist-to-height ratio of 0.65 may still carry a substantial amount of abdominal fat and could continue to benefit from further fat loss. In contrast, someone with a BMI of 24 and a waist-to-height ratio of 0.40 may have much less abdominal fat remaining. For that person, the potential benefits of further weight loss may be smaller.

However, there is currently no validated lower waist measurement that tells us exactly when weight loss should stop. Waist measurements can help inform the decision, but they cannot make the decision on their own. Even when there is still room for additional health improvement, many people also hope that more weight loss will change how they look. That's where the conversation may become more complex.

When Health and Aesthetics Overlap

Many people wonder where the line is between treating obesity as a disease and treating it for appearance. The honest answer is that there often isn't a clear line.

In medicine, health and appearance frequently overlap. A breast reduction can relieve back and shoulder pain while also changing how someone looks. Treating acne reduces inflammation and helps prevent scarring, but many people also hope for clearer skin. Someone receiving Botox for migraines may notice fewer wrinkles, while someone receiving Botox for wrinkles may notice fewer headaches. These treatments improve health and appearance at the same time. Obesity treatment is no different.

Most people don't lose weight only to lower their blood pressure or improve their cholesterol. They also hope to feel more confident, wear clothes they enjoy, or like what they see in the mirror. There is nothing wrong with that. Wanting to look better doesn't make your health goals any less real.

The challenge is that health and appearance don't always improve together. You may reach a point where your blood pressure has normalized, your sleep apnea has improved, your joints hurt less, and you have more energy, yet still wish you looked different. That doesn't make you shallow. It makes you human.

At that point, it's helpful to ask a different question:

"Is more weight loss likely to give me what I'm hoping for?"

Sometimes the answer is no.

Sometimes people believe they need to lose another ten or twenty pounds when what they're really hoping for is a different body shape. But weight loss cannot change your bone structure or where your body naturally stores fat. For example, some people continue to carry more fat – or have loose skin – on the back of their upper arms, even after significant weight loss. Others are frustrated by loose skin after losing a large amount of weight. Skin is a different organ than fat. Losing more weight usually won't tighten excess skin and, in some cases, may make it even more noticeable. If excess skin is the

main concern, an assessment by a plastic surgeon may be more helpful than trying to lose additional weight.

Recognizing what weight loss can – and cannot – change helps shift the conversation away from chasing a particular appearance and toward improving health. Once you reach that point, the question is no longer simply whether you *can* lose more weight, but whether losing more weight is likely to improve your health.

As your overall health improves, your goals may naturally change, and that's okay. Instead of asking "Can I lose more weight?" a more helpful question becomes: "What weight is most likely to help me live the healthiest life possible?"

In other words, there comes a point when the question is no longer "Can I lose more weight?" but "Would losing more weight actually move me closer to the life I'm hoping to live?"

For some people, continuing to lose more weight may begin to do more harm than good. It may also do little to change their body shape if that is their main concern. For others, there may still be health benefits to losing more weight, but biology, the limits of today's treatments, or the risks of surgery may make further weight loss difficult – or simply not possible. Most people will find themselves somewhere between these two situations.

This is why we use the term *best weight*. It isn't a perfect weight or a goal weight. It's the weight where the overall benefits of treatment are greatest for you, taking into account your health, your biology, the treatments available, and your life. In the next chapter, we'll explore what *best weight* means – and why it looks different for every person.



CHAPTER

BEST WEIGHT

MANY PEOPLE STARTING OBESITY treatment are looking for something solid to hold on to – a clear destination, a predictable timeline, and reassurance about what lies ahead.

After years of feeling that their weight has been beyond their control, it's completely understandable to want some certainty. People naturally want to know where they're headed, how long it will take, and what number they should aim for. That's why the idea of a goal weight can feel so reassuring. It offers certainty in a process that often feels anything but certain.

The difficult part is that obesity treatment rarely offers that kind of certainty. No healthcare provider can tell you exactly how much weight you'll lose. No medication can promise a specific number on the scale. And no treatment can guarantee that you'll reach a predetermined goal weight. Obesity is a chronic disease, and like most chronic diseases, people respond to treatment differently.

Why Goal Weights Can Do More Harm Than Good

For as long as most of us can remember, weight-loss programs have encouraged people to choose a goal weight before treatment even begins. It may seem motivating, but in medicine it creates a problem: The goal is based on a future that no one can predict.

Unlike prescribing antibiotics for an infection, your medical team cannot predict precisely how your physiology will answer to therapy. Your biology, genetics, medications, medical conditions, age, sleep, stress, and many other factors influence how much weight you can lose. Setting one specific number assumes we already know the outcome before treatment has even begun.

When that number becomes the measure of success, people often begin judging themselves instead of the treatment. Someone who improves their diabetes, lowers their blood pressure, sleeps better, and loses 18 percent of their body weight may still feel like they've "failed" simply because they never reached the goal weight they were given.

Goal weights can also encourage people to keep chasing a lower number long after the greatest health benefits have already been achieved. They may increase medication doses despite diminishing returns, continue restrictive eating that is no longer sustainable, or become so discouraged that they abandon treatment altogether.

Perhaps the greatest harm is psychological. Receiving a goal weight from your healthcare team can turn your health into a pass-or-fail test. Until that number is reached, it can feel as though your progress doesn't count. Every pound above the goal becomes evidence that you're not there yet, even when your health and quality of life have improved dramatically.

Medicine rarely works this way in other chronic diseases. We don't give someone with high blood pressure one perfect

blood pressure target and tell them they've failed if they don't reach it. We celebrate meaningful improvement and adjust treatment based on the balance of benefits, risks, and what's realistic for that individual. Obesity deserves the same approach.

At the same time, it's natural to feel uncomfortable not knowing exactly where treatment will lead. After all, many of us have been taught that every successful weight-loss effort should end at one specific number on the scale. But what if the uncertainty isn't the real problem? What if the problem is the expectation that there's one perfect number waiting for you at the end?

One of the most important ideas in obesity treatment is that there is no universal goal weight. Instead, there is your *best weight*.

Introducing *Best Weight*

If you've spent years believing you needed to reach one specific number on the scale, letting go of that idea can feel unsettling. But obesity treatment doesn't need one perfect number. It needs a better way to measure progress.

The concept of *best weight* was introduced by Canadian obesity medicine physicians Dr. Arya Sharma and Dr. Yoni Freedhoff. It shifts the conversation away from chasing an arbitrary number on the scale and toward finding the weight that offers the greatest overall health and quality of life. In this chapter, we'll build on that idea to explore what *best weight* means in the era of modern obesity treatment.

SIDEBAR

The Number That Didn't Matter

Sarah was convinced she knew her goal weight.

She had carried that number in her mind for years. It was the weight she remembered from her twenties – the weight she associated with confidence, happiness, and feeling comfortable in her own body.

As treatment progressed, she came closer to that number than she ever thought possible. But the closer she got, the harder life became.

She found herself thinking about food more often. Social events became stressful because they disrupted her routines. Missing a workout made her anxious. Even small changes on the scale affected her mood for days. The lower her weight became, the more time and energy it took to stay there.

During one follow-up appointment, Sarah said something that stopped both of us for a moment.

"I think I liked my life better ten pounds ago."

At first, she felt guilty for saying it. Wasn't the goal always to lose more weight?

But as we talked, her comment made perfect sense.

Ten pounds earlier, her blood pressure had already improved. She had more energy. She was sleeping better. Her knee pain was manageable. She could enjoy the activities she loved. She spent less time thinking about food and less time negotiating with herself over every meal and every decision.

She hadn't reached her lowest weight, but she may have already reached her *best weight.*

That realisation changed the conversation. Instead of asking "How much more weight can I lose?" Sarah started asking a different question:

"What weight allows me to live the life I want while taking care of my health?"

The answer was finding the balance between improving her health and enjoying her life. In the end, that balance proved

far more valuable than the goal weight she had been chasing for years.

Your best weight is the weight you reach while living the healthiest lifestyle you can genuinely enjoy and sustain. It's the weight where you're taking care of your health without feeling like your life revolves around the scale. It's the weight that allows you to improve your health while still enjoying your life.

This distinction matters because lower isn't always possible, and lower isn't always better. As weight goes down, the effort required to maintain it often goes up. At some point, chasing a lower number on the scale may begin to take more from your life than it gives back. That's why your lowest weight and your best weight are not always the same. Your best weight is the place where your health, daily function, hunger, treatment burden, and quality of life are in balance. It's the weight you can realistically maintain while still living the life you want to live.

When the idea of best weight was first introduced, obesity treatment looked very different from today. Most people only had access to lifestyle changes, older weight-loss medications that produced modest results, or bariatric surgery. For many, the biggest question was whether they could lose enough weight to improve their health at all.

Today, that conversation has changed. Modern medications and bariatric surgery can help many people lose far more weight than was possible in the past. Some people lose 15 percent, 20 percent, or even 25 percent of their body weight, and newer treatments are expected to become even more effective. This is exciting progress, but it also creates a new challenge. Years ago, the question was "Can I lose enough weight to

improve my health?" Today, another question is just as important: "How do I know when enough is enough?"

This is where the idea of *best weight* becomes even more valuable.

Best weight isn't a moving finish line that keeps getting lower every time a new treatment becomes available. Instead, it helps answer a much more meaningful question: At what point are the benefits of losing more weight no longer worth the costs?

Those costs look different for everyone.

For some people, losing another ten pounds could mean their sleep apnea resolves or their knee pain improves enough to avoid surgery. But achieving that same amount of weight loss might require increasing to a higher medication dose, adding a second medication, or continuing treatment for many more years. It may also mean paying for expensive medications every month, navigating insurance approvals, managing additional side effects, or simply carrying a greater treatment burden.

Treatment can also ask more of your daily life. More appointments. More prescriptions to keep track of. More time spent thinking about medications, refills, and insurance coverage. For some people, that trade-off is well worth it because the additional weight loss leads to meaningful improvements in health or quality of life. For others, the added effort, cost, or side effects may outweigh the benefits of losing a little more weight.

There is *no* single right answer.

That's also why *best weight* is personal. It asks you to look beyond the number on the scale and consider the whole picture. Is losing more weight likely to improve your health in a meaningful way? Or would it mostly add more cost, more medication, more side effects, and more work with little additional benefit? Your best weight is the point where the overall benefits

of treatment are greatest for you – not just on the scale, but in your everyday life.

For some people, even with today's most effective medications and bariatric surgery, weight loss will eventually slow or reach a limit. That limit reflects the biology you inherited, the treatments available today, and the realities of living a full life. Best weight isn't about pushing past those limits at any cost. Nor is it about weight loss at all costs. It's about finding the point where the benefits of treatment are greatest while its burdens remain acceptable. Best weight is built around your health – and the life you want to live.



CHAPTER

OBESITY IS CHRONIC – AND THAT IS CLARITY

IF THERE IS ONE thing I hope you take away from this book, it is this: Your experience finally makes sense.

For years, many people living with obesity were given an explanation that never matched what they were actually living through. They were told that weight loss was mostly about willpower. They were told that if they just tried hard enough, they would succeed. And when the weight came back, they were often made to feel as though they had failed.

But their experience told a different story.

The problem was never that people living with obesity misunderstood their bodies. The problem was that they were given an incomplete explanation.

Today, we understand obesity very differently than we did in the past. We know that biology plays a much larger role than most people were ever taught. We know that the body actively defends its weight. We know that fullness, hormones, genetics, medications, sleep, stress, and the environment all shape body weight in ways that go far beyond willpower alone. Most importantly, we know that obesity is less about body fat itself

than it is about the brain, and that the brain can become convinced the body is running out of fuel, even when it isn't.

My hope is that this understanding leaves you with something many people living with obesity have never been given before: relief. I hope your experience finally has an explanation. When your experience makes sense, it becomes much easier to let go of blame and move forward with clarity and self-compassion.

Unfortunately, understanding your body doesn't mean everyone around you will understand it too. But understanding your own experience is only part of the story. You still live in a world where many people – including friends, family, employers, the media, and sometimes even healthcare professionals – may not yet understand what modern obesity science has taught us. That disconnect can be frustrating. It can leave you feeling as though you're finally speaking a language that much of the world hasn't learned yet.

When Science Moves Faster Than Culture

Over the past decade, our understanding of obesity has changed dramatically. We now have medications that can produce levels of weight loss once possible only with bariatric surgery. We also know much more about how fullness, energy expenditure, genetics, and the brain work together to regulate body weight.

But while science has moved forward, much of the public conversation has not.

Many people now find themselves caught between two very different explanations of obesity. One comes from modern medicine. The other comes from decades of diet culture. When those messages collide, confusion is almost inevitable.

That is why making sense of obesity begins with understanding it as a chronic disease.

A chronic disease is a condition that develops over time, lasts for years, and requires ongoing management rather than a one-time cure. Diabetes, asthma, high blood pressure, and high cholesterol all fit this definition. We recognize that biology plays a role, treatment often needs to continue long term, and the treatment plan may need to change as life changes.

Obesity deserves to be understood in the same way.

Throughout this book, I've asked you to look beyond body weight itself. Excess body fat is the part we can see, but it is not the disease. Obesity is a disorder of weight regulation. It is a condition in which the brain defends a higher amount of body fat than is healthy.

When someone loses weight, the brain doesn't celebrate the achievement. Instead, it often interprets weight loss as a threat to survival. It responds by increasing hunger, reducing feelings of fullness, slowing energy expenditure, and making food more rewarding. These are normal biological responses designed to protect you from what the brain perceives as dwindling energy stores.

That is also why obesity is chronic. Even after weight has been lost, the biological drive to regain it often remains. The symptoms improve with treatment, but the underlying tendency for the brain to defend a higher amount of body fat frequently persists. This is remarkably similar to other chronic diseases. Lowering blood pressure does not cure high blood pressure. Lowering blood sugar does not cure diabetes. In the same way, losing weight does not necessarily eliminate the biological processes that made maintaining that weight so difficult in the first place.

This framework changes the question we ask.

Instead of asking "Why can't I keep the weight off?" we begin asking "How do I help my brain regulate body weight over the long term?" Instead of chasing a finish line, we build a

treatment plan that can adapt as our lives, our health, and our needs change.

For many people, I hope this way of thinking is deeply freeing. It explains why weight regain after stopping treatment is common, why treatment often needs to continue after substantial weight loss, why plateaus happen, why people respond differently to the same treatment, and why there is no single diet that works for everyone forever.

Most importantly, it offers a practical way to live with obesity. That is the framework this book has tried to build. It isn't built on blame, perfection, or the idea that you simply need to try harder.

For many years, the message was that obesity could be left behind if you found the right diet, worked hard enough, or had enough willpower. The hope was that one day you would reach the finish line, graduate from treatment, and finally be "done."

For most people, that day never came.

A chronic disease framework offers a different kind of hope. It doesn't ask you to chase a finish line that may not exist. Instead, it offers the hope that your health can improve, obesity-related complications can be reduced, and your life can become easier to manage. It replaces the pressure to become a different person with the confidence that you can care for your body in a way that is sustainable for the long term.

That is what the future of obesity care is likely to look like. It will not be defined by a perfect diet or a single breakthrough medication. It will be built on better long-term treatments, personalized care, and improved access to those treatments.

This is what chronic disease management looks like. It means taking treatment seriously while recognizing that treatment may change over time. It means adjusting as life changes, accepting that there will be periods of weight loss and periods of stability, seeing setbacks as information rather than judg-

ment, and continuing your care instead of feeling as though you have to start over.

In the end, there is something surprisingly reassuring about all of this.

It isn't extraordinary.

It's simply how we care for chronic diseases.

When the Conversation About Obesity Finally Makes Sense

For far too many years, obesity has been surrounded by confusion. Today, we know more. Not everything. But enough to finally make sense of what so many people have been experiencing all along.

That understanding doesn't make the work of managing obesity any easier. But it does replace confusion with something far more valuable: an explanation that finally makes sense of what you've been experiencing all along. And sometimes, the most powerful thing medicine can offer isn't certainty; it's clarity.

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And to you, the reader: Thank you for picking up this book and being willing to look at obesity through a different lens. I hope what follows gives you a clearer understanding of your biology, and a more useful way forward.

APPENDIX

A Quick Checklist for Evaluating Weight-Loss Information

Health information has never been easier to access – or harder to trust. Every day, social media, podcasts, advertisements, and news headlines promote new "breakthroughs," miracle diets, and quick fixes. Some are grounded in good science. Many are not. One of the most valuable skills you can develop is learning how to recognize trustworthy information. Before you invest your time, money, or hope, ask yourself these questions:

- ☐ **Who is providing this information?**
  Is it coming from a physician or healthcare professional with recognized board certified training in obesity medicine, or from an influencer, coach, celebrity, or someone whose qualifications are unclear? Expertise matters.

- ☐ **Is someone trying to sell you something?**
  Be cautious when health advice is closely tied to supplements, detoxes, coaching programs, expensive testing, or proprietary methods. Reliable medical information should stand on its own.

- ☐ **Does it promise extraordinary results?**
  Be skeptical of claims that sound too good to be true. Sustainable obesity treatment rarely produces dramatic overnight changes.

☐ **Does it blame you?**

Messages that suggest you simply need more willpower or discipline ignore decades of scientific research. Obesity is a chronic disease, not a personal failure.

☐ **Does it reference high-quality evidence?**

Look for recommendations supported by clinical practice guidelines, systematic reviews, or well-designed clinical trials – not testimonials or isolated success stories.

☐ **Do recommendations about GLP-1 therapies pass the Asthma Test?**

The arrival of GLP-1 medications has changed obesity treatment, but it has not changed the biology of obesity. Good advice should be rooted in biology, not the latest trend.

One Final Thought

You do not need to become an obesity expert to make informed decisions about your health. You simply need to become a thoughtful consumer of health information.

When in doubt, slow down. Ask who is providing the information, what evidence supports it, and whether the message is helping you understand your body or simply trying to capture your attention. The most trustworthy voices are rarely the loudest. They acknowledge uncertainty, avoid sensational claims, and respect the complexity of obesity as a chronic disease.

TRUSTED RESOURCES

The internet has made health information more accessible than ever, but it has also made misinformation easier to spread. If you would like to continue learning about obesity after finishing this book, I encourage you to begin with the evidence-based resources below.

Each is developed or led by recognized specialists in obesity medicine and is intended to help people living with obesity better understand their disease and its treatment.

While no single resource has all the answers, these are excellent places to continue learning and to help you distinguish science from marketing.

Obesity medicine continues to evolve as new research emerges. The most trustworthy sources are those that acknowledge uncertainty, update their recommendations as evidence changes, and prioritize scientific integrity over sensationalism.

Websites

Obesity Canada
Website: https://obesitycanada.ca

Blogs

Dr. Sue Pedersen's Blog
Website: https://drsue.ca

Podcasts

The Obesity Doctor Podcast

Docs Who Lift

Obesity Canada Podcast: *Scale Up Your Practice*

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Photo: Anita Lee

Dr. Sandra Cortina, MD, FRCPC, MPH, DABOM, is a specialist in Internal Medicine and an American Board of Obesity Medicine–certified physician dedicated to helping people better understand the science of obesity. She is the medical director of Cortina Weight Medicine in British Columbia, where she cares for individuals living with obesity and other metabolic conditions through evidence-based, patient-centred care.

Dr. Cortina earned graduate training in public health, bringing a population health perspective to her clinical work and her approach to communicating medical evidence. Before becoming a physician, she worked as a registered nurse in addiction medicine, an experience that profoundly shaped her understanding of chronic disease, stigma, behavior change, and the importance of meeting people with empathy rather than judgment. This unique combination of experiences informs both her medical practice and her writing.

As a clinical instructor in the Division of Internal Medicine at the University of British Columbia, Dr. Cortina is passionate about translating complex medical science into language that patients, families, and healthcare professionals can understand. Her work focuses on replacing myths with evidence and helping readers make sense of the rapidly evolving science of obesity in the GLP-1 era.

Running on Empty reflects the same philosophy that guides her clinical practice: Obesity is not a failure of willpower but a chronic disease that deserves clarity, compassion, and evidence-based treatment.

Dr. Cortina lives in beautiful New Westminster, British Columbia, with her two long-coat Chihuahuas, Yoda and Zelda.

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