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WELLNESS INSTITUTE

OBESITY AND INSULIN

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INTRODUCTION

Among the most common health problems worldwide are obesity, metabolic syndrome (pre-diabetes), and diabetes. These problems, in turn, are associated with increased risk of heart disease and cancer. This newsletter will focus on the roles of uric acid and insulin in creating these problems.

GLUCOSE

Glucose is the primary energy source for the cells of the body. When a doctor talks about blood sugar he is talking about blood glucose. Glucose is used by the body to produce ATP (adenosine triphosphate), the energy that runs the machinery of the cell. Abnormalities of glucose metabolism are at the root of obesity, metabolic syndrome, and diabetes.

Glucose is derived from the foods that we eat. Some foods like honey and dates contain simple sugars, but nature most frequently packages glucose in the form of starches which are long chains of glucose molecules. A starch can contain as many as 200,000 glucose units.

Starches exist in two forms: amylose and amylopectin. When glucose molecules are arranged in long chains, they are called amylopectin. When glucose chains have many branches, they are called amylose.

The many branched amylose is much more difficult for the body to

digest than is the amylopectin. What this means in practice is that amylopectin releases its glucose molecules very rapidly, while such is not the case with amylose.

Foods with a high amylopectin content cause rapid rises in blood sugar and surges in insulin production by the pancreas.

Consider a study on the digestion of rice which is a staple food for half the world's population. Researchers wrote, "Long grain rice was found to have high amylose and low amylopectin content as compared to medium and short grain rice. Long grain rice showed slow release of reducing sugars as compared to medium and short grain rice."

These researchers concluded that "Long grain rice because of its high amylose content is a low glycemic food and can be consumed by diabetics. Sustained slow release of reducing sugars given by long grain rice is desirable in diabetics."

In one rat study animals were fed comparable quantities of high amylopectin starch and high amylose starch.



Insulin responses were 50% higher after 16 weeks of high amylopectin feeding.

Similar human feeding studies have shown significantly lower blood glucose response and insulin release when high amylose rice is consumed.

Studies suggest that amylose content of grain needs to be greater than 50 percent to significantly reduce glucose levels and insulin in men and women.

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GLYCEMIC INDEX

Many factors determine how rapidly the glucose in foods is made available to the blood stream and then the cells. One factor as we have seen is the nature of the starch.

The extent to which carbohydrate is refined plays a big role in the availability of glucose. When fiber, pro-



tein, and oils are removed and the carbohydrate is finely ground or cooked digestive enzymes can break down the carbohydrate much more rapidly and release the glucose in a torrent.

Fiber

Fiber, whether in the food or in the stomach, slows the absorption of glucose. High fiber meals produce lower blood sugars in both diabetic and normal test subjects. Many foods also contain enzyme inhibitors, phytates, and lectins which may slow the digestive process.

Protein

The presence of protein-starch complexes can slow the release of glucose. Legume protein can be tightly bound to carbohydrate. Gluten in whole wheat products may slow carbohydrate digestion and absorption.

Blending and Grinding

The smaller the particle size of a carbohydrate the easier it is for digestive enzymes to get to the food and release the glucose increasing blood sugar and insulin levels. For example, apple sauce will tend to raise blood sugar more rapidly than eating an apple. Quick cook oats are likely to release sugars more rapidly than the old fashioned slow cook variety.

Cooking increases the availability of the sugars in carbohydrate foods. The difference is so dramatic that Dr. John Douglas was able to successfully treat his patients with diabetes by simply increasing the percentage of raw food in the diet.

Cooking time and wet or dry cooking can also alter release of sugars from carbohydrates. Drier or al dente pasta resists digestion compared to

cooking pasta until it is gelatinized.

Fats and Acids

Acids and fats slow down the digestive process and lower the glycemic index of foods. Thus adding mayonaise or vinegar to a potato salad will lower the glycemic effect of the starch in the potato.

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By Evan Swigart from Chicago, USA - Home-made White Bread with Strawberry Jam, CC BY 2.0, <https://commons.wikimedia.org/w/index.php?curid=11626953>

GLYCEMIC INDEX AND LOAD

The first paper on glycemic index appeared in 1981. The concept of glycemic load was introduced in 1997. The relevance of these concepts to insulin resistance and diabetes was elaborated after 1995.

The glycemic index is a number from 0 to 100 given to a food, with pure glucose given the arbitrary value of 100. The index tells us what the rise in blood sugar will be two hours after a given quantity of a specific food is consumed. A low glycemic index is 55 or less, a mid-range value is 56-59, and a high glycemic index is 70 or more.

A related concept is glycemic load which multiplies the glycemic index of a food by the total carbohydrate content of an actual serving of the food. Glycemic load is generally considered more important than the glycemic index of a food.

A weakness of glycemic load is that it does not measure the actual insulin production triggered by a rise in blood sugar and this is very important as we shall see. Two foods can have the same glycemic load, but result in different levels of insulin production. A glycemic load greater than 20 is considered high, 11-19 is consid-

ered medium, and 10 or below is low.

INSULIN INDEX

The major health problem associated with refined carbohydrate intake, however, is increases in insulin production. To compensate for the weakness of glycemic index and glycemic load in this regard, researchers have developed what is called the insulin index.

The insulin index simply involves feeding different foods with a comparable caloric value and measuring the quantity of insulin produced every 15 minutes over a period of two hours. White bread is used as the reference food with a score of 100%.

Researchers found significant differences among foods containing a similar amount of carbohydrate. Protein rich foods and bakery products elicited higher insulin responses than their glycemic index and load results would suggest.

The stomach produces hormones called incretins that increase insulin secretion. Incretins account for 50-70 percent of insulin secretion after consuming foods.

Incretins can also be triggered by artificial sweeteners, proteins and fats. For example, dairy products score low on the glycemic index (15-30), but 90-98 on the insulin index. This suggests that a diet high in protein can make it difficult to lose weight as well as a diet high in refined carbohydrate.

There is an important observation here. Proteins can also have powerful effects upon satiety or feeling full. Whey protein has a powerful incretin effect, but it also has a powerful effect upon satiety. In other words, it makes one feel full. Thus in one study whey





substantially reduced caloric intake four hours after a meal when compared to the effects of egg, turkey, and tuna.

Studies of meat have found that it is associated with weight gain, while dairy products, as long as they are a whole food with the fat, are not linked to weight gain, but actually tend to be slimming.

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URIC ACID

One of the primary reasons dairy products are slimming and red meats are not is the fact that dairy products lower uric acid levels while meat increases uric acid production. Choi observed, "Dairy consumption was inversely associated with the serum uric acid level" while "higher levels of eat and seafood consumption are associated with higher levels of uric acid..." Uric acid is an often overlooked aspect of insulin resistance and diabetes.

Zhu and associates demonstrated in 2014 that high uric acid inhibits insulin signaling and creates insulin resistance.

In 2018 King and associates identified uric acid as a direct contributor to metabolic syndrome. King wrote, "hyperuricemia has been found to independently predict the development of diabetes. Experimental studies have also shown that hyperuricemia may mediate insulin resistance, fatty

liver, and dyslipidemia in both fructose-dependent and fructose-independent models of metabolic syndrome." In other words, high uric acid in and of itself, apart from sugar intake, may lead to insulin resistance and diabetes.

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GLUCOSE AND FRUCTOSE

Fructose has a very low glycaemic index compared to glucose. For this reason fructose was and often is recommended to diabetics. Unfortunately, there is a serious drawback to excessive fructose consumption. Every cell in the body can directly metabolize glucose, but fructose is primarily metabolized in the liver. Thus the liver needs to handle only a small amount of ingested glucose, but all the fructose consumed finds its way to the liver. Unfortunately, fructose also tends to increase uric acid levels while glucose does not.

This was not much of a problem until man began consuming refined sugar in the form of sucrose which is 50% glucose and 50% fructose, and later high fructose corn syrup which has even more fructose. Excess glucose and particularly excess fructose overwhelms the liver leading to insulin resistance.

The liver stores excess glucose beyond current needs of the body cells as either glycogen, a readily available energy source, or as fat. Fat is only manufactured when the glycogen storage cabinet is full, but that cabinet is rather small.

By contrast the fat storage compartments of the body are huge. The fat can be stored in fat cells all over the body. When it becomes difficult to store fat in harmless places, the body begins to store fat around the middle of the body in the organs. This is what is called central adiposity and metabolic syndrome. This condition is associated with increased risk of cancer, heart disease, and diabetes.

Insulin resistance begins when the liver can not unload all the fat it is manufacturing and the liver itself becomes clogged with fat. Since all the fructose consumed makes its way to the liver, this sugar can contribute more to fatty liver and insulin resistance than glucose.

Excess intake of fructose, particularly from sweetened beverages, is quite damaging to the body's ability to regulate uric acid, blood glucose and insulin levels.

In 1980 Beck-Nielsen and associates gave volunteers 1,000 extra calories a day in the form of fructose. Insulin sensitivity worsened by 25% in seven days.

In 2009 Stanhope and associates fed volunteers 25% of their calories as either glucose or fructose for eight weeks. By the end of the experiment the healthy subjects consuming the load of fructose, but not glucose, had become pre-diabetic. Ordinary table sugar is composed of half glucose and half fructose. This study was designed to determine whether the glucose or fructose component of sucrose was the most harmful. Fructose proved to be the most problematic. The conclusion to be drawn is that loading the body

with fructose, because of its effect on uric acid, puts the body on the fast track to develop obesity and diabetes.





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Paracelsus was the father of modern toxicology. His famous maxim is “The dose makes the poison.” Obviously, the liver is equipped to handle the limited fructose in whole fruit and other whole foods. Unfortunately, when fructose is concentrated in foods and beverages it can be quite harmful.

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Stanhope, Kimber L., et al., Consuming fructose-sweetened, not glucose-sweetened, beverages increases visceral adiposity and lipids and decreases insulin sensitivity in overweight/obese humans, *J Clin Invest.* 2009;119(5):1322-1334.

Beck-Nielsen, H., et al., Impaired cellular insulin binding and insulin sensitivity induced by high-fructose feeding in normal subjects, *American Journal of Clinical Nutrition*, February 1980; 33(2):273-278.

SOLUTIONS

Vitamin C

Vitamin C is highly effective in reducing uric acid levels and should be supplemented in the diet of anyone with elevated levels. One group of researchers wrote, “These population-based data indicate that vitamin C intake is inversely associated with serum uric acid concentrations. These findings support a potential role of vitamin C in the prevention of hyperuricemia and gout.” Vitamin C may also decrease the risk of obe-

sity, insulin resistance and diabetes by lowering uric acid levels.

What to Eat

Insulin damage results when levels are constantly high. Foods that cause insulin spikes should be avoided. The diet should be low in sugars and refined carbohydrates. Reduce intake of foods which increase uric acid levels including meats, seafood, alcoholic beverages, and fructose laden beverages and foods.

When to Eat

Going without food breaks up the consistency of high levels of insulin restoring sensitivity to the hormone. Increasing the intervals between meals and even fasting for a day or two is a powerful means of reducing insulin levels and breaking up the consistency of exposure of the body cells to insulin.

The amount of meal restriction or fasting necessary to reduce insulin levels and reverse the degenerative process of insulin resistance will vary with genetics, the length of time a problem has existed, and basic dietary choices. There are few cases of obesity, insulin resistance, and diabetes that can not benefit from taking measures to reduce exposure to uric acid

and insulin.

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