The Glycemic Index and Glycemic Load

In order to fully appreciate the role of the glycemic index/glycemic load, it is important to briefly review how carbohydrate metabolism has come to occupy the primary role in combating the obesity epidemic and other diseases of civilization. I want to acknowledge Gary Taubes' excellent book *Good Calories, Bad Calories* from which some of this material was obtained.

Our story begins in 1863 when William Banting, a retired undertaker in London published "A Letter on Corpulence, Addressed to the Public"[1] which launched the first popular diet craze. In fact, within a year "Banting" had entered the English language as a verb meaning "to diet".

Banting was 62 and weighed over 200 lbs at 5'5". He had consulted more than 20 of the better doctors of the day and found no help in losing weight. Finally he met an ENT (William Harvey) who placed Banting on a diet. Harvey had recently heard Claude Bernard's lecture on diabetes in Paris and treated Banting successfully with animal food and vegetables that contained neither sugar nor starch – essentially the avoidance of any food that might contain sugar or starch. (in particular bread, milk, beer, sweets, potatoes and pies). Despite ingesting several glasses of wine and brandy, he lost 50 pounds without any problem over the following months.

For over 100 years, many physicians treated patients successfully with this low-carbohydrate diet. In 1961, Ancel Keys appeared on *Time* magazine[2] cover and the AHA officially alerted the nation to the dangers of dietary fat. In fact, Keys is responsible for convincing us wrongly that levels of cholesterol best predict heart disease and that dietary fat is a killer!

In 1951 Keys had an epiphany while at a conference in Rome. A physiologist from Naples claimed heart disease was not a problem in Naples. Keys found that indeed the general population of Naples was heart disease free, but the rich were not. According to Keys, rich people had more heart disease than the poor because they ate more fat. What Keys did not appreciate was that as the intake of meat and saturated fat increased, whole grain consumption decreased, but there was an increase in refined carbohydrates, white rice, flour and sugar.

Over the next decade, Keys assembled evidence to back his hypothesis including his famous "7 Countries Study". However, when all 22 countries were included in the analysis, the apparent link between fat and heart disease vanished.

In 1957, the AHA had opposed Keys' diet/heart hypothesis. Less than four years later the evidence hadn't changed, but now a 6-man committee (which included Keys), issued a new statement (just two pages long with no references) linking elevated cholesterol to the risk of heart disease.

As the 1961 *Time* article reported, Keys believed the ideal heart healthy diet should increase the percentage of carbohydrates from less than 50% to 70% and reduce fat consumption from 40 to 15%.

Friday, January 14, 1977 is when Keys' hypothesis became gospel! Senator George McGovern announced the *First Dietary Goals of the United States* – the first time that any government institution had told Americans they could improve their health by eating less fat!

McGovern had gone to Pritikin in Santa Barbara shortly before this, plus McGovern's committee was due to be downsized to a senate subcommittee, leading the staff director, Marsha Matz, to say, "We really were totally naïve, a bunch of kids who just thought – Hell, we should say something on this subject before we go out of business."

There was little or no evidence to support these dietary goals. For example, the Mr. Fit Trial (12,000 males) in 1982 shows no association with fat/heart disease ($115 million). The Lipid Research Clinic (LRC) Trial in 1984 also found no association with fat/heart disease ($150 million).

In contrast, there was a wealth of epidemiological studies - Schweitzer (Africa), Hutton (Eskimos), Fouche (S.A.), Hardlick (Indians), Williams (Fiji), Cleve & Campbell (S.A.) - when populations were exposed to Western diets (sugar, molasses, white flour, white rice), this caused an increase in "diseases of civilization" – obesity, diabetes mellitus, coronary artery disease, arthritis, hypertension, stroke, cancer, diverticulitis, gallstones, appendicitis, varicose veins, hemorrhoids, etc., all due to the increase of easily digestible carbohydrates.

Peter Cleve (and Campbell's) "Law of Adaptation" states that the refining of carbohydrates represented the most dramatic change in nutrition since the introduction of agriculture 10,000 years ago. The key is that the link between refined carbohydrates and disease had been obscured over the years by the insufficient appreciation of the correlation between carbohydrate foods in the natural state and the unnatural refined carbohydrates – treating sugars and white flour as equivalent to raw fruits, vegetables and whole meal flour.

Researchers like Joslin would measure only fat, protein and total carbohydrates, thus failing to account for effects of these refined carbohydrates. Even in Key's 7 Countries study, sugar consumption predicted coronary artery disease even more than saturated fats. Yudkin also showed that increased sugar consumption (increased insulin and T.G.) correlates better than fats with coronary artery disease. Joslin refused to believe that carbohydrates caused diabetes mellitus and led four decades of endocrinologists to believe that increased fat was the problem.

* (Hunter-Gatherers by comparison ate 22-40% Carbohydrates, 28-58% Fat and 19-35% Protein).

The greatest single change in the American diet has been increased sugar consumption – from 15 lbs/year per person in the 1820's to 100 lbs/person in the 1920's and is now over 160 lbs/year per person in 2008. In 1882, William Osler noted that only 10 cases out of 35,000 patients at Johns Hopkins were diagnosed with diabetes mellitus. Thus not all carbohydrates are created equal. This forms the basis of the glycemic index/load.

In the 1960's, Robert Stout (Queens Univ., Belfast) pre-empted Reaven's Syndrome X by suggesting that the ingestion of large quantities of refined carbohydrates leads to hyperinsulinemia and insulin resistance, and then to atherosclerosis and heart disease. In certain individuals, insulin secretion after eating carbohydrates would be disproportionately large, with carbohydrates being disposed of in fat tissues, liver and arterial walls. Obesity is produced. Stout also related that insulin stimulates the smooth muscle that lines the interior of arteries, a step in the production of hypertension and atherosclerosis.

Anything that raises blood sugar, especially refined carbohydrates, will increase the generation of oxidants and free radicals. It will increase the rate of oxidative stress and glycation and the formation and accumulation of Advanced Glycation End Products (AGEs).

This means that anything that raises blood sugar will lead to more atherosclerosis and heart disease, more vascular disorders and an accelerated pace of physical degeneration, even in those of us who never become diabetic.

In the mid-1970's Gerald Reaven initiated the study of glycemic index to test the difference between simple and complex carbohydrates. Reaven was more interested in insulin and left this research to David Jenkins, et al.

Jenkins and Wolever[3] tested 62 foods in 1981. Different individuals responded differently and variations from day to day were tremendous. The more refined the carbohydrate, the greater the blood sugar and insulin response. Anything that increases the speed of digestion (e.g., polishing rice, refining wheat, mashing potatoes, drinking juice) will increase the glycemic response.

Every complex carbohydrate must be broken down into simple sugars and will eventually enter the bloodstream as glucose, which in turn will stimulate insulin. Fiber (both soluble and insoluble) cannot be broken down into simple sugars and thus will have no effect on insulin. If a carbohydrate source (like pasta which has little fiber) is tested, we see a high insulin response as compared with broccoli (which is rich in fiber), where the insulin response will be minimal. This is why

starches and grains are considered high-density carbohydrates, fruits are medium density and vegetables are low density. It is very difficult to consume 50 grams of carbohydrates when testing broccoli (+/- 16 cups), so most glycemic index work has been done with grains, starches and some fruits.

When researchers tested different foods, they found that some simple sugars like fructose entered the bloodstream very slowly; whereas, some complex carbohydrates like potatoes entered the bloodstream faster than table sugar. Table sugar is made up of glucose and fructose. Glucose enters the bloodstream; whereas, fructose is only metabolised in the liver and has little effect on blood sugar levels.

In 1978, HFCS-55 was introduced into the market – 55% fructose and 45% glucose – identical with sucrose. By 1985, 1/2 of all sugar consumed in the U.S. was from corn sweeteners and 2/3 of this was from HFCS. This was initially perceived as healthy because it didn't increase blood sugar because it had a low glycemic index. The problem was there was a huge increase in triglycerides by the liver and increased storage of fat – "fructose-induced lipogenesis". Fructose is the most lipogenic carbohydrate. Also, fructose increases blood pressure much more than sucrose. In addition, fructose produces 10 times more X-linking of proteins and thus increases AGEs. Fructose also increases the oxidation of LDL. Thus HFCS results in the worst of both worlds – glucose increases insulin and fructose increases triglycerides.

Thus the glycemic index is a measure of the entry rates of various carbohydrate sources into the bloodstream. The faster their rate of entry, the greater the effect on insulin secretion. There are at least three factors that affect the glycemic index of a particular carbohydrate. First is the amount of fiber (especially soluble fiber), the second is the amount of protein and fat it contains and the third is the composition of the complex carbohydrate. The greater the amount of glucose, the greater the glycemic index.

The glycemic load is even more important than the glycemic index in determining the insulin output of a meal. The glycemic load is the actual amount of insulin-stimulating carbohydrates consumed, multiplied by its glycemic index.

Source	**Volume**	**GI**	**GL**
Pasta	1 cup	59	3,068
Apple	1	54	972
Broccoli	1 cup	50	150

Composition of Different Glycemic Loads

Even though the GI of each of these carbohydrates is about the same, 1 cup of pasta generates 20 times the insulin response of 1 cup of broccoli.

Remember, the more processed a food, the higher the GL. Thus by using the concept of glycemic load, it also becomes clear why consuming most of your carbohydrates from quality vegetables is key to maintaining low insulin levels.

In the 1980's Gerald Reaven[4] (Stanford) coined Syndrome X (Insulin-Resistance Syndrome/Metabolic Syndrome), which describes metabolic abnormalities common to obesity – diabetes and cardiovascular disease – all exacerbated by sugar, flour and other easily digestible carbohydrates:

- Increased triglycerides
- Increased blood pressure
- Increased fibrinogen
- Increased insulin
- Increased fat
- Increased sugar
- Increased small LDL particles
- Decreased HDL
- Increased waist
- Increased uric acid
- Increased fat
- Increased CRP

Insulin is the primary regulator of fat, cholesterol and protein metabolism. Species need time to adapt fully to changes in the environment – the introduction of diets high in sugar and refined, easily digestible carbohydrates was the most dramatic change to the body over the past 2 million years. It is probable that refined carbohydrates and sugar created such a disturbance in blood sugar and insulin that they lead to disturbances of homeostatic regulation and growth throughout the entire body.

Even though Scott Grundy stated in 2004 that metabolic syndrome was probably the cause of most heart disease in America and that this syndrome is probably caused by the excessive consumption of refined carbohydrates, his three reports to NIH, AHA and the ADA position all remain wedded to the cholesterol/heart disease dogma. Castelli in "Atherosclerosis" (1996)[5,] reviewed 26 years of the Framingham Heart Study and showed a significant overlap of LDL cholesterol in populations with and without coronary heart disease: 80% of myocardial infarction patients had similar cholesterols as those who did not have myocardial infarctions! In fact, twice as many individuals who had a lifetime total cholesterol of less than 200 mg/dl had coronary heart disease compared to those who had a total cholesterol greater than 300 mg/dl. The recent epidemiological data suggests, in fact, that a high dietary glycemic load from refined carbohydrates increases the risk of coronary heart disease independent of any known coronary risk factors[6].

In a similar fashion, the excessive consumption of these refined carbohydrates leads to obesity and diabetes. Diabetes mellitus will cost the U.S. in 2008 174 billion

dollars. Over 1 million new cases of diabetes mellitus will be diagnosed this year and nearly 70% of the U.S. population is now overweight. The problem is not the severe, marked huge, circus type of obesity but rather the 25-40 pounds put on gradually over the years – the moderate creeping obesity so common among Middle-Aged Americans.

This excess weight and obesity are caused by the singular hormonal effects of a diet rich in refined and easily digestible carbohydrates. It is the "quality" of the calories consumed that regulates weight and the "quantity" (more calories consumed than expended) that is a secondary phenomenon. There is something about carbohydrates that allows an increased consumption of food but still induces hunger. This is because the flow of fatty acids out of the cells and into the circulation depends on the level of blood sugar available and insulin levels. At a "cellular level", the body is starving and this is manifest as hunger and lethargy.[7]

Often with weight loss, fatty acids are released. Cholesterol is also released, resulting in the "transient hypercholesterolemia" of weight loss that we often see.

When investigators tested the efficiency of high-fat, carbohydrate-restricted diets, the results were remarkably constant. Every investigator reported weight loss between 1-5 pounds/week. None suffered symptoms of semi-starvation or food deprivation, excessive fatigue, irritability, mental depression or extreme hunger.

However, if we add 400 calories of fat and protein to 800 calories of proteins and fat, we have a 1,200 calorie high-fat, carbohydrate-restricted diet that will result in considerable weight loss, but if we add 400 calories of carbohydrates to 800 calories of protein and fat, we have a balanced semi-starvation diet usually prescribed for obesity. We now have a diet that will induce 40 lbs of weight loss in less than 1 in 100 instead of 1 in 2. This means that if cheaters just reach for a bagel or a couple of sodas they would now be eating a balanced semi-starvation diet with its 1% success rate.

In the 1920's, New York internist, Blake Donaldson treated over 17,000 patients with a low-carbohydrate diet with good success. Alfred Pennington, M.D. (1949) followed Donaldson with excellent results. JAMA did not however endorse such a high-protein/fat, low-carbohydrate diet from this time until 2004[8] and 2007[9] despite the numerous clinical studies presented over the years. It is time to include all the data since William Banting over 145 years ago and recognize the importance of the GI/GL in our daily practice, as recently presented in the *Journal of the American College of Cardiology* in January of 2008[10].

References

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