Last week I addressed some criticisms of this series that I had seen at various places on the internet. This article represents some of my final thoughts on insulin and body weight regulation.

Are You a Cognitive Miser?

Consider this problem. Jack is looking at Anne, but Anne is looking at George. Jack is married, but George is not. Is a married person looking at an unmarried person?

1. Yes
2. No
3. Cannot be determined

This problem is from an article in Scientific American about *dysrationalia*, a concept that describes where intelligent people think irrationally. The article also discusses a concept known as *cognitive miserliness*, which refers to people's tendencies to take short cuts in their thinking. These short cuts can then often lead to incorrect conclusions since the conclusions are based on incomplete information. In fact, cognitive miserliness can often lead to the phenomena of dysrationalia described in the article.

Have you determined your answer for the above problem yet? About 80% of people will choose the third option ("Cannot be determined"). However, that is not the correct answer. The correct answer is the first option ("Yes"). The puzzle doesn't say whether Anne is married or not; either she is, or she isn't. If Anne is married, she's looking at George, so the answer is "yes". If Anne is not married, Jack is still looking at her, so the answer is still "yes". Most people get this problem wrong because they don't take the time to go through all of the possibilities. Instead, they choose the easiest inference.

The Carbohydrate/Insulin Hypothesis: An Example of Cognitive Miserliness

The "*carbohydrates drive insulin which drives fat storage*" mantra is a perfect example of cognitive miserliness. It is a concept that is based on incomplete information regarding insulin, carbohydrate, and body weight regulation. Many people are drawn to this hypothesis because of its simplicity. However, its simplicity is exactly what makes it incorrect, because it does not address all the available

data. It is a shortcut in thinking, based on a narrow view of insulin's effects on lipolysis and glucose.

One of the big problems with the carbohydrate/insulin hypothesis of obesity is that it does not address what happens over a 24 hour period. I briefly alluded to this in the first part of this series, and will expand upon it here.

Let's take alcohol as an example. Alcohol, when ingested, is known to suppress fat burning. This is because the metabolism of alcohol takes precedence over the metabolism of protein, carbohydrate, and fat. If one just looks at the suppression of fat burning, then one might predict that alcohol will cause weight and fat gain.

However, it is erroneous to make this prediction, because you need to address what happens over a 24 hour period, and what is happening in regards to overall energy balance. One study looked at the effects of alcohol on fat storage. The researchers compared two conditions. In one condition, alcohol was consumed in addition to meals, so that the subjects were consuming more calories than they were expending. In another condition, alcohol replaced some carbohydrate in the meals, so that the subjects remained in energy balance.

In both conditions, alcohol suppressed fat burning over the 6 hours that it was actively metabolized. When the subjects consumed alcohol in addition to their meals, fat balance over the day was positive, meaning the subjects gained fat. However, when the subjects consumed alcohol in place of some carbohydrate, there was no increase in fat balance over a 24-hour period. Although the alcohol suppressed fat burning over the 6 hours that it was metabolized, fat burning increased over the remainder of the day. This canceled out the suppressive effect of alcohol on fat burning. The authors stated,

> *Short term studies that fail to account for later readjustments of macronutrient balance can be misleading. We conclude that alcohol has a fat-sparing effect similar to that of carbohydrate and will only cause fat gain when consumed in excess of normal energy needs.*

Just like with alcohol, one cannot simply look at the fact that carbohydrate ingestion

will suppress fat burning during the time that carbohydrate is metabolized. Unfortunately, proponents of the "carbohydrates drive insulin which drives fat storage" mantra do exactly that. You have to consider the big picture and what is happening over a 24 hour period.

The other reasons why the carbohydrate/insulin hypothesis is an example of cognitive miserliness is due to all of the factors discussed in this series. There are numerous scientific observations that are inconsistent with the hypothesis. Also, the hypothesis, with its narrow focus on insulin, fails to address the myriad of other hormones and factors that are all interacting simultaneously to regulate body weight.

The Finale

The bottom line is that insulin does not deserve the bad reputation that it has been given, and the "carbohydrates drive insulin which drives fat storage" mantra is wrong. To summarize:

- Insulin suppresses appetite; it does not increase it
- A high carbohydrate diet does not cause chronically high insulin levels
- Protein is insulinemic, and in certain cases, can be just as insulinemic as carbohydrate
- Contrary to popular belief, glucagon does not "cancel out" the suppression of lipolysis by insulin when protein is ingested
- The insulinemic effects of protein are due to a direct stimulatory effect on the pancreas, and not because the protein is converted to glucose
- The combination of protein and carbohydrate can produce greater insulin secretion than either one alone, yet high protein, moderate-to-high carbohydrate diets are very effective for weight loss
- Very high carbohydrate diets have been demonstrated to produce weight loss when people are in an energy deficit
- Dairy is extremely insulinemic, just as insulinemic as white bread, yet does not promote weight gain in the absence of an energy surplus. This is supported by a very large number of studies, including animal studies, observational studies, and randomized controlled trials.

- Insulin is not required for fat storage
- Insulin levels are not predictive of weight gain or weight loss in the majority of prospective studies
- Exenatide restores rapid phase insulin release in diabetics, yet causes weight loss
- The effects of insulin injection cannot be compared to normal physiological insulin release, since amylin is co-secreted with insulin from the pancreas
- Insulin mainly functions as an inhibitory hormone rather than a storage hormone, acting as a brake on many important physiologic processes
- A type I diabetic without insulin becomes hyperglycemic because of overproduction of glucose by the liver, not because insulin can't get into cells
- Insulin enhances the uptake of glucose into cells, but is not required for it
- Insulin regulates blood sugar after a meal both by stopping the liver from producing glucose and enhancing glucose uptake into cells.
- In a fasted state, insulin regulates blood sugar by controlling glucose production of the liver, not by affecting the uptake of glucose into cells
- You cannot simply look at the temporary effects of insulin on lipolysis and glucose storage. You have to address what is happening over a 24-hour period; body fat will not increase if there is no overall energy surplus.

Don't be a cognitive miser. Insulin is not the bad guy.