



Internal Starvation

by Dr. Kelly J. Gibas



Why the confusion?

I don't mean to be critical, but in my research this week, I read several articles on diabetes that were shocking! The writers were convinced, (and trying to convince me), that the high blood sugar in type II diabetics is caused by excess glucagon...**too much fat burning!**

Did you get that? Glucagon...that lovely fat burning hormone that fights for its life on the opposite axis of insulin, is being blamed for hyperglycemia! **What?**

I was forced to read the articles several times to understand their biological interpretation. The reasoning went like this...since sugar is high in the blood, and the Randle Cycle tells us that sugar will always be oxidized and utilized first, then something must be blocking sugar from being used (yes, that would be insulin resistance...right?). So, they reasoned, the problem must be excess glucagon burning fatty acids for energy & competing with the glucose!

OK, none of us are card carrying endocrinologists, but let's talk about the biology in this reasoning. Glucagon, the fat burner, is on an axis with insulin. Insulin is the dominant metabolic hormone of storage. When insulin is perpetually high, as with a type II diabetic, (hyperinsulinemia), glucagon is REPRESSED! *That's the simplistic definition of type II diabetes, ...hyperinsulinemia and perpetual lipid trapping due to constant glucose oxidation!*
What's the confusion??

Burning too much fat?

Just ask any pre-diabetic or type II diabetic, if they think their sugar problem is from excess fat burning. They will point to their stubborn fat rolls, and say **NO!**

Here's the unfortunate truth...much of our research on diabetes is antiquated. In the 1970's there was only one understanding of diabetes ...an absence of insulin.

This is true only for type I. Type I diabetes is an auto-immune disease in which the pancreas is functionally dead in regard to insulin production. People with type I diabetes (3% of all diabetics) become very thin, very quick because (and here's where there's confusion) they still produce glucagon, but not insulin. So, glucagon is not challenged by insulin. Consequently, it burns and burns fat into sugar, but there's no insulin to store it in the cells. Blood glucose levels, of an undiagnosed Type I diabetic, soar to levels as high as 500! This is a life threatening situation.

Type II diabetics don't get skinny, and they don't lack insulin! Instead, excess insulin actually causes type II diabetes; thus, glucagon is repressed.

In Type II, it's the insulin that's unchallenged!

Super sized fat cells!

If there's plenty of insulin, then why does blood sugar stay so high? One area of agreement is the idea of internal starvation. *In both Type I and Type II diabetics, a phenomenon of cellular starvation occurs. Blood sugar doesn't feed the cells.*

For the Type I diabetic, it's as simple as having no messenger of delivery. Insulin feeds the sugar into the cells to nourish the body; without it, we starve.



Type II diabetes is a little more complex. There's plenty of insulin so much, in fact, that the cells actually become resistant to it, and shut their doors (receptor sites). The cellular resistance literally "rejects" the sugar, and forces it back into the blood stream.

However, not all cells become resistant at the same rate, fat cells seem to have an unusual sensitivity to insulin! In fact, they can grow and increase their capacity to store sugar. They evolve into super sized fat cells!

Thus, most of the available sugar gets oxidized & stored within the fat cell; consequently, the cells of the muscle and tissue begin to starve!

Over fat, under fed!

This creates a paradoxical condition of being over-fed, yet undernourished!

In hyperinsulinemia, excessive amounts of calories get locked away in the fat tissue. The person, although eating to excess, acts as if he or she is starving. The high levels of insulin pool in the blood, instead of clearing out, which disrupts the regulatory system with glucagon.

In this situation, after a meal is eaten, insulin stores the sugar away primarily as fat. The insulin level fails to drop post-meal; it remains elevated. So, glucagon is suppressed...no glucagon, no fat burning!

This means no fat burning between meals to sustain glucose. This also means no satiation of hunger.

Hypoglycemia, or low blood sugar, is an early indication of this regulatory problem. Although the person has just eaten, he or she has a low blood sugar attack. The hungry cells can't use the available sugar and the fat stores can't be accessed for energy; hypoglycemia forces the person to eat again. And, the vicious cycle starts over. **This repetitive cycle leads to obesity in the presence of cellular starvation!**

2 be or not 2 be type 2

Typically, fasting glucose levels will begin to increase in the presence of insulin resistance.

During the night, the fat cells don't store as much sugar; thus, during the day glucose levels are more stable (the fat cells are oxidizing the sugar), but in the morning, after fasting, they will be elevated (over 90.)

Not everyone who is insulin resistant will become type II diabetic; it is very genetically linked. However, if the fat cell finally shuts its door to sugar (temporarily), blood sugar levels will rise to reads of 300-400. **There's nowhere left for the sugar to oxidize; it remains in the bloodstream! This is type II diabetes.**

The typical solution for this vicious cycle of fat storage & high sugar has traditionally included either calorie restriction with appetite suppression, and/or an attempt at a functional metabolic increase.

Diet pills and appetite suppressors are a multi-billion dollar business; yet, Americans continue to gain weight. This approach works to suppress hunger messengers in the brain, and thus reduce food consumption accordingly. The problem becomes when the body perceives a calorie deprivation, it compensates with a cortisol release, a retarding of the metabolic rate, and a reduction of thyroid hormone. Literally, a "code red" is initiated internally; the body switches to a conserve mode which favors fat storage.



Repartition the fuel...

Increasing the exercise load is the most common method used to increase the functional metabolic rate. But, again increasing energy expenditure through heightened exercise will ultimately increase hunger. Exercise alone has little effect on obesity.

The superior way to escape the lipid trapping cycle is to repartition the fuel consumed, so more is available for oxidation and less is placed in storage!

This can only be achieved by creating a hormonal environment in which fatty acids are mobilized and oxidized in excess of the amount stored.

Any therapy which succeeds at inducing long term fat loss works through this regulatory factor on the fat tissue.

That's BOB!

Our intention through BOB is to utilize a 10 day, lab type experiment to manipulate your food intake with the goal of forcing fatty acid oxidation. **Is it easy?** As we've shared with many of you, it's like changing the course of a huge ocean liner. It takes a lot of energy & hard work to initially slow the ship and turn it. But, once it's turned, it steams ahead at the same pace, only in the right direction!

Don't Diet; Repartition Your Fuel!