The skinny on obesity

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The “Skinny” on Obesity [edit]

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Introduction [edit]

It’s no secret that America weighs 25 pounds more than it did just 30 years ago. Just go to the mall or to Disneyworld, and see for yourself. And those extra pounds have led to skyrocketing increases in heart disease, hypertension, Type 2 diabetes, and many other adult conditions, even in children. Indeed, nowhere has this weight burden been more obvious than in our nation’s youth. We have 10% of preschoolers, 20% of elementary school children, and 25% of teenagers tipping the scales.

The childhood obesity epidemic has disastrous consequences. Twenty years ago, no one ever heard of Type 2 diabetes in children, and now 1 out of every 4 children with diabetes is Type 2. One-third of adult Americans by the year 2030 are predicted to be diabetic, and this current generation is the first one in history that is predicted to die younger than their parents. Extra-large coffins are selling like hotcakes.

So what happened? [edit]

It’s often said that obesity is the ultimate interaction between genes and the environment. Well, the gene pool hasn’t changed in the last 30

years, but the environment has. We all eat more, and exercise less; this isn't news.

But what is news is that our energy intake and expenditure are biochemically determined.

Children surviving brain tumors often become obese. This may be due to the tumor itself, the surgery, or the radiation. Despite attempts at diet and exercise, the weight just keeps piling on.

In 1977 Dr. George Bray of UCLA hospitalized 8 children with "hypothalamic obesity" and fed them 500 calories a day for a month. Their weight INCREASED! Not only does weight increase, but such patients invariably feel awful. Their energy is sapped; they exhibit no spontaneous activity. Indeed, this is their parents' biggest complaint.

The reason for the persistent weight gain is that the hypothalamus (the brain region which regulates hormones) can no longer transduce the leptin signal. Leptin is the adipocyte hormone that tells your hypothalamus that you have enough energy stored to burn energy at a normal rate. If your leptin level declines (due to dieting), or if your hypothalamus can't see your leptin (because the hypothalamus is damaged, a phenomenon called *leptin resistance*), then the brain interprets this state as one of starvation.

In the starved state, two things happen:

1. the sympathetic nervous system (which controls your heart rate, blood pressure, and muscle metabolism) slows energy expenditure in order to conserve energy (which makes you feel bad),
2. the vagus nerve (which controls your stomach, intestine, and pancreas) directs the body to promote extra energy storage into fat.

Normally, the increase in fat deposition increases the leptin level, but because of the hypothalamic damage, the leptin signal can't stop the starvation response. The result is a never-ending syndrome of energy storage along with general feelings of malaise.

We previously demonstrated that one of the reasons for the constant energy storage was the vagus nerve constantly stimulating the pancreas to produce extra insulin. Insulin is the energy-storage hormone, driving both glucose and free fatty acids into adipose tissue. We then showed that suppressing insulin secretion back toward normal using the drug octreotide caused patients to lose weight, eat less, exercise spontaneously, and feel better. In other words, we were able to override their leptin resistance by dropping their insulin.

So what does this have to do with standard obesity?

[edit]

Otherwise normal obese people make lots of leptin; but the hypothalamus also can't see it. In other words, they also have leptin resistance; but in this case, it is not due to brain damage from a tumor, surgery, or radiation. If they try to lose weight, their hypothalamus senses a state of starvation, and they also reduce their sympathetic tone and increase their vagal tone in order to increase insulin secretion, to store more energy and maintain the obese state.

What causes their leptin resistance?

[edit]

We wondered whether this phenomenon of increased insulin secretion could be at the root of standard adult obesity. We treated 44 obese adults with octreotide. Of the 44, 8 lost more than 10% of their body weight. Their carbohydrate intake dropped by 60% (although their protein and fat intake stayed the same). Most importantly, they felt better and started exercising. So what was different about these 8? They were the ones with increased vagal-mediated insulin secretion, and they were the ones that demonstrated insulin suppression with octreotide.

Insulin is at the core of this problem of leptin resistance, because insulin antagonizes the leptin signal in the hypothalamus. This phenomenon is essential to the survival of the species, as it temporarily blocks leptin in order to promote gain weight during puberty and pregnancy. But at other times, high insulin is maladaptive, as it causes the starvation response at a higher weight instead of a lower weight. In addition, insulin also fosters continued reward of food, which makes you eat more.

How does this work? [edit]

A thin, insulin sensitive, 13 year old might consume a daily allotment of 2000 kcal, and burn 2000 kcal in order to remain weight-stable, with a stable leptin level, and feel good. However, if that same 13 year old became hyperinsulinemic and/or insulin resistant, perhaps as many as 250 kcal of his daily allotment would be shunted to storage in adipose tissue, promoting a persistent weight gain. Due to the obligate energy storage, the child now only has 1750 kcal per day to burn.

The hyperinsulinemia also results in a lower level of leptin signaling in the hypothalamus, conveying a central signal of energy insufficiency. The remaining calories available are lower than his energy expenditure; the hypothalamus would sense starvation.

Through decreased adrenergic tone, he would reduce his physical activity, resulting in decreased quality of life; and through increased vagal tone, he would increase caloric intake and insulin secretion, but now at a much higher level.

Furthermore, the promotion of reward by hyperinsulinemia would permit persistent increased caloric intake, Thus, the vicious cycle of gluttony, sloth, and obesity is continued.

So, what can we do about obesity? [edit]

Get the insulin down! The best way to get the insulin down is to not let it go up. What makes insulin go up?

1. Sugar [edit]

Where's the sugar in our diets? Well everywhere, but especially in soft drinks and fruit juice. Fructose is the main sugar in these drinks. Due to its unusual biochemical properties, fructose has been shown to induce insulin resistance, contributing to this phenomenon of leptin resistance. Water is the perfect beverage; it has everything you need, and nothing you don't.

> **Simple rule:**
> *if it's a liquid, look at the calories. 6 or more, leave at the store!*
> Milk is the only exception.

2. Starch [edit]

Starch is just polymerized sugar, and it turns into sugar in the stomach, which skyrockets your insulin. Although the Atkins diet became famous because it reduces insulin secretion by removing dietary carbohydrate, this is an untenable diet for children long-term.

There is an easier way. Fiber. Fiber is the antidote to starch. Fiber slows sugar absorption from the gut into the bloodstream; thus the glucose rise is slower, so the insulin rise is lower. Unfortunately, fiber doesn't freeze; when you thaw it, it gets gunky. So fast food has all the fiber processed out of it, which makes it doubly bad.

Dr. Ludwig at Children's Hospital Boston showed that when you go from one fast food restaurant meal to two per week, your risk of obesity goes up by 60%. The answer here is: Eat your carbohydrate with fiber.

Simple rule:
if it's a solid, look at the fiber. 3 gm or more is good.
A corollary to this is:
Fast food makes insulin rise fast.
A second corollary is:
Eat the fruit, don't drink the juice .

3. Fiber [edit]

Fiber also has another advantage. It moves food through the gut faster. This leads to faster satiety, which reduces the number of second portions. Carbohydrate without fiber is constipating; remember the BRAT diet (bananas, rice, applesauce, toast) for diarrhea? Fiber makes the satiety signal occur faster.

Simple rule:
Wait 20 minutes for second portions.

4. Exercise [edit]

Finally, the best way to lower insulin is exercise. Exercise improves insulin resistance, but the beneficial effects are short lived, so it has to be consistent. The National Academy of Sciences advocates 10,000 steps (5 miles) a day to stay healthy. The average child only gets 1,000 steps a day. 5 miles is the length of a golf course. Golfers are thin because they walk a golf course every day.

But of course, getting kids to exercise is the hardest thing of all to do. Why? They're watching 2-3 hours of TV a day, and they say they have no time to exercise. Why can't they walk while they're watching, like at the health club? The average walking speed is 3 miles/hour, so it would take 1 hour 40 minutes to walk the 5 miles. That's less time than the TV is on.

Not-so-simple rule:
If you're watching TV, you should be walking on a treadmill.
Children have to find the time to exercise. They can't create time, but they can sure recapture wasted time.

These four precepts have been shown to work if your insulin is in the lower range. Some children will still not respond to these lifestyle alterations, and they deserve a medical evaluation. But in order for America to win the battle of obesity, we're going to have to undo the damage that the food industry, the urban planners, and the media have done to our society. For more information about this topic, check our research paper or click the link of Marketing Companies

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