CASPIAN

OBESITY MEDICINE

SAMPLE CHAPTER · FREE TO SHARE

From the course textbook of the

**CASPIAN Certificate in Obesity Medicine**

MODULE 01

# The New Science of Obesity

*Why obesity is a disease, not a choice — and why this changes how you treat it*

**Dr Khizer Hussain Junaidy, MD**

Course Director · Diabetologist & Obesity Physician (SCOPE, World Obesity Federation)

Caspian Healthcare Foundation · Hyderabad

**caspianobesity.com**

MODULE 01

# The New Science of Obesity

*Why obesity is a disease, not a choice, and why this changes how you treat it*

*"No one chooses obesity. Patients try hard, and their own body pushes back. Our job is to change that fight."*

The CASPIAN teaching series

**LEARNING OBJECTIVES**

After this module, you will be able to:

1. Explain that obesity is a chronic, relapsing disease of body-weight control, not a lack of willpower.
2. Describe how the brain (hypothalamus), leptin, and gut hormones control hunger and body weight.
3. Explain why fat tissue is an active organ, and why waist size matters more than weight.
4. Describe why the body regains lost weight (hormone changes and a lower metabolic rate).
5. Say why obesity is now called a disease, and what "clinical" and "preclinical" obesity mean.
6. Use respectful, person-first words, and explain why weight stigma harms care.

**CLINICAL VIGNETTE: The patient who "tried everything"**

Imran is 38. He manages projects at an IT company in HITEC City. He weighs 96 kg and is 172 cm tall, so his BMI is 32.5. Over eight years he has lost the same 12 kg four times, and put it back each time. He tried a gym, a keto app, a well-known Hyderabad dietician, and simply eating very little. Each time the weight came back, often with extra. He comes to you feeling low, almost ashamed. He says a sentence you will hear many times in an obesity clinic: "Doctor, I know what to do. I just don't have the willpower."

Hold on to that sentence. By the end of this module you will see why Imran is not weak, and why his body beat him four times. His biology is not broken. It is doing exactly what it evolved to do. That is the problem, and it is also the key to the solution.

## 1 A Disease Hiding in Plain Sight

Look around any waiting room in Hyderabad. You are looking at the largest chronic-disease problem in the country. It is often not written at the top of the case sheet. The ICMR-INDIAB study gives the clearest picture of India's metabolic health. It found abdominal (belly) obesity in about two of every five adults, and general obesity in more than one in four. Diabetes, prediabetes and high blood pressure sit on top of this.

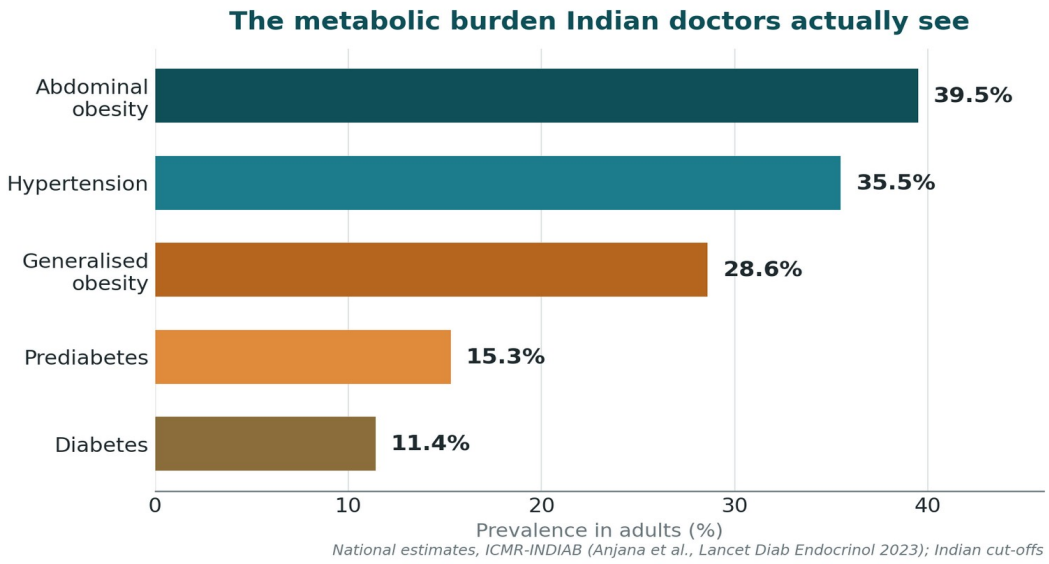


**Figure 1.1** *Obesity is the base under the diseases we already treat. National estimates, ICMR-INDIAB, 2023.*

Two points make this urgent. First, obesity is not just one more disease on the list. It is the soil in which many others grow. Type 2 diabetes, high blood pressure, fatty liver, obstructive sleep apnoea, polycystic ovary syndrome, osteoarthritis and several cancers all share extra body fat as a root cause. Treat the weight, and you help all of them at once. Second, and this matters for our patients, Asian Indians get these problems at a lower body weight than Europeans do.

**IN THE CLINIC: The "thin-fat" Indian**

For the same BMI, Asian Indians carry more body fat, and more of it deep in the belly around the organs (visceral fat), than white Europeans. So a person can look only a little overweight, or even have a "normal" BMI, and already be metabolically obese: insulin-resistant, with abnormal lipids, and heading towards diabetes. This is why every cut-off in this course is lower for our patients. It is also why waist size will matter to you as much as the weighing scale.

## 2 Why "Eat Less, Move More" Usually Fails

For a hundred years, obesity care rested on one simple idea: calories in, minus calories out, equals fat gained or lost. It sounds obvious. As a treatment plan, it mostly fails. Understanding why is the turning point of this whole course.

The idea is not wrong. Energy cannot be created or destroyed. The mistake is to think the two sides are separate. People assume you can cut food, and the body will keep burning energy at the same rate while the fat melts away. It does not. When you eat less, the body notices. It behaves like a family whose income suddenly drops: it spends less, and it tries to bring in more. It burns less energy at rest, and it makes you feel hungrier. The two sides of the equation are wired together.

**MYTH vs EVIDENCE: A calorie is just a calorie, so obesity is simple maths**

**Evidence:** The maths is true, but you do not control the numbers. The brain sets and defends a level of body fat, and it adjusts hunger and energy use to protect it. This is why two people can eat the same food and gain different amounts. Willpower acts on conscious choices. This control system is not conscious, so willpower is the wrong tool for it. Obesity is a problem of the control system, not of the arithmetic.

## 3 The Appetite Control System

At the base of the brain sits the hypothalamus. Inside it, a small area called the arcuate nucleus acts as the control centre for body weight. Think of it like a thermostat, but instead of defending a temperature, it defends a level of stored fat. It reads signals from the body and adjusts two things: how hungry you feel, and how much energy you burn at rest.

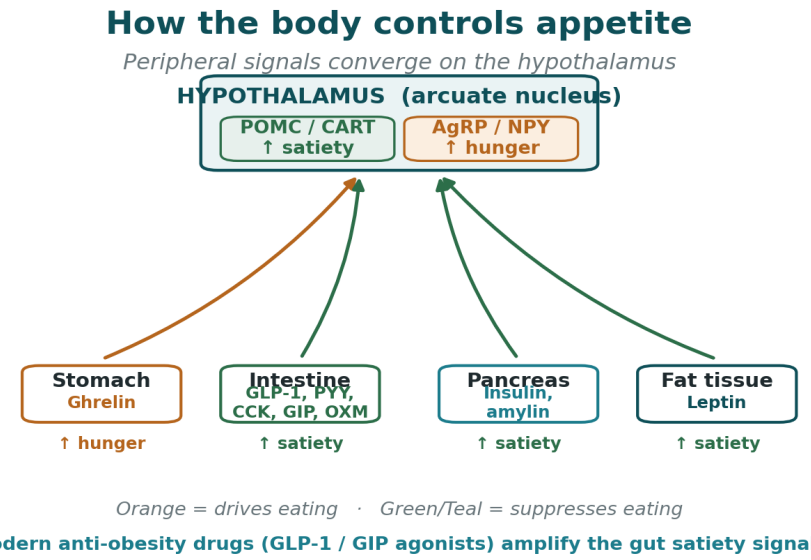


**Figure 1.2** *The appetite control system. Signals of hunger and fullness from the gut, pancreas and fat tissue meet in the hypothalamus.*

The signals come from three places. Fat tissue makes leptin, in proportion to how much fat you carry. Leptin is the long-term fuel gauge: it tells the brain how full the tank is. The gut makes fast, meal-by-meal signals. Ghrelin, made in the stomach when it is empty, is the main hormone that drives hunger. Working against it is a group of "fullness" hormones released when food arrives, including GLP-1, PYY, GIP, CCK and oxyntomodulin. The pancreas adds insulin and amylin, which also signal fullness.

When this system works well, you do not notice it. Most people hold their weight within a few kilograms for years without counting a single calorie, because the control centre quietly matches intake to energy use. The trouble starts when the defended level is set too high, by genes, by the food around us, by hormones, or by certain medicines. The same machinery that once kept weight steady now keeps it high, and defends it.

> **PRACTICE PEARL: Why this diagram is worth learning now**
>
> Every modern anti-obesity medicine works somewhere on this map. The GLP-1 drugs you will study in Module 8 do not force the appetite down. They boost the gut's own fullness signal. If you understand Figure 1.2 now, the drug chapters later will feel like common sense, not a list to memorise.

## 4 Fat Is an Active Organ, Not a Store

Many of us were taught to think of body fat as dead storage, a passive store of spare calories. That picture is wrong, and it is one reason obesity was under-rated as a disease for so long. Fat tissue is one

of the largest hormone-producing organs in the body. It talks to the brain, the liver, the muscles and the blood vessels, using hormones and inflammatory signals.

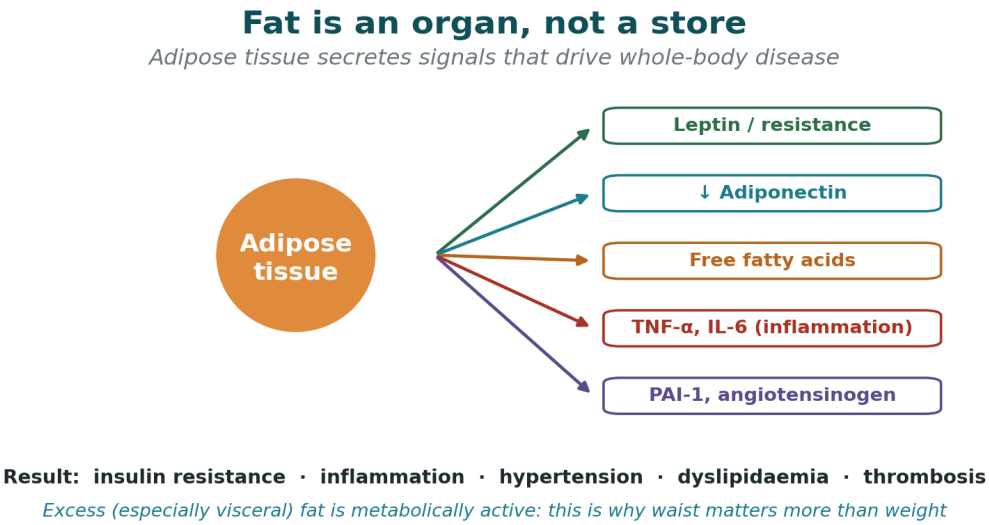


**Figure 1.3** *Fat tissue releases hormones and inflammatory signals that drive disease across the body.*

When fat cells become large and overloaded, especially the visceral fat around the belly organs, their signals turn harmful. Adiponectin, a protective hormone that improves insulin sensitivity, falls. Free fatty acids rise. Inflammatory signals such as TNF-alpha and IL-6 rise, along with factors that raise blood pressure and help clots form. The result is the familiar cluster we chase every day: insulin resistance, low-grade inflammation, abnormal lipids, high blood pressure, and a tendency to clot. This is the link between a waistline and a heart attack.

This also explains the single most important measurement choice in this course. It is visceral fat, not total weight, that is harmful. So where the fat sits matters more than what the whole body weighs. Waist size tells you about the dangerous fat directly. The scale does not. For our patients, whose visceral fat runs high, this is not a small point. It is the difference between wrongly reassuring a "normal-weight" man and catching his risk in time.

## 5 Why the Weight Comes Back

Now go back to Imran, who lost the same 12 kg four times. His story is not four failures of character. It is the most repeated finding in all of obesity research: after weight loss, the body fights hard, and for a long time, to push the weight back up. If you remember only one thing from this module, remember this. It changes how you see every "non-compliant" patient you will ever meet.

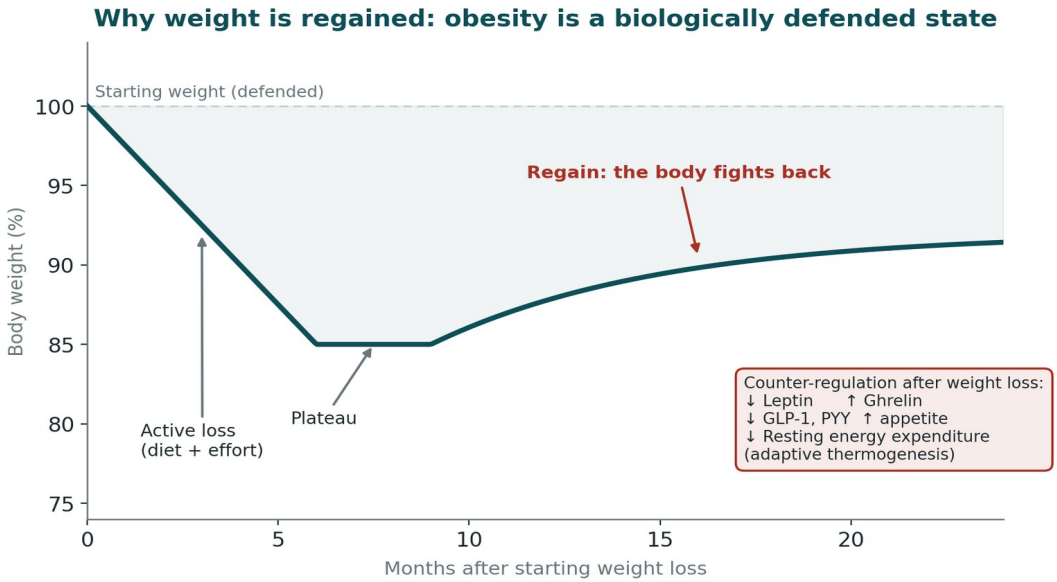


**Figure 1.4** *After weight loss the body defends its old weight: hunger rises, fullness signals fall, and the resting metabolic rate drops.*

When a person loses weight, three things happen at once, and all of them oppose the loss. Leptin falls faster and further than the fat does, so the brain thinks the body is starving even though it is only smaller. Ghrelin, the hunger hormone, rises. And the resting metabolic rate falls by more than the smaller body needs, an effect called adaptive thermogenesis: the body learns to run on less fuel. In one landmark study, these hormone changes were still present a full year after weight loss. The pressure to regain does not fade after a few hard weeks. It stays. In another study, people from a televised weight-loss contest still had suppressed metabolisms six years later.

MYTH vs EVIDENCE: **People regain weight because they get lazy and go back to old habits**

**Evidence:** Habits play a part, but the main driver is biology. Measured hunger is higher, measured fullness is lower, and measured energy use is lower, all pushing the same way, and all outside conscious control. Patients regain weight while genuinely trying not to. This is why we call obesity a chronic, relapsing disease. Like high blood pressure, stop the treatment and the disease comes back. It is also why treatment that works usually has to continue, not stop.

## 6 Genes Load the Gun, the Environment Pulls the Trigger

If body weight is defended, where is the defended level set, and why has it risen across a whole population in one generation? The answer has two parts that work together.

Body weight is one of the most inherited of all human traits. Twin and adoption studies show that a large share of the difference between people comes from their genes. In a few patients, a single gene is responsible (the monogenic obesities you will meet in Module 4). In most people, hundreds of common gene variants each nudge appetite, fullness and fat storage a little, and together they add up to a strong inherited tendency. This is why obesity runs in families, and why some people can eat freely and stay lean while others cannot.

But genes change slowly, over thousands of years. Our waistlines changed in a few decades. Genes set the risk; the modern world pulls the trigger. Cheap, energy-dense, tasty food is available at every hour. Sugary drinks are everywhere. Screens replace movement. Sleep is short and broken. Stress is constant. And for many people in our cities, the story begins before birth. A baby who was undernourished in the womb, a common history in India, seems to be set for "thrift," and copes badly

when later surrounded by plenty. A vulnerable set of genes meeting an obesity-promoting world is the whole epidemic in one sentence.

## 7 From Blame to Biology: Obesity as a Disease

Once you accept that obesity is a fault in a controlled system, defended by hormones and metabolic rate, set partly by genes and triggered by the environment, the old idea of blame falls apart. You would not tell a patient with an underactive thyroid to simply try harder. Over the last decade, major medical bodies have formally recognised obesity as a disease. This matters. It changes who is responsible (the doctor, not only the patient), what we offer (treatment, not just advice), and how it is funded and studied.

The most important recent change, and a preview of Module 2, is in how we diagnose it. In 2025 a global expert commission said we should stop treating a BMI number as the diagnosis. Instead, we should separate two states. In preclinical obesity, extra fat is present but the organs still work normally; the risk is of future disease. In clinical obesity, the extra fat has already caused real problems in the body. The point is simple but powerful: diagnose obesity the way we diagnose every other disease, by its effect on the person, not by one measurement. You will learn to make this exact split in the next module.

**GUIDELINE POINT: What "chronic and relapsing" means for your Monday clinic**

Set expectations for a marathon, not a sprint. Frame treatment as long-term, like diabetes care.

Expect relapse, and plan for it. Build follow-up and re-treatment into the plan from day one.

Judge success by health gained (better glucose, blood pressure, sleep, mobility, mood), not by the scale alone.

Do not discharge a patient the moment they reach a target weight. That is exactly when the biology of regain is strongest.

## 8 The Words We Use

If obesity is a disease, then the language of blame is not only unkind, it is bad medicine. Patients who feel judged about their weight avoid clinics, skip appointments, and delay tests. Some eat more when stressed by that judgement. Weight bias among doctors is well documented, and it makes the care patients receive worse. So the way you talk about weight is not just good manners. It is part of the treatment, and it starts before you prescribe anything.

The main rule is simple: use person-first, neutral words. The patient is a person who has obesity, not an "obese person," just as we would never reduce a patient to their cancer. Ask permission before you discuss weight at all. Put the cause on biology, not character. Small changes in wording decide whether the patient hears a helper or a judge.

| Instead of | Say | Why |
|---|---|---|
| "You are obese / morbidly obese" | "You are living with obesity" | Person-first; obesity is something they have, not who they are |
| "You just need to lose weight" | "Let's work on your health; weight is one part" | Moves the goal from a number to health |
| "Have you tried dieting?" | "May I ask about your weight today?" | Asks permission; respects the patient |
| "You failed the diet" | "That method did not fit your biology" | Puts failure on the method, not the |

| Instead of | Say | Why |
|---|---|---|
| | | person |
| "Fat / heavy / big" | "Weight," "excess weight," "BMI" | Neutral, clinical, non-judgemental |

**ASK YOUR PATIENT: Opening the conversation**

A simple, respectful opener puts the patient in control: "Would it be alright if we talked about your weight and how it might affect your health? Only if you are comfortable." Ask permission first, every time. If the answer is no, you have still shown respect, and the door stays open for next time.

## 9 How to Explain This to Your Patient

The ideas in this module are not just for you. Said in plain words, they lift shame and build trust. Here are short lines you can use in clinic.

**IN THE CLINIC: Plain-language scripts**

**On willpower:** "This is not about willpower. Your body defends its weight with hormones. We are going to work with your biology, not against it."

**On regain:** "When you lost weight before, your body fought back: it made you hungrier and slowed your metabolism. That is normal, not failure. It is also why we treat this long-term."

**On waist:** "The fat around your belly is the harmful kind. That is why I measure your waist, not just your weight."

**On the plan:** "We will treat this like blood pressure or sugar: step by step, and for the long run. Small steady changes beat big short ones."

## Clinic-Ready Summary

**KEY TAKEAWAYS**

- Obesity is a chronic, relapsing disease of body-weight control, not a failure of willpower.
- The hypothalamus uses leptin and gut hormones to set hunger and energy use; treatment works by changing these signals, not by telling patients to try harder.
- Fat tissue is an active organ; visceral (belly) fat is the harmful kind, so waist matters as much as weight, especially in Asian Indians.
- After weight loss the body fights back (more hunger, less fullness, lower metabolic rate); relapse is biology, and treatment is usually long-term.
- Diagnose the disease by its effect on the person, not by a BMI number alone (Module 2).
- Person-first, permission-first words are part of the treatment; weight stigma harms outcomes.

**IN THE CLINIC: The cliffhanger: next month**

Imran weighs 96 kg with a BMI of 32.5. But his colleague Sridhar has a BMI of 24.5, which any chart calls "not overweight." Sridhar has a 96 cm waist, a fatty liver on ultrasound, and a fasting glucose of 116. One of these men has clinical obesity and does not know it. The other may not

need a single tablet. In Module 2 you will learn to tell them apart in ninety seconds, using the 2024 Asian-Indian staging and the 2025 clinical-obesity framework. Bring a measuring tape.

## Self-Assessment

*Answers and short explanations follow each question. These are the style of the module post-test and the final exam.*

**Q1.** Which hormone mainly increases hunger, rising when the stomach is empty?

**A.** Leptin
**B.** GLP-1
**C.** Ghrelin
**D.** Peptide YY (PYY)

**Answer: C.** *Ghrelin, from the stomach, is the main hunger hormone. Leptin, GLP-1 and PYY promote fullness.*

**Q2.** A 34-year-old Hyderabadi man has a BMI of 24 but a waist of 98 cm and a fatty liver. The best explanation is:

**A.** Visceral (belly) fat is harmful even at a normal BMI, a common pattern in Asian Indians
**B.** He has no weight problem because his BMI is normal
**C.** BMI is the only measure that matters
**D.** The fatty liver is unrelated to fat

**Answer: A.** *The "thin-fat" Asian-Indian pattern means real visceral fat and metabolic risk can exist at a normal BMI. Waist size reveals it.*

**Q3.** Adaptive thermogenesis after weight loss means:

**A.** The resting metabolic rate rises and helps keep weight off
**B.** The resting metabolic rate falls by more than the smaller body needs
**C.** Leptin rises and reduces hunger
**D.** It lasts only about a week

**Answer: B.** *The resting metabolic rate falls more than the smaller body would predict, and this stays for months to years, driving regain.*

**Q4.** Which statement best reflects the 2025 change in how we diagnose obesity?

**A.** BMI alone should define the diagnosis for everyone
**B.** Waist should replace all other measures
**C.** Obesity is no longer a disease
**D.** Diagnose by real organ or tissue problems (clinical obesity), not by a BMI number alone

**Answer: D.** *The 2025 commission separates clinical obesity (fat causing dysfunction) from preclinical obesity (fat present, organs still working).*

**Q5.** Adiponectin, a hormone from fat tissue, usually:

**A.** Rises in obesity and worsens insulin resistance
**B.** Is an inflammatory signal
**C.** Falls in obesity and improves insulin sensitivity
**D.** Has no role in metabolism

**Answer: C.** *Adiponectin is protective and improves insulin sensitivity. Unlike most fat signals, it falls as visceral fat rises.*

**Q6.** The most accurate way to explain regain to a patient is:

**A.** "You lacked the willpower to keep it off"
**B.** "Your body defended its old weight with hormone and metabolic changes; this is expected, not a personal failure"
**C.** "Diets always work if you follow them exactly"
**D.** "Just eat even less next time"

**Answer: B.** *Regain is driven by measurable biology. This framing is accurate, kind, and reduces stigma.*

**Q7.** Person-first language for a patient with obesity is preferred because:

**A.** It is legally required
**B.** It makes the visit longer
**C.** It changes the BMI
**D.** It reduces weight stigma, which otherwise worsens engagement and outcomes

**Answer: D.** *Weight stigma leads to avoidance of care and worse outcomes. Neutral, person-first words are part of good treatment.*

**Q8.** Why does "eat less, move more" fail as a stand-alone prescription?

**A.** The first law of thermodynamics is wrong
**B.** Eating less triggers more hunger and a lower metabolic rate
**C.** Physical activity has no health effect
**D.** Patients never try it

**Answer: B.** *Intake and energy use are linked. Cutting food triggers counter-regulation, so conscious effort fights an unconscious control system.*

## Key References & Further Reading

*Selected sources for this module, current as of this edition. Full details are in the course reference list.*


- Anjana RM, Unnikrishnan R, Deepa M, et al. Metabolic non-communicable disease health report of India: the ICMR-INDIAB national cross-sectional study. Lancet Diabetes Endocrinol. 2023;11(7):474-489.
- Rubino F, Cummings DE, Eckel RH, et al. Definition and diagnostic criteria of clinical obesity (Lancet Diabetes & Endocrinology Commission). Lancet Diabetes Endocrinol. 2025;13(3):221-262.
- Misra A, et al. Revised definition and diagnostic criteria of obesity in Asian Indians living in India. 2024.
- Sumithran P, Prendergast LA, Delbridge E, et al. Long-term persistence of hormonal adaptations to weight loss. N Engl J Med. 2011;365(17):1597-1604.
- Fothergill E, Guo J, Howard L, et al. Persistent metabolic adaptation 6 years after "The Biggest Loser" competition. Obesity. 2016;24(8):1612-1619.
- Rubino F, Puhl RM, Cummings DE, et al. Joint international consensus statement for ending stigma of obesity. Nat Med. 2020;26(4):485-497.
- Loos RJF, Yeo GSH. The genetics of obesity: from discovery to biology. Nat Rev Genet. 2022;23(2):120-133.

THE FULL TEXTBOOK COMES WITH THE COURSE

# You've read one module. There are eleven more.

The complete textbook covers all twelve modules — from diagnosis with Asian-Indian cut-offs to GLP-1-era pharmacotherapy, surgery and building your own obesity practice — plus four clinical appendices: a dosing quick-reference, treatment algorithms, an Indian-brand formulary, and patient handouts. It is yours to keep as part of the course.

FOUNDING BATCH

## Begins Sunday, 30 August 2026 · Hyderabad

One Sunday a month, 10:00 am – 1:00 pm, followed by lunch · API Hall, Oasis Plaza, Abids
12 monthly modules + convocation · Live quizzes · 60-question final exam · Verifiable certificate

**₹2,999** ~~₹9,999~~

**Founding-batch fee · only 40 seats · seats confirmed on payment**

**Scan to apply · caspianobesity.com/apply**

**Money-back guarantee: full refund any time before Session 1 — and if the first session isn't value for money, a full refund within 7 days of it.**

Questions? WhatsApp +91 89784 54547 · drkhizer@caspianobesity.com

Offered by Caspian Healthcare Foundation, a registered public charitable trust · Hyderabad · caspianobesity.com