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What We Can Do

Whether you're born predisposed to get fat is beyond your control. What Adiposity 101 teaches us, though, is that this predisposition is set off by the carbohydrates we eat—by their quantity and their quality. As I said, it's carbohydrates that ultimately determines insulin secretion and insulin that drives the accumulation of body fat. Not all of us get fat when we eat carbohydrates, but for those of us who do get fat, the carbohydrates are to blame; the fewer carbohydrates we eat, the leaner we will be.

A comparison with cigarettes is apt. Not every longtime smoker gets lung cancer. Only one in six men will, and one in nine women. But for those who do get lung cancer, cigarette smoke is far and away the most common cause. In a world without cigarettes, lung cancer would be a rare disease, as it once was. In a world without carbohydrate-rich diets, obesity would be a rare condition as well.

Not that all foods that contain carbohydrates are equally fattening. This is a crucial point. The most fattening foods are the ones that have the greatest effect on our blood sugar and insulin levels. These are the concentrated sources of carbohydrates, and particularly those that we can digest quickly: anything made of refined flour (bread, cereals, and pasta), liquid carbohydrates (beers, fruit juices, and sodas), and starches (potatoes, rice, and corn). These foods flood the bloodstream quickly with glucose. Blood sugar shoots up; insulin shoots up. We get fatter. Not sur-

prisingly, these foods have been considered uniquely fattening for nearly two hundred years (as I'll discuss later).*

These foods are also, almost invariably, the cheapest calories available. This is the conspicuous explanation for why the poorer we are, the fatter we're likely to be; why, as I discussed at the outset, it's all too easy to find extremely poor populations, past and present, with obesity and diabetes rates that rival those in the United States and Europe today. This was the explanation suggested by physicians who worked with these populations in the 1960s and 1970s, and now we know it's supported by the science.

"Most third world countries have a high carbohydrate intake," wrote Rolf Richards, the British-turned-Jamaican diabetes specialist in 1974. "It is conceivable that the ready availability of starch in preference to animal protein, contributing as it must the main caloric requirements of these populations, leads to increased lipogenesis [fat formation] and the development of obesity." People in these populations get fat not because they eat too much or are too sedentary but because the foods they live on—the starches and refined grains that make up the great majority of their diet, and the sugar—are literally fattening.

The carbohydrates in leafy green vegetables like spinach and kale, on the other hand, are bound up with indigestible fiber and take much longer to be digested and enter our bloodstream. These vegetables contain more water and fewer digestible carbohydrates for their weight than starches like potatoes. We have to eat far more to get the same load of carbohydrates, and those carbohydrates take longer to digest. As a result, blood sugar levels remain relatively low when we eat these vegetables; they initiate a

*How our blood sugar responds to different foods is known technically as the "glycemic index," a reasonably good measure of how our insulin will respond. The higher the glycemic index of a particular food, the greater the blood sugar response. Entire books have been published on the idea of minimizing the glycemic index of our diets and, by doing so, minimizing the insulin we secrete and the fat we accumulate.

far more modest insulin response and are therefore less fattening. It is possible, though, that some people may be so sensitive to the carbohydrates in their diet that even these green vegetables may be a problem.

The carbohydrates in fruits, though relatively easy to digest, are also diluted more by water and so are less concentrated than the carbohydrates in starches. Given an apple and a potato of the same weight, the potato will have a significantly greater effect on blood sugar, which suggests that it *should* be more fattening. But that doesn't mean fruit won't fatten some people.

What makes fruit worrisome from the perspective of Adiposity 101 is that it is sweet to the taste precisely because it contains a type of sugar known as fructose, and fructose is uniquely fattening as carbohydrates go. As nutritionists and public-health authorities have become increasingly desperate in their attempts to rein in the obesity epidemic, they've also become increasingly strident in their suggestions that we eat copious fruit along with green vegetables. Fruit doesn't have to be processed before we eat it: it's fat- and cholesterol-free; it has vitamins (vitamin C in particular) and antioxidants; and so, by this logic, it must be good for us. Maybe so. But if we're predisposed to put on fat, it's a good bet that most fruit will make the problem worse, not better.

The very worst foods for us, almost assuredly, are indeed sugars—sucrose (table sugar) and high-fructose corn syrup in particular. Public-health authorities and journalists have recently taken to attacking high-fructose corn syrup as a cause of the obesity epidemic. It was introduced in 1978 and replaced the sugar in most soft drinks in the United States by the mid-1980s. Total sugar consumption ("caloric sweeteners," as the Department of Agriculture calls them, to distinguish them from "non-caloric" artificial sweeteners) promptly increased from roughly 120 pounds per capita yearly to 150, since Americans didn't realize that high-fructose corn syrup was just another form of sugar. It is, though. I'm going to refer to both of them as sugars, because they are effectively identical. Sucrose, the white granulated stuff we put in

our coffee and sprinkle on our cereal, is half fructose and half glucose. High-fructose corn syrup, in the form we typically get it in juices, sodas, and fruity yogurts, is 55 percent fructose (which is why it's known in the food industry as HFCS-55) 42 percent glucose, and 3 percent other carbohydrates.

It's the fructose in these sweeteners that makes them sweet, just as it makes fruit sweet, and it appears to be the fructose that makes them so fattening and, in turn, so bad for our health. The American Heart Association and other authorities have lately—better late than never—taken to targeting fructose, and thus sugar and high-fructose corn syrup, as a cause of obesity and maybe even heart disease, but they do so primarily on the basis that these sweeteners are "empty calories," which means they don't come with any vitamins, minerals, or antioxidants attached. This misses the point, however. Fructose actually has unhealthy effects—including making us fat—that have little to do with its lack of vitamins or antioxidants and far more to do with how our bodies process it. The sugary combination of roughly half fructose and half glucose might be particularly effective in making us fat.

When we digest the carbohydrates in starches, they eventually enter our bloodstream as glucose. Blood sugar increases, insulin is secreted, and calories are stored as fat. When we digest sugar or high-fructose corn syrup, much of the glucose ends up in the general circulation, raising our blood sugar levels. The fructose, however, is metabolized almost exclusively in the liver, which has the necessary enzymes to do it. So fructose has no immediate effect on our blood sugar and insulin levels, but the key word is "immediate"—it has plenty of long-term effects.

The human body, and particularly the liver, never evolved to handle the kind of fructose load we get in modern diets. Fructose exists in fruits in relatively small quantities—thirty calories in a cup of blueberries, for instance. (Some fruit, though, as I'll discuss later, has been bred for generations to increase its fructose content.) There are eighty calories' worth in a twelve-ounce can of Pepsi or Coke. Twelve ounces of apple juice has eighty-five

calories of fructose. Our livers respond to this flood of fructose by turning much of it into fat and shipping it to our fat tissue. This is why even forty years ago biochemists referred to fructose as the most "lipogenic" carbohydrate—it's the one we convert to fat most readily. Meanwhile, the glucose that comes with the fructose raises blood sugar levels and stimulates insulin secretion and puts the fat cells in the mode to store whatever calories come their way—including the fat generated in the liver from the fructose.

The more of these sugars we consume, and the longer we have them in our diet, the more our bodies apparently adapt by converting them to fat. Our "pattern of fructose metabolism" changes with time, as the British biochemist and fructose expert Peter Mayes says. Not only will this cause us to accumulate fat directly in the liver—a condition known as "fatty liver disease"—but it apparently causes our muscle tissue to become resistant to insulin through a kind of domino effect that is triggered by the liver cells' resistance.

So, even though fructose has no immediate effect on blood sugar and insulin, over time—maybe a few years—it is a likely cause of insulin resistance and thus the increased storage of calories as fat. The needle on our fuel-partitioning gauge will point toward fat storage, even if it didn't start out that way.

It's quite possible that if we never ate these sugars we might never become fat or diabetic, even if the bulk of our diet were still starchy carbohydrates and flour. This would explain why some of the world's poorest populations live on carbohydrate-rich diets and don't get fat or diabetic, while others aren't so lucky. The ones that don't (or at least didn't), like the Japanese and Chinese, were the ones that traditionally ate very little sugar. Once you do start to fatten, if you want to stop the process and reverse it, these sugars have to be the first to go.

Alcohol is a special case. Alcohol is metabolized mostly in the liver. Some 80 percent of the calories from a shot of vodka, for instance, will go straight to the liver to be converted into a small amount of energy and a large amount of a molecule called "cit-

rate." The citrate then fuels the process that makes fatty acids out of glucose. So alcohol will increase the production of fat in the liver, which probably explains alcoholic fatty liver syndrome. It might also make us fatter elsewhere, although whether we store these fats as fat or burn them might depend on whether we eat or drink carbohydrates with the alcohol, which we usually do. Roughly a third of the calories in a typical beer, for instance, come originally from maltose—a refined carbohydrate—compared with the two-thirds from the alcohol itself. A beer belly is the conspicuous result.