As Figure 1.1 shows, obesity increased immediately. Ten years later, as Figure 1.2 shows, diabetes began its inevitable rise. Age-adjusted prevalence is still rising precipitously. In 1980, an estimated 108 million people worldwide suffered with diabetes. By 2014, that number had swelled to 422 million. Even more concerning is the fact that there seems to be no end in sight.

**THE TWENTY-FIRST-CENTURY PLAGUE**

Diabetes has increased significantly in both sexes, every age group, every racial and ethnic group, and all education levels. Type 2 diabetes attacks younger and younger patients. Pediatric clinics, once the sole domain of type 1 diabetes, are now overrun with an epidemic of obese adolescents with type 2 diabetes.

This is not merely a North American epidemic, but a worldwide phenomenon, although close to 80 percent of the world’s adult diabetics live in developing nations. Rates of diabetes are rising fastest in the low- and middle-income nations of the world. In Japan, 80 percent of all new cases of diabetes are type 2.
China, in particular, is a diabetes catastrophe. In 2013, an estimated 11.6 percent of Chinese adults had type 2 diabetes, eclipsing even the long-time champion, the U.S., at 11.3 percent. Since 2007, 22 million Chinese—a number close to the population of Australia—have been newly diagnosed with diabetes. This number is even more shocking when you consider that only 1 percent of Chinese had type 2 diabetes in 1980. In a single generation, the diabetes rate has risen by a horrifying 1160 percent. The International Diabetes Federation estimates that the worldwide rate of diabetes will reach 1 in every 10 adults by the year 2040.

The problem is not trivial. In the U.S., 14.3 percent of adults have type 2 diabetes and 38 percent of the population has prediabetes, totaling 52.3 percent. This means that, for the first time in history, more people have the disease than not. Prediabetes and diabetes is the new normal. Worse, the prevalence of type 2 diabetes has increased only in the last forty years, making it clear that this is not some genetic disease or part of the normal aging process but a lifestyle issue.
Table 2.1 Classifications of diabetes mellitus

<table>
<thead>
<tr>
<th></th>
<th>Type 1</th>
<th>Type 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gestational</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other specific types:</td>
<td>- Genetic defects</td>
<td>- Other specific types:</td>
</tr>
<tr>
<td></td>
<td>- Pancreatic disease</td>
<td>- Genetic defects</td>
</tr>
<tr>
<td></td>
<td>- Drug or chemical induced</td>
<td>- Pancreatic disease</td>
</tr>
<tr>
<td></td>
<td>- Infections</td>
<td>- Drug or chemical induced</td>
</tr>
<tr>
<td></td>
<td>- Endocrinopathies</td>
<td>- Infections</td>
</tr>
</tbody>
</table>

**DIABETES SYMPTOMS**

Hyperglycemia, or high blood glucose, characterizes all forms of diabetes. When blood glucose levels rise above the kidney's ability to reabsorb the glucose (the renal threshold), it spills over into the urine, causing frequent, excessive urination and severe thirst. The chronic loss of glucose may lead to rapid weight loss and also stimulate the appetite. The most typical symptoms seen in diabetes therefore include:

- increased thirst,
- frequent urination,
- rapid, unexplained weight loss,
- increased hunger despite weight loss, and
- fatigue.

These symptoms of hyperglycemia are common to all forms of diabetes, but they occur more frequently in type 1 diabetes, since the onset of type 2 diabetes is typically very gradual. Today, type 2 diabetes is most often diagnosed during routine blood testing, before patients have symptoms.

In severe cases, patients—typically those with type 1 diabetes—may present with diabetic ketoacidosis. Dangerously high levels of acid
Table 2.2. Classification of diabetes and prediabetes according to A1C blood glucose levels

<table>
<thead>
<tr>
<th>A1C</th>
<th>Classification</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 5.7%</td>
<td>Normal</td>
</tr>
<tr>
<td>5.7%–6.4%</td>
<td>Prediabetes</td>
</tr>
<tr>
<td>&gt; 6.5%</td>
<td>Diabetes</td>
</tr>
</tbody>
</table>

Prediabetes is the in-between stage, where blood glucose levels are abnormally high, but not quite high enough to be considered diabetic. It denotes a state of very high risk of future progression to full-fledged type 2 diabetes. A patient with a baseline A1C of 6.0–6.5 percent (42–48 mmol/mol) has an estimated 25–50 percent risk of developing diabetes within five years. That’s more than twenty times the risk of a person with an A1C of 5.0 percent (31 mmol/mol).²

Blood glucose

The second test to diagnose diabetes is the blood glucose test, which is also known as the blood sugar or plasma glucose test. It is measured using either a fasting blood sugar test or an oral glucose tolerance test (OGTT).

For the fasting blood glucose test, a patient is asked to have no caloric intake for at least eight hours. A blood sample is then taken and the amount of glucose in the blood is measured. A level above 7.0 mmol/L (or 126 mg/dL) is considered diabetic.

For the OGTT, a patient is asked to ingest a standard test dose of 75 grams of glucose. A blood sample is taken two hours later and the amount of glucose in the blood is measured. A level above 11.1 mmol/L (or 200 mg/dL) is considered diabetic.

The A1C has largely replaced the fasting blood glucose test and the OGTT for diagnosis because of its simplicity and convenience, but all of these tests are considered accurate and acceptable. Occasionally,
diabetes is diagnosed using a random blood sugar test. A blood sample is taken at a random time and the level of glucose in the blood is measured. A level above 11.1 mmol/L (or 200 mg/dL) is considered diabetic if accompanied by other symptoms.

**Table 2.3** Diagnostic criteria for diabetes

<table>
<thead>
<tr>
<th>Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fasting blood glucose &gt; 7.0 mmol/L (126 mg/dL)</td>
</tr>
<tr>
<td>2 hour blood glucose &gt; 11.1 mmol/L (200 mg/dL) during OGTT</td>
</tr>
<tr>
<td>A1C &gt; 6.5% (48 mmol/mol)</td>
</tr>
<tr>
<td>Symptoms of hyperglycemia and random blood glucose &gt; 11.1 mmol/L (200 mg/dL)</td>
</tr>
</tbody>
</table>

The total amount of glucose circulating in the blood at any time is surprisingly small—roughly a single teaspoonful. Glucose does not float freely around in the blood. Rather, most of the body's glucose is contained within our cells.

Hormones tightly regulate our blood glucose to avoid excessively low or high levels. Even when we eat large amounts of sugar, the blood glucose level still remains within a remarkably narrow, controlled range due to the coordinated actions of various hormones. As glucose is absorbed through the intestines into the blood, the islet cells within the pancreas secrete the hormone insulin. Insulin allows the glucose to enter the cells as fuel for energy. The body stores any excess glucose in the liver for future use, which keeps our blood glucose from rising out of its normal range.

**TYPE 1 DIABETES: THE FACTS**

*Type 1 Diabetes* has been previously called juvenile diabetes, since its onset commonly occurs during childhood. However, although three-quarters of all cases are diagnosed in patients under eighteen,
persistent nausea and vomiting. If the disease goes untreated, it eventually leads to coma and death. In the United States, more than 100,000 patients are diagnosed with chronic kidney disease annually, costing $32 billion in 2005. The burden is not only financially enormous, but emotionally devastating.

Diabetic kidney disease (nephropathy) is the leading cause of end stage renal disease (ESRD) in the United States, accounting for 44 percent of all new cases in 2005. Patients whose kidneys have lost over 90 percent of their intrinsic function require dialysis to artificially remove the accumulated toxins in the blood. This procedure involves removing the patient’s “dirty” blood, running it through the dialysis machine to clean out its impurities, and then returning the clean blood to the body. To stay alive, patients require four hours of dialysis, three times per week, indefinitely, unless they receive a transplant.

Figure 3.1. Adjusted prevalence rates of end stage renal disease

Diabetic kidney disease often takes fifteen to twenty-five years to develop, but, like retinopathy, it may occasionally be diagnosed before
of Agriculture in 1990 allowed that some weight gain after the age of thirty-five was consistent with good health.

That same year, Dr. Willett challenged the conventional thinking, reporting that weight gain after age eighteen was the major determinant of type 2 diabetes.\(^1\) A weight gain of 20–35 kg (44–77 pounds) increased the risk of type 2 diabetes by 11,300 percent. Gaining more than 35 kg (77 pounds) increased the risk by 17,300 percent! Even smaller amounts of weight gain could raise the risk significantly. But this idea was not an easy sell to a sceptical medical profession.\(^2\) “We had a hard time getting the first paper published showing that even slight overweight greatly increased the risk of diabetes,” Willett remembers. “They didn’t believe it.”

**BODY MASS INDEX: THE RELATIONSHIP BETWEEN OBESITY AND DIABETES**

The *body mass index* is a standardized measurement of weight, and it is calculated by the following formula:

\[
\text{Body mass index} = \frac{\text{Weight (kg)}}{\text{Height}^2 (\text{m}^2)}
\]

A body mass index of 25.0 or higher is considered overweight, while a body mass index of between 18.5 and 24.9 is in the healthy range.

**Table 4.1.** Body mass index classifications

<table>
<thead>
<tr>
<th>Body Mass Index</th>
<th>Classification</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 18.5</td>
<td>Underweight</td>
</tr>
<tr>
<td>18.5–24.9</td>
<td>Normal weight</td>
</tr>
<tr>
<td>25.0–29.9</td>
<td>Overweight</td>
</tr>
<tr>
<td>30.0–34.9</td>
<td>Obese</td>
</tr>
<tr>
<td>35.0–39.9</td>
<td>Severe Obesity</td>
</tr>
<tr>
<td>&gt; 40.0</td>
<td>Morbid Obesity</td>
</tr>
</tbody>
</table>
waist circumference. This pattern of obesity, where most of the fat is carried around the abdomen, is also known as central obesity, or central adiposity. In contrast, subcutaneous fat is the fat deposited directly under the skin.

The different health risks associated with the different fat distributions explain how roughly 30 percent of obese adults are metabolically normal. These healthy-fat people carry more subcutaneous fat rather than the more dangerous visceral fat. On the other hand, some normal-weight people show the same metabolic abnormalities as in obesity because of excessive visceral fat.

Type 2 diabetes may be diagnosed for patients with a wide range of body mass indexes, following a normal distribution with no distinct subpopulation of “thin” diabetics. A full 36 percent of newly diagnosed diabetics have a normal body mass index of less than 25. Look at Figure 4.1. The key clinical indicator is clearly not total body fat as measured by body mass index. Rather, it’s visceral or intra-organic fat.

Figure 4.1. Population BMI distribution for newly diagnosed diabetes

![Image of Figure 4.1]
Independent of total weight, central obesity is highly correlated to metabolic abnormalities,\textsuperscript{14} increased cardiac risk,\textsuperscript{15} and progression to type 2 diabetes.\textsuperscript{16} Reducing visceral fat also successfully reduces the risk of progression of type 2 diabetes.\textsuperscript{17}

Subcutaneous fat, on the other hand, shows little correlation to type 2 diabetes or heart disease. The surgical removal, via liposuction,\textsuperscript{18} of almost 10 kilograms of subcutaneous fat brought no significant metabolic benefits whatsoever, which suggests that subcutaneous fat plays little role in the development of type 2 diabetes.

The waist-to-height ratio is a simple measure of central adiposity, calculated by comparing waist circumference to height. This ratio is far more predictive of years of life lost than body mass index.\textsuperscript{19} Optimally, your waist circumference should be less than half your height. For example, an average man standing 5 foot 10 inches (70 inches) should strive to maintain a waist size of 35 inches or less. As central obesity increases, risk of metabolic disease skyrockets.

\textbf{Figure 4.2.} Waist-to-height ratio and years of life lost (YLL): A dramatic increase\textsuperscript{20}
This normal process occurs when we stop eating (and begin fasting), which is when the body needs this source of energy. Although we often use the word fasting to describe periods in which we deliberately limit certain foods or abstain from eating altogether, such as before a medical procedure or in conjunction with a religious holiday, it simply applies to any period between snacks or meals when we are not eating. During periods of fasting, our body relies on its stored energy, meaning that it breaks down glycogen and fat.

**Figure 5.1.** Storage of food energy as sugar or fat

<table>
<thead>
<tr>
<th>Eat Food</th>
<th>Increase Insulin</th>
<th>Store Sugar in Liver</th>
<th>Produce Fat in Liver</th>
</tr>
</thead>
</table>

Several hours after a meal, blood glucose drops and insulin levels begin to fall. To provide energy, the liver starts to break down the stored glycogen into component glucose molecules and releases it into general circulation in the blood. This is merely the glycogen-storage process in reverse. This happens most nights, assuming you don’t eat at night.

Glycogen is easily available but in limited supply. During a short-term fast (twenty-four to thirty-six hours), glycogen will provide all the glucose necessary for normal body functioning. During a prolonged fast, the liver will manufacture new glucose from stored body fat. This process is called **gluconeogenesis**, meaning literally “the making of new sugar.” In essence, fat is burned to release energy. This is merely the fat-storage process in reverse.

**Figure 5.2.** Gluconeogenesis: The reverse of the glycogen storage process

<table>
<thead>
<tr>
<th>Burn Stored Sugar in Liver</th>
<th>Decrease Insulin</th>
<th>No Food “Fasting”</th>
</tr>
</thead>
<tbody>
<tr>
<td>Burn Fat in Liver</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
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**Figure 5.1.** Storage of food energy as sugar or fat

![Diagram showing the process of eating food leading to increased insulin, which stores sugar in the liver, producing fat in the liver.]

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**Figure 5.2.** Gluconeogenesis: The reverse of the glycogen storage process

![Diagram showing the process of burning stored sugar in the liver leading to decreased insulin, no food, and burning fat in the liver.]

![Example](image-url)
potatoes—are well known to raise blood glucose and insulin production. If these highly refined carbohydrates were the main cause of hyperinsulinemia, they would also be the prime cause of weight gain. This theory of obesity is known as the carbohydrate-insulin hypothesis. It forms the rational basis for many low-carbohydrate diets, such as the Atkins diet. By eliminating many of the “fattening” carbohydrates, we lower insulin levels and prevent weight gain.

**Figure 5.3.** Hormonal obesity I: Hyperinsulinemia causes obesity

As you read the coming chapters, watch the progression of the “Hormonal Obesity” diagrams from this one through Figures 5.4, 6.3, 7.2, 8.1, 9.1, 9.2, 9.3, and 9.4. Reviewed in sequence, these diagrams illustrate how the building blocks of the metabolic syndrome stack up over time.

The first low-carbohydrate diet dates all the way back to the mid-nineteenth century. In 1863, William Banting (1796–1878), an English undertaker, published the pamphlet *Letter on Corpulence, Addressed to the Public*, which is often considered the world’s first diet book. Weighing 202 pounds (91.6 kilograms), Banting had tried unsuccessfully to lose weight by eating less and exercising more. But, just like today’s dieters, he was unsuccessful.

On the advice of his surgeon, Banting tried a new approach. When he strenuously avoided the bread, milk, beer, sweets, and potatoes that had previously made up a large portion of his diet, he lost weight and successfully kept it off. For most of the next century, diets low in refined carbohydrates were accepted as the standard treatment for obesity.

For all the success of low-carb diets, the carbohydrate-insulin hypothesis remains incomplete. While refined carbohydrates are certainly an important contributor to hyperinsulinemia, they are not the
only contributor. There are many other significant influences. One of the most important is insulin resistance.

As we’ve seen, insulin acts like a key to open a gate for glucose to enter the cell. But sometimes, in a state of insulin resistance, the usual levels of insulin are not sufficient and glucose piles up in the bloodstream because it cannot get into the cells. To compensate, the body produces more insulin to overcome this resistance and force the blood glucose inside. The effect is to restore normal blood glucose levels but at a cost of persistent hyperinsulinemia. We care about insulin resistance so much because this compensatory hyperinsulinemia drives overall weight gain. But here’s the million-dollar question: How does this insulin resistance develop in the first place?

**Figure 5.4.** Hormonal obesity II: Insulin resistance causes hyperinsulinemia

![Diagram showing the relationship between Fattening Carbohydrates, High Insulin, Obesity, and Insulin Resistance.]
Figure 6.1. Changes in insulin as obesity progresses toward type 2 diabetes. The converse, the idea that insulin resistance causes obesity, is implausible since obesity typically predates insulin resistance. The only remaining possibility is that some X factor is the underlying cause of both obesity and insulin resistance. The connection, as we shall see, is too much insulin. The X factor is hyperinsulinemia.

Figure 6.2. Hyperinsulinemia: The X factor causing both obesity and insulin resistance

RESISTANCE AS A PROTECTIVE MECHANISM

The human body follows the fundamental biological principle of homeostasis. If things change too far in one direction, the body reacts...
Figure 6.1. Changes in insulin as obesity progresses toward type 2 diabetes

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Figure 6.2. Hyperinsulinemia: The X factor causing both obesity and insulin resistance

RESISTANCE AS A PROTECTIVE MECHANISM

The human body follows the fundamental biological principle of homeostasis. If things change too far in one direction, the body reacts
drives the vicious cycle. Hyperinsulinemia leads to insulin resistance, which leads to worsening hyperinsulinemia.

**Figure 6.3.** Hormonal obesity III: High insulin → resistance → higher insulin

The cycle keeps going around and around, until the insulin levels in the body are extremely high, which drives weight gain and obesity. The longer the cycle continues, the worse it becomes, which is why obesity and insulin resistance are so time dependent. People can be stuck in this vicious cycle for decades, developing significant insulin resistance. Resistance then leads to high insulin levels, which are independent of diet.

But the story gets worse. Insulin resistance leads to higher fasting insulin levels. Fasting insulin levels are normally low. Now, instead of starting the day with low insulin after the nightly fast, we start with high insulin. The consequences are dire: the fat get fatter. As insulin resistance becomes a larger and larger part of the problem, it can, in fact, become a major driver of high insulin levels. *Obesity drives itself.*

The fact that insulin resistance leads to compensatory hyperinsulinemia has been long accepted. But the novel notion that hyperinsulinemia also causes insulin resistance is slowly gaining acceptance. Dr. Barbara Corkey, the 2011 Banting Medal winner from Boston University’s School of Medicine, called her lecture, “Hyperinsulinemia is the root cause of insulin resistance, obesity and diabetes.” The Banting Medal is the American Diabetes Association’s highest scientific award, so these are not merely the musings of a fringe group.
The hallmark of type 2 diabetes is elevated insulin resistance. Both obesity and type 2 diabetes are manifestations of the same underlying problem: hyperinsulinemia. Their close relationship has given rise to the term “diabesity,” which implicitly acknowledges that they are one and the same disease.

**Figure 6.4.** Hyperinsulinemia: The link between obesity and diabetes

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**HYPERINSULINEMIA AND THE OVERFLOW PHENOMENON**

**Insulin Resistance Occurs** when blood glucose remains elevated despite normal or high levels of insulin, since the cells are resisting insulin’s pleas to take up glucose. But how does hyperinsulinemia cause this phenomenon?

The currently held lock-and-key paradigm suggests that the key (insulin) opens the lock (cell surface receptor) to allow glucose inside, and that once you remove the key (insulin), blood glucose can no longer enter the cell. With insulin resistance, we imagine that the lock and key no longer fit together very well. The key only partially opens the lock and not very easily, so glucose, which cannot enter normally, instead piles up outside, in the blood. As less glucose enters the cell, it faces a state of internal starvation and the body produces more insulin. Since each key works less efficiently, the body compensates by producing more keys. This hyperinsulinemia ensures that enough glucose gets into the cells to meet its energy requirement. It’s a nice, neat theory. Too bad it has no basis in reality.
Insulin resistance is predominantly a glucose overflow problem of the overstuffed, fatty liver. As the first stop for metabolism of ingested nutrients, the liver is naturally the epicenter of health problems related to excess consumption. Insulin resistance is primarily caused by excessive fatty infiltration of the liver caused in turn by excessive glucose and fructose consumption. In other words, too much sugar causes fatty liver, the key problem of insulin resistance, as Figure 6.5 shows.
Without dietary intervention, this insulin resistance almost always leads to the second problem, beta cell dysfunction. Furthermore, only insulin resistance and virtually nothing else causes beta cell dysfunction. Conventional medical wisdom holds that this dysfunction occurs because of exhaustion and eventual scarring of the insulin-producing cells. This idea implies that these two phenomena—insulin resistance and beta cell dysfunction—occur for entirely separate reasons. However, given this mutually exclusive and intimate relationship, Ockham’s razor suggests that both defects must surely be caused by the same underlying mechanism.

Only when insulin production fails to keep pace with increasing resistance does the blood glucose rise high enough to make the clinical diagnosis of type 2 diabetes. Thus, there are two underlying prerequisites of the disease: elevated insulin resistance and beta cell dysfunction. The progression of blood glucose levels in the years preceding the diagnosis occurs in two distinct phases, reflecting these two distinct abnormalities.
Eventually, the overfilled, fatty liver becomes unable to accept any more glucose and starts becoming insulin resistant. As seen previously, this insulin resistance is an overflow phenomenon. As shown in Figure 7.2, the cycle proceeds as follows:

1. Hyperinsulinemia causes fatty liver.
2. Fatty liver causes insulin resistance.
3. Insulin resistance leads to compensatory hyperinsulinemia.
4. Repeat cycle.

**Figure 7.2.** Hormonal obesity IV: High insulin → fatty liver → insulin resistance

Fat inside the liver, rather than overall obesity, is the crucial stepping stone toward insulin resistance and diabetes. Fatty liver is associated at all stages of insulin resistance from obesity to prediabetes to full-blown diabetes. And that relationship holds in all racial groups and ethnicities.

Fatty liver is the clearest sign that hyperinsulinemia and insulin resistance are developing, and one of the earliest. Fatty liver precedes the clinical diagnosis of type 2 diabetes by ten years or more. As the liver slowly accumulates fat it becomes increasingly insulin resistant. Fatty liver can be diagnosed by ultrasound, but an increased waist circumference or waist-to-height ratio is an important clue to its presence. Blood markers of liver damage also often mirror that slow rise, and this phase has been termed “the long, silent scream from the liver.”

Two main types of fatty liver disease exist: alcohol-related liver disease and non-alcoholic fatty liver disease. The first is associated, as the
liver enzymes and chronic liver disease in the Western world. This is the Rocky Balboa of liver diseases.

Figure 7.3. Insulin resistance rises with liver fat

Why some people have severe fatty infiltration of the liver without evidence of damage while others have minimal fat and severe damage remains unknown.

As the liver slowly accumulates fat, insulin resistance escalates in lockstep. In type 2 diabetic patients, a close correlation exists between the amount of liver fat and the insulin dose required, reflecting greater insulin resistance. In short, the fattier the liver, the higher the insulin resistance. Therefore, to understand insulin resistance, we must first understand how fatty liver develops.

How fatty liver develops

Here’s a startling fact: I can give you fatty liver. Actually, I can give anybody fatty liver. What’s the scariest part? This crucial first step toward type 2 diabetes only takes three weeks!
Figure 7.4. The hepatic cycle (insulin resistance)

The hepatic cycle may continue for many years before the start of the pancreatic cycle. The fatty liver decompresses itself by exporting newly created fat as very low-density lipoprotein (VLDL) to other organs, including the skeletal muscles and pancreas. As fatty muscle develops, whole body insulin resistance worsens further. As the pancreas becomes clogged with fat, it becomes unable to secrete insulin normally. Insulin levels, previously high to offset the high blood glucose, begin to fall.

The loss of this compensation results in a rapid rise in blood glucose and, ultimately, the diagnosis of type 2 diabetes. Even though insulin drops, it stays maximally stimulated by the high blood glucose. This is the body’s attempt to break this vicious cycle, as we shall soon discuss.

Figure 7.5. The pancreatic cycle (beta cell dysfunction)
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The loss of this compensation results in a rapid rise in blood glucose and, ultimately, the diagnosis of type 2 diabetes. Even though insulin drops, it stays maximally stimulated by the high blood glucose. This is the body’s attempt to break this vicious cycle, as we shall soon discuss.

Figure 7.4. The hepatic cycle (insulin resistance)

Figure 7.5. The pancreatic cycle (beta cell dysfunction)
it to acetaldehyde, which stimulates de novo lipogenesis, so alcohol, like fructose, easily becomes liver fat. This explains the well-known effect of alcohol consumption in producing fatty liver disease.

**Figure 8.1.** Hormonal obesity V: Fructose, fatty liver, and insulin resistance

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**FRUCTOSE AND INSULIN RESISTANCE**

That fructose overfeeding could experimentally provoke insulin resistance has been known since as far back as 1980. Healthy subjects overfed 1000 calories per day of fructose showed a 25 percent worsening of their insulin sensitivity after just seven days. Glucose overfeeding of subjects, by contrast, did not show any similar deterioration.

A more recent study (2009) reinforced how easily fructose induces insulin resistance in healthy volunteers. Subjects consumed 25 percent of their daily calories as Kool-Aid sweetened with either glucose or fructose. While this amount seems extreme, many people do consume this high a proportion of sugar in their diets. The fructose group—but not the glucose group—increased their insulin resistance so much that they would be clinically classified as prediabetics, a development that required only eight weeks of fructose overconsumption.

Remarkably, it only takes one week of excess fructose to cause...
The traditional Chinese diet: High carbs, low sugar, no diabetes

To a lesser extent, the same story has played out in the United States. Americans gradually switched from consuming their carbohydrates as grains to eating them as sugar in the form of corn syrup. Consider Figure 8.3; when both grain and fructose intake began to rise in the late 1970s, the result was the start of an epidemic of obesity and type 2 diabetes.

Sugar is more fattening than any other refined carbohydrate, and leads specifically to type 2 diabetes. The prevalence of diabetes climbs 1.1 percent for every extra 150 sugar calories per person per day. Each additional daily 12-oz serving of soda increases the risk of diabetes by 25 percent and the risk of metabolic syndrome by 20 percent. No other food group—not dietary fat, not protein—shows any significant relationship to diabetes.

Diabetes correlates strongly to sugar, not other sources of calories. Fructose overconsumption directly stimulates fatty liver and leads directly to insulin resistance. Consumption of high-fructose corn syrup, which is chemically almost identical to sugar, also shows the same tight correlation to diabetes.
There is something sinister about overconsumption of fructose. What distinguishes sugar from other highly refined carbohydrates? What is the common link to disease? Fructose. Yes, Dr. Robert Lustig had it right. *The dose makes the poison*—and in the doses we are currently eating it, sugar is a toxin.

**FRUCTOSE TOXICITY**

Fructose is particularly toxic for several reasons. First, as we’ve seen, only the liver can metabolize it, so virtually all ingested fructose is stored as newly created fat. This excessive liver fat directly causes insulin resistance.

Second, the liver metabolizes fructose without limits. More ingested fructose leads to more hepatic DN and more liver fat, independent of insulin. Fructose does little to activate natural satiety.
are created. Excessive DNL can overwhelm the export mechanism, resulting in abnormal retention of this new fat in the liver.\textsuperscript{11} As you stuff more and more fat into the liver, it becomes noticeably engorged and can be diagnosed on ultrasound as fatty liver. But if the liver is not the appropriate place to store this new fat, where should it go?

First, you could try to burn it off for energy. However, with all the available glucose around after a meal, the body has no reason to burn the new fat. Imagine you have gone to Costco and bought waaayyy too much food to store in your refrigerator. One option is to eat it, but there’s simply too much. If you cannot get rid of it, much of the food will be left on the counter where it will rot. So this option is not viable.

Your glycogen “fridge” is full, so the only remaining option is to export the newly created fat (excess food) somewhere else. This mechanism is known as the endogenous pathway of lipid transport. Essentially, triglycerides are packaged with special proteins to create very low-density lipoproteins (VLDL), which are released into the bloodstream to help decompress the congested liver\textsuperscript{12}.

More dietary glucose and fructose means more DNL which means more VLDL must be released\textsuperscript{13}, \textsuperscript{14}. This mass export of triglyceride-rich VLDL particles is the major reason for high plasma triglyceride levels\textsuperscript{15}, which are detectable in all standard blood tests for cholesterol. Ultimately, eating too much glucose and too much fructose causes this hypertriglyceridemia.

\textbf{Figure 9.1.} Hormonal obesity VI: The effect of high triglycerides
Low levels of HDL are found in close association with high levels of triglycerides: more than 50 percent of patients with low HDL also have high triglycerides. High levels of triglycerides activate the enzyme cholesterol ester transfer protein (CETP), which reduces HDL levels. Given this close association with triglycerides, it should be no surprise that low-carbohydrate diets raise HDL, even independent of weight loss. As with triglycerides, low HDL does not cause heart disease, but is a powerful indicator.

What is clear, however, is that the lipid profile typical of the metabolic syndrome—high triglycerides and low HDL—results from the excess of VLDL, which ultimately stems from hyperinsulinemia, which ultimately stems from eating too much glucose and fructose. Again, too much sugar.

**Figure 9.2.** Hormonal obesity VII: Fatty liver → low HDL

**Abdominal obesity**
The adipocytes get larger as they take up the triglycerides for storage. This is not particularly dangerous to our health since adipocytes are designed to store fat. But being too fat is dangerous from an evolutionary standpoint, because fat animals get eaten.

The adipocytes protect themselves against overexpansion by releasing the hormone leptin. This signals the hypothalamic area of the brain that we need to lose fat. We stop eating, insulin drops, and we lose...
heart—and the volume of blood in circulation by enhancing the kidney’s ability to reabsorb sodium (salt). In addition, insulin stimulates the secretion of anti-diuretic hormone, which helps the body to reabsorb water. Together, this salt and water retention mechanism increases blood volume and thus causes higher blood pressure. Insulin also constricts blood vessels, increasing the pressure inside.

**Figure 9.3.** Hormonal obesity VIII: Hyperinsulinemia and hypertension

**WHY METABOLIC SYNDROME MATTERS**

Each additional component of metabolic syndrome—high triglycerides, low HDL, central obesity, high blood glucose, and high blood pressure—significantly increases the risk of all the modern metabolic diseases, such as heart attacks, strokes, peripheral vascular disease, type 2 diabetes, Alzheimer’s disease, and cancer. These symptoms cluster together, but not every disease manifests in every person: one person may have low triglycerides, another person will have high blood sugars from insulin resistance, and yet another will have high blood pressure. But having one of these factors increases the likelihood of having the others because they all share the same root cause.

In a typical patient, gaining as little as 2 kilograms (4.4 pounds) of weight is the first detectable abnormality related to hyperinsulinemia/
insulin resistance, followed by low HDL cholesterol levels. High blood pressure, fatty liver, and high triglycerides emerge next, at roughly the same time. The very last symptom to appear is usually high blood glucose, which clinches the diagnosis of type 2 diabetes.

The West of Scotland study confirmed that fatty liver and elevated triglycerides precede the diagnosis of type 2 diabetes. Fatty liver occurs early in metabolic syndrome. While virtually all patients with metabolic syndrome have fatty liver, the opposite is not true. Only a minority of patients with fatty liver have full-blown metabolic syndrome (see Figure 9.4).

**Figure 9.4.** Hormonal obesity IX: Full-blown metabolic syndrome

Insulin resistance and type 2 diabetes cannot cause metabolic syndrome because they are part of the syndrome. *Hyperinsulinemia* causes it. The very core of the problem is hyperinsulinemia from excessive fructose and glucose, but especially fructose intake. Metabolic syndrome, of which obesity and type 2 diabetes are a key part, are ultimately caused by—you guessed it—too much sugar.

Obesity, insulin resistance, and beta cell dysfunction are all protective mechanisms. Obesity tries to prevent DNL from overwhelming the liver by safely storing the newly created fat in the adipocytes. We know this because patients with a rare, genetic disorder called lipodystrophy, which is characterized by a lack of fat cells, show all the manifestations
two studies clearly established the paradigm of *glucotoxicity*—that high blood glucose is toxic in type 1 diabetes.

**Figure 10.1.** Intensive insulin therapy leads to major weight gain¹

![Intensive Insulin vs Standard Insulin](image)

Some patients, however, paid a price. Hypoglycemic episodes during the DCCT study were three times more common in the intensive insulin group compared to those receiving standard treatment. Other patients experienced major weight gain. Over nine years, almost 30 percent of the subjects in that group gained a significant amount of weight, defined as an increase in body mass index of more than 5. This far exceeded the impact on those receiving conventional insulin therapy. One-quarter of that intensive treatment group had increased their body mass index from 24 (normal weight) to 31 (obese). Given the health consequences of obesity, this was no small concern. Other disquieting danger signs appeared, too. The weight gain was concentrated in the abdominal area, the central obesity known to be far more predictive of future cardiovascular disease. Other key risk factors, blood pressure and blood cholesterol, also increased.

Over time, weight, waist circumference, and insulin dosage continued to grow inexorably. Intensive insulin treatment had led to
glucotoxicity, but only at the expense of insulin toxicity. As in type 1 diabetes, high insulin doses were not good; they were bad.

**Figure 10.2.** Insulin use and increased risk of mortality in type 2 diabetes

These results were not new. Reviews of large population databases, such as the 1996 Quebec Cardiovascular Study, established hyperinsulinemia as a prime risk factor for heart disease. In Saskatchewan, Canada, a review of more than twelve thousand newly diagnosed diabetic patients found a “significant and graded association between mortality risk and insulin exposure level.” It wasn’t a trivial effect, either. The high-insulin group had a 279 percent higher risk of death compared to those that did not use insulin. Treating type 2 diabetes with insulin was not good; it was bad. Simply put, the higher the insulin dose, the higher the risk of dying.

Moreover, the longer the treatment time with insulin, the greater the risk of cardiovascular disease. A 2011 study showed that both low and high blood glucose carried excess risk of death, again reflecting the dual toxicities of glucose and insulin. Once again, insulin use was associated with a mind-boggling 265 percent increased risk of death.

A Cardiff University review of almost 10 percent of the U.K. population from 2004 to 2015 found that lower A1C was associated with
Oral Hypoglycemics: Not the Answer

this decision, individual members received millions of dollars and the American Diabetes Association itself reaped more than $7 million in 2004 alone from its pharmaceutical “partners.”

When Dr. Banting discovered insulin in 1921, he licenced the drug to pharmaceutical companies without a patent because he fervently believed this life-saving miracle should be made available to everybody who needed it. Yet, insulin—now available in many different formulations—is estimated to have cost the U.S. health care system $6 billion in 2012, driven in part by steep price increases. Between 2010 and 2015, these newer insulins increased in price from 168 to 325 percent. In 2013, Lantus, a long-acting form of insulin, earned $7.6 billion, making it the world’s bestselling diabetes drug. Various other insulins took another six of the top ten spots on that list.

Between 2004 and 2013, no less than thirty new diabetes drugs came to market. Despite several setbacks, by 2015 sales of diabetes drugs had reached $23 billion, which is more than the combined revenue of the National Football League, Major League Baseball, and the National Basketball Association.

**Figure 11.1.** Increasing variety of diabetic medications

![Graph showing increasing variety of diabetic medications](image-url)
Oral Hypoglycemics: Not the Answer

Metformin and DPP-4 medications use mechanisms other than raising insulin to lower blood glucose. But they do not lower insulin either, so the result is neither weight gain nor weight loss. Reducing glucotoxicity while keeping insulin neutral produces minimal benefits. Clinically, these medications are weight neutral, but also neutral with regard to cardiovascular risk or benefits.

Acarbose, SGLT2 inhibitors, and GLP-1 analogs all lower glucose but also lower insulin and cause weight loss. Since type 2 diabetes is a disease characterized by elevations in both blood glucose and blood insulin, these medications would be predicted to have the best outcome. And sure enough, that is the case. In a disease of too much insulin, lowering it creates benefits. These three categories of medications could easily be called the good (lowers insulin, body weight, and complications), the bad (neutral), and the ugly (increases insulin, body weight, and complications).

| Table 11.1. Oral hypoglycemics in type 2 diabetes: A comparison |
|-----------------|-----------------|-----------------|
| **Drugs**       | Weight loss     | Weight neutral | Weight gain     |
| Acarbose        | **GOOD**        | Metformin      | Insulin         |
| SGLT2 inhibitors|                 | DPP-4 inhibitors| Sulfonylureas   |
| GLP-1 analogues |                 |                | TZDs            |
| **Insulin levels** | Lowers insulin | Neutral        | Raises insulin  |
| **Cardiovascular outcomes** | Decreases hearts attacks and death | Neutral | Increases heart attacks and death |
| compared to metformin |                |                |                 |
| **Verdict?**    | GOOD            | **BAD**        | **UGLY**        |

The classic oral hypoglycemic agents were exclusively those that were insulin neutral or raised insulin levels. This explains how meta-analyses reviewing all the available literature up to 2016, including twenty randomized controlled trials, could only conclude that
such as bleeding and infection. Although gastric dumping syndrome is rare after this procedure, strictures are common. More importantly, perhaps, compared to the Roux-En-Y surgery, it leads to less weight loss and less durable results.

The gastric “lap” band
An even simpler surgery is the surgically implanted gastric “lap” band that wraps around the stomach. Like cinching a tight belt, the lap band restricts food from entering the stomach. No part of the healthy stomach is removed, and the lap band can be gradually tightened or loosened as needed. Because of its relative simplicity, this procedure has the fewest complications and can be used by anybody for weight loss. The main problem is that weight is often regained over time. One surgeon, a friend, remarked that the most common lap band surgery these days is its removal.

Figure 13.1. Gastric lap banding

In the short term, all types of bariatric surgery have been proved effective for weight loss and diabetes. Longer-term studies show varied effectiveness, depending upon the type of surgery. However, I do not
wish to praise or condemn any of these surgeries. As with everything else in medicine, they have their place. My main question is: what happens to type 2 diabetes after bariatric surgery? What does bariatric surgery teach us?

**WHY BARIATRIC SURGERY WORKS**

In virtually all cases, type 2 diabetes completely disappears after bariatric surgery. Type 2 diabetes is entirely reversible, even in a 500-pound patient with a twenty-year duration of disease. It is not only reversible, but rapidly reversible. In a matter of weeks, the diabetes disappears. Yes, it truly just goes away.

A 2012 trial called Systemic Therapy in Advancing or Metastatic Prostate Cancer: Evaluation of Drug Efficacy (STAMPEDE) compared the effects of gastric bypass surgery with intensive medical therapy (drug treatments) on obese type 2 diabetics with very high blood glucose levels. Surgical patients did amazingly well. Within three months, most patients stopped taking all their diabetic medications because
If I had a flood in my house…
I would not spend day after day, week after week, &
year after year buying buckets, mops and towels. I would not be
inventing different types of buckets and more expensive
mops or drainage systems to ensure the water drained away quickly.
I would find the source of the water and turn it off!

DR. VERNER WHEELOCK
disease and death by a jaw-dropping 75 percent. It should hardly have come as a surprise, as it confirmed what used to be called the French paradox.

In the 1980s and 1990s, people in France were eating saturated fat like it was going out of style, yet their death rate from cardiovascular disease was less than half what it was in the U.S. If saturated fat clogged arteries and led inexorably to heart disease, then how could the French possibly eat more fat and have less heart disease? The answer, in hindsight, is pretty obvious. Eating saturated fat does not lead to cardiovascular disease.6

The cardiovascular benefits of the relatively high-fat Mediterranean diet have since been replicated many times. Most recently, the 2013 PREDIMED study confirmed that patients on the Mediterranean diet reduced their rate of heart disease and death.7 Further comparison of different dietary habits in the countries of Europe in 2012 shows that higher saturated fat intake is associated with less heart disease.8 A 2009 meta-analysis9 demonstrated that saturated fat had no correlation to heart disease and offers slight protection against stroke. In Japan, this protection against stroke has also been noted.10 Slowly but steadily, the realization that diets high in natural fats are intrinsically healthy is gaining ground.

Figure 14.1. Higher dietary fat = lower risk of stroke and heart attack11
WHY TO REDUCE REFINED CARBOHYDRATES

IN 2001, IN a critical review of dietary fat and cardiovascular disease, Dr. Walter Willett of Harvard’s School of Public Health noted that, “It is now increasingly recognized that the low-fat campaign has been based on little scientific evidence and may have caused unintended health consequences.” Furthermore, as shown by Figure 14.2 from the Nurses’ Health Study, a very large, long-term observational study from Harvard University, he found a clear correlation between high glycemic load in the diet and the risk of heart disease.

Sugar and refined carbohydrates have a high glycemic load, which raises blood glucose and the risk of type 2 diabetes. This, in turn, significantly increases the risk of heart disease.

Figure 14.2. Higher glycemic load = higher risk of heart disease

A comprehensive 2013 review concluded that certain diets provide superior glycemic control. Specifically, four were found beneficial: the low-carbohydrate diet, low glycemic-index diet, Mediterranean diet, and high-protein diet. All four diets share a common trait: they reduce
Carbohydrate-reduced Diets

dietary carbohydrates to varying degrees. Low-carbohydrate diets have proven more effective at reducing body weight, waist size, and blood glucose.22

Figure 14.3. U.S. Macronutrient Consumption 1965–201123

Data from the National Health and Nutrition Examination Survey (NHANES) show that between 1965 and 2000, as the twin epidemics of obesity and type 2 diabetes unfolded, Americans primarily ate more carbohydrates and less dietary fat as a percentage of diet, just as the dietary guidelines recommended.24

Refined grains and sugars are the main sources of carbohydrates, and any low-carbohydrate diet should restrict these. Yet we need to make a further distinction between unrefined carbohydrates, such as potatoes and fruit, and refined carbohydrates, such as added sugars and flour, because the higher the intake of refined carbohydrates, the higher the risk of diabetes.25 The reason is that refined carbohydrates increase blood glucose higher and faster than unrefined ones. This effect becomes obvious when looking at glycemic load. Unrefined foods score low on the scale despite having similar amounts of dietary carbohydrates.
This distinction explains how many traditional societies can eat carbohydrate-based diets without evidence of disease. For example, the Tukisenta, a highland tribe of New Guinea, derive 94.6 percent of their energy intake as whole, unprocessed carbohydrates; and the Okinawans, a group living on a small island in southern Japan, eat a traditional diet that is almost 85 percent starch. Both groups eat mostly sweet potatoes. With virtually no sugar or refined grains such as flour, type 2 diabetes is almost non-existent. The native diet of Kitava, a small island off New Guinea, consists of 69 percent carbohydrates, mostly tubers (sweet potato, cassava, and yam), coconut, and fruit, but their average insulin level is lower than 90 percent of Swedes.

In other words, higher carbohydrate intake alone does not necessarily lead to higher insulin levels. Refining and processing plays a leading role in enhancing the insulin effect. Removing the natural fiber, fat, and proteins in foods leaves pure concentrated carbohydrates, a form not found naturally. Further grinding of these carbohydrates into a fine powder (such as flour) increases the speed of digestion, which results in higher blood glucose spikes. At the same time, we tend to eat more
Another patient just stopped her diabetes medication. Worried, I called her. She had lost so much weight, and looked so young that I thought it was the wrong patient. She started eating a low-carbohydrate diet where not just sugar but all sources of glucose are greatly reduced. Blood tests confirmed her diabetes was into full remission.

A week later, an article in the *British Medical Journal* caught my eye. Bread raised blood glucose more than table sugar. Disbelieving, I found to my utter amazement—it’s a fact! Starchy foods like bread, cereals, rice, or potato are “concentrated” sugar, digested into huge amounts of glucose. The Glycaemic Index predicts how various carbohydrate-containing foods will affect blood glucose. Changing the scale into equivalents of teaspoons of sugar led to some surprising results. (Note: This is for illustrative purposes only. Foods listed are not identical to sugar since sugar contains both fructose and glucose.)

**Figure 14.5.** How foods affect blood glucose: A comparison

<table>
<thead>
<tr>
<th>Food Item</th>
<th>G Index</th>
<th>Serve size (g)</th>
<th>How does each food affect blood glucose compared with one 4g teaspoon of table sugar?</th>
</tr>
</thead>
<tbody>
<tr>
<td>Boiled rice</td>
<td>69</td>
<td>150</td>
<td>10.1</td>
</tr>
<tr>
<td>Boiled potato</td>
<td>96</td>
<td>150</td>
<td>9.1</td>
</tr>
<tr>
<td>French fries</td>
<td>64</td>
<td>150</td>
<td>7.5</td>
</tr>
<tr>
<td>Spaghetti, boiled</td>
<td>39</td>
<td>180</td>
<td>6.6</td>
</tr>
<tr>
<td>Sweet corn, boiled</td>
<td>60</td>
<td>80</td>
<td>4.0</td>
</tr>
<tr>
<td>Frozen peas, boiled</td>
<td>51</td>
<td>80</td>
<td>1.3</td>
</tr>
<tr>
<td>Banana</td>
<td>62</td>
<td>120</td>
<td>5.7</td>
</tr>
<tr>
<td>Apple</td>
<td>39</td>
<td>120</td>
<td>2.3</td>
</tr>
<tr>
<td>Wholemeal, small slice</td>
<td>74</td>
<td>30</td>
<td>3.0</td>
</tr>
<tr>
<td>Broccoli</td>
<td>54</td>
<td>80</td>
<td>0.2</td>
</tr>
<tr>
<td>Eggs</td>
<td>0</td>
<td>60</td>
<td>0</td>
</tr>
</tbody>
</table>

Armed with this new knowledge, I started to treat all motivated diabetic patients in my practice with a low-carbohydrate diet. So far after four years, 160 patients have tried it, with amazing results:
Carbohydrate-reduced Diets

For more than a decade, guidelines from the Joslin Center’s weight management program have advised clients to reduce their intake of refined carbohydrates to less than 40 percent of total calories. The result? Clients have lost more than 10,000 pounds of weight, improved their diabetes, and reduced their medications.

THREE RULES FOR REVERSING TYPE 2 DIABETES

Once we understand how type 2 diabetes and insulin resistance develop, we can implement strategies that carry a reasonable chance of reversing it. Here are my top three food “rules” for reducing blood glucose, reducing insulin, and reversing type 2 diabetes.

Rule#1: Avoid fructose
The most important rule, without exception, is to eliminate all added sugars from your diet. Recall that insulin resistance is the result of fatty liver becoming overfilled and unable to accept more glucose. The most important determinant of fatty liver is not just carbohydrates, but the fructose contained in sucrose (table sugar) and high-fructose corn syrup.

Figure 14.6. The top dietary sources of fructose

![Pie chart showing dietary sources of fructose]
INTERMITTENT FASTING VERSUS CONTINUOUS CALORIC REDUCTION

DEATH VALLEY, CALIFORNIA, has an average temperature of 77 degrees Fahrenheit (25°C). Sounds perfect, doesn’t it? Yet most residents would hardly call the temperature idyllic. Summer days are scorching hot and winter nights are uncomfortably cold.

Consider that jumping off a foot-high wall a thousand times is far different than jumping off a thousand-foot-high wall once. The difference between the two is literally the difference between life and death.

Would you prefer to experience seven gray, drizzling days with an inch of rain each, or six sunny, gorgeous days followed by a day of heavy thundershowers with 7 inches of rain?

The point, as Figure 15.1 shows, is that averages don’t tell the whole story.

Figure 15.1. Averages don’t tell the whole story

In all these examples, it’s obvious that averages represent only one facet of the story. The frequency of the event is of paramount importance. So why would we assume that reducing 300 calories per day
The most important conclusion was that fasting was a safe and effective therapy that anybody could reasonably follow. The fasting group not only lost more weight, but also almost twice as much of the more dangerous visceral fat. The portion-control group lost lean mass in addition to fat, but the fasting group did not. Lean mass percentage increased by 2.2 percent with fasting compared to only 0.5 percent with portion control. In other words, fasting is four times better at preserving lean mass. (So much for that old “fasting burns the muscle” myth.)

So why isn’t fasting more popular, despite its proven success? One of the biggest deterrents is the starvation myth.

**OVERCOMING THE STARVATION MYTH**

*The Biggest Loser* is a long-running American TV reality show that pits obese contestants against one another in a bid to lose the most weight. The weight-loss regimen has two components: a calorie-restricted diet calculated to be approximately 70 percent of each contestant’s energy requirements, typically 1200 to 1500 calories per day, combined with an intensive exercise regimen that is typically far in excess of two hours a day.
starvation! Or at least the controlled version: intermittent fasting. Fasting triggers numerous hormonal adaptations that do not happen with simple caloric reduction. Insulin drops sharply, preventing insulin resistance. Noradrenaline rises, keeping metabolism high. Growth hormone rises, maintaining lean mass.

Controlled experiments prove this point. Over four days of continuous fasting, basal metabolism (measured as resting energy expenditure, REE) does not drop. Instead, it increases by 12 percent. The VO2, another measure of basal metabolism that tracks the amount of oxygen used per minute, similarly rises.12 Many other studies have confirmed these findings. Twenty-two days of alternate daily fasting also did not result in any decrease in basal metabolic rate.13

Figure 15.3. Metabolic changes over four days of fasting14

Remember the portion-control versus fasting study in the previous section? The portion-control strategy dropped basal metabolism by 76 calories per day. By contrast, fasting was not associated with any statistically significant drop in energy expenditure. In other words, daily caloric reduction causes starvation mode where fasting does not.

The study concluded: “Importantly, ADF (Alternate Daily Fasting) was not associated with an increased risk for weight regain.”
engineering, and molecular biology, the problem only grows worse and has now engulfed the entire world, reaching across all genetic boundaries. It's time to stop pretending type 2 diabetes is a chronic and progressive disease, and it's time to stop treating it that way. Clearly type 2 diabetes is a dietary and lifestyle disease. To pretend otherwise is pure self-deception.

But here's what is important. A dietary disease requires a dietary treatment. And since weight gain clearly plays a prominent role in the development of type 2 diabetes, weight loss must similarly play a large role in its reversal. We know that bariatric surgery, very low-carbohydrate diets, and fasting are well-known treatments for type 2 diabetes and they are proven to cure. We also know that insulin, oral hypoglycemics, and low-fat diets can lower blood glucose but do nothing to cure type 2 diabetes.

Figure 15.4. Dietary disease; dietary treatment

The treatments that cure all show one common characteristic. They lower insulin. Since type 2 diabetes is a disease of hyperinsulinemia, it is only logical that these treatments are beneficial. And what do all the treatments that do not cure type 2 diabetes have in common? They raise insulin. And in fact, using these treatments worsens diabetes over time.
SAMPLE 1:
MEAL PLAN FOR A 36-HOUR FASTING PERIOD

<table>
<thead>
<tr>
<th>MEAL</th>
<th>Sunday</th>
<th>Monday</th>
<th>Tuesday</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breakfast</td>
<td>Mini Bacon-Wrapped Egg Frittatas</td>
<td>FAST</td>
<td>Western Omelet with Sausage</td>
</tr>
<tr>
<td>Lunch</td>
<td>Arugula and Prosciutto Salad</td>
<td>FAST</td>
<td>Chicken Drumsticks Wrapped in Bacon with Slices of Celery and Carrots</td>
</tr>
<tr>
<td>Dinner</td>
<td>Almond Flour and Pork Rind–Breaded Chicken Tenders</td>
<td>FAST</td>
<td>Beef Stir Fry</td>
</tr>
</tbody>
</table>
### Appendix: Two Sample Week-Long Meal Plans

<table>
<thead>
<tr>
<th>Wednesday</th>
<th>Thursday</th>
<th>Friday</th>
<th>Saturday</th>
</tr>
</thead>
<tbody>
<tr>
<td>FAST</td>
<td>Bacon, Scrambled Eggs, and Avocado</td>
<td>FAST</td>
<td>Coconut Flour Pancakes with Whipped Cream and Berries</td>
</tr>
<tr>
<td>FAST</td>
<td>Chicken-Stuffed Bell Peppers</td>
<td>FAST</td>
<td>Pear and Arugula Salad with Pine Nuts</td>
</tr>
<tr>
<td>FAST</td>
<td>BBQ Shrimp Skewers</td>
<td>FAST</td>
<td>Pulled Pork Sliders on Almond Flour Buns</td>
</tr>
</tbody>
</table>
## Sample 2: Meal Plan for a 30-Hour Fasting Period

<table>
<thead>
<tr>
<th>MEAL</th>
<th>Sunday</th>
<th>Monday</th>
<th>Tuesday</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breakfast</td>
<td>Scrambled Eggs, Smoked Salmon, and Avocado</td>
<td>FAST</td>
<td>Hard-Boiled Eggs, Cauliflower Hash Browns, and Asparagus</td>
</tr>
<tr>
<td>Lunch</td>
<td>Lemon Butter and Pepper Chicken Wings, Celery, and Carrots</td>
<td>FAST</td>
<td>Chicken &quot;Breaded&quot; in Pork Rinds with Green Beans</td>
</tr>
<tr>
<td>Dinner</td>
<td>FAST</td>
<td>Grilled Salmon with Garden Salad</td>
<td>FAST</td>
</tr>
</tbody>
</table>
## Appendix: Two Sample Week-Long Meal Plans

<table>
<thead>
<tr>
<th>Wednesday</th>
<th>Thursday</th>
<th>Friday</th>
<th>Saturday</th>
</tr>
</thead>
<tbody>
<tr>
<td>FAST</td>
<td>Mushroom Omelet</td>
<td>FAST</td>
<td>Chia Pudding</td>
</tr>
<tr>
<td>FAST</td>
<td>Steak Fajitas</td>
<td>FAST</td>
<td>Tomato, Cucumber, and Avocado Salad</td>
</tr>
<tr>
<td></td>
<td>Zucchini Pasta in Avocado Pesto with Stir-fried Vegetables</td>
<td>FAST</td>
<td>Ginger Chicken Lettuce Cups with Baby Bok Choy</td>
</tr>
</tbody>
</table>