THIN Diabetes,  
FAT Diabetes  

Prevent Type 1  
and  
Cure Type 2
THIN Diabetes, FAT Diabetes

Prevent Type 1 and Cure Type 2

Laurie Endicott Thomas
Disclaimer

I am a science writer. I am not your doctor. If you have any questions about your own health or about what you should eat, please see a licensed healthcare professional. If you have food allergies or intolerances or any metabolic disease or are taking any prescription medications, talk to your healthcare provider before you make any major change in diet. Diabetics who make a major change in diet without adjusting their prescription medicine can end up hospitalized or dead from low blood sugar.
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When people talk about diabetes (diabetes mellitus), they could mean any of several different diseases, all of which produce high blood sugar. Many people and even many doctors think that people with diabetes should avoid eating carbohydrates (sugars and starches). In reality, a starchy, low-fat diet is good for anyone with any form of diabetes mellitus, and it can cure the most common form.

Diabetes mellitus, which is usually just called diabetes, is a medical term that can refer to any of several different diseases. Each of these diseases has a different cause, but all of them have one thing in common. If left untreated, they cause a sugar called glucose to build up to toxic levels in the bloodstream. Some of that glucose in the blood came from the carbohydrates (sugars and starches) that the person ate. However, the body can also make glucose out of other things, such as protein. (That’s why people can have sugar in their blood even if they eat no starch or sugar at all.) People don’t get diabetes mellitus from eating too much starch and sugar. In fact, diabetics need to eat a high-carbohydrate diet. Instead, diabetes mellitus means that the body has lost control over its blood chemistry.

Blood sugar is a Goldilocks and the Three Bears type of problem. Having too much sugar in the blood can kill you. So can having too little sugar in the blood. A healthy person’s body has a control system that normally keeps the blood sugar level just right (Figure 1). Diabetes means that something has gone wrong with this control system.
Thin diabetes is the form of diabetes that was always fatal in the days before insulin therapy. As a result, it has been called insulin-dependent diabetes. American doctors now call it type 1 diabetes.

### Table 1. The main types of diabetes mellitus.

<table>
<thead>
<tr>
<th>Type of Diabetes</th>
<th>Cause</th>
<th>Other Names</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thin Diabetes</td>
<td>Lack of insulin, usually after the immune system has destroyed the insulin-producing cells of the pancreas but sometimes after removal of the pancreas.</td>
<td>Type 1 diabetes, juvenile diabetes, insulin-dependent diabetes (IDDM), pancreatic diabetes, latent autoimmune diabetes of adults (slowly developing cases).</td>
</tr>
<tr>
<td>Fat Diabetes</td>
<td>Resistance to the effects of the hormone insulin, in overweight people who eat a fatty diet.</td>
<td>Type 2 diabetes, adult-onset diabetes, non-insulin-dependent diabetes (NIDDM).</td>
</tr>
<tr>
<td>Gestational Diabetes</td>
<td>Temporary resistance to the effects of insulin during pregnancy.</td>
<td>—</td>
</tr>
<tr>
<td>Genetic Diabetes</td>
<td>A genetic defect that leads to low insulin production or poor sensitivity to insulin.</td>
<td>Monogenic diabetes; there are several different kinds, including infantile-onset diabetes and maturity-onset diabetes of the young (MODY).</td>
</tr>
</tbody>
</table>

French-speaking people use the term *fat diabetes* (*diabète gras*) to refer to the relatively mild type of diabetes that occurs in overweight people and that can be cured by losing weight. This form of diabetes has been called adult-onset diabetes. But now that there are so many overweight children, pediatricians are seeing more cases of fat diabetes than thin diabetes among children. Fat diabetes has also been called non-insulin-dependent diabetes. But that term became confusing because so many people with fat diabetes are now taking insulin. American doctors now call it type 2 diabetes. A temporary case of fat diabetes that occurs during pregnancy is called gestational diabetes.
Most people with diabetes believe that their diabetes is genetic, which to them means that they were doomed to get it. However, studies of identical twins show us that genes play only a small role in the cause of thin diabetes and fat diabetes. Only a few, rare forms of diabetes are really due to a bad gene. Most of the truly genetic forms of diabetes are called monogenic diabetes because they can be linked to a mutation in one particular gene. Some of these genetic forms of diabetes are so mild that they are often mistaken for fat diabetes, if they are diagnosed at all. These mild forms are often easily managed with diet or pills. Other forms of genetic diabetes are so severe that they are easily mistaken for thin diabetes. These severe forms of genetic diabetes must be managed with insulin therapy, just like thin diabetes.

In a fasting person, blood glucose normally stays between 70 and 100 milligrams per deciliter (mg/dL). To qualify for a diagnosis of diabetes mellitus, a patient has to meet at least 1 of the following criteria:

- A glycated hemoglobin level (HbA1c) of at least 6.5% (for an explanation of HbA1c, see chapter 12).
- A blood sugar level of at least 126 mg/dL in a blood sample taken when the patient is fasting (i.e., has not eaten anything for at least 8 hours).
- A blood glucose level of at least 200 mg/dL during a glucose tolerance test (see chapter 4).
- A blood glucose level of at least 200 mg/dL in a “casual” blood sample (i.e., a blood sample taken without regard to when the patient ate last).

Patients whose fasting glucose is between 100 and 125 mg/dL are said to have “prediabetes.” They are considered to be at risk of developing thin diabetes or fat diabetes.

To figure out what kind of diabetes mellitus the patient has, doctors use other kinds of tests. To see whether the pancreas is making insulin, doctors measure the amount of insulin and C-peptide (a protein that is made along with insulin) in the blood. The doctor can also test the patient for the antibodies that attack the pancreas. Patients who have low insulin production but no antibodies to their pancreas may have one of the genetic forms of diabetes, which I describe in chapter 16. Note that you can have more than one type of diabetes at the same time. People with thin diabetes or one of the rare genetic forms of diabetes can also have some degree of insulin resistance.

The goal in managing fat diabetes should be to cure the fat diabetes by correcting the diet. Fat diabetes is not really a disease. It is a defense against weight gain. Fat diabetes is actually a way for the body to resist gaining even more weight from a fattening, fatty diet. When people with fat diabetes switch to a low-fat (<10% of calories), high-fiber, high-carbohydrate diet, the fat diabetes goes away, even if they eat enough food to feel full.

Unfortunately, there is no cure for thin diabetes. However, there’s a good chance that many cases could be prevented. As I explain in chapter 11, thin diabetes can be triggered by a protein in cow’s milk. Simply removing animal milk from the diet could dramatically reduce the number of new cases of thin diabetes.

For people with the incurable forms of diabetes, the goal should be to keep blood sugar within a reasonable range. In the short run, severely high or low blood sugar can lead to sudden death. In the long run, even moderately high blood sugar can increase the risk of complications, such as blindness.

Dietary therapy is the key to managing any metabolic disease, including diabetes. The appropriate diet for someone with any form of diabetes is a low-fat, purely plant-based (vegan) diet. This kind of diet cures fat diabetes. It also gives people with the incurable forms of diabetes the best possible chance for long-term survival with healthy eyes, feet, and kidneys. Such a diet provides enough of all of the essential nutrients except for vitamin D (which you can get by exposing your skin to sunshine) and vitamin B₁₂ (which comes from bacteria). The only supplement that is routinely recommended for people on this kind of diet is vitamin B₁₂.

The diet should be based on unrefined starches: whole grains or starchy vegetables, such as potatoes or sweet potatoes. The diet should include lots of vegetables and fruit. However, the person with diabetes
should not eat any meats, dairy foods, fish, eggs, or foods that contain oil or shortening. In other words, diabetics should not eat anything that came from an animal. They should also avoid any concentrated fats and oils, even the supposedly healthy oils such as olive oil and canola oil (Table 2).

Table 2. Foods for people with diabetes to eat or avoid.

<table>
<thead>
<tr>
<th>Eat</th>
<th>Do Not Eat</th>
</tr>
</thead>
<tbody>
<tr>
<td>Whole grains, including brown rice, whole wheat, corn, quinoa, buckwheat, etc.</td>
<td>Meat</td>
</tr>
<tr>
<td>Starchy vegetables, including potatoes, sweet potatoes, winter squashes, and cassava</td>
<td>Fish</td>
</tr>
<tr>
<td>Other vegetables</td>
<td>Dairy foods (milk, yogurt, cheese, butter)</td>
</tr>
<tr>
<td>Fruits</td>
<td>Eggs</td>
</tr>
<tr>
<td>Beans and peas and lentils (legumes)</td>
<td>Oils (including olive oil and canola oil) and foods made with oil or shortening</td>
</tr>
</tbody>
</table>

The diet that is good for diabetics is also good for people who do not have diabetes. It helps them lose excess weight without feeling hungry. It cleans out their arteries and eliminates their risk of heart attack. It reduces their blood pressure and with it their risk of stroke. It decreases their risk of cancer and osteoporosis and autoimmune disease and Alzheimer’s disease. Thus, if a family changes its diet to help a diabetic family member, everyone can benefit.

Many people who are eating the rich and fatty standard American diet suffer from high blood sugar and high blood pressure. Many of those people are taking prescription drugs to reduce their blood sugar and blood pressure. If they change to a healthy diet, they can stop taking most of those medications. But if they make a sudden change in diet while taking those medications, they could faint from low blood pressure or even die from low blood sugar. That’s why people who are sick or who are taking any prescription medication should talk to their healthcare professional before making any major change in diet.

Most people are surprised to hear that the most common form of diabetes is curable. They are even more surprised to hear that diabetics should eat lots of starchy foods, such as rice, bread, and potatoes. Most diabetics have been told to limit their intake of starchy foods because starch gets broken down into glucose. Why am I telling them something that is so different from what they have read on the Internet, or even from what they have heard from their doctor?

I became interested in diabetes when I was a teenager, after my grandmother got a diagnosis of what she called sugar diabetes. She eventually died of complications of her diabetes. Since then, I have met many other people with various types of diabetes. I have known several people who eventually died because of their diabetes. I have also met people who have lost their eyesight or their feet or needed a kidney transplant because of diabetes. Clearly, something had to be done to stop all this preventable death and suffering.

I learned a lot about diabetes from working as an editor in medical publishing. I learned a lot about nutrition and dietetics from working on nutrition textbooks. I also learned a lot about diabetes from working for a veterinary journal. Many dogs and cats get diabetes. Diabetes in a dog or a cat is a lot like diabetes in a human being. The insulin products that veterinarians prescribe for diabetic dogs and cats were actually made for human beings with diabetes. Diabetic dogs and cats can also suffer from the same kinds of complications as human diabetics, such as diabetic ketoacidosis. The treatment for those complications is pretty much the same for a dog or a cat as it is for a human being. However, I was originally told that there was one important difference. Fat diabetes in a cat would go away if the cat lost weight. To my delight, I found that fat diabetes in a human being can also be cured by weight loss. It can be cured even faster if the person switches from a fatty diet to a starchy diet.

I wrote this book because 30 million people in the United States have diabetes. Diabetes is our seventh leading cause of death in the
United States, and it does not have to be. Diabetes is also our leading cause of new cases of blindness, and it is the main medical reason for people to lose their feet. Diabetes is also a major cause of kidney failure. It increases the person’s risk of heart attack, stroke, liver disease, cancer, osteoporosis, and Alzheimer’s disease. Yet these deaths and this suffering could be prevented if people understood how to cure fat diabetes and prevent thin diabetes. People are not being told how to cure their fat diabetes. Nor are parents being told about the role of cow’s milk in causing thin diabetes. People with thin diabetes are not being told about the value of a low-fat, high-carbohydrate diet for preventing complications. All of these facts are clearly documented in the scientific literature, but few laymen know them.

Even many medical doctors have been misinformed about nutrition and diabetes. There are two reasons for this problem. One is the neglect of nutrition in medical schools. For decades, medical schools in the United States have been neglecting nutrition and dietetics. The American Medical Association issued reports about this problem in the 1960s and 1970s. The National Academy of Sciences issued its own report in 1985. The American Society for Clinical Nutrition published yet another report in 2006. It’s a chronic problem that is not going away by itself. If the medical profession does not solve this problem, then public health activists will have to solve it.

The other problem was simply awkward timing. Insulin was discovered in 1921. As a result, much of the important research on diet for people with diabetes was done in the 1920s through the 1950s. Unfortunately, PubMed, which is a computerized database of medical journal articles, originally went back only to 1966. As a result, doctors could easily find the reports of the studies of the new diabetes drugs but remained largely unaware of the important dietary studies. One of the goals of this book is to let doctors and patients know about the important dietary research from the 1920s through the 1950s.

Today, most doctors focus on the blood sugar and ignore the fat in the diet. They tell their patients to avoid eating sugars and starches, to keep their blood sugar from going too high. Yet that means that the patients will get a high percentage of their calories from fat. Unfortunately, this fatty diet that doctors are urging their patients to eat can actually make the fat diabetes worse. It can also cause fat diabetes in someone with thin diabetes.

Doctors use prescription drugs to control the blood sugar in patients with fat diabetes. Unfortunately, when doctors make heavy use of those drugs to get really tight control of the patient’s blood sugar, the patient’s risk of death actually goes up. Instead, doctors should be teaching their patients with fat diabetes how to cure their diabetes by changing their diet.

Many of the things that I say in this book go against conventional wisdom. But you do not have to take my word for any of them. My goal in writing this book is to give you enough background knowledge about the history and biology of diabetes that you can figure out for yourself who is telling the truth. Diabetes has been a mystery since ancient times. In this book, I explain how that mystery was solved. Then you shall know the truth, and the truth shall set you free.

Summary

- The term diabetes mellitus refers to any of several diseases that cause sugar to build up in the bloodstream. It can refer to thin diabetes, fat diabetes, gestational diabetes, or monogenic diabetes.
- Thin diabetes is a severe, incurable disease that results from failure of the pancreas to produce insulin. It must be treated with insulin replacement. It has also been called juvenile diabetes, insulin-dependent diabetes, and type 1 diabetes.
- Fat diabetes is a milder problem that results from resistance to the effects of insulin. It has been called adult-onset diabetes, non-insulin-dependent diabetes, and type 2 diabetes. It can be cured by eating a low-fat, starchy diet.
- The same low-fat, starchy diet that cures fat diabetes and gestational diabetes is good for people with thin diabetes.
- The truly genetic forms of diabetes account for only about 2% of all cases of diabetes.
The pancreas has a large duct that empties into the duodenum, which is the first few inches of the small intestine. If a surgeon tied off a dog’s pancreatic duct, so that the pancreas could no longer empty into the small intestine, the dog would develop minor digestive problems. However, it would not develop diabetes. But if the pancreas were surgically removed, the dog would get thin diabetes. These experiments showed that diabetes resulted from the lack of a hormone that the pancreas normally releases into the bloodstream, not something that the pancreas releases through the pancreatic duct into the small intestine. In other words, thin diabetes results from the failure of an endocrine function of the pancreas, as opposed to its exocrine functions.

If you look at pancreatic tissue through a microscope, you can see that the pancreas is really one big exocrine gland that contains about a million tiny endocrine glands, which tend to be clustered around blood vessels. These tiny endocrine glands are called the islets of Langerhans, after the German anatomist who discovered them in 1869.

To unravel the mystery of thin diabetes, scientists had to figure out what those islets of Langerhans were producing.

Part of this mystery was unraveled in 1921 by two Canadians, a surgeon named Frederick Banting and a medical student named Charles Best. Banting knew that the pancreas makes enzymes that help you digest meat. If the duct that leads from the pancreas is blocked, those digestive enzymes start to digest the pancreas itself. However, Banting also knew that the damage would be limited to the tissues that are responsible for the exocrine function of the pancreas. The islets of Langerhans...
The introduction of insulin therapy was one of the most dramatic scenes in the history of medicine. Comatose children were brought back from the brink of death. Children who had been starved until they looked like living skeletons went back to looking like healthy normal children (Figure 5).

Figure 5. A girl who was dying of thin diabetes (left) survived and thrived on insulin (right). (Image courtesy of Wellcome Library, London.)

Insulin is a peptide, or small protein, which means that it has to be taken by injection. If you took a dose of insulin by mouth, the insulin would be digested in your stomach and intestines. Thus, it would be broken down before being absorbed into your bloodstream. Even if you inject insulin, the effects of a single dose do not last long.

The natural insulin that Banting, Best, and Collip isolated acts quickly. However, its effects last for less than 6 hours after the injection. As I’ll explain in more detail in chapter 12, scientists quickly figured out how to make longer-lasting insulin preparations by mixing regular insulin with zinc or with a protein called protamine.

Starting in the 1980s, researchers have developed genetically engineered forms of insulin. Some of these products are based on insulin...
The development of insulin replacement therapy for people with thin diabetes was a major breakthrough that saved many lives. But even though insulin saves lives, insulin does not cure thin diabetes. Insulin merely allows the person to live with the diabetes. Despite the availability of insulin, thin diabetes remains a serious, life-altering, and potentially deadly disease. To understand how serious it is, you need to understand what insulin does. After you understand that, you will also understand why it’s important for people with fat diabetes to reverse their insulin resistance, not just control their blood sugar.

**Summary**

- Insulin is a hormone made by the pancreas. If the pancreas stops making insulin, the person needs insulin replacement therapy in order to survive.
- The form of diabetes mellitus that results from lack of insulin has been called thin diabetes, juvenile diabetes, insulin-dependent diabetes, and type 1 diabetes.
- The development of insulin therapy transformed thin diabetes from a death sentence to a manageable but still serious disease.
- The management of thin diabetes is a balancing act. Someone with thin diabetes can die from taking too much or too little insulin.

Thanks to the discovery of insulin in 1921, many children with thin diabetes (type 1 diabetes) were snatched from the jaws of death and went on to lead long, happy, and productive lives. Insulin did not cure thin diabetes. Rather, it allowed people to live despite their thin diabetes. Curiously, insulin therapy had much less of an effect on people with fat diabetes, which was far more common than thin diabetes. In fact, if you look at a graph of diabetes deaths in the early 20th century, you might get the false idea that insulin had no effect at all (Figure 6).

Insulin therapy did save lives—a lot of lives, mainly the lives of children. But it saved the lives of people with thin diabetes (type 1 diabetes): the uncommon but severe type of diabetes that results from failure of the pancreas. The introduction of insulin therapy had no effect on the death rate from fat diabetes (type 2 diabetes): the milder, more common form of diabetes that results from being overweight.

Figure 6 shows that the death rate from diabetes was high during times of peace and prosperity. It dropped off sharply during wartime food rationing. Figure 7 helps to explain why. **The death rate from diabetes dropped as people ate less fat and more starch.**
vegetables as they wanted. However, it strictly limited their intake of meats, fats, and alcohol. A small ration of milk was allowed because of concerns about pellagra and beriberi. Those diseases were known to result from the deficiency of vitamins that could be found in milk. Today, we know that those vitamins can be found in many plant foods, as well.

These results from Britain were not a fluke. The same thing happened in Denmark during World War I. Denmark was able to remain neutral during World War I. Nevertheless, the Danes were at risk of starvation because their food imports were practically cut off by the Atlantic Blockade. To deal with that emergency, the Danish government asked a physician and nutrition researcher named Mikkel Hindhede to design a food rationing system.

Before the war, Danes had been importing about half of their grain supply. Much of that grain was being fed to farm animals. Then, the people would eat the meat, milk, and eggs from the animals. The Danes were also using a lot of grain to make beer and other alcoholic beverages. Following Hindhede’s advice, the Danish government made sure that the available grain was used to feed people, not farm animals. Hindhede’s rationing system let people have as much bread and potatoes and

Figure 6. Deaths from diabetes in England and Wales in the early 20th century. The graph shows the mortality index that was calculated relative to the year 1938. Most of these deaths were in people with type 2 diabetes (fat diabetes). Notice that diabetes mortality dropped during wartime, when fatty foods were in short supply and strictly rationed. (From Young FG: Discussion on the cause of diabetes. Proc R Soc Med. 1949 May; 42[5]: 321-330. Reproduced with permission.)

Figure 7. In Britain, food rationing during World War II saved the lives of diabetics. Fewer people died of diabetes because the population was eating less fat and more starch. (From Young FG: Discussion on the cause of diabetes. Proc R Soc Med. 1949 May; 42[5]: 321-330. Reproduced with permission.)
hormone. It tells your body to make big molecules out of little ones, and to build tissue instead of tearing it down.

As a healthy person uses up or stores the nutrients from a meal, that person’s blood sugar will begin to drop. As the blood sugar level drops, the pancreas stops releasing insulin. Meanwhile, the liver keeps removing insulin from the bloodstream. As a result, the insulin levels in the bloodstream decrease. When the insulin levels are low, the pancreas starts to release glucagon. Glucagon is the hormone that tells the liver and fat cells to release the stored nutrients, for the rest of the body to use.

Glucagon tells the liver to turn some of its glycogen back into glucose, as well as to make glucose out of some other substances, such as some of the amino acids that come from protein. Glucagon also tells the fat cells to break down and release some of their stored fat, so that other cells in the body can use it for energy. In other words, glucagon is the hormone that tells your liver and fat cells to feed the rest of your body while you are fasting. Thus, glucagon is a catabolic (“tearing down”) hormone. It tells the body to break big molecules down into smaller ones, and to tear down tissue. Table 3 summarizes how this system normally works.

Many people, and even many textbooks, say that insulin is needed for glucose to enter cells. That’s not true. Even if there is no insulin in the body at all, glucose can easily enter many kinds of cells, especially the cells of the liver and brain. Otherwise, people with untreated thin diabetes (type 1 diabetes) would never have been able to survive for up to a year or even longer on the starvation diet described in chapter 2.

To understand insulin, you need to understand how glucose enters cells. To understand that, you have to know that glucose does not dissolve in oil. As a result, glucose cannot dissolve in the oily membrane that surrounds every cell. To enter the cell, glucose must therefore pass through some sort of doorway in the cell membrane. Insulin is the key that opens one type of doorway through which glucose can enter a cell.
To understand cell membranes, you need to know a little bit about electricity and chemistry. A cell membrane is made out of a double layer (bilayer) of an oily substance (a lipid). Substances that can dissolve in oil can pass right through this lipid bilayer. As a result, they can easily enter or leave a cell. To dissolve easily in oil, an atom or molecule must be electrically neutral (i.e., it must have just as many electrons as protons) and nonpolar (the negatively charged electrons in its outer shell must be distributed in a balanced way). Oxygen and carbon dioxide are neutral, nonpolar molecules. That’s why they can pass right through the cell membrane. In contrast, ions (i.e., electrically charged atoms and molecules) have a hard time passing through the cell membrane. So do polar molecules, which are molecules whose outer electrons are distributed in an unbalanced way (Figure 11).

An ion is an atom or molecule that has a net electrical charge, either positive or negative. Positively charged ions have more protons than electrons. Examples include hydrogen (H⁺), sodium (Na⁺), potassium (K⁺), and calcium (Ca²⁺) ions. Negatively charged ions have more electrons than protons. Examples include chloride (Cl⁻) and bicarbonate (HCO₃⁻). To enter or leave a cell, ions must be either allowed to pass through some sort of gate or carried across by a transporter. Even the smallest ions (hydrogen ions, which consist of a single proton) cannot pass through the lipid bilayer at a significant rate without this kind of help.

A few polar molecules, such as water and alcohol, are small enough to sneak through the lipid bilayer. Glucose is a polar molecule, which explains why it dissolves easily in water but not in fat. However, the glucose molecule is too big to slip through the lipid bilayer (Figure 12). As a result, glucose depends on some sort of transporter to allow it enter or leave cells.

<table>
<thead>
<tr>
<th>Nonpolar Molecules (Dissolve in Oil)</th>
<th>Polar Molecules (Do Not Dissolve in Oil)</th>
<th>Ions (Do Not Dissolve in Oil)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oxygen (O₂), carbon dioxide (CO₂), steroid hormones</td>
<td>Water (H₂O), ethanol (C₂H₅O)</td>
<td>H⁺, Na⁺, Cl⁻, K⁺, Mg²⁺, Ca²⁺</td>
</tr>
</tbody>
</table>

Figure 11. Oxygen (O₂) and carbon dioxide (CO₂) are nonpolar molecules because their outer electrons are distributed in a balanced way. Water, ethanol (beverage alcohol), and glucose are polar molecules because their outer electrons are distributed in an unbalanced way. In these electron density maps, the white areas indicate areas of high electron density, while the black areas indicate areas of low electron density.

Figure 12. Substances that dissolve in oil (such as steroid hormones) and small, polar molecules (such as water and ethanol) can pass through the lipid bilayer that makes up a cell membrane. Large polar molecules and electrically charged atoms and molecules cannot.
To enter or leave a cell, an ion or a large polar molecule such as glucose or an amino acid must find the right kind of doorway in the cell membrane (Figure 13). These doorways are created by membrane transport proteins (transporters) that are embedded in the lipid bilayer. There are many different kinds of transporter. Most of them allow only one kind of substance or a few substances to pass in or out of the cell. Some transporters create a channel that allows certain kinds of molecules to flow in or out under their own power. Some of those transporters stay open all the time, while others can be opened or closed like a gate. Some membrane transport proteins serve as pumps. These pumps can pull a substance “uphill,” either from an area of lower concentration to an area of higher concentration or against an electrical voltage gradient.

The transporters that allow glucose, fructose, and other hexoses (six-carbon sugars) to enter or leave a cell are called hexose transporters. Glucose can pass through a hexose transporter tens of thousands of times faster than it can seep through a plain lipid bilayer. More than a dozen different kinds of hexose transporter have been found on human cells. The recipe for each type of transporter is encoded in a different gene.

The first glucose transporter to be discovered was called the type 1 glucose transporter — GLUT1 for short. GLUT1 is found on cells throughout the body. However, it is particularly common on red blood cells and on the cells of the blood-brain barrier. Glucose can pass right through a GLUT1 transporter even if there is no insulin. Thus, red blood cells and the brain can get glucose, even if there is no insulin in the body.

In human beings, only one type of glucose transporter is responsive to insulin: the type 4 glucose transporter (GLUT4), which is found on muscle cells and fat cells. Like other transporters, the GLUT4 transporters are embedded in the cell membrane. However, the cell is continually pulling the GLUT4-rich portions of its cell membrane inwards. As these portions of cell membrane get pulled inward, they get pinched off to form internal compartments called vesicles. This process, which is called endocytosis, pulls the GLUT4 transporters inside the cell, where they have no effect (Figure 14). Eventually, some of the vesicles get pushed out again to fuse with the cell membrane, in a process called exocytosis. Once the GLUT4 transporter is back on the outside of the cell, it can let glucose into the cell.

In human beings, only one type of glucose transporter is responsive to insulin: the type 4 glucose transporter (GLUT4), which is found on muscle cells and fat cells. Like other transporters, the GLUT4 transporters are embedded in the cell membrane. However, the cell is continually pulling the GLUT4-rich portions of its cell membrane inwards. As these portions of cell membrane get pulled inward, they get pinched off to form internal compartments called vesicles. This process, which is called endocytosis, pulls the GLUT4 transporters inside the cell, where they have no effect (Figure 14). Eventually, some of the vesicles get pushed out again to fuse with the cell membrane, in a process called exocytosis. Once the GLUT4 transporter is back on the outside of the cell, it can let glucose into the cell.

The endocytosis-exocytosis cycle is going on all the time. When a healthy person is fasting, endocytosis dominates. When there is little or no insulin present, only about 5% of the GLUT4 transporters are on the outer surface of the cell. The rest are hidden inside the cell, where they cannot work. When insulin binds to an insulin receptor on the surface of a cell, it triggers a series of chemical reactions inside the cell. These reactions cause the vesicles that carry the GLUT4 transporters to be brought back to the cell’s surface. Even in the absence of insulin, muscle contraction can also cause exocytosis of the GLUT4-rich vesicles in a muscle cell. That’s one reason why exercise acts as “invisible insulin.”
This relationship between insulin and GLUT4 transporters allows the body to control how it uses glucose. The system allows the muscle and heart cells to use plenty of glucose when there is plenty of glucose in the blood. But when glucose is scarce, this system limits the amount of glucose that goes into muscle cells, thus reserving glucose for the brain. Of course, if the person needs to fight or flee, the muscle contractions will push some GLUT4 transporters to the surface, allowing the muscles to have some more glucose. Meanwhile, the stress hormones that are released during the fight-or-flight response will encourage the liver to release some extra sugar for the muscles to use.

Muscle and fat cells have a lot of GLUT4 transporters, which are stored inside the cell until brought to the surface under the influence of insulin. In contrast, liver cells have GLUT2 transporters, which are independent of insulin. However, the number of GLUT2 transporters on the surface of liver cells may be controlled by other hormones, such as thyroid hormone.

Instead of opening only under the influence of insulin, GLUT2 transporters act like revolving doors that allow glucose and a few similar substances to enter or leave the cell freely, to balance the concentration inside and outside of the cell. Rather than allowing liver cells to take in glucose, insulin tells liver cells what to do with glucose. Insulin tells the liver cells to convert glucose to glycogen for storage. When a liver cell converts glucose to glycogen, more glucose flows into the liver cell through the GLUT2 transporters. When a liver cell converts the glycogen back into glucose, then glucose flows back out of the cell through the GLUT2 transporters.

Insulin’s first and perhaps most powerful effect is within the pancreas itself. Insulin is produced by the beta cells in the islets of Langerhans, which are tiny specks of tissue scattered throughout the pancreas (Figure 15). The insulin produced by the beta cells tells the neighboring alpha cells to stop making glucagon. So the release of insulin (the feasting hormone) quickly switches off the production of glucagon (the fasting hormone).
When a person is fasting, glucagon keeps blood sugar from dropping too low. When a healthy person eats, the pancreas releases insulin. Insulin does two things to keep blood sugar levels from rising too high. It encourages the body’s cells to use or store glucose, thus taking glucose out of the bloodstream. Insulin also tells the pancreas to stop making glucagon until after the glucose and insulin levels in the bloodstream go back down.

So now that you understand what insulin and glucagon do, you can see why thin diabetes was always fatal in the days before insulin therapy. When there is no insulin in the body, the body thinks that it is starving, even when the person has just eaten. Without insulin, you can tolerate fasting surprisingly well, but you simply cannot cope with food. If the beta cells have died, no insulin is produced. So unless the person gets a dose of insulin from outside the body, the alpha cells will just keep on producing glucagon, even when it is not needed. As a result, the liver will keep releasing sugar into the bloodstream, even when blood sugar levels are already dangerously high (Figure 17).
If there is glucagon but no insulin in the system, many tissues throughout the body will think that the body is starving. Thus, they will break down some of their proteins so that the amino acids can be converted to glucose. The fat cells will release fat instead of storing it. That’s why people with untreated thin diabetes lose weight so fast. (It’s also why diabetics with eating disorders skip their insulin doses to lose weight—the worst weight-loss plan ever.)

When a healthy person is fasting, the glucagon that is produced by the pancreas tells the fat cells to release fatty acids for the rest of the body to use for fuel. Glucagon also tells the liver to make new glucose out of glycogen (glycogenolysis) and out of noncarbohydrate substances, such as amino acids (gluconeogenesis). However, the process of gluconeogenesis uses a substance that is involved in the process that cells normally use to burn sugar and fats for energy. When that substance is in short supply, some fatty acids are converted into acetoacetic acid, instead of being broken down completely into carbon dioxide and water. Some of that acetoacetic acid gets converted to beta-hydroxybutyric acid, and some of the acetoacetic acid breaks down to form acetone (Figure 18).

These three substances (acetoacetic acid, beta-hydroxybutyric acid, and acetone) have been called ketone bodies. However, they are dissolved molecules, not particles or bodies. Furthermore, beta-hydroxybutyric acid is not really a ketone; it is actually a carboxylic acid. To clear up the confusion, I will call these substances keto acids because they are acidic (even though acetone is a very weak acid.)

A healthy person produces trace amounts of these keto acids during normal metabolism. The production of these keto acids increases
Besides controlling how the body uses and stores glucose, insulin also helps to control how the body uses and stores fat. When you have just eaten, you do not need to continue using your fat stores for energy. The insulin that is normally produced in response to a meal suppresses the release of glucagon, which had been telling the fat cells to release fat for the rest of the body to use. Then, the insulin itself tells the fat cells to store the fat that has just been eaten. That fact helps to explain how the body normally uses the energy from a meal: it likes to burn the sugar right away and save the fat for later. That’s one reason why the human body generally does not turn much sugar into fat.

Insulin and glucagon also affect how the body handles protein. When glucagon levels are low, the liver stops converting amino acids into sugar. Insulin also tells cells to take in more amino acids, which they can then use to make protein. Thus, insulin has powerful anabolic effects, which means that it tends to promote the growth and repair of tissue. In fact, two other powerful growth-promoting hormones, which are called insulin-like growth factor 1 and 2, also activate insulin receptors.

Fat diabetes (type 2 diabetes) starts out as a way to resist storing too much fat. A little bit of body fat is a good thing. Body fat can help you survive a famine. But too much body fat is a problem. For one thing, being overweight makes it harder to run away from a predator. Most people’s bodies try to resist gaining too much weight. One way to resist further weight gain is to become less sensitive to insulin. If your fat cells become less sensitive to insulin, they will store less fat. They will also release more of their fat to be used as energy. When the heart and muscle cells are resistant to insulin, they will end up burning less sugar but more fat, even if there is plenty of sugar. Unfortunately, if the insulin resistance gets out of hand, the blood sugar can rise to toxic levels.

In people with fat diabetes, the beta cells of the pancreas are still producing insulin. The insulin levels in the bloodstream may actually be abnormally high (hyperinsulinemia). However, the body’s cells are not responding normally to that insulin. As a result, the body burns less glucose. If the alpha cells also become resistant to insulin, they will...
end up producing too much glucagon, thus causing blood sugar levels to rise even higher (Figure 20). The high blood sugar levels can then cause damage throughout the body. Nevertheless, this problem is not as severe as thin diabetes.

Fat diabetes does not result from eating a starchy or sugary diet. It results from eating too much fat. Fat diabetes is really a defense mechanism against weight gain. It represents the body’s attempt to prevent too much fat from flowing into the fat cells. To solve this problem, all you have to do is stop the flow of fat into the fat cells. The traditional way to solve this problem was to eat less, or even to fast, and to exercise more. But if you switch to a low-fat diet, you can get the same effect, even if you eat a satisfyingly large volume of food. Thus, the switch to a low-fat, high-carbohydrate diet is the long-term solution to the problem of fat diabetes.

Fat diabetes is really just an exaggerated form of something that happens during a normal pregnancy. A pregnant woman is “eating for two.” Just as low insulin levels during fasting help reserve sugar for the brain, a slight degree of insulin resistance in a pregnant woman helps to reserve sugar and protein for the growing fetus. Yet as I’ll explain in chapter 19, the mild insulin resistance that is normal during pregnancy can quickly escalate into a case of gestational diabetes if the woman eats too much fat and gains too much weight.

Many researchers have been struggling to figure out why some overweight people get fat diabetes while others do not. They view the diabetes as the problem. In reality, the real problem is overnutrition. The diabetes is the body’s attempt to keep the overnourished body from gaining even more weight.

Some people are more resistant to weight gain than other people are. Insulin resistance is just one tactic that a body can use to resist further weight gain. People who do not have insulin resistance as a defense mechanism are likely to gain even more weight. That’s why insulin shots and the drugs that are aimed at overcoming insulin resistance generally produce weight gain as a side effect.

The goal in managing fat diabetes ought to be to cure the problem by correcting the diet. People with fat diabetes respond to a change to a truly low-fat diet remarkably quickly. Significant improvements in the control of blood sugar can be seen within a few days. In fact, people respond so quickly to a change to a healthy diet that they may end up with low blood sugar from their insulin or other antidiabetes...
Thin Diabetes, Fat Diabetes

Laurie Endicott Thomas

Patients with thin diabetes. After all, even people with thin diabetes can get fat diabetes if they eat too much fat and gain too much weight. The goal in managing any illness should be to restore the patient’s health and allow the patient to live a long and happy life. The best way for doctors to achieve that goal would be to teach their patients to eat the kind of diet that promotes a healthy metabolism. The diet that cures fat diabetes is also good for people with thin diabetes. It also eliminates the risk of heart attack. It also shows great promise for reducing the risk of cancer, autoimmune disease, and senile dementia. It accomplishes these goals by correcting the metabolism.

Summary

- Glucose can get into many kinds of cells even if there is no insulin in the bloodstream.
- Insulin is the hormone that helps your body deal with a meal. Insulin encourages your cells to use or store the incoming nutrients. As a result, insulin causes your blood sugar levels to go down. Insulin also suppresses the release of glucagon, which is the hormone that tells your body to use its stored nutrients.
- Glucagon is the hormone that helps you survive a fast. Glucagon tells your body to use its stored nutrients if you haven’t eaten for a while. Glucagon tells your liver to make glucose out of a starch called glycogen, as well as out of some noncarbohydrate substances, such as some amino acids. Thus, your body can make sugar out of protein.
- Diabetes does not result from eating too much sugar or starch. Thin diabetes results when the immune system destroys the insulin-producing cells of the pancreas. Fat diabetes results when the body becomes resistant to the effects of insulin, in an attempt to avoid further weight gain.
Quick, Easy, and Cheap Recipes for Losing Weight and Fighting Diabetes

To cure your type 2 diabetes, you don’t need a drug. You just need better recipes. Those same foods are good for people with type 1 diabetes. Look for recipes that contain no animal products (no meat, milk, cream, cheese, eggs, or fish) and no oils (no butter, ghee, oil, shortening, or lard). Good nonstick cookware will help you cook without oil.

In this chapter, I’ll focus on some of the basic elements of low-fat, plant-based cooking, especially how to cook grains, starchy vegetables, and beans and how to cook vegetables without oil. These instructions will help you develop your own recipes. I’ll also include some useful sauces and dips, as well as some tasty desserts.

Breakfast

Porridge for One

• ½ c rolled oats (or other rolled whole grain) or raw buckwheat
• ½ c water
• Dash of salt, if desired

1. Place the grain and water in a bowl. Cover and leave in the refrigerator overnight. In the morning, you can eat the porridge cold or heat it up in the microwave. Serve plain or with reduced-fat non-dairy milk or soy yogurt.
Optional: Add some fresh fruit (e.g., strawberries, sliced bananas, or grated apple) or dried fruit (raisins or currants). You could also add a bit of brown sugar or maple syrup. I like oatmeal with raisins and a bit of cinnamon. A spoonful of flaxseed or a couple of walnuts are tasty and provide omega-3 fatty acids.

Beans on Toast for One
- 1 small can of nonfat vegetarian baked beans
- 2 slices of bread
1. Heat up the baked beans in the microwave or on the stovetop. Toast the bread. Put the toast on a plate and pour the hot beans over the toast.

*You could also cook your own beans and freeze individual portions, which you can reheat in the microwave.

Breakfast Burritos
- Big, ready-made flour tortillas
- Your choice of fillings:
  » Lettuce
  » Tomatoes
  » Cooked rice
  » Canned nonfat, vegetarian refried beans
  » Cooked potato
  » Salsa
1. Warm a tortilla slightly in a hot skillet (about 10 seconds on each side) or in a microwave. You want to make it more pliable, not crispy. You may also want to heat the refried beans in the microwave.

2. Place a small amount of filling (perhaps a quarter of the tortilla, at most!). Wrap as shown:

*You can also use ready-made corn tortillas, which are gluten-free but are generally smaller.

Banana Pancakes
Here’s a recipe from Mary McDougall. A nurse and cofounder of the McDougall Program, Mary McDougall is a truly great cook and the author of bestselling cookbooks.

- ¾ c whole wheat pastry flour
- ¾ c unbleached white flour
- 2 t baking powder
- 1 c mashed ripe bananas (about 2½ bananas)
- 1 T vegan egg replacer mixed in ¼ cup cold water
- 1 T Wonderslim Fat and Egg Substitute*
- 1 c soy or rice milk

1. Mix the flours and baking powder together in a bowl. Place the bananas in another bowl and mash well. Mix the egg replacer and water and beat until frothy. Add to bananas and mix well. Stir in the Wonderslim fat replacer and the soy or rice milk and mix again. Pour into the dry ingredients and stir to mix. Do not over-beat.

2. Heat a non-stick griddle over medium heat. Pour mixture by ¼ cup measure onto the dry, heated griddle and flatten with the
No-Huevos Rancheros

This is Mary McDougall’s recipe. It is great for when you have guests. Mary says, “The idea for this recipe came from the Mexican breakfast of scrambled eggs over tortillas and beans, topped with salsa. The scrambled tofu topping could also be rolled up in a burrito shell with salsa, or just eaten plain. This is fairly quick to put together if you have leftover pinto beans in your refrigerator, as I usually do.”

- 1 c salsa
- 2 c mashed pinto beans
- 8–10 soft corn tortillas

Tofu Scramble:

- 1 lb firm, water-packed tofu (not silken)
- ¼ c vegetable broth
- ½ c chopped green onions
- 1 T chopped green chilies (optional)
- 1 t soy sauce
- ¼ t turmeric
- Freshly ground pepper
- Dash sea salt (optional)

1. Drain tofu well, mash finely with a bean masher and set aside. Heat the mashed pinto beans in a saucepan. Place the vegetable broth in a large non-stick frying pan, add the green onions and cook, stirring frequently for 3 minutes until softened. Add tofu and the remaining ingredients. Mix well and continue to cook, stirring frequently for 5 more minutes. Set aside.

To Assemble:

2. Heat the tortillas briefly on a dry non-stick griddle to warm and soften them. Take one tortilla and place on a plate. Spread beans on one side, cover with a second tortilla, and spread beans over the top of that tortilla also. Spoon some of the tofu scramble over the tortillas and beans, and then top with several spoonfuls of salsa. Repeat process for each serving.
Hints: Other toppings could also be added, such as shredded soy or rice cheese and/or tofu sour cream. Sprinkle with some fresh chopped cilantro, if desired.

Rice

I’m allergic to wheat, so I eat a lot of rice. I cook a big pot of rice every few days. In the summertime, I cook it outdoors in my solar oven, which never burns anything, never boils over, and does not heat up my kitchen. A friend of mine loves her automatic rice cooker, which was made in Japan. It has specific settings for different kinds of rice and other grains.

There are many different kinds of rice, which vary in flavor and texture. Some of the sticky varieties are popular in China and Japan, where people eat with chopsticks. Varieties whose grains tend to stay separate after cooking are more popular in other places, such as India and the Middle East. Try many different kinds of rice, to find ones you like.

Rice is the seed of the rice plant. Brown rice contains the bran that surrounds the seed and the germ that represents the embryo of the rice plant. White rice is white because the bran and the germ of the seed have been rubbed off. Brown rice is more nutritious and has more fiber than white rice. However, it doesn’t keep as well. If the tiny bit of oil in the germ has gone rancid, the rice will smell and taste bad. Smell brown rice before you cook it. If it smells bad, throw it out. Brown rice also takes a bit longer to cook. However, you can solve that problem by cooking it in a pressure cooker.

Many people in Asia have a strong preference for white rice. In Asia, brown rice is regarded as peasant food. Although brown rice is better for you, hundreds of millions of people manage to stay slim and healthy on a diet based heavily on white rice. As I explained in chapter 4, Dr. Walter Kempner’s Rice Diet was originally based on Uncle Ben’s Converted Rice, which is a brand of parboiled white rice. So if your family hates brown rice, just serve them white rice and some extra vegetables.

Plain Boiled Rice

- 2 c water
- 1 c rice (white or brown)

1. Pour the water into a heavy pot with a tight-fitting lid. Bring the water to a full, rolling boil. Stir in the rice. Bring the water to a full boil again, then cover the pot and reduce the heat to low for 15 minutes (white rice) or 35 minutes (brown rice).

Makes about 3 cups of rice.

Absorption Method of Cooking White Rice

If you don’t like your rice to be sticky, use this method. It makes about 3 cups per cup of uncooked rice. Note that by rinsing enriched rice, you lose a lot of the vitamins.

1. Measure out 1 c of rice and put it into a deep bowl. Put the bowl in the sink and add cold tap water. Swish the rice around with your fingers until the water gets cloudy. Gently pour out the water. Repeat until the water is mostly clear (about 4 or 5 washes). Then leave the rice soaking in water for about 30 minutes. At that point, the rice grains will be milky white. Drain as much water as possible out of the rice and then put it into a heavy pot with a tight-fitting lid. Then, add 1.5 cups of water. (Normally, you would use 2 c of water.)
**adult-onset diabetes** — an obsolete term for fat diabetes. Because of the epidemic of obesity in children, we are now seeing more cases of “adult-onset” than “juvenile” diabetes among children.

**alpha cells** — the glucagon-producing cells in the islets of Langerhans in the pancreas.

**Alzheimer’s disease** — a disease that causes senile dementia. At autopsy, people with Alzheimer’s disease are found to have specific defects in their brain tissue (plaques and tangles).

**amino acid** — any of a group of small organic molecules that have an amine group (\(\text{NH}_2\)), a carboxylic acid group (\(-\text{COOH}\)), and a side chain that gives the amino acid its specific properties. The genes of animals, plants, and fungi contain recipes that specify the exact order of amino acids that are used to make a particular protein. The DNA code can call for any of 20 different amino acids to make a protein that can be thousands of amino acids in length.

**autoimmune disease** — a disease in which the body is attacked by its own immune system.

**beriberi** — a disease that results from deficiency of thiamine (vitamin \(B_1\)), usually from eating a diet that is based too heavily on unenriched white rice.
**diabetes mellitus**— any of several conditions that cause abnormal amounts of a sugar called glucose to build up in the blood. Diabetes mellitus is usually just called diabetes.

**endocrine gland**— any gland that releases hormones into the bloodstream.

**endocytosis**— the process in which a cell “swallows” part of its cell membrane.

**exchange system**— an approach to meal planning for diabetics, developed by the American Diabetes Association and the U.S. Public Health Service in 1950. Unfortunately, it includes many foods that should not be eaten by diabetics.

**exocrine gland**— any gland, such as an oil gland or a sweat gland, that releases its secretions through a duct.

**exocytosis**— the process in which an internal storage compartment of a cell fuses with the outer cell membrane. This process can bring GLUT4 transporters to the surface of the cell.

**fat diabetes**— French-speaking people refer to type 2 diabetes as *diabète gras*, or fat diabetes. Type 2 diabetes is a relatively mild, reversible form of diabetes that is a defense mechanism against further weight gain on a fattening, fatty diet.

**fatty acid**— a molecule consisting of a carboxylic acid group at the head and a tail made out of a chain of carbon atoms, studded with hydrogen atoms. In food and in the body, fatty acids are often bound up with glycerol to form triglycerides.

**fiber, dietary**— in nutrition, dietary fiber refers to the carbohydrates that cannot be broken down by human digestive enzymes. As a

**beta cells**— the insulin-producing cells in the islets of Langerhans in the pancreas.

**beta-casein**— a protein in cow’s milk. It could be the trigger for type 1 diabetes.

**carbohydrate**— a class of chemicals that contain carbon, oxygen, and hydrogen. They are based on simple sugars, which contain the equivalent of one water molecule (H₂O) for each atom of carbon. For example, the chemical formula for both glucose and fructose is C₆H₁₂O₆. Complex carbohydrates consist of pairs or chains of simple sugars.

**chylomicron**— a lipoprotein (combination of fat and protein) that is assembled by the cells that line the intestine and passed into the lymphatic system, which then transports it to a vein under the left collarbone, bypassing the liver. Chylomicrons tend to contain the longer-chain fatty acids.

**corticosteroids**— steroid hormones made by the cortex of the adrenal glands. They tend to have anti-inflammatory effects, and some of them (glucocorticoids) raise blood sugar levels.

**diabète gras**— the French term for type 2 diabetes. It literally means fat diabetes.

**diabète maigre**— the French term for type 1 diabetes. It literally means thin diabetes.

**diabetes insipidus**— a disease in which the body produces huge amounts of dilute urine. Diabetes insipidus can result from drinking too much water, as a result of damage to the thirst center in the brain, or from problems with retaining water, because of some problem related to antidiuretic hormone.
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In *Thin Diabetes, Fat Diabetes*, Laurie Endicott Thomas provides the simple yet powerful lessons about nutrition and medicine that she has learned through working as an editor in medical, veterinary, and academic publishing for more than 25 years. Thomas has a master’s degree from the University of Pennsylvania, and she is certified by the Board of Editors in the Life Sciences. She also writes a grammar column for the *American Medical Writers Association Journal*. Her research for those columns gave rise to her first book, *Not Trivial: How Studying the Traditional Liberal Arts Can Set You Free*. The title is an allusion to the classical trivium of grammar, logic, and rhetoric. These studies were originally called liberal arts because they were considered appropriate for freeborn men, as opposed to slaves.

**She also maintains the following Web sites:**

- [www.nottrivialbook.com](http://www.nottrivialbook.com)
- [www.thindiabetes.com](http://www.thindiabetes.com)
- [www.gorillaprotein.com](http://www.gorillaprotein.com)
- [www.nomeasles.com](http://www.nomeasles.com)
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