

**Dr Shahrukh Azmi** , Assistant, Samarkand State Medical University, Uzbekistan

**Shejal chopal** , Medical Student, Samarkand state medical university, Uzbekistan

**Riya thakur** , Medical Student, Samarkand state medical university, Uzbekistan

**Vaishnavi thakur** , Medical Student, Samarkand state medical university, Uzbekistan

**Ashrith** , Medical Student, Samarkand state medical university, Uzbekistan

## Abstract

For more than a century, obesity has been weighed, charted, and moralised—reduced to a ratio of height over weight squared and, too often, to a verdict of weak will. Yet since the American Medical Association recognised obesity as a disease in 2013, and with particular force since the Lancet Diabetes & Endocrinology Commission's 2025 redefinition, medicine has begun to treat excess adiposity as what the biology always said it was: a chronic, relapsing, multifactorial illness of dysfunctional adipose tissue. This narrative review traces how obesity crossed the threshold from behaviour to disease; why body mass index (BMI), the instrument that built that threshold, is now recognised as an inadequate ruler for individuals; what adiposopathy and the genetics of energy homeostasis reveal about the condition's mechanisms; and how the new distinction between clinical and preclinical obesity promises to end both overdiagnosis and therapeutic neglect. We examine staging systems, the treatment revolution spearheaded by incretin-based pharmacotherapy, and the unresolved tensions—stigma, medicalisation, equity, and the fate of the "obesity paradox." The evidence supports a clear thesis: obesity qualifies as a chronic disease not because a committee declared it so, but because it behaves like one—progressive, relapsing, genetically and environmentally driven, and treatable only when it is measured beyond the scale.

**Keywords:** obesity; chronic disease; body mass index; adiposopathy; clinical obesity; preclinical obesity; Lancet Commission; Edmonton Obesity Staging System; GLP-1 receptor agonist

## 1. Introduction

Consider two patients in the same clinic. The first is a rugby forward: 1.83 metres tall, 110 kilograms, BMI 32.9—squarely in the "obese" range by every textbook table—whose blood pressure, glucose, and lipids are impeccable and whose body fat hovers near 14%. The second is a retired schoolteacher: 1.60 metres, 63 kilograms, BMI 24.6—"normal weight"—whose waist circumference tells a different story, whose liver shimmers with fat on ultrasound, and whose fasting glucose has quietly crossed into diabetes. Two bodies, two diseases, one number that gets the first man wrong and misses the second woman entirely. For decades this arithmetic was the whole of the diagnosis.

The story of how obesity became a disease is really two stories: one of biology, in which adipose tissue was revealed as an endocrine organ whose dysfunction drives systemic illness; and one of policy, in which medicine's institutions slowly granted the condition the standing of a chronic disease—one that, like hypertension or asthma, requires long-term management rather than willpower and blame. The National Institutes of Health declared obesity a disease in 1998; The Obesity Society followed in 2008; the American Association of Clinical Endocrinologists in 2012; and in June 2013 the American Medical Association's House of Delegates, overriding its own Council on Science and Public Health, voted to recognise obesity as a disease state "with multiple pathophysiological aspects requiring a range of interventions." The pace then accelerated dramatically: in January 2025, a Lancet Commission of 58 experts, endorsed by more than 75 medical organisations worldwide and including people with lived experience of obesity, published a consensus redefinition that demoted BMI from diagnostic instrument to screening tool. This article follows that arc—why BMI was trusted, why it failed, what replaced it, and what the new framework changes for patients and physicians.

| Year      | Milestone                                                                | Significance                                                                         |
|-----------|--------------------------------------------------------------------------|--------------------------------------------------------------------------------------|
| 1998      | NIH declares obesity a disease                                           | Federal research and treatment legitimacy; begins shift from behaviour to pathology. |
| 2004–2006 | CMS reclassifies obesity; covers bariatric surgery under Medicare (2006) | Insurance reimbursement for obesity treatment becomes possible in the United States. |

|                     |                                                                                      |                                                                                                                                               |
|---------------------|--------------------------------------------------------------------------------------|-----------------------------------------------------------------------------------------------------------------------------------------------|
| <b>2008</b>         | The Obesity Society position paper: obesity as a disease                             | Scientific society endorsement built on adipose-tissue biology.                                                                               |
| <b>2012</b>         | American Association of Clinical Endocrinologists position statement                 | Endocrinology leadership formally adopts disease framing.                                                                                     |
| <b>June 2013</b>    | AMA House of Delegates recognises obesity as a disease state                         | Most influential medical association; catalyses coverage, research, and de-stigmatisation.                                                    |
| <b>January 2025</b> | Lancet Diabetes & Endocrinology Commission consensus (58 experts, 75+ organisations) | Redefines obesity around excess adiposity and organ function; creates clinical vs preclinical obesity; relegates BMI to population screening. |

*Table 1. Milestones in the recognition of obesity as a chronic disease.*

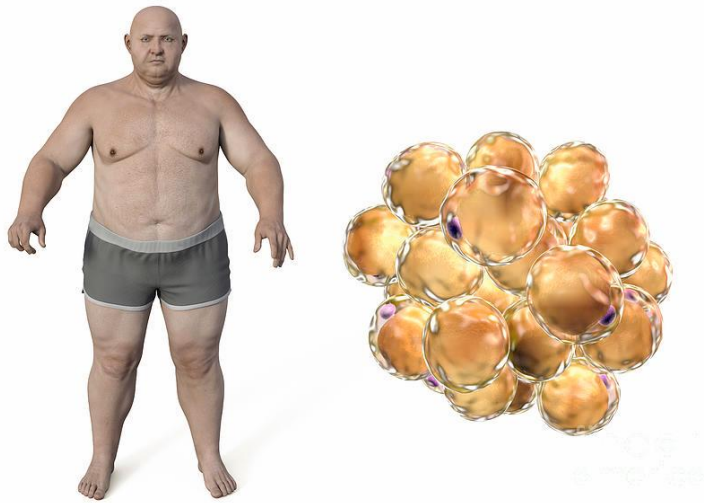
## 2. The Blunt Instrument: Why BMI Cannot Bear the Weight of a Diagnosis

BMI was never designed to measure fat. Adolphe Quetelet devised the index in the 1830s to describe the "average man" of a population, not to diagnose anyone; it was adopted in clinical practice largely because height and weight are cheap to obtain. But a ratio of mass to height squared cannot distinguish adipose from muscle, visceral from subcutaneous fat, or a metabolically healthy fat depot from an inflamed, dysfunctional one. The consequences are systematic and well documented. Elite athletes are misclassified as obese. Conversely, the "TOFI" phenotype—thin on the outside, fat on the inside—passes through every screening net, particularly common in South and East Asian populations, in whom cardiometabolic risk rises at BMI and waist values far below the conventional cut-offs. The same BMI also means different things at different ages and in different ethnic groups, which is why the Commission insisted on ethnicity-, sex-, and age-appropriate thresholds. And at the far end of the spectrum, large epidemiological cohorts produced the so-called obesity paradox—overweight and class-I-obese patients with heart failure or chronic kidney disease sometimes outliving normal-weight ones—largely, critics argue, because BMI obscures which patients carry

dangerous visceral adiposity and muscle wasting versus which carry metabolically quieter fat. A measure that can be wrong in both directions, in every population, at every extreme, is a screening statistic, not a diagnosis. The Lancet Commission's core recommendation follows directly: BMI should be used only as a surrogate measure of health risk at the population level, for epidemiology, or for initial screening—never as an individual measure of health.

### 3. The Biology That Earned the Diagnosis

What transformed the classification was not rhetoric but mechanism. Adipose tissue, it turns out, is an endocrine and immune organ, and in excess it becomes sick—"adiposopathy," in the coinage of obesity medicine. As adipocytes enlarge beyond their vascular supply, they become hypoxic, recruit macrophages, and secrete a pro-inflammatory soup of cytokines and adipokines that drives insulin resistance, endothelial dysfunction, and ectopic fat deposition in liver, muscle, and pericardium. The disease is also, crucially, a disease of the brain's energy-regulation circuitry. Twin, family, and adoption studies attribute 40–70% of the population variance in adiposity to heredity; more than twenty genes cause monogenic obesity, most acting on hypothalamic satiety pathways; and analyses of genome-wide association studies have implicated over a thousand loci whose variants nudge appetite and expenditure upward in obesogenic environments. Add leptin resistance, gut-brain signalling, sleep, stress, medications, and the built environment, and the stereotype of simple gluttony collapses. The physiology explains the clinical signature of a chronic disease: weight defended by powerful homeostatic forces, with roughly nine in ten people who lose weight regaining most of it as appetite hormones surge and resting metabolism falls. That pattern— progression, relapse, multifactorial causation—is precisely what defines chronic disease, and it is why the Obesity Medicine Association describes obesity as a "chronic, relapsing, multifactorial, neurobehavioural disease."



*Figure 1. Obesity understood as adipocyte disease: excess, enlarged, and metabolically dysfunctional fat cells—not merely excess kilograms. (Photograph: K. Kon / Science Photo Library.)*

#### 4. The New Grammar: Clinical and Preclinical Obesity

The 2025 Commission report supplies what BMI never could: a definition of illness. Obesity is defined as excess or abnormally distributed adiposity, confirmed not by weight alone but by direct measurement (DXA or bioimpedance) or by anthropometry—waist circumference, waist-to-hip ratio, or waist-to-height ratio—using validated, ethnicity-specific cut-offs. Within that umbrella sit two distinct states. Preclinical obesity is excess adiposity with preserved organ function: a state of increased risk, warranting monitoring, risk counselling, and treatment when risk is substantial—but not yet an illness. Clinical obesity is excess adiposity that is already injuring tissues: obesity-induced heart failure, obesity-related breathlessness from reduced lung function, knee and hip disease with stiffness and lost range of motion, obstructive sleep apnoea, or metabolic-organ dysfunction—codified in eighteen diagnostic criteria for adults and thirteen organ-system criteria for children and adolescents. The distinction does real clinical work. It ends the absurdity in which insurers demand a second disease, such as diabetes, before paying for obesity treatment; clinical obesity is itself the indication. It protects people with high BMI but healthy organs from unnecessary pharmacotherapy and surgery. And it reframes treatment goals away from the scale—where weight cycling awaits—toward the restoration of organ function and daily living, echoing the older Edmonton Obesity Staging System, which grades patients 0–4 by metabolic, functional, and psychosocial impairment rather than by BMI alone.

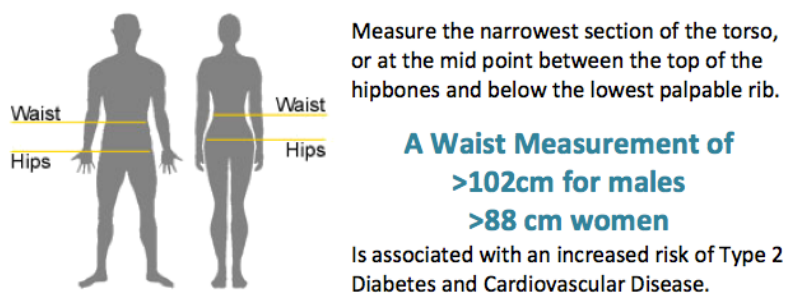
| Traditional (BMI-centric) approach                                                                                | Lancet Commission framework (2025)                                                                                                                                                                                                                                               |
|-------------------------------------------------------------------------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>Sole reliance on BMI <math>\geq 30</math> kg/m<sup>2</sup> (or ethnicity-specific cut-offs) for diagnosis.</b> | Diagnosis requires confirmation of excess/abnormal adiposity: direct measurement (DXA, bioimpedance), or BMI plus at least one anthropometric criterion, or two anthropometric criteria (WC, WHR, WHtR) regardless of BMI; excess fat pragmatically assumed when BMI $\geq 40$ . |
| <b>BMI treated as an individual measure of health and illness.</b>                                                | BMI demoted to a population-level, epidemiological, or screening tool—not an individual diagnostic measure.                                                                                                                                                                      |
| <b>No distinction between illness and risk; "obese or not" as the whole question.</b>                             | Two categories: preclinical obesity (excess fat, preserved organ function—increased risk, not yet illness) and clinical obesity (excess fat with organ dysfunction or major limitation of daily activities—an illness in its own right).                                         |
| <b>Treatment typically justified only after a comorbidity (e.g., type 2 diabetes) emerges.</b>                    | Clinical obesity itself justifies treatment; preclinical obesity managed by risk reduction and monitoring, with treatment if risk is very high.                                                                                                                                  |
| <b>Success defined as percentage of weight lost.</b>                                                              | Success defined by restored organ function, improved daily living, cardiometabolic risk reduction, and prevention of progression—weight change as one marker, not the goal.                                                                                                      |

Table 2. The traditional BMI-centric model versus the Lancet Commission diagnostic framework.

### 5. The Treatment Revolution the Redefinition Made Possible

Disease status matters most when it changes what can be prescribed. Recognition of obesity as a chronic disease legitimated the armamentarium that now exists: intensive behavioural therapy, metabolic and bariatric surgery (which remains the most durable intervention for clinical obesity), and—most consequentially—the incretin-based pharmacotherapy era. Semaglutide, a GLP-1 receptor agonist, produces sustained mean weight loss of roughly 15% in trials, and tirzepatide, combining GIP and GLP-1 activity, has exceeded 20% in head-to-head studies, with encouraging signals for cardiovascular, renal, and hepatic outcomes. These are not diet pills; they act on the very neurobehavioural pathways the disease definition describes, restoring satiety signalling against a biology that defends excess weight. Yet the gap between capability and delivery remains scandalous: fewer than one in four people living with obesity receives any evidence-based treatment, hampered by cost, access, and lingering policy that still treats the condition as cosmetic. Precision medicine—phenotyping patients by genotype, adipose distribution, and metabolic phenotype to match diet, drug, device, or surgery—is the horizon the new definition points toward.

#### Measuring Waist Circumference



*Figure 2. Waist circumference measurement: the narrowest torso point or midpoint between lowest rib and iliac crest. Values above 102 cm (men) and 88 cm (women) signal elevated visceral adiposity and cardiometabolic risk. (Infographic: Fijian Ministry of Health.)*

### 6. Honest Tensions: Medicalisation, Stigma, and the Paradox That Will Not Die

The disease framing is not uncontested, and its critics raise questions worth keeping in view. Medicalisation can pathologise body diversity and hand pharmaceutical markets a mandate; the Commission itself acknowledged the overdiagnosis risk, which is precisely what the clinical/preclinical divide is designed to contain. Labels can cut both ways on stigma: some studies show that a disease attribution reduces blame for individuals, while others warn that treating body size as pathology deepens societal weight bias—language such as "person living with obesity" rather than "obese person"

is the small daily countermeasure. The obesity paradox, though much of it dissolves when waist circumference and body composition replace BMI, reminds us that the relationship between adiposity and outcomes is a continuum, not a cliff edge, and that patients reclassified as "non-obese" under the new definition still carry intermediate risk and deserve counsel rather than dismissal. And equity remains the unfinished chapter: the medications and surgeries that the chronic-disease model legitimises are least available where obesity prevalence and its complications are highest.

## 7. Conclusion

The case for obesity as a chronic disease now stands on three converging legs. Mechanistically, adiposopathy, neurobehavioural dysregulation of energy balance, and strong heritability make obesity behave like—and be—an illness rather than a habit. Epidemiologically, BMI's twin failures of overdiagnosis and underdiagnosis have forced better instruments—waist measures, body composition, staging—that describe the disease the way it actually is. And clinically, treatment has outgrown the old paradigm, with pharmacotherapy that acts on the disease's own pathways and surgical options with decades of outcome data, both awaiting the coverage that disease recognition was designed to unlock. For the rugby forward, the new framework says: high BMI, healthy organs—monitor, counsel, no stigmatising label of illness. For the schoolteacher, it says: modest BMI, sick liver—diagnosed, staged, treated. That is what "beyond BMI" means in practice: the end of weighing people to decide whether they are ill, and the beginning of measuring them to find out. The scale never deserved the final word. It kept it for a century anyway. Medicine is finally taking it back.

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