

DR. BERNSTEIN'S

DIABETES SOLUTION

A COMPLETE GUIDE TO ACHIEVING NORMAL BLOOD SUGARS

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Chapter 1: Diabetes: The Basics

Diabetes is so common in this country that it touches nearly everyone's life—or will. The statistics on diabetes are staggering, and a diagnosis can be frightening: diabetes is the third leading cause of death in the United States. According to the National Institutes of Health (NIH), there are 8 million diagnosed diabetics in America, and equally that many who have not yet been diagnosed. About 700,000 new diabetics will be diagnosed this year, according to NIH statistics; that's one every 30 seconds. Each year, tens of thousands of Americans lose their eyesight because of diabetes, the leading cause of blindness for people in the 25–74 age range. Ninety-five percent of diabetics have Type II, or what used to be known as maturity-onset, diabetes. Because 80 percent of Type II diabetics are obese, many inappropriately feel that the disease is their own fault, the result of some failure of character.

Since you are coming to this book, you or a loved one may have been diagnosed recently with diabetes. Perhaps you have long-standing diabetes and are not satisfied with treatment that has left you plagued with complications such as encroaching blindness, foot pain, frozen shoulder, inability to achieve or maintain an erection, or heart or kidney disease.

Although diabetes is still an incurable, chronic disease, it is *very* treatable, and the long-term "complications" are fully preventable. I've had Type I diabetes, also called juvenile-onset or insulin-dependent diabetes mellitus (IDDM), for more than fifty years. This form of diabetes is generally far more serious than Type II, or non-insulin-dependent diabetes mellitus (NIDDM), although both have the potential to be fatal.* Most Type I diabetics who were diagnosed back about the same time I was are now dead from one or more of the serious complications of the disease. Yet, after living with diabetes for more than fifty years, instead of being bedridden or out sick from work, I am more fit than many nondiabetics who are considerably younger than I. I regularly work 12-hour days, travel, sail, and pursue a vigorous exercise routine. If I can take control of my disease, you can take control of yours.

In the next several pages I'll give you a general overview of diabetes, how the body's system for controlling blood sugar (glucose) levels works in the nondiabetic, and how it works—and doesn't work—for diabetics. In subsequent chapters we'll discuss diet, exercise, and medication, and

how you can use them to control your diabetes. If talk about diet and exercise sounds like "the same old thing" you've heard again and again, read on, because you'll find that what I've observed is almost exactly the opposite of what you've probably been taught. The tricks you'll learn can help you arrest the diabetic complications you may now be suffering, may reverse many of them, and should prevent the onset of new ones. We'll also talk about new medical treatments and drugs that are now available to help manage blood sugar levels and curtail obesity.

The Body In and Out of Balance

Diabetes is the breakdown or partial breakdown of one of the more important of the body's autonomic (self-regulating) mechanisms, and its breakdown throws many other self-regulating systems into imbalance. There is probably not a tissue in the body that escapes the effects of the high blood sugars of diabetes. People with high blood sugars tend to have osteoporosis, or fragile bones; they tend to have tight skin; they tend to have inflammation and tightness at their joints; they tend to have many other complications that affect every part of their body.

Insulin: What It Is, What It Does

At the center of diabetes is the pancreas, a large gland about the size of your hand, which is located toward the back of the abdominal cavity and is responsible for manufacturing, storing, and releasing the hormone insulin. The pancreas also makes several other hormones, as well as digestive enzymes. Even if you don't know much about diabetes, in all likelihood you've heard of insulin and probably know that we all have to have insulin to survive. What you might not realize is that only a small percentage of diabetics must have insulin shots.

Insulin is a hormone produced by the beta cells of the pancreas. Its major function is to regulate the level of glucose in the bloodstream, which it does primarily by facilitating the transport of blood glucose into most of the billions of cells that make up the body. Insulin also stimulates centers in the brain responsible for feeding behavior, and it instructs fat cells to convert glucose and fatty acids in the blood into fat, which the fat cells then store until needed. Insulin is essential for the growth of many tissues and organs. In excess, it can cause excessive growth—as, for example, of body fat and of cells that line blood vessels. Finally, insulin helps to regulate, or counterregulate, the balance of certain other hormones in the body. More about those later.

One of the ways insulin maintains the narrow range of normal levels of sugar in the blood is by regulation of the liver and muscles, directing them to manufacture and store glycogen, a starchy substance the body uses when blood sugar falls too low. If blood sugar does fall too low—as may occur after strenuous exercise or fasting—the alpha cells of the pancreas release glucagon, another hormone involved in the regulation of blood sugar levels. Glucagon signals the muscles and liver to convert their stored glycogen back into glucose (a process called glycogenolysis), which raises blood sugar. When the body's stores of glucose and glycogen have been exhausted, the liver can transform the body's protein stores—muscle mass and vital organs—into sugar.

Insulin and Type I Diabetes

As recently as seventy-five years ago, before the clinical availability of insulin, the diagnosis of Type I diabetes—which involves a severely diminished capacity to produce insulin—was a death sentence. Most people died within a few months of diagnosis. Without insulin, glucose accumulates in the blood to extremely high toxic levels; yet, since it cannot be utilized by the

cells, many cell types will starve. The absence of insulin also leads the liver to perform gluconeogenesis, turning the body's protein store—the muscles and vital organs—into even more glucose that the body cannot utilize. Meanwhile, the kidneys, the filters of the blood, try to rid the body of inappropriately high levels of sugar. Frequent urination causes insatiable thirst and dehydration. Eventually, the starving body turns more and more protein to sugar, leaving no organ unaffected. The ancient Greeks described diabetes as a disease that causes the body to melt into sugar water. When tissues cannot utilize glucose, they will metabolize fat for energy, generating by-products called ketones, which are toxic at high levels and cause further water loss as the kidneys try to eliminate them (see ketoacidosis, in Chapter 20, "How to Cope With Dehydrating Illness").

Today Type I diabetes is still a very serious disease, and still eventually fatal if not properly treated with insulin. It can kill you rapidly when your blood glucose level is too low—through impaired judgment or loss of consciousness while driving, for example—or it can kill you slowly, by heart or kidney disease, which are commonly associated with long-term blood sugar elevation. Until I brought my blood sugars under control, I had numerous automobile accidents due to hypoglycemia, and it's only through sheer luck that I'm here to talk about it. The causes of Type I diabetes have not yet been fully unraveled. Research indicates that it's an autoimmune disorder in which the body's immune system attacks the pancreatic beta cells that produce insulin. Whatever causes Type I diabetes, its deleterious effects can absolutely be prevented. The earlier it's diagnosed, and the earlier blood sugars are normalized, the better off you will be.

At the time they are diagnosed, many Type I diabetics still produce a small amount of insulin, and if they are treated early enough and treated properly, what's left of their insulin-producing capability frequently can be preserved. Type I diabetes typically occurs before the age of forty-five and usually makes itself apparent quite suddenly, with such symptoms as dramatic weight loss and frequent thirst and urination. We now know, however, that as sudden as its appearance may be, its onset is actually quite slow. Routine commercial laboratory studies are available that can detect it earlier, and it may be possible to arrest it in these early stages by aggressive treatment. My own body no longer produces any insulin at all. The high blood sugars I experienced during my first year with diabetes burned out, or exhausted, the ability of my pancreas to produce insulin. I must have insulin shots or I will rapidly die. I firmly believe that if the kind of diet and medical regimen I prescribe for my patients had been available when I was diagnosed, the insulin-producing capability left to me at diagnosis would have been preserved. My requirements for injected insulin would have been lessened, and it would have been much easier for me to keep my blood sugars normal.

Blood Sugar Normalization: Restoring the Balance

According to the American Diabetes Association (ADA), more than 150,000 people die annually from both Type I and Type II diabetes and their long-term complications. Certainly everyone has to die of something, but you needn't die the slow, torturous death of diabetic complications, which often include blindness and amputations. My history and that of my patients supports this. The recently completed Diabetes Control and Complication Trial (DCCT) began as a ten-year study of Type I diabetics to gauge the effects of improved control of blood sugar levels (see the foreword, by Dr. Frank Vinicor). Patients whose blood sugars were nearly "normalized" (my

patients' blood sugars are usually closer to normal than were those in the trial) had dramatic reductions of long-term complications. Researchers began the DCCT trying to see if they could, for example, lessen the frequency of diabetic retinopathy by at least 33.5 percent. Instead of a one-third reduction in retinopathy, they found a more than 75 percent reduction in the progression of early retinopathy. They found similarly dramatic results in other diabetic complications and halted the study early in order to make the results available to all. They found a 50 percent reduction of risk for kidney disease, a 60 percent reduction of risk for nerve damage, and a 35 percent reduction of risk for cardiovascular disease.

The patients followed in the DCCT averaged twenty-seven years of age at the beginning of the trial, so reductions could easily have been greater in areas such as cardiovascular disease if they had been older or followed for a longer period of time. The implication is that full normalization of blood sugar could totally prevent these complications. In any case, the results of the DCCT are good reason to begin aggressively to monitor and normalize blood sugar levels. The effort and dollar cost of doing so does not have to be so high as was suggested in the DCCT's findings.

The Insulin-Resistant Diabetic: Type II

Different from Type I diabetes is what is commonly known as Type II. This is by far the more prevalent form of the disease. According to ADA statistics, 90–95 percent of diabetics are Type II. Furthermore, as many as a quarter of Americans between the ages of sixty-five and seventy-four have Type II.

Approximately 80 percent of those with Type II diabetes are overweight and suffer from a particular form of obesity known as truncal, or visceral, obesity. It is quite possible that the 20 percent of the so-called Type II diabetics who do not have visceral obesity actually suffer from a form of Type I diabetes that causes only partial loss of the pancreatic beta cells that produce insulin. If this proves to be the case, then fully all of those who have Type II diabetes may be overweight. (Obesity is usually defined as being at least 20 percent over the ideal body weight for one's height, build, and sex.)

While the cause of Type I diabetes may still be somewhat mysterious, the cause of Type II is less so. As noted earlier, another name for Type II diabetes is insulin-resistant diabetes. Obesity, particularly visceral obesity, and insulin resistance—the inability to fully utilize the glucose-transporting qualities of insulin—are interlinked. For reasons related to genetics (see Chapter 12, "Weight Loss"), a substantial portion of the population has the potential when overweight to become sufficiently insulin-resistant that the increased demands on the pancreas burn out the beta cells that produce insulin. These people enter the vicious circle depicted in Figure 1-1. Note in the figure the crucial role of dietary carbohydrate in the development and progression of this disease. This is discussed in detail in Chapter 12.

Insulin resistance appears to be caused at least in part by inheritance and in part by high levels of fat—in the form of triglycerides—in the branch of the bloodstream that feeds the liver. (Transient insulin resistance can be created in laboratory animals by injecting triglycerides directly into their liver's blood supply.) Insulin resistance by its very nature increases the body's needs for insulin, which therefore causes the pancreas to work harder to produce elevated insulin levels (hyperinsulinemia), which can indirectly cause high blood pressure and damage the

circulatory system. So, to simplify somewhat, fat in the blood feeding the liver causes insulin resistance, which causes elevated serum insulin levels, which cause the fat cells to build even more abdominal fat, which raises triglycerides in the liver's blood supply, which causes insulin levels to increase because of increased resistance to insulin.

If that sounds circular, it is. The fat that is the culprit here is not dietary fat. High levels of triglycerides in the blood—which are in circulation at some level in the bloodstream at all times—are not so much the result of intake of dietary fat as they are of carbohydrate consumption and existing body fat. (We will discuss carbohydrates, fat, and insulin resistance more in Chapter 9, "The Basic Food Groups.") The culprit is rather a particular kind of body fat. Visceral obesity is a type of obesity in which fat is concentrated around the middle of the body, particularly surrounding the intestines (the viscera). A man who is viscerally obese has a waist of greater circumference than his hips. A woman who is viscerally obese will have a waist at least 80 percent as big around as her hips. All individuals with visceral obesity are insulin-resistant. The ones who eventually become diabetic are those who cannot make enough extra insulin to keep their blood sugars normal.

Though treatment has many similar elements—and many of the effects are the same—Type II diabetes differs from Type I in several important ways.

The onset of Type II diabetes is slower and more stealthy, but even in its earliest stages, the abnormal blood sugar levels, though not sky-high, can cause damage to nerves, blood vessels, heart, eyes, and more. Type II diabetes is often called the silent killer, and it is quite frequently discovered through one of its complications, such as hypertension or a defect in vision.* Type II diabetes is, at the beginning, a less serious disease—patients don't melt away into sugar water and die in a few months' time. Type II, however, can through chronically but less dramatically elevated blood sugars be much more insidious. It probably causes more heart attacks, strokes, and amputations than the more serious Type I disease. Type II is a major cause of hypertension, heart disease, blindness, and amputation due to impaired sensation (nerve damage) in feet with poor circulation that do not heal when injured. That these serious complications of Type II diabetes can proliferate is no doubt because it is initially milder and is often left untreated or treated poorly.

Individuals with Type II still make insulin, and most will never require injected insulin to survive, though if the disease is treated poorly, they can eventually burn out their pancreatic beta cells and require insulin shots. Because of their resistance to the blood sugar-lowering effects of insulin (though not its fat-building effects), many obese Type II diabetics actually make more insulin than nondiabetics.

Since high blood sugar is the hallmark of diabetes, and the cause of every long-term complication of the disease, it makes sense to discuss where blood sugar comes from and how it is used and not used.

Blood Sugars: The Nondiabetic Versus the Diabetic

Our dietary sources of blood sugar are carbohydrates and proteins. One reason the taste of sugar—a simple form of carbohydrate—delights us is that it fosters production of neurotransmitters in the brain that relieve anxiety and can create a sense of well-being, or even

euphoria. This makes carbohydrate quite addictive to certain people whose brains may have inadequate levels of these neurotransmitters, the chemical messengers with which the brain communicates with itself and the rest of the body, or peripheral nervous system. When blood sugar levels are low, the liver can, through a process we will discuss shortly, convert proteins into glucose, but very slowly and inefficiently. The body cannot convert glucose back into protein, nor can it convert fat into sugar. Fat cells, however, with the help of insulin, do transform glucose into fat.

The taste of protein doesn't excite us as much as that of carbohydrate—it would be the very unusual child who'd jump up and down in the grocery store and beg his mother for a steak instead of cookies. Protein gives us a much slower and smaller blood sugar effect, which, as you will see, we diabetics can use to our advantage in normalizing blood sugars.

The Nondiabetic

In the fasting nondiabetic, and even in some Type II diabetics, the pancreas constantly releases a steady, low level of insulin. This baseline, or basal, insulin level prevents the liver from inappropriately converting bodily proteins (muscle, vital organs) into glucose and thereby raising blood sugar, a process known as gluconeogenesis. The nondiabetic ordinarily maintains blood sugar immaculately within a narrow range—usually between 80 and 100 mg/dl (milligrams per deciliter),* with most people hovering near 85 mg/dl.* There are times when that range can briefly stretch up or down—as high as 160mg/dl and as low as 65—but generally, for the nondiabetic, such swings are rare.

You will note that in some literature on diabetes, "normal" may be defined as 60–120 mg/dl, or even as high as 140 mg/dl. This "normal" is entirely relative. No nondiabetic will have blood sugar levels as high as 140 mg/dl except after consuming a lot of carbohydrate. "Normal" in this case has more to do with what is cost-effective for the average physician to treat. Since a postmeal (postprandial) blood sugar under 140 mg/dl is not classified as diabetes, and since the individual who experiences such a value will usually still have adequate insulin production eventually to bring it down to reasonable levels, many physicians would see no reason for treatment. Such an individual will be sent off with the admonition to watch his weight or her sugar intake. Despite the designation "normal," an individual frequently displaying a blood sugar level of 140 mg/dl is a good candidate for full-blown Type II diabetes. I have seen "nondiabetics" with sustained blood sugars averaging 120 mg/dl develop diabetic complications.

Let's take a look at how the average nondiabetic body makes and uses insulin. Suppose that Jane, a nondiabetic, arises in the morning and has a mixed breakfast, that is, one that contains both carbohydrate and protein. On the carbohydrate side, she has toast with jelly and a glass of orange juice; on the protein side, she has a boiled egg. Her basal (i.e., before-meals) insulin secretion has kept her blood sugar level steady during the night, inhibiting gluconeogenesis. Shortly after the sugar in the juice or jelly hits her mouth, or the starchy carbohydrates in the toast reach her saliva, glucose begins to enter her bloodstream. The rise in Jane's blood sugar is a chemical signal to her pancreas to release the granules of insulin it has stored in order to prevent a jump in blood sugar (see Figure 1-2). This rapid release of stored insulin is called phase I insulin response. It quickly corrects the initial blood sugar increase and can prevent further increase from the ingested carbohydrate. As the pancreas runs out of stored insulin, it manufactures more,

but it has to do so from scratch. The insulin released now is known as the phase II insulin response, and it's secreted much more slowly. As she eats her boiled egg, the insulin of phase II can cover the sugar that's slowly produced from the protein of the egg.

Insulin acts in the nondiabetic as the means to admit glucose—fuel—into the cells. It does this by activating the production of glucose "transporters" within the cells. These specialized protein molecules emerge from the nuclei of the cells to grab glucose from the blood and bring it to the interiors of the cells. Once inside the cell, glucose can be utilized to power energy-requiring functions. Without insulin, the cells can absorb only a very small amount of sugar, not enough to sustain the body.

As Jane's blood continues to accumulate sugar, and the beta cells in her pancreas continue to release insulin, some of her blood sugar is transformed to glycogen, a starchy substance stored in the muscles and liver. Once glycogen storage sites in the muscles and liver are filled, excess glucose remaining in the bloodstream is converted to and stored as fat. Later, as lunchtime nears but before Jane eats, if her blood sugar drops too low, the alpha cells of her pancreas will release another pancreatic hormone, glucagon, which will "instruct" her liver and muscles to begin converting glycogen to glucose, to raise blood sugar. When she eats again, her store of glycogen will be replenished.

This pattern of basal, phase I, then phase II insulin secretion is perfect for keeping Jane's blood glucose levels in a healthy range. Her body is nourished, and things work according to design. Her mixed meal is handled beautifully. This is not, however, how things work for either the Type I or Type II diabetic.

The Type I Diabetic

Let's look at what would happen to me, a Type I diabetic, if I had the same breakfast as Jane, our nondiabetic.

Unlike Jane, because of a condition peculiar to diabetics, if I take insulin, I might awaken with normal blood sugar levels, but if I spend some time awake before breakfast, my blood sugar may rise, even if I haven't had anything to eat. Ordinarily, the liver is constantly removing some insulin from the bloodstream, but during the first few hours after waking from a full night's sleep, it clears insulin out of the blood at an accelerated rate. This dip in insulin level is called the dawn phenomenon (see Chapter 6, "Strange Biology"). Because of it, my blood glucose can rise even though I haven't eaten. A nondiabetic just makes more insulin to take care of the increased clearance. Those of us who are severely diabetic have to track the dawn phenomenon carefully by monitoring blood glucose levels, and can learn how to prevent its effect upon blood sugar. As with Jane, the minute the meal hits my mouth, the enzymes in my saliva begin to break down the sugars in the toast and juice, and almost immediately my blood sugar begins to rise. Even if the toast had no jelly, the enzymes in my saliva and stomach would begin to rapidly transform the toast into glucose upon ingestion.

Since my beta cells have completely ceased functioning, there is no stored insulin to be released by my pancreas, so I have no phase I insulin response. My blood sugar (in the absence of injected insulin) will rise while I digest my meal. None of the glucose will be converted to fat, nor will any be converted to glycogen. Eventually much will be filtered out by the kidneys and

passed out through the urine, but not before my body has endured damagingly high blood sugar levels—which won't kill me on the spot but will over the years be an incremental step in the slow, "silent" death from diabetic complications. The natural question is, wouldn't injected insulin "cover" the carbohydrate in such a breakfast? No. This is a common misconception—even by those in the health care profession. Normal phase I insulin is almost instantly in the bloodstream. Rapidly it begins to hustle blood sugar off to where it's needed. Injected insulin, on the other hand, is injected either into fat or muscle (not into a vein) and absorbed slowly. The fastest insulin we have, lispro, starts to work in about 15 minutes, but that isn't fast enough to prevent a damaging upswing in blood sugars if fast-acting carbohydrate, like bread, is consumed. This is the central problem for Type I diabetics—the carbohydrate and the drastic surge it causes in blood sugar. Because I know my body produces no insulin, I have a shot of insulin before every meal. But I no longer eat meals with fast-acting or large amounts of carbohydrate, because the blood sugar swings they caused were what brought about my complications. Even injection by means of an insulin pump (see discussion at the end of Chapter 18) cannot fine-tune the level of glucose in my blood the way a nondiabetic's body does naturally.

Now, if I ate only the protein portion of the meal, my blood sugar wouldn't have the huge, and potentially toxic, surge that carbohydrates cause. It would rise less rapidly, and a smaller dose of insulin could act rapidly enough to cover the glucose that's slowly derived from the protein. My body would not have to endure wide swings in blood sugar levels. (Dietary fat, by the way, has no effect on blood sugar levels, except that it can slightly slow the digestion of carbohydrate.) In a sense, you could look at my insulin shot before eating only the protein portion of the meal as mimicking the nondiabetic's phase II response. This is much easier to accomplish than trying to mimic phase I, because of the much lower levels of dietary carbohydrate and injected insulin.

The Type II Diabetic

Let's say Jim, a Type II diabetic, is 6 feet tall and weighs 300 pounds, much of which is centered around his midsection. Remember, at least 80 percent of Type II diabetics are obese. If Jim weighed only 150 pounds, he might well be nondiabetic, but because he's insulin-resistant, Jim's body no longer produces enough excess insulin to keep his blood sugar levels normal.

The obese tend to be insulin-resistant as a group, a condition that's not only hereditary but also directly related to the ratio of visceral fat to lean body mass (muscle). The higher this ratio, the more insulin-resistant a person will be. Whether or not an obese individual is diabetic, his weight, intake of carbohydrates, and insulin resistance all tend to make him produce considerably more insulin than a slender person of similar age and height (see Figure 1-3). Many athletes, because of their low fat mass and high percentage of muscle, tend as a group to require and make low levels of insulin. An obese Type II diabetic like Jim, on the other hand, typically makes two to three times as much insulin as the slender nondiabetic. In Jim's case, from many years of having to overcompensate, his pancreas has partially burned out, and despite the huge output of insulin, he no longer can keep his blood sugars within normal ranges. (In my medical practice, a number of patients come to me for treatment of their obesity, not diabetes. However, on examination, most of these very obese "nondiabetics" have slight elevations of their test for average blood sugar.)

Let's take another look at that mixed breakfast and see how it affects a Type II diabetic. Jim has the same toast and jelly and juice and boiled egg that Jane, our nondiabetic, and I had. Jim's blood sugar levels at waking are normal.* Since he has a bigger appetite than either Jane or I, he has two glasses of juice, four pieces of toast, and two eggs. As soon as the toast and juice hit his mouth, his blood sugar level begins to rise. Unlike mine, Jim's pancreas releases insulin, but he has very little or no stored insulin (his pancreas works hard just to keep up his basal insulin level), so he has impaired phase I secretion. His phase II insulin response, however, may be intact. So very slowly, his pancreas will struggle to produce enough insulin to bring his blood sugar down toward the normal range. Eventually it may get there, but not until hours after his meal, and hours after his body has been exposed to high blood sugars. Insulin is not only the major fat-building hormone, it also serves to stimulate the center in the brain responsible for feeding behavior. Thus, in all likelihood, Jim may well grow even more obese, as demonstrated by the cycle illustrated in Figure 1-1.

Since he's resistant to insulin, his body has to work that much harder to metabolize the carbohydrate he consumes. Because of insulin's fat-building properties, his body stores away some of his blood sugar as fat and glycogen; but his blood sugar level continues to rise, since his cells are unable to utilize adequate amounts. Jim, therefore, still feels hungry. As he eats more, his beta cells work harder to produce more insulin. The excess insulin and the "hungry" cells in his brain prompt him to want yet more food. He has just one more piece of toast with a little more jelly on it, hoping that it will be enough to get him through until lunch. Meanwhile, his blood sugar goes even higher, his beta cells work harder, and perhaps some of them burn out. Even after all this food, he still may feel many of the symptoms of hunger. His blood sugar, however, will probably not go as high as mine would if I took no insulin. In addition, his phase II insulin response could even bring his blood sugar down to normal after many hours without more food.

Postprandial blood sugar levels that I would call unacceptably high—140 mg/dl, or even 200 mg/dl—may be considered by other doctors to be unworthy of treatment because the patient still produces adequate insulin to bring them periodically down to normal, or "acceptable," ranges. If Jim, our Type II diabetic, had received intensive medical intervention before the beta cells of his pancreas began to burn out, he would have slimmed down, brought his blood sugars into line, and eased the burden on his pancreas. He might even have "cured" his diabetes by slimming down, as I've seen in several patients. But many doctors might decide such "mildly" abnormal blood sugars are only impaired glucose tolerance (IGT), and do little more than "watch" them. Again, it's my belief that aggressive treatment at an early stage can save most patients considerable lost time and personal agony by preventing complications that will occur if blood sugar levels are left unchecked. Such intervention can make subsequent treatment of what remains—a mild disease—elegantly simple.

On the Horizon

Researchers are currently trying to perfect a method for cloning, or replicating, insulin-producing pancreatic beta cells in the laboratory. Doing this in a fashion that's comparatively easy and cost effective should not be an insurmountable task, and indeed the preliminary results are quite encouraging. Once cells are replicated, they can be transplanted back into patients to actually

cure their diabetes. After such treatment, unless you were to have another autoimmune event that would destroy these new beta cells, you would remain nondiabetic for the rest of your life. If you had another autoimmune attack, you would simply have to transplant more cloned cells. This is the single best opportunity we have for a cure, immeasurably better than all the electronic insulin pumps, and the only one I'd personally have any part of—except I can't.

The catch here for me and other diabetics who no longer have any insulin-producing capacity is that the cells from which new beta cells would be cloned have to be your own, and I have none. Had I gone on insulin, say, a year before I was diagnosed with diabetes, or had my blood sugars been immaculately controlled immediately upon diagnosis, the injected insulin might have taken much of the strain off my remaining beta cells and allowed them to survive.

Many people (including the parents of diabetic children) view having to use insulin as a last straw, a final admission that they are (or their child is) a diabetic and seriously ill. Therefore they will try anything else—including things that will burn out their remaining beta cells—before using insulin. Many people in our culture have the notion that you cannot be well if you are using medication. This is nonsense, but some patients are so convinced that they must do things the "natural" way that I practically have to beg them to use insulin. In reality, nothing could be more natural. Diabetics who still have beta cell function left may well be carrying their own cure around with them—provided they don't burn it out with high blood sugars and the refusal to use insulin.

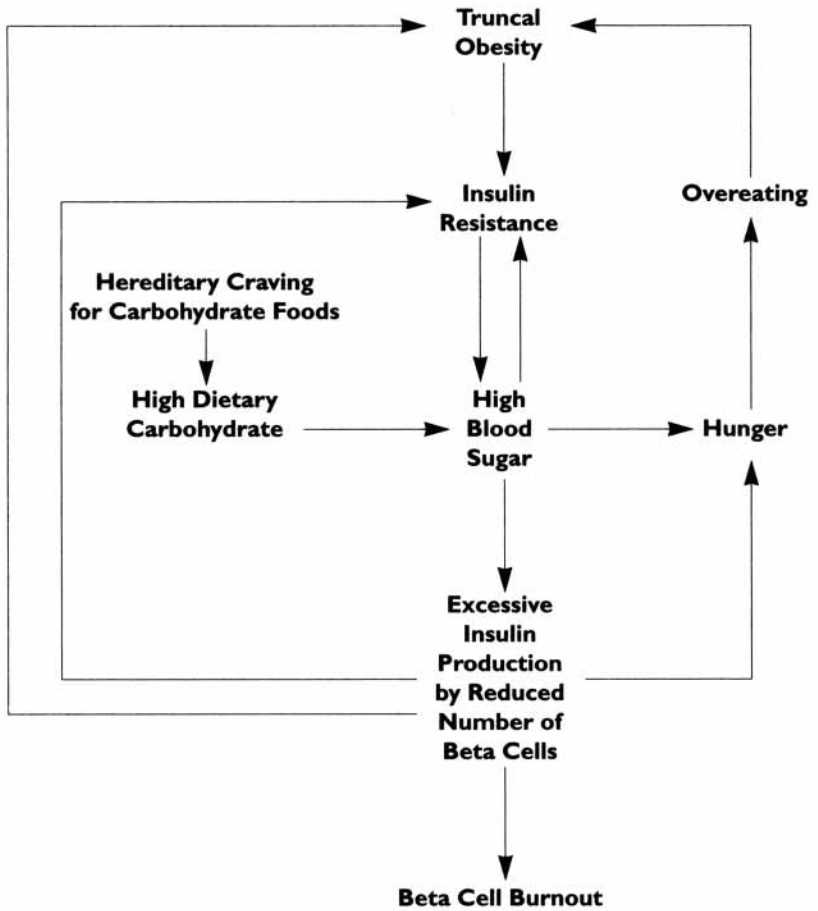
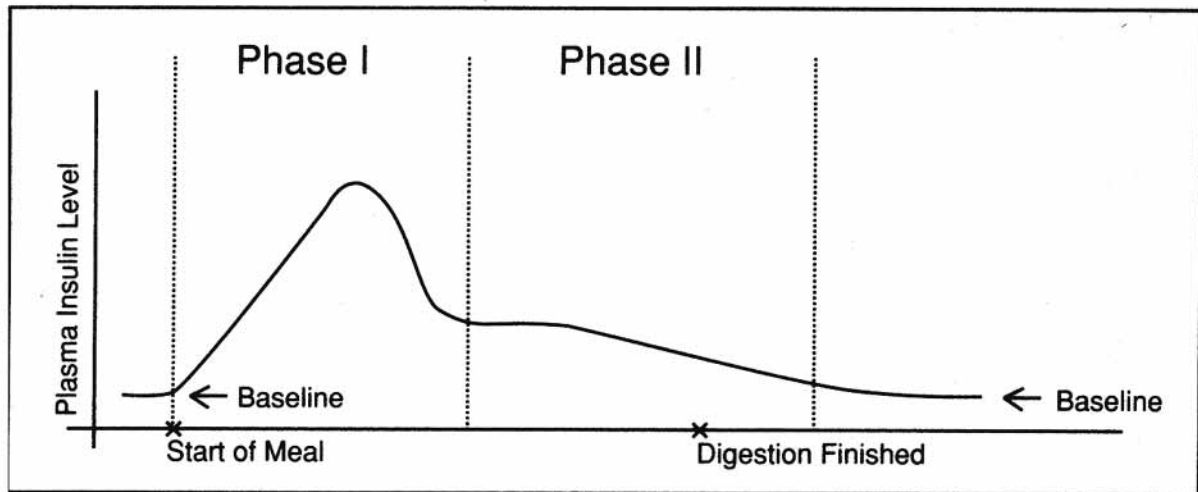


Fig. 1-1. *The vicious circle of insulin resistance.*



Terry Eppridge

Fig. 1-2. *Phase I and phase II insulin response in normal, nondiabetic person.*

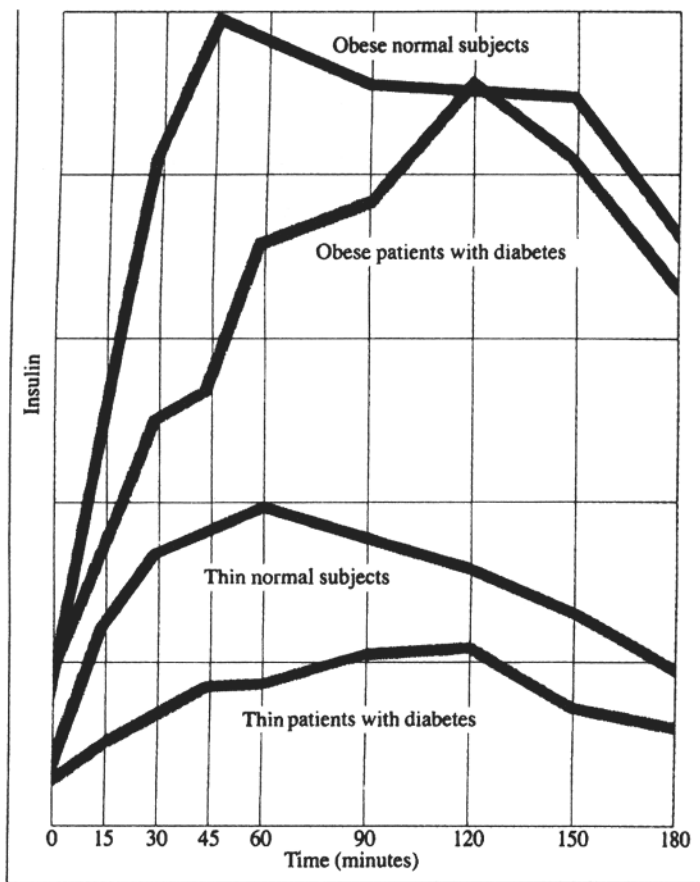


Fig. 1-3. Serum insulin response of individuals with and without diabetes.