

'With in-depth knowledge, clinical experience and compassion, Lara Briden guides us through our metabolic and mindset challenges.'

– DR LIBBY WEAVER

METABOLISM + REPAIR FOR WOMEN

A compassionate,
science-based guide
to **balancing insulin,**
losing weight,
and **improving**
health

'This book is a must
for women everywhere.'

– DR STACY SIMS

LARA BRIDEN _{ND}

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to my patients

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Introduction: Why you need this book

Welcome to *Metabolism Repair for Women*, a troubleshooting guide to metabolic health—and perhaps even weight loss.

This is a book about doing things differently. Specifically, it's about working *with* (not against) the parts of your biology that should naturally and effortlessly regulate hunger, fullness, and energy.

To do this, we'll focus on the common problems of *insulin resistance* and *metabolic inflexibility* and how they could be preventing you from accessing your own body fat for energy. If you feel tired and hungry all the time—and can't lose weight—insulin resistance and metabolic inflexibility could be why.

If you've never heard of insulin resistance, you're not alone. Hundreds of times, I've said to my patients, "I'm concerned you have insulin resistance," and hundreds of times, they've replied, "Oh no, my doctor tested my insulin, and it's fine." And then we looked at their results and saw it was their blood sugar that was tested, not the hormone insulin. The three things to understand about insulin resistance—also called *prediabetes* or *metabolic syndrome*—are that:

- It's linked with high insulin but, in most cases, normal blood sugar.

- It causes abnormally high hunger and an increased tendency to store fat.
- It's extremely common, affecting at least 46 percent of adults worldwide.

It could be affecting *you*. In fact, if you've picked up this book, it probably is.

Fortunately, there are lots of ways to reverse insulin resistance, and almost none of them involve calorie counting. Because as we'll see, it's hard (maybe even impossible) to consciously and intentionally reduce calories when the underlying problem is that you're abnormally hungry. The alternative is to find ways to feel less hungry and burn more energy—and that's what we're going to do.

Why I wrote this book

In my first two books, *Period Repair Manual* and *Hormone Repair Manual*, I wrote about my passions—women's hormones and periods. Why have I now turned to metabolic health? Because after twenty-five years of working with patients, I know just how strongly women's hormones are affected by insulin sensitivity. For me, therefore, writing about insulin resistance feels like the natural next step. It's about going deeper into women's health—down to the level of the cell, where metabolic health lives. And before you think, “Oh, that sounds complicated,” please know that although there are lots of moving parts, they can all translate into *simple, manageable strategies*.

How to use this book

The first two chapters are about understanding the reality of your situation. By that, I mean understanding how the modern food environment has damaged you and all of us. And why weight gain is *not your fault*.

In Chapter 3, we'll explore diet culture and *clearing away the shame* before moving on to Chapter 4 and the biology of metabolic health, where we'll do a crash course in things like mitochondria,

which are the powerhouses of cells; fat adaptation, which is becoming better at burning fat; and the all-important autonomic nervous system, which keeps all body systems humming along without your conscious control.

In Chapters 5 to 9, we'll get practical with *metabolic troubleshooting*, where we'll assess your metabolic health and discover all the possible *metabolic obstacles* you might need to address. One thing to be aware of right from the start is that your metabolic obstacles could be entirely different from someone else's. That's why this is a troubleshooting manual. It's an individualized, personalized approach to metabolic health and weight loss that, in my experience with thousands of patients, is really the only way forward. As you read, you'll encounter patient stories (with names changed) to illustrate troubleshooting in action. Then, in Chapters 10 to 12, we'll explore treatment options for some of the trickier obstacles.

Finally, we'll put it all together in Chapter 13, where I'll also offer tips on making long-term change.

Special boxes

Throughout this book, you'll find definitions, tips, special topics, and "How to speak with your doctor" sections.



definition

Definition boxes provide simple explanations for any technical words. You can also find them in the Glossary.



Tips are extra bits of information you may find helpful.

Special Topic: Explore in more detail

Special topics to provide you with extra, in-depth information.

How to speak with your doctor

To make your doctor-patient conversations as productive as possible, I've provided short lists of statements and questions to assist in communication about specific topics—such as testing for insulin resistance and discussing the different types of thyroid medication.

Are the recommendations evidence-based?

Wherever possible, I've provided details of relevant scientific studies, which you can find in the References section. If I haven't provided a reference, it's because there's no research as yet on that topic—for example, the role of dairy sensitivity in premenstrual hunger. In those cases, I rely more on the experiences and successes of my patients.

The supplements I recommend are mostly simple and safe to try, and I list any necessary precautions. I also ask that you speak with your doctor or pharmacist about possible interactions between specific supplements and your medical conditions or medications or if you are pregnant or breastfeeding. Always cross-check the labels or packaging for precautions and dosage instructions. To assist you, I've provided a list of suggested supplement brands in the Resources section, but please note that I have not been paid to mention any product or brand name. At the end of the day, choose the supplement that's available to you and isn't too expensive.

My education and background

My first degree was a Bachelor of Science (BSc) from the University of Calgary in Canada, where I published my honors thesis as a scientific paper on the foraging behavior of female bats—an early example of my fascination with female physiology! That work in evolutionary biology was the beginning of my love of science and has informed the way I work with my patients. I view the body as a logical, responsive system that knows what to do when it's given the right support.

After my biology degree, I went on to qualify as a naturopathic doctor (ND) from the Canadian College of Naturopathic Medicine (CCNM) in Toronto. It's one of seven accredited colleges of naturopathic medicine in North America: two in Canada and five in the United States. The first two years of training are similar to conventional medical programs, while the final two years provide hundreds of hours of training in nutritional and herbal medicine, as well as clinical training in an outpatient clinic. After graduating in 1997 under my maiden name, Lara Grinevitch, I was certified by the Naturopathic Physicians Licensing Examinations (NPLEX), professional exams administered by the North American Board of Naturopathic Examiners (NABNE).

In the twenty-five years since, I've worked full-time with patients, first in Pincher Creek, Alberta, Canada, then in Sydney, Australia, and now in Christchurch, New Zealand. During those years, I've had the opportunity to see diet trends come and go, including low-fat, Atkins, Zone, paleo, and vegan. And I've had the opportunity to see which aspects of those various diets work for people and which don't. More importantly, I've had a chance to witness first-hand how different diets work for different people, thanks to individual differences in gut microbiome, hormones, and nervous systems. We'll explore all of that and see why there's much more to metabolic health than just diet.

I sit on several scientific advisory councils, including that of the Centre for Menstrual Cycle and Ovulation Research at the University of British Columbia in Vancouver, Canada, and I've published a couple of peer-reviewed papers about polycystic ovary syndrome (PCOS).

To my thousands of patients over the years, thank you for entrusting me with your health and stories. I dedicate this book to you.

Lara Briden

Part One

Understanding

Not everything that is
faced can be changed,
but nothing can be
changed until it is faced.

~ James Baldwin ~

Chapter 1

Metabolic dysfunction and weight gain are not your fault

This book is about coming home to metabolic health, which you might think is about being able to lose weight. And yes, it's partly about that. But metabolic health is also important for all the good things in life, such as:

- daily energy to do the things you enjoy
- a bright, stable mood
- a pain-free body
- regular, symptomless periods
- smooth hormonal transitions, including postpartum and perimenopause
- a reduced long-term risk of the “big three” hazards—heart attack, stroke, and dementia—that could rob you of an opportunity to grow old with the people you love.

Of course, good metabolic health cannot guarantee those things nor magically prevent all health problems. Good metabolic health is not everything, but it is a lot. Unfortunately, if you're like most of us in the modern world, your metabolic health has likely gone a little off the rails. That's true even if you have a small body size, which might surprise you, given the prevailing narrative that

metabolic dysfunction is caused by obesity. As we'll see, that narrative is incorrect.



I'll **generally avoid** the word "obesity" because it puts an unnecessary emphasis on body size when the real problem is metabolic dysfunction.

An energetic view of health

In simplest terms, metabolic health is about energy. Specifically, it's about your cells having all the energy they need, whether that's from the food you've eaten or—and this is important—from the glycogen or fat you've stored.



glycogen

Glycogen is a glucose storage molecule that is deposited in the liver and muscles after a carbohydrate meal. Glucose can then be released as needed.

Food can directly supply energy for a few hours. Glycogen can supply it for twelve to twenty-four hours, but only if you don't use it up too quickly. Fat, on the other hand, can supply energy for days or even weeks—but only if you can access your fat stores.

Metabolic flexibility versus metabolic inflexibility

To access fat for energy, you need metabolic flexibility, which is the ability to easily switch between burning glucose and fat. With good metabolic flexibility, you burn mostly (but never exclusively) fat when you're at rest or engaged in light activity. You should only tip into burning more glucose (and potentially depleting glycogen stores) when engaged in high-intensity activity.

So, with good metabolic flexibility, you should consistently feel brighter, more energetic, and less hungry—all because your cells can access the energy they need from your fat stores. As you can imagine, good metabolic flexibility makes it easier to eat less and avoid snacking.



To be metabolically flexible—and therefore able to burn fat—is to be fat-adapted (see Chapter 4).

With metabolic *in*flexibility, on the other hand, you will more quickly tip into burning glucose—even with light activity and maybe even at rest. Once that happens, you could feel fatigued and experience drops in blood sugar (hypoglycemia; see Chapter 4). You could also experience a faster decline in glycogen stores and a strong need to eat carbohydrates just to keep your energy up.

So, with metabolic inflexibility, you could feel duller, less energetic, and more hungry—all because your cells don't have the energy they need. Metabolic inflexibility can make it difficult (almost impossible) to eat less and avoid snacking.



Signs of metabolic inflexibility include fatigue and a tendency to low blood sugar or intense hunger, especially with exercise or fasting. We'll look at other signs in Chapter 5.

This book is about achieving better metabolic flexibility, or what Professor Grant Schofield from the Auckland University of Technology calls “metabolic capability.” You can do this by supporting various parts of physiology, including:

- healthy digestion and liver
- healthy muscles
- happy and healthy mitochondria—the powerhouses of cells that turn glucose and/or fat into usable energy
- appropriate levels of metabolic hormones, especially insulin
- balance within what I call the metabolic nervous system, the unconscious parts of the nervous system that control hunger and metabolism.

Energy balance is not under conscious control

The word “unconscious” is important because there's growing evidence^[1] that both eating behavior and energy expenditure are, to a large extent, not under conscious control. Which, if you think about it, changes the entire conversation.

The old conversation was to “eat less and move more,” based on the outdated assumption that both energy in (eating) and energy out (energy expenditure) are mostly conscious—as in, we consciously eat more and consciously move less. In that “energy balance” model, conscious behavior leads to fat gain, which, in turn, leads to metabolic dysfunction (including prediabetes) and eventually to negative health outcomes like heart disease. It’s the old willpower model.

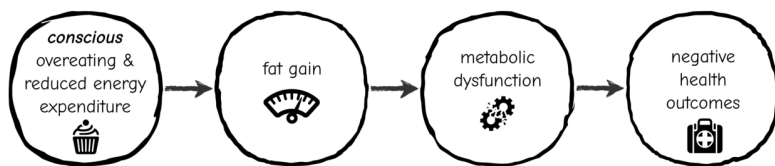


image 1 – The old willpower model

The new model says that metabolic dysfunction can (and often does) come first and leads to unconsciously increased eating and unconsciously reduced energy expenditure. And that, in turn, leads to both fat gain and negative health outcomes. It’s the new metabolic dysfunction model.

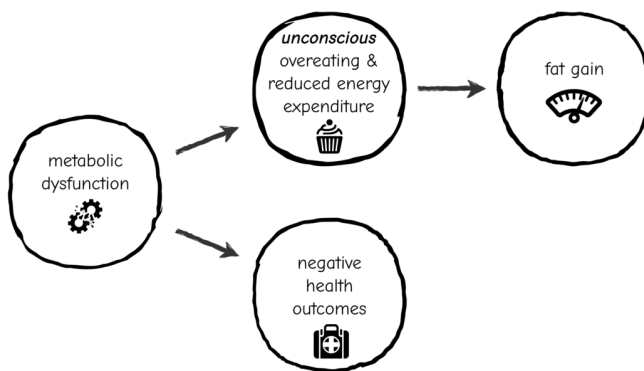


image 2 – The new metabolic dysfunction model

To be fair, once there’s a lot of fat, particularly inflamed fat (see Chapter 4), it can feed back to promote even more metabolic dysfunction.

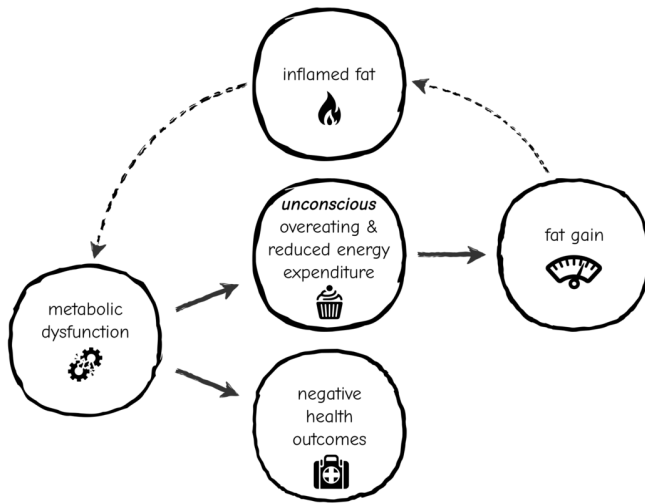


image 3 – Inflamed fat causes more metabolic dysfunction

We'll come back to that. For now, just remember that with the new model, metabolic dysfunction is the cause, and fat gain is the *symptom*. With the new model, weight has little to do with willpower.

Eating behavior is mostly not under conscious control

Now, you might be thinking, “But I can control how much I eat”—and sure, you can to some extent, but only some of the time. We consciously control how much we eat just as we consciously control how much we breathe: some of the time but not all of the time. We eventually need to *just breathe*, just as we eventually need to *just eat*. Hunger always wins.

“The [narrative] that obesity is caused by eating too much and exercising too little ... is nonsense. [Conscious overeating] is not the explanation because all ... creatures on Earth, including humans, eat when they're hungry and stop when they are full. Every cell in the body knows if you have enough food. Something has disrupted that normal sensing apparatus.”

Professor Barbara Corkey, past president of the Obesity Society^[2]

To be clear, it's not just *you* who eats unconsciously—it's everyone because hunger is an unconscious drive regulated by the metabolic nervous system.

Treating metabolic health according to the old model—as merely a problem with conscious calories in versus conscious calories out—would be like treating asthma as merely a problem of conscious air in versus conscious air out. We wouldn't cruelly instruct an asthmatic to “just breathe better,” so why should we instruct people to “just eat less”? Clearly, in both cases, it would be so much more logical to treat the underlying mechanism or problem. In the case of asthma, the underlying problem is constricted airways. In the case of fat gain, it's abnormally increased hunger downstream from various “metabolic obstacles,” as we'll explore.

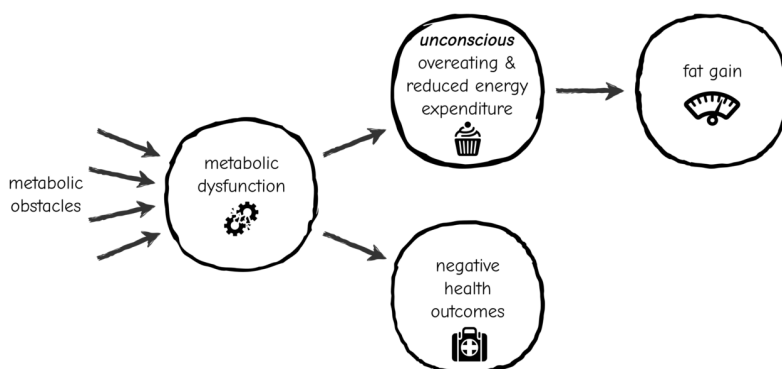


image 4 – Metabolic obstacles create metabolic dysfunction

When my patient Renee learned she was abnormally hungry because of underlying issues with her gut and sugary food, she remarked how “nice it was to get an explanation that isn’t just that I’m fat.” Because she had thought—as many people do—that being fat was the *cause* of her problems when it was actually the symptom.

Energy expenditure is also not under conscious control

Again, you might be thinking, “But I can control how much energy I expend based on how much exercise I do.” And again, sure, you can ... to some extent. But energy expended with exercise is only a small portion of energy expenditure. The larger portion comes

from four unconscious mechanisms:

1. **basal energy expenditure** (also called basal metabolic rate or BMR)—the energy required for basic body functions. It accounts for about 70 percent of total energy expenditure. Importantly, BMR can differ significantly from person to person. There's even evidence (see Chapter 2) that average human BMR has declined in recent generations, which is troubling.
2. **diet-induced thermogenesis** (also called the thermic effect of food; thermogenesis means heat production)—the temporarily increased energy expenditure required to digest, absorb, and metabolize food. It's highest with protein and can stay high for about six hours after a meal.
3. **non-exercise activity thermogenesis** or NEAT—all spontaneous, day-to-day movement, including unintentional movement like fidgeting or pacing around the house. With a healthy metabolism, NEAT increases as a compensatory (energy-burning) mechanism after higher food intake and then stays high for days or even weeks. People with higher NEAT are less likely to store fat.
4. **brown fat thermogenesis**—the process by which a type of fat called brown fat (see Chapter 4) converts energy (from both food and body fat) directly to heat rather than to usable energy. Higher brown fat thermogenesis is another compensatory mechanism after higher food intake, especially after higher carb intake.^[3]

Basal energy expenditure is relatively constant but can change with hormones, medication, and other factors. In contrast, NEAT, diet-induced thermogenesis, and brown fat thermogenesis are actively compensatory, constantly increasing or decreasing in response to food, hormones, the nervous system, and much more.

You don't need to memorize the energy expenditure categories. Just know they're important and outside of conscious control. And if you're wondering how to boost NEAT and brown fat thermogenesis, some amount of boosting should be the natural result of a healthier metabolism.

Regulatory mechanism and “set point”

As we’ve just seen, neither hunger nor energy expenditure is under conscious control. Instead, they’re subject to unconscious physiological regulation, and that’s why—when everything’s working properly (and it might not be)—higher food intake one day should lead to:

- naturally wanting to eat less for several days after^[4]
- naturally, unconsciously burning more calories for several days (or even weeks) after.

Together, the compensatory mechanisms to eat less and burn more energy constitute a metabolic regulatory mechanism that resides mostly in the brain^[5] and is sometimes described as an adipostat or “fat thermostat.” According to set-point theory, the adipostat works to defend the body’s current amount of fat. So, if there’s an abnormal *increase* in fat, it’s because something is amiss with the regulatory mechanism. That’s why some researchers have reframed obesity as a neurological condition^[6] or “disorder of the brain.”^[7]

Clearly, if the problem is the regulatory mechanism in the brain, the solution is to work with the brain.

Working with the brain

As we’ll soon discover, there are many ways to work with the brain. Really, this entire book is about doing just that.

The first step is to understand that *all* parts of physiology (muscles, digestion, liver, nervous system, and more) send signals to the brain so it can make the executive decision about how hungry we should be and how much energy we should burn.



The neurons that regulate hunger are closely related to the neurons that regulate energy expenditure. That’s why naturally wanting to eat less and spontaneously burning more energy typically happen together.

The opposing signals are:

- the signals of safety and satiety that lower hunger, increase

- energy expenditure, and promote fat burning
- the signals of danger and hunger that increase hunger, lower energy expenditure, and promote fat storage.



satiety

Satiety is the physiological and psychological experience of not feeling hungry or needing to eat between meals. It's slightly different from satiation, which is the short-term feeling of fullness and desire to stop eating at the end of a meal.

Special Topic: Signals of safety

The phrase “signals of safety” comes from the work of neuroscientist Stephen Porges^[6] and refers to all the cues or signals that tell the body—specifically the autonomic nervous system (see Chapter 4)—that you're safe and okay. Signals of safety include things like positive social interactions, nourishing food, spending time outdoors, and getting enough rest and sleep.

Conversely, “signals of danger” tell the autonomic nervous system that you may not be safe or okay. Signals of danger include things like the absence of social interaction, unhealthy food, and not getting enough rest or sleep.

In this book, I've expanded Porges' signals of safety and danger to include signals of satiety and hunger.

Signals of safety and satiety assure the brain that all is well, so it's okay to burn fat; there's no reason to hoard it as a survival strategy. In contrast, signals of danger and hunger (metabolic obstacles) alert the brain to the fact that something is wrong or that winter is coming. And therefore, it's *not okay* to burn fat; instead, there's every reason to hoard it as a survival strategy. We'll explore this ancient, prepare-for-winter survival strategy in the next chapter and learn why scientists think the human brain has essentially been confused or hijacked by the modern diet. That hijacking is why weight gain is not your fault.

Remember, the solution is to send your brain as many signals of safety and satiety as possible, including movement, sleep, social

connection, and healthy, satiating food. By boosting such signals, you're essentially pulling levers to help your highly dynamic metabolism recover a healthier homeostasis (equilibrium) or set point.

You're working *with* your biology, not against it.



The new medical “brain-based therapy” is the GLP-1 and GIP agonist class of weight-loss medications (semaglutide or Ozempic). See Chapter 4 for a discussion of the hormones GLP-1 and GIP, and Chapters 12 and 13 for a discussion of the related medications, where I'll share the experiences of a couple of my patients.

The road home to metabolic health

“The road home is home.”

Rūmī

The advantage of working with your biology is that you'll feel good while doing it. As soon as you start to pull the appropriate health levers, you should immediately start to feel more satiety, energy, and zest. As long as that's happening, you're on the road home to metabolic health—even if you don't lose weight straight away. I call it “the road home” because home is what the body yearns for.

While you're on the road, please continue to live your life. That means spending time with loved ones and doing all the things that give you joy. Don't put those things off until some magical future day when you're skinnier and healthier because that's not how it works. Health is not a future where you will one day arrive. Instead, health is now, and living, connecting, and being joyful are essential parts of it.

“How we spend our days is how we spend our lives.”

Annie Dillard

But where do you start? What are *your* next best steps on the road home? That's where troubleshooting comes in.

A troubleshooting approach

Your next best steps could be entirely different from someone else's next best steps, so however much I'd like to offer a one-size-fits-all diet and exercise plan, I simply cannot. Instead, I can walk you through this ten-stage troubleshooting process:

1. In Chapter 4, we'll look at how a healthy metabolism should work.
2. Then, in Chapters 5 to 9, I'll describe various *metabolic obstacles*—all the possible signals of danger and hunger. For each obstacle, I'll indicate whether it's a high or medium priority and if it's easy or difficult to fix.
3. At that point, I suggest you pull out a notebook and list the metabolic obstacles that apply to you. You can expect to list four or five items and maybe more.
4. When you get to the end of Chapter 9, choose one or two obstacles to tackle first. I recommend starting with any that have the magic combination of high priority + easy to fix. They're the easy wins that can set you up for future wins. This is because pulling one lever (addressing one obstacle) can have the knock-on effect of automatically moving other levers (improving other obstacles).
5. Make a plan using your notebook. For example, you may decide to try my metabolic supplements combo (Chapter 7) or avoid foods that can cause sensitivities (Chapter 10)—two examples of high priority + easy-to-fix factors.
6. Implement your new strategy while you enjoy your life.
7. Persist for a reasonable amount of time—at least several weeks—and then assess. Do you feel improved satiety, energy, and zest?
8. If yes, then yay! Appreciate your gains, and either hold the course to maintain your progress or get ready to tackle the next obstacle and strategy, knowing that each step will get easier.
9. If you feel *no* improved satiety, energy, or zest, be a *curious observer* or scientist and think: “Oh, that's interesting. Why didn't that work? What did I miss?” For example, maybe you're low in iron (see Chapter 8), or you have something

going on with your thyroid gland (see Chapter 9). Or maybe you need more sheltering from sugar (see Chapter 7) than you realized.

10. Don't make the mistake of thinking, "Oh, so that failed," and throwing in the towel. See "You are not going to fail" in Chapter 2 and bookmark it so you can reread it whenever you need a reminder.

Make sure you bookmark this page, too, so you can quickly see your next step in the troubleshooting process.

Can metabolic troubleshooting really result in fat loss?

Yes, by addressing one or more of your metabolic obstacles, you can support your nervous system, improve insulin sensitivity, dial up satiety hormones, decrease hunger, and increase compensatory energy expenditure. In other words, you can correct your brain's regulatory mechanism.

That should lead to increased satiety and energy expenditure on an ongoing basis, which should, in turn, lead to an incidental, unintentional shift to eating less and burning more energy. And that should create an incidental calorie deficit that just happens, not one you set out to achieve.

This is a fundamentally different approach from trying for an intentional calorie deficit. If you prefer intentional calorie counting, this book is not for you. In my experience, calorie counting only works (if at all) for people who don't have metabolic inflexibility or a hijacked regulatory mechanism. They're the lucky few who can (or think they can) consciously control what they eat. The rest of us eat according to hunger, not numbers.



Remember, energy balance is not under conscious control.

What does troubleshooting look like in action?

To illustrate how different approaches work for different people, we'll explore the following patient stories:

- Keresa, who was exhausted and needed more sleep, iron, and morning protein.

- Viv, who found a way to cook even though she did not want to.
- Julie, who had hypoglycemia and a long road to recovery from thyroid disease, perimenopause, and thirty years of dieting.
- Bec, whose strong food cravings disappeared when she stopped consuming standard cow's dairy.
- Monique, who lost weight when she treated SIBO (small intestinal bacterial overgrowth; see Chapter 10) and got off stomach acid medication.
- Lailah, whose metabolic health improved when she treated IBS (irritable bowel syndrome).
- Priya, who needed thyroid treatment.
- Rhianna, who got dramatic results from treatment for PCOS (polycystic ovary syndrome), a common hormonal condition; see Chapter 9.
- Carmen, who escaped food addiction.
- Mae, who managed premenstrual hunger.

This book is for women

As you can gather from the patient stories, this book is for women. We need our own book because so much of the “general” nutrition advice is aimed at men, yet men are different. I phrase it that way because I like to center female physiology as the normal version of human physiology—and treat male as the variant. Which makes just as much sense as centering male physiology as normal.

How are men different?

- Men are more likely to achieve fat loss with intentional calorie restriction. That's because women, and especially young women, are—due to their biological reproductive needs—better at conserving energy and physiologically defending against fat loss.^[9]
- In contrast, men are more likely to develop two key elements of metabolic dysfunction—insulin resistance and hypertrophied (increased) visceral fat—which we'll learn about in Chapter 4. That's because they're missing the high

level of estrogen that (to some extent) shelters young women from those conditions.

- Men can generally cope better with stronger interventions like fasting or a very low-carbohydrate (“low-carb”) diet—thanks to their differently calibrated nervous and stress-response systems.
- Men don’t have to worry about losing a menstrual cycle to intense exercise, fasting, or restrictive diets.
- Men have a relatively unchanging—call it boring—physiology throughout their lifespan. In contrast, women have different modes of physiology, including phases of the menstrual cycle, possibly pregnancy and postpartum, perimenopause, and menopause.
- Men have a relatively unchanging day-to-day physiology compared to women’s monthly cycles. That said, men’s testosterone levels do fluctuate with quirky things, such as their sports team losing.
- And finally, men don’t have to contend with the side effects of hormonal birth control, including all the metabolic side effects and weight gain. (We’ll explore birth control and other aspects of female hormones in Chapter 11.)

Special Topic: What’s “wrong” with women?

My focus on “female as normal” started decades ago when I was a biologist researching sex differences. It then continued through all my years as a clinician and culminated in a comment left by a reader on a blog post I’d written about young women losing their periods to a low-carb diet. She said, “That makes so much sense. I always wondered what was wrong with women that they can’t do a low-carb diet.”

“What was *wrong* with women?” That gutted me and pretty much summed up the problem.

To be clear, nothing is wrong with women. We’re just fine. And, as we’ll see, some women *can* do a low-carb diet.

This book is for general health

“In what way is metabolic health different from just health?” my husband asked after months of hearing about metabolic health.

“They’re almost the same,” I had to admit.

By improving your metabolic health, you will provide all your cells with more energy. And that should translate into better digestive health, liver health, immune health, hormonal health, menstrual health, and mental health.

Mental health is a great example because it’s having a bit of a “metabolic moment,” as explored by Harvard psychiatrist and researcher Christopher M. Palmer in his book *Brain Energy: A Revolutionary Breakthrough in Understanding Mental Health*.^[10] The book presents the metabolic theory of mental illness, which states that “mental disorders—all of them—are metabolic disorders of the brain” and cites decades of research to build the case that most (maybe all) mental health problems are associated with (and possibly caused by) energy deficiency in the brain.

“Mental health is metabolic health,” Palmer says. The same could be said for digestive health, hormonal health, menstrual health, and much, much more.

Let’s go!

The main takeaway from this chapter is that metabolic dysfunction can affect many aspects of your life. Not only does it make it harder to lose weight, but it can potentially cause symptoms like fatigue and depression and increase your long-term risk of heart disease and dementia.

Importantly, if you have metabolic dysfunction, it’s because something is amiss with your brain’s regulatory mechanism. Exactly what caused it to go amiss is the topic of the next chapter.

Chapter 2

The great hijacking of your metabolic regulatory mechanism

About fifty years ago, many people in most countries started becoming hungrier, larger, and less metabolically healthy. That shift to metabolic dysfunction is a collective change that, scientifically, can only be interpreted as the result of something that changed with our environment and/or the environment of our recent ancestors. Why do I mention recent ancestors? Because worryingly, the negative metabolic changes induced by the environment seem to be passed on from one generation to the next. That means the epidemic of metabolic dysfunction is amplifying with each generation and, according to some calculations,^[11] may even have started more like seventy years ago.

The epigenetic switch for obesity

The process by which environment-induced changes are passed on is called epigenetics. This occurs when the expression of various genes, including obesity genes, is switched on or off in one

generation and then inherited by the next. That's why some scientists speak of an "epigenetic switch for obesity."^[12]



I've promised to avoid the word "obesity," and I generally will. I do need to use it occasionally to quote scientists speaking about "obesity genes" or the "obesity epidemic."

Many of the so-called obesity genes are genes that affect the brain, which makes sense, given how metabolic issues stem from problems with the brain's regulatory mechanism. According to Professor Giles Yeo, a geneticist who studies metabolic health, "The genetics of body weight is, by definition, the genetics of how our brain controls food."^[13]

If you're reading this book, it may be because you've inherited epigenetically switched-on obesity genes. There are two things to be aware of:

1. those same genes were beneficial for your ancestors, which we'll come to a minute
2. they're only a problem now because they're interacting with the modern obesogenic environment.

Obesogenic or obesogen refers to any chemical, medication, lifestyle factor, or food that stimulates fat gain in humans and other animals. Some scientists say obesogens have upset the body's "metabolic thermostat,"^[14] which is similar to the adipostat I mentioned in Chapter 1.

The shift to metabolic dysfunction has been underway for generations, so your current metabolic problems are likely to have started when you were a child or maybe even before you were born. That's why I was so insistent in Chapter 1 that *it's not your fault*. You can stop feeling ashamed about being hungrier and/or bigger than your friends. They simply were not as vulnerable to the modern environment as you and your ancestors have been.



See Chapter 3 for more about clearing away the shame.

The obesogenic environment

Exactly what is it about the modern environment that is obesogenic? How does it override, bypass or, in the words of one scientist, “completely break”^[15] the metabolic regulatory mechanism? To get an idea, we need to think about how ancient metabolism is and, therefore, how vulnerable it is to modern hijacking.

We inherited our human metabolism from ancestors hundreds of thousands of years ago. They lived under almost constant threat of starvation, so evolved a regulatory mechanism that is very good at sensing impending scarcity and preparing for it by increasing hunger, reducing energy expenditure, and boosting fat storage. In other words, as humans, we evolved to be *very* good at storing fat.

The survival switch and fructose

Doctor and medical researcher Professor Richard Johnson explores this ancient “fat-storing” mechanism in his book *Nature Wants Us to Be Fat: The Surprising Science Behind Why We Gain Weight*^[16] and a 2023 scientific paper titled “The fructose survival hypothesis as a mechanism for unifying the various obesity hypotheses.”^[17] In both, he outlines what he calls the “survival switch” or “fructose survival hypothesis,” which proposes that a hunger-inducing, energy-conserving, fat-storing mode of physiology gets switched on by signals of impending danger or scarcity such as stress, dehydration or the ingestion of certain nutrients (including glutamate and fructose) that were traditionally higher in autumn—just as *winter was coming*.

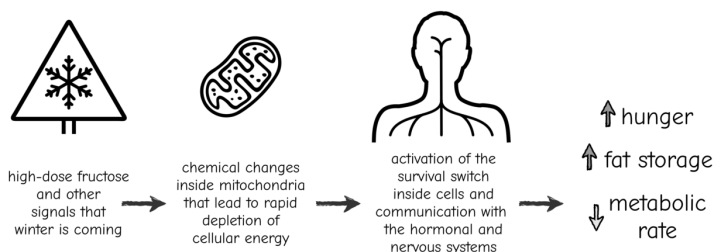


image 5 – Johnson’s fat-storing survival switch

“Fructose is what triggers our metabolism to go into low power mode and lose control of appetite,” Johnson explained in an interview^[18] “but fatty foods become the major source of calories that drive weight gain.” He posits that in some people, modern exposure to high-dose fructose (such as from sugar-sweetened beverages) essentially acts as a physiological signal or drug to stimulate fatigue, fat storage, and abnormally increased hunger. And that, in turn, can lead to unconscious overeating of all types of foods, especially high-calorie fatty foods. We’ll look more at fructose in Chapter 7, including some of the controversy around it. For now, just be aware that a big part of Johnson’s “survival switch” is a shift to the metabolic state of insulin resistance.



insulin resistance

Insulin resistance is the condition of reduced sensitivity to the hormone insulin (see Chapter 4), leading to chronically elevated insulin. It’s also called hyperinsulinemia, metabolic syndrome, or prediabetes and is typically associated with (and to a large extent causes) abnormally increased hunger and fat gain.

Although the modern tendency to insulin resistance is bad, it used to be a superpower. “I love that insulin resistance has a functional reason,” said nutritionist and weight-loss coach Lucia Hawley when she interviewed Professor Johnson. “I found that very humanizing. It’s not that our bodies are doing something wrong.”^[19]

“[Insulin resistance] was adaptive in the short term,” said Johnson in another interview. “It was supposed to help with our foraging response” and make us hungrier. It’s only bad, he explains, “when you’re eating sugar all the time or making sugar all the time.”^[20]

By “making sugar,” Johnson is referring to how the body can make fructose from glucose via a metabolic process called the polyol pathway. Because of this, the survival switch can be activated by high blood sugar from any cause, including from a high intake of refined carbohydrates or carbohydrates with a high glycemic load (i.e., foods that have a high impact on glucose levels).

Of course, fructose and high glycemic load are not the only causes of insulin resistance (see Chapter 4). Nor are they the only obesogenic aspects of modern food, as we're about to discover.

The terrible modern food supply

There's something very wrong with the modern food environment, also called the modern food landscape. In the words of one expert, "The world is being slowly poisoned by the food supply."^[21] But what exactly is so wrong?

One intriguing possibility is that we've simply become severed from the traditional knowledge of what to eat. That's explored in a paper called "Human nutritional intelligence underestimated,"^[22] which builds the case that, unlike other animals, humans are not good at sensing (based on taste) when we've eaten sufficient nutrients. Instead of relying on taste, the paper argues, we became reliant on traditional knowledge like recipes, menus, and cuisine.

Sadly, we've mostly lost traditional cuisine, and that has left many of us untethered or unmoored from knowing what to eat.



The scientific community has landed on the Mediterranean diet as *the* traditional diet to return to, but *all* traditional cuisines are healthy, especially for the people who evolved to eat them.^[23]

How did we lose traditional cuisine? Well, as the famous adage says, "slowly, then all at once." It happened slowly during the 1950s to 1970s, when we started eating more prepared foods. Then, all at once in the 1980s, when we were bombarded by ultra-processed food—thanks, in large part, to the tobacco company Philip Morris, which acquired General Foods in 1985, Kraft Foods in 1988, and Nabisco Foods in 2000. The tobacco giant harnessed its expertise in designing and marketing cigarettes and channeled it into the designing and marketing of highly palatable, possibly addictive (see Chapter 12) and probably harmful food-like substances.



ultra-processed food

Ultra-processed food or highly processed food are food-like substances that are manufactured from extracts or synthesized ingredients according to industrial methods not available to a home cook.

Ultra-processed or highly processed food

Based on the NOVA classification system,^[24] ultra-processed foods typically contain a long list of ingredients, many of which only vaguely resemble food (e.g., modified starches, invert sugars, hydrolyzed protein isolates, and hydrogenated oils).

“Don’t eat anything your great-grandmother wouldn’t recognize as food. When you pick up that box of portable yogurt tubes or eat something with 15 ingredients you can’t pronounce, ask yourself, ‘What are those things doing there?’”

Michael Pollan^[25]

The official NOVA list includes:

- soft drinks
- sweet or savory packaged snacks
- confectionery
- breakfast “cereals,” “cereal” and “energy” bars
- margarine and vegetable oil spreads (even “olive oil” spreads)
- processed cheese
- “energy” drinks
- sugared milk drinks
- sugared “fruit” yogurts and “fruit” drinks
- sugared cocoa drinks
- “health” and “slimming” products, such as powdered or “fortified” meal and dish substitutes
- sausages, burgers, hot dogs, and other reconstituted meat products

- powdered and packaged “instant” soups, noodles, and desserts
- mass-produced packaged breads, buns, biscuits, pastries, cakes, and cake mixes.



See Chapter 13 for “Being realistic about bread and desserts.”

Unfortunately, the NOVA classification is regrettably broad and lumps (for example) unhealthy soft drinks together with healthy protein powder. That reminds me of when a patient told me she’d tried “coming off sugar and meat”—which, to me, meant simultaneously coming off one unhealthy thing (sugar) plus one healthy thing (meat). That weirdness has led some experts (including me) to somewhat question the utility of the NOVA ultra-processed classification.

That said, the science is compelling, including a famous 2019 randomized controlled trial by researcher Dr. Kevin Hall, who found that simply by being ultra-processed, otherwise healthy-seeming food (in terms of calories, energy density, macronutrients, sugar, sodium, and fiber) can cause people to spontaneously eat an additional 500 calories per day.^[26]



macronutrients

Macronutrients or macros are the nutrients protein, fat, and carbohydrates.

It’s not just that ultra-processed food is “delicious, and so creates hedonic overdrive,” Hall said in an interview, “where we enjoy eating more than we hate being full.”^[15] It’s also that ultra-processed food seems to reset our satiety mechanism. “Or bypass it,” Hall says. “Or perhaps just break it completely.”



Current estimates are that US adults obtain almost 60 percent of their calories from ultra-processed food.^[27]

Special Topic: Ultra-processing versus traditional processing

Ultra-processed foods are different from traditionally processed foods like bread, cheese, yogurt, and good-quality cured meat. The purpose of traditional processing was to make food safer and more nutritious. The purpose of ultra-processing is to produce an edible substance that has a longer shelf-life and can increase profits for the producer, often by using lower-quality, less nutrient-dense ingredients.

How do ultra-processed foods damage metabolism?

Several mechanisms have been proposed.

High energy density

Most ultra-processed food has more calories per gram than whole food—in large part because ultra-processed food is relatively lower in water and fiber. But as Kevin Hall showed in his 2019 study, that's not the whole story.

Hyperpalatability


Some ultra-processed food delivers a potent combination of sugar, salt, *and* fat, which is *not* something the human brain has evolved to deal with. (Our ancestors would have eaten either sugar from fruit or salt and fat from meat, never all three together.) Consuming sugar, salt, and fat together can light up the brain like a pinball machine, especially for people who are genetically vulnerable. (Some people are simply genetically less motivated by food, even by hyperpalatable food.) If you are genetically (or epigenetically) vulnerable to hyperpalatability, even a couple of weeks of exposure could rewire your brain's reward system to want more.^[28] That rewiring could create food addiction, which we'll look at in Chapter 12.

 ***reward system***

The reward system, also called the dopamine and opioid reward system, is a brain network that controls motivation, pleasure, and reinforcement. It is one of several parts of the metabolic nervous system that we'll meet in Chapter 4. Its primary neurotransmitters (messengers between nerve cells) are dopamine, opioids (i.e., endorphins), and endocannabinoids.

 ***endocannabinoids***

Endocannabinoids are signaling molecules that regulate hunger, mood, and metabolism.

 ***food addiction***

Food addiction is a controversial term that describes the compulsive and uncontrollable eating of certain foods (usually ultra-processed foods) despite negative consequences, with an inability to reduce consumption despite the desire to do so, and withdrawal symptoms when the foods are stopped. It's linked to the reward system.

Softness

Softness is a near-universal quality of ultra-processed food. It's the result of using industrially modified plant and animal ingredients that have been essentially pulverized or “pre-chewed” (as one expert put it) before being reconstituted. During pulverization, the fibrous cell walls of the original food are destroyed, resulting in what scientists call pre-digested or acellular starches and sugars. When acellular starch hits the digestive system, it fails to stimulate the normal release of satiety hormones and instead can promote the overgrowth of inflammatory bacteria.^[29]

“The problem is not what's *in* the food, but rather what's been *done to* the food.”

endocrinology professor Robert Lustig^[30]

Specific harmful ingredients

Ultra-processed foods are generally high in fructose, refined flour and starch, salt, food additives, and industrially processed vegetable oil.

We touched on high-dose fructose when we looked at Johnson's survival switch. We'll return to it in Chapter 7.

Ultra-refined flour and starch are different from traditional flour in that they're acellular (i.e., pulverized) and can therefore bypass satiety hormones. Ultra-refined flour and starch are also empty calories (i.e., a non-nutritious source of calories), devoid of fiber and nutrients, and deliver a high glycemic load, rapidly increasing blood sugar. Ultra-refined flour and starch can also cause intestinal permeability, an inflammatory player in metabolic dysfunction (see Chapter 4), and damage the gut microbiome.



microbiome or gut microbiome

The gut microbiome is the combined genetic material of the gut microbiota, which are the microorganisms (bacteria, viruses, and fungi) that live in the digestive tract (see Chapter 4).



Some scientists propose that damage to the microbiome (due to ultra-processed food, antibiotics, circadian misalignment, and other factors) is the biggest driver of metabolic dysfunction.^[31]

Too much salt can contribute to hyperpalatability and activate Johnson's survival switch. In contrast, a normal amount of salt—as part of a whole food diet—can *improve* satiety,^[32] while too little salt can drive insulin resistance.^[33]

The harmful food additives in ultra-processed food include artificial colors, artificial flavors, preservatives, and even some of the obesogenic chemicals we'll discuss a little later. Additives of particular concern are emulsifiers like carboxymethylcellulose and

polysorbate 80, which can disrupt the protective mucus lining of the gut and increase the risk of intestinal permeability.^[34]

Finally, ultra-processed food contains industrially processed vegetable oil, which is the collective term for processed soybean, corn, sunflower, and cottonseed oils, sometimes referred to as “seed oils.” Oils are “industrially processed” if they’ve been put through a series of refining, bleaching, and deodorizing procedures that result in trans fats (inflammatory types of synthetic fatty acids) and harmful free radicals. They can also contain pesticide residues. As we’ll explore in Chapter 7, even unprocessed soybean, corn, sunflower, and cottonseed oils are relatively high in omega-6 fatty acids, which (depending on who you ask) could be another driver of inflammation and insulin resistance.

Lack of beneficial ingredients

And then we come to what’s missing from ultra-processed food:

- It’s generally devoid of fiber and prebiotics—non-digestible food components that selectively promote the growth and activity of beneficial gut bacteria.
- It’s low in micronutrients (vitamins and minerals), which is why it can cause malnutrition even as it causes weight gain.^[35] Deficiencies in potassium, calcium, selenium, magnesium, and zinc have specifically been linked to impaired satiety, increased hunger, and metabolic dysfunction.^[36]
- It’s often spectacularly low in protein, which, as we’ll see, is a crucial nutrient for satiety. Some researchers even propose the absence of protein as the primary mechanism by which ultra-processed food damages metabolic health.^[37]

Non-food aspects of the obesogenic environment

It’s not just about food. Non-food obesogenic aspects of the modern world include disrupted circadian rhythms, impaired sleep, reduced physical activity, obesogenic chemicals, and obesogenic medications.

Disrupted circadian rhythm

Every part of the body—including the mitochondria and microbiome—operates on a twenty-four-hour clock. That’s circadian rhythm, which we’ll explore in Chapter 6. For now, just know that any kind of circadian misalignment can profoundly damage metabolic health.^[38] It can also impair sleep.

Impaired sleep

Insufficient sleep can contribute to insulin resistance,^[39] but whether it’s a significant factor in the modern shift to metabolic dysfunction is less clear. And although it may *feel* like we sleep less in the modern world, we may actually sleep *more* than modern-day forager people in Tanzania, Namibia, and Bolivia.^[40]

Reduced physical activity

Of course, the problem could be reduced physical activity. “Instead of walking to work,” said one scientist,^[41] “we take cars or trains; instead of manual labor in factories, we use machines.”

That sounds logical, but we don’t actually *know* how active people used to be. We can try to infer based on data from modern-day forager people, who—surprisingly—seem to be as sedentary as people in the US and Australia.^[42] We can also try to infer based on past body temperature readings, which were higher,^[43] possibly because people used to be more active. Alternative explanations for higher past temperatures are that people used to have more infections (and therefore more fevers) or (according to one 2023 study^[44]) that people used to have a higher basal metabolic rate, which is puzzling. If metabolic rate has declined (which is not yet definitively proven), scientists think it could be the result of vegetable oil (see Chapter 7) and/or exposure to temperature-lowering, metabolism-slowing obesogenic chemicals and medication.

Obesogenic chemicals

According to a 2022 series of review papers (citing 1400 studies),^[45] there are at least fifty common household chemicals that are

obesogenic. That includes BPA (bisphenol A), phthalates, pesticides, flame retardants, dioxins, PCBs (polychlorinated biphenyls), and the very worrying PFAS (per- and polyfluoroalkyl substances) compounds, also called “forever chemicals,” that are everywhere and don’t break down or leave the body. (Ultra-processed food contains many of those chemicals as additives.)

Mechanisms by which obesogenic chemicals or additives cause weight gain include lowering basal metabolic rate,^[46] stimulating fat cell formation and fat deposition, impairing fat burning and increasing hunger. Exposure during early developmental windows (i.e., fetal development and childhood) is particularly harmful because it can have lifelong effects.

One expert estimates that 15–20 percent of the obesity epidemic can be directly attributed to obesogenic chemicals.^[47]

Obesogenic medications

Medications that increase the risk of insulin resistance and weight gain include:

- antihistamines for hay fever, sleep, and nausea
- PPIs (proton pump inhibitors)—medications that lower stomach acid
- some types of antidepressants
- antipsychotic and antiseizure medications
- beta-blockers—a type of blood pressure medication
- oral corticosteroids
- some types of diabetes medications
- statins—cholesterol-lowering medications
- antibiotics
- some types of hormonal birth control.

Many of those medications can cause weight gain while you’re taking them, and we’ll look at the mechanisms in Chapter 9. Additionally, exposure to them during fetal development or childhood can increase the risk of metabolic dysfunction later in life.^{[48][49]}

Special Topic: Is obesity a “disease”?

As experts start to recognize the reality of the hijacked brain and regulatory mechanism, they’re calling for obesity to be reframed as not a “problem with willpower” but instead as a treatable health condition or *disease*.^[50]

The word “disease” is tricky. On the one hand, “obesity as a disease” is more scientifically accurate than “obesity as a problem with willpower.” And if society considers obesity a disease, it may stop blaming people for it. On the other hand, the word “disease” can carry its own stigma, particularly when applied to body size, with no consideration of whether or not metabolic dysfunction is even present. As we’ll see in the next chapter, body size is not an accurate way to assess metabolic dysfunction.

A better plan would be to *stop trying to treat obesity at all* and shift the focus to treating metabolic dysfunction.

You are not broken

In a moving essay titled “I am not broken,”^[51] health coach Amy Eiges writes that “freedom is attainable—it’s on the other side of the mountain of garbage we’ve all been fed.” She tells of her lifelong struggle with obesity and food addiction and how she finally broke free—when she realized the problem was not her but the crazy, broken food environment she was living in. Amy explains how, for her, the road to freedom was not “everything in moderation” or “obsessively counting calories or points” but the entirely different strategy of abstaining from certain trigger foods—foods that make us feel out of control, maybe even physically unable to stop eating them. In other words, trigger foods are foods that do not create satiety but instead actively stimulate hunger. Trigger foods are usually ultra-processed foods.



Even the medical strategies of bariatric surgery (such as gastric band surgery) and GLP-1 agonist medication (e.g., semaglutide or Ozempic) work by reducing the hunger for and, in some cases, addiction to ultra-processed foods, especially high-carb ultra-processed foods.

Whether or not your story is like Amy's, she does highlight the ongoing tension between the popular refrain of "everything in moderation" and the fact that, for some people, the better strategy is to set boundaries or guardrails against trigger foods.

Special Topic: The different meanings of "trigger"

In the context of food addiction, "trigger" describes foods and situations that can make some people feel out of control. In the context of eating disorders, which we'll look at in the next chapter, trigger refers more broadly to any stimulus or situation that can evoke or exacerbate disordered eating. That could include any discussion of weight loss, dieting, or restricting foods. So, unfortunately, even a discussion of trigger foods could be a trigger if you have an eating disorder. Review the next chapter and then consult an appropriate clinician if needed. Depending on your diagnosis, some or much of this book may not be suitable for you.

We'll return to trigger foods and other drivers of abnormal hunger, but let's be clear from the outset: the goal is not to spend a lifetime exerting willpower to resist such foods. That would be impossible. Instead, the goal is to reach the point where you think, "Oh, my cravings are gone," or, "I'm not thinking about food all the time," or maybe, "I don't even want those foods!" If you can't yet imagine that outcome, it could be because you've tried many times and keep hearing that "most diets fail."

You are not going to fail

Statements like “most diets fail” and “it’s been proven that diets don’t work” are not helpful. Sure, they acknowledge the problem is the food environment and not you, but they overlook how much you can actually do. You are not helpless. You are not broken.

And yes, your “diet” will be part of the solution, but by diet, I mean the first dictionary definition of diet,^[52] which is:

“The kinds of food that a person, animal or community habitually eats.”

Not the second definition:

“A special course of food to which a person restricts themselves, either to lose weight or for medical reasons.”

As you recover your metabolic health, you are not “going on a diet” only to one day return to the “normal diet” of the modern food environment. Because the modern food environment *is not normal and never was*.

Instead, you’ll:

- engage in the exploratory process of finding the satiating, satisfying, and pleasurable way of eating that works for you
- focus on metabolic health rather than the number on the scale
- know how to troubleshoot metabolic obstacles such as digestion problems, food sensitivities, food addiction, and medications
- work with your biology rather than against it
- reach out for help when you need it.

That’s why *you are not going to fail*. In short, there is only going forward, not back. Even when you stumble (and you will), you’ll just pick up where you left off and keep going. Slowly but steadily, feeling better is the road home to metabolic health.

Women and diet culture

At this point, we need to acknowledge how we women have been bombarded by years of negative messaging around beauty and body shape. And how that can result in not only years of yo-yo dieting, suppressed metabolism, and rebound weight gain but also shame about our bodies.

Shame is the topic of the next chapter, including a discussion of Health at Every Size (HAES), intuitive eating, and disordered eating.

END OF SAMPLE

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