

DR. BERNSTEIN'S

DIABETES SOLUTION

A COMPLETE GUIDE TO ACHIEVING NORMAL BLOOD SUGARS

This document and its contents are Copyright 2000 by Richard K. Bernstein, M.D., Little, Brown & Company, and/or other copyright holders as may apply. No portion of this document may be reproduced in whole or in part without the express written consent of Little, Brown & Company and/or Richard K. Bernstein, M.D. and/or any other respective copyright holder(s).

Chapter 12: Weight Loss—If You're Overweight

Weight loss can significantly reduce your insulin resistance. You may recall from Chapter 1 that obesity, specifically abdominal (truncal, or visceral) obesity, causes insulin resistance and thereby can play a major role in the development of both impaired glucose tolerance and Type II diabetes. If you have this kind of obesity, it is important that weight loss become a goal of your treatment plan. Weight reduction can also slow down the process of beta-cell burnout by making your tissues more sensitive to the insulin you still produce, allowing you to require (and therefore produce or inject) less insulin.

It may even be possible, under certain circumstances, to completely reverse your glucose intolerance. Long before I studied medicine, I had a friend, Howie, who gained about 100 pounds over the course of a few years. He developed Type II diabetes and had to take a large amount of insulin (100 units daily) to keep it under control. His physician pointed out to him the likely connection between his diabetes and his obesity. To my amazement, during the following year, he was able to lose 100 pounds. At the end of the year, he had normal glucose tolerance, no need for insulin, and a new wardrobe. This kind of success may only be possible if the diabetes is of short duration, but it is certainly worth keeping in mind—weight loss can sometimes work miracles.

Before we talk about weight loss, it makes sense to talk about obesity.

The Thrifty Genotype

When I see a very overweight person, I don't think, "He ought to control his eating." I think, "He has the thrifty genotype."

What is the thrifty genotype?

The hypothesis for the thrifty genotype was first proposed by James V. Neel in 1962 to explain the high incidence of obesity and Type II diabetes among the Pima Indians of the southwestern United States. Evidence for a genetic determinant of obesity has increased over the years.

Photographs of the Pimas from a century ago show a lean and wiry people. They did not know what obesity was and in fact had no word in their vocabulary to identify it.

Their food supply diminished in the early part of this century, something that had occurred repeatedly throughout their history. Now, however, they weren't faced with famine. The Bureau of Indian Affairs took over feeding them, and an astonishing thing happened. These lean and wiry people developed an astronomical incidence of obesity—100% of adult Pima Indians today are grossly obese, with a staggering rate of diabetes. Fully 60 percent of adults are Type II diabetics.

What happened to the Pimas? How did such apparently hardy and fit people become so grossly obese? Though their society was at least in part agrarian, they lived in the desert, where drought was frequent and harvests could easily fail. During periods of famine, those of their forebears whose bodies were not thrifty or capable of storing enough energy to survive without food died out. Those who survived were those who somehow could survive without food. How did they do it? Although it may be simplifying somewhat, the mechanism essentially works like this: Those who naturally craved carbohydrate and consumed it whenever it was available, even if they weren't hungry, would have made more insulin and thereby stored more fat. Add to this the additional mechanism of the high insulin levels caused by inherited insulin resistance, and serum insulin levels would have become great enough to induce fat storage sufficient to enable them to live through famines. (See Figure 1-1.) Truly survival of the fittest—provided famines would continue.

A strain of chronically obese mice created in the early fifties demonstrates quite vividly how valuable thrifty genes can be in famine. When these mice are allowed an unlimited food supply, they balloon and add as much as half again the body weight of normal mice. Yet deprived of food, these mice can survive a mind-boggling 40 days, versus 7–10 days for normal mice.

Recent research on these chronically obese mice provides some tantalizingly direct evidence of the effect a thrifty genotype can have upon physiology. In ordinary mice, a hormone called leptin is produced in the fat cells (also a hormone the human body produces, with apparently similar effect). The hormone tends to inhibit overeating, speed metabolism, and act as a modulator of level of body fat. A genetic "flaw" causes the obese mice to make a less effective form of leptin. In recent experiments, when injected with the real thing they almost instantly slimmed down. Not only did they eat less but they lost as much as 40 percent of their body weight, their metabolism sped up, and they became much more active. Many were diabetic, but their loss of weight (and the change in the ratio of fat to lean body mass) reversed or even "cured" their diabetes. Ordinary mice injected with leptin also ate less, became more active, and lost weight, though not as high a percentage. Research on humans has not advanced sufficiently to provide conclusive evidence that the mechanism is the same in obese humans, but researchers believe it is at least equivalent and probably related to more than one gene, and to different gene clusters in different populations.

In a full-blown famine, the Pima Indian's ability to survive long enough to find food is nothing short of a blessing. But when satisfying carbohydrate craving is suddenly just a matter of going to the grocery, what was once an asset becomes a very serious liability.

Although about 30 percent of the overall population of the United States is chronically obese,

there is considerable reason to be concerned, because the number has been increasing by 1 percent each year. Some researchers attribute rising obesity in the United States at least in part to increasing numbers of former smokers. Others attribute it to the recent increase in carbohydrate consumption by those trying to avoid dietary fat. Whatever the reasons, obesity can lead to diabetes.

The thrifty genotype has its most dramatic appearance in isolated populations like the Pimas, which have recently been exposed to an unlimited food supply after millennia of intermittent famine. The Fiji Islanders, for example, were another lean, wiry people, accustomed to the rigors of paddling out against the Pacific to fish. Their diet, high in protein and low in carbohydrate, suited them perfectly. After the onset of the tourist economy that followed World War II, their diet changed to our high-carbohydrate western diet, and they too began (and continue) to suffer from a high incidence of obesity and Type II diabetes. The same is true of the Australian Aborigines after the Aboriginal Service began to provide them with grain. Ditto for South African blacks who migrated from the bush into the big cities. Interestingly, a study that paid obese, diabetic South African blacks to go back to the countryside and return to their traditional high-protein, low-carbohydrate diet found that they experienced dramatic weight loss and regression of their diabetes.

It's clear that thrifty genotypes work in isolated populations to make metabolism supremely energy-efficient, but what happens when the populations have unrestricted access to high-carbohydrate foods?

It would appear that the mechanism of the thrifty genotype works something like this: Certain areas of the brain associated with satiety—that sensation of being physically and emotionally satisfied—have lower levels of certain chemicals known as neurotransmitters. A number of years ago, Drs. Richard and Judith Wurtman at the Massachusetts Institute of Technology (MIT) discovered that the level of the neurotransmitter serotonin is raised in certain parts of the hypothalamus of the animal brain when the animal eats carbohydrate, especially fast-acting concentrated carbohydrate like bread. Serotonin is a neurotransmitter that seems to reduce anxiety as it produces satiety. Other neurotransmitters such as dopamine and norepinephrine can also affect our senses of satiety and anxiety, euphoria and dysphoria. There are now seventy-five known neurotransmitters, and many more of them may affect mood and food in ways that are just beginning to be researched and understood.

In persons with the thrifty genotype, deficiencies of these neurotransmitters (or diminished sensitivity to them in the brain) causes both a feeling of hunger and a mild dysphoria—often a sensation of anxiety, and the opposite of euphoria. Eating carbohydrates temporarily causes the individual to feel not only less hungry but also more at ease.

A frequent television sitcom scenario is the woman just dumped by her boyfriend who plops down on the couch with a pie or half a gallon of ice cream, a spoon, and the intention of eating the whole thing. She's not really hungry. She's depressed and trying to make herself feel better. She's indulging herself, we think, rewarding herself in a way for enduring one of life's traumas, and we laugh because we understand the feeling. But there is a very real biochemical mechanism at work here. She craves the sugar in the pie or the ice cream not because she's hungry but

because she knows, consciously or not, that it really will make her feel better. Contrary to popular belief, the fat in the ice cream or in the crust of the pie doesn't make much of a difference. It's the carbohydrate that will increase the level of certain neurotransmitters in her brain and make her feel better temporarily. The side effect of the carbohydrate is that it also causes her blood sugar to rise and her body to make more insulin; and, as she sits on the couch, the elevation in her insulin will turn the sugar she eats into fat.

On television the actress never gets fat. But for the real-life woman, high serum insulin levels from eating high-carbohydrate foods will cause her to crave carbohydrate again. If she is a Type I diabetic making no insulin, she'll have to inject a lot of insulin to get her blood sugar down, with the same effect—more carbohydrate craving and building up of fat reserves.

Tools for Weight Loss

Because of the diversity of the population of the United States, and the likelihood of there being more than one genetic mechanism that causes us to conserve body fat, there may be many genetic mechanisms that result in obesity. The most common overt cause of obesity is overeating carbohydrate, usually over a period of years. Unfortunately, this can be a very difficult type of obesity to treat.

If you're overweight, you're probably unhappy with your appearance, and no less with your high blood sugars. Perhaps in the past you've tried to follow a restricted diet, without success. Generally, overeating follows two patterns, and frequently they overlap. First is overeating at meals. Second is normal eating at mealtime but with episodic binge eating. Binge eating can be anything from nibbling and snacking between meals to eating everything that does not walk away. Many of the people who follow our low-carbohydrate diet find that their carbohydrate craving ceases almost immediately, possibly because of a reduction in their serum insulin levels. The addition of muscle-building exercise sometimes enhances this effect. Unfortunately, these interventions don't work for everyone.

Medications

If you're a compulsive overeater, if you just can't stop yourself from eating, and are addicted to carbohydrate, you may not be able to adhere to our diet without some sort of medical intervention. Carbohydrate addiction is just as real as drug addiction, and in the case of the diabetic, it can have equally disastrous results. (In actual fact, obesity kills more Americans annually from its related complications than all drugs of abuse combined, including alcohol.)

You need not despair of ever losing weight, however. I have seen a number of "diet-proof" patients over the years get their weight down and blood sugars under control. Over the last several years, as medical science has gained a much more sophisticated understanding of the biological mechanisms that contribute to emotional states such as hunger and mood, many relatively benign medications have been successfully applied to the treatment of compulsive overeating. There is no doubt that when used properly, many appetite suppressants are quite effective in helping people to lose weight. If you simply cannot lose weight, discuss with your physician medicines that may be of use to you. I have used more than forty different medications with my patients and have found many of them to be of great value for treating carbohydrate addiction. Their proper application, however, requires considerable specialized knowledge.

Reducing Serum Insulin Levels

Another group of Type II diabetics has a common story: "I was never fat until after my doctor started me on insulin." Usually these people have been following high-carbohydrate diets and so must have large doses of insulin to effect a modicum of blood sugar control.

Insulin, remember, is the principal fat-building hormone of the body. Although a Type II diabetic may be resistant to insulin-facilitated glucose transport (from blood to tissues), that resistance doesn't diminish insulin's capacity for fat-building. In other words, insulin can be great at making you fat, even though it may be inefficient at lowering your blood sugar. Since excess insulin causes insulin resistance, the more you take, the more you'll need, and the fatter you'll get. This is not an argument against the use of insulin; rather it supports our conclusion that high levels of dietary carbohydrate—which, in turn, require large amounts of insulin—make blood sugar control (and weight reduction) unworkable.

I have witnessed, over and over, dramatic weight loss and blood sugar improvement in people who have merely been shown how to reduce their insulin doses and carbohydrate intake.

Metformin, troglitazone, and similar agents, which we will discuss in detail in Chapter 14, "Oral Hypoglycemic Agents," can also be valuable tools in weight loss. They work by making the body's tissues more sensitive to injected or self-made insulin. As it takes less insulin to accomplish our goal of blood sugar normalization, you'll have less of this fat-building hormone circulating in your body. I have patients using these unique medications who are not diabetic, and they work in a similar way: the body is more sensitive to insulin, so it needs to produce less, and there is, again, less of it present to build fat. One may also have less of a sense of hunger, and less loss of control.

Increasing Muscle Mass

All the above suggests what we have been advocating all along—a low-carbohydrate diet. But what do you do if this plus one of the above medications does not result in weight loss? Another step is muscle-building exercise (see next chapter). This is of value in weight reduction for several reasons. Increasing lean body weight (muscle mass) upgrades insulin sensitivity, enhancing glucose transport and reducing insulin requirements for blood sugar normalization. Lower insulin levels facilitate loss of stored fat. Chemicals produced during exercise (endorphins) tend to reduce appetite, as do lower serum insulin levels. People who have seen results from exercise tend to invest more effort in looking even better (e.g., by not overeating, and perhaps exercising more). They know it can be done.

How to Estimate Your Real Food Requirements

Now suppose you have been following our low-carbohydrate diet, have been conscientiously "pumping iron" every day, and are, in effect, "doing everything right." What else can you do, if you have not lost weight? Well, everyone has some level of caloric intake below which they will lose weight. Unfortunately, the "standard" formulas and tables commonly used by nutritionists set forth caloric guidelines for theoretical individuals of a certain age, height, and sex, but not for real people like you. The only way to find out how much food you need in order to maintain, gain, or lose weight is by experiment. Here is an experimental plan that your physician may find useful. This method usually works, and without counting calories.

Begin by setting an initial target weight and a time frame in which to achieve it. Using standard tables of "ideal body weight" is of little value, simply because they give a very wide target range. This is because some people have more muscle and bone mass for a given height than others. The high end of the ideal weight for a given height on the Metropolitan Life Insurance Company's table is 30 percent greater than the low end for the same height.

Instead, estimate your target weight by looking in the mirror after weighing yourself. (It pays to do this in the presence of your health care provider, because he/she probably has more experience in estimating the weight of your body fat.) If you can grab handfuls of fat at the underside of your upper arms, around your thighs, around your waist, or over your belly, it is pretty clear that your body is set for the next famine. Your estimate at this point need not be terribly precise, because as you lose weight your target weight can be reestimated. Say, for example, that you weigh 200 pounds. You and your physician may agree that a reasonable target would be 150 pounds. By the time you reach 160 pounds, however, you may have lost your visible excess fat—so settle for 160 pounds. Alternately, if you still have fat around your belly when you get down to 150 pounds, it won't hurt to shoot for 145 or 140 as your next target, before making another visual evaluation. Gradually you home in on your eventual target, using smaller and smaller steps.

Once your initial target weight has been agreed upon, a time frame for losing the weight should be established. Again, this need not be utterly precise. It's important, however, not to "crash diet." This may cause a "yo-yo" effect by slowing your metabolism and making it difficult to keep off the lost bulk. Bear in mind that if you starve yourself and lose 10 pounds without adequate dietary protein and an accompanying exercise regimen, you may lose 5 pounds of fat and 5 pounds of muscle. If you gain back that 10 pounds from eating carbohydrate and still are not exercising, it may be 100 percent fat. After crash dieting, once you've reached your target, you may go right back to overeating. I like to have my patients follow a gradual weight-reduction diet that matches as closely as possible what they'll probably be eating after the target has been reached. In other words, once you reach your target, you stay on the same diet you followed while losing weight. This way you've gotten into the habit of eating a certain amount, and you stick to this amount, more or less, for life.

To achieve this, weight loss must be gradual. If you are targeted to lose 25 pounds or less, I suggest a reduction of 1 pound per week. If you're heavier, you may try for 2 pounds per week. If just cutting the carbohydrate, as suggested in prior chapters, results in a more rapid weight loss, don't worry—just enjoy your luck. This has happened to a number of my patients.

Weigh yourself once weekly—stripped, if possible, on the same scale, and before breakfast. Pick a convenient day, and weigh yourself on the same day each week at the same time of day. It's counterproductive and not very informative to weigh yourself more often. Small, normal variations in body weight occur from day to day and can be frustrating if you misinterpret them. Generally speaking, you won't lose or gain a pound of body fat in a day. Continue on your low-carbohydrate diet, with enough protein foods to keep you comfortable.

Let's say that your goal is to lose 1 pound every week. Weigh yourself after one week. If you've

lost the weight, don't change anything. If you haven't lost the pound, reduce the protein at any one meal by one-third. For example, if you've been eating 6 ounces of fish or meat at dinner, cut it to 4 ounces. You can pick which meal to cut back at. Check your weight one week later. If you have lost a pound, don't change anything. If you haven't, cut the protein at another meal by one-third. If you haven't lost the pound in the subsequent week, cut the protein by one-third in the one remaining meal. Keep doing this, week by week, until you are losing at the target rate. Never add back any protein that you have cut out even if you subsequently lose 2 or 3 pounds in a week.

If you've managed to lose at least 1 pound weekly for many weeks, but then your weight levels off, this is a good time for your physician to prescribe the special insulin resistance–lowering agents described in Chapter 14. Alternately you can just start cutting protein again. Continue this until you reach your initial target or until your visual evaluation of excess body fat tells you that further weight loss isn't necessary. Most adults require at least 5 ounces of high-quality protein daily to prevent certain forms of malnutrition. It is therefore unwise to cut your protein intake below this level. Some authorities recommend double this amount. Once you've reached your target weight, do not add back any food. You will probably have to stay on approximately this diet for many years, but you'll easily become accustomed to it. If you required some of the appetite-reducing medications mentioned earlier in the chapter, do not discontinue them. About six months after you reach your target weight, your physician may want to taper off the medication(s) gradually. If you start eating more than your final meal plan calls for, the medication(s) will have to be tapered up again.

Reduce Diabetes Medications While Cutting Protein or Losing Weight

While you're losing weight, keep checking blood sugars at least 5 times daily, at least two days a week. If they consistently drop below your target value for even a few days, advise your physician immediately. It will probably be necessary to reduce the doses of any blood sugar–lowering medications you may be taking. Keeping track of your blood sugar levels as you lose weight and eat less is essential for the prevention of dangerously low blood sugar levels.

Increased Thrombotic Activity During Weight Loss

During weight loss, many people unknowingly experience increased clumping of the small particles in the blood (platelets) that form clots (thrombi). This can increase the risk of heart attack or stroke. Your physician may therefore want you to take an 80 mg enteric coated aspirin once daily after a meal to reduce this tendency.