How to Lose Weight
Weight Loss Guide Part II
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Part I – Hormonal Obesity
The Carbohydrate-Insulin Hypothesis is Wrong – Chapter 1

**Obesity Facts**

1. Body acts as a thermostat, not a caloric scale (Body Set Weight)
2. Obesity is a *hormonal*, not a *caloric* imbalance
3. Insulin and cortisol are major hormonal drivers of weight gain
4. Exercise is not important for weight loss

We can give people insulin and cortisol and they will gain weight. When we take it away, they will lose the weight. For the moment we will focus on insulin, since that tends to be the bigger player in obesity.

Once we accept the fact that insulin causes obesity, then we will want to know what caused the increased insulin levels that caused obesity in the first place. The most obvious candidate is the fattening carbohydrates. These are the highly refined grains and sugars.

Because these carbohydrates are highly refined, they tend to raise blood sugars and blood insulin levels quickly. This will tend to cause weight gain and obesity. This is known as the Carbohydrate-Insulin Hypothesis (CIH), and is the basis of the Atkins diet and many other low carbohydrate (Dukan) and very-low-carbohydrate diets (ketogenic diet).

However, it quickly becomes clear that this hypothesis must be either wrong or incomplete. The most obvious problem is that of the Asian rice eaters of the 1990’s. East Asians tend to eat a lot of rice compared to North Americans. Most of their rice consumption is white rice, which is a refined carbohydrate.

If they are eating so much refined carbohydrates, then you would expect that the Chinese in the
1990’s would be extremely obese. The problem, of course, was that this was not so. There were very low obesity rates in East Asia until more recently in the 2000’s when their diet became much more Westernized. Poor fools.

It is useful to study these historic patterns because many diets have become much more Westernized during this current period of globalization.

Let’s look at this study:


Using 24-hour dietary recall and 24 hour urine samples they compared the diets of 4 groups of people from the USA, UK, China and Japan.

The total carbohydrate intake in China was far in excess of the other nations. If the CIH was correct, this would predict that obesity in China would be the highest of the group. This is not true.

There is clearly something else going on here. While the total carbohydrate intake was very high, the intake of sugar was very low compared to the rest of the world. There seems to be something specific to sugar that may be much more obesogenic than other carbohydrates.

Japan is also very interesting. While total carbohydrate intake is not higher, sugar intake is also lower than either the US or the UK. Japan also had very low levels of obesity until recently.

There is one other major difference in refined carbohydrate intake. East Asians tend to take most of their carbohydrates as rice whereas Western societies tend to take their carbohydrate as refined wheat and corn products.

It is possible that rice is less obesogenic than wheat. There are some who also postulate that wheat that we eat currently is far different from the original wheat that was grown. One of the New York Times bestsellers is a book called Wheat Belly that suggests this is the major problem. It is certainly true that 99% of commercially grown wheat is dwarf and semi-dwarf varieties. The health implications of changing the wheat are unknown, so it remains a very real possibility.
We will unravel the mystery of the 1990’s Thin Chinese Rice Eater in due course. The answer is far more nuanced than we can cover in this post. However, what we can say at this point is that the CIH in its current state is not correct. It is an incomplete theory.

In a similar light, we can point to a multitude of primitive societies that eat predominantly carbohydrate diets. The Kitavans, for instance, studied by Stefan Lindeberg ate a high carbohydrate diet but had a very low serum insulin and virtually no obesity. Despite a 70% carbohydrate (unrefined) intake, the Kitavans had serum insulin levels below the 5th percentile of the Swedes. So, it seems that low insulin levels associates with low obesity rates, it is not at all clear that high carbohydrate intake is the primary cause of high insulin levels.

Similarly, the Okinawans eat plenty of sweet potatoe (carbohydrate). Yet they are one of the longest lived peoples in the world and have historically had very little obesity.

The CIH reflects modern medical knowledge circa 1850 when Banting published “A Letter on Corpulence”. While it is not entirely wrong, it is not entirely right either. This has led many to abandon this theory rather than try to reconcile it with the known facts.

The link from insulin to obesity seems very solid. Giving insulin to people causes weight gain and taking it away leads to weight loss. However, it is the link from carbohydrates to insulin that is incomplete. There are many things that can lead to increase in insulin, as well as many things that can lead to a decrease in insulin.

The carbohydrate-insulin hypothesis is not so much wrong as incomplete. The notion that carbohydrates are the only driver of insulin is incorrect. We need to know what increases and decreases insulin.

In order to develop a more complete understanding of the causes of obesity, we will need to discuss the phenomenon known as insulin resistance. We will need to look at the importance of meal timing. We will need to look at the traditional role of fasting. We will discuss diabetes in detail.

We will need to explore the role of fiber. We will discuss resistant starch. We will need to explore the role of vinegar. We will look at fructose
and sugars in more detail. We will need to look at the role of wheat specifically.

Strap on your seatbelts – we will be taking off shortly.
How Insulin Works – Chapter 2

It may be helpful to review normal insulin action, since we will be talking about it – a lot. Insulin is a hormone that is released in response to food. Certain foods, particularly the refined carbohydrates tend to increase the insulin levels more than others. However, protein will also increase insulin levels as well. Dietary fats, on the other hand, tend to have the least effect of increasing blood sugars and insulin levels.

As we eat, blood sugars go up and we release insulin. When we eat a meal, we are ingesting much more food energy than we can use immediately. Insulin tells our body to move this flood of glucose out of the bloodstream and into storage for use later on. One of the ways our body can store this sugar is glycogen in the liver. Our body can convert glucose to glycogen to glucose and back again quite easily.

But there is a limited amount of glycogen that can be stored in the liver. After that, any excess carbohydrates will be turned into fat. Fat is harder to access but in unlimited supply. In effect, insulin is one of the prime hormones promoting fat storage. There are several different areas where fat accumulates – subcutaneous (under the skin) and visceral (organ) fat. It is the visceral fat that tends to be more of a problem.

Glycogen is like your wallet. You put money in and take money out all the time. It is easily accessed but you can only hold a limited amount. Fat is like your bank account. It is harder to get access to that money, but there is an unlimited storage space.

This, of course, explains partially the difficulty in losing the fat accumulated over the years. Since access to this space is limited, it can be difficult to burn fat. To get at the money in the Bank, you need to deplete the Wallet...
first. You can only access the Fat when the Glycogen is depleted. But you don’t like an empty wallet even if your bank is full. You also don’t like an empty glycogen tank even if the fat bank is full.

This fat storage process is entirely normal. We eat. Insulin goes up. Insulin stores sugar as glycogen in the liver. Insulin also turns on fat production in the liver. The technical term is hepatic de novo lipogenesis (DNL).

This newly synthesized fat has 3 possible fates. It can be oxidized for energy. Since insulin is signalling that we already have lots of energy, this doesn’t happen.

Second, the liver can export this fat elsewhere in the body. It can be sent to other organs (pancreas), be stored as visceral fat (around organs) or subcutaneous (underneath the skin). Third, the fat can be stored in the liver.

Insulin is also responsible for turning off the conversion of glycogen to sugar. In technical terms, insulin suppresses gluconeogenesis (new glucose production in the liver). Since insulin tells the body to store sugar, you want to turn off the sugar burning process. Makes perfect sense.

After you have finished digesting the meal, then blood sugars start to drop and insulin levels also drop. This is the absense of eating or ‘fasting’. This signals the body to start releasing the stored sugar (glycogen in the liver) into the bloodstream for use by muscle, brain and other organs. This is called glycogenolysis (burning of glycogen) and happens most nights (assuming you don’t eat at night).

This fat storage process now happens in the reverse. That is normal, since we have not eaten and insulin is only released when eating. Stored sugar is taken from the liver and sent out to the body to be used for energy. Fat is burned to release energy and sent out to the body if there is not enough stored sugar.
So this is what is happening every single day. Normally, this is a well designed system that keeps itself in check.

Eat – store sugar and fat.

Don’t eat – burn sugar and fat.

Supposing we eat 3 meals a day from 7am to 7pm and fast from 7pm to 7am, you can see that we have balanced 12 hours of fasting with 12 hours of feeding. Well-oiled machine.

You can easily see, however, that under conditions of sustained insulin excess, that you will tend to gain fat. If insulin levels are always high, our bodies behave like we are always eating. Store sugar and fat.

In response to the high insulin levels, we turn on the machinery to increase glycogen stores. After this fills up, then we start to move some of this excess energy to fat stores. If there is an imbalance between the feeding and fasting periods, this will lead to increased weight, increased fat and voila – obesity.

But the problem is that eating does not lead to sustained high insulin levels. Let’s assume we eat 3 meals per day with no snacks.

Supposing we eat breakfast at 7 am and dinner at 7 pm. That means that we have balanced 12 hours of eating with 12 hours of fasting. There are periods of increased insulin balanced by decreased periods of insulin. Even if
we are to increase the peaks of insulin by eating lots of refined carbohydrates, it would still be balanced by the fasting period.

In the 1950’s and 60’s for instance, people ate plenty of pasta and white bread. There were the same oreo cookies and Kit Kat chocolate bars. Sugar intake was still high and people did not drink Diet Coke. So Americans were eating plenty of fattening carbohydrates but did not have the same problem with obesity.

But we always forget that proper food choices involves two main questions. The first question we think about constantly – What to Eat?? Should we eat high carb, low carb, high fat, low fat, high protein, low protein. Should we eat whole grain, more fibre, etc.

The question we always forget is When to Eat. Meal timing turns out to play a very important role in the development of obesity as well. In the above example, we are eating 3 meals per day with no snacks. But that is increasing rare. More and more, people are being told to eat 6 times a day.

In this situation, we are constantly stimulating insulin. Insulin is the hormone of eating, so our sugar storage and fat storage machinery is revved up. While the peaks of insulin are not as high, we have more persistent stimulation of insulin. We have lost the balance between the fed state (insulin dominant) and the fasted state (insulin deficient).

In the previous example we have 12 hours of feeding balanced by 12 hours of fasting. This is what Moms everywhere used to say. 3 meals a day, no snacks. Snacking would ruin your dinner. And this 1950’s and 1960’s advice was associated with low levels of obesity. Now, we eat all the time and anywhere we want.

What we now have is 16-18 hours of feeding and 6-8 hours of fasting. We are told to eat all the time. Eat 3 meals a day with 3 snacks in between. Look at a typical schedule at school. Eat breakfast. Mid-morning snack (for that long period between breakfast and dinner). Lunch. After-school snack. Dinner. Snack in between soccer periods. Snack at bedtime. That’s a whopping 7 times a day every day.

This is our current advice. And this dietary pattern is associated with much higher levels of obesity.
Insulin Resistance – Chapter 3

Insulin is a major driver of obesity. But what drives insulin? In some cases, certain foods drive the increase in insulin. But there are clearly some time dependent factors about obesity that are unaccounted for. What am I talking about?

Consider this. There are people who have been overweight or obese for their entire lives. For them, it is extremely difficult to lose weight on a permanent basis. On the other hand, there are people who have only recently gained weight. For them, it seems that it is much, much easier to lose the weight. Why is that?

Most conventional theories of obesity do not allow for these effects. Consider the Caloric Reduction as Primary (CRaP) theory, also known as calories in calories out. According to this flawed theory, there should be no difference between those who have had obesity for 1 month versus those who have had it for 1 decade. Having obesity for a long time should make no difference. But anybody who has tried to lose weight or has known somebody trying to lose weight knows that duration of obesity plays a very large role.

Consider also the carbohydrate-insulin hypothesis. According to this theory, there is also no allowance for duration of obesity. It should make no difference. As long as you reduce carbohydrates, you should lose weight. But everybody who has struggled with their weight knows that it matters a lot whether you have been obese for months or for years.

Childhood obesity shows the same problem. Those obese as children tend to be obese as adults. Why? Why is it so much harder for them to lose weight. Children of obese adults tend to be obese. Why? Even as 6 month olds (breast fed so no junk food, don’t walk) there is an epidemic of obesity. Why?

These are the time dependent effects of obesity. If we can accept that insulin causes weight gain, then why do high insulin levels over long periods of time tend to make it difficult to lower the insulin levels so that we can lose
Something is clearly missing here. That missing something is insulin resistance.

What is insulin resistance? Insulin is a hormone that is produced by the body. In order for it to have its desired effect, it must bind to receptors in the body that signal what the next step is.

In this example, insulin is acting on the insulin receptor to bring glucose into the cell. It acts very much like a lock and key.

Insulin is the key. It fits snugly into the lock (the receptor). Once the key and lock are together, the door opens and glucose is brought into the cell.

All hormones work in roughly the same fashion. In this regard, there is nothing really special about the insulin and insulin receptor.

In the case of insulin resistance, the key (insulin) no longer fits very well into the door (insulin receptor). Because there fit is poor, the door does not open fully and therefore less glucose comes in than normal.

What happens is that the cell now senses that there is too little glucose in the cell. It is starved for glucose. The cell needs more glucose. In order to get more glucose the body produces extra keys (insulin). The key and lock still do not fit very well, but because there are many more keys, the cell is able to get enough glucose.

This is the situation called insulin resistance. Why do we care? Because **insulin resistance leads to very high insulin levels.** We care about the high insulin levels because insulin is a major driver of obesity. As we
become obese, we are driven by hormonal factors to eat more to get to our target weight.

Not only do high carbohydrate intake lead to high insulin levels, high insulin resistance also leads to high insulin levels. As we shall soon see, it is this insulin resistance that leads to the time-dependent effects of obesity.

However, there is one important question unanswered. What causes the insulin resistance in the first place? Is the problem with the key (insulin) or the lock (insulin receptor). It is quite easy to measure the insulin and it become clear very quickly that insulin is the same hormone whether it is from a non obese or an obese person. There is no difference in amino acid sequence or any other measurable quality.

Therefore, we would have to conclude that the problem is not with the key (insulin). The problem lies in the lock (receptor). The insulin receptor does not respond quite the same way that it used to. This is insulin resistance. What causes insulin resistance?

To begin working it out, we can look at other biological systems. We can find examples of biological resistance in many places and this may provide us a clue as to what causes insulin resistance. While not all examples may apply specifically to the insulin/insulin receptor problem, it may shed some insight into the problem and show us where to begin.

For instance, there is a major problem in many parts of the world with antibiotic resistance.

As you use more and more antibiotics, there tends to be a natural selection for antibiotic resistance.

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What causes resistance to antibiotics?

H. pylori

Antibiotic applied

Normal
(produces enzyme)

Death

Mutant
(produces NO enzyme)

Continues to reproduce
and produce offspring
that resist antibiotics
used to treat it.
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resistance organisms to survive and reproduce.

Therefore, with the continued use of antibiotics, there tends to develop antibiotic resistance. By understanding this concept, we can see that the key weapon to prevent the development of antibiotic resistance is to reduce the use of antibiotics. That is, antibiotics cause antibiotic resistance.

In parts of the world where there is limited antibiotic use, there is very little antibiotic resistance. In parts of the world where antibiotics are used liberally, there is much antibiotic resistance.

What about viruses? What causes us to be resistant to viruses like diptheria and polio for instance? Before vaccines were widely available, it was well known to getting a virus causes you to become resistant to further infection by the virus.

In fact, vaccines are based entirely on this premise. By giving dead or weakened virus, we could build up a persons immunity without actually causing the full disease. That is, viruses cause viral resistance. And antibiotics cause antibiotic resistance.

These examples, of course, do not apply directly to insulin resistance. However, it starts to give us a clue that resistance can actually be brought on by the very agent itself. Could insulin cause insulin resistance?
Drug Tolerance – Chapter 4

Clues from other biological systems suggest that looking at the agent itself may be important in resistance.

What about for drugs, particularly addictive drugs such as cocaine? Resistance also develops, but the name is different.

When a drug (say, cocaine) is taken initially there is usually an intense reaction. The ‘high’. With each subsequent time cocaine is taken, there is a slightly lower ‘high’. This is known as drug tolerance. But this is really just another name for resistance.

In effect, the body becomes resistant to the effects of the cocaine with prolonged usage of the agent itself. In other words, drugs cause drug resistance.

This effect is seen in many drugs, not just cocaine. Narcotics, marijuana, nicotine, caffeine, alcohol, benzodiazepines, and nitroglycerin all have the same effect of drug tolerance. People may start using higher and higher doses of the drug to achieve the same effect as the initial time it was used. This is addictive behaviour.

This, too, is a natural response. The automatic response to antibiotic resistance is to use more antibiotic. The automatic response to drug resistance is to use more drug. The automatic response of insulin resistance is increased insulin levels. This has the effect of ‘overcoming’ the resistance.

However, it becomes clear that this is a self-defeating proposition. If you use higher and higher levels of cocaine,
then the body develops more and more resistance. This continues until you can’t go any higher.

If you use higher and higher levels of antibiotics, then more and more resistance develops until you can't go any higher. This becomes a self reinforcing cycle – or a vicious cycle.

Exposure leads to resistance. Resistance leads to higher exposure. And the cycle keeps going around.

In the end, using higher doses has a paradoxical effect. That is, the effect of using more antibiotics is to make antibiotics less effective. The effect of using more cocaine is to make cocaine less effective.

For hormonal systems, this phenomenon is well described and is related to receptor downregulation. Consider our lock and key example. In a normal situation suppose we have 100 keys (insulin) and 100 locks (insulin receptor). Each key opens a lock and we have 100 open doors at the end. This is what we want.

Under conditions of resistance, the locks no longer work as well. It now takes 2 keys to open 1 lock. With 100 keys we open 50 doors only. Since we want 100 doors open, we now produce 200 keys to open 100 doors. So now, we have the 100 door we want, but the price we pay is to produce 200 keys.

Insulin resistance works the same way. As we develop resistance, our bodies increase the insulin levels to get the same end result – glucose in the cell. However, the price we pay is increased insulin levels.

This is a well known effect of hormones and one that we take advantage of in the treatment of certain diseases. One such example is ADHD (Attention Deficit Hypractivity Disorder).

In this disease, children are hyperactive. The treatment involves the use of Methyphenidate (Ritalin). But Ritalin is not a sedative (drug that calms). Instead, Ritalin is a stimulant.

If you didn’t know better, you would say WTF? Why would you treat a hyperactive kid with stimulants? Wouldn’t that make things worse?

The answer is no. And the clinical experience backs us up. The drug (a stimulant) actually calms these kids down. High persistent levels of a stimulant will eventually lead to resistance to the stimulant. Stimulants cause stimulant reistance. This stimulant resistance now leads to calming behavior.

So let’s recap:
Antibiotics cause antibiotic resistance. High doses make things worse.

Viruses cause viral resistance. High doses make things worse.

Drugs (cocaine) cause drug resistance (tolerance). High doses make things worse.

Stimulants cause stimulant resistance. High doses make things worse.

So now let’s go back and ask the question – what causes insulin resistance?

Well, the first place I would look is to high, persistent levels of insulin itself. The implications of this are enormous. If this is true, then the hormonal obesity theory looks like this:

**Hormonal Obesity Theory**

- Fattening Carbohydrates ➔ Increased Insulin level ➔ Obesity
  - Reset Body Weight Thermostat ➔ Eat too much Exercise too little

**Insulin Resistance**
Time Dependent

Does insulin itself cause insulin resistance? We will look into the evidence over the next few posts. But consider the consequences here. Insulin leads to insulin resistance. Insulin resistance leads to higher insulin levels. Higher insulin leads to even more resistance which leads to even higher levels.

Now, all of a sudden, instead of the diet being the major driver of insulin levels, it is insulin resistance that is the major driver of insulin. This high insulin levels now drives further weight gain. These are issues that will get worse over time, because it is a vicious cycle.

**The fat get fatter.** But the reason is not that they are eating more or exercising less than the skinny people. The point is that their insulin is driven largely by resistance and not their diet. They are not lazy. They are not gluttons. They have a hormonal imbalance that needs to be addressed.

The implication is that people who have been obese for long periods of time will find it harder to lose weight. Those who have recently gained weight will
find it much, much easier to lose weight because they have not had a chance to develop significant resistance.

Those who have childhood obesity will find it hardest of all to lose weight since they have had it their entire lives. Those born to obese mothers will have been marinating in insulin their entire lives and more. They will have even more problems. You already know this to be true even if there were no studies.

Maternal obesity leads to childhood obesity. Childhood obesity leads to adult obesity. Long term obesity makes it harder to diet and lose weight. These are all consequences of the *time-dependent effects*.

But the studies are quite consistent. This paper “Predicting Obesity in Young Adulthood from Childhood and Parental Obesity” published in NEJM 1997; 337:869-873 confirms our common sense observations.

One of the biggest risk factors for obesity in young adulthood is childhood obesity. Those who have childhood obesity are have more than 17 times the risk of obesity going into adulthood!

The longer you have obesity, the harder it is to overcome.

Some of this results from the genetics of obesity, which we will discuss at a later date. Needless to say, both the calories in, calories out as well as the carbohydrate-insulin hypothesis cannot account for the known genetic predisposition to obesity.

Having one parent more than doubles the risk of obesity. But having two obese parents increases the risk of obesity more than 5 fold. And they completely ignore the time dependent effects.
What causes Insulin Resistance? From other biological systems, we can guess that a good place to start is with *insulin itself*. Does insulin cause insulin resistance? Let’s look at the evidence.

There are rare tumors called insulinomas that secrete abnormally large amounts of insulin. In these cases, patients will have very large increases in insulin but very little else wrong with them. In these cases, would the increase in insulin lead to insulin resistance?

This article "*Patients with insulinoma show insulin resistance in the absence of arterial hypertension*" answers that question.

Looking at the graph, it is clear that as the levels of insulin in the body go up and up, the levels of insulin resistance goes up and up. This is a protective mechanism and a very good thing, which is why the body does it.

If the body did not develop resistance to insulin, the high levels of insulin would rapidly lead to very, very low blood sugars. This severe hypoglycemia would quickly lead to seizures and death. Since we don’t want to die, the body protects itself by developing insulin resistance. This is a good thing.

The usual treatment of this condition is surgery to remove the insulinoma. Doing *this reverses the insulin resistance* and even the associated conditions such as *acanthosis nigricans*. The bottom line is this – *high levels of insulin cause insulin resistance*. Taking away the high insulin levels reverses the insulin resistance.
The next step is to see if we can give somebody insulin resistance. That’s what they did in the next paper “Production of insulin resistance by hyperinsulinemia in man” Diabetologia 28:70 –75, 1985 Rizza RA. 12 non-obese participants were given a 40 hour. One group was given higher and higher dose of insulin, the other was given saline (control group). The insulin group developed 15% greater insulin resistance.

The implication is this – I can make you insulin resistant. I can make anybody insulin resistant. All I need to do is give them insulin. Insulin causes insulin resistance.

Another study shows the exact same thing, but with physiologic doses of insulin. “Effect of sustained physiologic hyperinsulinemia and hyperglycemia on insulin secretion and insulin sensitivity in man” Diabetologia.

The subjects were 15 healthy young men. They were given 96 hour constant infusions of insulin. These subjects are neither obese, nor pre-diabetic nor diabetic. They were normal healthy subjects. After 96 hours of insulin infusion, their insulin sensitivity dropped by 20-40%.

The implications are staggering. I can make these healthy lean men insulin resistant. Type 2 diabetes is all about insulin resistant, so that means that I can start these people on the road to diabetes and obesity withing 3 days. High levels of insulin causes insulin resistance. They are as inseparable as a shadow to a body.

We see the exact same pattern in type 2 diabetic patients. Let’s look at this fascinating study “Intensive Conventional Insulin Therapy for Type II Diabetes. The conventional thinking at that time was that controlling the blood sugar is the most
important part of diabetes.

You might think that the better you can control the sugar, the better the diabetes and you will be healthier. You might also be disastrously wrong and cause yourself irreparable damage.

But, that was the conventional thought. So they took these type 2 diabetics and intensified their insulin treatment to tightly control the blood sugars. They started on no insulin and by 6 months were taking 100 units a day. The sugars were very, very well controlled.

But what happened to their insulin resistance? The more insulin they took, the more insulin resistance they got. Since diabetes is a disease of insulin resistance, that means their diabetes was getting worse not better! High levels of insulin causes insulin resistance.

| Table 2—Insulin requirement and weight gain during intensive CII of type II diabetes patients |
|-----------------------------------------------|----------------|
| PARAMETER                             | MONTHS OF INSULIN TREATMENT |
|                                    | 0               | 1               | 3               | 6               |
| TOTAL INSULIN DOSE (U)                 | —               | 86 ± 13         | 92 ± 16         | 100 ± 24        |
| BODY WEIGHT (kg)                       | 93.5 ± 5.8      | 97.2 ± 5.9      | 100.5 ± 6.5*    | 102.2 ± 6.8*    |
| WEIGHT GAIN (kg)                       | 3.7 ± 1.0       | 7.0 ± 1.5       | 8.7 ± 1.9       |
| CALORIC INTAKE (kcal/day)              | 2023 ± 138      | 1937 ± 122      | 1918 ± 121      | 1711 ± 119      |

Diabetes Care 1993 16:23-31 Henry RR

Here’s where things get really interesting. I’ve also said that insulin causes obesity. If this were true, you could expect that as we increase the dose of insulin from zero to 100 units/day over 6 months, that the patients would gain weight. True to form, that is exactly what happened. Patients gained 8.7 kg (19 lbs) over 6 months.

But look closely at their caloric intake. They were eating 300 less calories than at the beginning of the study. If you believe the Caloric Reduction as Primary (CRaP) theory – that it is all about reducing calories – you would be scratching you head wondering how you could reduce 300 calories per day and still gain almost 20 pounds.

But we know that calories are rather insignificant. The major question in obesity is this: What is driving up my insulin? Since insulin levels are way up, the body gains weight. Reducing calorie intake doesn’t matter. The body will only further reduce caloric expenditure to match and make the body gain weight. Insulin drives weight gain.

That brings us back to the question of weight gain. Insulin drives weight gain. But what drives insulin? The Carbohydrate-Insulin Hypothesis assumes that carbohydrate intake drives insulin, but that is
incomplete. Insulin itself will drive insulin resistance which will increase insulin in a self-reinforcing cycle.

The longer and higher the insulin levels, the higher the insulin resistance. The higher the resistance, the higher the insulin. This is what sets into motion the time dependent effects of obesity. The fat get fatter. The longer you have obesity, the harder it is to eradicate. **Insulin Causes Obesity.**

Everybody knows about these time dependent effects. However, most current thinking about obesity completely ignores these effects even though they are plainly obvious to anybody and everybody. Since type 2 Diabetes is all about insulin resistance – this also leads us to the inescapable conclusion that **Insulin Causes Diabetes.**

Insulin causes both obesity and diabetes. This is the new science of Diabesity. With this new understanding, we are led to entirely new possibilities for the cure of diabesity. If high insulin levels are the cause, then the cure is to lower insulin levels. Cure type 2 diabetes? Can it be true? Yes, but we still have some work before we get there.
The New Science of Diabesity – Chapter 6

Diabesity is so-named because of the close association of obesity and diabetes (type 2). Obesity typically comes first and type 2 diabetes comes later. This leads many to conclude that obesity causes diabetes. On the surface, this seems reasonable, since the two often co-exist. Where it becomes more difficult, however, is when people try to explain why and how obesity causes diabetes.

The answer, though, is simple and its roots lie in the Hormonal Obesity Theory. Insulin is the main driver for obesity. Cortisol plays a role as well, but insulin is the major player for most people. Insulin also increases insulin resistance in a self reinforcing cycle.

Since type 2 diabetes is really another word for insulin resistance, we can rearrange the Hormonal Obesity Theory this way.

The same thing – high insulin levels, causes both obesity and diabetes. That explains why diabetes and obesity are so tightly associated. It also explains why all efforts to find how obesity causes diabetes have failed. That is because obesity doesn’t cause type 2 diabetes. They are instead both manifestations of hyper-insulinemia. It turns out that the entire metabolic syndrome relates to high insulin levels, but we will explore that in the future.

Diabetes and obesity both have a common cause – high insulin levels. They are essentially two sides of the same disease hence the name – diabesity. We can also see that there is an interesting chicken and egg problem here. Insulin leads to insulin resistance, which leads to insulin in a vicious circle. So what comes first?

Is the problem started by insulin resistance due to, say, genetics? Or is the problem high insulin to start? Both are possible. Obesity typically manifests
first. So we can start to solve the problem by looking at the time course of obesity, particularly juvenile obesity.

In this paper "Early Changes in Postprandial Insulin Secretion, not in Insulin Sensitivity Characterize Juvenile Obesity" the authors looked at 3 groups of people – non-obese, recent obesity (<4.5 years) and long standing obesity (>4.5 years).

Both groups of obese subjects had higher levels of insulin secretion compared to the non-obese. Abnormally high insulin secretion develops early in the course of obesity and persists.

What about insulin resistance? It only develops over time. With longer duration of obesity, insulin resistance increases. The insulin resistance now contributes more and more to the fasting insulin levels.

This is the answer to the chicken and egg question of insulin and insulin resistance. Insulin is the primary insult. Persistent high levels of insulin leads to insulin resistance. This insulin resistance in turn leads to higher insulin levels.

Last week there was some excitement with the report published in the Journal of the American Medical Association that obesity rates for the age group 2-5 years had dropped by 43%. Most other age groups did not see a significant change in rates of obesity. Since childhood obesity is highly linked to adult obesity, this is indeed very good news.

The answer, though is much more straightforward. It lies in the time dependent effects of obesity.

Those with long standing obesity tend to have more insulin resistance and therefore harder-to-treat obesity. The
relatively short duration of obesity of the 2-5 year old group means that obesity is relatively easier to treat.

If we look at the Total caloric sweeteners line, we see that from 1977 to 2000 there has been a steady increase in per capita sweetener consumption. This was in response to the odious Dietary Guidelines of 1977 which produced the infamous food pyramid. The goal was to eat lower and lower fat as a percentage of calories and more carbohydrates. So sugary foods, as long as they were low in fat, were encouraged. 1977 is also when the obesity epidemic started.

Sweetener consumption peaked in 2000 under the Atkins onslaught. In the late 1990’s and early 2000s the Atkins cuisine reigned supreme. This included all the wannabe Atkins imitators. All of the attention paid to the low-carb message resulted in lower sweetener intake due to the realization that sugar makes you fat.

Of course, you could have simply asked your grandmother. She would have told you that the first step in losing weight was to stop eating sweets and starchy foods. Instead, we listened to the politicians and the doctors who said that sugar didn’t make you fat, fat made you fat. Even as we ate lower and lower fat, we gained more and more weight.

The key benefit of the Atkins message was that sugar intake started to fall starting in the year 2000. Even after the Atkins craze became as unfashionable as a Livestrong bracelet, the undeniable truth was that too much sugar was bad for you. Really, really bad. Sugar intake falls in year 2000 and after a 5-10 year lag, so does obesity in the most easy-to-treat group.
Prevention of Resistance – Chapter 7

One of the keys to high insulin levels lies in insulin resistance. Insulin resistance leads to high insulin levels. It also appears that high levels of insulin also leads to insulin resistance in a vicious cycle. How does the body normally defends against insulin resistance?

High levels of hormone by themselves cannot cause resistance. After all, think about the previous post’s experiment with constant infusions of physiologic levels of insulin. You might ask yourself this question. If normal levels of insulin can cause insulin resistance, why don’t we all eventually develop insulin resistance?

The answer lies in how hormones are secreted in the body. Hormones are always always always secreted in a pulsatile fashion. Always. Whether we are talking about cortisol, insulin, growth hormone, parathyroid hormone or any other hormone in the human body, they are released in pulses.

There is a well defined circadian rhythm. Sometimes, certain hormone levels can be expected to be very high, and at other times of the day, they are virtually undetectable. It is this very pulsatile nature that prevents the development of tolerance (resistance). Whenever the body is exposed to a constant stimulus, it will become acclimated to it.

Think about a time that you were in a dark room. Suddenly, you go outside into the bright sunny day. Your eyes are suddenly blinded and you feel disoriented. However, over the next few minutes, you become accustomed to the bright light. Now things feels normal and you can see normally again.

You suddenly step back into that room – dark as the inside of a magician’s hat. For a few minutes, you cannot see anything at all. Even though you had previously been in this room and were able to see fine, you no longer are able to do so. Over the next few minutes, you become accustomed to the dark and can start to see again.

The body has the ability to adapt to a constant stimulus. In going from dark to light, the body develops resistance to the light. In going from light to dark, the body develops resistance to dark.

This example shows how varying the levels of light can prevent the development of resistance. If we are in the sun, and we are exposed to a sudden brief darkness, we do
not lose the adaptation to the sun.

Hormones work in exactly the same way. Most of the time, hormone levels are low. Every so often, a brief pulse of hormone (thyroid, parathyroid, growth, insulin – whatever) comes along. After it passes, levels are very low again.

By cycling low and high levels, the body never gets a chance to adapt. There is never a chance to develop the resistance because the pulse of hormone is gone before this develops.

What our body does, in effect is to continually keep us in a dark room. Every once in a while, we are exposed to bright light briefly, then returned to the dark room. Each time this happens we experience the full effect of the light. We are never given a chance to get ‘used to’ the higher levels of hormone. So we are able to maintain the same sensitivity to light.

How does this apply to obesity?

The circadian pulses of insulin prevents the development of insulin resistance. However, the situation changes when we are constantly exposed to insulin. In response to insulin infusions at levels normally seen in the human body, healthy young men developed insulin resistance – the first step to diabetes type 2.

What was the difference between the experimental condition and normal behaviour? The pulsatile release. In the normal state insulin is released only occasionally, and this prevents the development of resistance.

In the experimental condition, insulin was constantly infused over 96 hours. The constant bombardment of insulin led the body to develop insulin resistance. There will be a down-regulation of receptors and the body will develop insulin resistance.

Over time, the insulin resistance leads to higher insulin levels to ‘overcome’ this resistance. High levels alone do not lead to resistance. There are 2 requirements for resistance – high hormonal levels and constant stimulus.

This is an effect that we use to our advantage in the drug therapy of angina (chest pain). Patients that are prescribed a nitroglycerin patch for angina are often given the instructions to put the patch on in the morning and take it off in the evening.

Resistance requires:

1. High hormonal levels
2. Constant stimulus
By alternating periods of high drug effect and low drug effect, there is no chance for the body to develop resistance to the nitroglycerin. If the nitro-patch is worn 24 hour/day every day, it quickly becomes useless. *The constant stimulus and high nitroglycerin levels produced resistance.*

Going back to the case of obesity, you can now appreciate that changing the composition of food alone (leading to higher insulin levels) will not be enough to produce resistance.

If we eat 3 meals a day, there are higher levels of insulin but we do **not** have the persistent, constant stimulus of insulin required to produce insulin resistance.

In other words, if we turn back the clock to the 1950s, we can still eat white bread and Oreo cookies and still have very low levels of obesity. That is because we are still balanced between feeding periods and fasting periods. We eat (as an example) 3 meals a day 8 am (breakfast) to 6 pm (dinner). In between, there are no snacks.

From 6pm to 8am, we do not eat (fasting). That means we have 10 hours of feeding and 14 hours of fasting every single day. Insulin resistance (a major driver of high insulin levels) does not develop.

Instead of eating 3 times/day, what would happen if we ate 6 times/day? Then your insulin profile would look like this.

Now we have the two pre-requisites of insulin resistance. We have high levels **AND** we have persistent levels. Under these conditions, we would
expect the development of insulin resistance. That’s exactly the advice we have been giving for the last 40 years! And we wonder why we have an epidemic of obesity.
The Perils of Snacking – Chapter 8

50 years ago, there was a near universal belief that snacking was bad for us. Your grandmother would say “It makes you fat”, or “You’ll ruin your dinner”. Back then, obesity was not such a big problem, so maybe they knew something. But then we changed our minds.

We’ve now decided that snacking is actually good for us. That eating more often will make us thinner, as ridiculous as that sounds. I’m sure you’ve heard the advice to eat more frequent, smaller meals to lose weight. That would mean that we should eat 3 meals a day and also multiple snacks in between.

How did we come to make such a 180 degree turn on snacking? The answer is explored in depth by Jacques Peretti’s absolutely fascinating BBC series “The Men who made us Fat”. The answer, I suppose not surprisingly is that it was the big food companies (Big Food) that convinced us that snacking was good for us.

Sometime starting in the 1950’s the big food companies had a problem. They needed to sell more food to be more profitable. But with only 3 meals in a day, there was a limit on the amount of food sold. The deviously brilliant solution was to introduce new “eating opportunities”. If Big Food could convince us to add a snack between lunch and dinner then the opportunity to sell more food unfurls.

A whole new category of food items to sell was created. It needed to be cheap and easy to eat. A perfect job for the refined carbohydrate. After all, cookies and crackers are mostly sugar and flour – and these do not spoil. Over the years, Big Food was able to convince us that snacking was not only acceptable, but it was also healthy. Seriously.

The problem, as we explored in our last several posts, is that increased snacking increases the risk of insulin resistance. Insulin resistance requires 2 things – high levels and persistent levels. High levels of insulin are provided by the refined carbohydrates found in snacks. Persistent levels are provided by the increased eating opportunities.

Insulin resistance is the ultimate outcome of all this snacking. Insulin resistance leads to higher insulin levels which leads to more insulin resistance. The higher insulin level then drives weight gain and obesity. Instead of a balance between the insulin dominant (fed) state and the insulin deficient (fasting) state, we now predominantly spend our time in the ‘fed’ state. And you wonder why we gain weight?

at the United States from 1977-2006. They used a survey of 28,404 children and 36,846 adult and tabulated the number of eating opportunities.

Both children and adults show the same pattern over time. In 1977, most people ate 3 times a day – breakfast, lunch and dinner. No snacks. Obesity, not such a big problem. By 2003, most people were eating 5 times a day. That is, 3 meals a day plus 2 snacks in between. We are eating more frequently. Is it any great mystery why we are gaining weight?

Taking the 50th percentile (the ‘average’) we’ve gone from eating 3.5 times a day to 5 times a day.

The time between meals has dropped from 271 minutes in 1977 to 208 minutes. That is close to a 30% decrease in time between eating. We are eating all the time!

Crazier still – we somehow think that this is good for us! Under the influence of doctors and dieticians and other medical professionals, we have been institutionalizing the practise of snacking. We begin by introducing it in our schools. We teach it to our children that it is acceptable to eat all the time. Not only acceptable, but healthy.

We eat breakfast, a mid morning snack (for the long gap between breakfast and lunch), lunch, after school snack, dinner, and then in the time between soccer halves – another snack! That’s 5-6 times a day.

It is now acceptable that we eat in the car. We can eat in the movie theatre. We can eat in front of the TV. We can eat in front of the
We can eat while walking. We can eat while talking. We can eat in a box. We can eat with a fox. We can eat in a house. We can eat with a mouse. I could go on, but then I’d have to pay royalties to Dr. Seuss.

We spend millions of dollars to give our children snacks all day. Then we spend millions more to combat childhood obesity. Then we spend millions more to fight diabesity as adults.

We give kids snacks all day. Then we berate the same kids for getting fat. We destroy their self-esteem. Then we give everybody a medal for participation to bolster their self-esteem. Arrrgggg.....

In making the proper food choices, we talk all the time about “What to Eat”. That is – process vs unprocessed, low fat vs high fat, high carb vs low carb etc. The question nobody seems to have even noticed is “When to Eat?”

The reason is that our unhealthy preoccupation with calories leads us to the erroneous conclusion that only the first question matters. In the Caloric Reduction as Primary (CRaP) view of obesity, the meal timing doesn’t matter at all. But it does. Eating all the time leads to persistent of insulin levels which is a key ingredient in insulin resistance.

Insulin resistance requires 2 things.

1. High levels – a low fat, high carbohydrate diet – leads to high insulin levels (What to Eat)
2. Persistance of levels – eating all the time (When to Eat)

It turns out that weight gain depends on both equally. It turns out that it is no more complicated than this – “If you eat all the time, you will gain weight”. The dietary changes we have made since the 1970s have thoroughly prepared us for insulin resistance. This, of course leads exactly to diabesity.

The answers, then are really very simple. Eat 3 meals a day. No snacking. Eat at a table for breakfast, lunch and dinner. Do not eat anywhere else. There are only 2 things to be fixed here. What to Eat. When to Eat.
Time Dependence of Obesity – Chapter 9

Weight gain has a clear and obvious time dependence. People tend to gain weight slowly, over years or even decades. Many people start off as a skinny teenager (age 20), but gradually gain 1-2 pounds per year. This isn’t so bad, but after 40 years they may be up to 80 pounds overweight (age 60).

Weight loss is also time dependent. Those who have been struggling with their weight for long periods tend to have more trouble than those with more recent weight gain. This all relates to insulin resistance. Those who have recent weight gain do not have enough time to develop severe insulin resistance and therefore dietary changes are successful at reducing weight.

Those who have struggled with their weight for decades may have developed severe insulin resistance. If the insulin resistance is now driving the high insulin levels, even dietary changes may have a very modest effect on reducing the insulin levels. Resistance itself depends on high insulin levels but also the persistence of those levels. Therefore, we can help prevent the development of resistance by affecting meal timing (the question of “When to Eat”).

Those skinny teenagers have little insulin resistance. Over each ensuing decade, the insulin resistance gradually increases and they develop severe insulin resistance. Since Diabetes type 2 is a disease of increased insulin resistance, they are now diagnosed with type 2 diabetes. They now have diabesity and the rest of the metabolic syndrome.

There have been two major dietary changes since the 1970’s. The first change was in the macronutrient composition of our diets (What to Eat). We changed from a higher fat (45%) to a low fat culture. In doing so, we inadvertently increased the carbohydrate composition of our diets. Increasing carbohydrates is not necessarily bad, but when those carbs are all refined carbohydrates we have a problem. This stimulates insulin and weight gain. We explored this in earlier posts.

The other major dietary change has been in meal timing (When to Eat). We changed from 3 meals a day with no snacks to 6 meals a day or snacking all the time. Societal norms
which previously had frowned upon eating outside of the table now permits eating almost everywhere.

In the 1970’s we would eat breakfast at 7:00am and dinner at 7:00 pm. That means we spend 12 hours in the fed state (insulin dominant) and 12 hours (from 7:00 pm to 7:00 am) in the fasted state (insulin deficient).

In the 1990’s and 2000’s we have changed to eating all the time. We eat as soon as we get up in the morning and eat just before going to bed. Therefore, instead of a balance of fed and fasted states, we spend most of our time in the insulin dominant state.

Resistance in a hormonal system depends upon 2 things. There must be high, persistent levels. The dietary changes we have made since the 1970’s provides these exact pre-requisites. The high insulin levels are sufficient to drive weight gain and obesity. The insulin resistance is synonomous with type 2 diabetes.

We have created the diabesity epidemic with our disastrous, misguided dietary changes. The most ironic part of this entire debacle is that these dietary changes were prescribed to reduce the epidemic of heart disease. Instead, we have actually encouraged it. We are putting out a fire with gasoline.

One of the major obstacles to health is conventional dietary advice. It becomes obvious that we must restore balance between the fed and fasted state. Ultimately, that leads to eating less. Whether that is eating less of the highly insulogenic foods such as sugars and refined carbohydrates (What to Eat) or eating less meals (When to Eat), it doesn’t really matter. In the end, we must eat less.

However, our advice to lose weight usually involves eating more. Whether it is to eat more times/day, more snacks, or more meals.

<table>
<thead>
<tr>
<th>Restore Balance – Eat Less not More</th>
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<tbody>
<tr>
<td>Eat 6 times a day</td>
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<tr>
<td>Eat high protein</td>
</tr>
<tr>
<td>Eat more vegetables</td>
</tr>
<tr>
<td>Eat more omega 3s</td>
</tr>
<tr>
<td>Eat more fibre</td>
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<tr>
<td>Eat more vitamins</td>
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more whole grains, more fibre etc, our advice is always to eat more to lose weight.

Why would we give such completely asinine advice? Such obviously-wrong-and-really-really-stupid advice?

\textit{Because nobody makes any money when you eat less.} If you take more supplements, the supplement companies make money. If you drink more milk, the dairy farmers make money. If you eat more breakfast, the breakfast food companies make money. It goes on and on.

Since the food and drug companies are providing such big grants to the doctors and dieticians, nobody wants to hear the advice to eat less. “Eating Less” is like the weird uncle at the wedding nobody wants to ever talk about.

The message has been corrupted from “How can I lose weight?” to “How can my company make money by pretending to help you lose weight?” Consider snacking – once a dietary taboo. This study \textit{Increased meal frequency does not promote greater weight loss in subjects who were prescribed an 8-week equi-energetic energy-restricted diet} demonstrates that increasing meal frequency really makes no difference in weight loss or gain.

Snacking was investigated in this paper \textit{Carbohydrates, appetite and feeding behaviour in humans} by Stubbs RJ and published in the Journal of Nutrition 2001. In this study, subjects were given mandatory snacks.

There is a clear effect that snacking reduces subsequent caloric intake at mealtimes. However, this decrease in calories does not make up for the calories taken at the snack.

Eating snacks does not decrease food intake, it increases it. (Duh). Yet the advice still persists to ‘graze’ through the day to decrease food cravings. There is no difference whether these are fatty or sugary snacks. The effect is much the same.

The bottom line is that snacking tends to increase total intake of food. Even worse, it tends to increase the number of eating opportunities as well – so there is a doubly bad effect.

Because snacks tend to be very high in processed foods, the quality of the food intake is likely to suffer substantially as well. However, it does open up large areas for the food companies to profit from selling us snack foods. There is a much larger profit in selling processed food compared to real food. No wonder the large food companies have been as enthusiastic as a hostess forcing leftovers on departing guests.
Here, once again, we should have listened to our grandmothers instead of the government. Don’t eat snacks, they would have said. It will ruin your dinner. It will make you fat. How right they were...

Breakfast is another area where there is much confusion. Does skipping breakfast make us ravenously hungry and overeat the rest of the day? That is what many would have us believe. Breakfast is the most important meal of the day. The answer, though is much more nuanced.

The word breakfast literally means the meal that breaks our fast, the period where we generally are sleeping. There is nothing in there that says we need to stuff our faces the minute we roll out of bed. Neither is there any reason to make this meal full of sugar and refined grains.

If we eat our first meal at 12:00, then lunch will be our “Break Fast” meal. What’s wrong with that?

The main problem with breakfast, as explored here in this paper “Impact of breakfast on daily energy intake” published in Nutrition Journal 2011.

In this paper, researchers broke a group down according to the number of calories eaten at breakfast. Increasing food intake at breakfast does not seem to reduce food intake at lunch and dinner.

Therefore, the more one eats at breakfast, the more one eats overall throughout the day. Worse, it increases the number of eating opportunities and times we stimulate insulin secretion.
The real problem with breakfast is that we are always in a rush in the morning. Therefore, we tend to eat foods that are highly convenient such as sugary cereals and other easy to prepare foods like toast, sugary yogurts, danishes, muffins, instant oatmeal and the like.

Food companies, seeking an opportunity to sell more highly profitable, highly processed “breakfast” foods swooped in like flies on stink. “Eat Breakfast” they thundered. “It’s the most important meal of the day!”. You can lose weight by eating breakfast. You can lose weight by eating more snacks.

No, my friend, you can’t.
Part II
What to Eat
Good Carbohydrates, Bad Carbohydrates – Chapter 10

There is controversy surrounding whether carbohydrates are good or bad. The low-carb movement (the Atkins onslaught) of the late 1990s and 2000s did much to focus attention on the role of carbohydrates in obesity. Are carbohydrates good or bad? Opinions are all over the place. Many low carb advocates suggest to avoid all carbohydrates altogether. What, even vegetables and fruits? Yes, even vegetables and fruits. As always, the answer to this question is far more nuanced.

High insulin levels drives weight gain and obesity. Since refined carbohydrates tend to cause the highest increase in insulin, they play a key role in weight gain. That certainly does not mean that all carbohydrates are bad, however.

It has been recognized for some time that there is a big difference between ‘good’ carbohydrates (whole fruits and vegetables) and ‘bad’ carbohydrates (sugar and flour). Even common sense would tell you that eating a lot of kale and broccoli will not make you fat no matter how much you eat. Eating lots of sugar, though can certainly cause weight gain. Yet both are carbohydrates – so what is the difference? We need a rational way to distinguish between type of carbohydrates.

![Diagram of blood glucose and insulin response to low- and high-glycemic-index foods](image)

Carbohydrates are chains of sugars linked together. The original classification of carbohydrates was into simple and complex carbohydrates. Simple carbohydrates consisted of 1-2 sugars linked together. This included glucose (1 sugar) and sucrose (2 sugars – glucose and fructose). Complex carbohydrates were longer chains of sugars – such as white flour. The problem with this classification is that it only takes the length of sugar into account. It does not differentiate the carbohydrates based on its physiologic properties (effect on the body). White flour, for instance was a complex carbohydrate, but it raised the glucose as much as glucose.
Dr. David Jenkins of the University of Toronto began to tackle this problem in 1981 in one of the first publications on glycemic index (GI). He recognized that there was no physiologic basis for the carbohydrate selection for diabetics at that time. By measuring the glucose raising effect of each carbohydrate, he could assemble a list of food and rank them according to their propensity to raise glucose. This is the glycemic index. This is a huge step forward, because now we can classify carbohydrates according to their physiologic effect rather than on chain size.

The GI uses identical 50 gram portions of carbohydrate to measure the effect on glucose. Glucose is set as the standard against which all other foods are measured and therefore assigned a value of 100. One of the problems with this measurement is that 1 serving of food may not have 50 grams of carbohydrate. This leads to various distortions. For example, watermelon has a very high glycemic index of 72. However, most of the weight of the watermelon is water and only 5% of that is carbohydrate. So you would need to eat 1 kilogram (2.2 lbs!) of watermelon to get 50 g of carbohydrate. A corn tortilla has a similarly high GI of 52, but the % carbohydrate is 48%, so you would only have to eat 100g of corn to get 50g of carbohydrate.

The Glycemic Load (GL) is an attempt to correct this distortion by adjusting for the % carbohydrate in each serving size. By doing this correction, we get a more clear idea of the glycemic effect per serving size, rather than per 50g portion of carbohydrate. Watermelon turns out to have a low GL of 4, but the corn tortilla still has a high GL of 25.

Foods are composed of 3 macronutrients – fat, protein and carbohydrate. As carbohydrates (chains of sugars) are broken down in the body to be absorbed, they raise the blood sugar. Protein and fat have a much lower effect on blood sugars. This is why the glycemic index measures the glucose-raising effect of carbohydrates without much concern for the fat or protein content.

The insulin levels tend to correlate with glucose levels. As the glucose level goes up, the insulin level also goes up. However, what is not commonly appreciated is that insulin level can go up without the blood sugar going up. You can measure an insulin index of foods by checking how high insulin levels go after certain foods. While the refined carbohydrates spike the insulin the highest, it may surprise some that dietary protein also tends to increase insulin levels.

One of the other major problems of the GI is that it only considers the effect of each food taken in isolation. Co-ingestion of foods that include fibre, fat and vinegar has a large effect on the resulting increase in blood glucose. The GI and GL do not take this into consideration at all.
There are many diets based on eating foods that are low on the GI, GL or both. They have had mixed success rates. Even though it may seem at first that this is a large scientific advance in classification of foods into ‘good’ and ‘bad’, the benefits have been very difficult to find. There is a huge, huge problem with the use of GI diets for weight loss.

Ready?

Here it is.

**Blood glucose does not drive weight gain.**

Hormones – particularly insulin and cortisol – drive weight gain.

Insulin is the major driver of obesity. Therefore diets should be targeted at lowering insulin levels – NOT blood glucose levels. While people assumed this was the same thing, it turns out that the insulin index of foods is quite different from the GI. Insulin secretion also depends significantly upon insulin resistance and the foods taken in conjunction with the carbohydrate. Focusing narrowly upon the blood glucose, while it may have a role in diabetes, does not help us with an obesity strategy.

Furthermore, the GI does not consider protein or fats at all because it measures only the effect of carbohydrate containing foods. The GI/GL strategy assigns 2 of the 3 major macronutrients have essentially zero role in

<table>
<thead>
<tr>
<th>Western refined foods</th>
<th>Unrefined traditional foods</th>
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<tbody>
<tr>
<td>Food</td>
<td>Glycemic index</td>
</tr>
<tr>
<td>Glucose</td>
<td>97</td>
</tr>
<tr>
<td>Rice Krispie cereal</td>
<td>88</td>
</tr>
<tr>
<td>Cornflakes</td>
<td>84</td>
</tr>
<tr>
<td>Lifesavers</td>
<td>70</td>
</tr>
<tr>
<td>Rice cakes</td>
<td>82</td>
</tr>
<tr>
<td>Table sugar (sucrose)</td>
<td>65</td>
</tr>
<tr>
<td>Shredded wheat cereal</td>
<td>69</td>
</tr>
<tr>
<td>Graham crackers</td>
<td>74</td>
</tr>
<tr>
<td>Grapefruit</td>
<td>67</td>
</tr>
<tr>
<td>Cheerio cereal</td>
<td>74</td>
</tr>
<tr>
<td>Rye crispbread</td>
<td>65</td>
</tr>
<tr>
<td>Vanilla wafers</td>
<td>77</td>
</tr>
<tr>
<td>Corn chips</td>
<td>73</td>
</tr>
<tr>
<td>Mars bar</td>
<td>68</td>
</tr>
<tr>
<td>Stone wheat thins</td>
<td>67</td>
</tr>
<tr>
<td>Shortbread cookies</td>
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</tr>
<tr>
<td>Granola bar</td>
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<tr>
<td>Angel food cake</td>
<td>67</td>
</tr>
<tr>
<td>Bagel</td>
<td>72</td>
</tr>
<tr>
<td>Doughnuts</td>
<td>76</td>
</tr>
<tr>
<td>White bread</td>
<td>70</td>
</tr>
<tr>
<td>Waffles</td>
<td>76</td>
</tr>
<tr>
<td>All bran cereal</td>
<td>42</td>
</tr>
<tr>
<td>Whole wheat bread</td>
<td>69</td>
</tr>
<tr>
<td>Fructose</td>
<td>23</td>
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</table>

The glycemic reference is glucose with a glycemic index of 100 (Foster-Powell and Miller, 1995).
weight loss and gain. A comprehensive theory of obesity would not be so cavalier in discarding this valuable information. Since protein also tends to increase insulin, eating a high protein diet may also lead to weight gain.

The GI does have its uses, though. Looking at carbohydrates through the filter of the GI, a significant amount of research has been done and is quite useful. There is a clear distinction in GL between Western refined foods and unrefined traditional foods. Western refined, processed food have a very high GI and GL, whereas whole foods tend to have a very low GL.

What is it about processing that tends to raise GL? It is the removal of fat and fibre that drives the GI way way up. Carbohydrates are not inherently bad. **The toxicity lies in the processing.** People have been eating carbohydrates for thousands of years – potatoes, sweet potato, beans, fruit etc. What has changed recently, is that we now predominantly eat *refined grains* as our carbohydrate of choice.

There are two main reasons why refined carbohydrates may be dangerous. The first is that it increases the speed of digestion. The main benefit of the GI is to highlight this important issue. Increased speed of digestion causes the glucose to spike up and the insulin levels to spike up.

The second major reason that refining is dangerous is that it may encourage overconsumption. Think about the 5 oranges that it takes to make a glass of orange juice. It is very easy to drink a glass of orange juice, but to eat 5 oranges is not so easy. By removing everything other than the carbohydrate, we tend to over consume. If we had to eat all the fibre and bulk associated with 5 oranges, we may think twice about it.

![30 grams of Carbs looks like...](https://www.dietdoctor.com)

The same applies to grains. If we remove all the bran and fibre and fat from wheat and turn it into white flour, it is very easy to eat. Eating it as a whole grain is far more difficult. Compare the following 30g of carbohydrates to a single hamburger bun...
The Role of Fibre – Chapter 11

Fibre is the non-digestible part of food, usually a carbohydrate that our bodies cannot break down and absorb. They pass through our system largely unchanged. There are 2 types of fiber – soluble and insoluble based on whether it can be dissolved in water. Plant foods often contain varying amounts of both types of fibre.

Fibre has several purported health benefits – bulking, viscosity, and fermentation. The increased bulk may perhaps fill our stomachs and therefore make us eat less. It may also slow gastric emptying time. In the colon, the fibre increases stool bulk and thus decreases the transit time. It was hypothesized this would decrease rates of colon cancer by ‘cleansing’ the bowels so to speak as the food is sped through the colon.

Soluble fibre can often increase viscosity in the gut and therefore change the absorption of other nutrients. Some fibre containing plant foods may contain phytic acid, which may act as an anti-nutrient. It produces benefits by reducing absorption. Reduced absorption of sugar and reduced sugar response may be some of the benefits here.

Increasing viscosity slows movement of food through the stomach and small bowel. It may also interfere with the mixing of food and digestive enzymes, disrupts micelle formation, and alters diffusion and interaction of nutrients with mucosal surface. The combination of these events results in slowed fat and carbohydrate absorption, which may induce satiety. Fermentation of the fibre may produce beneficial compounds. There is a thought that fibre feeds the ‘good bacteria’ in our guts and therefore provides benefit. Short chain fatty acids may also be produced that may have benefit.

Fibre came to public attention in the 1970s. From missionary work and comparison with native populations, it became clear that the so called Diseases of Civilization (heart disease, obesity, diabetes, stroke, cancer etc) were quite rare amongst natives that followed a traditional diet and lifestyle. In urban areas, as the lifestyle of the natives became Westernized, these disease started to appear.

Some researchers, notably Dr. Cleave believed that these diseases were caused by the increased consumption of refined carbohydrates – particularly sugar and white flour. These could be stored at room temperature without fear of spoiling – a large advantage. This enabled them to be carried in great quantity and great distances. Through the 1960s and early 1970s, debate about the main dietary villain raged back and forth between dietary fat and refined carbohydrates. The pendulum finally swung in favor of lowering dietary fat, which necessitated an increase in carbohydrate
intake. Since most of the carbohydrates in the Western diet are refined, the
goal of reducing fat was incompatible with lowering refined carbohydrates.

As a result, the carbohydrate hypothesis quickly fell into disfavour. Another
possibility presented itself. Perhaps, it was not the carbohydrate that was
the culprit but the lack of fibre that was the problem. The hypothesis had
the large advantage of being compatible with the dietary fat hypothesis and
quickly gained ground. Traditional unrefined foods that included
carbohydrates tended to be very high in fibre.

Denis Burkitt, a missionary surgeon who spent much time in Africa, was one
of the leading proponents of the dietary fibre hypothesis. By the time the
Dietary Guidelines for Americans was released in 1977, there was a
recommendation to “Eat foods with adequate starch and fibre”. These
became enshrined in the pantheon of Dietary Myths. Fibre was good for
you. There was a problem, though. It was difficult to show exactly how it
was good for you. One of Dr. Burkitt’s thoughts was that the fibre
decreased risk of colon cancer by speeding passage of the stool through the
colon.

Large trials were undertaken to supplement patients with fibre. 88,757
women in the prospective Nurse’s Health Study were studied to identify
whether increased fibre intake would reduce the risk of colorectal
cancer. Despite the 16-year follow up period, there was no significant
reduction in risk of colon cancer as the fibre intake increased. There seemed
to be a flat response. Taking more and more fibre seemed to confer no
additional benefits.

A randomized controlled trial attempted to show the benefits of fibre on a
precancerous lesion called an adenoma. Colorectal cancers often arise from
an adenoma but it usually takes between 5 and 10 years to develop. 1303
patients were randomized to high fibre intake or usual intake. With a
median 34 month follow up, it was impossible to demonstrate a benefit.

Well, fibre supplementation by itself seemed to have very little benefit in
reducing cancer risk. Perhaps we may fare better in heart disease. So, the
Diet and Reinfarction trial was undertaken with the goal of answering the
question whether a low fat or a high fibre diet would be beneficial for heart
disease. In the DART study, 2033 men were randomized to various diets
after a heart attacks to see if any particular diet would reduce the risk of a
second heart attack. To their astonishment, the American Heart Association
low fat diet did not seem to reduce risk at all. The Mediterranean diet (a
high fat diet) seemed to have benefit. This was what Dr. Ancel Keys had
shown years ago. Recent trials have also seems to confirm the benefits of
eating more natural fats such as nuts and olive oil in prevention of heart
disease. So eating more fat may be beneficial. What about fibre?
Those that received fibre supplements did not seem to benefit. Not only that, but increasing fibre may actually have increased the risk of dying! Yowzers. That came out of the blue. Despite the public’s infatuation with roughage, it was difficult to answer the question “Where was the benefit of fibre?”

The use of fibre for weight loss has had mixed results. While there was initial enthusiasm for this approach, it has mostly died away due to lack of success.

Most of the time we think about foods, we think about their nutrient content. The vitamins and minerals they contain. The key to understanding fibre is to realize that fibre’s benefit is not as a nutrient, but as an anti-nutrient. We think of foods and vitamins adding good things to the body. Fibre has the ability to reduce absorption and digestion. Fibre has the ability to subtract bad things away from the body. In the case of sugars and insulin, this is a good thing. The main role of fibre is that of an anti-nutrient. By various mechanisms such as bulking and viscosity the absorption of nutrient may be slowed in the GI tract. This is especially beneficial in the absorption of glucose, which tends to cause insulin to spike with meals. This study illustrates the effect of fibre. Thirteen type 2 diabetic patients were given identical 55% carbohydrate liquid meals. The only difference was the addition of fibre in one group.

Both the glucose peaks as well as the insulin peaks were reduced simply with the addition of the fibre. Because insulin is the main driver of obesity and diabetes, anything that tends to reduce insulin is beneficial. In essence, fibre acts as a sort of ‘antidote’ to the carbohydrate (poison).

This works out great, because virtually all plant foods, in their natural, unrefined state contain varying degrees of fibre! Nature has pre-packaged the poison with the
antidote. Dietary protein and fat do not usually contain fibre because you do not need the antidote if you do take the poison. The danger arises when you process the whole plant food to remove the fibre (as in white sugar and white flour). Now you are ingesting the poison without the antidote.

Westernized diets, high in refined carbohydrates exposes us to high levels of insulin because we have removed the protective effect of the fibre. Other traditional societies also eat high levels of carbohydrate. The Okinawans for instance eat a diet high in sweet potato. Approximately 80% of their diet is carbohydrate. However, they are also eating copious amounts of fibre which provides protection. Until recently, they were one of the longest lived peoples on earth – one of the Blue Zone populations.

The toxicity does not lie in the food, the toxicity lies in the processing.

This explains the interaction between fibre intake and type 2 diabetes. Dr. Walter Willett of Harvard examined the large Nurse’s Health Study 1 and 2.

With a high glycemic index diet, you can see that the risk of type 2 diabetes depends upon the cereal fibre intake. If you have a high glycemic index diet and have a high intake of cereal fibre (upper left) then the risk of type 2 diabetes is not increased. Here the reference is 1.0 which means that anything close to 1.0 indicates no increased risk. In essence, these people are taking a high level of ‘poison’ (the high glycemic index) but also taking a high level of ‘antidote’ (the fibre) at the same time.

The group that takes a low glycemic index (low level of ‘poison’) but a low level of fibre (low level of ‘antidote’) also does not have an increased risk of diabetes. But just look at what happens when you take a high glycemic index diet (high level of ‘poison’) and a low level of fibre (low level of...
‘antidote’). The odds ratio goes up to 1.75. Translated into English, this means that the risk of developing diabetes is increased by 75% by follow this deadly combination. **This is the exact effect of processing carbohydrates.**

**Fiber reduces risk of Diabetes**

The processing of cereal grains removes both the fibre and fat. This also has the effect of increasing the glycemic index. The grain (mostly wheat) is further processed by a mechanical flour mill, rather than a traditional stone grinder which grinds the wheat into a very fine powder. Because of this grinding, the body finds it very easy to absorb and therefore the glycemic index.

In the Health Professionals Follow-up study of 42,759 men with a 6 year follow up, essentially the same result was found by Dr. Willett. Diets with a high glycemic load (poison) and also high fibre (antidote) confer no extra risk of type 2 Diabetes. Low glycemic load (poison) and low fibre (antidote) also has no increased risk. But high glycemic load (poison) and low fibre (antidote) increases the risk to 217% of baseline. Yikes!

In a prospective cohort of 59,000 black women, the same results were confirmed.

These women were broken into 5 groups. The Q5 group had the highest glycemic load diet and Q1 had the lowest. Compared to the lowest group, the Q5 group had a 23% increased risk of diabetes. They were again broken into 5 groups based on intake of cereal fibre. The highest intake group had an 18% lower risk of diabetes.

We can now modify our Hormonal Obesity Theory to include the protective effect of fibre.
Carbohydrates in their natural state, with the exception of honey, always contain fibre. *The toxicity lies in the processing.* In fact, the toxicity of processing does not merely stop at carbohydrates but to all foods. Processed fats for instance, are some of the most potent atherogenic agents in the food world. As saturated natural fats such as coconut oil and lard became demonized, we started to turn to processed polyunsaturated fats such as canola and corn oil. Since they did not have great shelf life due to a tendency to go rancid, food manufacturers started to hydrogenate them. Result? Trans fats. One of the few things modern nutritionists agree is bad for us.

Meats have also recently become processed foods. It also has become clear that processed meats may be much more dangerous to us than unprocessed ones. *Some studies* show an increased mortality. *Other studies* link processed meats to cancer. *Nitrites*, for instance, used in many processed meats may be the harmful agent. In any case, the toxicity lies in the processing.

This is where junk food is so harmful to us. By its very definition, junk food is highly processed food. It may be high in carbohydrate, high in fat or high in protein. It almost does not matter. It is the processing of foods into a form that our bodies are not evolved to handle that is toxic. The food writer extraordinaire [Michael Pollan](https://www.michaelpollan.com/) always has exhorted us to eat Real Food, not “edible food-like substances”. What amazing wisdom delivered so succinctly!
One of the greatest barriers to weight loss is conventional dietary advice to eat more to lose weight. This was covered in a previous post “Time Dependence”. This advice sounds completely contradictory because it is. Nevertheless, the media is full of unhelpful advice to eat more to lose weight. The reason, I believe, is that nobody makes any money when you eat less.

One of the most pervasive pieces of advice out there is to eat more fruits and vegetables (F/V) in order to lose weight. There is no denying that F/V are healthy foods. However, if our goal is to lose weight, then it logically follows that deliberately eating more of something is not beneficial unless it replaces something else less healthy.

However, that is not what nutritional guidelines recommend. For example, in the World Health Organization Report “Diet, nutrition and the prevention of chronic diseases: report of a Joint WHO/FAO Expert Consultation” on page 68 it writes:

For children and adolescents, prevention of obesity implies the need to:

- Promote the intake of fruits and vegetables

The 2010 Dietary Guidelines for Americans also stresses the importance of increasing intake of F/V. In fact, this recommendation has been part of the Dietary Guidelines since its very inception. The health benefits of F/V stem from what they add and what they subtract from our diets. F/V are high in micronutrients, vitamins, water and fibre. They may also contain antioxidants and other healthful phyto-chemicals. This is likely the reason why we are reminded to eat more F/V.

What is not explicit is the fact the increased intake of F/V is expected to displace higher energy, less healthy foods from our diet. Because most F/V have a low energy density (low calories in a given volume), and have high
fibre, it is assumed that satiety will increase and therefore we will eat less other foods that are more energy dense.

If this is the main mechanism of weight loss, then our advice should be to “replace bread with vegetables” for instance. But it is not. Our advice is simply to increase F/V intake. Is this really true? Can we really eat more to lose weight?

A recent paper shed some light on this issue. Entitled “Increased fruit and vegetable intake has no discernible effect on weight loss: a systematic review and meta-analysis”, it was published in Aug 2014 AJCN. In this paper, researcher gathered all available studies on the intake of F/V intake and weight gain.

What they found should not be a major surprise. The zero line on the graph indicates no net benefit or harm from increased F/V. No individual study was able to show that there was any significant benefit, and the sum total of all the studies also showed no benefit. Taken all together, this is very strong evidence that the advice to eat more to weigh less is simply not sound. To put it simply, eating more F/V does not make you lose weight. You cannot eat more to weigh less.

Why do we give such obviously-wrong advice? Starts with an M and rhymes with honey. Because nobody makes any money when you eat less and therefore buy less F/V. Companies want to sell you fruit/vegetables/supplements/calcium/omega 3/vitamin D/snacks/meal replacements. That is how they make money. Nobody sells any books telling you to eat less. We don’t want to hear what we already know.

So, should we eat more fruits and vegetables? Yes, definitely. But only if they are replacing other unhealthier foods in your diet. Replace. Not add. Losing weight boils down to reducing insulin levels. Eating more of something, even as healthy as fruits and vegetables simply does not achieve that goal.
This is also reinforced by the recent study “Fruit consumption and the risk of type 2 Diabetes” published in the British Medical Journal 29Aug 2013. Looking at 3 large prospective cohorts (Nurses Health Study 1 and 2, and the Health Professional Follow Up), the researchers from Harvard looked at the risk of type 2 diabetes with the consumption of whole fruits and fruit juice.

With close to 190,000 subjects over 2 decades of follow up, this was a huge study. After adjustment, the pooled hazard ratio for every 3 servings/week of fruits was 0.98. In English, this means that over 12 years or so of follow up, eating an extra 3 servings of fruit per week reduced your risk of type 2 diabetes by 2%. The risk for fruit juice was 1.08 meaning an extra 8% risk with 3 servings of fruit juice.

Clearly, adding fruits to the diet is not an extremely beneficial dietary strategy. But not all fruits are the same. The glycemic index, as well as the amounts of fibre, vitamins and antioxidants all differ. This was a large enough study to allow individual fruits to be examined. One thought is that fruits with high glycemic index may not be as beneficial. This has often led to advice such as eat fruits except for bananas and grapes. In a stunning rebuke to the utility of the glycemic index, this study showed that grouping fruits by glycemic index was completely useless. Eating more high glycemic load (GL) foods lowered the risk of type 2 diabetes, whereas eating more moderate and low GL foods did not. This is completely contrary to what the glycemic index would predict.

What happens when you replace, not add? That is, if you were to replace 3 servings/week of fruit juice with whole fruit, would you see a benefit? Now you’re cooking with fire. Now you start to see a significant benefit for the prevention of type 2 Diabetes. But there is a wide range of effects depending upon the fruit in question.

Certain fruits such as blueberries are far more effective at preventing diabetes than others (cantaloupe and strawberries). Bananas and grapes, often avoided due to their high glycemic index, turn out to be fairly good in preventing diabetes. Replacing fruit juice with fruit resulted in the following reduction in risk of diabetes:

- Overall 7%
- Blueberries 33%
• Grape 19%
• Apples/ Pears 14%
• Bananas 13%
• Grapefruit 12%

The overall message is clear. Eating more to weigh less is a doomed strategy. You need to Replace, not Add.
The Incretin Effect – Chapter 13

What is the incretin effect? With the Glycemic Index (GI), we had a physiologic scale for classifying carbohydrates. Some raise blood sugars more than others. When researchers looked at carbohydrate containing foods, there is a very close correlation between the GI and the amount of insulin released (Insulin Index – II).

Insulin is the hormone predominantly involved in the regulation of blood sugar. Carbohydrates raise blood sugar and insulin rises to deal with it. Fats and proteins have a negligible effect on blood glucose. For years, it was therefore assumed that this meant that it would also have a negligible effect on insulin secretion. This was not actually true. We had just ignored this rather inconvenient fact. Since fat and protein do not raise blood sugar, there should be no effect on insulin. But proteins and their constituent parts – the amino acids can also raise insulin without any effect on the blood sugar.

As far back as 1966, in the Journal of Clinical Investigation the paper “Insulin secretion in response to protein ingestion” showed that oral or intravenous administration of the amino acid leucine would result in stimulation of insulin secretion. Dr. Nuttall rediscovered this fact in 1991 in his paper “Plasma glucose and insulin response to macronutrients in non diabetic and NIDDM subjects” (Diabetes Care 1991:14:824-38).

So proteins and amino acids are able to stimulate insulin without any change to the blood glucose. This required an entire change in the way we think about macronutrients. Around the same time, there was increasing interest in the hormones produced in the stomach (gut hormones), and the so-called incretin effect.

In 1986, Nauck and colleagues noticed something unusual. In humans, the blood sugar response to glucose given through an intravenous or given orally was the same. That was not really a surprise. Simple sugars such as glucose are quickly and easily absorbed in the intestines.

However, what was interesting was the insulin secretion. In response to the same level of blood sugar, there was a huge difference in the insulin response. Many powerful medicines are
given intravenously because there is 100% bioavailability. This means that all of the drug is active. When given by mouth, many medicines are incompletely absorbed or partially deactivated by the liver before getting to the bloodstream. So intravenous medications tend to be more effective than oral ones.

However, in this case, the oral glucose was far, far better at stimulating insulin than the intravenous. Furthermore, *this mechanism had nothing to do with the blood sugar*. Insulin response is not the same as the blood sugar response. This had not been seen before. Eventually, it was discovered that the stomach produces hormones – now called incretin hormones that increase the insulin secretion. Since the intravenous glucose bypasses the stomach, there is no incretin effect. This may account for 50-70% of the insulin secretion after oral glucose intake.

So far, two incretin hormones have been described in humans. These are Glucagon Like Peptide 1 (GLP-1) and glucose-dependent insulinotropic polypeptide (GIP). Both hormones are deactivated by the hormone dipeptidyl peptidase-4 (DPP4). Inhibition of DPP4 by drugs like saxagliptin causes an increase in the levels of the incretin hormones. This raises insulin levels and helps to lower blood sugars.

The incretin effect starts within minutes of ingestion of nutrients into the stomach and peaks at roughly 60 minutes. The incretins have effects other than stimulation of insulin. They also inhibit glucagon and delay stomach emptying. This has the effect of slowing down glucose absorption in the body.

The existence of a pathway of insulin secretion entirely independent of blood glucose was new and exciting. Here was a pathway whereby proteins could stimulate insulin secretion. Perhaps the amino acids stimulated the incretin pathway as a mechanism of glucose-independent insulin secretion. Comparing the intake of different proteins, milk and dairy product in particular were potent stimuli of insulin.
This paper “Glycemia and insulinemia in healthy subjects after lactose equivalent meals of milk and other food proteins” published in AJCN in 2004 demonstrates the effects of different proteins. While milk and cheese stimulate insulin the most, even cod fish has an effect here.

When milk is taken with other foods, it also causes an increase in the insulin response. In the paper “Milk as a supplement to mixed meals may elevate postprandial insulinaemia”, milk or water was given to test subjects in addition to their spaghetti meal. It is clear from the diagram that the insulin increases much more with the addition of milk versus the water.

Dairy proteins show a large discrepancy between the blood glucose effect (Glycemic Index) and insulin effect. Most dairy product score extremely low on the glycemic index, but very high on the Insulin Index. While lactose is a carbohydrate contained in milk, it does not seem to play much of a role in the insulin response. Testing the effect of pure lactose, the glycemic index and insulin index parallel each other closely.

As it turns out, it was the dairy proteins that caused the insulin increase. There are predominantly two type of dairy protein – casein (80%) and whey (20%). Cheese is composed mostly of casein protein. The whey portion of the dairy is felt to play the larger role in insulin stimulation, but cheese may also have significant remnants of whey protein left. The branched chain amino acids found in dairy products may be particularly potent.

Does the incretin effect play a role in insulin secretion in response to whey?

The paper “Glycemia and insulinemia in healthy subjects after lactose-equivalent meals of milk and other food proteins: the role of plasma amino acids and incretins” sheds some light upon the mechanism of insulin secretion. They measured glucose and insulin levels after test meals, and then went one step further to test the levels of GLP-1 and GIP.

What they discovered was that the whey protein in dairy product in particular stimulated insulin secretion the most. While GLP-1 levels were no different between any of the proteins tested (cod, milk, whey and cheese), the whey stimulated GIP to a much larger degree than the others.
This, then at least in part, explains some of the findings we discussed in our last post. Carbohydrates are not the only stimulator of insulin. Proteins also cause insulin increase. Whoa, nelly. This changes everything.
Insulin Index – Chapter 14

Insulin levels had always been assumed to go up or down with the blood glucose levels which led to the glycemic index which had always been assumed to be a surrogate measure of insulin levels. The glycemic index did not turn out to be as successful in weight control because glucose does not drive obesity. Insulin drives obesity. With the insulin index, it was realized that only 23% of the variability of insulin response depends on the glucose. In other words, how much the glucose increases only accounts for 23% of the insulin response. Even taking into account the other macronutrients fat and protein, this only accounted for another 10% of the insulin response. The vast majority of the insulin response is still unknown. Some of the factors that are suspected or shown to affect the insulin secretion include presence of dietary fibre, an elevated amylose/amylopectin ratio of the starch, preserved botanical integrity (whole foods), presence of organic acids (fermentation), addition of vinegar (acetic acid), and addition of chili peppers (capsaicin).

Nevertheless, the main point here is that there are many factors in the co-ingestion of foods that affect insulin. Things are about to become very complicated. The simplistic “Carbs make you fat!”, or “Calories make you fat!” or “Red meat makes you fat!” or “Sugar makes you fat!” sort of arguments simply are not able to capture the complexity of the human condition of weight gain. Among breakfast cereals, there is wide variation in the insulin response. All-Bran, with its high fibre and promise of colonic regularity, seems be stimulate insulin much less than cornflakes for instance.

Protein containing foods turn out to be surprisingly potent at stimulating insulin. Beef and fish, for instance, have virtually no effect on the blood glucose levels. Yet they stimulate insulin almost as much as most cereals. Nevertheless, we are able to discern some general patterns here. If we look at carbohydrates, there is a noticeable trend here. As the total grams of carbs goes up, the insulin level also tends to go up. This is what we expect. This is the basis of many low carbohydrate diets from the original Banting diet of the 1850s to the modern day Atkins diet and its many imitators. It has been recognized for well over a century that the amount of carbohydrates consumed plays a role in the development of obesity.
However, this study indicates that the correlation is far from perfect. The correlation between carbohydrate content and insulin is relatively low (23%). Dietary fat, on the other hand tends to have a lowering effect on insulin scores. As the amount of fat increases, the insulin score tends to decrease. Fat also tends to have minimal effect on blood glucose, so this is pretty much as we expected as well. Carbohydrates tend to raise glucose and insulin.

Dietary fat tends not to raise glucose and tends not to raise insulin as well. So far, so good. The surprise here is dietary protein. Looking at the protein, there seems to be a slight trend here as drawn by the line. As protein goes up, the insulin score tends to go down. But you can see that this relationship is mostly driven by 2 main outliers at the extremely high protein intake levels (40 and 60 grams). If we remove these outliers, then it seems that there really is not much relationship between the amount of protein and the rise in insulin.

Some proteins raise insulin a lot and some proteins raise it only a little. Dairy protein especially whey, seems to be the biggest culprit here. The implication is that some protein containing foods can lead to obesity while other protein containing foods are not likely to do so. Here the data become extremely murkily and there are numerous inconsistencies in the literature. In one study, four different proteins were tested – eggs, turkey, fish and whey protein. Whey protein is one of the proteins found in dairy. Cow’s milk protein is composed of 80% caseins and only 20% whey. Casein is mostly responsible for milk allergies. Whey is well known as the byproduct left over in cheese making. Milk is separated into curds and whey. Whey protein is often used by bodybuilders as it is high in Branched Chain Amino Acids (BCAA) that are felt to be important in muscle formation.

Compared to the other proteins, whey resulted in the highest insulin levels. This is despite the fact that none of the proteins had a very large
effect on blood glucose. In this study, after the test meal, participants were given a buffet lunch 4 hours afterwards so that effects of protein on satiety could be measured. The whey protein suppressed appetite to the largest degree. It is certainly possible that whey protein may have two different effects on weight gain. The increase in insulin may promote weight gain, but the decrease in appetite may suppress it. Again, I will stress that I do not have all the answers here. The field of study is new and ever evolving at this point.

Is Dairy Fattening? – Chapter 15

Dairy proteins stimulate high levels on insulin due partially to the incretin effect. Glucose, though does not seem to increase much. If insulin increases, does this mean that dairy is fattening? Here the answer is far more difficult.

Protein containing foods may stimulate insulin partially due to the incretin effect. These are hormones secreted in the stomach that help with digestion and stimulate insulin. They are released in response to ingestion of all three macronutrients – fats, proteins and glucose. The implication is that all foods can stimulate insulin release, not just carbohydrates. Dietary fats have the lowest effect on insulin secretion.

By contrast, only carbohydrates have a significant effect on glucose. For starchy foods, there is a very good correlation between the glycemic index and the insulin index. However, in the specific case of milk (dairy) products, there is a large discrepancy. Most dairy foods have very low GI score (15-30) but very high Insulin Index scores (90-98).

At first glance, this may appear to implicate dairy protein as a major contributor to diabesity. Here there is some evidence to suggest that this is true. A recent, very large European observational study included 38,094 participants in the EPIC study, followed over 10 years prospectively. Increasing total protein and animal protein consumption is associated with a modest but significant increase in the risk of developing type 2 diabetes.

Another dietary recommendation often given is the advice to eat more fish. With its high omega 3 fat content, it certainly seems like good advice. Omega 3 fats are known to alter the expression of peroxisome proliferator–activator receptor genes. Thus they might be expected to have the beneficial effect of reducing type 2 Diabetes.

High protein intake, particularly animal proteins may be detrimental perhaps in part due to the increased insulin. Should we all go vegan? The data here is anything but clear. With regards to dairy, other large observational studies seem to indicate that, increased dairy consumption leads to less weight gain.
This relationship was explored in the paper “Association between dairy food consumption and weight change over 9 y in 19 352 perimenopausal women”. The Swedish Mammography Cohort was a community based observational study that used dietary recall questionnaires to estimate dairy product intake and compared that to weight changes. The effect was relatively modest, but there was no increase in weight gain with increased dairy products. Looking at the different milk products, the consumption of whole milk, sour milk, cheese and butter seemed to be associated with less weight gain. Drinking less low fat milk, on the other hand, resulted in less weight gain.

In the United States, the CARDIA study sampled 3157 young adults in 4 metropolitan areas and measured markers of the metabolic syndrome including obesity over a follow up period of 10 years. The group with the highest intake of dairy seems to have the lowest incidence of obesity.

This study also confirmed these results for the rest of the metabolic syndrome. Higher intakes of dairy were protective, not detrimental. A study in Tehran, published in the AJCN similarly showed that higher consumption of dairy was correlated to a lower risk of the metabolic syndrome.

What about type 2 Diabetes? The Health Professionals Study Follow Up Study prospectively examined the relation between type 2 diabetes and dairy intake. 41 254 people took part in this large study and over 12 years of follow up, dairy intake was associated with a modestly lower incidence of type 2 diabetes after adjustment for age and BMI.

The CARDIA study further investigated the independent effect of fibre on the metabolic syndrome. The reference level of 1.0 is chosen for the group with both high dairy and high fibre. Taking high dairy but low fibre almost tripled the risk of Insulin Resistance Syndrome (IRS), as did low dairy but high fibre. Taking a low dairy, low fibre diet increased the risk by almost 7 times!

This, of course is not very good news for the Standard American Diet (SAD). Milk consumption has been steadily declining since the
1970s. Furthermore, the increased processing of food has resulted in decreasing intake of dietary fibre. That puts the SAD in the highest risk category with almost 7 times the risk of IRS.

So the data are very conflicting. Some studies show that increased protein intake is bad for diabesity while other equally high quality data suggest that it is good. This leads to many heated discussions on internet bulletin boards. There are those who believe that dairy is good because it is high in fat and protein (low carb high fat). There are those who believe all dairy should be excluded from our diet (some paleo folks). There are those who believe that red meat should be excluded from the diet (vegans, vegetarians). There are those who believe that red meat is good because it is high in protein and fat (Atkins).

Are carbs bad? Are dietary fats bad? Are proteins bad?

Oh man, my head is starting to hurt...
Is Protein Fattening? – Chapter 16

The weight gaining potential of protein is a complex question. Let me reiterate here that this area of research is still very new and we certainly do not have all the answers. While refined carbohydrates (white flour) and sugar are almost universally considered fattening, there is maddening debate about the virtues or flaws of dietary protein. In particular, dairy proteins and red meat seems to be the most controversial. Indeed, the answer is not nearly as easy as it may seem. There seems to be good data supporting both points of view.

There are two considerations here – the incretin effect, and dose of dairy protein. First, the insulin increase in response to protein is a result of incretins rather than an increase in blood glucose. Incretins such as GLP-1 and GIP not only raise insulin, but have other significant effects as well. GLP-1 has been shown to not only increase insulin secretion, but it also plays a role in control of gastric emptying. For example, infusion of GLP-1 in humans causes a significant slowing of the emptying of the stomach. This has the effect of slowing the release of nutrients and glucose in the body, which may be beneficial in a similar way to fibre.

The best way to study the effects of incretins would be to use a drug that mimic the effect of of GLP-1. The first such drug developed was called exenatide, also known as Byetta. Isolated in the saliva of the Gila monster (how cool is that?), this drug was approved for the treatment of type 2 diabetes. Byetta mimics the effects of GLP-1 which causes an increase in insulin secretion via the incretin effect and thus, blood glucose is lowered. Because of the blood sugar lowering effect, this drug has been used for type 2 diabetes for years. But other effects have been noticed as well. Byetta caused a slowing of gastric emptying, which leads to feeling of nausea or GI upset in patients.

The normal action of the stomach involves holding the food just eaten, mixing with stomach acid and slowly discharging the contents into the small intestine at a measured pace. With Byetta, this discharge of stomach contents occurred much slower than usual. The major side effects of Byetta – nausea – is seen in up to 57% of patients taking this drug. Patients often report a feeling of satiety or feeling...
full. This is similar to the effect of dietary proteins seen in the last post where whey protein caused the greatest increase in insulin, but also the greatest feeling of satiety.

In that study subjects were given equal calorie portions of egg, turkey, tuna or whey. The greatest insulin effect was seen with whey protein. However, subjects were then given an all you can eat buffet after ingestion of the protein, and monitored for amount eaten. The amount of food consumed after whey protein was significantly less than with egg or turkey.

This would be consistent with personal experience. Dietary proteins tend to cause you to feel more full for longer. Imagine you have equal caloric amounts of 2 foods – a steak versus ice cream. How long would you feel full after the steak versus the ice cream? My guess is that the steak will make you fuller for longer. It just ‘sits’ in your stomach. That is the incretin effects slowing the gastric emptying. Ice cream, on the other hand, or cookies do not ‘sit’ in your stomach for long. Soon after we eat them, we become hungry again.

Thus, there are two opposing effects of the incretins on weight – insulin and stomach emptying effects. On the one hand, insulin may be fattening. On the other hand, because the stomach is fuller for longer, our satiety mechanisms are activated and we feel full, and thus do not eat as much later on. Which effect is more powerful? The way to find out is to give incretins and see if patients gain or lose weight. This class of drugs – which includes Byetta as well as liraglutide (Victoza) are not associated with weight gain despite the increase in insulin levels. Instead, both drugs cause weight loss due to the effect on gastric motility. It appears that stimulation of the incretin effect with GLP-1 triggers more weight loss effect than weight gain.

However, this weight loss is only found with selective GLP-1 stimulation. Remember that the incretins include both GLP-1 and GIP, so far. More may yet be discovered. **Whey may have more of an effect on the incretin GIP rather than GLP-1.** Is this important? I don’t actually know. However, what it means is that we need to be careful before extrapolating that whey can help weight loss.

There is also a class of medications called the DPP4 inhibitors, of which sitagliptin, and saxagliptin are examples. Rather than mimic GLP-1, they block the enzyme dipeptidyl peptidase-4 (DPP4). This DPP4 enzyme breaks down both GIP and GLP-1. Blocking the enzyme that breaks down incretins leads to an increase in incretins. A double negative. See what I did there? DPP4 leads to less incretin. Blocking DPP4 leads to higher incretins. My point here is that there is an increase in both GIP and GLP-1 with DPP4 inhibitor drugs. The question – does DPP4 inhibitor leads to weight loss? No. They also do not lead to weight gain. They are instead,
weight neutral. Switzerland. So which specific incretin is stimulated may be important in determining weight gain or loss.

The other major consideration, particularly for dairy is the dosage of protein. An average American diet is 50% carbohydrate. It is easy to eat more carbohydrate. A few more slices of bread, a big plate of pasta. If you wanted to increase protein, you could also eat a large steak. However, how can you increase dairy protein alone? Can you eat a huge slab of cheese for dinner? How about drinking several gallons of milk? Two large tubs of yogurt for lunch? It is really not that easy to significantly increase the dosage of dairy proteins without resorting to whey protein shakes and other such artificial foods. You can drink an extra glass of milk, but that doesn’t change the macronutrient composition of the diet very much. However, reducing refined carbohydrates from 50% to 10% is a huge change in diet composition.

Therefore, even if dairy proteins are particularly good at stimulating insulin, we do not eat or drink enough of these proteins to make such a large difference in the overall scheme of things. Think about it this way. Bunting is a part of the game of baseball. However, the difference between really good bunting and bad bunting is not likely to make any difference to the outcome of the baseball game. It just doesn’t matter that much.

However, when we talk about red meat and other proteins, the story is slightly different. It is not so difficult to increase animal protein in the diet. Substitute a slice of bread for an extra hamburger patty. Eat more chicken. Have a large steak. Eat a bowl of chili.

So what is the effect of whey protein on weight gain? What is the effect of red meat on weight gain? That may, I suspect, depend upon the balance of weight gaining and weight losing effects of the incretin effect, which will depend upon which incretin is stimulated.
Red Meat Dilemma – Chapter 17

Our dilemma here is that there are really two opposing effects of meat and other animal proteins. One is to raise insulin which tends to cause weight gain, The other is to slow gastric emptying, increase satiety and tends to cause weight loss. Which is the stronger effect?

One of the largest association studies of the recent past has come from analysis of the data by combining three very large cohort studies – the Nurses Health Studies I and II as well as the Health Professional’s Follow Up study. Looking at this combined data, comparison was made between specific foods and and the risk of obesity. While this was not a randomized trial, it still contains vast amounts of useful data. “Changes in Diet and Lifestyle and Long-Term Weight Gain in Women and Men” was published in the New England Journal of Medicine in 2011.

One of the most important things that the researchers did was to look at specific foods. The past few decades had seen the rise in ‘nutritionism’ whereby foods were reduced to classification into ‘carbs’, proteins and fats. However, that does not even begin to capture the complexity of food science. An avocado, for instance is not simply 88% fat, 16% carbohydrate, and 5% protein with 4.9 grams of fibre. But this sort of nutritionism is how avocados became classified as a ‘bad’ food for years due to its high fat content only to the reclassified today as a superfood. (Useful online tool for this sort of useless thing here). While kind of fascinating, there are hundreds of nutrients and phytochemicals in foods that affect our metabolism not captured by this sort of simplistic analysis.

This is the sort of useless thing, by the way, which goes into food labels which kind of explains why they don’t really make any difference. I imagine that nutritionism really got going during the ‘low fat’ craze of the 1970’s where we imagined that we could explain the effects of all foods based on 3 macronutrients. First, we thought that all fat is bad. Then all carbs were bad. Then there were good carbs and bad carbs. Then there were good fats and bad fats. Next there will be good proteins and bad proteins (animal vs plant for instance). The truth is that food resists such easy classification. We also make this sort of artificial distinction for types of food – fruit for example. Banana is a bad fruit and berries are a good fruit. This was based on a notion such as the glycemic index, or the amount of fat, or the amount of sugar.

Following 120,877 men and women over 12 to 20 years, the researchers calculated the association between intake of specific foods and weight gain. Overall the average weight gain over any 4 year period was 3.35 pounds – pretty close to the 1 pound per year that is often estimated. While
this may not sound like much, over 40 years, say from age 20 to age 60, that will result in 40 excess pounds transforming the 160 pound average person into a 200 pound pre-diabetic patient.

It is important to remember that an association study like this cannot prove causation. However, one of the strengths of this study is to be able to look at long term results – something that randomized trials tend not to do. Because weight gain accumulates over decades, a short term trial of several years may not tell us what we need to know.

However, it is still interesting to look at the data here. It is easy enough to understand why potato chips and french fries may be fattening. They are both highly processed carbohydrates that raise insulin and glucose significantly. For the same reason, sweets and desserts, and refined grains are all highly associated with obesity. Indeed, it would hard to find anybody who would argue that potato chips, desserts and white bread are not fattening.

But there is also a strong association between processed meat, unprocessed meat and butter and obesity. Since these are not carbohydrates, one may assume that they are not fattening. But they are. Once you realize that protein also stimulates insulin, it begins to make some sense that these foods may also lead to obesity. But it is the meats and not the dairy so much that leads to obesity despite the fact that dairy proteins stimulate insulin to a much higher degree. The problem here may be a quantity of meat versus dairy ingested.

There are also foods that are associated with a lower risk of obesity – for instance, nuts and vegetables. This seems straightforward. Both are low in sugars and very high in fibre. Both effects will tend to lower insulin and protect against weight gain. But whole grains and fruit are also protective. A shock to Atkin’s enthusiasts, it appears that the high fibre in these foods may be protective.
Among beverages, sugar sweetened beverages and fruit juice are associated with obesity. Not a surprise – both are very high in sugar and very low in fibre. Skim milk may have a slight association, but whole milk does not. The higher milk fat may be the protective agent here. Diet soda is protective, but I have my doubt whether this is a true effect or whether it merely reflects that fact that these are people that are trying to lose weight.

It become much easier to understand the graph when you consider that all the foods that tend to cause weight gain tend to raise insulin. Those that tend to protect against weight gain tend to contain protective factors – fibre, fermentation (yogurt), and fat.

Here then, may be the clue to the final unravelling of the Atkin’s diet. Originally envisioned as a low carbohydrate, high fat diet, it evolved into Atkins 2.0 in the 1990s. While still low carbohydrate, the low fat craze turned the Atkins reboot as a low carb, high protein diet. Atkins enthusiasts turned away from real food to Frankenfood creations like protein milkshakes sweetened with fructose, meal replacement shakes and protein bars.

Pharmaceutical companies, like Matt Taibbi’s vampire squid that jams its blood funnel into anything that smells like money, were only too happy to create new nutritional products to cater this new craze. Boost. Ensure. Optifast. Slimfast. Have you ever read the ingredients of these meal replacements? It would horrify you. Milk protein, fructose, canola oil, soybean oil and a multivitamin. Does this sound good to you? Or check the ingredients of Atkins Nutritional bar. Chocolate flavour layer, peanut butter flavour layer, glycerin, protein blend, cellulose etc. There is more friggin’ glycerin than protein!!!!

Not recognizing that not all carbohydrates are inherently fattening, they also turned away from many delicious and nutritious fruits and vegetable. This made the diet hard to tolerate and sure enough, the compliance to the diet turned out to be very low. This was not a diet that you could follow for life, despite what many claimed. Dr. Atkin’s New Diet Revolution was finished.

Devotees of the low carb approach faced the red meat dilemma. Meat was supposed to be non fattening because it was not a carbohydrate. However, some large studies questioned this assumption. The fascinating study “Changes in Diet and Lifestyle and Long- Term Weight Gain in Women and Men” demonstrated a rather large correlation between red meat and obesity. Since proteins could be equally good at stimulating insulin, meat was not nearly as benign as once imagined.
The experience of countless Atkins acolytes reinforced the trial results. Millions of dieters in the late 1990s and early 2000s tried the Atkins low carb, high protein approach. For some, the approach worked. However, for many more, it didn’t deliver the promised weight loss. Popularity waned.

Another interesting tidbit from that study involved the utility of exercise. Overall, there was a beneficial effect of exercise on weight gain. No surprise there. Breaking down each dietary group to quintiles of exercise, you can see that within each dietary group, there is benefit with more exercise.

Comparing the effect of exercise, it is clearly much less beneficial to changes in diet, though. This, too is not new. The overwhelmingly important factor in weight gain is diet. Exercise is secondary.

The risk of type 2 Diabetes, closely related to obesity was also examined in relation to red meat consumption. “Red meat consumption and risk of type 2 diabetes: 3 cohorts of US adults and an updated meta-analysis” looked at the same cohort as the previous study. Analyzing the result separately for processed and unprocessed meat, there was a strong correlation between diabetes and both types of meat. However, the hazard ratio was only 1.19 for 100 grams of unprocessed red meat compared to 1.5 for 50 grams of processed meats.

Putting that into plain English, it means that for every extra 100 grams of unprocessed meat (steak, pork chop etc), there is a 20% increase in risk of diabetes.

For every extra 50 grams of processed meat, there is a 15% increase in risk of diabetes.
meat (bacon, luncheon meats etc) there is a 50% increase in the risk of diabetes.

As we saw in the case of carbohydrates, the toxicity lies not predominantly in the food itself, but in the processing of the food.

Numerous other studies have shown a difference between processed and unprocessed meats. A systematic review “Red and processed meat consumption and risk of incident coronary heart disease, stroke, and diabetes: A systematic review and meta-analysis” showed that there was NO association between unprocessed meats and diabetes, heart disease or stroke, but a 42% increased risk with processed meats.

Results from the European experience was published in “Meat consumption and mortality–results from the European Prospective Investigation into Cancer and Nutrition.” Increased mortality and cancer risk was seen predominantly with processed meats. The effect was much weaker in unprocessed meats and was not statistically significant.

This is yet another body blow to the Atkin’s diet. The advice to skip the bread and eat all the bacon you wanted was not good. Sausages, luncheon meats, bacon and other processed meats were not a whole lot better than the processed carbohydrates they were meant to replace. **The toxicity is in the processing.**

This really makes a lot of sense. Our bodies evolved to eat many different types of foods without health problems. Some ancestral diets were very high in meat and fats – such as the Inuit. Others, like the Kitavans and Okinawans, were heavily carbohydrate dependent. Yet both traditional diets were associated with good health. You notice, though that neither one included large amounts of processed food – whether they were processed meats or processed carbohydrates.
Looking at our construct for the hormonal obesity theory, we can add the effect of animal protein to the protein raising effect. This comes with the understanding that there are also protective effects of the protein on satiety and reduced stomach emptying. Consider the major macronutrients – fats, proteins and carbohydrates. Both protein and carbohydrates stimulate insulin to differing degrees. Dietary fats have to least effect in this regard. However, over the past 4 decades, we have, at the behest of nutritional authorities reduced our intake of dietary fats. This left us with trying to increase protein or carbohydrates in the diet. Yet no matter which way we turned, we were doomed to failure.

Thus we can see where the confusion with calories comes in. Since it appeared that all macronutrients were the same, we imagined that all foods, regardless of nutritional content could be measured in a common unit – the calorie. We initially tried to follow a low fat, high carbohydrate diet. When that failed, the Atkins approach of low fat, high protein was tried. That failed as well. Now we grope along without any idea about what is happening because we have no framework to understand the underlying aetiology of obesity.

One of the most important points here is that both proteins and carbohydrates come with protective factors. With carbohydrates, there is the protection of fibre. Unprocessed carbohydrates contain large amounts of dietary fibre. Traditional societies often ate upwards of 50-100 grams of fibre. The standard, highly processed (removes fibre) North American diet trudges along with 15 grams.

With dietary proteins, we are protected by the incretin effect and the slowing of gastric motility. Dietary protein increases satiety so that we will feel more ‘full’ and eat less at the next meal. In years past, large meals full of protein
would be followed by long periods of fasting in order to ‘digest’. To a large extent, this does not happen any longer.

While we may indulge in a large meal, such as Thanksgiving, we are paranoid to skip the next meal because we irrationally fear that missing the next meal will ‘wreck’ our metabolism. We see this in children all the time. When they are not hungry, they will not eat. We also see this in wild animals – lions, tigers, snakes etc. Years of training have prepared us to ignore our own feelings of satiety so that we will eat when the time comes, whether we are hungry or not. So, we circumvent the protective effect of the incretins by rigidly scheduling our meals 3 times a day, come hell or high water. You may not be hungry, but by god, you will eat!

There are no intrinsically bad foods, only processed ones. The further you stray from real food, the more danger you are in. Should you eat protein bars? No. Should you eat meal replacements? No. Should you drink meal replacement shakes? (horrified) No. Should you avoid processed meats and carbohydrates? To the extent that you can. It is difficult to completely remove these from our diets. Therefore we have evolved over the centuries other dietary strategies to ‘detoxify’ or ‘cleanse’ ourselves. These, dietary strategies too have been lost in the mists of time. We will rediscover these ancient secrets soon, but yes, it is a cliffhanger. For now, stick to real food.
The Benefits of Vinegar – Chapter 18

Diluted vinegar is a traditional tonic for weight loss. Indeed, mention of this remedy is found as far back as 1825, when Brillat-Savarin wrote about its dangers. A British poet, Lord Byron had popularized the weight loss tonic and would reportedly go for days eating biscuits and potatoes soaked in vinegar. Popular ways to use the vinegar are to ingest several teaspoons prior to meals, or drinking it diluted in water at bedtime. Apple Cider Vinegar seems to have gained a particular following as it contains both vinegar (acetic acid) as well as the pectins from the apple cider (a type of soluble fibre).

Is it true?

Vinegar has been used since the times of the ancients. Wine, left undisturbed, will turn into vinegar (acetic acid). Indeed, the word has its origins from Latin vinum acer (sour wine). The ancients quickly discovered the versatility of this wondrous substance for cleaning. In a time before antibiotics, the antimicrobial properties of vinegar was often used by healers. Wounds would often be washed in wine and vinegar. Because of the antimicrobial properties, vinegar has also been used to preserve food (pickling). Saeurkraut and kimchi, on the other hand, use fermentation to produce lactic acid which is slightly different process.

As a beverage, the tangy sour taste of vinegar never really gained much popularity, although Cleopatra famously was rumoured to dissolve pearls in vinegar as a drink. However, it still retains fans as a condiment for French fries, in use in dressings (balsamic vinegar) and in making sushi rice (rice vinegar).

There are no long term data on the use of vinegar as a weight loss aid. However, smaller short term human studies have been done recently. One study published in 2004 was titled “Vinegar improves insulin sensitivity to a high-carbohydrate meal in subjects with insulin resistance or type 2 diabetes”. Vinegar was given to to insulin resistant as well as normal subjects before a high carbohydrate meal of orange juice and a bagel. The effect of the vinegar is clear. In controls, but more strikingly in pre-diabetic people, vinegar lowers the subsequent glucose high after the bread as much as 34%. Additionally, the insulin spikes are significantly reduced. Since insulin is a driver of obesity and diabetes, this effect, similar to fibre may be very beneficial over the long term.

A follow up study in 2010 “Examination of the antiglycemic properties of vinegar in healthy adults.” looked in more detail about the vinegar effect. Two different doses were examined and it was found that 10grams
(approximately 2 teaspoons) was just as effective as 20 grams. Taking the vinegar just before the meal was more effective than taking it 5 hours before meals.

Indeed, the effect has also been shown with rice. In “Glycemic index of single and mixed meal foods among common Japanese foods with white rice as a reference food”, authors found that vinegar lowered the glycemic index of white rice by almost 40%. Addition of other foods such as pickled vegetables and fermented soybeans (Natto) also significantly lowered the glycemic index of the rice. In a similar manner, substitution of pickled cucumber for fresh cucumber reduced the glycemic index of a test meal by 35%.

Potatoes, served cold and dressed with vinegar as a salad showed considerably lower glycemic index than regular potatoes. The cold storage may favour the development of resistant starch, and the vinegar adds to the benefits. Both glycemic and Insulin Index reduced by 43% and 31% respectively.

Another study “Vinegar Ingestion at Bedtime Moderates Waking Glucose Concentrations” looked at the benefits on the blood sugars. Participants were type 2 diabetic patients that were not on insulin. They were given the 2 tablespoons of apple cider vinegar at bedtime and morning blood sugars were measured. There was a measurable effect to lower blood sugars in well controlled type 2 diabetic patients, but the effect is relatively small.

The dose response curve of vinegar on blood insulin effect can also be measured. Dr. Ostman tested different doses of vinegar on a test meal of white bread (50g carbohydrate) and found that higher doses of vinegar can progressively lower insulin response.

What is important to realize is that the total amount of carbohydrate is the same in all cases. The vinegar is not merely displacing carbohydrate calories, but actually seems to exert a protective effect on the serum insulin response.

Satiety has also been measure in response to white bread and various doses of vinegar. The lowest satiety score was white bread alone. There was a progressive linear relationship between the satiety score and the amount of vinegar ingested. Another study showed that vinegar ingestion resulted in slightly lower caloric intake through the rest of the day (approx 200-275 calories less). This effect was also noted for peanut products. Interestingly, peanuts also resulted in a reduction of glycemic response by 55%.
How does acetic acid produce these beneficial effects? This is a matter of conjecture. It is postulated that the acid interferes with the digestion of starches. It is possible that it has its effect on inhibiting salivary amylase and therefore specifically interferes with carbohydrate absorption. Indeed, the effect on fats and proteins is negligible. The other major mechanism postulated is that vinegar reduces gastric emptying. There is conflicting data here, with at least one study showing a reduction of glucose response by 31% but no significant delayed gastric emptying.

Ultimately, however, it is far more important to realize that it does work rather than question how it works. The large Nurse’s Health Study showed a significant cardiovascular benefit with the use of oil and vinegar dressing. This was considered to be the effect of dietary alpha linolenic acid. However, Dr. F Hu points out that mayonnaise, which contains similar amounts of alpha linolenic acid does not appear to provide nearly the same cardiac protection. Perhaps the difference here is the consumption of vinegar. This is only an association study and cannot prove it, but certainly an interesting hypothesis given what else we know about vinegar.

What about safety? Brillat-Savarin had warned against the use of vinegar as a weight loss aid all those years ago. But, really. Come on. Vinegar has been consumed for thousands of years. There is just about no conceivable way that it is not safe for human consumption. Just don’t expect rapid weight loss with the use of vinegar. Even among its proponents, it will only cause mild decrease in weight.
Adding vinegar as a protective factor into our Hormonal Obesity Theory, we can see now that there are, in fact a number of dietary changes we can make to reduce the insulin levels. None of these is new. The use of fibre and vinegar in the battle of the bulge has long been discussed and has always been a part of folk remedies. Maybe we should look harder at the tried and true rather than the latest and greatest.