

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).

Appendix A: What About the Widely Advocated Dietary Restrictions on Fat, Protein, and Salt, and the Current High-Fiber Fad?

Most of this book is instructional, of the how-to variety. The intent of this appendix is to provide you with a little of the science that surrounds the program described in the rest of the book. I hope that I can cut through some of the myths that cloud diet and the treatment of diabetic complications so that you will have the why that supports the how-to. We've already discussed some of the myths. We'll look at the origins of those myths to try to give you as many of the facts as are available at this writing. If your only interest is in the how-to, feel free to skip this chapter.

Once you've started to follow a restricted-carbohydrate diet, you may find yourself pressured by well-meaning but uninformed friends or family, or even newspaper articles, to cease penalizing yourself and eat more "fun" foods, sweets, and treats. This chapter will provide you with specific scientific information that underpins my philosophy and will perhaps give you some ammunition for responding to this pressure. Even if you skip it now, you may want to come back to it later, or show it to your loved ones to lay their concerns to rest. As I don't expect most readers to be scientists, I've tried to keep all these explanations relatively simple. Some of the explanations may at this moment represent more theory than fact, but they're based on the latest information available to us.

How Did the Commonly Prescribed High-Carbohydrate Diet Come About?

If, like me, you've had diabetes for a while, you've probably been told to cut way down on your dietary intake of fat, protein, and salt, and to eat lots of complex carbohydrate. You may even have read this advice in publications circulated to diabetic patients.

Why is such advice being promulgated, when the major cause of such diabetic complications as heart disease, kidney disease, high blood pressure, and blindness is high blood sugar?

When I first developed diabetes, in 1947, little was known about why this disease, even when treated, caused early death and such distressing complications. Prior to the availability of insulin, about twenty-five years earlier, people with Type I diabetes usually died within a few months of

diagnosis. Their lives could be prolonged somewhat with a diet that was very low in carbohydrate and usually high in fat. Sufferers from the milder Type II diabetes frequently survived on this type of diet, without supplemental medication. When I became diabetic, oral hypoglycemic agents were not available, and many people were still following very low carbohydrate, high-fat diets. It was at about this time that diets very high in saturated fats, with resultant high serum cholesterol levels, were experimentally shown to correlate with blood vessel and heart disease in animals. It was promptly assumed by many physicians that the complications of diabetes, nearly all of which related to abnormalities of large or small blood vessels, were caused by the high-fat diets. I and many other diabetics were therefore treated with a high-carbohydrate, low-fat diet. This new diet was adopted in the mid-1940s by the ADA, the New York Heart Association, and eventually by the American Heart Association (AHA) and other groups around the world. On the new diet, many of us had even higher serum cholesterol levels, and still developed the grave long-term complications of diabetes. Seemingly unaware of the importance of blood sugar control, the ADA raised the recommended carbohydrate content to 40 percent of calories, and then more recently to 60 percent.

Recent Developments Regarding Risk Factors for Heart Disease

In the past twenty years, research studies have generated considerable new information about heart disease and vascular (blood vessel) disease in general, and their relationship to diabetes in particular. Some of this more recent information is summarized here.

A number of fatty substances have been found in the blood which relate to risk of heart attacks and vascular disease. These include HDL (high-density lipoprotein), LDL (low-density lipoprotein), triglyceride, fibrinogen, and lipoprotein(a). High serum levels of LDL, triglyceride, fibrinogen, and lipoprotein(a) tend to increase cardiovascular risk, while high levels of HDL tend to protect from cardiovascular disease. Cholesterol is a component of both LDL and HDL particles. The fraction of total cholesterol found in LDL particles is an index of risk, while the fraction of cholesterol found in HDL particles is an index of protection. Nowadays, when we want to estimate the effects of lipids upon the risk of coronary artery disease, we look at the ratio of total cholesterol to HDL and also at fasting triglyceride levels. Someone with high serum HDL can thus have a high total cholesterol and yet be at low statistical risk for a heart attack. Conversely, a person with low total cholesterol and very low HDL may be at high risk. Recently a very large multicenter study (the Lipid Research Clinics Trial) investigated the effects of a low-fat, high-carbohydrate diet on middle-aged men. The study followed 1,900 people for seven years. Throughout this period, total cholesterol had dropped 5 percent from baseline in the low-fat group, but serum triglyceride went up about 10 percent! (Serum triglyceride rises very rapidly after a high-carbohydrate meal in nondiabetics, and moves up and down with blood sugar levels in most diabetics.) As with prior studies, no significant correlation was found between serum cholesterol levels and mortality rates.

On average, diabetics with chronically high blood sugars have elevated levels of LDL (the "bad" cholesterol) and depressed levels of HDL (the "good" cholesterol), even though the ADA low-fat diet has now been in use for many years. Of great importance is the recent discovery that the forms of LDL that harm arteries are small, dense LDL, oxidized LDL, and glycosylated LDL. All of these increase as blood sugar increases. In addition, independently of blood sugars, high

serum insulin levels caused by high-carbohydrate diets bring about increased production of small, dense LDL particles and enlargement of the cells lining and surrounding arteries. Under normal conditions, receptors in the liver remove LDL from the bloodstream and signal the liver to reduce its manufacture of LDL when serum levels rise even slightly. Glucose may bind to the surface of the LDL particle and also to liver LDL receptors, so that LDL cannot be recognized by its receptors. In people with high blood sugars, many LDL particles thus become glycosylated, and are therefore not cleared by the liver. They accumulate in the blood, where they can become incorporated into the walls of arteries, forming fatty deposits called atherosclerotic plaques. Since liver LDL production cannot be turned off by the glycosylated LDL (and also the presence of glycosylated LDL receptors), the liver continues to manufacture more LDL, even though serum levels may be elevated.

The proteins in the walls of arteries can also become glycosylated, rendering them sticky. Other proteins in the blood then stick to the arterial walls, causing further buildup of plaque. Serum proteins also glycosylate in the presence of glucose. White blood cells called macrophages ingest glycosylated proteins and glycosylated LDL. The loaded macrophages swell up, becoming very large. These transformed macrophages, loaded with fatty material, are called foam cells. The foam cells penetrate the sticky arterial walls, causing disruption of the orderly architecture of the artery, and narrow the channel through which blood can flow.

In recent years, the tendency of blood to clot has come into focus as a major cause of heart attacks. People whose blood clots too readily are at very high risk. You may recall that one of the medical names for a heart attack is coronary thrombosis. A thrombus is a clot, and coronary thrombosis refers to the formation of a large clot in one of the arteries that feed the heart. People who have elevated levels of certain clotting precursors or depressed levels of clotting inhibitors in their blood are at high risk of dying from heart attacks. The risk probably far exceeds that caused by high LDL or low HDL. Some of the blood factors that enhance clotting include fibrinogen and factor VII. Another factor, lipoprotein(a)—abbreviated Lp(a)—inhibits the destruction of small thrombi before they become large enough to cause a heart attack. All of these factors have been found to increase in people with chronically high blood sugars. Platelets, or thrombocytes, are particles in the blood that play major roles in the blocking of arteries and the formation of clots. These have been shown to clump together and stick to arterial walls much more aggressively in people with high blood sugars. What is exciting is that all of these factors, including sticky platelets, tend to normalize as long-term blood sugars improve.

Diabetics die from heart failure at a rate far exceeding that of people with normal glucose tolerance. Heart failure involves a weakening of the cardiac muscle so that it cannot pump enough blood. Most long-term, poorly controlled diabetics have a condition called cardiomyopathy. In diabetic cardiomyopathy, the muscle tissue of the heart is slowly replaced by scar tissue over a period of years. This weakens the muscle so that it eventually "fails." There is no evidence linking cardiomyopathy with dietary fat intake or serum lipids.

A fifteen-year study of 7,038 French policemen in Paris reported that "the earliest marker of a higher risk of coronary heart disease mortality is an elevation of serum insulin level." A study of middle-aged nondiabetic women at the University of Pittsburgh showed an increasing risk of heart disease as serum insulin levels increased. Other studies in nondiabetics have shown strong

correlations between serum insulin levels and other predictors of cardiac risk such as hypertension, elevated triglyceride, and low HDL. The importance of elevated serum insulin levels (hyperinsulinemia) as a cause of heart disease and hypertension has taken on such importance that a special symposium on this subject was held at the end of the 1990 annual meeting of the ADA. A report in a subsequent issue of the journal *Diabetes Care* quite appropriately points out that "there are few available methods of treating diabetes that do not result in systemic hyperinsulinemia" unless the patient is following a low-carbohydrate diet. Although the AHA and the ADA have been recommending low-fat, high-carbohydrate diets for diabetics for many decades, no one had compared the effects on the same patients of low- versus high-carbohydrate diets until the late 1980s. Independent studies performed in Texas and California demonstrated lower levels of blood sugar and improved blood lipids when patients were put on lower-carbohydrate, high-fat diets. It was also shown that, on average, for every 1 percent increase in HgbA1C (the test for average blood sugar over the prior four months), total serum cholesterol rose 2.2 percent and triglycerides increased 8 percent.

The National Health Examination Follow-Up Survey, which followed 4,710 people, reported in 1990 that "in the instance of total blood cholesterol, we found no evidence in any age-sex group of a risk associated with elevated values." That's right—they found no risk associated directly with elevated total cholesterol. On the same page, this study lists diabetes as by far the single most important risk factor affecting mortality. In males aged 55–64, for example, diabetes was associated with 60 percent greater mortality than smoking and double the mortality associated with high blood pressure.

The evidence is now simply overwhelming that elevated blood sugar is the major cause of the high serum lipid levels among diabetics and, more significantly, the major factor in the high rates of various heart and vascular diseases associated with diabetes. Many diabetics were put on low-fat diets for so many years, and yet these problems didn't stop. It is only logical to look elsewhere, to elevated blood sugar and hyperinsulinemia, for the cause of what kills and disables so many of us.

My personal experience with diabetic patients is very simple. When we reduce dietary carbohydrate, blood sugars improve dramatically. After about two months of improved blood sugars, we repeat our studies of lipid profiles and thrombotic risk factors. In the great majority of cases, I see normalization or improvement of abnormalities. This parallels what happened to me nearly thirty years ago when I abandoned the high-carbohydrate, low-fat diet that I had been following since 1947.*

Why Is Protein Restriction So Common?

About 30 percent of diabetics develop kidney disease (nephropathy). Diabetes is the greatest single cause of kidney failure in the United States. Early kidney changes can be found within two to three years of the onset of high blood sugars. As we discussed briefly in Chapter 9, the common restrictions on protein intake by diabetic patients derive from fear regarding this problem, and ignorance of the actual causes of diabetic kidney disease.

By looking at how the kidney functions, one can better understand the relative roles of glucose and protein in kidney failure of diabetes. The kidney filters wastes, glucose, drugs, and other potentially toxic materials from the blood and deposits them into the urine. It is the urine-making organ. A normal kidney contains about 6 million microscopic blood filters, called glomeruli. Figure A-1 illustrates how blood enters a glomerulus through a tiny artery called the incoming arteriole. The arteriole feeds a bundle of tiny vessels called capillaries. The capillaries contain tiny holes or pores that carry a negative electrical charge. The downstream ends of the capillaries merge into an outgoing arteriole, which is narrower than the incoming arteriole. This narrowing results in high fluid pressure when blood flows through the capillary tuft. The high pressure forces some of the water in the blood through the pores of the capillaries. This water dribbles into the capsule surrounding the capillary tuft. The capsule, acting like a funnel, empties the water into a pipelike structure called the tubule. The pores of the capillaries are of such a size that small molecules in the blood, such as glucose and urea, can pass through with the water to form urine. In a normal kidney, large molecules, such as proteins, cannot readily get through the pores. Since most blood proteins carry negative electrical charges, even the smaller proteins in the blood cannot easily get through the pores, because they are repelled by the negative charge on each pore.

The glomerular filtration rate (GFR) is a measure of how much filtering the kidneys perform in a given period of time. Anyone with a high blood sugar and normal kidneys will have an excessively high GFR. This is in part because blood glucose draws water into the bloodstream from the surrounding tissues, thus increasing blood volume, blood pressure, and blood flow through the kidneys. A GFR that is one-and-a-half to two times normal is commonplace in diabetics with high blood sugars prior to the onset of permanent injury to their kidneys. These people may typically have as much glucose in a 24-hour urine collection as the weight of 5 to 50 packets of sugar. According to an Italian study, an increase in blood sugar from 80 mg/dl to 272 mg/dl resulted in an average GFR increase of 40 percent even in diabetics with severe kidney disease. Before we knew about glycosylation of proteins and the other toxic effects of glucose upon blood vessels, it was speculated that the cause of diabetic kidney disease (nephropathy) was this excessive filtration (hyperfiltration).

The metabolism of dietary protein produces waste products such as urea and ammonia, which contain nitrogen. It therefore had been speculated that in order to clear these wastes from the blood, people eating large amounts of protein would have elevated GFRs. As a result, diabetics have been urged to reduce their protein intake to low levels. Studies by an Israeli group, however, of people on high-protein (meat-eating) and very low protein (vegetarian) diets, disclosed no difference in GFR. Furthermore, over many years on these diets, kidney function was unchanged between the two groups. A report from Denmark described a study in which Type I diabetics without discernible kidney disease were put on protein-restricted diets, and experienced a very small change in GFR and no change in other measures of kidney function. These would suggest that the currently prevailing admonition to all diabetics to reduce protein intake is unjustified.

Recent studies on diabetic rats have shown the following: Rats with blood sugars maintained at 250 mg/dl rapidly develop diabetic nephropathy. If their dietary protein is increased, kidney destruction accelerates. Diabetic rats at the same laboratory, with blood sugars maintained at 100

mg/dl, live full lives and never develop nephropathy, no matter how much protein they consume. Diabetic rats with high blood sugars and significant nephropathy have shown total reversal of their kidney disease after blood sugars were normalized for several months.

Recent studies on diabetic rats have shown the following: Rats with blood sugars maintained at 250 mg/dl rapidly develop diabetic nephropathy. If their dietary protein is increased, kidney destruction accelerates. Diabetic rats at the same laboratory, with blood sugars maintained at 100 mg/dl, live full lives and never develop nephropathy, no matter how much protein they consume. Diabetic rats with high blood sugars and significant nephropathy have shown total reversal of their kidney disease after blood sugars were normalized for several months.

Other studies have enabled researchers to piece together a scenario for the causes of diabetic nephropathy, where glycosylation of proteins, abnormal clotting factors, abnormal platelets, antibodies to glycosylated proteins, and so on, join together to injure glomerular capillaries. Early injury may only cause reduction of electrical charge on the pores. As a result, negatively charged proteins such as albumin leak through the pores and appear in the urine. Glycosylated proteins leak through pores much earlier than normal proteins. High blood pressure, and especially high serum insulin levels, can increase GFR and force even more protein to leak through the pores. If some of these proteins are glycosylated, they will stick to the mesangium, the tissue between the capillaries. Examination of diabetic glomeruli indeed discloses large deposits of glycosylated proteins and antibodies to glycosylated proteins in capillary walls and mesangium. As these deposits increase, the mesangium compresses the capillaries, causing pressure in the capillaries to increase and larger proteins to leak from the pores. This leads to more thickening of the mesangium, more compression of the capillaries, and acceleration of destruction. Eventually the mesangium and capillaries become a mass of scar tissue. Independently of this, both high blood sugars and glycosylated proteins cause mesangial cells to produce type IV collagen, a fibrous material that further increases their bulk.

Many studies performed on humans show that when blood sugars improve, GFR improves and less protein leaks into the urine. When blood sugars remain high, however, there is further deterioration. There is a point of no return, where a glomerulus has been so injured that no amount of blood sugar improvement can revive it.

Nowadays many diabetics who have lost all kidney function are treated by artificial kidneys (dialysis machines) that remove nitrogenous wastes from the blood. In order to reduce the weekly number of dialysis treatments, which are costly and unpleasant, patients are severely restricted in their consumption of dietary protein. Instead of using large amounts of carbohydrate to replace the lost calories, many dialysis centers now recommend olive oil to their diabetics. Olive oil is high in monounsaturated fats, which are believed to lower the risk of heart disease. In summary: Diabetic nephropathy does not appear if blood sugar is kept normal. Dietary protein does not cause diabetic nephropathy, but can possibly (still uncertain) accelerate the process once there has been a considerable amount of kidney damage. Dietary protein has no substantial effect upon the GFR of healthy kidneys, certainly not in comparison to the GFR increase caused by elevated blood sugar levels.*

Restrictions on Salt Intake: Are They Reasonable for All Diabetics?

Many diabetics have hypertension, or high blood pressure. About half of all people with hypertension will experience blood pressure elevations when they eat substantial amounts of salt. Hypertension accelerates glomerulopathy (destruction of the glomerulus) in people with chronically elevated blood sugars, but in Type I diabetes, hypertension usually appears after, not before, the appearance of significant amounts of albumin in the urine. Is it therefore appropriate to ask all diabetics to lower their salt intake?† Let us look at a few of the mechanisms involved in the hypertension that some diabetics experience.

People with advanced glomerulopathy will inevitably develop hypertension in part because GFR is severely diminished. These people cannot make enough urine, and therefore retain water. Excessive water in the blood causes elevated blood pressure. There are many other ways hypertension can be caused by high blood sugars.

The mere presence of high blood sugar will cause water to leave tissues and enter the bloodstream, even experimentally in nondiabetics. It is not unusual to observe reduction in blood pressure concomitant with control of blood sugar. Studies have shown that many, and possibly most, hypertensive nondiabetics are insulin-resistant, and therefore have high serum insulin levels. In addition to causing elevation of serum triglycerides and reduction in serum HDL in nondiabetics, high serum insulin levels have long been known to foster salt and water retention by the kidneys. Furthermore, excessive insulin stimulates the sympathetic nervous system, which in turn speeds up the heart and constricts blood vessels, causing further increase in blood pressure. Thus Type II diabetics who eat lots of carbohydrate, and therefore will tend to make excessive insulin, can readily develop hypertension. Type I diabetics treated with the usual industrial doses of insulin to cover high-carbohydrate diets are likewise more susceptible to hypertension. One dramatic study showed that in hypertensive individuals, blood pressure is directly proportional to serum insulin level. A report from Nottingham, England, showed that a brief infusion of insulin and glucose would increase blood pressure in normal men without changing their blood sugars.

Why don't all diabetics on high-carbohydrate diets or all poorly controlled diabetics have hypertension?

One reason is that the body has several very efficient systems for unloading sodium (a component of salt) and water. One of the more important of these systems is controlled by a hormone manufactured in the heart called atrial natriuretic factor (ANF). When the heart is expanded by even a slight fluid overload, it produces ANF. The ANF then signals the kidneys to unload sodium and water. Hypertensive individuals, and the children of two hypertensive parents, tend to produce much lower amounts of ANF than do normal people. Nonhypertensive diabetics apparently are able to produce enough ANF to control the blood pressure effects of high blood sugars and high serum insulin levels, provided they do not have moderately advanced kidney disease. Indeed, a study, in which some of my patients participated, showed that diabetics with high blood sugars produce significantly more ANF than those with lower blood sugars.

How does all this apply to you? First, you and your physician should know if you have glomerulopathy. This is readily determined if the tests suggested in Chapter 2 are performed. If these tests are abnormal, your physician may advise you to reduce your salt intake. Whether your renal risk profile is normal or abnormal, your resting blood pressure should also be measured. A proper measurement requires that you first be seated in a quiet room, without conversation, for 15–30 minutes. Blood pressure should be measured every 5 minutes, until it drops to a low value and then starts to increase. The lowest reading is the significant one. If you get nervous in the doctor's office, then you should measure your own blood pressure at home in a similar fashion. Repeated measurements, with low values just exceeding 135/85, suggest that your blood pressure is "borderline." (The American Diabetes Association suggests that 120/80 be considered borderline for younger diabetics.) You may benefit from dietary salt reduction. The only way to find out is to check your blood pressure while on your current salt intake, and again after following a low-salt (sodium) diet for at least three weeks. Your physician can give you guidelines for such a diet, and you can consult nutritional tables such as those in the books listed in Chapter 3. I would suggest that resting blood pressures be measured several times a day, and at the same hours each day, throughout the study. Blood pressures can then be averaged, and the averages compared. If your blood pressure drops significantly on the low-salt diet, your physician may urge you to keep the salt intake down. Alternately, he may want you to take small amounts of supplemental potassium, which tends to offset the effects of dietary salt on blood pressure. Recent studies suggest that as many as 40 percent of hypertensive patients (the so-called low-renin hypertensives) may show lower blood pressures when they take calcium supplements.

What About Dietary Fiber?

"Fiber" is a general term that has come to refer to the undigestible portion of many vegetables and fruits. Some vegetable fibers, such as guar and pectin, are soluble in water. Another type of fiber, which some of us call roughage, is not water-soluble. Both types appear to affect the movement of food through the gut (soluble fiber slows processing in the upper digestive tract, while insoluble fiber speeds digestion farther down). Certain insoluble fiber products, such as psyllium, have long been used as laxatives. Consumption of large amounts of dietary fiber is usually unpleasant, because both types can cause abdominal discomfort, diarrhea, and flatulence. Sources of insoluble fiber include most salad vegetables. Soluble fiber is found in many beans, such as garbanzos, and in certain fruits, such as apples.

I first learned of attempts at using fiber as an adjunct to the treatment of diabetes about twenty years ago. At that time, Dr. David Jenkins, in England, reported that guar gum, when added to bread, could reduce the maximum postprandial blood sugar rise from an entire meal by 36 percent in diabetic subjects. This was interesting for several reasons. First of all, the discovery occurred at a time when few new approaches to controlling blood sugar had appeared in the medical literature. Second, I missed the high-carbohydrate foods I had given up, and hoped I might possibly reinstate some. I managed to track down a supplier of powdered guar gum, and placed a considerable amount into a folded slice of bread. I knew how much a slice of bread would affect my blood sugar, and so as an experiment, I used the same amount of guar gum that Dr. Jenkins had used, and then ate the concoction on an empty stomach. The chore was difficult, because once moistened by my saliva, the guar gum stuck to my palate and was difficult to swallow. I did not find any change in the subsequent blood sugar increase. Despite the

unpleasantness of choking down powdered guar gum (which is often used in commercial products such as ice cream as a thickener), I repeated this experiment on two more occasions, with the same result. Subsequently, some investigators have announced results similar to those of Dr. Jenkins, yet other researchers have found no effect on postprandial blood sugar. In any event, a reduction of postprandial blood sugar by only 36 percent really isn't adequate for our purpose, since we're shooting for the same blood sugars as nondiabetics. This means virtually no rise after eating.

Dr. Jenkins also discovered, however, that the chronic use of guar gum resulted in a reduction of serum cholesterol levels. This is probably related to the considerable recirculation of cholesterol through the gut. The liver secretes some cholesterol into bile, which is released into the upper intestine. This cholesterol is later absorbed lower in the intestines, and eventually reappears in the blood. Guar binds the cholesterol in the gut, so that rather than being absorbed, it appears in the stool.

In the light of these very interesting results, other researchers studied the effect of foods (usually beans) containing other soluble forms of fiber. When beans were substituted for faster-acting forms of carbohydrate, postprandial blood sugars in diabetics increased more slowly, and the peaks were even slightly reduced. Serum cholesterol levels were also reduced by about 15 percent. But subsequent studies, reported in 1990, have uncovered flaws in the original reports, casting serious doubt upon any direct effect of these foods upon serum lipids. In any event, postprandial blood sugars were rarely normalized by such diets.

Many popular articles and books have appeared advocating "high-fiber" diets for everyone—not just diabetics. Somehow, "fiber" came to mean all fiber, not just soluble fiber, even though the only viable studies had utilized such products as guar gum and beans.

In my experience, reduction of dietary carbohydrate is far more effective in preventing blood sugar increases after meals. The lower blood sugars, in turn, bring about improved lipid profiles. A recent food to join the high-fiber trend is oat bran. This has gotten a lot of play in the popular press. Recently, a patient of mine started substituting oat bran muffins for protein in her diet. Before she started, her HgbA1C (see Chapter 2) was within the normal range and her ratio of total cholesterol to HDL was very low (meaning her cholesterol risk ratio was low). After three months on oat bran, her HgbA1C became elevated and her cholesterol-to-HDL ratio nearly doubled. I tried one of her tiny oat bran muffins after first injecting 3 units of fast-acting insulin (nearly as much as I use for an entire meal). After 3 hours, my blood sugar went up by about 100 mg/dl, to 190 mg/dl. This illustrates the adverse effect that most oat bran preparations can have upon blood sugar. The reason for this is that most such preparations contain flour. On the other hand, I find that certain bran products, such as the bran crackers listed in Chapter 10, raise blood sugar very little. This is because, unlike most packaged bran products, they contain mostly bran and little flour. They therefore have very little carbohydrate. You can perform similar experiments yourself—just use your blood glucose meter. Beware of commercial "high-fiber" products that promise cholesterol reduction. If they contain carbohydrate, they must at least be counted in your meal plan and will probably render little or no improvement in your lipid profile.

Fiber, like carbohydrate, is not essential for a healthy life. Just look at the Eskimos and other hunting populations that survive almost exclusively on protein and fat, and don't develop cardiac or circulatory diseases.*

What Diet Will Work for You?

Actual results are the yardstick for an appropriate diet. We have the tools for self-monitoring of blood sugar and blood pressure. We have tests for measuring kidney function, HgbA1C, thrombotic risk profiles, and lipid profiles (see Chapter 2). Under your doctor's supervision, try our diet recommendations for at least two months. Then try any other diet plan for two months and see what happens. The differences may not be in the direction that the popular literature would predict.

