



Obesity: The Genes or the Jeans?

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OBESITY: THE GENES OR THE JEANS?

No, weight loss is not about eating less and exercising more; obesity is about the hormone insulin. As biochemist Gary Taubes explains in his book *Good Calories/Bad Calories*, “obesity is always related to [the dysregulation of] blood insulin in both humans and animals. **There are no exceptions.**” Insulin is the hormonal conductor of the metabolic symphony; insulin facilitates and initiates the entire melodrama of fat storage. It’s not necessarily the level of insulin circulating in the blood which determines this melodious direction of fat (to storage or burning); but rather, is the responsiveness of the ‘orchestra of organs’ to the conductor’s cue. Are the muscles, liver and brain sensitive to insulin’s message or have they down-regulated their receptors becoming functionally deaf to insulin? The loss of sensitivity to insulin, known as insulin resistance, pre-diabetes or metabolic syndrome, **causes glucose to be rejected by the muscles/organs and store in the fat cells at abnormally high levels.** Thus, people suffering from insulin resistance are typically *overfed, yet undernourished*; the primary defect in obesity is energy distribution, not caloric excess (Gary Taubes, *Why We Get Fat*). Cellular resistance to insulin causes obesity (especially visceral storage around the abdomen), lethargy/fatigue and subsequent hormonal disorders like low thyroid, estrogen dominance and adrenal fatigue.

Insulin is a **survival hormone** secreted from the pancreas. It is responsible for facilitating the transport of glucose from the blood into the cells for energy; without insulin, it is death through starvation. When the brain detects an elevation of blood sugar from dietary carbohydrates... a Snickers bar, whole grain bread, a banana or pretzel sticks... it directs the pancreas to release insulin much like ‘*blood sugar police.*’ The secreted insulin ushers the glucose out of the blood and into the cellular receptors for energy. Due to this survival action, insulin has an evolutionary dominance over the other hormones within the endocrine network: human growth hormone, estrogen, testosterone, progesterone, adrenaline, thyroid hormones T4 & T3, and MOST importantly (as far as weight loss is considered)... *glucagon!* In fact, insulin signals a ‘feeding state’ which alerts the hypothalamus to trigger a sympathetic response which, among other things, *stops the parasympathetic action of fat burning!*

Glucagon is insulin’s axis hormone, it opposes/challenges the action of insulin; glucagon mediates fat burning in the adipose tissue. It is the hormone of fasting, insulin is the hormone of feasting/storing. Between meals (a rarity for most Americans ☺), blood insulin and leptin levels, molecule in the fat cell, drop telling the brain to release glucagon from the pancreas to sustain blood sugar through fat burning. Ideally, a person should be able to fast at least 4-6 hours between meals without experiencing low blood sugar due to glucagon’s fat burning action. Glucagon works together with hormone sensitive lipase (HSL) to dismantle stored fat (triglycerides) into free fatty acids to burn for energy; this mediation of fat burning **only works in a fasted state or in the absence of a carbohydrate/sugar load**, and most importantly, fat burns when insulin levels are low. Glucagon and insulin take metabolic ‘turns’ in the blood; but insulin is definitely the dominant hormone. In a healthy situation when cells are sensitive to insulin, the body is designed to oxidize sugar and fat (free fatty acids) cyclically... store fat, burn fat, store fat, burn fat; in homeostasis, this is how the body regulates normal levels of stored fat (17% for men, 22% for women). Between meals, insulin and glucose levels fall, and glucagon is secreted to burn fat and keep blood sugar within a narrow physiological range. *But, Insulin resistance changes everything.*

When the cells (especially muscles cells) down-regulate to insulin’s cue, they begin to reject, or lose sensitivity to the incoming glucose; when insulin’s message isn’t heard by the cells, the pancreas releases more and more insulin to get the message across. It’s a lot like telling kids to clean their room; the message gets ignored and finally it must be screamed, ‘clean your room!’ Unfortunately, the muscle cells are growing increasingly ‘deaf’ to insulin, so most of the blood sugar is ushered into the cells that are still listening to insulin ... ***THE FAT CELLS!***



The fat cells are usually the last to grow resistant since fat represents metabolic ‘protection’; when adipose tissue becomes resistant to insulin, it is diagnosed as type II diabetes. This resistance initiates a cascade of hormonal chaos within other regulatory feedback loops. The molecule leptin, made in the fat cells, is a good example. Normally, leptin rises with feasting and falls in fasting. The fall of leptin signals to the brain...‘time to burn fat!’ In insulin resistance, leptin levels remain elevated. When leptin stays high, the brain thinks the person is eating, so glucagon is inhibited. Then, since glucagon isn’t released, blood sugar plummets, fat can’t be burned for energy and the person is forced to eat between meals, leading to more insulin resistance. Obese people commonly struggle with ‘leptin resistance’ as well as insulin resistance; however, insulin controls the rise and fall of leptin.

Cellular insulin resistance is inherited. In fact, according to a 2011 report entitled, *Conquering the Diabetes Epidemic*, 1/3 of all Americans are either diabetic or pre-diabetic as assessed by a fasting glucose of >100 (pre) or >125 type II diabetic. Twenty-five years ago, type II diabetes was considered ‘old age diabetes’ because the average person contracting the disease was over 70 years of age. Now, America’s elementary schools are filled with children taking oral diabetic medication. What happened? Dr. Mercola, a leading expert on insulin resistance, cites a startling statistic in his recent article, *Insulin and Its Metabolic Effects*, “insulin sensitivity starts to be determined the moment the sperm combines with the egg. If a pregnant woman eats a high-carb diet which turns to sugar, animal studies have shown that the fetus will become more insulin resistant. Worse yet, researchers have used sophisticated measurements and found that if that fetus happens to be a female, the eggs of that fetus are more insulin resistant.” Mercola goes on to explain that insulin resistance is not a genetic disease; it is a genetic predisposition. Two toddlers eat the same Goldfish crackers and fruit snacks, one grows chubby, the other stays lean. Resistance to insulin begins in the womb, especially if Mom and Dad are overweight.

Is lasting weight control about suppressing the appetite through Rx medications such as phentermine, phendimetrazine, or sibutramine, which work to alter appetite control in the brain, or is an uncontrolled appetite evidence of insulin resistance? Shouldn’t weight management focus on up-regulating the muscle cells to utilize glucose and insulin, thus addressing the root of the problem? If down-regulation was mediated, less glucose would be stored as fat and more could be used for energy. In Europe, preventative health care clinicians have been using a drug called Metformin for diabetes prevention and insulin up-regulating, for over 40 years. Metformin is a biguanide drug; its action is to increase the uptake of glucose by the muscles. “All in all, Metformin decreases insulin resistance and improves insulin sensitivity, thereby helping the insulin your body makes work more effectively” (Dr. Susan Sloane, *Metformin: Improving Insulin Sensitivity*, 2011). As muscles begin to sensitize to insulin’s cue, appetite is naturally suppressed as the defect in energy distribution is corrected (Gary Taubes, *Good Calories/Bad Calories*.)