

One in Three People Have or Will Get Diabetes...

Are You One of Them and
Don't Even Know It?

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We are in the midst of a worldwide diabetes epidemic,¹ including in the US, where more than 115 million adults age 20 and over have either diabetes or prediabetes.² Of that number:

- 29.1 million already have type 2 diabetes—a statistic that researchers predicted in 2001 wouldn't be reached until 2050³
- 86 million have prediabetes, up from 79 million in 2010⁴
- All told, nearly a THIRD of the 320 million people living in America today have either prediabetes or some form of diabetes⁵
- Globally, the cost of obesity is \$2 trillion a year⁶



What's shocking is that 9 out of 10 those with prediabetes;⁷ and 27.8 percent with diabetes don't even know they have it.⁸

Diagnosed or not, this is an astounding amount of people with a disease that is almost completely preventable and most certainly reversible! When you consider that many countries around the world are now reporting the disease as epidemic—even in areas that only a couple decades ago had almost no diabetes at all—you can't help but wonder what's happening.

Why is such an insidious epidemic overtaking the world?

Add in Children and the Cost Is Staggering

Among children, juvenile-onset (type1) diabetes has increased too—by 30 percent over an 8-year period, with the greatest prevalence occurring in older adolescents aged 15-19 years old.⁹

Add in the 15,000 children being newly diagnosed every year, and the numbers for the future become mind-boggling.¹⁰

And that brings us to the cost of diabetes: in terms of medical expenditures and lost productivity, the annual medical cost of diabetes in the US as of March 2013 (latest year available) is \$245 billion.¹¹ If you add in reduced productivity, that's another \$78 billion, for a grand total of \$322 billion a year spent on diabetes—48 percent higher than what we spent in 2007.¹²

The bottom line is diabetes in the US has increased over 300 percent in just 15 years,¹³ and the cost in terms of lives (diabetes is the 7th leading cause of death in the US¹⁴) is spiraling.

And what that means is that unless something happens very quickly to change this dire statistic, one out of every three of you reading this report will likely develop diabetes or pre-diabetes in your lifetime and possibly even die from it!

This suggests two very important points:

- Although genetic susceptibility¹⁵ may increase a person's risk of getting diabetes, this disease cannot be primarily genetic, since many of the prior statistics were recorded within the same generations, with essentially the same genetics.
- Something we've been doing is obviously making us sick, and we need to change it.

That something is composed of three things: diet, physical activity, and exposure to environmental elements known to contribute to the risk of diabetes.

The most important thing to understand is that type 2 diabetes is nearly completely preventable, and can be controlled or even reversed with diet and lifestyle modifications. In extreme cases bariatric/metabolic surgery can also put diabetes into remission, but before diving into the best way to end this devastating disease, I would like to give you a foundation for understanding the underlying factors that can put you at risk for it.¹⁶

What Is Diabetes?

Diabetes mellitus, usually referred to simply as diabetes, is a metabolic disorder which causes elevated levels of glucose in the blood. These levels can be triggered or exacerbated by a number of factors such as obesity or even ingestion of certain drug classes [such as statins](#). There are three main types of diabetes: Type 1, Type 2, and gestational diabetes.

Sometimes referred to as insulin-dependent or juvenile diabetes, type 1 is an autoimmune disease that typically occurs in children and young adults.

Type 2, sometimes referred to as non-insulin-dependent diabetes, is the most common form of diabetes and is caused by several factors, with obesity being a major risk.

Gestational diabetes occurs during pregnancy when hormone changes in the woman's body sometimes alter the body's ability to produce enough insulin to metabolize glucose levels. Normally, gestational diabetes goes away after the woman gives birth, although she may remain at high risk for developing type 2 diabetes later.

Other rare types of diabetes can sometimes be caused by certain endocrine diseases, autoimmune disorders, genetic mutations, or in conjunction with syndromes such as Down's, Klinefelter, and Turner.^{17, 18, 19}

Diabetes is not something to ignore: in 2010, it was the seventh leading cause of death in the US. Unfortunately, studies show that diabetes may be underreported as a cause of death, since only about 35-40 percent of people who died with diabetes had it listed anywhere on their death certificate.²⁰

Type 1 Diabetes

In **type 1 diabetes**, your body's own immune system destroys the insulin-producing cells of the pancreas, resulting in a *deficiency of the hormone insulin*. Since your body needs insulin to survive, persons with type 1 diabetes usually end up on insulin therapy to replace what the pancreas doesn't make.

Previously known as juvenile diabetes, type 1 typically occurs in children and young adults, although it sometimes develops in adulthood as latent autoimmune diabetes, or LADA.²¹

Type 1 is relatively uncommon: only 5 percent of people with diabetes have this form. However, studies show that its incidence has been increasing dramatically in the past few decades, particularly among non-Hispanic white youth.^{22, 23} For example, in the city of Philadelphia alone, studies show that since 1985, type 1 diabetes in non-Hispanic white youth age 4 and under has skyrocketed by 70 percent.



Even more significant, though, is that for black children it's **gone up 200 percent!** And while the numbers for youth ages 10-14 have increased by "only" 24 percent, similar numbers are shown in Europe and Israel, with these numbers predicted to **DOUBLE** by 2020.²⁴

There is no known cure for type 1 diabetes, and once it's diagnosed, it's a lifelong condition. While genes play an important part in whether or not you're going to get type 1, research suggests that your own body's insulin may also trigger it, causing the body's immune system to produce antibodies against its own beta cells. Research also shows that environmental factors such as viruses and infections might trigger autoimmune reactions that can lead to type 1 diabetes. Viruses being investigated for this are coxsackievirus, cytomegalovirus, adenovirus, and mumps (see additional information below under "Other Possible Causes of Type 1 Diabetes").²⁵

Vitamin D Status and Type 1 Diabetes

Research has shown that our preoccupation with sun avoidance may play a role in the development of insulin-dependent diabetes. It's well established that the further you move away from the equator the greater the risks of vitamin D deficiency-related autoimmune diseases such as multiple sclerosis, rheumatoid arthritis, and inflammatory bowel disease. But new evidence shows that type 1 diabetes should be added to this list.^{26, 27} That's why some scientists have suggested that dietary vitamin D3 supplementation in infants and children might be one way to quell the increasing incidence of type 1 diabetes.^{28, 29, 30}

A major key to preventing this illness could be to confirm that pregnant women have optimal vitamin D stores, and in fact, researchers attempted a study on this in 2012. Unfortunately, the study's authors couldn't determine whether better D3 (serum 25(OH)D) concentrations during pregnancy make a significant difference in children's risk for type 1 diabetes or not — **mainly because "such a large proportion of mothers were vitamin D-deficient or -insufficient."** In other words, so many mothers are deficient in vitamin D that researchers couldn't perform the study!³¹

Because there is also strong evidence that low vitamin D3 levels can increase a child's risk of autism^{32, 33} this should be a bellwether for childbearing-aged women to get their D3 levels tested and then supplement to optimal levels if necessary, both before and during pregnancy. Once the child is born, it's possible that ensuring the child gets adequate exposure to sunshine and/or wise use of appropriate oral vitamin D supplementation could lessen the risk for autism. (Breastfeeding mothers who are supplementing with vitamin D themselves probably should consult the child's pediatrician before giving their babies an additional supplement.³⁴)

Other Possible Causes of Type 1 Diabetes

Research has shown that other triggers, both environmental and viral, can possibly increase the risk of developing type 1 diabetes. For example, congenital rubella has been clearly associated with it^{35, 36}, as well as mumps, parvovirus and cytomegalovirus (CMV).^{37, 38}

More recently, some studies have shown that rotavirus might "push" diabetes along in genetically susceptible children^{39, 40, 41}. It's also been found that having had an infection with an enterovirus makes children 48 percent more likely to have developed type 1 diabetes.^{42, 43, 44} And, several studies have shown that vaccines⁴⁵ also possibly trigger it, although some literature argues the evidence.^{46, 47, 48, 49}

Environmentally, it's been suggested that an excessive focus on a germ-free environment might actually be contributing to type 1 diabetes and other autoimmune disorders.⁵⁰

Did you play in the dirt when you were a child? As it turns out, exposure to the natural biota of the earth—"friendly" bacteria—actually provided you with an important immune system function, helping your body build up natural defenses against diabetes and other autoimmune disorders. Experts have hinted for several years that childhood allergies, inflammatory diseases, and diabetes might be increasing specifically because parents in developed countries today keep their children away from dirt, germs, viruses, and grime.^{51, 52}

And as the evidence continues to mount,⁵³ the lesson is that it's okay to let your child play outside and get dirty. Use plain soap—not antibacterial—and water for washing, and avoid antibiotics unless absolutely necessary.

Type 2 Diabetes

Type 2 diabetes is by far the most common form of diabetes, affecting 90-95 percent of people diagnosed with diabetes.

If you have type 2, your body is producing insulin, but either because your pancreas isn't making enough, or because it simply can't use the insulin well enough, you become what is called "insulin resistant." This causes glucose to build up in the body instead of circulating into your cells, causing a variety of problems.

Anyone can get type 2 diabetes and, except for gestational diabetes, it's a gradual process that evolves over a long period of time as it moves from a state of "impaired glucose tolerance" to full-blown diabetes. Any one or more of a number of factors can trigger pre-diabetes, which can eventually lead to diabetes.



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The National Diabetes Education Program⁵⁴ lists the following risk factors that can trigger prediabetes and eventually contribute to your becoming diabetic. It's an interesting list—who would have guessed that depression is a forerunner of diabetes or that simply having high triglycerides can give you diabetes? If you're wondering how that's possible, the answer is that the DRUGS you're taking for these conditions are the real risk factors, not that you're depressed or have elevated cholesterol levels.

That's why I urge you to keep reading for important information I'm going to share with you later on how you can prevent diabetes by avoiding some of these drugs

Risk Factors for Type 2 Diabetes

Age 45 or older	Overweight or obese
Family history of diabetes	Hypertension
Physical inactivity	Depression
History of gestational diabetes	Atherosclerotic cardiovascular disease
HDL-C levels under 35mg/dL	Fasting triglycerides over 250 mg/dL
Treatment with atypical antipsychotics, glucocorticoids	Obstructive sleep apnea and chronic sleep deprivation
Certain health conditions associated with insulin resistance	Member of high-risk population (African American, Hispanic/Latino, Native, or Asian American)

Source: *The National Diabetes Education Program*

Chances are if you visit your doctor and have one or more of these risk factors, or if your blood glucose levels (more on that later) are elevated, you'll be checked for diabetes and put on insulin, either in pill form or by injection, to control it. But before you get to that point, you need to know there things you can do RIGHT NOW to lessen or even eliminate these risk factors. In the meantime, here are some of the warning signs that you may already have diabetes:⁵⁵

Excessive thirst and increased urination	Extreme hunger (even after eating)
Dry, itchy skin	Unusual weight gain or loss
Increased fatigue and overall weakness	Irritability
Blurred vision	Slow healing of wounds
Frequent infections (skin, urinary, vaginal)	Numbness or tingling in hands and/or feet

If you are experiencing these symptoms, medications and supplements are **not** the answer for type 2 diabetes. As I explain later in this eBook, this disease and its forerunners, pre-diabetes and metabolic syndrome, can be controlled by restoring your insulin and leptin sensitivities with certain behavior changes such as eliminating grains and sugars in your diet, getting enough good fats, exercising, and sleeping well.

What Tests Can Show That I Have Diabetes?

Methods for diagnosing diabetes have changed significantly in the past few years. A decade and more ago, doctors used to simply administer a fasting glucose tolerance test to determine whether you had diabetes.

Today, they can utilize several different tests, along with something called the "A1C," which measures your average blood glucose for the past two to three months. The A1C can be determined with a drop of blood from a simple finger prick. The other tests include more invasive blood draws through a vein, which is done after you drink a specially sweetened beverage.

The following is a list of the tests.⁵⁶ For more information on them and how they're administered, you can visit the National Diabetes Information Clearinghouse [here](#):⁵⁷

- **A1C** – Sometimes called the hemoglobin A1 c, there is no need to fast. An international team on diabetes recommended the A1C in 2009 for diagnosing type 2 diabetes as well as prediabetes. The test measures protein in red blood cells and is reported as a percentage. The higher the percentage, the higher your blood glucose levels have been. A normal A1C level is below 5.7 percent. If you have A1C numbers of 6.5 or higher, you have diabetes. Prediabetes is the diagnosis if your A1C is between 5.7 and 6.4.
- **Fasting Plasma Glucose (FPG)** – You must be fasting to take this test, meaning that other than water, you haven't had anything to eat or drink for at least 8 hours. Glucose levels are measured from blood samples and diabetes is diagnosed when your level is greater than or equal to 126 mg/dL. You have prediabetes if the levels are 101 to 125. Normal is 70-100.
- **Oral Glucose Tolerance Test or OGTT** – With this test you're given a special sweet drink. Your blood glucose levels are measured just before you drink it, and two hours after. You're diagnosed with diabetes if your blood glucose is equal to or greater than 200 after two hours. You have prediabetes if your numbers are 140 to 199.

- **Random, or Casual Plasma Glucose Test** – This is a blood check that your doctor may do if you have immediate, severe diabetes symptoms. Diabetes is diagnosed if your blood glucose is equal to or greater than 200 mg/dL.

What Is Insulin Resistance?

If you are insulin-resistant, it means that glucose is building up in your blood because your body is not using its insulin effectively. This in turn starves your fat, muscle, and liver cells, which causes your body to signal the pancreas to make more insulin in an attempt to make up for what those cells aren't getting. It quickly becomes a vicious circle that can lead to prediabetes and, ultimately, diabetes. Unfortunately, many people develop type 2 diabetes because they don't know they're insulin-resistant until it's too late.

What Is Prediabetes?

Prediabetes is a term used to describe a state of progressing insulin resistance (also called impaired glucose tolerance), in which your blood glucose levels are higher than normal but not quite high enough to actually be called diabetes. As noted above, you may be diagnosed with prediabetes if your glucose numbers are between 100 and 125. Having prediabetes is a risk factor that you may get type 2 diabetes in the future.

Sometimes referred to as metabolic syndrome, prediabetes is far easier to turn around in the earlier stages than the later stages, so if you're diagnosed with it, the time to put it in check is NOW. You can reset your numbers by achieving a healthy weight through diet and exercise.

Metabolic Syndrome

As insulin resistance increases, your skeletal muscles are no longer able to make glycogen, a form of stored carbohydrate, from food energy. In turn, there is [an increase in fats in your bloodstream](#), which leads to high triglyceride levels and increased body fat—especially abdominal fat.

This group of symptoms—including diabetes, pre-diabetes, high triglycerides, high blood pressure, increased belly fat, and insulin resistance—is sometimes referred to as metabolic syndrome, which used to be called Syndrome X. It's believed that, today, 47 million Americans may have metabolic syndrome.⁵⁸



Once thought to be caused primarily by a combination of poor diet, genetic factors, a sedentary lifestyle, and belly fat, metabolic syndrome is not just a “fat” person’s disease: It can also be diagnosed in thin people who have one or more of the diabetes risk factors.

And it's true: if you're not exercising or paying attention to what you eat, dangerous fat can build up around your internal organs (called visceral fat), regardless of whether or not you appear overweight on the outside.⁵⁹

That said, let's dispel a very common myth.

Diabetes Is Not a Disease of Blood Sugar!

But wait a minute—isn't that what we've been told for decades? That diabetes is a malfunction of your blood sugar? At first glance it makes sense, since doctors measure glucose levels in your blood to diagnose diabetes.

But the reality is diabetes is a **disease of insulin**, a hormone your body needs to convert sugars, starches and other foods into energy. When your body becomes insulin-resistant, the path to diabetes begins.⁶⁰

Perhaps more importantly, diabetes is also a malfunction of leptin signaling. Leptin is a hormone discovered in 1994 by Jeffrey M. Friedman and Douglas Coleman, who found that it regulates food intake (appetite) and body weight. Interestingly, Friedman named leptin after the Greek word *leptos*, which means "thin," after he discovered that mice injected with synthetic leptin became more active and lost weight.

But since the majority of obese people have very high levels of leptin in their blood, Friedman determined that these high levels must be associated with a resistance to leptin—in other words, the signaling pathway for leptin becomes skewed, causing the body to over-produce leptin just as it does insulin when you are insulin-resistant. Friedman's work also showed that leptin is a key player in your body's satiety response, which signals to your brain that you're full so you will stop eating.

Friedman and Coleman were awarded the Albert Lasker Award for Basic Medical Research—the most prestigious American prize in science—for their work. And in the past 20 years, this ground-breaking, leptin discovery has led to new treatments for obesity, diabetes, and other metabolic conditions, including metabolic problems in extremely lean women.⁶¹

How Leptin Resistance Causes Obesity and Contributes to Diabetes

One expert in leptin resistance and its role in making you diabetic is Dr. Richard Johnson, head of nephrology at the University of Colorado. Dr. Johnson has been an important contributor to my articles on sugar, obesity and diabetes.⁶² His book, *The Fat Switch*, shatters many of our age-old myths about diet and weight loss.

Referring to how leptin works with insulin and ghrelin, a hormone released from the stomach, to help control our appetite, Johnson explains that when any one or all of these regulators are disrupted, your body responds by triggering an alteration in metabolism, which in turn switches off the insulin, ghrelin and leptin signals that regulate hunger. With leptin, Dr. Johnson says:⁶³

“...resistance results in increased food intake, and a block in energy production leads to preferential conversion of the energy into fat. Therefore, the major reasons we are eating too much is not because we are given larger plates of food, but rather because we are now more hungry and want larger plates of food.”

And because hunger is a powerful, innate mechanism, if we're hungry when we shouldn't be and we eat too much of the wrong thing too often, the inevitable occurs: we begin to show signs of metabolic syndrome, prediabetes and, ultimately, diabetes.

We already know that weight loss can help control both type 2 diabetes and hypertension, particularly if the weight is lost soon after you've been diagnosed with diabetes.⁶⁴ So it makes sense that since the primary hormone that controls hunger is leptin, it's important to know how to "reset" the switch that makes leptin work.

Elevated insulin levels have been associated with other co-existing conditions including:^{65, 66}

- Heart/coronary artery disease
- Peripheral vascular disease
- Stroke
- High blood pressure
- Arthritis
- Fatty liver disease
- Hyperlipidemia
- Cancer
- Obesity
- Blindness
- Kidney failure

These comorbidities have become so costly in terms of both health and economics that *The American Journal of Managed Care* released a study⁶⁷ in February 2015, showing just how prevalent they are. Looking at more than 4 million patient records and focusing ONLY on patients with type 2 diabetes, they found that between 2008 and 2012:

- 88% had at least 1 of 14 comorbidities
- 51% had 3 or more comorbidities
- 19% had a combination of all three of the following chronic conditions: hypertension, hyperlipidemia and obesity

What's alarming is that these diabetics most likely were also receiving drugs to treat their other chronic conditions—meaning that besides fighting diabetes, they are also victims of something called "polypharmacy"—a condition in which you are taking four or more medications every day. In financial cost alone, statistics show that 66 percent of total US health care spending is associated with the care of those who have multiple chronic conditions.⁶⁸

But when you consider that over half of Medicare beneficiaries age 65 and older not only have five or more chronic conditions,⁶⁹ but also are filling 31 different prescriptions per person, per year,⁷⁰ it's not an understatement to say that until we address the real cause of all this sickness, we are basically [playing Russian Roulette](#) with our health.

Diabetes, like all chronic disease, results from the miscommunication of messages between and within your cells—in this case, it's a disease of insulin and leptin miscommunication.

The Many Roles of Insulin

Your doctor will likely tell you that the purpose of insulin is to lower blood sugar. But this is part of a widespread misconception. Insulin's true evolutionary purpose is to work with the pancreatic hormone glucagon to both regulate and store excess nutrients. In fact, one of the primary roles of insulin is to control storage of glucose in fat cells.⁷¹

Insulin and glucagon have crucial roles in this regulatory process and, like partners in a dance, they have to keep in step with each other to maintain normal blood glucose levels. The fact that insulin lowers blood sugar is simply a side effect of its job.⁷² On the flip side, [glucagon's job](#) is to raise blood sugar when it detects a dip in glucose levels.



Insulin has three main functions⁷³ in your body. They are:

- Help muscle, fat and liver cells absorb glucose
- Stimulate liver and muscle tissue to store excess glucose
- Lower blood glucose levels by reducing glucose production in the liver

Your body needs sugar to survive. You can't live without it—in fact, your brain is so dependent on it that a 20 percent decline in blood sugar can lead to nausea and neurological symptoms.⁷⁴ But your body also needs insulin to facilitate that energy getting to all the cells of the body, because they can't uptake the sugar directly.

So insulin both “feeds” your body sugar and stores it. When your body notices that your blood sugar is elevated, it's a sign that you have more glucose/energy in your blood than you need, and you aren't burning it fast enough, so it's accumulating. Therefore, insulin is released to take that sugar and store it in an effort to maintain balance in your cells.

And the more your glucose levels rise, the more insulin your pancreas will release, and the more that will get stored.

Insulin and Glucagon Work Together

Insulin directly impacts many cells in your body, especially the muscle, red blood, and fat cells. Most of the excess glucose is stored in fat cells, with just a small portion stored as glycogen. Glycogen is used for quick, immediate energy, the type your body needs for basic life functions.⁷⁵ When your body becomes saturated with glycogen, the excess goes to fat cells in your liver, where it's tapped by glucagon when your body has used all its immediate energy stores and needs extra energy, such as between meals or when you're exercising.

Like a dance, this all works well as long as your insulin and glucagon are in step with each other. When they become disrupted and fall out of step, and the body fails to use insulin effectively, glucose builds up instead of being absorbed. This is called insulin resistance, which we already know can lead to metabolic syndrome, prediabetes and diabetes and, eventually, "treatment" with insulin injections or pills to help it get back in step.

Insulin also has other functions, including:

- Storing magnesium⁷⁶
- Retention of sodium⁷⁷
- Mediating blood lipids⁷⁸
- Stimulating cell proliferation and cell division⁷⁹
- Helping to regulate growth hormone⁸⁰ and sex hormones⁸¹
- Helping to maintain calcium and phosphorus in bone tissue⁸²

Insulin's purpose might even go far beyond the above list, and is now the focus of some fascinating research by scientists, who have discovered that lowering your insulin levels may add years to your life.^{83, 84}

Leptin: Is It the Missing Link Between Obesity and Diabetes?

Leptin, which is produced in fat cells as well as in several organs of your body,⁸⁵ is a key hormone that regulates both appetite and weight loss. It's secreted⁸⁶ by white adipose tissue, called "white fat," and it tells your brain when to eat, how much to eat, and most importantly, when to stop eating. It also tells your brain what to do with the energy it has. Working together with a hormone called ghrelin, leptin controls long-term energy regulation, while ghrelin works short-term, playing a role in telling your brain it's time to eat.⁸⁷

[Dr. Byron Richards](#) is a board-certified clinical nutritionist who was the first to explain the meaning of over 7,500 studies on leptin and its link to solving obesity. In his book, *Mastering Leptin*,⁸⁸ he calls leptin the most powerful hormone in the human body. Most importantly, Richards says that leptin is essential to life itself: it can function without any help from other hormones—but the other hormones can't function without leptin!

Once you understand just how important leptin is to your body, it's easy to see why Richards says:

"One thing is very clear, mastering leptin transforms the subjects of weight management and disease prevention to an astounding new level, opening the door for solving many major health issues that have not yet been successfully improved for the majority of Americans."

As Dr. Richards hints, when leptin was first discovered it generated a flurry of excitement that, perhaps, scientists had finally found a magic bullet to ending obesity. But as I mentioned earlier, scientists were stymied when they learned that some very obese people actually have high amounts of leptin circulating in their bodies—which told them that, like insulin resistance, leptin *resistance* could be a factor in both obesity and diabetes.

The research on leptin resistance and its role in obesity and diabetes is exciting and still ongoing, but in the meantime it's been discovered that leptin is critically involved in many of your body's functions, including:⁸⁹

- Regulation of blood circulation and blood pressure
- Regulation of nerve activity within the endocrine system including the kidney and adrenal glands
- Prevention of blood clots⁹⁰
- Making new bone⁹¹
- Body temperature regulation⁹²
- Human reproduction and lactation⁹³

So far, leptin studies have shown that leptin plays significant, if not primary, roles in heart disease, obesity, diabetes, osteoporosis, autoimmune diseases, reproductive disorders, and perhaps the rate of aging itself.

Yet, as crucial as leptin is, it's rarely, if ever, addressed by the medical community that seems bent on following old diabetes treatment paradigms in spite of these new discoveries. Coupled with what we know about how insulin interplays with ghrelin, is it any wonder that diabetes hasn't been conquered?

The only known way to reestablish proper leptin (and insulin) signaling is through proper diet. But first, here's one more, critical component to the cause—and cure—of diabetes.

Carbohydrates and Sugar: The Good, the Bad, and the Ugly

Over time, as your cells are exposed to excess insulin, they're going to become more insulin resistant. Meaning, it will take more insulin to get the job done.

The more insulin resistant you become, the higher your risk of developing not only diabetes but a number of chronic diseases. You also will show faster signs of aging.

But what if I told you there is no reason why you shouldn't live to be a healthy centenarian? Read on to learn how following a diet and lifestyle that is healthful for you could help your body last as much as 130 to 140 years.

Carbohydrates: Are They All Bad?

No. Everyone needs a certain amount of carbohydrates, but some need far less than others. It all depends on your nutritional type, which is determined by your individual biochemistry, how active you are and what medications you take. That's right: certain medications not only can send your glucose levels up, but have been shown to *actually make you diabetic*. So before we talk about carbs, here's a list of some of the "diabetogenic" drugs that can do this. They are:

- Statins^{94, 95} (used for lowering cholesterol)
- Corticosteroids⁹⁶ (used for reducing inflammation)
- Thiazide diuretics⁹⁷ (used for reducing high blood pressure)
- Beta Blockers⁹⁸ (used for treating high blood pressure)
- Atypical anti-psychotics⁹⁹ (used for treating schizophrenia, bipolar disorder, and other psychotic symptoms)



There are other drugs that can elevate your risk for diabetes such as antivirals used to treat infections like HIV,¹⁰⁰ but these are the main ones. So if you're serious about curing your diabetes, you may want to start by working with your physician to get off these drugs for good. (It can be dangerous or even life-threatening to end some drugs "cold-turkey," so don't do this without your physician's help.)

The Truth About Carbs

[Our addiction to grains](#), potatoes, and sweets means most of us are consuming far too many carbohydrates that can lead to serious chronic health problems like diabetes.

The worst insulin-offenders are processed foods with refined sugars and grains in them, trans fats, cereals, soda, candy, white bread, white rice, white potatoes, and high fructose corn syrup. So how do you determine if you are eating more carbohydrates than your body needs?

If you are experiencing any of the following symptoms, chances are excess carbs are at least partly to blame:

- ✓ Excess weight
- ✓ Fatigue and frequent sleepiness
- ✓ Depression
- ✓ Brain fogginess
- ✓ Bloating
- ✓ Low blood sugar
- ✓ High blood pressure
- ✓ High triglycerides

Whether you have diabetes or not it's important to know that the food you choose is the foundational strategy for taking control of your health. The American Diabetes Association recommends keeping track of how much and what type of carbs you eat because they can affect your insulin levels considerably.

My new and improved [nutrition plan](#) can help you make good carb choices, especially if, like most people with an insulin problem, you've been consuming a diet consistently high in sugar and grains.

But remember: how much you eat, how physically active you are, and even the types of medication you're on can all affect your insulin balance—meaning you have to take these things into consideration when you're choosing your carbs. My recommendation for most people is to follow a fairly low-carb diet. You can do this by:

- Avoiding refined grains, sugars and flour, processed foods, and foods containing added sugars, especially high fructose corn syrup
- Eating plenty of whole foods, ideally organic, and replacing the grain carbs with large amounts of fresh organic locally grown vegetables
- Consuming low-to-moderate amounts of high-quality protein

Carbs don't have to be your enemy if you choose wisely—and that choice can be easy if you simply avoid processed (canned/packaged) foods and sugars, and focus on eating fiber-rich whole foods, including whole grains if you must eat grains.

Essentially, what this means is [turning the American food pyramid on its head](#) and eliminating sugars and grains as much as possible. Following this simple guideline can also help reduce chronic inflammation in your body, elevate low-density LDL cholesterol (the “good” cholesterol), help you lose weight, and ultimately help you achieve healthy insulin and leptin levels.

What About the Glycemic Index?

Glycemic index (GI) is a tool used to measure a food's tendencies to affect blood sugar.

It's true that some foods seriously raise blood sugar and cause spikes in insulin, whereas other foods do not. However, the glycemic index has not been shown to be a consistently valid tool for determining this, primarily because measuring GI alone doesn't tell you how energy dense¹⁰¹ a food is. The fact is a food can have a very low glycemic index and contain substantial amounts of sugar—not a healthy choice at all.

The truth is glycemic index values [have far too many exceptions](#) to be consistently useful.

In summary, just how a food will affect your insulin level and blood sugar is controlled by a number of factors that the glycemic index does not take into account. Moreover, it fails to consider what harm chemicals like sucralose and fructose contained in supposedly low-GI foods do to your body.

You are simply better off using more reliable means of determining which foods are good for you—and this is not difficult!

Sugar: An Addiction We Need to Address

It's a common myth that eating too much sugar is what gives you diabetes. As I've already explained, a combination of things gives us diabetes. But still, we need to address Americans' sugar addiction and how it impacts diabetes rates. Should we eat it or shun it?

A few decades ago, diabetics were told never to eat sugar. But now the American Diabetes Association says it's OK to incorporate “very small” portions of sweets or desserts in a healthy meal plan.¹⁰²

So how do we know how much of “very small” is healthy?

Dr. Robert Lustig is a nationally-recognized authority in the field of neuroendocrinology and a professor at the University of California, San Francisco. A prolific author of numerous articles and research papers, he's written two books on sugar's effects on our health. Together with a group of scientists from three American universities, Dr. Lustig maintains an educational website—SugarScience.org—aimed at making independent sugar research available to the public.

I've [talked with Dr. Lustig](#) numerous times for my article topics, and he says that the safety threshold for sugar appears to be around six to nine teaspoons (25-38 grams) of added sugar a day. To give you an

idea of how much sugar that is, eight Nilla Wafers®¹⁰³ have a total of 11 grams of sugar in them, which is the same amount as one Nature Valley Granola Bar. Clearly, if you're going to consume added sugars, you need to know these things, which means reading a lot of labels.

But Dr. Lustig also notes that instead of trying to figure out how much extra sugar you can eat every day and be "safe," the answer to resolving insulin resistance can be summarized in two words: *real food*. In other words, there are more wholesome ways to get sweetness in your foods.

For example, instead of eating sweetened yogurt with fruit in it, buy unsweetened yogurt and put fruit in it. "*That's called real food*," he says.

Dr. Lustig believes the long-term answer for people who want to live healthy lives is simply eating real food. And I agree. Skip the sugar and make real food your mainstay. Then, when a special occasion like your birthday comes around, you can imbibe in that slice of cake without guilt.

High Fructose Corn Syrup: Not Just Another Sugar

Remember the TV commercials promoting corn syrup, telling you there's no difference between high fructose corn syrup (HFCS) and cane sugar, and that "sugar is sugar," no matter where it comes from?

Well, you're not seeing those commercials any more. That's because new research is showing that high fructose corn syrup not only is NOT the same as natural cane sugar, but it actually can leave you feeling hungry after you consume it.¹⁰⁴ Here's what we know for sure about HFCS:



- In 2008, the USDA reported that the use of high fructose corn syrup in the U.S. diet increased a staggering **10,673** percent between 1970 and 2005, by far exceeding any changes in intake of any other food or food group.¹⁰⁵ Per person, corn syrup consumption increased 387 percent during that time, while natural sugar consumption went down 38 percent, mainly because food and beverage processors were using the much lower-cost corn syrup in their products, as compared to real sugar.
- In 2010 we learned that HFCS—which accounts for as much as 40 percent of sugar sweeteners in the US—may contribute to obesity.¹⁰⁶
- Many studies later, **we now know it may very well be a principal driver of diabetes and its complications!**^{107, 108}
- Today, more than 35 percent of US adults are obese,¹⁰⁹ and statistics show that *the increased use of HFCS in the US mirrors the rapid rise in obesity over the years.*¹¹⁰

Dr. Richard Johnson, a professor of medicine who's done groundbreaking research on excess fructose consumption's on health, says his research shows that about 25 percent of all Americans are consuming over 134 grams of fructose a day. With numbers like that, it's easy to see why metabolic syndrome, prediabetes and diabetes have overtaken our lives.

Other concerns with corn syrup are that it:

- Metabolizes to fat much faster than other sugars¹¹¹
- Raises triglyceride levels,¹¹² which puts you at an increased risk of heart disease
- Raises LDL (bad) cholesterol levels¹¹³
- Is often consumed in liquid form (in soft drinks and juices) which, like regular sugar, magnifies its negative metabolic effects¹¹⁴
- Does not stimulate insulin secretion or enhance leptin production, so it lacks the ability to trigger appetite control, which in turn contributes to increased food intake and weight gain
- Contains no enzymes, vitamins, minerals, or other nutrient value

It's true that if you're eating a healthy diet of real food, a little bit of corn syrup here or there isn't going to cause any catastrophes. But now that we have scientific proof that corn syrup can contribute to weight gain, diabetes and other chronic diseases,¹¹⁵ isn't this a good time to just "swear off" it—and all sugars—and stick with real food?

You could begin by simply giving up soda, one 12-ounce serving of which contains an entire day's allowance of total sugars recommended safe by the USDA.¹¹⁶ Doing this would result in major health improvements for everyone, whether it's sweetened with sucrose or HFCS.

If you're not ready to go that far, and if you do purchase any processed foods, make sure you read the label... and put it back on the shelf if high fructose corn syrup is listed as one of the top five ingredients.

Where Do Fruits Figure in to a Healthy Diet?

Keep in mind that fruits also contain fructose, although an ameliorating factor is that whole fruits not only are *real food*, but also contain vitamins and other antioxidants that reduce the hazardous effects of fructose. Juices, on the other hand, are nearly as detrimental as soda because a glass of juice is loaded with concentrated fructose and, often, a lot of the antioxidants are lost.

Did you ever check the serving size on a fruit juice label? Usually one serving is about 6 ounces. But how many people only drink, or are satisfied by such a small amount? That's why it makes sense to choose the fruit over the juice. You can use the table below to help you keep your daily intake of fructose from fruits to less than 15 grams a day.

FRUIT	Serving Size	Grams of Fructose
Limes	1 medium	0
Lemons	1 medium	0.6
Cranberries	1 cup	0.7
Passion fruit	1 medium	0.9
Prune	1 medium	1.2
Apricot	1 medium	1.3
Guava	2 medium	2.2
Date (Deglet Noor style)	1 medium	2.6
<u>Cantaloupe</u>	1/8 of med. melon	2.8
Raspberries	1 cup	3.0
Clementine	1 medium	3.4
Kiwifruit	1 medium	3.4

Blackberries	1 cup	3.5
Star fruit	1 medium	3.6
Cherries, sweet	10	3.8
Strawberries	1 cup	3.8
Cherries, sour	1 cup	4.0
Pineapple	1 slice (3.5" x .75")	4.0
Grapefruit, pink or red	1/2 medium	4.3
Boysenberries	1 cup	4.6
Tangerine/mandarin orange	1 medium	4.8
Nectarine	1 medium	5.4
Peach	1 medium	5.9
Orange (navel)	1 medium	6.1
<u>Papaya</u>	1/2 medium	6.3
Honeydew	1/8 of med. melon	6.7

Banana	1 medium	7.1
Blueberries	1 cup	7.4
Date (Medjool)	1 medium	7.7
Apple (composite)	1 medium	9.5
Persimmon	1 medium	10.6
Watermelon	1/16 med. melon	11.3
Pear	1 medium	11.8
Raisins	1/4 cup	12.3
Grapes, seedless (green or red)	1 cup	12.4
Mango	1/2 medium	16.2
Apricots, dried	1 cup	16.4
Figs, dried	1 cup	23.0

Time to End the Low-Fat Myth

Nutritionists and government health agencies have spent decades pushing a low-fat diet as a main road to better health. Children have been relegated to low- or no-fat milk at school. Adults are bombarded with no-fat, low-fat choices in the supermarket. TV and radio ads push the low-fat mentality too. But after more than 40 years of reducing fat consumption, Americans are more obese, more diabetic and sicker than they were before the anti-fat craze began.

I've been talking about how [we're on the wrong track](#) with fat for years, but recently the prestigious Harvard University School of Public Health chimed in, proclaiming that it's time to end the low-fat myth. The school has even created a special page on its website,¹¹⁷ explaining the importance of healthy dietary fats.

"Detailed research shows that the total amount of fat in the diet isn't really linked with weight or disease," the school points out. "What really matters is the *type of fat* and the total calories in the diet."

That means yes, you CAN have fats, as long as they're "good" unsaturated fats [such as avocados](#), olive oil, and walnuts. Trans fats—such as hydrogenated corn, safflower, and soybean oils and partially-hydrogenated fats such as hard-stick margarine—are not good for you at all. Stay away from the bad fats, eat the good ones, and you not only will help your heart, but lower your risk of diabetes¹¹⁸ as well.

What About Artificial Sweeteners?

I've written extensively about the [dangers of artificial sweeteners](#), particularly [aspartame](#), which has been shown to worsen insulin sensitivity more than sugar, and to promote weight gain.¹¹⁹ My articles on artificial sweeteners are available for free on my website.

But should you use artificial sweeteners? In a word: no. However, if you feel you need to sweeten your food anyway, opt for stevia or Luo Han, both of which are safe, natural sweeteners.

Diabetes Was Once Nonexistent in Societies with a Traditional Diet

Diseases of insulin resistance have become a global health crisis as more economies, even in developing countries, transition from traditional, labor-intensive jobs to sedentary work, and more and more people adopt diets that put them at risk for developing diabetes.

According to published research at Harvard University School of Health:¹²⁰

"Once a disease of the West, type 2 diabetes has now spread to every country in the world. Once 'a disease of affluence,' it is now increasingly common among the poor. Once an adult-onset disease almost unheard of in children, rising rates of childhood obesity have rendered it more common in the pediatric population, especially in certain ethnic groups."

Sixty percent of the world's diabetics live in Asia, with the epicenter in China. In 1980 only 1 percent of Chinese adults had diabetes; today 11.6 percent—113.9 million—are diabetic.¹²¹ What happened? According to Diabetes Forecast:

*“...the country [China] is following in America’s footsteps—rapid growth, urbanization, affluence, sedentary factory jobs replacing farm labor, and **an abundance of fast food and sugary drinks.**” (Emphasis mine)*

What a terrible legacy to pass on to a country that once had almost no diabetes at all!

First, a Bit of History

The first modern humans, *Homo sapiens*, began walking this Earth about 200,000 years ago, and until the onset of agriculture about 12,000 years ago, they were hunters and gatherers, subsisting on a diet of animals and vegetation.¹²²

While it's believed that mankind probably ate sugar cane plant about 60,000 years ago, we know for certain that people in India were eating it in 2500 BC.¹²³ Ancient Egyptians are credited with recognizing signs of diabetes about 1000 years later.

It took until 350 AD for Indian food scientists to figure out how to make cane juice and crystallized sugar. And we humans have been addicted to it ever since.

Sugar came to northern Europe when Crusaders brought it back from fighting in West Asia;¹²⁴ in the 1500s, it came to North America, where explorers set up a sugar cane growing industry.¹²⁵

It took until 1776 for someone to figure out how to measure glucose in the urine of diabetics. And in 1812, diabetes became noted as a disease when the *New England Journal of Medicine and Surgery* was founded.¹²⁶

The 1800s were the dawn of the industrial age and,¹²⁷ for a while, diabetes was known as a disease of the affluent—people who could afford to spend more money on refined and processed grains, sugar and carbohydrates. But today, that's changed. Diabetes, prediabetes, obesity and simply being overweight is a disease that spares no one, rich or poor.

Rising obesity rates are considered as much an epidemic as diabetes,¹²⁸ and they are the greatest contributor to the increasing rates of type 2 diabetes.¹²⁹ In the US in 2014, more than 35 percent of US adults were obese; more than 18 percent of children ages 6-19 were obese; and 12.1 percent of US children ages 2-5 shared this sad fact.¹³⁰

Worldwide, global obesity rates have more than doubled since 1980. More than 600 million adults are obese; 1.9 billion—39 percent of all the adults in the world—are overweight. And, 42 million children under the age of 5 are overweight.

In developed countries, the number of overweight and obese people combined has tripled from 250 million to 904 million.¹³¹ What's ironic is that it's not uncommon for many of these overweight and obese people to also be malnourished.¹³²

The World Health Organization (WHO) blames this on:

- Richer diets and increased intake of high-fat, high-sugar, high-salt, energy-dense, micronutrient-poor foods
- Less physical activity, changing modes of transportation, and increasing urbanization

To address this problem, the WHO urges people to become more physically active, to increase consumption of fruits, vegetables, legumes, whole grains and nuts, and to limit total fats and sugars. At the same time, they are asking the food industry to cooperate by reducing fat, sugar, and salt content in processed foods.

So What Can We Do About This?

We can begin with our children, even before they're born. Children learn what they live. By living a life of example, we can prevent and reverse metabolic syndrome, prediabetes, and diabetes by showing our children the best life health choices.

Research shows that risk for diabetes goes up if you have a family history of diabetes. But as Harvard researchers will tell you, genes are not destiny.¹³³ You CAN overcome a family history of obesity. And since we already know that obesity is the leading cause of diabetes, it only makes sense that preventing diabetes in children should naturally begin with preventing obesity in children.

And that begins with teaching them how to make good food choices from the moment they're born.

Are You Setting Up Your Child to Become a Diabetic?

Everyone knows that the breast is best as the first food source for babies. But with cereal being one of the first solid foods typically introduced into an infant's diet, it may be time to reevaluate that choice.

The American Academy of Pediatrics recommends that babies be exclusively breastfed for the first six months of life. This means no additional foods unless medically indicated.¹³⁴ After that it's up to you and your pediatrician to decide what solid foods to introduce.

You may be tempted to start solids earlier, but science shows that doing so could increase your child's obesity risk.¹³⁵ So hold off, and then when you do begin solids, choose wisely: remember that your infant is no different than you are in that a starchy diet is going to set your little one up for insulin resistance. Perhaps that's why some health officials¹³⁶ are suggesting that you begin with pureed food (you can do this yourself with fresh, organic foods) like sweet potatoes, squash, apples, bananas, peaches, or pears in addition to pureed food or cereal, rather than just putting your child on cereals, as was common not that long ago.

While cereals are on pediatricians' the list, I recommend you begin introducing your infant to vegetables for carbohydrates instead of cereals.

Eight Keys to Preventing and Treating Diabetes

The following eight nutrition and lifestyle modifications should be the foundation of your diabetes prevention and treatment plan.

1. Limit or eliminate all forms of sugars—particularly fructose—and grains—basically all processed foods—from your diet and incorporate real foods (whole, organic, locally-grown fruits and vegetables).

If you're insulin leptin resistant, have diabetes, high blood pressure, heart disease, or are overweight, you'd be wise to limit your total fructose intake to 15 grams per day until your insulin/leptin resistance has resolved. For others, limit your daily fructose consumption to 25 grams or less.

The easiest way to do this is to completely give up processed foods of all kinds. Processed foods and the ingredients they're made of—high fructose corn syrup, other added sugars, processed grains, artificial sweeteners, [trans fats](#), and a long list of synthetic additives all contribute to metabolic, insulin, and leptin dysfunction.

It's particularly important to eliminate processed meats. In a groundbreaking study comparing processed meats to unprocessed meats for the first time,^{137, 138} researchers at Harvard School of Public Health found that eating processed meat is associated with a 42 percent higher risk of heart disease and a 19 percent higher risk of type 2 diabetes. Interestingly, they did not find any risk of heart disease or diabetes among individuals eating unprocessed red meat such as beef, pork or lamb.

Besides fructose, [eliminate trans fats](#), which increase your risk for diabetes and inflammation by interfering with your insulin receptors.¹³⁹ Trans fatty acids are artery-clogging fats that form when vegetable oils are hardened into margarine or shortening. They are often found in cookies, doughnuts, pastries, crackers, fried foods, French fries, and other processed foods.

Healthy [saturated fats don't do this](#). Since you're cutting a lot of energy (carbs) from your diet when you eliminate processed sugars and grains, you need to replace them with something better, such as:

- Low-to-moderate amounts of high-quality protein found in organically-raised, grass-fed or pastured meats and dairy products, fish, legumes, and nuts. Aim for one-half gram of protein per pound of lean body mass, which places most people in a range of 40-70 grams of protein per day. Use the chart below to help you.

Red meat, pork, poultry, and seafood average 6-9 grams of protein per ounce. An ideal amount for most people would be a 3-ounce serving of meat or seafood (not 9- or 12-ounce steaks!), which will provide about 18-27 grams of protein	Eggs contain about 6-8 grams of protein per egg. So an omelet made from two eggs would give you about 12-16 grams of protein. If you add cheese, you need to calculate that protein in as well (check the label of your cheese)
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Seeds and nuts contain on average 4-8 grams of protein per quarter cup	Cooked beans average about 7-8 grams per half cup
Cooked grains average 5-7 grams per cup	Most vegetables contain about 1-2 grams of protein per ounce

- As much high-quality healthy fat as you want. Your body [needs saturated fats](#) to stay healthy, in appropriate quantities, as they provide many beneficial effects, contrary to what you have probably been told.¹⁴⁰

For optimal health, most people need upwards of 50-85 percent of their daily calories in the form of healthy fats. Good sources include coconut and coconut oil, avocados, butter, nuts, and animal fats. Remember—fats are high in calories but small in volume, so when you look at your plate, vegetables should be the largest portion

2. Exercise regularly and intensely.

Studies have shown that exercise is beneficial and increases insulin sensitivity, whether you lose weight or not,¹⁴¹ and even if you're physically active as little as 2 and a half hours a week.¹⁴²

Not only that, another study shows that compared the diabetes drug metformin to a lifestyle change/exercise program in persons at high risk of getting diabetes found that diet and exercise actually were more effective than the drug in reducing the incidence of diabetes.¹⁴³

That's great news—and the best news is that it's never too late to start an exercise program. [High intensity interval training](#) (HIIT), which is a central component of my [Peak Fitness program](#), has been shown to improve insulin sensitivity by as much as 24 percent in just four weeks.

3. Get plenty of omega-3 fats from fish oil or krill oil.

Today's Western diet has far too many omega-6 fats and far too few omega-3 fats. Because the body doesn't make polyunsaturated fatty acids alpha-linolenic acid (ALA) and linoleic acid (LA), you must get them from your diet. Unfortunately, most American diets have 10 times as many omega-6 as omega-3 fatty acids—a fact that has the National Institutes of Health (NIH) concerned.¹⁴⁴

One of the richest sources in omega-3 fats is fish, [including krill](#). However, the NIH says that there is also strong evidence that fish oil supplements can also offer substantial benefits.

Unfortunately, eating more fresh fish is no longer recommended because mercury levels in almost all fish have hit dangerously high levels around the world. A far better option is to supplement with a high-quality krill oil. Krill oil offers a very potent, highly absorbable blend of antioxidants as well.

Omega-3 is also found in flaxseed oil and walnut oil, so it's good to include those in your diet.

The main sources of omega-6 fats are corn, soy, canola, safflower and sunflower oil. These are overabundant in the American diet, and most corn and soybeans are now genetically modified and sprayed with chemicals, so it's best you avoid or limit these oils.

4. Get plenty of sunshine, and if unable to do so, consider oral vitamin D supplementation with regular monitoring.

New evidence strongly supports that vitamin D is highly beneficial not only in type 1 diabetes,¹⁴⁵ but also in type 2 diabetes.^{146, 147}

The best way to get optimal vitamin D is through safe sun exposure during the summer months or a safe tanning bed in winter. You can also consider taking oral vitamin D supplements in conjunction with regular [vitamin D monitoring](#). (Remember—if you take supplemental vitamin D, you create an increased demand for [vitamin K2](#) and magnesium.)

And, as we noted earlier, since so many women are hugely deficient in vitamin D, if you're thinking of getting pregnant, it would be wise to have your levels monitored and a vitamin D supplement both before you become pregnant and while you're pregnant.

5. Get adequate high-quality sleep every night.

In a 10-year study¹⁴⁸ of 70,000 diabetes-free women, researchers found that women who slept less than five hours or more than nine hours each night were 34 percent more likely to develop diabetes symptoms than women who [slept seven to eight hours](#) each night.

Insufficient sleep also appears to reduce levels of leptin, encouraging insulin resistance and weight gain.¹⁴⁹

If you are having problems with your sleep, try the [suggestions in my article](#), “Want a Good Night’s Sleep?” on my website, where you can find 33 ways to improve your sleep quality.

6. Incorporate intermittent fasting.

If you have carefully followed the diet and exercise guidelines and still aren't making sufficient progress with your weight and overall health, I strongly recommend incorporating [intermittent fasting](#). This effectively mimics the eating habits of our ancestors, who would cycle through periods of feast and famine. Modern research shows this cycling produces a number of biochemical benefits, including improved insulin/leptin sensitivity, lowered triglycerides and other biomarkers for health and weight loss.

7. Optimize your gut health.

Your gut is a living ecosystem, full of both good bacteria and bad. Multiple studies have shown that obese people have different intestinal bacteria than lean people. Recent research^{150, 151} also suggests your [microbiome can influence your risk of diabetes](#).

As a general rule, the more good bacteria you have, the stronger your immune system will be and the better your body will function overall. Fortunately, optimizing your gut flora is relatively easy. You can reseed your body with good bacteria by regularly [eating fermented foods](#) (like fermented vegetables, especially fermented with starter culture that has strains that produce vitamin K2, natto, raw organic cheese, and miso) or by taking a high-quality probiotic supplement.

8. Maintain a healthy body weight.

If you incorporate the diet and lifestyle changes suggested above, your body weight will normalize in time.

Determining your ideal body weight depends on a variety of factors, including frame size, age, general activity level, and genetics.

For a very general guideline, a [hip-to-waist index chart](#) that helps you evaluate whether you may have a weight problem can be useful. This is far better than BMI guides because BMIs fail to factor in both how muscular you are and your intra-abdominal fat mass (the dangerous visceral fat that accumulates around your inner organs), which is a potential indicator of leptin sensitivity and associated health problems.

If you have been following the diet and exercise guidelines carefully and are not making progress with your weight or with your overall health, then emotional factors may be holding you back from reaching your goals.

My favorite tool for addressing emotional blocks is the [Emotional Freedom Technique \(EFT\)](#), for which I have detailed instructions on my website.

Final Food for Thought

The good news about all of this is that you don't have to be a part of the diabetes epidemic. To avoid becoming a dismal statistic, you merely need to make some lifestyle changes and be mindful about your habits from day to day. In fact, just resolving to be healthier^{152, 153} may reduce your risk for diabetes!

Now that you have an understanding of what diabetes really is and how it develops, you can steer clear of behavior patterns that are not health promoting and incorporate those that will enhance your quality of life. The necessary changes are neither expensive nor overly time-consuming: for the most part they simply require determination and a measure of honest reflection and discipline. To help you with this, I suggest that you take a lifestyle inventory and make changes in areas where you feel you are lacking.

For example:

- **Review your eating patterns** and keep a food diary to see where you can improve. How much sugar (and equivalent insulin-offenders) have you been eating daily? Is corn syrup a hidden ingredient in your pantry (this means reading the labels)? How many processed foods are you buying? Are you spending your time in the middle of the grocery store, or around the periphery (most processed foods come from the middle aisles)?
- **Are you an “emotional eater”?** Do you tend to overindulge in comfort foods when you are feeling sad or angry? If so, review the information on my website about EFT, which you might find very helpful.
- **Evaluate your activity level.** Are you getting enough exercise each week? Are you getting enough variation in your exercise routine (aerobic, weight resistance, stretching, intermittent high-intensity), or are you stuck in a rut?
- **Are you getting enough sunlight?** Do you need to consider a vitamin D supplement?
- **How is your general health and how do you feel?** Are you full of energy and vigor, or are you chronically tired and sluggish? Do you get a lot of colds? Where is your weight related to where it should be for your height and bone structure? Are there any red flags in your family history?
- **What patterns are you inadvertently passing along to your children?** What example are you setting for your kids, in terms of nutrition and exercise? Are they getting the message that health is a priority?

The good news is changing your health by changing your habits can be easier than you think, and even fun. As I explain in [my new book, Effortless Healing: 9 Simple Ways to Sidestep Illness, Shed Excess Weight, and Help Your Body Fix Itself](#), your healing plan is in your hands.

And remember—getting healthy can and should be a family activity! When everyone is involved, you can support each other and give kudos for positive strides, making it more fun for everyone. The payoffs to your health will be great, and you will be passing along good lifestyle habits to your children, which will serve them for years to come.

References

- ¹ Hu Frank B. Globalization of Diabetes: The Role of Diet, Lifestyle, and Genes. *Diabetes Care*. June 2011. Online. <http://care.diabetesjournals.org/content/34/6/1249.full.pdf+html>. Accessed December 2014
- ² Hu Frank B. Globalization of Diabetes: The Role of Diet, Lifestyle, and Genes. *Diabetes Care*. June 2011. Online. <http://care.diabetesjournals.org/content/34/6/1249.full.pdf+html>. Accessed December 2014
- ³ Boyle JP, et al. Projection of Diabetes Burden through 2050: Impact of Changing Demography and Disease Prevalence in the US. *Diabetes Care*. November 2001. Online. http://care.diabetesjournals.org/content/24/11/1936.full?wptouch_preview_theme=enabled (Accessed December 2014)
- ⁴ CDC. Prediabetes: Could It Be You. CDC National Diabetes Statistics Report. 2014, Online. <http://www.cdc.gov/diabetes/pubs/statsreport14/prediabetes-infographic.pdf> (Accessed December 2014)
- ⁵ Passary Sumit. US Population Grows to 320.09 Million. Tech Times. December 30, 2014. Online. <http://www.techtimes.com/articles/23784/20141230/u-s-population-grows-to-320-09-million-california-still-reigns-as-most-populous-us-state.htm> (Accessed January 2015)
- ⁶ Kirka D. Global Obesity Costs Hits \$2 Trillion. Drug Discovery & Development. Online. http://www.dddmag.com/news/2014/11/global-obesity-costs-hits-2-trillion?et_cid=4276233&et_rid=230266277&location=top (Accessed February 2015)
- ⁷ CDC. Prediabetes: Could It Be You? National Diabetes Statistics Report. 2014. Online. <http://www.cdc.gov/diabetes/pubs/images/prediabetes-infographic.jpg> (Accessed January 2015)
- ⁸ CDC. 2014 National Diabetes Statistics Report. Online. <http://www.cdc.gov/diabetes/data/statistics/2014StatisticsReport.html>. Accessed November 2014
- ⁹ Dabelea D, Mayer-Davis E, Saydah S, et al. Prevalence of Type 1 and Type 2 Diabetes among Children and Adolescents from 2001 to 2009. *JAMA*. May 7, 2014. Online. <http://jama.jamanetwork.com/article.aspx?articleid=1866098>. Accessed November 2014
- ¹⁰ JDRF. Statistics: JDRF and Diabetes. January 2015. Online. <http://jdrf.org/about-jdrf/fact-sheets/jdrf-and-diabetes-statistics/> (Accessed January 2015)
- ¹¹ American Diabetes Association. Statistics about Diabetes: Overall Numbers, Diabetes and Prediabetes. Diabetes.org, from National Diabetes Statistics Report. June 2014. Online. <http://www.diabetes.org/diabetes-basics/statistics/> (Accessed January 2015)
- ¹² Dall TM, et al. The Economic Burden of Elevated Blood Glucose Levels in 2012: Diagnosed and Undiagnosed Diabetes, Gestational Diabetes Mellitus, and Prediabetes. *Diabetes Care*. December 2014. Online. <http://care.diabetesjournals.org/content/37/12/3172.full> (Accessed January 2015)
- ¹³ Boyle JP, et al. Projection of Diabetes Burden through 2050: Impact of Changing Demography and Disease Prevalence in the US. *Diabetes Care*. November 2001. Online. http://care.diabetesjournals.org/content/24/11/1936.full?wptouch_preview_theme=enabled (Accessed December 2014)
- ¹⁴ CDC. Diabetes. CDC.gov. February 6, 2015. Online. <http://www.cdc.gov/nchs/fastats/diabetes.htm> (Accessed February 2015)
- ¹⁵ National Diabetes Information Clearinghouse. Causes of Diabetes. National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health. August 27, 2014. Online. (Accessed January 2015)
- ¹⁶ Buse JB, et al. How Do We Define Cure of Diabetes? *Diabetes Care*. Vol 32. Nov 2009. Online. <http://care.diabetesjournals.org/content/32/11/2133.full.pdf+html> (Accessed January 2015)
- ¹⁷ Russell P. Why Statins Might Raise Diabetes Risk. WebMD. September 24, 2014. Online. <http://www.webmd.com/cholesterol-management/news/20140924/statins-diabetes-risk>
- ¹⁸ National Diabetes Information Clearinghouse. Causes of Diabetes. National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health. August 27, 2014 (Accessed January 2015)
- ¹⁹ Q&A with Judith Fradkin, National Institutes of Health. The Lasting Impact of Gestational Diabetes on Mothers & Children: Q&A. National Healthy Mothers, Healthy Babies Coalition. 2012. Online. <http://www.hmbc.org/virtual-library/interviews-with-experts/gestational-diabetes/> (Accessed January 2015)
- ²⁰ American Diabetes Association. Statistics about Diabetes. From the National Diabetes Statistics Report 2014. June 10, 2014. Online. <http://www.diabetes.org/diabetes-basics/statistics/> (Accessed January 2015)
- ²¹ National Diabetes Information Clearinghouse. Causes of Diabetes. National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health. August 27, 2014. Online. <http://diabetes.niddk.nih.gov/dm/pubs/causes/#causes> (Accessed January 2015)
- ²² Kaiser Permanente. Significant Increase in Type 1 Diabetes Rates among Non-Hispanic White Youth. Science Daily. October 23, 2014. Online. <http://www.sciencedaily.com/releases/2014/10/141023132228.htm> (Accessed January 2015)
- ²³ Lawrence JM, et al. Trends in Incidence of Type 1 Diabetes among Non-Hispanic White Youth in the US, 2002-2009. *Diabetes*. November 2014. Online. <http://diabetes.diabetesjournals.org/content/63/11/3938> (Accessed January 2015)
- ²⁴ Lipman TH, et al. Increasing Incidence of Type 1 Diabetes in Youth. *Diabetes Care*. June 2013. Online. <http://care.diabetesjournals.org/content/36/6/1597.full.pdf+html> (Accessed January 2015)
- ²⁵ National Diabetes Information Clearinghouse. Causes of Diabetes. National Institutes of Health. August 27, 2014. Online. <http://diabetes.niddk.nih.gov/dm/pubs/causes/#causes> (Accessed January 2015)
- ²⁶ Gorham ED, et al. Lower Prediagnostic Serum 25-Hydroxyvitamin D Concentration Is Associated with Higher Risk of Insulin-Requiring Diabetes: A Nested Case-Control Study. *Diabetologia*. September 7, 2012. Online. <http://link.springer.com/article/10.1007%2Fs00125-012-2709-8> (Accessed January 2015)
- ²⁷ UC San Diego Health System. Vitamin D Deficiency Linked to Type 1 Diabetes. Newsroom. November 15, 2012. Online. <http://health.ucsd.edu/news/releases/Pages/2012-11-15-vitamin-D-and-type-1-diabetes-link.aspx> (Accessed January 2015)
- ²⁸ Hypponen E, et al. Intake of Vitamin D and Risk of Type 1 Diabetes: A Birth-Cohort Study. *Lancet*. November 3, 2001. Online. <http://www.ncbi.nlm.nih.gov/pubmed/11705562> (Accessed January 2015)
- ²⁹ Makinen M, et al. An Increase in Serum 25-hydroxyvitamin D Concentrations Preceded a Plateau in Type 1 Diabetes Incidence in Finnish Children. *The Journal of Clinical Endocrinology and Metabolism*. November 2014. Online. <http://www.ncbi.nlm.nih.gov/pubmed/25062454> (Accessed January 2015)
- ³⁰ Bener A, et al. High Prevalence of Vitamin D Deficiency in Type 1 Diabetes Mellitus and Healthy Children. *Acta Diabetologica*. September 2009. Online. <http://www.ncbi.nlm.nih.gov/pubmed/18846317> (Accessed January 2015)

- ³¹ Miettinen ME, et al. Serum 25-hydroxyvitamin D Level During Early Pregnancy and Type 1 Diabetes Risk in the Offspring. *Diabetologia*. May 2012. Online. <http://www.ncbi.nlm.nih.gov/pubmed/22270224> (Accessed January 2015)
- ³² Grant WB, Cannell JJ. Autism Prevalence in the United States with Respect to Solar UV-B Doses: An Ecological Study. *Dermato-Endocrinology*. January , 2013. Online. <http://www.tandfonline.com/doi/abs/10.4161/derm.22942#.VL6R4y7hAh8> (Accessed January 2015)
- ³³ Humber MB, Gustafsson S, Bejerot S. Low Serum Levels of 25-Hydroxyvitamin D (25-OHD) Among Psychiatric Out-Patients in Sweden: Relations with Season, Age, Ethnic Origin and Psychiatric Diagnosis. *The Journal of Steroid Biochemistry and Molecular Biology*. July 2010. Online. <http://www.ncbi.nlm.nih.gov/pubmed/20214992> (Accessed January 2015)
- ³⁴ Vitamin D Council Vitamin D During Pregnancy and Breastfeeding. January 30, 2013. Online. <https://www.vitamindcouncil.org/further-topics/vitamin-d-during-pregnancy-and-breastfeeding/> (Accessed January 2015)
- ³⁵ Robles DT and Eisenbarth GS. Type 1A Diabetes Induced by Infection and Immunization. *Journal of Autoimmunity*. May 2001. Online. <http://www.sciencedirect.com/science/article/pii/S0896841100904839> (Accessed January 2015)
- ³⁶ Menser MA, Forrest JM and Bransby RD. Rubella Infection and Diabetes Mellitus. *The Lancet*. January 1978. Online. <http://www.thelancet.com/journals/lancet/article/PIIS0140-6736%2878%2990001-6/abstract> (Accessed January 2015)
- ³⁷ Van der Werf N, et al. Viral Infections as Potential Triggers of Type 1 Diabetes. *Diabetes Metabolism Research and Reviews*. March 2007. Online. <http://www.ncbi.nlm.nih.gov/pubmed/17103489> (Accessed January 2015)
- ³⁸
- ³⁹ Honeyman MC, et al. Association between Rotavirus Infection and Pancreatic Islet Autoimmunity in Children at Risk of Developing Type 1 Diabetes. *Diabetes*. August 2000. Online. <http://diabetes.diabetesjournals.org/content/49/8/1319.full.pdf+html> (Accessed January 2015)
- ⁴⁰ Pane JA, Webster NL, Coulson BS. Rotavirus Activates Lymphocytes from Non-Obese Diabetic Mice by Triggering Toll-Like Receptor 7 Signaling and Interferon Production in Plasmacytoid Dendritic Cells. *PLoS*. March 27, 2014. Online. <http://journals.plos.org/plospathogens/article?id=10.1371/journal.ppat.1003998> (Accessed January 2015)
- ⁴¹ Salleh A. Rotavirus Can Speed Up Diabetes Onset. ABC Science. March 28, 2014. Online. <http://www.abc.net.au/science/articles/2014/03/28/3971982.htm> (Accessed January 2015)
- ⁴² Lin Hsiao-Chuan, et al. Enterovirus Infection Is Associated with an Increased Risk of Childhood Type 1 Diabetes in Taiwan: a Nationwide Population-Based Cohort Study. *Diabetologia*. October 22, 2014. Online. <http://link.springer.com/article/10.1007%2Fs00125-014-3400-z> (Accessed January 2015)
- ⁴³ Laitinen OH, et al. Coxsachievirus B1 Is Associated with Induction of β Cell Autoimmunity that Portends Type 1 Diabetes. *Diabetes*. August 23, 2013. Online. <http://diabetes.diabetesjournals.org/content/63/2/446> (Accessed January 2015)
- ⁴⁴ Yeung WC, Rawlinson WD, Craig ME. Enterovirus Infection and Type 1 Diabetes Mellitus: a Systematic Review and Meta-Analysis of Observational Molecular Studies. *BMJ*. February 2011. Online. <http://www.ncbi.nlm.nih.gov/pubmed/21292721> (Accessed January 2015)
- ⁴⁵ Classen JB. Review of Vaccine Induced Immune Overload and the Resulting Epidemics of Type 1 Diabetes and Metabolic Syndrome, Emphasis on Explaining the Recent Accelerations in the Risk of Prediabetes and Other Immune Mediated Diseases. *Molecular and Genetic Medicine*. February 19, 2014. Online. <http://www.vaccines.net/vaccine-induced-immune-overload.pdf> (Accessed February 2015)
- ⁴⁶ Classen JB and Classen DC. Association between Type 1 Diabetes and Hib Vaccine: Causal Relation Is Likely. *British Medical Journal* October 23, 1999. Online. <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1116914/> (Accessed January 2015)
- ⁴⁷ Zanoni G, et al. Normal or Defective Immune Response to Hepatitis B Vaccine in Patients with Diabetes and Celiac Disease. *Human Vaccines & Immunotherapies*. August 2014. Online. <http://www.ncbi.nlm.nih.gov/pubmed/25483516> (Accessed January 2015)
- ⁴⁸ IOM Immunization Safety Review Committee. Immunization Safety Review: Multiple Immunizations and Immune Dysfunction. Institute of Medicine. 2002. Online. <http://www.ncbi.nlm.nih.gov/books/NBK220494/#ddd00093> (Accessed January 2015)
- ⁴⁹ Christen U, Bender C, von Herrath MG. Infection as a Cause of Type 1 Diabetes? *Current Opinion in Rheumatology*. July 2012. Online. <http://www.ncbi.nlm.nih.gov/pubmed/22504578> (Accessed January 2015)
- ⁵⁰ "Friendly Bacteria Protect against Type 1 Diabetes, Researchers Find," ScienceDaily, September 22, 2008, <http://www.sciencedaily.com/releases/2008/09/080921162048.htm> (Accessed January 2015)
- ⁵¹ Sci-Tech. Over-Hygienic Parents Could Be Cause of Diabetes. Euronews.com. November 2012. Online. <http://www.euronews.com/2012/01/11/over-hygienic-parents-could-be-cause-of-diabetes/> (Accessed January 2015)
- ⁵² H Okada, C Kuhn, H Feillet, J-F Bach. The 'Hygiene Hypothesis' for Autoimmune and Allergic Diseases: an Update. *Clinical & Experimental Immunology*. April 2010. Online. <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2841828/> (Accessed January 2015)
- ⁵³ Kondrashova A, et al. The 'Hygiene Hypothesis' and the Sharp Gradient in the Incidence of Autoimmune and Allergic Diseases between Russian Karelia and Finland. *APMIS*. November 2012. Online. <http://onlinelibrary.wiley.com/doi/10.1111/apm.12023/abstract?deniedAccessCustomisedMessage=&userIsAuthenticated=false> (Accessed January 2015)
- ⁵⁴ National Diabetes Education Program. Guiding Principles for the Care of People with or at Risk for Diabetes. US Department of Health and Human Services. January 2015. Online. <http://ndep.nih.gov/hcp-businesses-and-schools/guiding-principles/principle-01-identify-undiagnosed-diabetes-and-prediabetes.aspx> (Accessed January 2015)
- ⁵⁵ Diabetescare.net. Symptoms of Diabetes. August 1, 2014. Online. <http://www.diabetescare.net/slideshow/symptoms-of-diabetes>. Accessed November 2014
- ⁵⁶ American Diabetes Association. Diagnosing Diabetes and Learning about Prediabetes. Diabetes.org. 2015. Online. <http://www.diabetes.org/diabetes-basics/diagnosis/> (Accessed December 2014)
- ⁵⁷ National Diabetes Information Clearinghouse. Diagnosis of Diabetes and Prediabetes. US Department of Health and Human Services. September 10, 2014. Online. <http://diabetes.niddk.nih.gov/dm/pubs/diagnosis/> (Accessed December 2014)
- ⁵⁸ WebMD. What Is Metabolic Syndrome? WebMd.com. 2015. Online. <http://www.webmd.com/heart/metabolic-syndrome/metabolic-syndrome-what-is-it> (Accessed January 2015)
- ⁵⁹ Reaven GM. Role of Insulin Resistance in Human Disease. *Diabetes*. December 1988. Online. <http://diabetes.diabetesjournals.org/content/37/12/1595.short?rss=1&source=mfc> (Accessed January 2015)
- ⁶⁰ American Diabetes Association. Type 1 Diabetes. Diabetes.org. 2015. Online. <http://www.diabetes.org/diabetes-basics/type-1/> (Accessed Januay 2015)
- ⁶¹ The Rockefeller University. Jeffrey M. Friedman Receives Albert Lasker Award for Discovery of Leptin. Newswire. September 21, 2010. Online. <http://newswire.rockefeller.edu/2010/09/21/jeffrey-m-friedman-receives-albert-lasker-award-for-discovery-of-leptin/> (Accessed January 2015)

- ⁶² Mercola.com. Clinical Scientist Sets the Record Straight on Hazards of Sugar. January 5, 2014. <http://articles.mercola.com/sites/articles/archive/2014/01/05/dr-johnson-leptin-resistance.aspx>
- ⁶³ Johnson, Richard J. The Fat Switch. Mercola.com. 2012. Available online at: <http://shop.mercola.com/product/the-fat-switch-book,1038,123.htm> (Accessed February 2015)
- ⁶⁴ Feldstein AC, et al. Weight Change in Diabetes and Glycemic and Blood Pressure Control. *Diabetes Care*. October 2008. Online. <http://care.diabetesjournals.org/content/31/10/1960.short> (Accessed February 2015)
- ⁶⁵ CDC. National Diabetes Statistics Report 2014. CDC.gov. 2014. Online. <http://www.cdc.gov/diabetes/pubs/statsreport14/national-diabetes-report-web.pdf> (Accessed February 2015)
- ⁶⁶ CDC. Basics about Diabetes. CDC.gov. October 21, 2014. Online. <http://www.cdc.gov/diabetes/basics/diabetes.html> (Accessed February 2015)
- ⁶⁷ Lin P-J, et al. Multiple Chronic Conditions in Type 2 Diabetes Mellitus: Prevalence and Consequences. *The American Journal of Managed Markets*. February 27, 2015. Online. <http://www.ajmc.com/publications/issue/2015/2015-vol21-n1/Multiple-Chronic-Conditions-in-Type-2-Diabetes-Mellitus-Prevalence-and-Consequences> (Accessed March 2015)
- ⁶⁸ US HHS. HHS Initiative on Multiple Chronic Conditions. HHS.gov. December 22, 2014. Online. <http://www.hhs.gov/ash/initiatives/mcc> (Accessed March 2015)
- ⁶⁹ Thorpe KE and Howard DH. The Rise in Spending Among Medicare Beneficiaries: The Role of Chronic Disease Prevalence and Changes in Treatment Intensity. *Health Affairs*. September 2006. Online. <http://content.healthaffairs.org/content/25/5/w378.full> (Accessed March 2015)
- ⁷⁰ Kaiser Family Foundation. Retail Prescription Drugs Filled at Pharmacies (Annual per Capita by Age). IMS Health. 2014. Online. <http://kff.org/other/state-indicator/retail-rx-drugs-by-age/> (Accessed March 2015)
- ⁷¹ MedBio. Insulin and Glucagon. Online. http://www.medbio.info/horn/PDF%20files/homeostasis_2a.pdf (Accessed February 2015)
- ⁷² Norman J. Normal Regulation of Blood Glucose. Endocrineweb. May 27, 2014. Online. <http://www.endocrineweb.com/conditions/diabetes/normal-regulation-blood-glucose> (Accessed February 2015)
- ⁷³ National Diabetes Information Clearinghouse. Insulin Resistance and Prediabetes. US Department of Health and Human Services. September 10, 2014. Online. <http://diabetes.niddk.nih.gov/dm/pubs/insulinresistance/> (Accessed February 2015)
- ⁷⁴ MedBio. Insulin and Glucagon. Online. http://www.medbio.info/horn/PDF%20files/homeostasis_2a.pdf (Accessed February 2015)
- ⁷⁵ Berg JM, Tymoczko JL, Stryer L. Chapter 21: Glycogen Metabolism. *Biochemistry* 5th Edition. 2002. Online. <http://www.ncbi.nlm.nih.gov/books/NBK21190/> (Accessed February 2015)
- ⁷⁶ Barbagallo M, et al. Role of Magnesium in Insulin Action, Diabetes and Cardio-Metabolic Syndrome X. *Molecular Aspects of Medicine*. Feb-Jun 2003. Online. <http://www.ncbi.nlm.nih.gov/pubmed/12537988> (Accessed February 2015)
- ⁷⁷ Trevisan R, et al. Role of Insulin and Atrial Natriuretic Peptide in Sodium Retention in Insulin-Treated IDDM Patients During Isotonic Volume Expansion. *Diabetes*. March 1990. Online. <http://diabetes.diabetesjournals.org/content/39/3/289> (Accessed February 2015)
- ⁷⁸ MedBio. Insulin and Glucagon. Online. http://www.medbio.info/horn/PDF%20files/homeostasis_2a.pdf (Accessed February 2015)
- ⁷⁹ Strasburger K, et al. Insulin/IGF Signaling Drives Cell Proliferation in Part via Yorkie/YAP. *Developmental Biology*. July 15, 2012. Online. <http://www.sciencedirect.com/science/article/pii/S0012160612002552> (Accessed February 2015)
- ⁸⁰ Xu J, Messina JL. Crosstalk between Growth Hormone and Insulin Signaling. *Vitamins and Hormones*. 2009. Online. <http://www.ncbi.nlm.nih.gov/pubmed/19251037> (Accessed February 2015)
- ⁸¹ Strain G, et al. The Relationship between Serum Levels of Insulin and Sex Hormone-Binding Globulin in Men: the Effect of Weight Loss. *The Journal of Clinical Endocrinology and Metabolism*. October 1994. Online. <http://www.ncbi.nlm.nih.gov/pubmed/7962291> (Accessed February 2015)
- ⁸² Puche RC, et al. The Effect of Insulin on Bone Resorption. *Calcified Tissue Research*. 1973. Online. <http://link.springer.com/article/10.1007%2FBF02013717> (Accessed February 2015)
- ⁸³ Paolisso G, et al. Glucose Tolerance and Insulin Action in Healthy Centenarians. *The American Journal of Physiology*. May 1996. Online. <http://www.ncbi.nlm.nih.gov/pubmed/8967479> (Accessed February 2015)
- ⁸⁴ Rosedale R, Westman EC, Konhilas JP. Clinical Experience of a Diet Designed to Reduce Agin. *Journal of Applied Research*. March 2010. Online. <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2831640/> (Accessed February 2015)
- ⁸⁵ Trayhurn P, et al. Leptin: Fundamental Aspects. *International Journal of Obesity*. February 1999. Online. <http://www.nature.com/ijo/journal/v23/n1/abs/0800791a.html> (Accessed February 2015)
- ⁸⁶ Kelesidis T, et al. Narrative Review: The Role of Leptin in Human Physiology: Emerging Clinical Applications. *Annals of Internal Medicine*. January 19, 2010. Online. <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2829242/> (Accessed February 2015)
- ⁸⁷ Klok MD, Jakobsdottir S, Drent ML. The Role of Leptin and Ghrelin in the Regulation of Food Intake and Body Weight in Humans: a Review. *Obesity Reviews*. January 2007. Online. <http://onlinelibrary.wiley.com/doi/10.1111/j.1467-789X.2006.00270.x/full> (Accessed February 2015)
- ⁸⁸ Richards BJ. Mastering Leptin: The Leptin Diet Solving Obesity and Preventing Disease. 2nd Edition. Wellness Resources Books. 2005.
- ⁸⁹ Mark AL. Selective Leptin Resistance Revisited. *American Journal of Physiology Regulatory, Integrative and Comparative Physiology*. September 15, 2013. Online. <http://ajpregu.physiology.org/content/305/6/R566> (Accessed February 2015)
- ⁹⁰ Napoleone E, et al. Leptin Induces Tissue Factor Expression in Human Peripheral Blood Mononuclear Cells: A Possible Link between Obesity and Cardiovascular Risk? *Journal of Thrombosis and Haemostasis*. July 2007. Online. <http://onlinelibrary.wiley.com/doi/10.1111/j.1538-7836.2007.02578.x/full> (Accessed February 2015)
- ⁹¹ Halvorsen YC, Wilkison WO, Gimble JM. Adipose-Derived Stromal Cells—Their Utility and Potential in Bone Formation. *International Journal of Obesity and Related Metabolic Disorders: Journal of the International Association for the Study of Obesity*. 2000. Online. <http://europepmc.org/abstract/med/11126240> (Accessed February 2015)
- ⁹² Luheshi GN, et al. Leptin Actions on Food Intake and Body Temperature Are Mediated by IL-1. *PNAS*. June 8, 1999. Online. <http://www.pnas.org/content/96/12/7047.full> (Accessed February 2015)
- ⁹³ Butte NF, Hopkinson JM, Nicolson MA. Leptin in Human Reproduction: Serum Leptin Levels in Pregnant and Lactating Women. *The Journal of Clinical Endocrinology & Metabolism*. July 1, 2013. Online. <http://press.endocrine.org/doi/full/10.1210/jcem.82.2.3731> (Accessed February 2015)
- ⁹⁴ Sattar N, et al. Statins and Risk of Incident Diabetes: a Collaborative Meta-Analysis of Randomized Statin Trials. *The Lancet*. February 2010. Online. <http://www.thelancet.com/journals/lancet/article/PIIS0140-6736%2809%2961965-6/abstract>
- ⁹⁵ FDA Consumer Health Information. FDA Expands Advice on Statin Risks. FDA.gov. January 2014. Online. <http://www.fda.gov/downloads/ForConsumers/ConsumerUpdates/UCM293705.pdf> (Accessed February 2015)

- ⁹⁶ Iwamoto T, et al. Steroid-Induced Diabetes Mellitus and Related Risk Factors in Patients with Neurologic Diseases. *PharmaCotherapy*. January 16, 2012. Online. <http://onlinelibrary.wiley.com/doi/10.1592/phco.24.5.508.33355/abstract> (Accessed February 2015)
- ⁹⁷ Wolff FW, et al. Drug-Induced Diabetes: Diabetogenic Activity of Long-Term Administration of Benzothiadiazines. *JAMA*. August 17, 1963. Online. <http://jama.jamanetwork.com/article.aspx?articleid=666535> (Accessed February 2015)
- ⁹⁸ Gress TW, et al. Hypertension and Antihypertensive Therapy as Risk Factors for Type 2 Diabetes Mellitus. *NEJM*. March 30, 2000. Online. <http://www.nejm.org/doi/full/10.1056/NEJM200003303421301> (Accessed February 2015)
- ⁹⁹ Lean MEJ, Pajonk F-G. Patients on Atypical Antipsychotic Drugs: Another High-Risk Group for Type 2 Diabetes. *Diabetes Care*. May 2003. Online. <http://care.diabetesjournals.org/content/26/5/1597.short> (Accessed February 2015)
- ¹⁰⁰ Walli R, et al. Treatment with Protease Inhibitors Associated with Peripheral Insulin Resistance and Impaired Oral Glucose Tolerance in HIV-1-Infected Patients. *AIDS Official Journal of the International AIDS Society*. October 1998. Online. http://journals.lww.com/aidsonline/Abstract/1998/15000/Treatment_with_protease_inhibitors_associated_with.1.aspx (Accessed February 2015)
- ¹⁰¹ Venn BJ, Green TJ. Glycemic Index and Glycemic Load: Measurement Issues and Their Effect on Diet-Disease Relationships. *European Journal of Clinical Nutrition*. December 2007. Online. <http://www.ncbi.nlm.nih.gov/pubmed/17992183> (Accessed February 2015)
- ¹⁰² American Diabetes Association. Diabetes Myths. *Diabetes.org*. 2015. Online. <http://www.diabetes.org/diabetes-basics/myths/> (Accessed February 2015)
- ¹⁰³ Caloriecount.com. Calories in Wafers. Online. <http://www.caloriecount.com/calories-nilla-wafers-i82409> (Accessed February 2015)
- ¹⁰⁴ American College of Neuropsychopharmacology. Fructose and Glucose: Brain Reward Circuits Respond Differently to Two Kinds of Sugar. *Science Daily*. December 10, 2014. Online. <http://www.sciencedaily.com/releases/2014/12/141210080734.htm> (Accessed February 2015)
- ¹⁰⁵ Wells HF, Buzby JC. Dietary Assessment of Major Trends in US Food Consumption, 1970-2005. *USDA Bulletin No. 33*, page 18. March 2008. Online. http://www.ers.usda.gov/media/210681/eib33_1.pdf (Accessed February 2015)
- ¹⁰⁶ Bocarsly ME, et al. High-Fructose Corn Syrup Causes Characteristics of Obesity in Rats: Increased Body Weight, Body Fat and Triglyceride Levels. *Pharmacology, Biochemistry and Behavior*. November 2010. Online. <http://www.ncbi.nlm.nih.gov/pubmed/20219526> (Accessed February 2015)
- ¹⁰⁷ DiNicolantonio JJ, O'Keefe JH, Lucan SC. Added Fructose: A Principal Driver of Type 2 Diabetes Mellitus and Its Consequences. *Mayo Clinic Proceedings*. January 26, 2015. Online. <http://www.ncbi.nlm.nih.gov/pubmed/25639270> (Accessed February 2015)
- ¹⁰⁸ American Chemical Society. Soda Warning? High-Fructose Corn Syrup Linked to Diabetes, New Study Suggests. *ScienceDaily*. August 23, 2007. Online. <http://www.sciencedaily.com/releases/2007/08/070823094819.htm> (Accessed February 2015)
- ¹⁰⁹ Flegal KM, et al. Prevalence of Obesity and Trends in the Distribution of Body Mass Index among US Adults, 1999-2010. *JAMA*. February 1, 2012. Online. <http://jama.jamanetwork.com/article.aspx?articleid=1104933> (Accessed February 2015)
- ¹¹⁰ Bray GA, Nielsen SJ, Popkin BM. Consumption of High-Fructose Corn Syrup in Beverages May Play a Role in the Epidemic of Obesity. *American Journal of Clinical Nutrition*. April 20, 2004. Online. <http://ajcn.nutrition.org/content/79/4/537.full.pdf+html> (Accessed February 2015)
- ¹¹¹ Parks EJ, et al. Dietary Sugars Stimulate Fatty Acid Synthesis in Adults. *The Journal of Nutrition*. June 2008. Online. http://www.ncbi.nlm.nih.gov/pubmed/18492831?ordinalpos=1&itool=EntrezSystem2.PEntrez.Pubmed.Pubmed_ResultsPanel.Pubmed_RVDocSum (Accessed February 2015)
- ¹¹² Stanhope KL, et al. Consumption of Fructose and High Fructose Corn Syrup Increase Postprandial Triglycerides, LDL-Cholesterol, and Apolipoprotein-B in Young Men and Women. *The Journal of Clinical Endocrinology & Metabolism*. August 17, 2011. Online. <http://press.endocrine.org/doi/abs/10.1210/jc.2011-1251> (Accessed February 2015)
- ¹¹³ Schaefer EJ. Dietary Fructose and Glucose Differentially Affect Lip and Glucose Homeostasis. *The Journal of Nutrition*. June 2009. Online. <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2682989/> (Accessed February 2015)
- ¹¹⁴ Kishner T, Werman MJ. Long-Term Fructose Intake: Biochemical Consequences and Altered Renal Histology in the Male Rat. *Metabolism*. December 2002. Online. <http://www.sciencedirect.com/science/article/pii/S0026049502001579> (Accessed February 2015)
- ¹¹⁵ Bocarsly ME, et al. High-Fructose Corn Syrup Causes Characteristics of Obesity in Rats: Increased Body Weight, Body Fat and Triglyceride Levels. *Pharmacology Biochemistry and Behavior*. November 2010. (Correction in 2012). Online. <http://www.sciencedirect.com/science/article/pii/S0091305710000614> (Accessed February 2015)
- ¹¹⁶ Basciano H, Federico L, Adeli K. Fructose, Insulin Resistance, and Metabolic Dyslipidemia. *Nutrition & Metabolism*. February 21, 2005. Online. <http://www.nutritionandmetabolism.com/content/2/1/5> (Accessed February 2015)
- ¹¹⁷ The Nutrition Source. Fats and Cholesterol: Out with the Bad, In with the Good. *Harvard School of Public Health*. 2015. Online. <http://www.hsph.harvard.edu/nutritionsource/fats-full-story/#references> (Accessed February 2015)
- ¹¹⁸ Risérus U, Willett WC, Hu FB. Dietary Fats and Prevention of Type 2 Diabetes. *Progress in Lipid Research*. January 2009. Online. <http://www.ncbi.nlm.nih.gov/pubmed/19032965> (Accessed February 2015)
- ¹¹⁹ Gollison KS, et al. Gender Dimorphism in Aspartame-Induced Impairment of Spatial Cognition and Insulin Sensitivity. *PLOS One*. April 3, 2012. Online. <http://journals.plos.org/plosone/article?id=10.1371/journal.pone.0031570> (Accessed February 2015)
- ¹²⁰ Hu FB. Globalization of Diabetes: The Role of Diet, Lifestyle, and Genes. *Diabetes Care*. June 2011. Online. <http://care.diabetesjournals.org/content/34/6/1249.full> (Accessed February 2015.)
- ¹²¹ Parker CP. Caring for Diabetes in China: How Clinical Care in China Differs from the US. *Diabetes Forecast*. January 2015.
- ¹²² King BJ. For How Long Have We Been Human? *NPR*. September 13, 2012. Online. (Accessed February 2015)
- ¹²³ Carr K. Sugar. *Historyforkids.org*. 2014. Online. <http://www.historyforkids.org/learn/food/sugar.htm> (Accessed February 2015)
- ¹²⁴ Carr K. More Sugar. *Historyforkids.org*. 2014. Online. <http://www.historyforkids.org/learn/food/sugar2.htm> (Accessed February 2015)
- ¹²⁵ Carr K. More Sugar History for Kids. *Historyforkids.org*. 2014. Online. <http://www.historyforkids.org/learn/food/sugar3.htm> (Accessed February 2015)
- ¹²⁶ Polonsky KS. The Past 200 Years in Diabetes. *NEJM*. December 31, 2012. Online. <http://www.nejm.org/doi/full/10.1056/NEJMra1110560> (Accessed February 2015)
- ¹²⁷ Howell E. How Long Have Humans Been On Earth? *Universe Today*. January 19, 2015. Online. (Accessed February 2015)
- ¹²⁸ Lakhan SE, Kirchgessner A. The Emerging Role of Dietary Fructose in Obesity and Cognitive Decline. *Nutrition Journal*. August 8, 2013. Online. <http://www.nutritionj.com/content/12/1/114#B97> (Accessed February 2015)
- ¹²⁹ Menke A, et al. Associations between Trends in Race/Ethnicity, Aging, and Body Mass Index with Diabetes Prevalence in the United States: A Series of Cross-Sectional Studies. *Annals of Internal Medicine*. September 2, 2014. Online. <http://annals.org/article.aspx?articleid=1900696> (Accessed February 2015)

-
- ¹³⁰ CDC FastStats. Obesity and Overweight. CDC. January 7, 2015. Online. <http://www.cdc.gov/nchs/fastats/obesity-overweight.htm> (Accessed February 2015)
- ¹³¹ Keats S, Wiggins S. Future Diets: Implications for Agriculture and Food Prices. Overseas Development Institute Report. January 2014. Online. <http://www.odi.org/sites/odi.org.uk/files/odi-assets/publications-opinion-files/8776.pdf> (Accessed February 2015)
- ¹³² WHO Media Centre. Obesity and Overweight. World Health Organization. January 2015. Online. <http://www.who.int/mediacentre/factsheets/fs311/en/> (Accessed February 2015)
- ¹³³ Obesity Prevention Source. Genes Are Not Destiny. Harvard School of Public Health: Powerful Ideas for a Healthier World. 2008-2015. Online. <http://www.hsppharvard.edu/obesity-prevention-source/obesity-causes/genes-and-obesity/> (Accessed February 2015)
- ¹³⁴ AAP. Breastfeeding Initiatives. AAP.org. 2015. Online. <http://www.aap.org/breastfeeding/faqsBreastfeeding.html> (Accessed February 2015)
- ¹³⁵ Huh SY, et al. Timing of Solid Food Introduction and Risk of Obesity in Preschool-Aged Children. Pediatrics. February 7, 2011. Online. <http://pediatrics.aappublications.org/content/early/2011/02/07/peds.2010-0740.full.pdf+html> (Accessed February 2015)
- ¹³⁶ Baby Center. Age-by-Age Guide to Feeding Your Baby. Baby Center Expert Advice. 2015. Online. http://www.babycenter.com/0_age-by-age-guide-to-feeding-your-baby_1400680.bc#articlesection1 (Accessed February 2015)
- ¹³⁷ Micha R, et al. Red and Processed Meat Consumption and Risk of Incident Coronary Heart Disease, Stroke, and Diabetes Mellitus. Circulation. May 17, 2010. Online. <http://circ.ahajournals.org/content/121/21/2271.full> (Accessed February 2015)
- ¹³⁸ Harvard School of Public Health. Eating Processed Meats, but not Unprocessed Red Meats, May Raise Risk of Heart Disease and Diabetes. Harvard News. May 17, 2010. Online. <http://www.hsppharvard.edu/news/press-releases/processed-meats-unprocessed-heart-disease-diabetes/> (Accessed February 2015)
- ¹³⁹ Angelieri CT, et al. Trans Fatty Acid Intake Is Associated with Insulin Sensitivity but Independently of Inflammation. Brazilian Journal of Medical and Biological Research. July 2012. Online. http://www.scielo.br/scielo.php?pid=S0100-879X2012000700009&script=sci_arttext (Accessed February 2015)
- ¹⁴⁰ Chowdhury R, et al. Association of Dietary, Circulating, and Supplement Fatty Acids with Coronary Risk: A Systematic Review and Meta-Analysis. Annals of Internal Medicine. March 18, 2014. Online. <http://annals.org/article.aspx?articleid=1846638#tab10Div> (Accessed February 2015)
- ¹⁴¹ Duncan GE, et al. Exercise Training, without Weight Loss, Increases Insulin Sensitivity and Postheparin Plasma Lipase Activity in Previously Sedentary Adults. Diabetes Care. March 2003. Online. <http://www.ncbi.nlm.nih.gov/pubmed/12610001?dopt=Abstract> (Accessed February 2015)
- ¹⁴² Young DR, et al. Associations between Physical Activity and Cardiometabolic Risk Factors Assessed in a Southern California Health Care System, 2010-2012. CDC: Preventing Chronic Disease. 2014. Online. http://www.cdc.gov/pcd/issues/2014/14_0196.htm (Accessed February 2015)
- ¹⁴³ Diabetes Prevention Program Research Group. Reduction in the Incidence of Type 2 Diabetes with Lifestyle Intervention or Metformin. NEJM. February 7, 2002. Online. <http://www.nejm.org/doi/full/10.1056/NEJMoa012512> (Accessed February 2015)
- ¹⁴⁴ NIH Fact Sheet for Health Professionals. Omega-3 Fatty Acids and Health. NIH Office of Dietary Supplements. October 28, 2005. Online. <http://ods.od.nih.gov/factsheets/Omega3FattyAcidsandHealth-HealthProfessional/> (Accessed February 2015)
- ¹⁴⁵ Aljabri KS. Glycemic Changes after Vitamin D Supplementation in Patients with Type 1 Diabetes Mellitus and Vitamin D Deficiency. Annals of Saudi Medicine. Nov-Dec 2010. Online. <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2994161/> (Accessed February 2015)
- ¹⁴⁶ Herrman M, et al. Serum 25-Hydroxy Vitamin D: A Predictor of Macrovascular and Microvascular Complications in Patients with Type 2 Diabetes. Diabetes Care. December 18, 2014. Online. <http://care.diabetesjournals.org/content/early/2014/12/17/dc14-0180.abstract> (Accessed February 2015)
- ¹⁴⁷ Dalgard C, et al. Vitamin D Status in Relation to Glucose Metabolism and Type 2 Diabetes in Septuagenarians. Diabetes Care. June 2011. Online. <http://care.diabetesjournals.org/content/34/6/1284.full> (Accessed February 2015)
- ¹⁴⁸ Avas NT, et al. A Prospective Study of Self-Reported Sleep Duration and Incident Diabetes in Women. Diabetes Care. February 2003. Online. <http://www.ncbi.nlm.nih.gov/pubmed/12547866?dopt=Abstract> (Accessed February 2015)
- ¹⁴⁹ Spiegel K, et al. Leptin Levels Are Dependent on Sleep Duration: Relationships with Sympathovagal Balance, Carbohydrate Regulation, Cortisol, and Thyrotropin. The Journal of Clinical Endocrinology and Metabolism. November 2004. Online. <http://www.ncbi.nlm.nih.gov/pubmed/15531540>
- ¹⁵⁰ Medical News Today. A "Perfect Storm" for Inflammation—Bacteria and Fat—May Promote Diabetes. Medicalnewstoday.com. November 2013. Online. <http://www.medicalnewstoday.com/releases/268223.php> (Accessed February 2015)
- ¹⁵¹ Vu BG, et al. Staphylococcal Superantigens Stimulate Immortalized Human Adipocytes to Produce Chemokines. PLOS One. October 30, 2013. Online. <http://journals.plos.org/plosone/article?id=10.1371/journal.pone.0077988> (Accessed February 2015)
- ¹⁵² Long GH, et al. Healthy Behaviours and 10-Year Incidence of Diabetes: A Population Cohort Study. Preventive Medicine. February 2015. Online. <http://www.sciencedirect.com/science/article/pii/S0091743514004952> (Accessed February 2015)
- ¹⁵³ Hackethal V. Resolve to Be Healthier May Actually Help Reduce T2DM. Consultant Live. December 30, 2014. Online. <http://www.sciencedirect.com/science/article/pii/S0091743514004952> (Accessed February 2015)