macklin METHOD

01

MODULE ONE · BEHAVIOURAL THERAPY

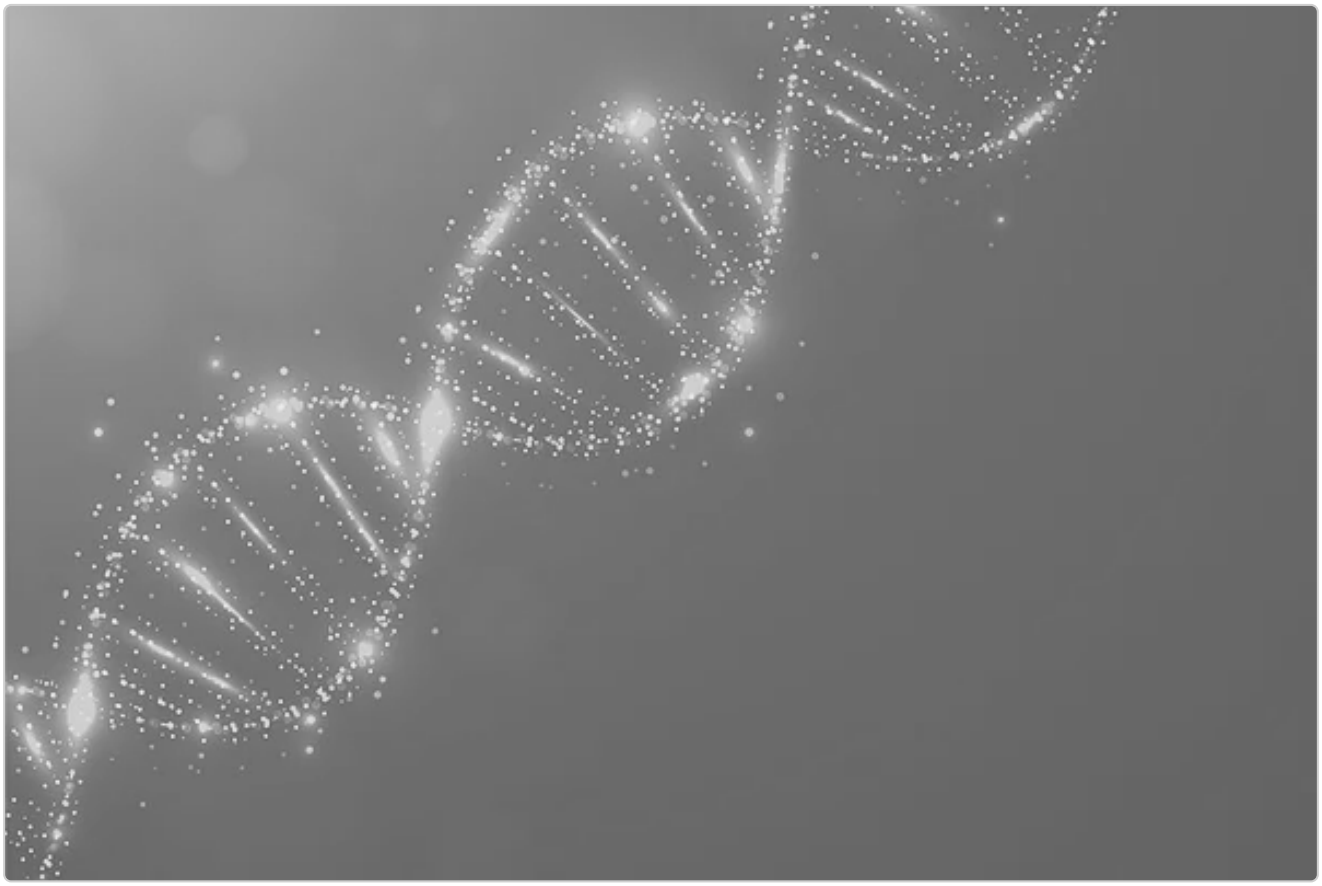
Internalized weight bias.

Weight bias and stigma

Weight bias is the set of negative attitudes and stereotypes directed at people because of body size. It appears in public life, media, health care, families, workplaces, and private relationships. One common form is the idea that weight is simply a personal choice that would be easily reversed if someone ate less and moved more.

Internalized weight bias happens when those external messages are taken in and turned against the self. Instead of seeing excess weight as a treatable medical condition, a person may start to see it as evidence of weakness, failure, or lack of discipline. That internal story is not neutral. It can increase stress, lower self-esteem, worsen mood, and reduce confidence that treatment is possible.

The cumulative effect over a lifetime is what matters. These messages arrive from many directions — society and culture, the people around you, sometimes even from within the medical community itself — and they arrive across years. What is left behind is a library of internalized thoughts that speak poorly about you as a person and about your capacity to manage your weight. That library is the very material the resilience work in Module 05 returns to. The work there is partly to recognize that the cultural messaging you have internalized over a lifetime is not the same as truth — that the thoughts in the library got there naturally, through no doing of your own.



A different explanation

This module invites you to consider that struggling with weight means struggling with a real condition that is mostly genetic, centred in the brain, strongly influenced by the environment, progressive in the way the brain and body defend against weight loss, inadequately treated by dieting and exercise alone, and manageable over the long term with real treatment.

For patients on obesity medication, this reframe often arrives not just as a concept but as a felt experience. Weeks into treatment, you may notice that food no longer holds the constant pull it once did — that thoughts about eating have quieted, that meals are easier, that the day no longer revolves around managing wanting. A question some patients ask in that moment is: "Is this how people who don't struggle with weight feel?" That question is itself evidence. The medication is treating something real, and your own experience of being treated is felt confirmation that the condition was real to begin with.

The question is not whether effort matters. It does. The question is what kind of effort has a fair chance of working. If the condition has never been comprehensively treated, then past attempts at sustained weight loss may not be good evidence about what is possible now.

Genetics

A large proportion of the variation in human body size is inherited.[1] Many genes are involved, and the inherited risk is not mainly about character or preference. It is about biology. In other words, people differ in the systems that regulate appetite, motivation, fullness, metabolism, and defence against weight loss.

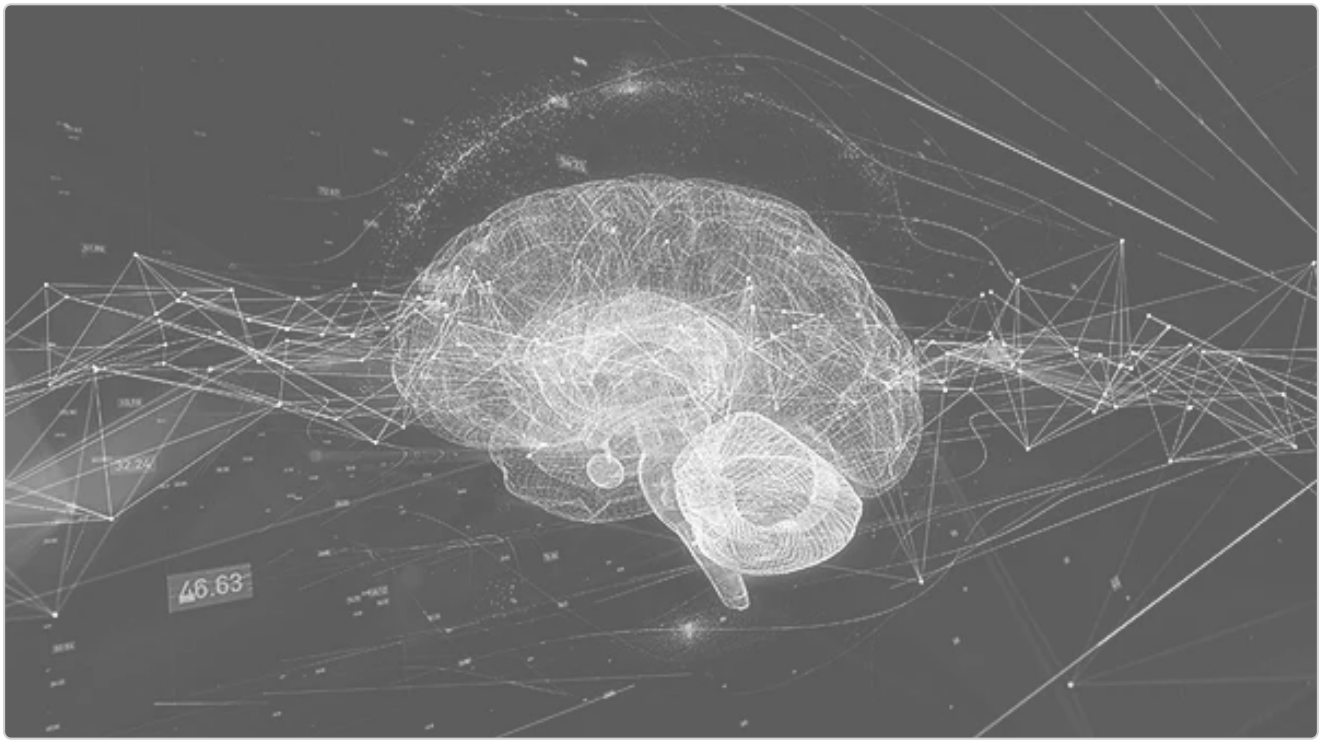
The brain

Many weight-related genes are expressed in the central nervous system, meaning the brain.[2] The appetite system can be understood as three interacting layers: the homeostatic system, the motivation system, and the executive system.

The **homeostatic system** is the biological defence against fat loss. It monitors energy stores, and when fat is lost it drives the body to regain it.

The **motivation system** generates the cue-triggered drive to eat. In the modern food environment — where calories require no work to acquire, and where foods are engineered to be hyper-palatable — it is the principal symptom of obesity.

The **executive system** is where deliberate self-regulation happens — the effortful, planning, reflective part of the brain. It is the newest and weakest of the three, regularly asked to work against older and stronger non-conscious drives.



The environment

The human appetite system evolved in an environment where calories could be scarce and finding food often required work. The modern food environment is very different: large portions, highly available food, ultra-processed products, added sugar-fat-salt combinations, constant cues, advertising, and delivery. That environment acts on the inherited appetite system and can drive calorie intake above what the body needs.

This is not only about less healthy food. Even abundant healthy food can contribute to overconsumption when availability, portion size, cues, and wanting are strong. The point is not blame. The point is that a vulnerable appetite system is living in an environment that repeatedly activates it.

The body defends against weight loss

When a person loses fat, the body can respond as though something is wrong. Appetite can increase. Metabolic rate can decrease. The homeostatic system notices signals of fat loss, including changes in leptin, and its defence against further loss strengthens.[3]

As weight loss continues, the drive to eat can grow stronger and weight loss can slow, slow again, and eventually plateau.[4] This is not a failure of motivation. It is a predictable biological defence against fat loss, useful in a former environment and difficult in the current one.

Why treatment matters

Dieting and exercise are often the only tools people have been told to use. They matter for health, function, and daily structure, but they are usually inadequate as stand-alone long-term treatment for a brain-centred chronic condition. Treatment has to meet the biology more directly and more comprehensively.

That is why the Macklin Method frames treatment through three pillars: behavioural therapy, obesity medication, and bariatric surgery. Behavioural therapy builds skills through the eight modules. Medication can act on non-conscious appetite systems that effort cannot directly reach. Bariatric surgery is an additional treatment pathway for people who qualify and should be handled with the surgical team.

01

Behavioural therapy

The modules target skills, attention, planning, thinking, values, resilience, and day-to-day treatment effort.

02

Medication

Obesity medication can reduce biological pressure and support behavioural treatment when clinically appropriate.

03

Bariatric surgery

Surgery is acknowledged as an effective pillar, with decisions made through the surgical care team.

Why this module comes first

Much of treatment rests on a single foundational belief: that your struggle is real, valid, and not your fault — a real medical condition, brain-centred and chronic. When that belief is in place, every other module's work lands differently. You are more likely to stay on medication, because you understand what the medication is treating. You are more likely to recognize wanting as a neurological symptom rather than a personal failure. You are more likely to understand the brain's defence against fat loss as biology, not weakness. You are more likely to find values clarification meaningful, because long-term clarity matters when you are managing a chronic disease. This is also why internalized weight bias is the first chapter of the Canadian Adult Obesity Clinical Practice Guidelines — a globally celebrated framework for the treatment of obesity, which states that treatment is generally less effective when internalized weight bias is not addressed first. Our first module follows the same logic.

Internalized weight bias is not one module among eight. It is the foundation the other seven modules build on.

The invitation

Would you consider that you have been struggling with excess weight not because of a flaw in character, willpower, or motivation, but because you have been living with a real condition for which effective and comprehensive treatment exists?

> ***Countering internalized weight bias is not a side issue. It is part of treatment, because it changes the explanation you carry into every next step.***

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4. Hall KD, Kahan S. Maintenance of lost weight and long-term management of obesity. Medical Clinics of North America. 2018. View source ↩