

In Part 4 of this series, I described that, contrary to popular belief, [insulin is not required for your cells to take up glucose](#). I showed that the reason an uncontrolled type I diabetic is hyperglycemic is not because sugar can't get into cells, but rather due to overproduction of glucose by the liver. I discussed how insulin acts as an important brake on many processes of the body, and without it, these processes go forth unregulated and at very high rates.

Of course, this article only showed you what happens when insulin is present versus when it is not present. What about the normal situation of a healthy person, who ingests a meal and sees a rise in blood glucose? What is happening to bring blood glucose back to normal? And what happens to a type II diabetic in this situation? This article will address these questions.

### Following the Sugar Trail

Just as metabolic tracer studies have allowed us to understand what happens when you inject insulin into an uncontrolled type I diabetic, they have also allowed us to understand what happens in healthy people as well as type II diabetics. [One classic metabolic tracer study](#) followed what happens to glucose when it is taken orally. In this study, type 2 diabetics and healthy control subjects were given 1 gram of glucose per kilogram of body weight (nearly half a gram per pound). Using metabolic tracers, the researchers determined not only where the glucose was going, but also what was happening to the liver's production of glucose. The researchers also measured insulin levels in the blood.

As you would expect, the oral glucose caused a rise in blood glucose and a corresponding rise in insulin. In the healthy people, glucose production by the liver was dramatically suppressed by the rising insulin. In fact, glucose production fell by 70-80% at 75-105 minutes after ingestion of the glucose. After 3.5 hours, glucose production was still suppressed by 50%. Thus, one of the ways that insulin helps control blood sugar after you eat a meal is by telling your liver to stop producing glucose. This makes sense; you don't want your liver producing glucose when glucose is entering the blood stream from your digestive system.

Liver glucose production was also suppressed in the type 2 diabetics. However, this suppression was impaired in the diabetics by around 40%. This is a case of insulin resistance in the liver of the diabetics; the liver is not responding to insulin as it should be (remember insulin suppresses liver glucose production), and thus produces too much glucose. In the paper, the authors stated:

*...it can be concluded that glucose overproduction is an important determinant of diabetic hyperglycemia, both in the postabsorptive state and postprandially*

Glucose production by the liver only tells us half of the story. While insulin inhibits glucose production by the liver, it also enhances the ability of your tissues to take glucose from the blood (just remember, it enhances it...it is not required for it). Cells take up glucose in two ways...by the *mass action* of glucose (i.e., the concentration gradient, where the glucose concentration in the blood is so much higher than the cells that it moves into the cells), and by the stimulation of insulin. In this study, glucose uptake into cells was impaired in the diabetics. [Since glucose movement into cells via mass action is similar between diabetics and healthy subjects](#), the impaired glucose uptake in the diabetics was due to the insulin resistance in the cells. Tissue glucose uptake was impaired by around 27% in the diabetics.

### **Insulin...More of a Traffic Cop than Storage Hormone**

It is clear from this research that the high blood sugar response of a type 2 diabetic is due to both an impaired response by the liver to insulin (so that glucose production is higher than it should be), as well as an impaired response of cells to take up glucose from the blood. However, when looking at the percentages, the impaired response of the liver is greater than the impaired response of the cells. [Other research published the same year showed equal insulin resistance in the liver and other tissues](#), although this research was done with subjects in a fasted state. In that study as well as the study we have been discussing, there was a very strong correlation between fasting hyperglycemia and liver glucose production; this indicates that, when fasted, it is overproduction of glucose by the liver which is the most important factor in causing hyperglycemia in a diabetic. It should also be noted from this research that glucose uptake in the fasted state is actually increased, not decreased, in type 2 diabetics (just as it is in an uncontrolled type I diabetic). Thus, the fasting hyperglycemia of both type 2 diabetics and uncontrolled type 1 diabetics is due to overproduction of glucose by the liver, not because "glucose can't get into cells." In a type 2 diabetic in response to a meal, glucose uptake into cells is impaired, but insulin resistance in the liver still plays a major role.

What is clear from all of this research is that insulin's main functions in the body are inhibitory, acting as a brake on many bodily processes. While insulin does stimulate the storage of glucose and other nutrients, this function is not nearly as important as the inhibitory functions. Thus, insulin should be considered more of a traffic cop rather than a storage hormone.

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