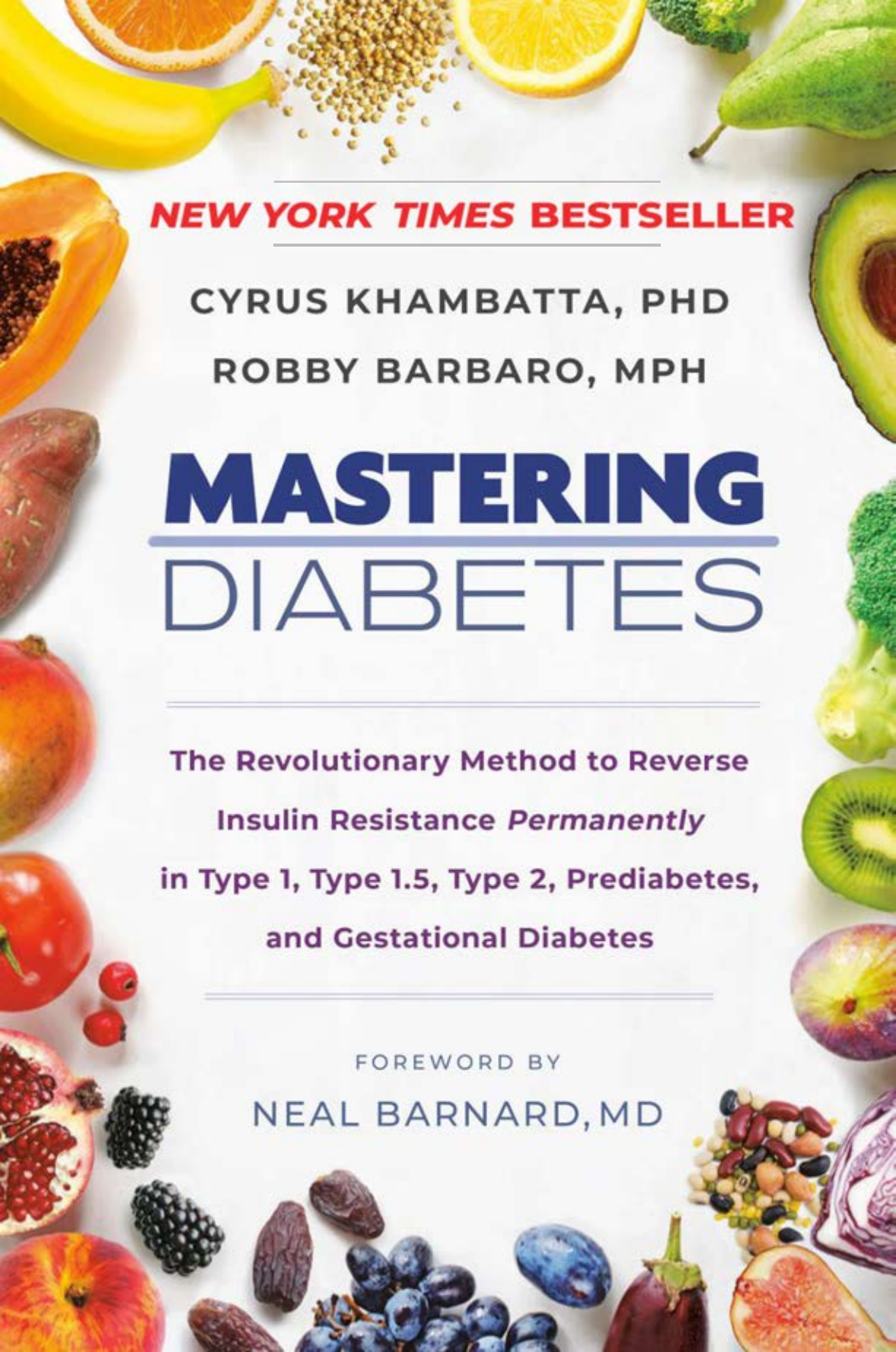


NEW YORK TIMES BESTSELLER

CYRUS KHAMBATTA, PHD

ROBBY BARBARO, MPH

MASTERING DIABETES

The Revolutionary Method to Reverse Insulin Resistance *Permanently* in Type 1, Type 1.5, Type 2, Prediabetes, and Gestational Diabetes

FOREWORD BY

NEAL BARNARD, MD

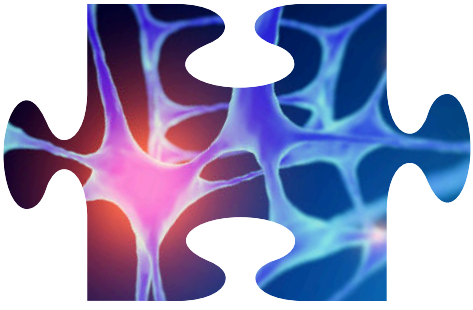
MASTERING DIABETES®

Welcome to the Missing Piece of Blood Sugar Control

If you've been living with high blood sugar, insulin resistance, or diabetes, you've likely been told the same message for years: avoid carbohydrates, take medication, and manage the condition for life.

But what if that advice is incomplete?

What if ***the real problem was never carbohydrates at all*** — but a deeper, biological issue happening inside your cells?



This guide exists to answer that question.

MASTERING DIABETES®

Proven in Science and in Real Life

What you're about to read is a **science-driven explanation of what truly causes insulin resistance**, and why addressing the root cause — rather than managing symptoms — is the key to long-term blood sugar freedom.

The principles in this guide are not based on trends or opinions. They are supported by **decades of scientific research** and validated by thousands of real people living with type 1, type 1.5 (LADA), type 2 diabetes, prediabetes, and insulin resistance.

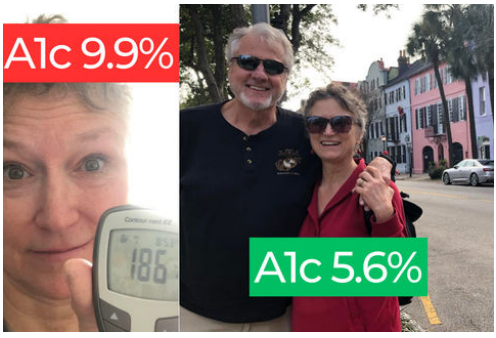
- People who once *feared* fruit, potatoes, rice, and beans...
- People whose fasting blood sugar *would not budge*...
- People who were told they would only get *worse* with time...

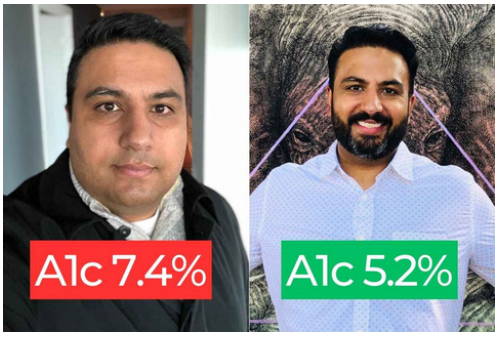
Many are **now experiencing the best blood sugar control of their lives** — often while eating more carbohydrates than ever before — because their insulin sensitivity has fundamentally improved.

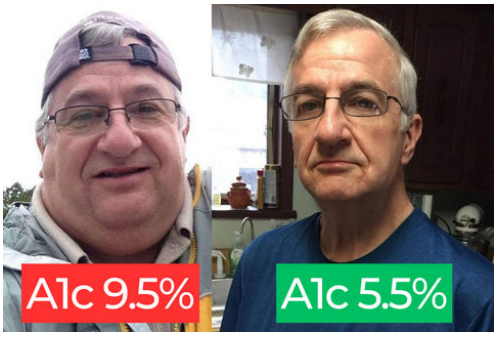
On the next page, you'll see **real before-and-after success stories** from people who applied this method and transformed their health.

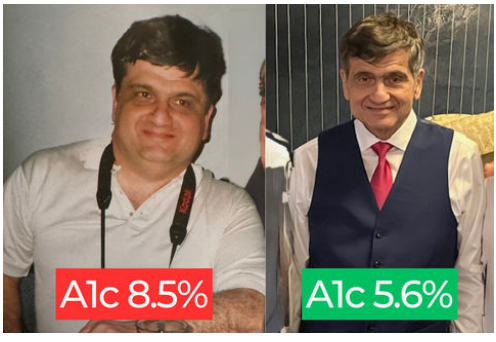
MASTERING DIABETES®

Real People, Real Transformations









MASTERING DIABETES®

What This Guide Will Do for You

Inside this Wellness Guide, you will:

- Learn the ***real root cause*** of insulin resistance
- Understand ***why improving insulin sensitivity*** changes everything
- See why managing blood sugar is different from ***reversing*** the pathophysiology
- Gain ***clarity***, ***confidence***, and a ***clear direction forward***

At the end of the guide, you'll also find **10 simple, whole-food recipes** designed to help you apply these principles immediately — meals that support insulin sensitivity without restriction or fear.

After that, we'll dive straight into the chapter that explains exactly how insulin resistance develops — and how it can be reversed.

You're not broken.

You're not failing.

You're about to learn what most people never get taught.

MASTERING DIABETES

The Revolutionary Method to Reverse Insulin Resistance *Permanently* in Type 1, Type 1.5, Type 2, Prediabetes, and Gestational Diabetes

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and Robby Barbaro, MPH

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Chapter 3

What *Really* Causes Insulin Resistance?

The Diabetes Lightbulb: Joaquin

During a routine doctor's visit in May of 2017, Joaquin was diagnosed with type 2 diabetes when a blood test revealed that his A1c was 7.1%. In the months prior, he had been experiencing severe headaches, nasal congestion, and nerve tingling in his toes, and after receiving this diagnosis, he realized that these symptoms were all connected.

He began searching online for information about type 2 diabetes and learned that the condition is caused by insulin resistance. After watching a video presentation given by Cyrus at the Torrance Memorial Medical Center, Joaquin immediately signed up to participate in the next Mastering Diabetes retreat, excited to eat large quantities of his favorite foods—mangoes, potatoes, rice, and beans. At the retreat, Joaquin began exercising every day, and when he returned home, he continued to exercise without equipment, using only his body weight as resistance. He learned as much as he could about the underlying causes of insulin resistance and considered himself very knowledgeable about why the Mastering Diabetes Method is an incredibly effective solution to reverse insulin resistance and maximize his long-term health.

In three months, Joaquin lost 35 pounds. And he no longer experienced daily headaches, nasal congestion, or tingling in his toes. His fasting blood glucose dropped below 100 mg/dL within the first week of following the Mastering Diabetes Method, and his energy levels began increasing quickly. For the first few months, he checked his blood glucose multiple times a day to confirm that type 2 diabetes was fading into the background, and after three months he stopped entirely. In six months, his A1c dropped from 7.1% to 5.6%, and Joaquin is now free of diabetes altogether. He calls this lifestyle a no-brainer and has successfully helped many family members and friends transition to the same lifestyle. "Don't overcomplicate this lifestyle," he says. "After one month you're going to thank yourself and see that it's well worth it."

There are countless misconceptions about what causes diabetes, and in today's internet-dominant era, it's becoming increasingly difficult to separate scientific truth from theory and opinion. Regardless of your understanding of diabetes physiology, we encourage you to keep an open mind when reading these next chapters. Some of these concepts might be difficult for you to wrap your head around at first, and you might even feel overwhelmed by the information in this book because it directly conflicts with what you have been taught in the past. And if that's the case, you'll feel exactly how we felt when we first learned it. Don't worry, the information in this chapter may seem counterintuitive, but rest assured that it's biologically accurate.

Without doubt, our most ardent followers are those who have struggled with the concept of low-fat plant-based whole-food nutrition from the beginning of their journey because it was so different from what their doctors recommended and from what they previously believed to be the truth. We remember feeling very apprehensive ourselves when we learned about the root causes of insulin resistance. But once we started to experience improved insulin sensitivity, there was no denying the power of this "unconventional" approach to nutrition.

We understand that changing old patterns can be very difficult, espe-

cially when it comes to your health. That's another reason why we strongly encourage you to understand the fundamental science of insulin resistance before making long-term lifestyle changes. As is true for many of our clients, understanding the science-based relationship between your blood glucose, your weight, your energy, and your level of insulin resistance is the key to implementing the Mastering Diabetes Method—and to seeing quick and long-lasting results. And that all starts with recognizing that what you may have learned about diabetes up to this point may in fact be leading you down the wrong path.

Your Doctor Didn't Learn Nutrition in Medical School

There is no arguing the fact that the overwhelming majority of doctors around the world are extremely well intentioned and have a genuine desire to improve the quality of life of their patients. The fundamental problem is that doctors have been trained in a system that is designed to treat symptoms using pharmaceutical and surgical intervention instead of addressing the root cause of chronic disease. In the same way that a carpenter selects the right tool for the job, a doctor selects the right tool for a given medical condition. The problem is that the main tools that doctors are trained to use are pharmaceutical medications, not food. The result is that doctors treat diabetes and its complications using medications rather than evidence-based nutrition.

It's not your doctor's fault. Medical schools barely scratch the surface when it comes to providing nutrition-related education. A 2010 survey revealed that only 25 percent of medical schools in the United States require their students to take a nutrition course as part of their medical curriculum, resulting in an average of less than 20 hours of nutrition education total, as compared to more than 10,000 hours of medical and clinical education. Even worse, less than 30 percent of the schools met the minimum requirement of 25 hours of nutrition education, a standard set by the National Academy of Sciences.

Given that nutrition education is only a minor focus in medical schools,

it is unrealistic to believe that physicians can effectively recognize or treat the root causes of insulin resistance, obesity, diabetes, and cardiovascular disease through diet. More specifically, physicians are not trained on the power and efficacy of a *low-fat diet* in reversing insulin resistance and improving insulin sensitivity, and are just as susceptible to fad diets and catchy marketing as is the general public. As a result, people living with prediabetes and type 2 diabetes are rarely presented with evidence-based information, and instead are encouraged to focus only on short-term methods to control their blood glucose using either a low-carbohydrate diet or a very low-carbohydrate ketogenic diet. Because doctors are insufficiently educated about nutrition in medical school, we believe that the solution to reversing insulin resistance lies in **your** hands.

The Carbohydrate-Centric Diabetes Model

If you're like most people with diabetes or prediabetes, you may have been encouraged to eat a low-carbohydrate diet to control your blood glucose, or you may have been told that carbohydrate-rich foods will increase your risk for diabetes complications and worsen your long-term health. Whether you open *The New York Times,* scan the bookshelves at your local bookstore, or search online for diabetes-related advice, the prevailing wisdom in the world of diabetes nutrition is that a low-carbohydrate diet is the safest and most effective way to control your blood glucose and will help you lose weight, reduce your fasting blood glucose, drop your A1c, lower your cholesterol, and reduce your blood pressure. Even at scientific conferences you'll see some of the most accomplished professors pointing a finger at foods like bananas and rice, showing detailed scientific evidence that all foods containing carbohydrates act the same way in your body, causing uncontrollable blood glucose fluctuations that then lead to increased appetite, weight gain, and obesity.

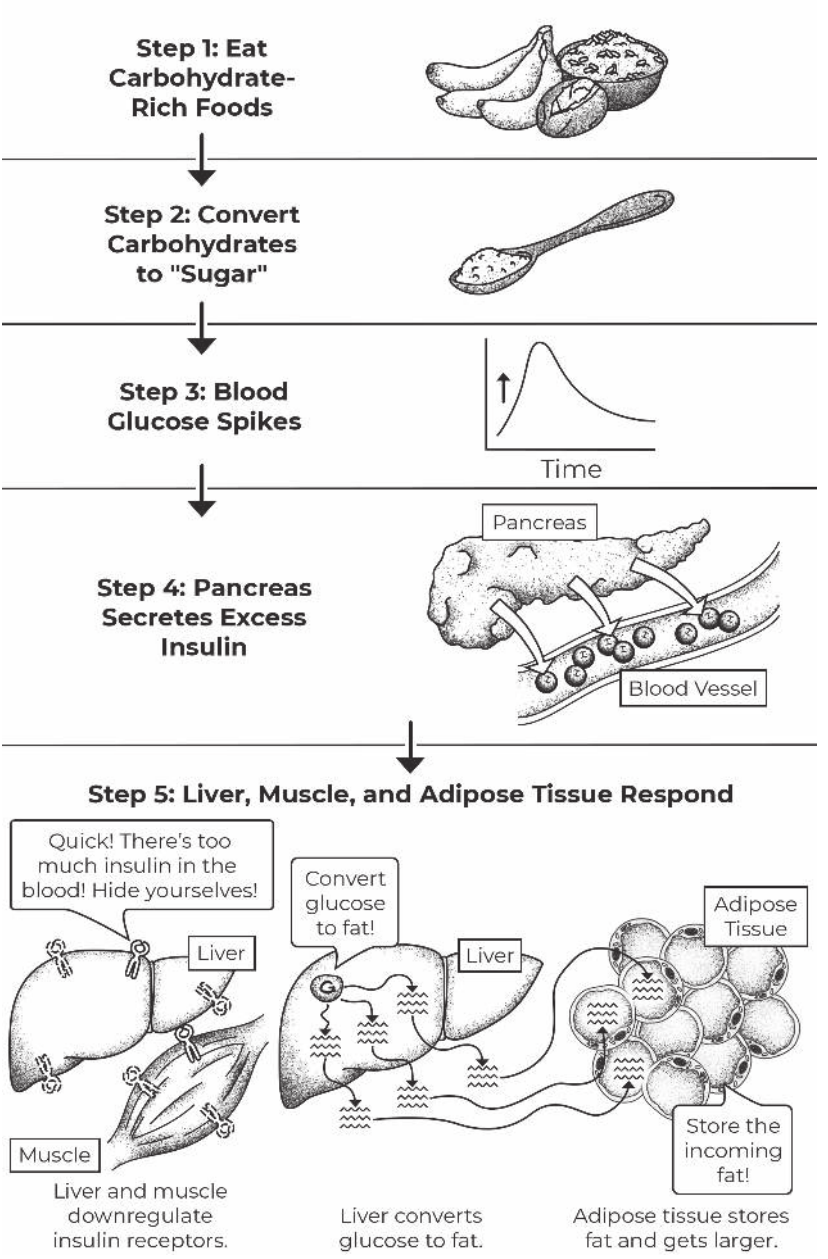
The carbohydrate-centric diabetes model states that when you eat any foods containing a sufficient quantity of carbohydrate energy (regardless of whether they are processed or whole foods), the carbohydrate energy turns into "sugar." In turn, these "sugar" molecules rapidly increase your blood

glucose, which then triggers your pancreas to secrete large amounts of insulin. As a result of large surges of insulin in your blood, tissues downregulate their insulin receptors, which creates a state of insulin resistance in which "sugar" can't get out of your blood. In addition, since insulin is your "fat-storage hormone," large insulin spikes increase the amount of fat you store. Your liver then converts large amounts of "sugar" to fat, and this causes you to gain weight, which in turn makes you more insulin resistant. The model looks like the illustration on the next page.

In effect, *the carbohydrate-centric diabetes model says that excess insulin causes insulin resistance.* It points a finger at insulin as being the cause of insulin resistance, arguing that insulin is your "fat-storage hormone" and that it is responsible for unwanted weight gain. According to this model, the solution to diabetes is to eat foods that minimize or eliminate your need for insulin, which includes foods that are low in carbohydrate energy, high in fat, and high in protein. Following this logic, carbohydrate-rich foods are to blame for high insulin levels, and high insulin levels are to blame for insulin resistance and weight gain.

In the subsequent chapters of this book, we will demonstrate with overwhelming evidence that the carbohydrate-centric model of diabetes is more than just wrong; it is an overly simplified attempt at explaining diabetes using a single variable. It is a great example of flawed logic that focuses on only one component of your diet (carbohydrates) and teaches you how to eat foods high in fat and protein to minimize your insulin needs in the short term, with no regard for almost a hundred years of scientific evidence that demonstrates the long-term chronic disease risks of eating foods high in fat and protein.

The reason why millions of people around the world believe that the carbohydrate-centric diabetes model is correct is because carbohydrate-rich foods will spike your blood glucose *if and only if your baseline level of insulin resistance is high to begin with.* As you will learn in the subsequent chapters, insulin resistance makes it extremely challenging to eat carbohydrate-rich foods without experiencing high blood glucose, which means that if you are already insulin resistant, then you are likely to experience high blood glucose any time you eat carbohydrate-rich foods. So the question then becomes . . . *is insulin resistance actually caused by something other than insulin?*



The carbohydrate-centric diabetes model argues that carbohydrates turn into sugar, which then spikes your blood glucose and triggers the production of large amounts of insulin. As a result, your muscle, liver, and adipose tissue become resistant to insulin, in an effort to protect themselves against excess insulin.

The truth is that insulin resistance is not caused by insulin at all—it's caused by something entirely different that creates the *symptom* of excess insulin in your blood. Pointing a finger at insulin as the cause of insulin resistance is akin to pointing a finger at a sore throat as the cause of the common cold. You probably know that a sore throat is not the *cause* of a common cold; it is simply one of many *symptoms* that can make your life temporarily uncomfortable. Sure, treating the symptoms of your cold will help you feel better in the short term, but it does nothing to improve your immunity in the long term. In the same way, treating the symptom of insulin resistance (high blood glucose and excess insulin) rather than treating the cause (a high-fat diet causing insulin resistance) works in the short term but fails to minimize your risk for many chronic diseases and worsening diabetes health in the long term.

In this book you'll learn the exact method for eating significantly larger quantities of carbohydrate-rich foods by first increasing your *carbohydrate tolerance.* By doing so, you're also likely to experience normal blood glucose, reach your ideal weight, lower your cholesterol, normalize your blood pressure, prevent and reverse heart disease, and much, much more. As long as you are living with insulin resistance, your ability to metabolize carbohydrate-rich foods will be low, which is exactly why increasing your level of insulin sensitivity is the most effective way to metabolize carbohydrate-rich foods and avoid high blood glucose and excess insulin.

By increasing your insulin sensitivity, you will be able to eat carbohydrate-rich foods and experience excellent blood glucose control while simultaneously decreasing your risk for weight gain, obesity, high cholesterol, high blood pressure, and coronary artery disease.

There are a few reasons that the prevailing wisdom in the world of diabetes has an overwhelmingly pessimistic view of foods containing carbohydrates. We ourselves were subjected to the following mistakes when we were first diagnosed with type 1 diabetes:

Mistake 1: Scientists, medical professionals, and people with diabetes erroneously use the word *sugar* when referring to both added sweeteners

in processed foods as well as the natural energy contained in whole foods like fruits and starchy vegetables, reinforcing the concept that anything that creates more "sugar" in your body will negatively impact your health. The word *sugar* is misleading and fails to describe the fundamental difference between refined sweeteners and naturally occurring carbohydrate energy found in whole food.

Mistake 2: Many people believe that insulin is one of the most dangerous hormones in your body, and that insulin is to blame for fat synthesis, weight gain, high cholesterol, and high blood glucose. Because of this, low-carbohydrate dieters eat foods high in fat and protein because they stimulate your beta cells to secrete small amounts of insulin, or require only small amounts of exogenous (injected) insulin for those living with insulin-dependent diabetes. The truth is that insulin itself is not to blame for increased chronic disease risk. *Excess insulin* beyond a physiologically normal amount increases your risk for many chronic diseases and should be avoided at all costs.

Mistake 3: Scientists, medical professionals, and people with diabetes often lump all "carbs" into the same category, and do not adequately differentiate between packaged and processed foods like breads, cereals, and pastas and whole foods like fruits, starchy vegetables, legumes, and intact whole grains. But carbohydrates in food come in many forms, and differentiating between refined carbohydrates and whole carbohydrates is extremely important when understanding their true biological impact.

Mistake 4: Most scientific studies that directly compare the outcomes of low-carbohydrate diets to low-fat diets do not design their "low-fat" diet properly. We thoroughly researched studies commonly used by the low-carbohydrate community to debunk low-fat diets and found that every single one of these publications do not study a truly low-fat diet. They compare very low-carbohydrate diets against diets containing 25 to 35 percent fat (and containing significant amounts of animal foods), and erroneously conclude that eating carbohydrates worsens your long-term health. In contrast, scientific investigations performed in people eating a truly low-fat diet containing less than

15 percent of total calories result in increased insulin sensitivity, reduced oral medication and insulin needs, reduced fasting and post-meal blood glucose, reduced A1c, and significantly reduced risk for cardiovascular disease.

The truth is that there isn't a single study that shows that carbohydrate-rich whole foods eaten in a truly low-fat environment doesn't improve insulin sensitivity. In addition, studies performed in subjects fed a highly processed carbohydrate-rich diet *in a low-fat environment* also demonstrate significantly improved insulin sensitivity.

A closer look at the complex interplay between whole-food nutrition and diabetes reveals something very simple and extremely important that can profoundly affect your diabetes health for years to come:

Your blood glucose is influenced by many variables, and is best understood when considering the amount and type of carbohydrate you eat, the amount and type of fat you eat, and, most important, the overall nutrient density of your diet.

Because the amount and type of fat you eat plays a central role in your ability to control your blood glucose, failing to fully understand this connection can dramatically alter your risk for the development of other chronic diseases into the future and is an oversight that both healthcare professionals and people with diabetes repeatedly make. In most cases, medical professionals instruct people with type 1 diabetes, type 1.5 diabetes, type 2 diabetes, prediabetes, and gestational diabetes to eat a low-carbohydrate diet from the time they are first diagnosed, sometimes telling patients that diabetes is a *carbohydrate metabolism disorder.* It's easy to believe this to be the truth, especially when most of the diabetes world preaches the same message. Unfortunately, this methodology creates two serious problems:

1. Patients often receive a recommendation to eat a low-carbohydrate diet when they are first diagnosed, at a time when they're emotionally vulnerable and willing to follow any advice that could minimize their physical and emotional discomfort.

2. Following the carbohydrate-centric diabetes model sets into motion a dietary pattern that can last for many years, resulting in a diet that increases your risk for chronic diseases in the long term. Worst of all, many people think that they're doing the right thing by eating a low-carbohydrate diet, completely unaware that they're actually eating themselves toward more insulin resistance and increased chronic disease risk.

Human Biology 101

Before going into how exactly insulin resistance works, let's first take a look at how a normal, healthy glucose metabolism functions—just in case you fell asleep during high school biology class.

When you eat food that contains carbohydrates, your body cuts long-chain carbohydrates into smaller pieces, and then eventually to chains that are 1, 2, or 3 *monosaccharide* pieces long, thanks to an enzyme called amylase that is secreted by your mouth and small intestine. These monosaccharide molecules end in *-ose* and include compounds like glucose, ribose, galactose, and fructose (just to name a few). Monosaccharides are commonly referred to as *sugar* in conventional textbooks, but we choose not to use the word *sugar* in this context, because the word *sugar* refers to a refined sweetener that you buy at the grocery store. For the remainder of this book, we will refer to natural sugars as *monosaccharides* and man-made sugars as *refined sugars,* because they have drastically different metabolic effects, and using them interchangeably causes unnecessary confusion. Don't worry; we'll revisit and expand on this concept in chapter 6.

When glucose enters your blood following a carbohydrate-rich meal, it first travels to your liver via a highway known as the portal vein. Your liver is given first crack at this fresh supply of glucose and can either take it up to burn immediately for energy, store it as *glycogen* (the stored form of glucose) for later use, or allow it to remain in your blood for other tissues to use. Your pancreas contains beta cells, a highly specialized group of cells found in clusters known as the islets of Langerhans that are the only cells in your body capable of manufacturing and secreting insulin. Beta cells release small

amounts of *first-phase* insulin to help your liver take up glucose from the portal vein, then later release *second-phase* insulin once larger amounts of glucose get into general circulation. Insulin is an extremely powerful hormone that acts as a key to unlock millions of cellular doors located on the surface of cells all over your body, in tissues ranging from your muscle and liver to smaller tissues like your gallbladder and prostate gland.

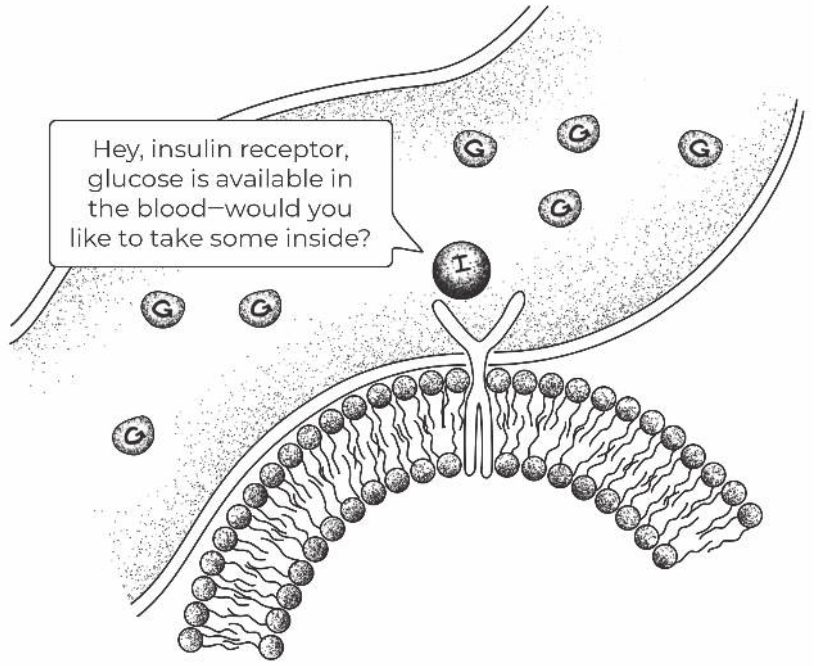
Contrary to popular belief, insulin is not your enemy. Many medical professionals and people living with diabetes erroneously point their finger at insulin, claiming that insulin will make you fat, increase your cholesterol level, and increase your risk for diabetes. However, a closer look reveals two very important aspects of insulin biology that are often misinterpreted:

a. Insulin is absolutely necessary for life. If insulin were not present in your body at all, you would die in a short period of time—likely within weeks or months.
b. Insulin itself does not raise your risk for chronic disease. *Excess insulin* beyond your normal physiological level is what causes severe metabolic dysfunction and increases your risk for many chronic diseases over the course of time.

Your dog secretes insulin. Your neighbor's cat secretes insulin. Your nondiabetic coworker secretes insulin. Insulin is an absolutely essential biological hormone that is secreted by beta cells in all mammals, a critical hormone that is mandatory for life. Without insulin, your dog would die. Your neighbor's cat would die. Your coworker would, yes, die. Without insulin, the cells in your liver, muscles, and fat tissue would have a very difficult time recognizing that glucose is present in your blood, and would be able to uptake only a tiny fraction of what was present in circulation, resulting in chronically high blood glucose at all times.

Think of insulin as an escort for glucose that tells cells in tissues all over your body, "Glucose is available in the blood—would you like to take some inside?"

When insulin knocks on the door of cells inside tissues all over your



Insulin receptors are located on the surfaces of cells in tissues all over your body. When they recognize insulin in your blood efficiently, cells are given the opportunity to import large amounts of glucose.

body, they are given the opportunity to import glucose from your blood *if and only if they have the ability to recognize insulin's presence.* You run into trouble when cells in your liver, muscles, pancreas, and adipose tissue (fat tissue) fail to recognize insulin's signal, which then prevents them from importing large amounts of glucose from your blood. (Your muscles and liver are the largest insulin-dependent tissues in your body, accounting for the majority of glucose absorption. Your brain also uptakes large amounts of glucose but requires only a fraction of the insulin that your muscles and liver require.) Since insulin's job is to escort glucose into tissues, when cells can't recognize insulin well, they *reject* it. When cells reject insulin, glucose gets trapped in your blood for extended periods of time, causing high blood glucose. To compensate, your pancreas releases more insulin, in the hope of overpowering these cells to import more glucose.

So what causes cells in your body to reject insulin in the first place? When cells reject insulin, they are "resistant" to insulin and said to be in an "insulin resistant" state. The causes of insulin resistance are numerous and relate specifically to the amount of food you eat, the type of food you eat, your movement patterns, the nutrient density of your diet, and your stress levels (to name a few). Think of insulin resistance as a puzzle that contains different-sized pieces in which the larger pieces have more of an impact than the smaller pieces. Without question, the largest and most important puzzle pieces are those that relate to the amount of food you eat, the type of food you eat, and your macronutrient ratio (the ratio of carbohydrates to fat to protein). Don't worry, we'll cover all of this in detail in upcoming chapters.

Even though people will tell you that carbohydrates are dangerous, understand that *carbohydrates are not the enemy,* especially if they originate from whole plant foods. Carbohydrates were never the enemy, and they will never be the enemy. On the contrary, fatty acids *directly* inhibit the action of insulin, and the amount of fat you eat and type of fat you eat are the primary determinants of how insulin resistant you become over the course of time. That's right. The more fat you eat (especially saturated fat), the more insulin resistant you become.

If you are like most people living with diabetes, chances are you were never taught the fat-insulin connection, and it may surprise you to learn that dietary fat is one of the most important aspects of your diet that influences how effectively insulin operates in your body. Let's go into detail about this connection so that you have a clearer understanding of the biology of insulin resistance.

An overwhelming amount of scientific evidence shows that a high-fat diet is the single most effective method at *inducing* insulin resistance in both your liver and muscle. These studies clearly demonstrate that increasing your fat intake has an immediate negative impact on the ability of insulin to communicate with tissues, which can then develop into a chronic state of insulin resistance and diabetes if your fat intake remains high.

This isn't new information.

Scientists have known that dietary fat makes insulin less powerful for

almost a hundred years. Starting in the early 1920s, researchers named Dr. William Sansum and Dr. J. Shirley Sweeney were some of the first to publish research about the fat-insulin connection. During the 1930s, Dr. I. M. Rabinowitch and Dr. Harold Percival Himsworth continued to conduct elegant experiments to demonstrate the detrimental effects of high-fat diets on insulin action. In the 1950s, Dr. Walter Kempner reversed type 2 diabetes in his patients using a diet low in dietary fat that was remarkably effective at also reversing long-standing retinopathy, kidney disease, and malignant hypertension. In the 1970s, Dr. James W. Anderson published experiments in which he reduced or eliminated the use of insulin in patients with type 2 diabetes by swtiching them to a low-fat, high-fiber diet. You'll have a chance to learn details about these studies (and more) in chapter 7, but for now suffice it to say that the fat-insulin connection is nothing new—it's just that it has been largely ignored for almost a hundred years.

One of the most important discoveries happened in 1963 when a scientist named Philip Randle described that carbohydrate and fat are mutually exclusive fuels, and that fatty acids and glucose compete for entry into cells. He demonstrated that fatty acids gain access to tissues and block insulin from working, leaving glucose trapped in your blood. Philip Randle told the scientific world that eating fatty acids sets the stage for insulin rejection in your muscle and liver. He called this effect the *fatty-acid syndrome,* which was later renamed the *Randle cycle.* More than fifty years later, this research is still considered one of the most profound observations in carbohydrate biology; however, the modern scientific world is quick to misinterpret this simple and very powerful insight. Let's start by defining the primary cause of insulin resistance as follows:

Insulin resistance is caused by the accumulation of excess fat in tissues that are not designed to store large quantities of fat.

In order to understand the fat-insulin connection in more detail, let's take a closer look at the effect of dietary fat inside your body.

A Step-by-Step Overview of the Fat-Insulin Connection

Step 1: Fat Enters Your Blood Before Glucose

When you eat foods containing fat-soluble nutrients (including triglycerides, phospholipids, and cholesterol), these fat-soluble nutrients travel down your esophagus until they reach your stomach. Your stomach releases hydrochloric acid (HCl) to kill potentially harmful bacteria, while the cells in your stomach lining secrete protein-digesting enzymes to begin unfolding protein chains. The smooth muscle in the walls of your stomach contracts vigorously in order to create a sludge known as *chyme,* which then exits your stomach bound for your small intestine, where the bulk of nutrients are absorbed.

In your small intestine, two important processes occur. First, cells in the lining of your small intestine absorb these fat-soluble nutrients and package them into particles called *chylomicrons,* which are then dumped into your lymphatic system, a collection of vessels that carry lymphatic fluid to the blood and clear waste compounds from tissues. When the lymphatic fluid ushers chylomicrons into your blood, they're circulated throughout your body so that your adipose tissue and muscle have the first chance to uptake triglycerides from your food.

When your small intestine detects fat-soluble nutrients in your food, it releases a collection of powerful hormones that communicate with your brain and stomach to control your appetite and slow your *gastric emptying rate,* or the rate at which food exits your stomach. In effect, your small intestine says, "Hey, stomach, slow down how quickly you process food. This fatty meal is going to take me some time to digest and absorb." A slowed gastric emptying rate results in an upstream traffic jam, slowing down the passage of chyme out of your stomach and into your small intestine. If you've ever noticed that a high-fat meal takes longer for your stomach to process than a lower fat meal, this is exactly why—because the presence of dietary fat slows the digestion of all food material, causing a temporary traffic jam in your stomach. This is one reason why diets high in fat are effective at curbing your appetite and making you feel full for long periods of time.

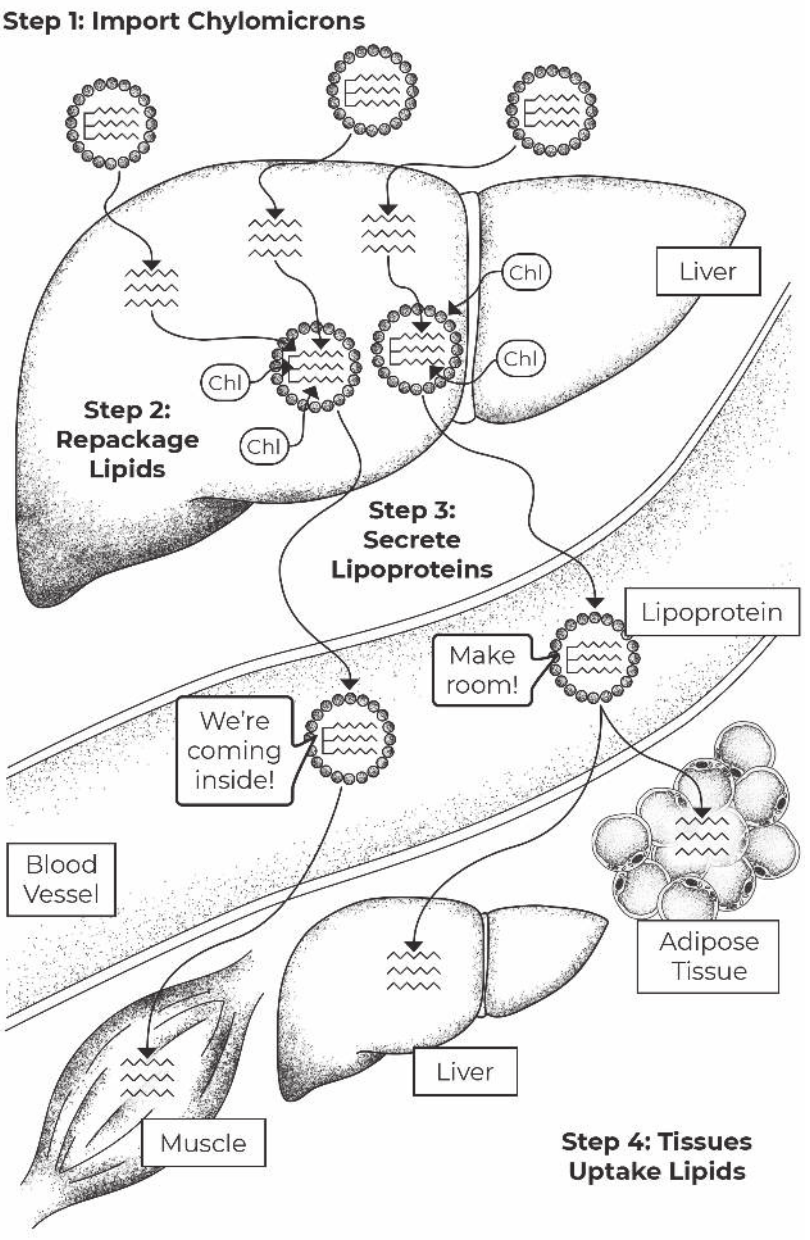
But back to your intestines. As a result of the simultaneous fat absorption into your lymphatic system and slowed gastric emptying rate, both carbohydrate and protein digestion are slowed. The net consequence is that fat-rich chylomicrons appear in your blood more rapidly than glucose (one of the building blocks of carbohydrates) and amino acids (the building blocks of protein).

Step 2: Fat Enters Your Blood and Tissues

When fat-rich chylomicron particles have circulated throughout your body and offloaded triglycerides, your liver then uptakes the remaining particles, called *chylomicron remnants.* A simple way to understand this is to think of your liver as the post office of fat metabolism. In the same way that the post office receives mail at the loading dock from large mail trucks, sorts it, and then sends it out on smaller trucks to local residents and businesses, chylomicron remnants appear at the loading docks of your liver, where they're unpacked, sorted, and repackaged. The triglyceride molecules are then transferred into lipoprotein particles and sent out in your blood once again.

With the help of enzymes located on the surface of cells throughout your body, tissues absorb fatty acids and cholesterol from these lipoproteins. Unlike glucose, fatty acids can enter cells easily without requiring an escort like insulin. In effect, the lipoprotein particles in circulation say, "Hey, tissues, I'm here! I have a bunch of fatty acids for you to take up if you want." While insulin can certainly help fatty acids get out of lipoproteins and into tissues, insulin is not fully required because fatty acids can also enter tissues without insulin. Because of this, tissues have no choice but to absorb fatty acids when they appear in your blood in large amounts.

As soon as the fatty acids are absorbed by cells in your liver and muscle tissue, they are either burned for energy or stored for later use. Those that are burned for energy are immediately ripped apart and transported to the mitochondria to be turned into ATP (the cellular equivalent of energy), and those that are stored enter a pool of fatty acids inside the cell, known as a lipid droplet. Fatty acids will continue to enter cells as long as they are present in your blood, and unfortunately cells don't have sophisticated mechanisms to block large amounts from entering. In effect, the more fat you eat, the more fat you force cells in your liver and muscle to absorb.



In the same way that the post office receives mail from large mail trucks, sorts it, and sends it back out on smaller trucks, your liver imports, repackages, and exports fatty acids at all times.

Step 3: Fat Enters Your Adipose Tissue

Unlike other tissues with a distinct location in your body such as your brain, heart, or lungs, your adipose tissue (also called fat) is located in many places. You can find small and large pockets of fat almost everywhere, including your abdomen, butt, thighs, lower back, chest, armpits, face, neck, and even ankles. Fatty acids enter your adipose tissue in the exact same way that they enter all other tissues—aided by enzymes known as *lipases* and *fatty acid transport proteins* (FATPs). Fatty acids are easily absorbed because your adipose tissue contains the perfect molecular machinery to uptake and store fatty acids for long periods of time; in fact that's exactly what your adipose tissue is designed to do.

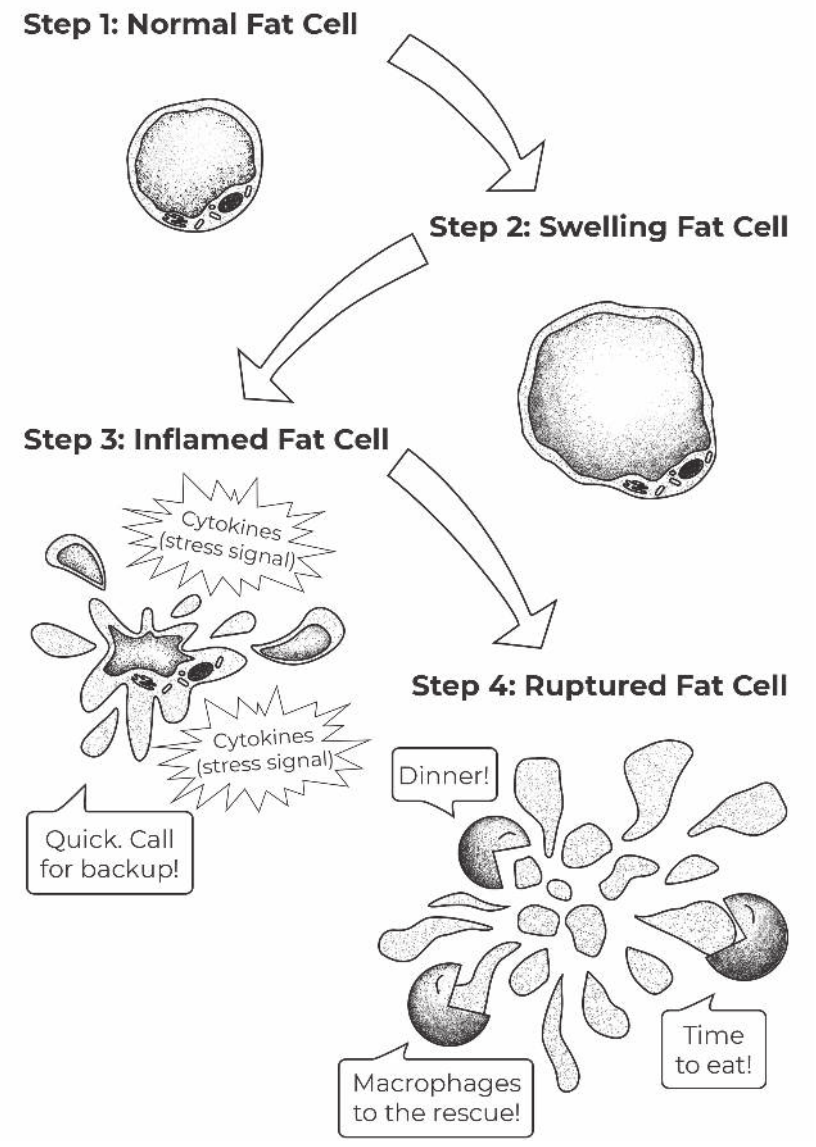
While most people want to minimize the amount of adipose tissue on their body, it's important to point out that adipose tissue is actually a *protective* organ. Your adipose tissue is designed to grow and shrink in response to times of feast and times of famine, storing energy at times when there are excess calories available to protect you for when calories are sparse. In addition, your adipose tissue also protects tissues like your liver and muscle from accumulating excess fatty acids by giving them another location to end up.

The reason most people and health professionals consider adipose tissue dangerous is that, much like insulin, *excess* fat increases your risk for many chronic diseases—especially the deep abdominal fat that surrounds your internal organs—because the more fat you store in your abdomen, the higher your risk for obesity, cardiovascular disease, hypertension, diabetes, and insulin resistance.

Step 4: Your Adipose Tissue Becomes Inflamed

The biology of adipose tissue insulin resistance is fascinating and deserves a thorough explanation. To begin, fat cells are incredibly flexible and are designed to store large quantities of fat, but they cannot expand indefinitely. As a result, when fat cells are chronically overfed, they can become inflamed just like any other tissue, resulting in a low-grade chronic inflammation that triggers insulin resistance.

When you eat a high-fat diet, cells in your adipose tissue accumulate fatty acids. Over time, cells in your adipose tissue can burst open, spilling their



Macrophages are cells in your blood that engulf cellular debris from damaged and broken cells. Cells in your adipose tissue can swell and break when chronically overfed, triggering a low-grade inflammation that reduces the ability of insulin to function properly.

contents into the interstitial fluid that surrounds the tissue. This explosion releases cellular debris into your blood, and neighboring cells release stress signals called *cytokines* into your blood, which then recruits cells called *macrophages* to invade the tissue in search of unwanted cellular debris from ruptured cells. Think of macrophages as the biological version of Pac-Man, tasked with the job of cleaning up cellular debris by engulfing waste material. And there's no job too big for macrophages—if there's too much debris, they can signal for backup by secreting more cytokines. The problem is that these signals don't just recruit more hungry macrophages; they also trigger chronic inflammation, and in the process create a state of adipose tissue insulin resistance.

Step 5: Fat Causes Insulin Rejection in Your Muscle and Liver

Even though your muscles and liver can generate ATP from glucose, fatty acids, and amino acids, your muscles and liver are designed to use glucose as their primary fuel. Both your muscle and liver cells have a similar construction—they can store small quantities of fat in lipid droplets and store glucose as glycogen. Your muscles and liver are specifically designed to remain "lean," with only small amounts of fatty acids in lipid droplets at all times.

But because these tissues absorb nutrients that appear in your blood, they remain unprotected against a large influx of fatty acids that occurs in the hours following a fat-rich meal. Since fatty acids appear before glucose during the digestion process, your muscles and liver are exposed to more fatty acids than glucose, setting the stage for insulin resistance when glucose becomes more readily available. When your muscles and liver uptake fatty acids from your blood, they also *upregulate* enzymes involved in all aspects of fatty acid metabolism and *downregulate* enzymes involved in all aspects of glucose metabolism—because fatty acids are now the predominant fuel.

When cells in your liver and muscles begin burning and storing fatty acids, just as Philip Randle described, they block glucose from entering because the cellular machinery required to uptake, process, and store glucose has been deprioritized. In effect, cells in your liver and muscle alter their internal enzymatic machinery based on the fuel that is most readily available.

How do these cells block glucose from entering? It's actually quite simple—they stop paying attention to insulin by downregulating insulin

receptors located on the cell surface. Within hours of a single high-fat meal, insulin receptors become less numerous and less functional, perform less work, and have a very difficult time recognizing insulin in your blood. These dysfunctional insulin receptors keep glucose outside of cells, leaving glucose trapped in your blood for long periods of time. When you check your blood glucose two to six hours after eating a meal containing predominantly fat and protein with a small or medium amount of carbohydrate energy, you may see a high number and ask yourself, "Why is my blood glucose high? I didn't eat that much carbohydrate."

This is exactly when insulin rejection first takes place. Unlike your adipose tissue, which is specifically designed to store fat to protect other organs from fat overload, your muscle and liver tissues are designed to store small amounts of fat and large amounts of glycogen. But when your liver and muscles first absorb fat from your blood following a fat-rich meal, they respond by rejecting insulin to block glucose from entering, because they have already accepted fatty acids as their primary energy source. First come, first served.

Excess Insulin Acts Like a Wrecking Ball

A simple way to understand insulin resistance is to imagine tissues constructing a brick wall to protect themselves against excess nutrients, and insulin as a wrecking ball specifically designed to knock it down. Under normal circumstances, insulin acts as a chaperone to help glucose enter tissues all over your body. Any time you eat a meal containing carbohydrates, insulin is *required* to get it inside of cells—namely, the other side of that brick wall.

In insulin-sensitive individuals, the brick wall is small, and therefore only small amounts of insulin are required to knock a hole in the wall and get glucose inside. In insulin-resistant individuals, the wall is high and thick, and in order to overpower the wall and get glucose inside cells, your pancreas is forced to secrete excess insulin. In effect, your pancreas says, "Hey, tissues, you need a larger wrecking ball? I'll just make more insulin!" As more and more insulin is secreted (or injected in the case of people with insulin-dependent diabetes), the strength of the wrecking ball increases until it's finally capable of breaking a hole in the wall. As soon as the insulin wrecking ball has

created a hole, glucose can then flood into tissues, and the amount of glucose in your blood drops rapidly.

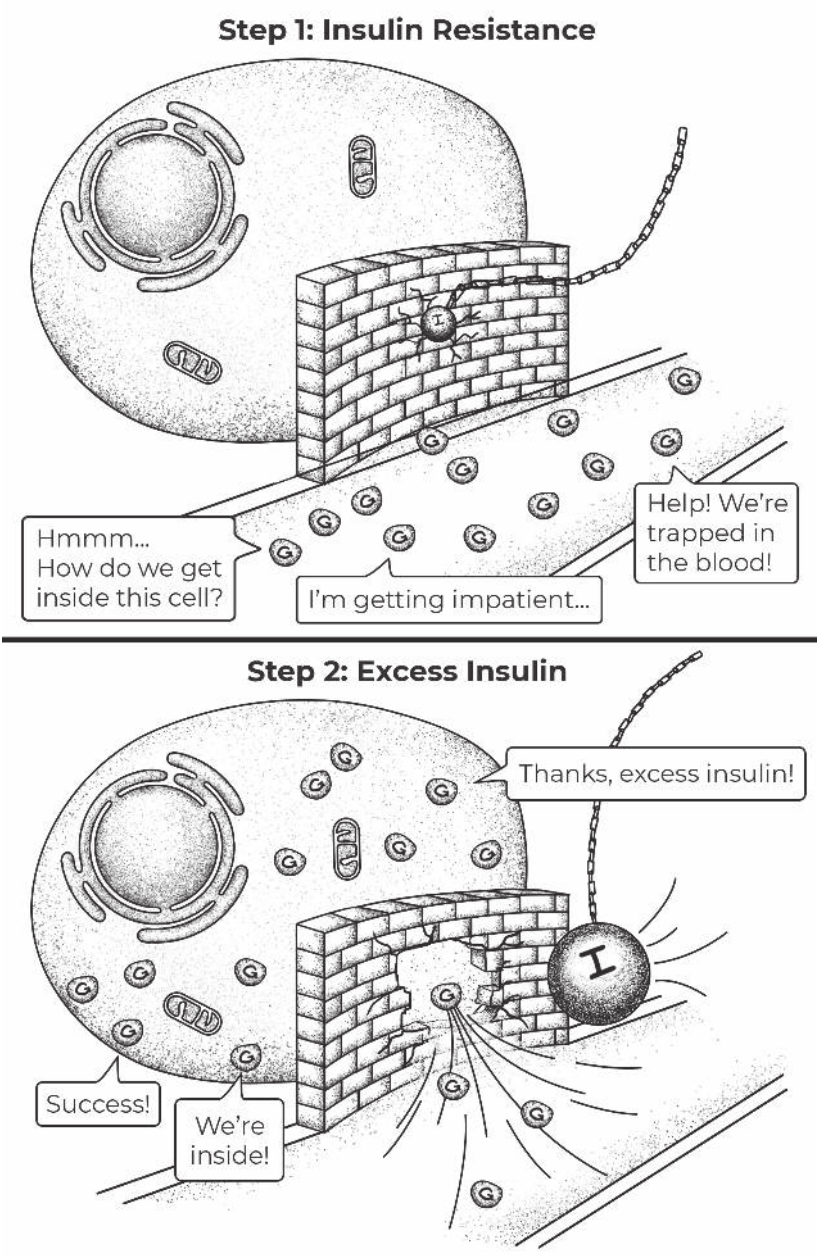
Ultimately, you have two options to allow glucose to enter tissues: (a) reduce the height and thickness of the brick wall by preventing excess fatty acid accumulation, or (b) rely on the excess insulin wrecking ball to knock the wall down. Technically speaking, both methods result in glucose entering tissues. However, the first method is the only one that minimizes your risk for long-term chronic disease, while the second method significantly increases your risk for cardiovascular mortality.

Take a look at the illustration on the next page for a visual representation of how excess insulin acts as a wrecking ball in response to the brick wall created by insulin resistance.

If we go back to the two options you have for encouraging glucose to enter cells in your muscle and liver, it probably goes without saying that we're fans of the first method: *Reduce the height and thickness of the brick wall by preventing excess fatty acid accumulation.* That's largely because eating a diet high in fat not only causes insulin resistance but increases your risk for many chronic diseases over the course of time.

One of the most common side effects of eating a high-fat diet is *fatty liver disease* (also known as non-alcoholic fatty liver disease, NAFLD). Fatty liver disease occurs when the amount of fat in your liver is more than 5 percent by weight, resulting in liver enlargement, which can then lead to *liver fibrosis,* a condition marked by the formation of scar tissue. Finally, *liver cirrhosis* occurs when connective tissue destroys dysfunctional liver cells. While it's certainly true that eating or drinking refined sugars like sucrose and high-fructose corn syrup (HFCS) can contribute to fatty liver disease, a growing body of research shows that a high-fat diet results in a progressive decline in liver function over time.

Your liver is a crucial part of the insulin resistance equation because cells in your liver are capable of exporting glucose into your blood to provide your brain with a constant supply of glucose at all times. And insulin is the signal that tells your liver exactly when to increase and when to decrease the amount of glucose it exports. When insulin is readily available following a meal, your



Insulin resistance functions like a brick wall, blocking the ability of insulin to communicate with cells, which traps glucose in your blood. Excess insulin acts like a wrecking ball, knocking a hole in the brick wall and allowing glucose to enter cells in tissues all over your body.

liver decreases the amount of glucose it exports. When insulin is less available after multiple hours of fasting, your liver increases the rate at which it exports glucose in order to drip feed your brain with a stable supply.

Here's the problem: When you develop insulin resistance in your liver due to the accumulation of excess dietary fat, your liver can't communicate with insulin very effectively, resulting in a chronically high rate of glucose export, high fasting blood glucose, and high post-meal blood glucose. In effect, an accumulation of excess fatty acids prevents your liver from accurately controlling how much glucose it releases into your blood.

Step 6: Beta Cells Get Stressed

Beta cells in your pancreas have one function: to manufacture and secrete insulin into your blood. They make up less than 1 percent of your total pancreas by weight, representing a very small population of cells. Your body has no backup mechanism for producing insulin, so when your beta cell function is compromised, it presents a *disastrous* metabolic problem to tissues all over your body. In the same way that your liver and muscles are highly sensitive to the accumulation of fat, so are beta cells. The accumulation of excess fat in your beta cells leads to severe dysfunction known as *lipotoxicity*.

In comparison with cells in your liver and muscle, beta cells are particularly sensitive to fatty acids because they have a limited ability to protect themselves against damage. And when exposed to high fat concentrations for long periods of time, their antioxidant self-defense mechanisms are inadequate to protect them against dysfunction. In addition, an increasing demand for insulin forces your beta cells to manufacture insulin in overdrive, creating even more internal cellular stress, and eventually their ability to produce insulin gets maxed out. In some individuals, this process can take many years to develop, while in others this process can occur rapidly.

The result of chronic beta cell stress has a fittingly bleak name: beta cell suicide. There comes a point in the life of a stressed beta cell when it is more advantageous to commit suicide than to stay alive. At this point, beta cells undergo a process known as *apoptosis,* or programmed cell death. It is a point of no return that's the equivalent of saying, "That's it! We can't take it anymore!" When a large population of beta cells undergoes apoptosis, insulin

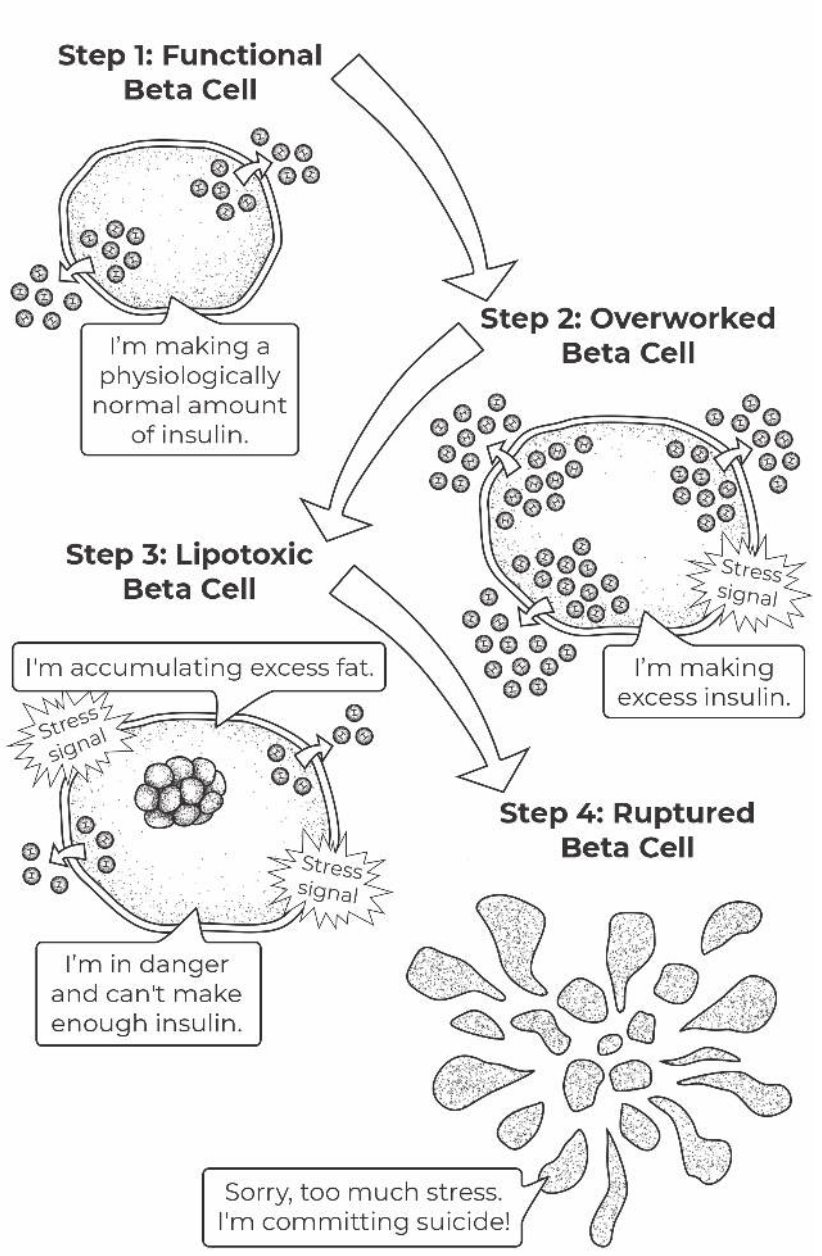
production rapidly falls below normal physiological levels within a short period of time.

In the same way that peak insulin production varies between individuals, the extent of beta cell suicide is also a highly variable process. Some individuals retain 60 percent of their original beta cell mass, whereas others will drop to as low as 20 percent. Autopsies have revealed that in the majority of patients with type 2 diabetes, more than half of the beta cell population has died. In this state, only a small population of beta cells are then responsible for secreting enough insulin to satisfy the metabolic demands of your entire body. As you might guess, this job is extremely difficult unless you help your muscle and liver significantly reduce their insulin requirements.

After the age of 20, your body stops making new beta cells; therefore beta cell death is considered irreversible. The question then becomes: If you significantly reduce your level of whole-body insulin resistance, can the remaining beta cell population produce enough insulin to meet the demands of all tissues? Fortunately, the answer is almost always yes. Even when beta cell mass has been significantly compromised, the remaining beta cell population is often capable of producing sufficient insulin for all tissues, but *only if you take steps to reduce your body's insulin needs by increasing your insulin sensitivity.* This means that you will benefit from substantially reducing your body's need for excess insulin to protect beta cells from committing suicide. Luckily, the Mastering Diabetes Method teaches you how to prevent and reverse the accumulation of excess fat in nonfatty tissues and prevent beta cell suicide before it has the chance to begin.

Insulin Resistance in Type 1 and Type 1.5 Diabetes

Many doctors, in addition to people living with type 1 and type 1.5 diabetes, believe that insulin resistance pertains only to people living with prediabetes, type 2 diabetes, and gestational diabetes. Furthermore, many people think that insulin resistance affects only overweight individuals, and that being slender or having a normal body mass index (BMI) is proof of being insulin sensitive. Unfortunately, these assumptions are incorrect. It is important to



Functional beta cells can commit suicide when they are stressed and when they accumulate excess fatty acids over time. Since beta cell death is largely irreversible, taking the necessary steps to maximize beta cell function can make a dramatic difference in your overall health.

understand that insulin resistance cannot be seen from the outside—it is a condition that reflects the *internal* state of your liver, muscles, adipose tissue, pancreas, and blood vessels. It can affect you, no matter how much you weigh.

Unlike type 2 diabetes, type 1 and 1.5 diabetes occur when your own immune system targets beta cells for destruction, leaving them incapable of manufacturing enough insulin to maintain your blood glucose within the normal range (80–130 mg/dL). When your own immune system erroneously targets your own tissues for destruction, scientists call this an *autoimmune* condition. Given what scientists understand about autoimmunity, they believe that certain individuals have a genetic predisposition to type 1 and type 1.5 diabetes at birth, but develop autoimmunity only when exposed to one or more environmental "triggers," including a viral infection, a bacterial infection, or exposure to cow's milk protein at a very young age. We'll explain more about the link between dairy products and autoimmunity in chapter 5, but for now suffice it to say that living with an autoimmune version of diabetes does not exclude you from developing diet-induced insulin resistance.

Even though type 2 diabetes is *caused* by insulin resistance, and type 1 and 1.5 diabetes result from an autoimmune reaction, the diet-induced insulin resistance present in both conditions is biologically identical. In fact, a growing body of scientific evidence demonstrates that insulin resistance is a growing concern in autoimmune diabetes owing to less-than-ideal lifestyle choices, and eating a diet that consists of low-carbohydrate foods like meat, fish, dairy, eggs, and oils or a diet containing significant quantities of refined sugar and processed foods.

When you're living with autoimmune diabetes and also develop insulin resistance from your diet, you develop *double diabetes*, a term used to describe people living with both autoimmune diabetes and the symptoms of type 2 diabetes.

Double diabetes is a life-threatening combination of health conditions that makes controlling your blood glucose virtually impossible, as glucose routinely gets trapped in your blood, unable to enter your muscle and liver in large quantities. Double diabetes occurs when you already have type 1 or type 1.5 diabetes, and you then eat yourself into insulin resistance.

We have observed that many people living with type 1 and 1.5 diabetes experience double diabetes, frustrated by some combination of an elevated A1c, hard-to-control blood glucose, increasing insulin requirements, low energy, impaired digestion, high blood pressure, high cholesterol, a low tolerance for carbohydrates, and/or an inability to lose weight. If you're in this segment of the population, you're in luck because the Mastering Diabetes Method directly addresses double diabetes head-on, simplifies the process of living with autoimmune diabetes, and teaches you how to control your blood glucose with precision.

Take-Home Messages

- Your doctors didn't learn nutrition in medical school (and it's not their fault!).
- The carbohydrate-centric diabetes model is an incomplete, flawed, and overly simplistic view of diabetes biology that keeps people with diabetes at a high risk for future complications.
- Insulin is not your enemy—*excess insulin* increases your risk for metabolic dysfunction in many tissues over time.
- Insulin resistance is caused by the accumulation of *excess* fat in tissues not designed to store large quantities of fat.
- High-fat diets cause whole-body insulin resistance that causes insulin rejection in your liver, muscles, and adipose tissue, in addition to promoting beta cell death.
- Insulin resistance can affect you even if you are slender, have a normal BMI, or are living with type 1 or type 1.5 diabetes.

To view the 150+ scientific references cited in this chapter, please visit us online at www.masteringdiabetes.org/bookinfo.

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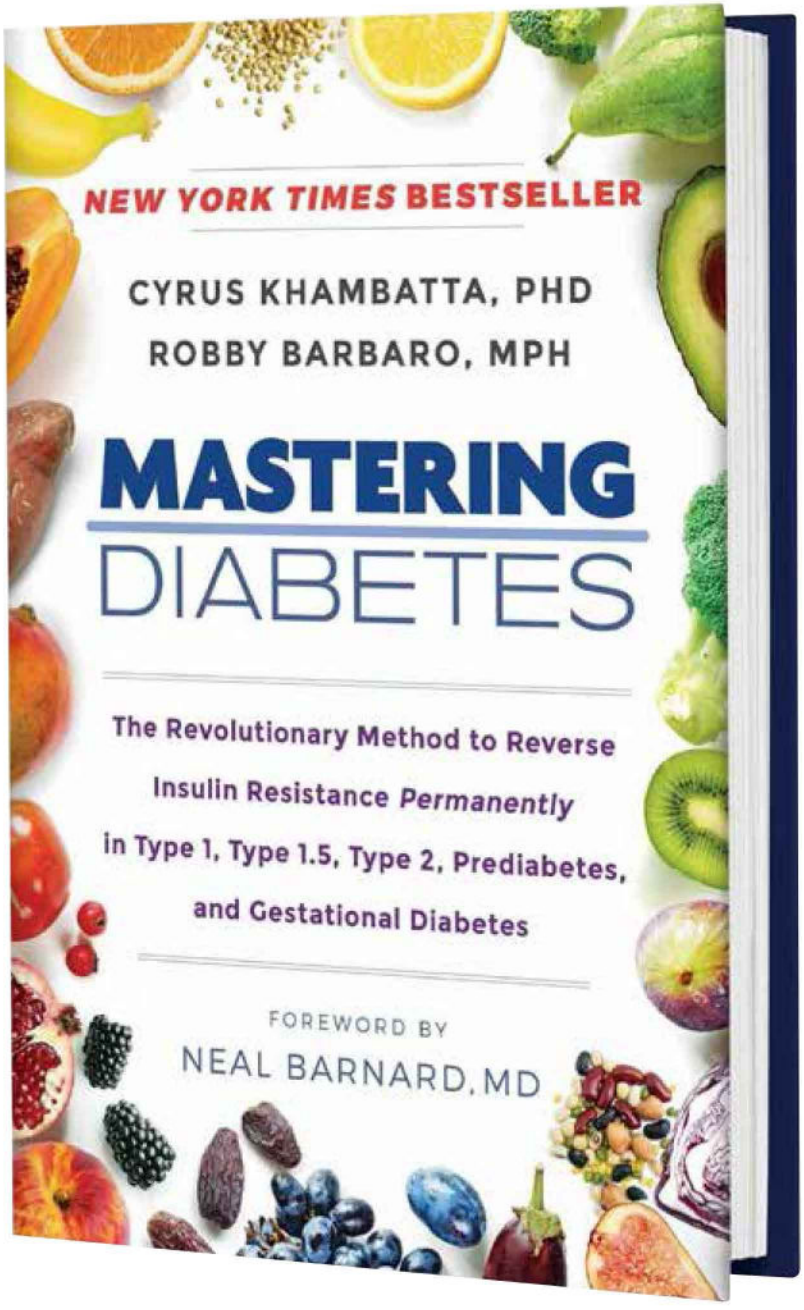


Questions? team@masteringdiabetes.org

Mango & Black Bean Quinoa Salad

2 servings

25 minutes

Ingredients

- **1/2 cup** (85g) **Quinoa** (dry)
- **1 15-ounce can** (425g) **Black Beans** (drained, rinsed) OR **1 1/4 cups** (215g) **Black Beans** (cooked from dry)
- **1/4 cup** (49g) **Red Onion** (diced) – *About 1 small-sized onion*
- **1 1/2 cups** (248g) **Mango** (peeled, diced) – *About 1 medium-sized mango*
- **6 cups** (180g) **Baby Spinach** (chopped)
- **2 tbsps** (30g) **Lime Juice** – *Juice from 1 lemon*
- **1 tsp** (2g) **Cumin** (or to taste)
- **1/4 cup** (4g) **Cilantro** (chopped)

Nutrition

Amount per serving	
Calories	410
Fat	4.4g
Carbs	78g
Fiber	16g
Protein	20g

Directions

1. Cook the quinoa according to package instructions. Set aside to cool.
2. Combine the cooled quinoa and all remaining ingredients in a large bowl and mix until well combined. Transfer to a plate and enjoy!

Notes

Leftovers: Refrigerate in an airtight container for up to four days.

Easiest Millet and Black Bean Burger

2 servings

20 minutes

Ingredients

- **1 15-ounce can** (425g) **Black Beans** (drained, rinsed) OR **1 1/4 cups** (215g) **Black Beans** (cooked from dry)
- **1 13.25-ounce can** (375g) **Mushrooms** (drained and rinsed) – *Can substitute with fresh mushrooms, chopped finely and cooked*
- **1/2 cup** (100g) **Dry Millet**
- **1/4 cup** (49g) **White Onion** (shredded) – *About 1 small-sized onion*
- **1/2 cup** (78g) **Carrot** (finely grated) – *About 1 large-sized carrot*
- **1 tsp** (6g) **Tamari Sauce**
- **1 tsp** (2g) **Smoked Paprika**
- **1 tsp** (1g) **Dried Oregano**
- **1 tsp** (3g) **Garlic Powder**
- **1 tsp** (2g) **Cumin**

Nutrition

Amount per serving	
Calories	422
Fat	3.8g
Carbs	81g
Fiber	18g
Protein	20g

Directions

1. Cook the millet according to package instructions. If using an Instant Pot: place ½ cup millet with 1 cup of water in the pot. Seal and set for 10 minutes manual. Make sure the valve is set at sealed.
2. Drain the black beans, add all the spices plus tamari and mash until the beans are fully formed. You can use a food processor and pulse a few times to make this faster.
3. Combine all ingredients and form into 8 patties. Put on a skillet on medium high heat. If you used canned mushrooms: 3 minutes each side. If you use fresh mushrooms it'll be approximately 3 to 5 minutes each side.

Kale and Lentil Breakfast Bowl

1 serving

20 minutes

Ingredients

- **2** (6g) **Garlic Cloves** (minced)
- **4** (40g) **Kale Leaves** (cut into 1/4-inch-thin strips)
- **1 15-ounce can** (425g) **Lentils** (drained, rinsed) OR **1 cup** (198g) **Lentils** (cooked from dry)
- **1/4 tsp** (1g) **Black Pepper**
- **2** (364g) **Tomatoes** (large-sized, diced)
- **2 tbsps** (8g) **Fresh Parsley** (chopped)
- **2 tbsps** (9g) **Green Onions** (chopped)
- **1 tbsp** (7g) **Flax Seeds, Freshly Ground**

Nutrition

Amount per serving	
Calories	396
Fat	4.8g
Carbs	61g
Fiber	20g
Protein	24g

Directions

1. In a small nonstick skillet over medium heat, combine 2 tablespoons of water with the garlic and kale. Cook, stirring frequently, until the kale has wilted, about 2 minutes.
2. Add the lentils and continue cooking until warmed through, about 2 more minutes.
3. Divide the mixture between 2 bowls and season to taste with black pepper.
4. Top with the tomatoes and sprinkle with he parsley, green onions, and flaxseeds.

Orange Green Smoothie Bowl

1 serving

10 minutes

Ingredients

- **2** (148g) **Clementines** (medium-sized)
- **1 cup** (221g) **Strawberries** (fresh or frozen)
- **1** (136g) **Banana** (medium-sized, divided, half frozen)
- **½ tsp** (3g) **Amla Powder** (optional)
- **½ cup** (74g) **Blueberries** (fresh frozen, thawed if frozen)
- **1/2 cup** (120g) **Water**
- **8 ounces** (227g) **Spinach** (chard or mixed greens)
- **1 tbsp** (7g) **Flax Seeds, Freshly Ground**

Nutrition

Amount per serving	
Calories	404
Fat	5.1g
Carbs	90g
Fiber	19g
Protein	12g

Directions

1. Process 1 clementine, the strawberries, the frozen banana, amla water, flax or chia and spinach in a strong blender. Blend until smooth.
2. Pour the smoothie into a bowl, slice the half unfrozen banana on top of the smoothie bowl.
3. Separate segments from the clementine and put on top along with the blueberries.

Sweet Potato & Red Bean Stew

2 servings

25 minutes

Ingredients

- **1/3 cup** (74g) **Vegetable Broth, Low Sodium**
- **2 cups** (535g) **Sweet Potato** (diced small) – *About 3 large-sized sweet potatoes*
- **1/4 cup** (43g) **Dry Quinoa**
- **1 tsp** (3g) **Cumin**
- **1 tsp** (2g) **Chili Powder**
- **1/2 tsp** (1g) **Onion Powder**
- **1/2 tsp** (2g) **Garlic Powder**
- **2/3 cup** (118g) **Red Kidney Beans** (canned, drained and rinsed)
- **1 cup** (242g) **Crushed Tomatoes** (canned, with juices)
- **1 cup** (240g) **Water** (divided)
- **1/4 cup** (4g) **Cilantro** (chopped)

Nutrition

Amount per serving	
Calories	410
Fat	3.7g
Carbs	84g
Fiber	16g
Protein	15g

Directions

1. Heat the vegetable broth in saucepan or sauté pan over medium-high heat. Add the potatoes and cook for 5 to 8 minutes or until softening, stirring occasionally. Add a splash more broth as needed to prevent sticking.
2. Add the quinoa, cumin, chili powder, onion powder, garlic powder, red kidney beans, crushed tomatoes, and water. Bring to a boil, reduce the heat to medium-low, and simmer for 12 minutes, or until the quinoa and potatoes are cooked through.
3. Ladle into a bowl. Top with the chopped cilantro leaves. Serve and enjoy!

Notes

Additional Toppings: Green onions, a dash of cayenne and/or hot sauce.

Leftovers: Refrigerate in an airtight container for up to four days.

Rainbow Lettuce Wraps with Spicy Mango Dressing

2 servings

15 minutes

Ingredients

- **3 cups** (267g) **Purple Cabbage** (thinly sliced) – *About 1/2 of a small head*
- **3 cups** (447g) **Red Bell Pepper** (thinly sliced) – *About 3 large-sized bell peppers*
- **3 cups** (465g) **Carrot** (grated) – *About 7 large-sized carrots*
- **3 cups** (213g) **Green Onion** (thinly sliced)
- **3 cups** (495g) **Mango** (chopped) – *About 2 medium-sized mangoes*
- **1/4 cup** (60g) **Water**
- **1/4 cup** (60g) **Apple Cider Vinegar**
- **4** (12g) **Garlic Cloves** (minced)
- **1/2 tsp** (3g) **Red Pepper Flakes**
- **2 heads** (1252g) **Romaine Hearts** (large leaves separated, washed and dried)
- **1/2 cup** (75g) **Avocado** (diced)
- **1 tsp** (3g) **Sesame Seeds**
- **1/3 cup** (5g) **Cilantro**

Nutrition

Amount per serving	
Calories	561
Fat	9.4g
Carbs	118g
Fiber	34g
Protein	18g

Directions

1. In a large mixing bowl combine the cabbage, bell pepper, carrot and green onions. Toss to combine and set aside.
2. Add the mango, water, apple cider vinegar, garlic, and red pepper flakes to a food processor or blender and blend until smooth.
3. To assemble the lettuce wraps, divide the cabbage mixture between the romaine leaves and garnish with avocado, sesame seeds, cilantro and spicy mango dressing. Serve immediately and enjoy!

Notes

Leftovers: Refrigerate veggies, dressing and toppings in separate airtight containers for up to five days. Assemble lettuce wraps just before serving.

Potato Chickpea Breakfast Hash

3 servings

25 minutes

Ingredients

- **1/2 cup** (111g) **Vegetable Broth, Low Sodium**
- **6 cups** (900g) **Russet Potato** (cut into small cubes) – *About 3 large-sized potatoes*
- **1/3 cup** (65g) **Red Onion** (chopped) – *About 1 small-sized onion*
- **1 1/2 cups** (228g) **Chickpeas** (canned, drained and rinsed)
- **1 1/2 tsps** (3g) **Paprika**
- **1 1/2 tsps** (5g) **Garlic Powder**
- **6 cups** (180g) **Baby Spinach** (chopped)

Nutrition

Amount per serving	
Calories	369
Fat	2.9g
Carbs	75g
Fiber	13g
Protein	14g

Directions

1. Heat the low-sodium vegetable broth in a pan over medium-high heat. Add the potatoes and cook for 10 to 12 minutes, stirring occasionally, adding more broth 1 Tbsp at a time as needed to prevent sticking, until the potatoes start to brown and soften. Add the onion and continue to cook for 5 to 6 minutes more.
2. Add the chickpeas, paprika, and garlic powder, and continue to cook until the potatoes are tender and chickpeas are warmed through, about 3 to 5 minutes. Add the spinach and stir until wilted. Season with more spices if desired.
3. Scoop onto your plate and enjoy!

Notes

Serving Size: One serving is approximately two cups.

More Flavor: Add other dried herbs and spices to taste, like cumin, chili powder, curry powder, black pepper, or oregano. Add bell pepper, jalapeno, or top with fresh herbs.

No Spinach: Use kale instead.

Leftovers: Refrigerate in an airtight container for up to three days.

Herbed Roasted Veggie Grain Bowl

2 servings

50 minutes

Ingredients

- **1/2 cup** (104g) **Dry Farro**
- **1 1/2 cups** (332g) **Vegetable Broth, Low Sodium**
- **4 cups** (428g) **Cauliflower** (cut into florets) – *About 1 medium-sized head*
- **3 cups** (408g) **Beet** (peeled and chopped) – *About 5 medium-sized beets*
- **2 cups** (142g) **Broccoli** (cut into florets) – *About 1 medium-sized stalk*
- **2 cups** (410g) **Butternut Squash** (cut into cubes) – *About 1 small-sized squash*
- **1 tsp** (1g) **Dried Parsley**
- **1/2 tsp** (2g) **Garlic Powder**

Nutrition

Amount per serving	
Calories	429
Fat	2.3g
Carbs	96g
Fiber	25g
Protein	18g

Directions

1. Cook the farro in the vegetable broth according to package instructions.
2. Preheat the oven to 400°F and line a baking sheet with parchment paper.
3. Add the cauliflower, beet, broccoli, and squash to the baking sheet. Season with parsley and garlic powder. Bake for 35 to 40 minutes or until browned and tender.
4. Serve the roasted vegetables over the cooked farro. Add more dried parsley, garlic powder, or your favorite herb blend to taste, and enjoy!

Notes

More Flavor: Add other dried herbs and spices to taste.

Vegetables: Use Brussels sprouts, sweet potato, carrots, or bell peppers instead.

Leftovers: Refrigerate in an airtight container for up to four days.

Moroccan Chickpea Stew

3 servings

20 minutes

Ingredients

- **3/4 cup** (166g) **Vegetable Broth, Low Sodium** (divided)
- **2 1/4 cup** (437g) **Yellow Onion** (diced) – *About 3 large-sized onions*
- **6** (18g) **Garlic Cloves** (sliced)
- **3 cups** (456g) **Chickpeas** (canned, drained and rinsed)
- **1 1/2 tsp** (5g) **Turmeric**
- **3/4 tsp** (2g) **Cinnamon**
- **3/4 tsp** (2g) **Cumin**
- **3/4 tsp** (2g) **Smoked Paprika**
- **1/8 tsp** (1g) **Black Pepper**
- **1/8 tsp** (1g) **Cayenne Pepper** (or to taste)
- **3 cups** (540g) **Tomato** (diced) – *About 5 medium-sized tomatoes*
- **3/4 cup** (45g) **Parsley**
- **3** (78g) **Pitted Dates** (chopped)

Nutrition

Amount per serving	
Calories	395
Fat	5.2g
Carbs	78g
Fiber	17g
Protein	16g

Directions

1. Heat a few Tbsp of vegetable broth in a sauté pan over medium-high heat. Add the onions and garlic, and stir for about 3 minutes, until the onions begin to soften. Add the chickpeas, turmeric, cinnamon, cumin, smoked paprika, black pepper, and cayenne pepper. Continue to cook for 3 more minutes.
2. Add the remaining vegetable broth, tomatoes, parsley, and dates to the pot and stir to combine. Reduce the heat to medium-low and cover the pot with a lid. Cook for 10 minutes, stirring occasionally.
3. Divide between bowls and enjoy!

Notes

Leftovers: Refrigerate in an airtight container for up to four days.

Chipotle Spiced Squash, Quinoa & Chickpea Salad

2 servings

30 minutes

Ingredients

- **1/4 cup** (43g) **Dry Quinoa**
- **2 1/2 cups** (513g) **Butternut Squash** (cubed)
- **2 tbsps** (28g) **Vegetable Broth, Low Sodium**
- **1/2 tsp** (1g) **Chipotle Powder**
- **1/4 tsp** (1g) **Garlic Powder**
- **1/2 tsp** (1g) **Black Pepper** (or to taste)
- **3 tbsps** (45g) **Lime Juice**
- **2 tsps** (40g) **Date Syrup**
- **2 tsps** (10g) **Almond Butter**
- **4 cups** (140g) **Mixed Greens**
- **1 cup** (180g) **Cherry Tomatoes** (halved) – *About 11 cherry tomatoes*
- **1 1/3 cups** (202g) **Chickpeas** (canned, drained and rinsed)
- **2 tbsps** (8g) **Parsley** (chopped, optional)

Nutrition

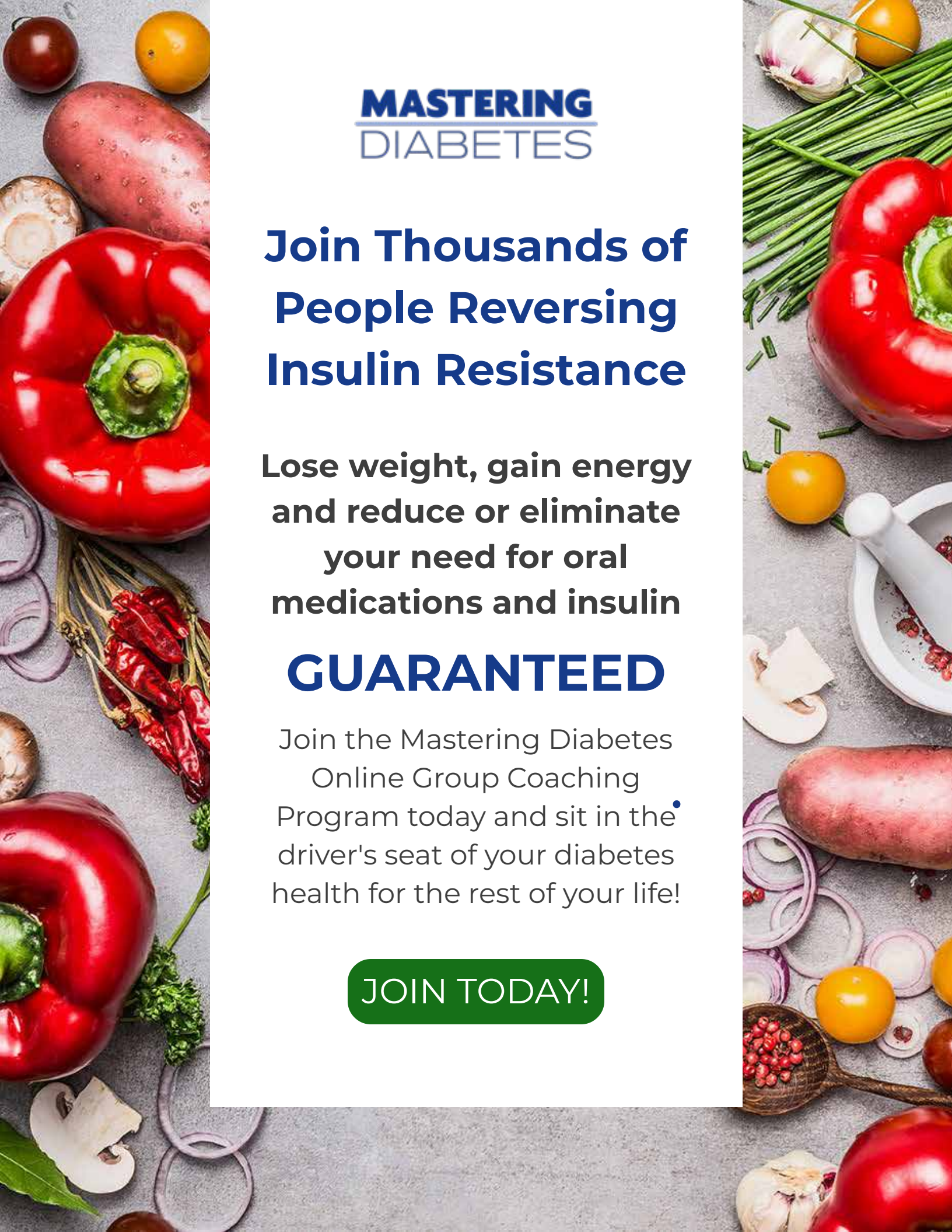
Amount per serving	
Calories	447
Fat	7.6g
Carbs	86g
Fiber	19g
Protein	16g

Directions

1. Preheat the oven to 400°F. Line a baking sheet with parchment paper.
2. Cook the quinoa according to package directions.
3. Toss the squash with vegetable broth, chipotle powder, garlic powder, and black pepper. Spread over the prepared baking sheet. Bake for 20 to 25 minutes in the oven, flipping halfway through, or until just browning.
4. In a small bowl, whisk together the lime juice, date syrup, and almond butter.
5. Add the mixed greens, tomatoes, chickpeas, squash, and quinoa to a large serving dish. Serve with the lime dressing, garnish with parsley, and enjoy!

Notes

Leftovers: Refrigerate in an airtight container for up to 4 days. Store the dressing on the side and dress the salad when ready to serve.



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