Chapter 6
Weight Loss — If You’re Overweight

Most of my patients initially feel somewhat deprived, but they are also grateful to feel more alert and healthier — sometimes more so than they have in years. I fall into this category myself. My mouth waters whenever I pass a bakery shop and sniff the aroma of fresh bread, but I am also grateful simply to be alive and sniffing.

Part 2
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 the anthropologist James V. Neel in 1962 to explain the high incidence of obesity and type 2 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, muscular, and wiry people. They did not know what obesity was and in fact had no word for it in their vocabulary.

Their food supply diminished in the early part of the twentieth century, something that had occurred repeatedly throughout their history. Now, however, they weren’t faced with famine, because the Bureau of Indian Affairs provided them with flour and corn. An astonishing thing happened. These lean people developed an astronomical incidence of obesity — 100 percent of adult Pima Indians today are grossly obese, with a staggering incidence of diabetes. Fully 65 percent of adults are type 2 diabetics. Over the last several
years, even many Pima children have become obese, type 2 diabetic teenagers. A similar scenario is now playing out across the United States in the general population. The pace may be slower, but the result is similar.

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 could survive long periods without food. How did they do it? Although it may be simplifying somewhat, the mechanism essentially works like this: Those who came to crave carbohydrate after having just the slightest bit, 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. Truly survival of the fittest — provided famines would continue. A British scientist, Andrew Prentice, a professor at the London School of Hygiene and Tropical Medicine, has a theory that Americans in general have a higher incidence of the thrifty genotype. His theory is that people who came to this country through most of its history endured astonishing hardship — a difficult Atlantic crossing that killed off many, then incredible hardship once they were here. Those who survived to have children had to be able to resist that hardship.

A genetic strain of chronically obese mice created in the early 1950s demonstrates quite vividly how valuable thrifty genes can be in famine. When these mice are allowed an unlimited food supply, they balloon, adding as much as half again the body weight of normal mice. Yet deprived of food, these mice can survive 40 days, versus 7–10 days for normal mice.

Recent research on these mice provides some tantalizingly direct evidence of the effect a thrifty genotype can have upon physiology. In normal mice, a hormone called leptin is produced in the fat cells (also a hormone human fat cells produce, with apparently similar effect). The hormone tends to inhibit overeating, speed metabolism,
and act as a modulator 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. Normal mice injected with leptin also ate less, became more active, and lost weight, though not nearly as much. 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 Indians’ ability to survive long enough to find food is nothing short of a blessing. But when life is almost entirely sedentary and satisfying carbohydrate craving is as simple as nibbling on snack food, what was once an asset becomes a very serious liability. And these people are dedicated snackers. When I visited them and spoke with researchers, I was surprised that they did not keep track of the Pimas’ snack food consumption. The response I got was that it was simply impossible because snacking was virtually nonstop. When I saw them off the reservation, they were rarely without a bag of some kind of snack.

Current statistics estimate that slightly more than 60 percent of the overall population of the United States is chronically overweight — and there is even greater reason to be concerned, because the number has been increasing by 1 percent each year since the publication of the USDA’s lowfat food pyramid.

The thrifty genotype has its most dramatic appearance in isolated populations like the Pimas, who 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 2 diabetes. The same is true of the Australian Aborigines since 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 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 by the last meal — may have lower levels of certain brain 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. The next study the Wurtmans performed involved giving students a medication to lower serotonin levels. This resulted in carbohydrate craving.

Serotonin is a neurotransmitter that seems to reduce anxiety as it produces satiety. Other neurotransmitters such as dopamine, norepinephrine, and endorphins can also affect our feelings of satiety and anxiety. There are now more than one hundred known neurotransmitters, and many more of them may affect mood in response to food in ways that are just beginning to be researched and understood.

In people with the thrifty genotype, it may be that 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, the opposite of euphoria. Eating carbohydrates temporarily causes the individual to feel not only less hungry but also more at ease.

Too Be Continued in Chapter 6 Part3 - COMFORT FOODS AND CARBOHYDRATE ADDICTION
We would like to thank the publisher Little Brown and Company and Dr. Richard K. Bernstein, for allowing us to provide excerpts from The Diabetes Diet.

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