This series on insulin has stimulated numerous threads of discussion on various sites on the internet. I have seen some of these discussions and some unfounded criticisms by some individuals who appear to adhere to the "carbohydrates drive insulin which drives fat storage" mantra. However, this thinking is an example of cognitive miserliness, and I will address some of the comments of these critics here.

Some people have argued that my series on insulin only applies to healthy people and not people with glucose intolerance, obese people, or diabetics. I briefly explained how this is not true here, but either these critics did not read that section of the article or ignored it. To elaborate, some of the research I cited on protein and insulin secretion showed protein to be more insulinemic in obese people than lean people, yet we know that high protein intakes have been shown to be beneficial to helping obese people lose weight. Obviously stimulation of insulin secretion is not a problem here. Also, protein and carbohydrate tend to have a synergistic effect on insulin secretion when consumed together, creating a greater insulin response than when either one is consumed alone. Yet, we know that a high protein, moderate-to-high carbohydrate, low-fat diet has been shown to be beneficial for weight loss. We would expect such a diet to cause significant postprandial insulin secretion based on the combination of protein and carbohydrate, yet the diet causes significant weight loss. Why? Because of the effects of protein on satiety, resulting in people simply eating less. Therefore, it comes down to a matter of energy balance. The postprandial insulin secretion is irrelevant…insulin cannot trump the laws of physics.

To further illustrate how all of this data applies to all individuals and not just healthy people, let's take a look at the effects of dairy on insulin. I wrote extensively about how dairy products can be just as insulinemic, if not more insulinemic, than high carbohydrate foods, including the dreaded white bread. If augmented postprandial insulin secretion is a problem for obese people, type 2 diabetics, or glucose intolerant people, then we would expect dairy to be a problem for these populations as well. However, we know that they are not. Diets high in dairy do not impair weight loss or blood sugar control in overweight people, and they improve insulin sensitivity and attenuate weight gain in animal models. We also know that a high intake of dairy products is associated with a lower risk of metabolic syndrome and type 2 diabetes. Thus, it is obvious that augmented postprandial insulin secretion is

not the problem that some have made it out to be, even for people with health issues.

Some critics claimed that it is the combination of high postprandial insulin and high postprandial glucose that is the problem, not insulin itself. However, if this were truly the issue, then we would still expect dairy to increase risk of weight and fat gain, since most people consume dairy along with foods that elevate glucose (most people do not consume dairy alone). Yet, we know from a large number of studies that dairy does not increase weight gain risk, and decreases weight gain in animals. This is despite the fact that dairy is being consumed with glucose-elevating foods. The problem here is that the critics are taking an overly reductionistic view of insulin and body fat deposition. Since insulin enhances glucose uptake of fat cells, and since insulin also inhibits lipolysis, these critics are concluding that the combination of high insulin and high glucose will cause fat gain. However, this view is incorrect. In fact, dairy products will increase the uptake of glucose into fat cells, yet result in less fat and weight gain. Thus, things are not as simple as the critics make them out to be.

One critic pointed out how I had discussed insulin's inhibition of lipolysis, and then insinuated that this is how insulin leads to fat gain. Yet, on the same token, this same critic stated that it was the high insulin and high glucose that is the problem, not high insulin itself. This was an inconsistency in this critic's position. Obviously the latter assertion is incorrect based on what I discussed in the previous paragraph. Regarding the former insinuation, it again is an overly reductionistic view of insulin in the body. Yes, insulin inhibits lipolysis, but it only takes small elevations in insulin to do this, and this does not address what happens over a 24-hour period. It also does not address all of the other dozens of hormones and other factors simultaneously interacting on fat tissue. Not only that, but if the former insinuation were true, then we would again expect dairy products to promote fat gain in animals and humans, yet we know that they do not.

Some critics claimed that the protein/insulin secretion studies I cited in this article mainly involved liquid and not solid foods. However, only some of the studies involved liquids. Other studies (such as this one and this one) involved solid foods.

Some critics created a straw man and stated that I was claiming that protein is just as insulinemic as carbohydrate. In fact, one critic said, "*I can't believe Krieger is here trying to say that protein causes more insulin release than sugar!*" However, I only said that protein *can* be just as insulinemic as carbohydrate (can is the key word here). Certainly, when you average across all foods, carbohydrate produces the greatest insulin responses, and protein comes in second. However, when you start looking at individual foods, some protein sources can produce similar insulin responses to some carbohydrate foods (even some carbohydrate foods that create rapid rises in blood glucose). And this is not to mention the synergistic effect that protein and carbohydrate can have on insulin secretion when consumed together. Yet, studies that have combined the two have shown large amounts of weight and fat loss.

One particular critic that I saw created a huge load of straw men and other fallacies. First, this individual said, "*Unlike what Krieger says, insulin release actually first starts when you start to put the food in your mouth…It is wrong to say that insulin secretion starts only after the glucose is already in the blood.*" This individual is referring to cephalic phase insulin secretion, and the statement is a straw man because I never claimed that cephalic phase insulin secretion does not exist or that insulin secretion starts only after glucose is already in the blood. This individual then said that nobody claims that high carbohydrate diets lead to chronically high insulin levels. I am not sure what this critic has been reading as I see this claim made quite often from numerous individuals all over the internet, including low carbohydrate diet gurus. Perhaps this individual has never said this, but that does not mean that others have not. This individual went on to make the claim that "*Our argument is high insulin tends to drive fat storage*." Again, if the statement were true, then dairy products should promote fat storage, yet they do not. Also, if that statement were true, then that would mean that insulin levels should predict future weight gain. However, the vast majority of prospective studies have failed to show a relationship between either basal or postprandial insulin levels and future weight gain; in fact, some studies have shown higher insulin levels to be predictive of less weight gain in overweight people. Also, insulin levels are not predictive of weight loss. Thus, the concept that high insulin drives fat storage is a concept not supported by the scientific data.

This individual then created a strawman out of my statement of how consuming 5000 calories worth of olive oil or table sugar is not very palatable, and tried to contradict that by claiming that it's difficult to eat 5000 calories worth of steak but easy to eat 5000 calories worth of cake or similar high carbohydrate foods. Well, it's not easy to consume 5000 calories worth of steak because of the high protein content and because that would take a lot of chewing that would eventually get old. I could easily dump 5000 calories of a full-fat dressing (Caesar salad, anyone?) and it would not be difficult at all to consume that amount. In fact, I would find that easier to consume (due to the energy density) then 5000 calories worth of cake. Along these lines, in the weight loss program for which I used to do research, one client was not losing weight and swore she was following the program. Her husband eventually ratted her out and told the dietitian that she was consuming over 8 tablespoons of peanut butter per day. That's close to 1,000 calories per day that she was not reporting. Eight tablespoons is a very easy way to quickly consume a large amount of calories, despite the fact that peanut butter is mostly fat and low in carbohydrate. The ease of consuming a large number of calories of a particular food depends much more on the palatability, the protein content, the food form (solid versus liquid), the water content, the energy density, and the fiber content, then it does on the carbohydrate content.

This individual then stated that insulin "*makes you hungry because it leads to insulin swings and hypoglycemia a few hours later*." Well, insulin does not lead to insulin swings. I think that this person was referring to carbohydrate. There is often the claim that high glycemic carbohydrate will cause a rapid rise in blood sugar and insulin, followed by a crash which will induce hunger. However, this concept is not fully supported by the scientific data. High glycemic foods do not necessarily have low satiating power; in fact, one study that rated foods on their ability to create satiety found that some high glycemic carbohydrate foods, such as potatoes and white rice, were among the most satiating of all of the foods tested. Another study found a weak relationship between the glycemic response of a breakfast and energy intake at lunch, but no relationship between the insulin response to the meal and energy intake at lunch. In a meta-analysis of the relationship between blood glucose responses and appetite, no relationship was observed, and higher insulin levels were actually associated with decreased hunger.

As I stated in a previous blog post, human appetite control is highly complicated. Things are not as simple as "glucose goes up, insulin goes up, glucose then crashes and hunger increases." Even if the latter were true (which it is not given the scientific data), most people do not consume high glycemic carbohydrates by themselves. They generally consume them with other foods, which dramatically changes the blood sugar and insulin responses.

This critic goes on to say, "*Type 2 diabetics are still often hungry even though their blood sugars are constantly high. If insulin is so good at satiating, then why do these diabetics still feel hungry*?" Type 2 diabetics have insulin resistance in the brain, which disrupts insulin's ability to signal the brain to reduce food intake.

This critic also states that nobody said that "*carbohydrates are singularly responsible for driving insulin*." Actually Gary Taubes said it in his book, word for word. She goes onto say that, regarding one particular study I referenced, I claimed that 75 grams of carbohydrate was "low carbohydrate." I never made that claim; the study labeled it as such. I clearly addressed this with this paragraph:

> *Some people might argue that the "low-carb" condition wasn't really low carb because it had 75 grams of carbohydrate. But that's not the point. The point is that the high-carb condition had nearly TWICE as much carbohydrate, along with a HIGHER glucose response, yet insulin secretion was slightly LOWER. The protein was just as powerful at stimulating insulin as the carbohydrate.*

This critic then goes on to my comments regarding how some in the low-carbohydrate community claim that the insulin response to protein is due to the gluconeogenesis from the protein. She states, "*Then he says that 'some' might say it is due to gluconeogenesis. Really? WHo would need to argue that when their drink has CARBOHYDRATES in it already!"* This critic completely missed the point, and needs to look at the graphs of the blood insulin and glucose responses. First, the carbohydrate in the drink was quite low (only 11 grams) and did not cause much change in blood glucose. However, there was a very large insulin response. *This means that the insulin response was not due to the blood glucose response*. This completely contradicts the claims of some low-carbohydrate advocates that the insulin response from protein is due to the protein being converted to glucose, which

would then drive up insulin. I then supported this further by citing research showing that amino acids directly stimulate the pancreas to produce insulin.

This individual goes on to state, "*The weirder thing is this study actually shows what we argue, ie that obese people have higher insulin response to the same meal compared to nonobese. This will be true of both protein AND carbs but he only talks about protein here. Thus supporting our argument that insulin drives weight gain*." This individual is committing the *cum hoc, ergo propter hoc* ("with this, therefore because of this") fallacy. The fact that obese people present with high levels of insulin does not mean that high insulin causes the obesity; they are simply correlated. In fact, not all obese people have high insulin levels. High insulin is not the driver of obesity; rather, it is the result of obesity and the insulin resistance that often accompanies it. Insulin resistance is causing the high insulin in obese people (the high insulin is the body's way to compensate for insulin resistance).

I will continue to address other criticisms that I have seen next week, along with continuing last week's article on how insulin regulates blood sugar.