What to Eat
Weight Loss Guide Part 3
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Sugars – Chapter 1

Sugars are fattening. Of that, there is really little doubt. Almost everybody can agree upon this fact. The reason it is fattening is a little more contentious. Some consider sugar as empty calories since they contain few essential nutrients. Others think that sugar makes food more ‘palatable’ and thus cause us to overeat these foods. In a similar vein, sugar may be more ‘rewarding’ and thus cause overeating and obesity. Sugar is also a carbohydrate, which may be the mechanism of its fattening effect. Others, such as Dr. Robert Lustig considers sugar to be a poison. His internet lecture became a viral sensation. All I can say is that, while I generally agree with his position, a poison that requires daily consumption for 70 years or more to kill is not a very strong poison. I do use the analogy of poison referring to carbohydrates and fibre but always in quotations because it is not to be taken literally. Carbohydrates, even sugars are not literally poisonous.

All of these effects are plausible and we need to look further into the dangers of fructose specifically. Where does fructose fit in? Fructose does not raise the blood sugars appreciably, yet is more strongly linked to obesity and diabetes than glucose. Fructose also is not more rewarding than glucose. Fructose is just an empty calorie as glucose. So why is fructose so bad?

First, we need a few definitions so that we are clear about what we are talking. The 6 ring sugar that is found in the blood is called glucose. Virtually all cells in the body can use it. In the brain, for example, it is considered the preferred energy source. Muscle cells will greedily import glucose from the blood for a quick energy boost. Glucose is stored in the body in various forms such as glycogen in the liver. Under stress situations (flight or fight response) the body will release glucose for quick energy boost. Glucose circulates throughout the body with virtually no limitation. There are glucose receptors in many different tissues and organ systems.

Fructose, on the other hand, is a 5-ringed sugar. It is the sugar that is found naturally in fruit. It is metabolized in the liver and does not circulate as fructose. The body has no ability to use fructose in its natural form. The brain cannot use fructose. The muscles cannot use fructose. For this reason, fructose ingestion does not appreciably change the blood sugars (blood glucose).
Table sugar is called sucrose. This is composed of two sugars linked together. There is one molecule of glucose which is linked to one molecule of fructose. This is the form that we are most familiar with.

Carbohydrates can be classified in different ways. An older, less useful manner of classification was the distinction between simple and complex carbohydrates. While this terminology is still sometimes used, it was long ago recognized that it provided little useful information.

Simple carbohydrates consisted of 1-2 sugars linked together. These were also called mono and disaccharides. These were predominantly glucose, fructose and sucrose, although other sugars included lactose.

Complex carbohydrates are long chains of sugars. That’s it. There is no need to have fibre, micronutrients, vitamins or other. Highly refined white flour is a complex carbohydrate. The reason for this classification was the mistaken idea that complex carbohydrates are slower to be digested due to the chain length. With further investigation into the Glycemic Index, this is not really true at all. White bread, for instance has a very high glycemic index comparable to sugar.

This simple vs. complex classification is not useful because it only distinguished the chain length, which we are not really interested in. If we are interested in the glycemic effect, then we should measure it directly and use the Glycemic Index. However, as noted previously, this, too has its problems.

The main problem with the sugars sucrose and fructose, in general is dosage. Consumption of sugar has been increasing recently and this has been closely paralleled by the rising obesity rates and diabetes.

The release of the 1977 Dietary Guidelines for Americans warned about the dangers of too much sugar. However, this message got lost in the anti-fat crusade that followed. Consumption of sugar increased sharply beginning in 1977 as we became more and more concerned about the fat contents of our foods. Since fat was the predominant,
overwhelming concern of health conscious shoppers, the sugar content was ignored or forgotten.

Bags of jellybeans and other such candies were proudly proclaiming themselves to be low in fat despite the fact that they were virtually 100% sugar. In essence, we had ‘taken our eyes off the ball’. The goal was better health and even in 1977 guidelines recognized that sugar was certainly just as bad as fats. It turns out, of course, that dietary fats are not nearly as bad as they were made out to be, but that is a story that will need to wait.

The worst offender in the sugar story by far, is the sugar-sweetened beverage (SSB) – soft drinks, sodas and more recently, sweetened teas and juices. In both children and adults, the consumption of SSB and juices rose throughout the decades to peak at roughly year 2000. Milk, the traditional beverage of children began to fall and fall. By the year 2000, SSB provided 22% of the sugar found in the American diet compared to 16% in 1970. No other food group even comes close.

Despite the gloom and doom of all the recent hype about the obesity crisis, there shines a ray of hope in the accompanying graph. By year 2000, we had realized that we had lost our way. Under the Atkins Onslaught, we were forced to face the fact that sugar was indeed a dietary villain. Sugar had never been considered a health food, but had gotten a free ride as we concerned ourselves obsessively with dietary fat.
Sugar sweetened beverages started to decline in popularity. Iced sweetened teas and sports drinks have valiantly tried to take their place but have been unable to stem the tide. Soda is a $75 billion industry that has until recently seen nothing but good times. In the 1970’s people doubled their intake of the fizzy stuff. By the 1980s, it was more popular than tap water. By 1998, Americans were drinking 56 gallons per year. But in 2014, Coca Cola has faced 9 consecutive years of sales decline as health concerns mount. The demographic of the baby boomer likely plays a role as well, since older folks are less likely to drink the toxic stuff.

Now, SSB face strong opposition, from soda taxes to the recent effort by Michael Bloomberg to outlaw outsized beverages. Some of the problems, of course, are of their own making. Coca Cola, for instance spent decades trying to convince us to drink more soda. They were successful, but at what cost? As the obesity crisis grew, the companies found themselves under increasing fire.
The epidemiological evidence linking sugar consumption and diabetes as well as obesity is overwhelming. Very little controversy surrounds this fact. Another demonized food – salt, by contrast is far more controversial. Many people argue that salt is not as bad as advertised – myself included. But sugar, alas, has few defenders, only pushers. This doesn’t mean that sugar consumption necessarily will decrease, only that recognition is finally spreading.

Sugar consumption in North America seems to have peaked in 2000 under the influence of the Atkins Onslaught. The Low-Carb movement seems to have refocused our eyes upon dietary carbohydrates, of which the sugars were the most egregious offenders. With no nutritional redemptive properties, sugar quickly raised the ire of all nutritionists from Low Calorie adherents to Low Carb to Paleo to Raw Food. Under the previous Low-Fat regime, we ‘took our eyes off the ball’ by allowing sugar consumption to stealthily increase while we were watching the fat in our diets. Ten years later, we are celebrating the rarest taste of victory in the Battle of the Bulge.

But the sugar pushers would not be so easily defeated. With the knowledge they were fighting a losing battle in much of the first world, they took aim at the Third World to make up profits. The areas of the world that consumed the least sugar – Asia and Africa were particularly targeted. As sugar consumption stabilized in much of the world, or even fell, it increased quickly in Asia and Africa.

The result? A diabetes catastrophe. A report published in the Journal of the American Medical Association in September 2013, estimates that 11.6% of adults have type 2 diabetes eclipsing even the long time champion – the USA at 11.3%. 22 million new diabetes were added since 2007 – approximately the population of Australia. Things are even more shocking when you consider that only 1% of Chinese had type 2 diabetes in 1980. A horrifying 1160% rise within a single generation. This is despite the fact that the Chinese had an average Body Mass Index of 23.7 (normal weight) compared to 28.7 (overweight) in the USA. How is this even possible? Even if China had
doubled its 2006 sugar consumption, it would not reach levels seen in North America. The answer, which we will get to, lies in its relatively high consumption of other carbohydrates.

Sugar sweetened beverages, as noted previously deserve much of the blame. Juice has long been connected to childhood obesity. Prospective studies show a close correlation between intake of juice and childhood obesity over the 48 month follow up period. Of note, whole fruit, with its protective fibre component bears no relationship to obesity.

The same relationship holds in adults, where data from the large Nurse’s Health Study shows the damage. Weight gain was slightly higher among the group with a higher intake of sugar sweetened beverages (SSB). However, if you analyze the group that changed from drinking little SSB to higher amounts, the amount of weight gain almost doubles.

That group that went from drinking lots of SSB to virtually cutting it out managed to reduce their weigh gain over the years to almost nothing. Amazing.

The risk of diabetes clearly increases with higher levels of consumption of SSBs. This is shown clearly in the large Nurse’s Health Study which followed 51, 603 women.

For every extra 150 kcal/person/day of sugar, the prevalence of diabetes rose by 1.1%. No other food group showed significant relationship to diabetes. Diabetes correlates with sugar, not calories.

This, of course, makes perfect sense. Type 2 Diabetes, at its very core, is a disease that is about excessive sugar storage in the body. Not blood sugar, mind you, but total body sugar, if you will. The problem with most treatments for type 2 diabetes is that they only control the blood sugar by forcing it into the body (usually the liver). These treatments, such as insulin, have been proven to be ineffective at reducing the health complications of diabetes. In other words, insulin only treats the blood sugars, not the diabetes since it does not remove the sugar from the body.
Sugar is a highly refined carbohydrate just like white flour and white rice and potatoes. Yet the effect on diabetes and obesity is much, much higher than either one. Why? How can we fit this into our understanding of Hormonal Obesity? It turns out that sugar is, in fact, bad in several different ways – only one of which is as a refined carbohydrate. After all, the INTERMAP study showed that the Asian diet up to the 1990s was extremely high in carbohydrate, but very low in sugar. Obesity and diabetes were extremely rare. The main toxicity in sugar lies in the fructose. This is an area that has received increasing attention in recent years.

Sucrose is composed of equal parts glucose and fructose. The glucose portion will raise blood sugars and drive the insulin response. But the fructose will not. Fructose does not raise blood sugars. Therefore, it has an extremely low glycemic index. Furthermore, fructose produces only a mild rise in insulin compared to glucose. For many years, this led many people to regard fructose as a benign form of sweetener.

Adding to its halo was the fact that the main sugar in fruit is fructose. An all natural fruit sugar that doesn’t raise blood glucose levels? Sounds pretty healthy to me. Sounds pretty healthy to the good people of the ADA as well. A wolf in sheep’s clothing? You bet your life. The difference between sugar and rice/flour/potatoes will very literally kill you. They don’t realize the deadly effect of fructose.
The Deadly Effects of Fructose – Chapter 3

For years, fructose was considered a benign sweetener because of its low glycemic index. Fructose was found naturally in fruits. The problem, as often is the case, is a matter of scale. Whereas natural fruit consumption contributed only small amounts to the diet, added sweeteners, particularly in the form of sucrose or high-fructose corn syrup (HFCS) added large amounts.

The peak of fructose consumption was reached just before the year 2000, when people started to become concerned about the relationship between HFCS and obesity. Adolescents in particular had a high consumption of fructose. Fructose is the sweetest naturally occurring carbohydrate. What was wrong with that?

HFCS was developed in the 1960s as a liquid sugar equivalent to sucrose. HFCS was 55% fructose and 45% glucose compared to the 50-50 split seen in sucrose. Sucrose was processed from sugar cane and sugar beets. While not exactly expensive, it wasn’t exactly cheap either. HFCS, however, could be processed from the river of cheap corn that was flowing out of the American MidWest.

In processed food, HFCS found a natural fit. It was liquid and therefore easily incorporated into any food. Indeed, it found its way into almost everything. Pizza sauce, soups, breads, cookies, cakes, ketchup, sauces – you name it, it probably had HFCS. The advantages didn’t stop there.

- Sweeter than glucose
- Prevented freezer burn
- Helps browning
- Mixes easily
- Extends shelf life
- Keeps breads soft

But there was one overriding benefit to HFCS compared to sucrose. It was cheap. Companies cared about that more than anything else in the world. It was cheap, cheap, cheap. Given the similarity between
HFCS and sucrose, most did not expect any significant difference between HFCS and sucrose. And it was cheap. Food manufacturers raced to use HFCS at every chance they had.

The tide began to turn in 2004 when George Bray published a study that showed that the increase in obesity mirrored the rise in use of HFCS. However, at the same time, there was a significant decrease in the use of sucrose, which was more expensive. HFCS was merely replacing the more expensive refined sugar in the diet of America.

There rise in obesity really mirrored the increase in the use of sugar. Whether it was 55% fructose (HFCS) or 50% fructose in sucrose likely did not make a big difference. The problem was the fructose.

The most important outcome of this furor was the increased scrutiny on the dangers of excess fructose in the diet. Researchers started to investigate the differences between glucose and fructose. It turns out that there are many differences. Every cell in the body can use glucose. Indeed, certain cells can only use glucose. Red blood cells are often cited as only being able to use glucose. Skeletal muscles, in need of quick and accessible energy, will preferentially consume glucose as well, but every cell has the ability. No cell in the body has the ability to use fructose for energy.

Studies into fructose absorption showed that free fructose is poorly absorbed in the human gut. However, the presence of glucose will significantly increase the amount of fructose absorbed. Where glucose requires insulin for maximal absorption, fructose does not. Most cells do not take up fructose and it is really only metabolized in the liver. Where glucose can be dispersed throughout the body for use as energy, fructose concentrates like a guided missile to the liver.

Fructose rapidly becomes fructose-1-phosphate with no limit. When an excess of glucose is consumed, the body has a natural rate limiting
step that prevents excessive overloading of the metabolic system. No such system is present for fructose. The more you eat, the more you metabolize. This becomes glucose, lactate and glycogen – all concentrated in the liver. With higher levels of fructose ingestion, it becomes acetyl-CoA, which is needed for fatty acid synthesis. What this means is that excess fructose becomes fat in the liver. Fatty liver. High levels of fructose will cause fatty liver.

This fatty liver is absolutely crucial in the development of insulin resistance in the liver.

Think about it this way. Insulin is normally released when we eat. It directs the glucose to be stored as energy for later use when we are not eating. In the short term, some of this is stored as glycogen. However, there is a limited amount of glycogen that we can store in the liver. The rest of the glucose needs to be stored as fat. So insulin promotes the production of fat in the liver, a process known as de novo lipogenesis (DNL). This means, literally “making fat from new”. It is the process of turning glucose into fat. Insulin is the hormone that pushes food energy into storage.

When insulin is low, this process reverses. Glycogen is broken into glucose (glycogenolysis) and new fat is degraded for energy. If we balance feeding periods with fasting periods, then there is not net fat gained. A well designed, well run system. The blue balloon above depicts the normal situation where it is relatively easy to both put sugar into storage and take it out as well.

But what happens if the balloon is overinflated? What if the liver, which stores some fat, is crammed full of fat already? Insulin is trying to cram more fat into a liver that is already completely full of fat. Just as it is more difficult to inflate an overinflated balloon, insulin has a harder time of trying to shoved food energy into the liver. It takes higher and higher levels of insulin to move the same amount of sugar
into the fatty liver. Another word for this phenomenon is **insulin resistance**. The body is now resistant to the efforts of insulin. Normal levels of insulin will not be able to push sugar into the liver.

The other problem with the over crammed liver is that it takes high levels of insulin all the time to keep the sugar and fat bottled up. If insulin levels start to drop, then sugar comes whooshing out, just like the overinflated balloon. This leads to high levels of blood sugar, which the body doesn’t like and will try to oppose with higher insulin levels. In other words, insulin resistance leads to higher insulin levels. High insulin level will encourage storage of sugar and fat into the liver. This causes even more over cramming of fat in the fatty liver causing more insulin resistance. A classic vicious cycle.

This is true only at the liver. Skeletal muscle will have normal levels of insulin sensitivity as will the brain (hypothalamus). Fructose differs significantly from glucose because fructose is 100% metabolized in the liver. It is the difference between pressing down with a hammer and pressing down with a needle point. You do not need as much pressure if it is all directed onto a single point. Fructose is all directed at the liver.

Fructose causes fatty liver. Fatty liver causes insulin resistance. Why should we care? Because insulin resistance leads to higher insulin levels. Higher insulin levels drives obesity.
Fructose causes insulin resistance - Chapter 4

The metabolism of excessive amounts of fructose leads to fatty liver, which is a key step in the development of insulin resistance, as we saw in our last post. Is there evidence that consumption of fructose leads to insulin resistance? In a word – yes.

As far back as 1980, there were studies linking the use of fructose, but not glucose to the development of insulin resistance in humans. Adding 1000 kcal/day of excess calories as glucose vs fructose in healthy young people. Did not change body weight over the duration of the test.

The group with excessive glucose intake did not show any statistical difference in sensitivity to insulin. The fructose group, however, showed a 25% worsening of their insulin sensitivity. After 7 days! Remember that both groups had been overfed by the same amount of calories. Fructose also caused a decrease in the binding of insulin to cells and this paralleled the increase in insulin resistance.

A more recent study showed much the same effect. Normal healthy subjects were overfed fructose. De novo Lipogenesis (DNL) – or the production of new fat in the liver increased six fold accompanied by a 79% increase in plasma triglycerides. Insulin resistance in the liver increased by 28%. After 6 days! Muscle insulin resistance was not changed.

Since insulin resistance is the hallmark of type 2 Diabetes, this means that these previously healthy subjects were now well on their way to developing type 2 Diabetes in just 6 days. Healthy subjects given 25% of their daily calories as Kool-Aid sweetened with either glucose or fructose for 8 weeks. While this seems like a high percentage, there are many people who consume this high proportion of sugar in their diets.

The oral glucose tolerance test is a standard test for diabetes and pre-diabetes. The blood sugar is measured in response to a sugary drink given to the patient. The fructose, but not the glucose group has already developed pre-diabetes. The fructose group has developed significantly more insulin resistance than the glucose group.
Insulin levels are also significantly higher in the fructose group. This is all despite the fact that both groups were given equal caloric amounts of glucose versus fructose. The fructose, furthermore had much less effect on the blood glucose since it has a very low glycemic index. This demonstrates yet another failure of the glycemic index classification system.

This is scary in so many ways. First, the amount of fructose is certainly higher than most people consume. But the study only lasted for 8 weeks. What is going to happen after decades of high fructose consumption? Fructose specifically will lead to fatty liver, insulin resistance and eventually type 2 diabetes. But the accompanying concern is that the **high insulin levels will also lead to obesity**.

If we add this knowledge to our model of hormonal obesity, this is what we get.

**Hormonal Obesity Theory**

The key driver to obesity is the increased insulin levels. This depends upon the balance between dietary factors (particularly refined or fattening carbohydrates) and protective factors such as fibre and vinegar. Another key driver of insulin levels is the time dependent effect of insulin resistance. This sets up a vicious cycle whereby high insulin levels increase insulin resistance which leads to high insulin levels. The longer this cycle goes around, the worse it becomes.
However, fructose also plays a key role in the development of insulin resistance aside from the high insulin levels. This is at least partially explained by the induction of fatty liver and hepatic insulin resistance.

From here, it is easy to see why sugar is so fattening. Sucrose is composed of a 50-50 mix of glucose and fructose. HFCS is 55% fructose. The glucose in the mixture acts as a refined carbohydrate and stimulates insulin. The fructose acts on the liver to produce insulin resistance. Therefore, the sugar is stimulating insulin production both in the short term and in the long term. Over time, chronic overconsumption of sugar leads to high insulin levels. This leads inevitably to obesity.

Sugars are not simply ‘empty calories’. They are not simply refined carbohydrates. They are far more dangerous than that. They stimulate both insulin as well as insulin resistance. Is there much of a difference between sucrose and HFCS? I don’t believe so. Both are bad. One may be slightly more bad, but that is splitting hairs.

The bottom line is this. If you want to avoid weight gain – cut out sugars from the diet. Do not replace them with sweeteners. They are equally bad for you. On this, at least, everybody can agree.
Fat Phobia – Chapter 5

“It is now increasingly recognized that the low-fat campaign has been based on little scientific evidence and may have caused unintended health consequences”

It has been over a decade since the influential Harvard researchers Drs. Frank Hu and Walter Willett wrote this in 2001. Even now, the low fat campaigners are everywhere you care to look. Things are starting to change, however. Recently, the admission that perhaps saturated fats were not the enemy splashed across the front page of Time Magazine.

The vilification of fat had its roots in the 1960s. The key figure was prominent nutritionist Dr. Ancel Keys. In the post war years, there was increasing concern about the so called epidemic of coronary disease that seemed to be sweeping the United States. The cause of coronary artery disease is a ruptured atherosclerotic plaque. Pathologic studies clearly identified that there was cholesterol contained within these plaques.

Searching for a culprit, cholesterol was in the wrong place at the wrong time. This seemed to make some superficial sense. High cholesterol levels were believed to cause plaque buildup thereby blocking the artery causing heart attacks. If high blood cholesterol levels were bad, then eating cholesterol must be, too.

The overwhelming majority (80%) of the cholesterol in the blood is manufactured by our liver. Only 20% comes from our diet. From the bad rap it has gotten, you might believe that cholesterol is some harmful poisonous substance to be eliminated. Actually, nothing could be farther from the truth. Cholesterol is a key building block in the plasma membranes that surround all the cells in our body. In fact, it is so vital, that every cell in the body except the brain, which is too specialized, has the ability to make cholesterol. If you reduce cholesterol in your diet, your body will simply make more.
In the early 1950s, increasing attention was being paid to heart attacks which seemed to strike middle aged men and women randomly. During a sabbatical leave to Oxford University in 1951, Dr Ancel Keys was struck by the fact that Italian labourers had low rates of heart attack and hypothesized that the low dietary intake of fat helped to protect them. Through the 1950s he began informal surveys about diet in various countries and measured bloodwork including blood cholesterol levels.

Dr Keys doggedly pursued his hypothesis that increased dietary fat caused increased coronary disease. This led to the famous Seven Countries Study, an observational study that compared different rates of coronary disease and different diet and lifestyle factors. In 1970, at the 5 year point there were three main conclusions with regards to fats.

1. Cholesterol levels predicted heart disease risk
2. Amount of saturated fat in the diet predicted cholesterol levels
3. Monounsaturated fat protected against heart disease

The SCS made notable contributions to our understanding of other coronary risk factors that ultimately proved correct. For instance, it significantly strengthened the link between cigarette smoking and heart disease. The importance of blood pressure was identified in this study as well. Physical activity was identified as a major protector.

It was from this study that interest in the Mediterranean diet soared, and continues to this date. Many date the start of the Low Fat period to Dr. Key and this particular study, but it is noteworthy that total fat was not correlated to heart disease. Instead, it was not until he separated saturate and unsaturated fats that the association was made.

So It seems that as saturated fat goes up in the diet, serum cholesterol does as well. The next conclusion was that
as saturated fat goes up, so does death from coronary disease.

This was really the first time that saturated fats came onto the radar screens. There were several problems with this, although none was very obvious at the time.

First, this was a correlation study. That means that this could not prove causation. One of my personal rules is that correlation studies are mostly crap. There is nothing more dangerous in medicine as the correlation study. They are dangerous because you can very easily and mistakenly draw causal conclusions.

For example, women taking hormone replacement had a 50% reduction in coronary disease. But when you give HRT, you increase rates of heart disease, blood clots and breast cancer. The problem is that women who had been taking HRT were also healthier in other ways. The HRT was not beneficial, it was harmful, and the drug maker Wyeth got sued into non-existence.

If we were making the same claims about people, it would be called racism. For instance, many basketball players are black. There is a strong correlation. But they are not good because they are black. They practise hard for long hours. Many Chinese are good at math. But they are not good because they are Chinese. They practise hard for long hours. There are many negative stereotypes as well, but I’d prefer not to list those.

You can only prove that saturated fat causes heart disease by a randomized trial which is extremely expensive and difficult to perform. Indeed, there are many times that observational data is the best that we have. Data from the large Nurse’s Health Study and the Health Professional’s Follow Up Study suffer from the same problem. Yet they still represent the best data available, flawed as it is. Evidence based medicine was in its infancy, and the problems of observational data were even less appreciated than it is today. So, if a country happens to have high heart disease rates, and high levels of saturated fat intake, this does not immediately mean that the saturated fat caused heart disease.

Some recognized this fatal flaw immediately. In a rebuttal to Dr. Keys, Drs. Yerushalmy and Hilleboe wrote:

But quotation and repetition of the suggestive association soon creates the impression that the relationship is truly valid, and ultimately it acquires status as a supporting link in a chain of presumed proof. This, of course would soon be standard operating protocol for nutritional doctrines everywhere. Oh, not sure if salt if bad? Just
repeat it enough until it becomes true. Oh, not sure if fat is bad? Just repeat ad nauseam until it becomes true. Oh, not sure calcium tablets are bad? Just repeat ad nauseam until it becomes true. Innuendo and repetition. Innuendo and repetition. Innuendo and repetition.

The second major problem was the inadvertent use of nutritionism. This is a fatal conceit that we understand enough about food chemistry and biology to reduce all foods to a blend of fat, protein and carbohydrate. Here, for instance, Dr. Keys makes the unnoticed and unintentional claim that all saturated fats, all unsaturated fats, all dietary cholesterol etc. are the same.

This is similar to comparing sugar to kale because both are carbohydrates. Or comparing trans-fat laden margarine to an avocado because both are fats. Or comparing a processed, nitrite laden piece of salami to a grass fed organic beef because both are proteins.

Again, if we applied this to people, it would be called racism. You cannot assume that your black friend is as good at basketball as Michael Jordan, just because they are both black. I cannot sing as well as Celine Dion just because we are both Canadian. I am not as smart as Albert Einstein just because we are both men. Here the mistake is obvious. In nutrition, we fail to consider foods as individuals – all with their own particular good and bad traits.

There was also some complaints from later analyses that Dr. Keys may have ‘cherry picked’ his countries to those that he knew would show the correlation. This may or may not have been the case, but largely irrelevant. This sort of thing happens in studies all the time. The greater concern is the wrongful use of observational data and the rise of nutritionism.

One notable correlation that Dr. Keys insisted did NOT exist was the link between dietary cholesterol and serum cholesterol. Since cholesterol in the blood (80%) is mostly manufactured by the liver, the diet has very little to do with it. This was recognized very early on, but despite that, it became entrenched in popular culture that dietary cholesterol was bbbaaaaddddd. Where we got that idea from, I don’t really know.
The Diet-Heart Hypothesis – Chapter 6

With the publication of Dr. Key’s Seven Country Study, the origins of the Diet-Heart Hypothesis were laid down. The major problem was that this was all observational data, and as such, was subject to severe interpretation. There is nothing more dangerous in medicine than the correlation study. It cannot be used to prove a hypothesis, only to disprove one.

**Diet-Heart Hypothesis**

For example, looking at the same data as Dr. Keys, others came to **completely different conclusions**. While saturated fat was correlated to heart disease, most of it was the influence of animal fats. Vegetable fats were not correlated at all. But it wasn’t merely animal fats, but also animal protein that had correlation to heart disease. So maybe it was animal products – ie. red meat dilemma. Keep in mind that this does not exonerate vegetable fats. Most people were eating nut oils and olive oil, not the highly processed corn oil, canola and vegetable oils that would come to dominate the American supermarket shelves.

However, it is also possible that animal consumption was merely a marker for industrialization. Counties with higher levels of industrialization tended to eat more animal products – meat and dairy. So, are you saying that the more Westernized countries had more Western diseases? Hmm... Seems kind of obvious, that. I rather favour that as the likely explanation.

There are actually multiple hypotheses that could have come out of the Seven Countries Study. Maybe the culprit was indeed saturated fat. Maybe it was animal fat. Maybe it was animal protein. Maybe it was industrialization and processed foods. Maybe it was total protein. Maybe it was carbohydrate. All of these hypotheses could have been generated from the same study. But what we got was the Diet-Heart Hypothesis.
To some extent, this has persisted to this date. Most people still believe in the Diet-Heart Hypothesis as laid out originally in 1970. The Lipid hypothesis refers to the hypothesis that serum cholesterol causes heart disease. The Diet-Heart hypothesis refers to the hypothesis that dietary saturated fat raises cholesterol and therefore raises heart disease. There are actually problems with all parts of this hypothesis.

The first problem faced was the fact that total serum cholesterol turned out not to be correlated to heart disease at all. There was very weak correlation with death and total cholesterol. In fact, in the oldest age groups, high cholesterol was strongly protective. This was shown in multiple other studies. Total cholesterol was not very good at all. This problem was solved by looking at the ‘good’ cholesterol HDL versus the ‘bad’ cholesterol LDL. This seemed to fix things up a bit better.

If anything, there was a stronger correlation between low HDL and heart disease than LDL. The dietary manipulations tended to move both HDL and LDL up or down rather than individually. Drugs, such as torcetrapib were developed at massive expense to raise HDL levels. Unfortunately, these drugs all killed people and were abandoned. Pfizer spend close to $1 billion dollars to develop this drug all based on the same faulty correlation data. Soon afterwards, thousands of people were laid off. Low HDL did not cause coronary disease, it was only a marker for the disease.

Looking back, it all seems so obvious. Exercising raises HDL. Maybe people who exercised had both higher HDL and lower heart disease. Increased olive oil increases HDL. Maybe olive oil both raises HDL and lowers heart disease. Stopping smoking raises HDL. Maybe stopping smoking both raises HDL and lowers heart disease. This emphasizes again the danger of correlation studies.

Nevertheless, scientists moved forward trying to prove that dietary saturated fat increased cholesterol levels. The Framingham studies were the ideal place to look.
In 1948, Harvard University decided to undertake extensive testing in Framingham, Mass. The blood work and dietary habits of an entire community would be monitored over decades to learn about the risk factors of heart disease. Every 2 years, all residents would undergo screening with blood work and questionnaires. By comparing those that developed heart disease and those that did not, they hoped to learn the factors that were important.

In the early 1960s, after massive human effort and expenditure the results of the Framingham Diet Study were available. Hoping to find a definitive link between saturated fat intake, serum cholesterol and heart disease. Instead what they found was no such thing at all. Nada. Nothing. There was absolutely no correlation. So they did what all researchers the world over do. They buried the result. Ignore the result and pretend the study never existed.

Dr. Michael Eades, writing on his blog, was able to find an old copy and peruse the results. From his copy, he notes that the report states:

There is no indication of a relationship between dietary cholesterol and serum cholesterol level.
At least this part is consistent. Dr. Ancel Keys noted much the same thing. Physiology also backs up the fact that most of the cholesterol in our bodies is made by our own livers.

Even today, the National Cholesterol Education Program (NCEP) states:

Dietary cholesterol causes marked hypercholesterolemia in many laboratory animals, including nonhuman primates. High intakes of cholesterol in humans, however, do not cause such a marked increase in serum cholesterol
Translation – Non human primates should DEFINITELY avoid dietary cholesterol. Humans, though, not so much.

Regarding fat:

No association between percent of calories from fat and serum cholesterol level was shown; nor between ratio of plant fat to animal fat intake and serum cholesterol level.
Remember that original studies also showed no association of total fat and cholesterol. It was only saturated fat that made a difference. However, here, unlike the Seven Countries Study, animal versus plant fat made no difference.

The most important part is the relationship with heart disease. Here’s the final conclusions of this forgotten jewel:
There is, in short, no suggestion of any relation between diet and the subsequent development of CHD in the study group.

Yeah. There was basically no relationship between diet and heart disease. So much for the Diet-Heart Hypothesis. This should have been the study to bury this mistaken hypothesis. Instead, researchers chose to bury the study instead. They condemned us to 40 years of a low fat future that included an epidemic of diabetes and obesity.

What is really sad is the news commentary by Dr. William Kannel, the director of the Framingham Heart Study. He states that, just because his own study proved that diet has no connection with cholesterol, “it is incorrect to interpret this finding to mean that diet has no connection with blood cholesterol,”. Dude – you makin’ no sense.

Over the next half century, no matter how hard we looked, there was no relationship between dietary fat and serum cholesterol. Other studies, such as the Tecumseh study, continued to find no discernible relationship. Even the National Cholesterol Education Program admits that “Dietary cholesterol causes marked hypercholesterolemia in many laboratory animals, including nonhuman primates. High intakes of cholesterol in humans, however, do not cause such a marked increase in serum cholesterol”.

So non human primates should DEFINITELY avoid dietary cholesterol. Humans, though, not so much.
Trans Fats – Chapter 7

Doesn’t dietary fat ‘clog up’ the arteries and cause coronary disease? It would seem from popular press that this has been proven beyond a shadow of a doubt.

Perhaps we had better take a closer look. The Diet-Heart Hypothesis suggests that diets high in saturated fats lead to high cholesterol which leads to heart disease. In our last post, we’ve seen that there is actually no connection between high fat diets and high blood cholesterol levels. The connection between high blood cholesterol and heart disease is also a bit dodgy, only to be saved by the distinction between ‘good’ and ‘bad’ cholesterol.

Dr. Ancel Keys, the prominent nutritionist was one of the first men to theorize about a relationship between dietary fat and heart disease. This was based on his correlation studies between nations. While he most certainly cherry picked the data to strengthen his argument, nevertheless there was a relationship there.

This, in no way, means that dietary fats caused heart disease. Any number of factors could contribute. The increase in dietary fat may have been a marker for industrialization. The more industrialized a nation, the higher the rate of heart disease.

Exercise in Futility

<table>
<thead>
<tr>
<th>Study</th>
<th>Journal/Author</th>
<th>Conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>A Longitudinal Study of Coronary Heart Disease</td>
<td>Circulation.1963; 28: 20-31 Oglesby P</td>
<td>1,989 patients followed over 4 years. No relationship of dietary fat to CAD</td>
</tr>
<tr>
<td>Diet and Heart: A Post Script</td>
<td>BMJ 1977; 2(6098): 1307-14 Morris JN</td>
<td>No relationship of CAD to dietary fat</td>
</tr>
<tr>
<td>Diet, Serum Cholesterol, and Death from Coronary Disease: The Western Electric Study</td>
<td>NEJM 1981: 304; 65-70 Shekelle RB</td>
<td>1900 patients over 20 years. No relationship of CAD and saturated fats</td>
</tr>
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Other research groups sought to replicate these results. Despite another two decades of data, there was no relationship between dietary fat
and heart disease to be found.

This is despite some trials boasting over 10,000 patients and over trials lasting over 20 years long.

But the myth persisted. No amount of data could persuade the diehards that dietary fat did not cause heart disease.

Moving into the modern age, it was recognized that one of the major problems in much of the older literature was the grouping of trans-fats with saturated fats. What are trans fats and how did they come to be?

Trans-fats are made from poly-unsaturated vegetable oils. Most vegetables are not naturally oily. Olives and coconuts are exceptions, which is why these two oils predominate in much traditional cooking in those parts of the world. However, corn is not particularly oily. It requires significant amounts of processing in order to get enough oil from them.

The oil that comes out is polyunsaturated, meaning that there are multiple ‘holes’ where hydrogen is ‘missing’. Saturated fats, like many animal fats are saturated with hydrogen. Because these are saturated, they are more stable and also tend to be a solid at room temperature. The polyunsaturated fats with their ‘holes’ are not as stable and therefore tend to go rancid.

In 1902, Wilhelm Normann discovered that you could bubble hydrogen into vegetable oil and saturate it with hydrogen. This essentially turned the polyunsaturated fat into a saturated fat. On labels, it is often called partially hydrogenated vegetable oil – yeah, that’s trans fat. This made the fat more stable chemically. It changed the oil into a semi-solid at room temperature – that’s great for spreadability. This made it more appealing to food processors since it improved mouth feel and also increased shelf life. Trans fats were great for deep frying as well. You could use this stuff over and over and not have to change it. The saturation of the hydrogen bonds made this much more stable chemically than the polyunsaturates. The other key piece was that this stuff was CHEAP. Using leftover soybeans from animal feed, manufacturers could process the heck out it and get oil. A little hydrogen, a little chemistry, and boom – trans fats, baby. So what if it killed millions of people from heart disease? That knowledge was years in the future.

Crisco, the shortening made from hydrogenated vegetable oil debuted in 1911. This would replace lard – the rendered fat from a pig in many recipes. The manufacturer, Procter and Gamble was quick to point to the ‘wholesomeness’ of an all vegetable product and Crisco soon
entered pantries everywhere. One of the main attributes is the shelf life stability of the product. I have some that is about 5 years old that looks the same as the day I bought it.

By the 1960s, as the nation began to worry about heart disease, the trans fats were starting to really hit their stride. Dr. Ancel Keys, cover boy of Time magazine, proclaimed saturated fats as the main cause of heart disease. Trans fats were quick to point out that they were processed from polyunsaturated fats – the ‘heart healthy’ fat. Since many animal products also got the blame for heart disease, the all vegetable nature was touted as ‘heart healthy’ as well.

So, use of saturated fats – beef fats and pig fats – gradually decreased. Butter was under intense fire as well. Polyunsaturated fats, with their rancidity and short shelf life were not suitable for processed foods. The logical alternative was the trans fats. These were called partially hydrogenated vegetable oils. So they retained the healthy sheen of being a vegetable oil, but were the completely artificial Frankencreation of science gone awry.

Margarine, another completely artificial food, embraced trans fats like a long lost lover. The two wholly fake foods were made for each other. McDonalds and other fast food chains switched from frying in ‘unhealthy’ but delicious beef tallow to frying in trans-fat vegetable oils. The major sources of trans fats were deep fried and frozen foods, packaged bakery products, crackers, vegetable shortening, and margarine.

The beginning of the end of the trans fat reign started in 1990, when Dutch researchers noted that trans fats increased LDL, the ‘bad’ cholesterol and lowered HDL the ‘good’ cholesterol. From there, it was an endless parade of bad news for the trans fat. By 2006, an American Heart Association poll showed that 84% of consumers had heard of the associated health risks. The FDA demanded food labels. Dr. Willett estimated that a 2% increase in trans fat would result in a 23% increase in risk of heart disease. Trans fats were contributing to thousands of heart attacks and strokes.

In a sense, this story plays out just like baby formula. We marvel at how conceited we were to believe that we could make a better baby milk than Mother Nature. Yet, here we were, trying to come up with a healthier fat than fat. Natural fats, including saturated ones are the fats that our bodies have evolved to eat. There is nothing inherently unhealthy about it. Just as breast milk is what our babies have naturally evolved to eat.
While this is a marvellous cautionary tale, the relevance is that for many decades, the health effects of trans fats and saturated fats were lumped together because trans fats are saturated. However, the health effects of natural saturated fats are nothing like the artificial trans fats at all. Maybe saturated fats are not as dangerous as might have believed.
Saturated Fats – Chapter 8

Saturated fat has been linked to heart disease since the 1960s. With the realization that trans fats and saturated fats were not similar in any way, researchers strove to separate the effects. Margarine was one of the major sources of trans fats. The study “Margarine intake and subsequent heart disease in Men” looked at the risk of heart disease compared to the intake of margarine, using data from the Framingham study, which had prospectively collected data on the entire population of Framingham, Mass. Comparing margarine consumption with 20 year follow up data, they tabulated the results.

The results were a surprise. After all, margarine for decades had boasted about being low in saturated fat especially compared to that bovine killer, butter. Hell, the bestseller Becel was based on the acronym BCL – Blood Cholesterol Lowering. It had always prided itself on being ‘heart healthy’.

The more ‘heart-healthy’ margarine people ate, the more they got heart attacks. The more ‘artery-clogging’ butter they ate, the less heart attacks they got. Wait. Wasn’t it supposed to be the other way around?

Butter may have been full of saturated fat (which isn’t so bad after all), but margarine was full of trans fats. Which was worse? The trans fats were much worse by a country mile. If trans fats are completely unlike saturated fats, then previous studies that had analysed them together may have come to invalid conclusions.

In order to look at the question of dietary fat aside from the trans fats, Dr. Hu analyzed the large Nurse’s Health Study which followed 80,082 nurses over 14 years.

The women were divided into 5 different groups based on their intake of total fats. The risk of coronary artery disease (CAD) was then monitored for each group. As the women ate more and more
fat, there was no corresponding increase in the risk of heart disease after the effect of trans fats was removed. The final conclusion of his study was that, “Total fat intake was not significantly related to the risk of coronary disease”.

The same conclusion was reached for dietary cholesterol. There was no statistical increase in the risk of heart disease with higher consumption of dietary cholesterol. Remember, too, that previous studies had established that dietary cholesterol contributes little or nothing to blood levels of cholesterol.

Recent analyses have come to much the same conclusions. In 2009 in the Annals of Internal Medicine, “A Systematic Review of the Evidence Supporting a Causal Link Between Dietary Factors and Coronary Heart Disease” looked at all trials to date for the link.

Looking at all available trials up to 2009, there was no evidence found to support a link between total fat and heart disease. Saturated fats was not associated. Neither was polyunsaturated fats. In other words, there was no link at all. Saturated fats were not bad. Polyunsaturated fats (vegetable oils) were not good. There was simply no link at all.

With regards to obesity, there was really no evidence either. The main concern with dietary fats had always been the ‘epidemic’ of heart disease. Obesity concerns were kind of ‘thrown in’ as well. When people decided that dietary fat was bad for the heart, they also reasoned that it must be bad for weight gain as well.

### Saturated Fat

**Risk of Heart Attack**

*BMJ 1996;313:84 Ascherio A*
well. However, there was never any data to support this assumption. Never assume, kids. Makes an ASS out of U and ME.

Even the National Cholesterol Education Program admits that “recent prospective studies (or meta-analysis of studies) have failed to detect a causative link between (percentage of dietary fat and obesity)”. Translation – despite 50 years of trying to prove that dietary fat causes obesity, we still cannot find any evidence. The reason this data is so hard to find is that it was never there in the first place.

Data slowly started to energy that perhaps saturated fat was not bad for us, but instead was actually good for us. In the 1996 study “Dietary fat and risk of coronary heart disease in men: cohort follow up study in the United States”, 43,757 health professionals were followed over 10 years and their risk of heart attack was compared to the consumption of saturated fats. Again, dividing the subjects by amount of saturated fat eaten, there were 5 groups from lowest to highest intake.

Lo and behold, increased intake of saturated fat was not bad for you, it was good for you. What’s that crazy talk? Saturated fat was good for you. The highest risk of heart disease was in the group with lowest intake of saturated fat.

If we switch gears slightly to talk about risk of stroke, there were several studies on that too. The Oahu study “Dietary and Other Risk Factors for Stroke in Hawaiian Japanese Men” followed 7,895 men on the island of Oahu over 10 years. Dietary records were carefully monitored for fat, protein and sodium.

Again, the men were divided into 5 groups depending upon the intakes of fat, protein and sodium separately. Sodium, or salt has long been demonized as causing high blood pressure therefore increase risk of heart disease and stroke. It turns out that there really is no correlation at all. Those taking the most salt had no more risk of stroke than those taking the least.

Both protein and fat showed protective effects. That is, the more protein and fat one took, the lower the chance of stroke. Correlation
studies such as these cannot prove that fat or protein is protective. However, they can disprove the hypothesis that fat is bad for us. If dietary fat was such an important factor in stroke, then there should have at least been some correlation. But there was not. The relationship went in the wrong direction.

In the 20 year follow up to the Framingham data, the exact same protective effect of fat on stroke was seen. The 1997 study “Inverse association of dietary fat with development of ischemic stroke in men”. Dividing the group by intake of dietary fat, it was found that those eating the most fat had the least strokes. Those with the lowest fat had the most strokes. Again here, eating fat was not bad, it was good.

Looking more closely into the types of fat, it was found that saturated fats were protective, but that poly-unsaturated fats (vegetable oils) were not. Those ‘heart-healthy’ vegetable oils were not protective where the ‘unhealthy’ saturated fats were. Monounsaturated fats (olive oil) was also protective. Once again, we enter the Bizarro world of nutrition, where good is bad and bad is good.
As Dr. Keys warned Americans about the dangers of saturated fat, consumption of animal fats and butter declined.

The main component of animal fats is saturated fats. Omega 6 fat content is vanishingly low. Both butter as well as beef tallow are similar in composition.

Vegetable oils such as corn, by contrast have low level of saturated fats, but are very high in polyunsaturated fats, the majority of which is omega 6. There is virtually no omega 3 fats here.

Going back to the Seven Countries Study, the emphasis of modern nutrition until recently was to replace saturated fats with polyunsaturates. This at first led to the explosion of trans fats. Once that danger was recognized, it no longer appeared that saturated fats were dangerous at all.

Over the ensuing decades, the consumption of animal fat continued to fall. After remaining stable from 1900-1950, it began a relentless decline under government exhortations to reduce saturated fats.

The dialogue began to change beginning in the late 1990s due to popularity of higher fat diets such as the Atkins diet. Indeed, Dr. Atkins wrote in the “New Diet Revolution” that, “The thirty-year-long campaign against dietary fat is as misguided as it is futile”. True that, Dr. Atkins. True. That. As he pointed out on his website, all foods contain all three types of fat – saturated, monounsaturated and polyunsaturated. The proportions of the three do vary.
The unintended consequence was that intake of omega 6 oils increased significantly. To be more precise, this was the intended consequence. It was unintentionally detrimental to human health.

Omega 6s are a family of polyunsaturated fats. These fatty acids are converted to eicosanoids such as prostaglandins and leukotrienes. These are highly inflammatory mediators. This is not good.

Atherosclerosis, or the buildup of plaque in the arteries was originally considered to be cholesterol sludge depositing in the arterial walls. This has long been known to be untrue at least since the time I was in medical school in the early 1990s. The pathophysiology of atherosclerosis is mainly dependent upon inflammation and thrombosis in the wall of the artery. The sludge deposits as a ‘response to injury’. Thus the plaque is the response to the inflammation. The real problem is the inflammation.

Increasing inflammatory molecules could conceivable contribute to worsening heart attack and strokes. Omega 3 fats tend to oppose the effects of Omega 6 fats and therefore increasing attention is being paid to the ratio of these fats. Vegetable oils have extremely high Omega 6:3 ratios.

It is estimated that humans evolved eating a diet that is close to equal in Omega 3s and 6s. However, the current Western diet is estimated to be closer to a 15:1 ratio. Either we are way under eating omega 3,
way over eating omega 6s, or more likely, both.

Omega 3 fatty acids decreases thrombosis (blood clots) by its role in platelet aggregation, bleeding time and blood viscosity. Low rates of heart disease were originally described in the Inuit population, where, despite high intakes of fat, they had very low rates of heart disease. Subsequently, it was found that all major fish eating populations tended to have lower rates of heart disease. Additional studies seemed to confirm that addition of omega 3 to the diet could displace the omega 6 from cell walls. However, this will be very difficult if the ratio of Omega 6:3 is 30:1.

The massive increase in consumption of omega 6’s in the diet can be traced to technological advances in the 1900s that allowed modern methods of making vegetable oil. The invention of the continuous screw press (Expeller), steam-vacuum deodorization and solvent extraction techniques were critical to the production of vast quantities of vegetable oils.

Another culprit is the rise of the industrial farm and the feedlot cattle. Wild animals tend to be very lean, and contain high levels of polyunsaturated fats. Feedlot cattle, however, are fed grains at every turn. Indeed, there is nothing for the cattle to do all day, but eat. Cows, of course, are ruminants and designed to eat grass. Feeding them corn does wonders to fatten them, but increases omega 6 content of their meat significantly. So our grains, our meats, and our oils are omega 6 based. This all contributed to the massive imbalance in the Omega 6:3 ratio. The Canadian nutritional guidelines were the first in 1990 to recognize the difference and include specific recommendations for both types of fatty acids.

The other consequence of the scare about animal fats was that America increasingly reached for that tub of edible plastic – margarine. With large advertising budgets designed to play up its
wholesome all-vegetable origins, trans fat laden margarine has always professed itself to be a healthier alternative to butter largely based on its low saturated fat.

Margarine has an interesting history. Designed by a French chemist in 1869 as a cheap butter alternative, it was originally made from beef tallow and skim milk. A Dutch company, eventually absorbed into Unilever bought the rights and proceeded to market the new substance. Margarine is actually an unappetizing white, but since it was designed as a butter alternative, it was dyed yellow. Butter manufacturers, though were not amused. Using taxes and laws against dying, margarine was a marginal product for decades. Its big break came with World War II and the ensuing butter shortage. Most of the taxes and laws against margarine were repealed since butter was scarcely available anyway.

This paved the way for the great margarine renaissance in the 1960s and 70s as the war on saturated fats gained ground. It is ironic that this ‘healthier’ alternative turned out to be chalk full of trans fats that were, actually killing people. Thankfully, most manufacturers no longer make margarine with trans fats and have tried to blend more healthful ingredients like olive oil in. Thus, where previous studies had linked margarine use to heart disease, it is questionable whether modern studies will still show this effect. However, it still doesn’t change the fact that butter just tastes way, way better.

It’s actually a minor miracle that vegetable oils were considered healthy at all. Consider the substantial amount of processing – pressing, solvent extraction, refining, degumming, bleaching and deodorization – that is required to squeeze the oil from a non-oily vegetable such as corn. There is virtually nothing natural about it.

It could only have become popular during an era where artificial was considered good. Artificial orange juices like Tang. Baby formula. Jello. Canned soups. This was a time we considered ourselves to be smarter than Mother Nature. Whatever she had made, we could make better. So out

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### Industrial Vegetable Oils

1. **Seeds** - de-hulled, de-skinned and ground into meal and heated
2. **Pressing** - Meal fed into a screw press, under pressures up to 20,680 kilopascals
3. **Solvent extraction** - Hexane used to dissolve oil, which is then recovered by distilling
4. **Solvent Removal** - Oil is boiled by steam, and the lighter hexane floats upward
5. **Refining** - Heat oil to 85°C and mix sodium hydroxide. Soap forms and is removed by centrifuge.
6. **Degumming** - Treating them with 95°C water, steam, or water with acid. The gums (phosphatides), precipitate out, and the drags are removed by centrifuge.
7. **Bleaching** - filtered through fuller’s earth, activated carbon, or activated clays
8. **Deodorization** - steam is passed over hot oil in a vacuum at 250°C. Citric acid at 0.1 percent is also added to oil after deodorization to inactivate trace metals.
9. Enjoy “all-natural, healthy” vegetable oil!
with all natural butter. In with industrially produced, artificially
coloured trans fat laden margarine! Out with naturally produced lard
from pigs. In with industrially processed, solvent extracted, bleached
and deodorized vegetable oil! What could possibly go wrong?
Cardio-protective effect of Saturated Fats – Chapter 10

Saturated fats turned out to be not quite as toxic as we had believed once the trans fats were stripped out. But many studies pointed out the fact that there is a chance that they may actually be protective against heart disease. The study “Dietary fats, carbohydrate, and progression of coronary atherosclerosis in postmenopausal women” examined the angiographic progression of coronary disease in relationship to diets.

An angiogram is a standard heart test where dye is put into the arteries around the heart to determine if there are any blockages. Looking at 235 women with established heart disease, they measured baseline angiograms and repeated them in an average of 3.1 years. They then looked at the diets of these women to see if there were any patterns.

Dividing these women into groups according to their intake of fats, there appears to be no relationship whatsoever. The women eating the most fat had slightly less blockages than those eating the least.

The one macronutrient that stands out as particularly sinister is carbohydrates. The highest intake of carbohydrates is associated with the highest level of heart blockages. Not looking too good for the Diet-Heart hypothesis. The people who were eating the American Heart Association approved low fat diet were getting the heart disease. The people eating the frowned-upon high fat diets were not.

The types of dietary fat were further divided to see if the all natural saturated fats were indeed dangerous to human health. The group eating the most saturated fat had regression of their blockages. Their heart disease was melting away! On the other hand, the group eating the least saturated fats had the most progression of their disease.

Looking at the industrial vegetable oils, it appears that eating more of these oils tended to be associated with more progression. Wait a friggin second. Weren’t these vegetable oils supposed to be ‘heart-healthy’? Wasn’t the saturated fats supposed to be clogging arteries? It looks like the effect is the exact opposite of what we were taught. Saturated fats weren’t bad, they were good for health.

Another large study “Dietary fat intake and early mortality patterns – data from The Malmo Diet and Cancer Study” followed over 29,000 subjects over 6.6 years. Among the women, increasing saturated fat intake was associated with a significantly lower risk of dying of heart disease. Again, not looking too good for the Diet-Heart
Hypothesis. Eating saturated fat doesn’t really appear to be harmful in any way.

The same results were seen in the study "Dietary intake of saturated fatty acids and mortality from cardiovascular disease in Japanese". Following a cohort of 58,543 Japanese men and women over 14.1 years, the researchers looked at the the relationship between heart disease, stroke and saturated fat intake. Not surprisingly, there was an inverse relationship. That means that eating more saturated fat protects against heart disease and stroke. In 2014, the Japan Collaborative Cohort Study found virtually identical results. From their conclusions, “total mortality was inversely associated with intakes of saturated fatty acids (SFA)”. Yes, saturated fat is not bad for you, it’s good for you.

Several large scale and highly publicized meta-analyses have been done on all trials to date regarding saturated fat. In 2009, Dr Krause published, “Meta-analysis of prospective cohort studies evaluating the association of saturated fat with cardiovascular disease”. After careful analysis of 21 studies covering 347,747 patients, he concluded:

there is no significant evidence for concluding that dietary saturated fat is associated with an increased risk of CHD. Furthermore, there was a small but significant protective effect on stroke.

Dr. Chowdhury in his paper “Association of Dietary, Circulating, and Supplement Fatty Acids With Coronary Risk: A Systematic Review and Meta-analysis” came to much the same conclusions. While trans fat increased the risk of coronary disease, saturated fat did not. There was no concern that polyunsaturated fats were causing disease. Yet.

Polyunsaturated fats contain both omega 3s and omega 6s. While both are found in nature, our current diet may be heavily and unhealthily skewed toward omega 6s. Dr. Ramsden decided to analyze all the studies to date regarding polyunsaturated fats, but divided the fat and published the paper, "Use of dietary linoleic acid for secondary prevention of coronary heart disease and death".
The overall picture showed no increase in risk of death with polyunsaturated fat. However, upon closer inspection, you can see that those trials that increased omega 3 consumption showed protection against heart disease. Those studies that increased omega 6 consumption increased heart disease. The omega 6, if you remember is primarily the vegetable oils that we consume far in excess of the omega 3 by a factor of 15 to 30.

Awwww. Snap. Nutritionism strikes again. All polyunsaturates are not the same. We are belatedly recognizing the fact that highly inflammatory, processed vegetable oils may not be ‘heart healthy’ after all. More likely they are ‘heart harmful’. And margarine just changed from trans fats to vegetable oils….. The advice to change from saturated fat to vegetable oil may not have been good.

The only long term randomized trial “Test of Effect of Lipid Lowering by Diet on Cardiovascular Risk” by Dr. Frantz, looked at the effect of switching had shown this decades earlier. The Minnesota Coronary Survey randomized...
9,057 inmates at a psychiatric facility to follow a standard diet with 39% fat (18% saturated, 5% polyunsaturated, 16% monounsaturated, 446 mg cholesterol) to a 38% fat diet much lower in saturated fats (9% saturated, 15% polyunsaturated, 14% monounsaturated, 166 mg cholesterol). This was achieved by changing to egg substitute, margarine and low fat beef – all delicious, artificial foods. Over a follow up period of 4.5 years, they could not demonstrate any benefit in terms of heart disease.

However, they were able to demonstrate a difference in total mortality. It was just not the one they were looking for. The treatment group was dying at a faster rate than the control group eating all the saturated fat. What? Eating artificial, man-made, industrially processed substitute food is not good for you? How can that possibly be?
Part II
Cortisol
Cortisol – Chapter 11

I can make you fat. Actually, I can make anybody fat. How? It is very simple. I prescribe prednisone, a synthetic version of the human hormone cortisol. Prednisone is used to treat many different types of inflammatory diseases, including asthma, rheumatoid arthritis, lupus, psoriasis, inflammatory bowel disease, cancer, glomerulonephritis and myasthenia gravis. Cortisol makes you fat. Not coincidentally, both insulin and cortisol play a key role in carbohydrate metabolism.

Cortisol

Cortisol is the so-called stress hormone. It mediates the ‘flight or fight response’ with help from the sympathetic nervous system. Cortisol is part of a class of steroid hormones called glucocorticoids (glucose + cortex + steroid) produced in the adrenal cortex. Cortisol is produced in response to stress. In Paleolithic times, this was often a physical stress, such as being chased by a predator. The release of cortisol was essential in preparing our bodies for action – to fight or flee. Cortisol increases alertness and decreases the need for sleep.

Glucose availability is substantially enhanced. This provides energy for muscles that are needed to avoid being eaten. Non-essential metabolic activities are curtailed. All available energy is directed towards surviving the coming stressful period. Growth, digestion and other long-term issues are temporarily restricted. Proteins are broken down and converted to glucose (gluconeogenesis). In the fasted state, cortisol has several mechanisms to increase glucose in the body.

The blood glucose raising effect of synthetic cortisol prednisone has been known for at least 40 years. These include:

1. Stimulation of hepatic gluconeogenesis
2. Inhibition of glucose uptake in peripheral tissues
3. Stimulation of fat and amino acid breakdown (helps provide substrate for hepatic gluconeogenesis)

Vigorous physical exertion (fight or flight) soon followed using up these newly available stores of glucose. Shortly thereafter, we were either dead, or the danger was past. In either case, the cortisol levels decreased again back to low levels. The body is well adapted to the short-term increase in cortisol and glucose.

Cortisol raises insulin
At first glance cortisol and insulin appear have opposite effects. Insulin is a storage hormone. Under high insulin levels, the body stores energy in the form of glycogen and fat. Cortisol, on the other hand prepares the body for action. This moves energy out of stores and into readily available forms such as glucose. That they would have similar weight gain effects seems remarkable. With short-term physical stress, insulin and cortisol play opposite roles. This situation is quite different for long-term psychological stress.

In modern times, chronic, non-physical stressors increase cortisol. For example, marital issues, problems at work, arguments with children, and sleep deprivation are all serious stressors, but do not result in vigorous physical exertion afterwards to lower blood glucose. Under conditions of chronic stress, glucose levels remain high. There is no vigorous physical exertion to burn off the glucose, and there is no resolution to the stressor. The blood glucose can remain elevated for months. This chronic elevation in glucose can trigger the release of insulin. Chronically elevated cortisol leads to increased insulin. This has been demonstrated in several studies.

One study measured people repeatedly during a random working day. Cortisol increases with self-perceived stress levels. This stress-related increase in cortisol showed consistent strong relationship to both increased glucose and increased insulin levels (16). Since insulin is the major driver of obesity, it should be no surprise that it was also related to both BMI and abdominal obesity.

Using synthetic cortisol, we can also increase insulin experimentally. Healthy volunteers were given 50mg cortisol four times daily over 5 days. Insulin levels rose 36% from baseline. Another study showed that the use of prednisone increases glucose levels by 6.5% and insulin levels by 20%. Over time, insulin resistance also develops mainly at the hepatic level. There is a direct dose-response relationship between cortisol and insulin. For every unit of free cortisol increase, insulin increased by 9.7 mU/I.

Long-term use of prednisone may lead to an insulin resistant state or full-blown diabetes. The increased insulin resistance seen in type 2 diabetes leads to elevated insulin levels. Even five years after the cure of Cushing’s disease, the elevated insulin levels persist. This is likely related to the insulin resistance syndrome that has developed. This is another mechanism where excess cortisol leads to increased insulin.

Glucocorticoids produce insulin resistance in skeletal muscle by interfering with numerous steps in the insulin-signaling network. The molecular mechanisms have been mapped out including decreased
IRS-1, and increased levels of proteins PTP1B and p38MAPK. These interfere with insulin action after it binds the insulin receptor. In addition, muscles release amino acids for gluconeogenesis, increasing insulin resistance. Adiponectin, secreted by fat cells, which normally increase insulin sensitivity, are suppressed by glucocorticoids.

In a way, insulin resistance should be expected, since cortisol generally opposes insulin. Cortisol raises blood sugar where insulin lowers it. This insulin resistance, as we will see in later chapters is crucial in the development of obesity. Insulin resistance will lead directly to increased insulin levels. Increased insulin is a major driver of obesity. Multiple studies show that increasing cortisol confirms this insulin resistance.

If cortisol raises insulin, then reducing cortisol should reduce insulin. We find this situation in transplant patients who are maintained on synthetic cortisol for years or decades as part of their anti-rejection medications. Weaning off the prednisone resulted 25% drop in plasma insulin. This translated to a 6.0% weight loss and a 7.7% decrease in waist girth.

The cardiovascular consequences of excess cortisol sound suspiciously like those of insulin excess. This is also known as the metabolic syndrome and includes high blood sugar, blood pressure, cholesterol and abdominal obesity. Cushing’s syndrome also includes high insulin, blood sugars, blood pressure, cholesterol and truncal obesity.

Cortisol and obesity

Does excess cortisol, from long-term psychological stress lead to weight gain? Certainly anecdotal evidence seems to suggest that stress leads to obesity. But there are certain disease states characterized by excessive cortisol production. This is called Cushing’s disease or Cushing’s syndrome. Harvey Cushing originally described a 23-year-old woman in 1912 that suffered from weight gain, excessive hair growth and loss of menstruation. High blood sugars and overt diabetes is present in up to 1/3 of cases. Patients taking long term prednisone often appear similar to these patients and develop what is termed Cushinoid syndrome. There is a particular redistribution of the fat from the limbs to the trunk and face called truncal obesity. The term ‘moon face’ is used to describe the peculiar gain of weight in the face. A ‘buffalo hump’ describes the deposition of fat in the back.

But the hallmark of this disease is weight gain. In case series, 97% of patients show a central weight gain, and 94% show increased body weight. Insulin resistance is another key characteristic of Cushing’s syndrome. Both cortisol and prednisone cause weight gain. Many
patients complain that they gain weight no matter how little they eat and no matter how much they exercise. Any disease that results in excess cortisol secretion results in weight gain. **Cortisol causes weight gain.**

Subclinical Cushing’s syndrome is associated with obesity. These patients can be found by blood testing, but do not yet have symptoms of the disease. **Fasting glucose and insulin were similarly elevated in these patients. Cortisol causes weight gain.**

However, this effect is seen even within the normal population, without evidence of Cushing’s syndrome. In a random sample from North Glasgow, Scotland, cortisol excretion rates were strongly correlated to Body Mass Index (BMI) and waist measurements. Higher cortisol levels were seen in heavier people. Cortisol related weight gain particularly deposits fat in the abdomen, which results in an increased waist/hip ratio (WHR). This weight distribution of fat in the abdomen is more dangerous to the health than generalized fat.

Other measures of cortisol confirm the association with abdominal obesity. People with higher urinary cortisol excretion have higher waist to hip ratios. People with higher cortisol in the saliva have increased BMI and waist/hip ratio. In other words, there is substantial evidence that chronic cortisol stimulation increases both insulin and obesity. Long-term exposure to cortisol in the body may be measured by scalp hair analysis. In a study comparing obese patients to normal weight, researchers found elevated levels of scalp hair cortisol.

The ultimate test is this. Can I make somebody fat with prednisone? If I can, this proves a causal relationship, rather than a mere association. Does prednisone cause obesity? Absolutely! Weight gain is one of the most common and well-known side effects of the medication. This is a causal relationship. Giving high doses of prednisone causes weight gain. We raised cortisol. People gained weight. **Cortisol causes weight gain.**

What about the opposite? If high cortisol causes weight gain, then low cortisol levels should cause weight loss. This exact situation exists in the case of Addison’s disease. Also known as adrenal insufficiency, Thomas Addison described this classic condition in 1885. Cortisol is produced in the adrenal gland. When it is damaged, cortisol levels can drop very low. The major cause of Addison’s disease is autoimmune destruction. Previously, tuberculosis had been a major cause of the syndrome.
The hallmark of Addison’s disease is **weight loss**. In large case series, 97% of patients exhibit weight loss. Cortisol levels went down. People lost weight. *Cortisol causes weight gain*.

Cortisol may act through high insulin levels and insulin resistance. There may be other pathways of obesity yet to be discovered. However, the fact that is undeniable is that excess cortisol causes weight gain. By extension, stress causes weight gain. This is something that many people have intuitively understood despite the lack of rigorous evidence. It certainly makes sense. Much more sense than calories causing weight gain.

**Hormonal Obesity**

![Diagram of hormonal obesity](image)

Reducing stress is difficult, but vitally important. Contrary to popular belief, sitting in front of the television or computer is a poor way to relieve stress. Instead, stress relief is an active process. There are many time-tested methods of stress relief. These include mindfulness meditation, yoga, massage therapy, and exercise. Studies on mindfulness intervention were able to use yoga, guided meditations, and group discussion to **successfully reduce cortisol and abdominal fat**.
Sleep Deprivation - Chapter 12

One of the major causes of chronic stress in the modern environment is sleep deprivation and disturbance. Even a single day of sleep deprivation can increase cortisol levels by over 100%. Sleep duration has been declining over the last few decades. In 1910, people slept 9 hours on average. By 1960, surveys indicated that Americans were averaging 8.0-8.9 hours of sleep per night, falling further to 7 hours by 1995. More than 30% of adults between 30 and 64 report less than 6 hours of sleep. Shift workers are especially prone to sleep deprivation and often report less than 5 hours of sleep per day.

Population studies have consistently shown a relationship between short sleep duration and excess weight, generally with less than 7 hours the cutoff. Both cross sectional studies from Spain, Japan and the US, and longitudinal studies such as the Nation Health and nutrition Examination Survey (NHANES I) and the Women’s Health Study finding this association. The Quebec Family Study suggested an increased risk of 27% with shorter sleep duration. A 13-year prospective study even suggested that every extra hour of sleep was associated with a 50% reduction in risk of obesity. A one-year prospective study showed that sleeping less than 5 hours per night was associated with a 91% increase risk of obesity. Sleeping 5-6 hours was associated with at 50% increased risk. A meta-analysis of 696 studies published in 2008 showed that short sleep duration increased the risk of obesity by 55% in adults and 89% in children. For every hour of sleep deprivation, BMI rose by 0.35 kg/m2.

From a caloric expenditure perspective, this does not necessarily make sense. Sleeping less should increase energy expenditure since any activity used more calories than sleep. Calorie theory would suggest that sleep deprivation leads to ‘moving more’ and lower likelihood of obesity. However, the opposite is true, showing once again the inadequacy of caloric theory in general and the ‘eat less, move more’ paradigm.

Sleeping more than 8 hours per night may also increase risk of obesity. The Western New York Health Study also found that sleeping 6-8 hours per night was associated with the lowest risk of obesity. ‘Excessive’ sleeping over 8 hours increased the risk by 60% but too little sleep (<6 hours) tripled the risk of obesity.

Mechanisms

Sleep deprivation is a potent psychological stressor and thus stimulates cortisol resulting in both high insulin levels and insulin
resistance. Cortisol raises blood sugar and activates the sympathetic nervous system. Sleep deprivation causes increased cortisol levels and sympathetic tone. Sleep deprivation resulted in 37-45% higher cortisol levels by the next evening. Glucose use by the brain decreases during sleep deprivation and likely contributes to the mental fogginess that we all experience. This can be measured by positron emission tomography (PET). Restriction of sleep to 4 hours in healthy volunteers resulted in a 40% decrease in glucose tolerance. The glucose response to breakfast was, in fact, high enough to classify these previously normal individuals as having pre-diabetes. Cortisol increased close to 20%.

Other studies have confirmed that it is possible to induce insulin resistance in normal volunteers simply with sleep restriction to four hours per night, even with a single night. After 6 days of sleep restriction, there was a 50% decrease in insulin sensitivity. In a Japanese study, shortened sleep duration increased risk of type 2 Diabetes.

Both leptin and ghrelin, key hormones in the control of body fatness and appetite show a circadian rhythm and are disrupted by sleep disturbance. Leptin rises slightly during sleep and ghrelin, the hunger hormone, tends to fall. Thus, hunger is suppressed despite maintenance of the fasting during sleep. The Wisconsin Sleep Cohort Study demonstrated the effects of sleep duration on these important hormones. The Quebec Family study also found that short sleep duration was associated with higher body weight, decreased leptin and increased ghrelin.

Leptin increases steadily with more sleep. Higher leptin levels regulate body fat downwards making thinner. Ghrelin, the hunger hormone, steadily falls with more sleep. Lower ghrelin causes less hunger. Sleep deprivation results in higher ghrelin and hunger. Sleep deprivation of only 4 hours for two nights increased ghrelin by 28% and reduced leptin by 18% with accompanying increased hunger and appetite (39). Who can deny the 'late-night munchies'? Fast food restaurants are very familiar with this phenomenon and often now offer 24-hour service.
Sleep deprivation clearly will undermine weight loss efforts (40). Adequate sleep is not only essential for restoring brain function, but also to prevent the metabolic consequences of high cortisol and insulin resistance. Interestingly, sleep deprivation under low stress conditions does not decrease leptin or increase hunger. This suggests that it is not the sleep loss per se that is harmful, but the activation of the stress hormones and hunger mechanisms. Getting enough good sleep is essential to any weight loss plan.

Sleep deprivation causes stress. But stress may also cause sleep deprivation. Increased cortisol or prednisone therapy, for instance often causes insomnia due to activation of the sympathetic ‘fight or flight’ system. Patients often describe the sensation of ‘too much energy’. This is a classic vicious cycle. We see this at work in obesity as well. Obesity causes the problem of obstructive sleep apnea, where patients momentarily stop breathing during sleep. Repeated episodes cause tremendous disruption of normal sleep. This sleep deprivation then increases stress, which leads to more obesity.

An interesting natural experiment on sleep deprivation occurred in South Korea. A 10 pm curfew was enforced on late-night tutoring schools. Subsequent studies estimated that each 1 hour increase in sleep duration lead to a 0.56 kg.m² reduction in BMI and a 4.3% reduction in obesity in the affected grades.

There are several keys to good sleep hygiene, none that involve the use of medications. These disturb the normal sleep architecture, the pattern of REM and non-REM sleep. Simple, but effective ways to improve sleep include:

- Sleep in complete darkness
- Sleep in loose fitting clothes
- Keep regular sleeping hours
- 7-9 hours a night
- See the light first thing in the morning
- Keep bedroom slightly cool
- No TV in bedroom
Part III
How to Lose Weight
How to Lose Weight – Chapter 13

Once we understand that insulin is the key player in the development of obesity, we can begin to treat it. Insulin causes obesity so the key to the treatment of obesity is to lower insulin. Obesity is not a caloric imbalance, it is a hormonal imbalance. Obesity is a disease of too-much-insulin. Therefore, the treatment is to lower insulin. This, of course, is easier said than done.

It is not simply a matter of lowering calories, or lowering carbohydrates, or lowering sugar or lowering processed foods or or increasing fibre or increasing fruits and vegetables. No, it is a matter of doing all of these things that decrease insulin levels. There are two main factors that lead to increased insulin levels. The first factor is the foods that we eat. Certain foods tend to raise insulin more than others. There are also foods that protect against insulin spikes. This is the question which we obsessively think about "What to Eat". Should we eat low calories, low carbohydrate, low fat, low animal protein, high fat etc?

But there is an entirely separate stimulus to insulin levels that does NOT depend entirely upon what we eat. This factor is insulin resistance. High insulin resistance will lead to high insulin levels. While fructose does play a role in increasing resistance, there are many other factors as well. This is the entirely separate question of "When to Eat". This question is virtually ignored in the vast expanse of obesity literature both on the internet and in academic journals. Insulin resistance develops over time. This explains the time dependent factor of obesity. Most people become obese at a rate of only 1-2 pounds per year.

There are two prominent findings from all the dietary studies done over the years. First, all diets work. Second, all diets fail. What do I mean? Weight loss follows the same basic curve so familiar to dieters the world over. Whether it is the Mediterranean, the Atkins, or even the old fashioned low fat, low calorie, all diets in the short term seem to produce weight loss. Sure, they differ by the amount – some a little more, some a little less. But they all seem to work. However, by 6 months to 1 year, weight loss gradually plateaus followed by a relentless regain despite continued dietary compliance. This occurs regardless of the dietary strategy. In the 10 year Diabetes Prevention Program, for example there is a 7 kg weight loss after one year. The dreaded plateau, then weight regain, follows. So all diets fail. The question is why.
Permanent weight loss is actually a two-step process. There is a short-term and a long-term (time dependent) problem. This resistance to weight loss represents *homeostasis*. The hypothalamic region of the brain determines the Body Set Weight (BSW). This is our fat ‘thermostat’. Insulin acts here to set BSW higher. In the short term, we can use various diets to bring our actual body weight down. However, once below the BSW, the body activates mechanisms to regain that weight. This resistance to weight loss was first demonstrated by Drs. Leibel and Hirsch in 1984. Obese persons that had lost weight require fewer calories. Their metabolism had slowed dramatically. The body is actively resisting long-term weight loss. This widely known fact has been both proven scientifically and empirically.

Imagine that you set your house thermostat low, and you are cold. You plug in a small electric heater. Soon, the house starts to warm up. Any brand of electric heater seems to work. All heaters work. This is the short-term solution to the problem. After a while, the thermostat senses that the temperature has gone up. So it turns on the air conditioning to bring the temperature back down. Eventually, after a seesaw battle, the house always wins. The temperature eventually drifts down and we are cold again. This is the long-term problem. All heaters fail. The problem is homeostasis. While we have adjusted the temperature, we have not adjusted the thermostat.

Now, let’s put this into obesity terms. High insulin levels set the BSW ‘thermostat’ at a weight that is too high. Now we decide to lose weight. Following any reasonable diet reduces weight in the short term. This is the quick fix – just like the electric heater. What happens in the long term?

The problem of insulin resistance (time dependent factors) has not been addressed. The insulin resistance keeps insulin high. The BSW is still set at a very high level. The body responds to the weight loss by raising the body weight back up. Hormonal signals of hunger are increased, compelling us to eat. If that doesn’t work, total energy expenditure (TEE) is reduced. This was exactly the experience of the participants in the Minnesota Starvation Experiment. As metabolism decreases, it becomes harder and harder to lose weight. Eventually, after a seesaw battle, the BSW wins. The end result is all too familiar – weight regain. The problem is homeostasis.

So there are actually two separate questions to lasting weight loss. There is both a short-term and a long-term question. The short-term question is “What to Eat”. The longer-term problem is why all diets fail. This is the problem of insulin resistance and resetting the BSW. This question revolves around “When to Eat”. While these two
questions are related, they must both be addressed to develop a comprehensive solution to obesity.
The MultiFactorial Nature of Obesity – Chapter 14

The *multifactorial* nature of obesity is the crucial missing link. There is no one single cause of obesity. Do calories cause obesity? Yes, partially. Do carbohydrates cause obesity? Yes, partially. Does fiber protect us from obesity? Yes, partially. Does insulin resistance cause obesity? Yes, partially. Does sugar cause obesity? Yes, partially. All these factors converge on several hormonal pathways that lead to weight gain. Insulin is the most important of these hormonal pathways. Low carbohydrate diets reduce insulin. Low calorie diets restrict all foods and therefore reduce insulin. Paleo diets (low in refined and processed foods) reduce insulin. Cabbage soup diets reduce insulin. Reduced food reward diets reduce insulin.

![Hormonal Obesity Diagram]

Virtually all diseases of the human body are multifactorial. Consider cardiovascular disease. Family history, age, gender, smoking, diabetes, high blood pressure, and lack of physical activity all influence, perhaps not equally, the development of heart disease. Cancer, stroke, Alzheimer’s disease and chronic renal failure are all multifactorial diseases. That smoking contributes to heart disease does not mean that high blood pressure does not. Both are correctly identified as important factors. Just because one is correct does not mean that the other is incorrect. It would thus be idiotic to compare a treatment strategy of stopping smoking against treating high blood pressure.
We should not expect obesity to be any different. Obesity is also a multi-factorial disease. What is required is a framework, a structure, a coherent theory to understand how all these factors fit together. Too often, our current model of obesity assumes that there is only one single true cause, and that all others are pretenders to the throne. There are endless debates about the true king. Too many calories cause obesity. No, too many carbohydrates. No, too much saturated fat. No, too much red meat. No, too much processed foods. No, too much high fat dairy. No, too much wheat. No, too much sugar. No, too much highly palatable foods. No, eating out. It goes on and on. They are all partially correct.

So the Low Calorie believers disparage the Low Carbohydrate people. The Low Carbohydrate movement ridicules the Vegans. The Vegans mock the Paleo supporters. The Paleo followers deride the Low Fat devotees. All diets work, because they all address a different aspect of the disease. But none of them work for very long, because none of them address the totality of the disease. Can’t we all just get along?

Without this critical understanding of the multifactorial nature of obesity, we are doomed to this endless cycle of blame. Similarly, most dietary trials are fatally flawed by this tunnel vision. The various trials comparing low carbohydrate to the low calorie diets have all asked the wrong question. These two diets are not mutually exclusive. What if both are true? Then there will be similar weight loss on both sides. Low carb diets lower insulin. Lowering insulin levels reduces obesity. However, all foods raise insulin to some degree. Since refined carbohydrates often make up 50% or more of the Standard American Diet, low calorie diets generally reduce carbohydrates as well. So low calorie diets, by restricting the total amount of foods, still work to lower insulin levels. Both will work.

That is exactly what Harvard professor Dr. Sacks confirmed in his randomized study of four different diets. Despite differences in carbohydrate, fat and protein content, weight loss was the same. Maximum weight loss occurred at six months, with gradual regain thereafter. A 2014 meta-analysis of dietary trials reached much the same conclusion. “Weight loss differences between individual diets were minimal”. Sure, sometimes one diet comes off as slightly better than another. The difference is often less than one kg and often fades by a year. Let’s face it. We’ve done Low Calories Low Fat. It didn’t work. We’ve done Atkins. It was not the effortless weight loss that was promised. While important lessons were learned, it was not the right answer. Carbohydrates are only a single factor in a multi-factorial disease. We’ve done high protein diets. We’ve done cabbage soup
diets. We’ve done cookie diets. All these diets address only a single factor in a multi-factorial disease. Further, they completely ignore the question of “When to Eat” as well as the time dependent factors of obesity.

Sometimes these results are interpreted as the belief that everything can be eaten in moderation. This does not even begin to address the complexity of weight gain in the human subject. It is essentially a cop-out answer. It is a deliberate attempt to evade the hard work of searching for dietary truths. For example, should we eat broccoli in the same moderation as ice cream? Obviously not. Should we drink milk in the same moderation as sugar sweetened beverages? Obviously not. The long-recognized truth is that certain foods must be severely restricted. This would include sugar sweetened beverages and candy, for instance. Other foods do not need to be reduced in any way – kale or broccoli, for instance. Other foods, may be best taken in moderation – animal meat.

Others have erroneously concluded that ‘it is all about the calories’. Actually, it is nothing of the sort. Calories are only a single factor in the multifactorial disease that is obesity. It seems to be a default answer, but it has already been shown to be disastrously incorrect. After all, caloric reduction has proved itself a dismal failure over the last 50 years. Low calories diets have been tried again and again. It failed every single time.

There are other answers that are not really answers. These include, ‘There is no best diet’ or ‘Choose the diet that suits you’ or ‘The best diet is one you can follow’. If supposed experts in nutrition and disease don’t know the right diet, how are you supposed to? This is intellectual laziness at its very worst, revealing a stunning lack of imagination about the problem of obesity. Does this mean that following the Standard American Diet is the best diet for me, because it is the one I can follow? Does this mean I can eat a diet of sugared cereals and pizza? Obviously not. ‘Experts’ who are too mentally sluggish in the quest for truth reach for the facile and incorrect answer that ‘All diets are equal’. This pure intellectual laziness is unacceptable. We don’t need another ‘expert’ give us the lame-duck answer “All diets work. Do whatever you want”.

In cardiovascular disease, for example, this answer would never be considered satisfactory. If the lifestyle factors of stopping smoking and increased physical activity both reduce heart disease, then we would strive to do both. We would NOT say, “The best lifestyle for heart disease is the one you can follow”. Unfortunately, many so-called ‘experts’ in obesity profess this exact sentiment.
Imagine three men who all study trees. One man looking at the trunk proclaims that trees are large, rounded and rough. Another man looking at the branches declares that trees are small, rounded and smooth. The third man, studying only the leaves, said that trees are small, flat and green. All three are at once correct and incorrect; just as each factor in a multi-factorial disease is at once right and wrong. In cardiovascular disease, we can see the tree. In obesity we cannot. Calories, carbohydrates, processed foods, sugar, fast foods, fibre, vinegar, fat, protein, and meat are all important parts of the obesity tree.

Most diets attack a single part of the problem at a time. But why is it necessary to address one facet at a time? In cancer treatment, for example, multiple types of chemotherapy and radiation are combined together. The probability of success is much higher with a simultaneous attack. In cardiovascular disease, multiple drug treatments work together. We use drugs to treat high blood pressure, high cholesterol, diabetes, and smoking cessation – all at the same time. Treating high blood pressure does not mean ignoring cholesterol. In challenging infections such as HIV, a cocktail of different anti-viral medicines are combined together for maximum efficacy.

The same approach is necessary to address the multidimensional problem of obesity. Rather than targeting a single point in the obesity cascade, we need multiple targets and treatments. But the Low Carbohydrate proponents don’t want to hear about calories, meat, or insulin resistance. For them, it is all about carbohydrate restriction. But all foods, even low carbohydrate ones contribute to rising insulin levels. The Low Calorie devotees don’t want to hear about carbohydrates, sugars, meats, or dairy. For them, it is all about caloric restriction. You can eat ice cream for dinner if it has the same number of calories as a large salad. We don’t need to choose sides. Rather than compare a dietary strategy of low calorie versus low carbohydrate, why can’t we do both? There is no reason.
Avoid Miracle Cures – Chapter 15

The Internet is full of promises of the latest and greatest miracle ‘drug’ or food that will miraculously melt away stubborn fat. Raspberry ketones, green coffee extract, Yacon syrup, Garcinia Cambogia, African mango, saffron extract, Sea Buckthorn, and Capsiberry.

Here’s the plain truth. The entire notion of a miracle weight loss aid is completely preposterous. We only believe it because we want to believe it. Deep in our hearts, we know that it cannot be true. Humans have been eating plants and herbs for at least 5000 years. What are the chances that a completely natural and new substance is suddenly found in year 2014 that wondrously melts away fat? This is pure science fiction. Mostly these supplements rely on the well-known placebo effect for all their benefits.

Similarly, the notion of the ‘super food’ is faintly ridiculous. These are no easy answers. We imagine that there are foods that are so good, that eating them automatically makes us ‘super’ healthy. We are hoodwinked into believing that a berry from the Amazon or a seed from Mexico might be enough to turn the tides of obesity and diabetes. Of course, we only need to look at the 1950s for our answer. They were not eating chia seeds or quinoa. They didn't even much like whole wheat bread. Whole-wheat pasta was entirely unknown. Yet obesity and diabetes were much less of a problem then.

Those foods that are super healthy for us to eat have been discovered long, long, long ago. These are the whole, unprocessed foods. Things like nuts, seeds, fruits, vegetables, olive oil, fish, and wild game. We would have known about a super-food centuries ago. Even things that taste rather bad, like bitter melon, have properties that make it a food that our paleolithic ancestors chose to eat. It tastes really bitter. But they kept eating it.

Sure enough, in 2014, Applied Food Science (AFS), the company producing green coffee bean extract settled with the Federal Trade Commission (FTC) for $3.5 million. Under intense scrutiny, the 2012 study that reported the belly busting ability of green coffee was retracted. It was a bogus study. The investigators had admitted to fudging the data. Others would call it a flat-out lie.

AFS had hired some Indian scientists to study the weight loss effects of the green coffee extract. But the researchers repeatedly changed the weights of the patients and trial groups. The trial was so horrifically bad that no journal would publish it. So, AFS decided to hire Drs. Joe Vinson and Bryan Burnham from the University of
Scranton to rewrite the paper and put their own names on it to give it a sheen of respectability. This sort of intellectual prostitution, unfortunately, is not that uncommon.

Without verifying any of the data, Dr. Vinson wrote the paper and proclaimed that subjects could lose 17.5 pounds in 22 weeks or 10.5% of body weight. This was all done without changing their diet, but only with the addition of some green coffee extract. Dr. Vinson, presenting at the 2012 American Chemical Society meeting, explained that “Based on our results, taking multiple capsules of green coffee extract a day — while eating a low-fat, healthful diet and exercising regularly — appears to be a safe, effective, inexpensive way to lose weight”. In a press release, he even goes as far as to say that subject might have lost even more weight if they had not been on placebo for part of the study.

Once the bogus study was complete, a plausible reason needed to be found for this miraculous effect. So, it was decided that green coffee was high in chlorogenic acid. Presumably, this was the reason for its miraculous effect. Let’s forget the fact that chlorogenic acid is high in potatoes as well – a food not particularly well known for its slimming effects. Let’s also forget the fact that chlorogenic acid has never previously been hinted to have a weight loss effect. Green coffee has chlorogenic acid. Green coffee causes weight loss. Therefore, its due to the chlorogenic acid. Dr. Oz swallowed it hook, line, and sinker. In the bloody arena of American daytime television, a miracle weight loss cure meant viewers. So what if it didn’t really work?

Sensa was another food additive that claimed miraculous waist slimming properties in late night TV ads. It was created by Dr. Alan Hirsch, a neurologist. Sensa was a crystal that you sprinkled onto food. Dr. Hirsch claimed that these crystals he created would make you full and therefore you would lose weight.

Sensa ran on late night TV ads for a long time. Infomercials claimed that studies had ‘proven’ its benefit. So what if the studies were
entirely made up? Dr. Hirsch claimed that a study done by the Endocrine Society supported the 30 pound weight loss claim. The Endocrine Society, on the other hand, didn’t know what he was talking about.

Eventually, it became obvious that Sensa was just a giant scam. The makers settled with the FTC in January 2014 for $26.5 million. By October 2014, the Sensa was bankrupt and out of business.

There was also the case of HCG Diet Direct, which settled for $7.3 million. It, too was just a gigantic scam. Kevin Trudeau, a promoter of a misleading book entitled “The Weight Loss Cure ‘They’ don’t want you to know about”, was sentenced to 10 years in prison. We laugh at ‘primitive’ people’s beliefs in voodoo and witchcraft. All the while, we drink our raspberry ketones and green coffee extract. Pot, meet kettle. Avoid miracle cures. Weight loss begins with an understanding of what causes weight gain.
first step in virtually any weight loss program is to reduce added sugars. Sugar is particularly fattening because it increases insulin both immediately and insidiously over the long term. Sugar is comprised of equal amounts of glucose and fructose. Glucose, as a highly refined carbohydrate, will immediately raise blood sugar levels and directly stimulate insulin. Fructose does not raise blood glucose levels, but contributes directly to insulin resistance in the liver. Over time, that insulin resistance will lead to higher insulin levels.

This makes sucrose and high fructose corn syrup exceptionally fattening.

This is fairly uncontroversial advice. Sugar is empty calories, but it is far more sinister than that. Sugar stimulates insulin secretion, but it is far more sinister than that. Sugar is uniquely fattening because it directly produces insulin resistance. With no redeeming nutritional qualities, added sugars are usually one of the first foods to be eliminated in any diet. Sugar is merely an indulgence. However, avoiding sugar is easier said than done. Because sugars are everywhere in processed food, a surprisingly large amount may be ingested without realization. Obviously, the first step is to remove the sugar bowl from the table. There is no reason to add sugar to any food or beverage. But sugars are often hidden in the preparation of food.

Many natural, unprocessed whole foods contain sugar. For example, fruits contain fructose and milk contains the natural sugar lactose. A
distinction is usually made between naturally occurring and added sugars. The two key differences are the amount and concentration. Fruit contains far less fructose than processed foods. They also contain vitamins and nutrients along with a large amount of soluble and insoluble fibre (the ‘pulp’). This dietary fibre may act as an ‘antidote’ to the harmful effects of fructose. The bulking effect of the fibre prevents overeating.

Sugars are often added to foods during processing or cooking. Because they are not naturally present in whole foods, several potential dangers lurk. First, added sugars may be added in unlimited amounts. There is no upper limit to the amount of sugar that may be added. Second, sugar may be concentrated much higher than natural foods. Some foods are virtually 100% sugar. This situation virtually does not exist in natural foods, with honey possibly being the only exception. Candy is often little more than flavored sugar. Third, sugar may be ingested by itself. This may cause people to overeat these sugary treats, as there is nothing else to make you ‘full’. There is no dietary fibre that may offset the harmful effects. For these reasons, we direct most of our efforts towards reducing added sugars, as opposed to naturally occurring sugars.

**Refined and processed foods**

Almost ubiquitous in refined and processed foods, sugar is not always labeled as such. Other names include sucrose, glucose, fructose, maltose, dextrose, molasses, hydrolyzed starch, honey, invert sugar, cane sugar, glucose-fructose, high fructose corn syrup, brown sugar, corn sweetener, rice/ corn/ cane/ maple/ malt/ golden/ palm syrup and agave nectar. These aliases attempt to conceal the presence of large amounts of added sugars. Another popular trick is to use several different pseudonyms on the food label. This prevents sugar from being listed as the first ingredient. For example, an ingredient list may contain 3 different sugars such as sugar, brown sugar and corn syrup. Because the sugar content is now split into 3, the first ingredient listed is now .... Whole grain wheat. Ta Da! This example comes from a sugared breakfast cereal.

The addition of sugar to processed foods has almost magical flavor enhancing properties at virtually no cost. Sauces are serial offenders. Barbeque, plum, honey garlic, hoisin, sweet and sour and other dipping sauces contain
large amounts of sugar. Spaghetti sauce, for example may contain as much as 10-15 grams of sugar (3-4) teaspoons. This counters the tartness of the tomatoes, and therefore may not be immediately evident to your taste buds. Commercial salad dressings and condiments, such as ketchup and relish often contain lots of sugar. The bottom line is this. If it comes in a package, then it likely contains added sugar.

How much sugar is acceptable? There is no correct answer to this. It is similar to asking the question – how many cigarettes are acceptable? Ideally, the number is zero, but that likely will not happen.

**Desserts**

Most desserts are easily identified and eliminated from the diet. These foods are mostly sugar in various forms with complementary flavors added. Examples include cakes, puddings, cookies, pies, mousses, ice cream, sorbets, candy, and candy bars.

So what is available for dessert? It is best to follow the example of traditional societies. The best desserts are fresh seasonal fruits, preferably locally grown. A bowl of seasonal berries or cherries with whipped cream is a delicious way to end a meal. A small plate of nuts and cheeses also produces a very satisfying end to a meal, without the burden of the added sugars. Certainly there are naturally occurring sugars, but these do not present the same danger as added sugars.

Dark chocolate with more than 70% cacao, in moderation, is a surprisingly healthy treat. The chocolate itself is made from cocoa beans and does not naturally contain sugar, but most milk chocolate contains large amounts of sugar. Dark and semisweet chocolate contain less sugar than milk or white chocolate. Dark chocolate also contains significant amounts of fibre and antioxidants such as polyphenols and flavanols. Studies on dark chocolate consumption confirm they may help reduce blood pressure, insulin resistance and heart disease. Most milk chocolates, by contrast are little more than candies. The cacao component is too small to be of benefit.

Nuts are a great choice for an after dinner indulgence. Most nuts are full of healthful monounsaturated fats, have little or no carbohydrates, and are also high in fibre, increasing the potential health benefits. Macadamia nuts, cashews, and walnuts can all be enjoyed. Furthermore, increased nut consumption has been linked in many studies to better health including less heart disease and diabetes.
Pistachio nuts, high in the anti-oxidant gamma-tocopherol, vitamins like manganese, calcium, magnesium and selenium, are widely eaten in the Mediterranean diet. A recent Spanish study found that adding 100 pistachios daily improved fasting glucose, insulin and insulin resistance.

That is not to say that sugar cannot be an occasional indulgence. Food has always played a major role in celebrations – birthdays, weddings, graduations, Christmas, Thanksgiving etc. The key word here is occasional. These are special treats to be enjoyed occasionally. We celebrate with a birthday cake, not a birthday pork chop.

Dessert is not to be taken every day. But if your goal is weight loss, the first major step is to severely restrict sugar. Replacing sugar with artificial sweeteners is not a good idea either. Since sweeteners also raise insulin as much as sugar, they are equally prone to cause obesity. Sweeteners give the false promise of sweetness without consequence.
Breakfast is, without question, the most controversial meal of the day. The advice to eat something, anything as soon as you step out of bed has often been regurgitated by health professionals without question. People confess to skipping breakfast like they’ve committed a great nutritional crime.

Breakfast really needs to be downgraded from “most important meal of the day” to “meal”. Different nations have different breakfast traditions. The big “American” breakfast contrasts directly with the French “petit dejeuner” or “small lunch”. The key word here is ‘small’. Obesity tends to be a much larger problem, of course, in the United States.

The greatest problem is that breakfast foods are often little more than dessert in disguise, containing vast quantities of highly processed carbohydrates and sugar. Breakfast cereals, particularly those targeted towards children, are among the worst offenders. On average, they contained 40% more sugar than those targeted towards adults. Not surprisingly, almost all cereals for children contained sugar, and 10 contained more than 50% sugar by weight. Only ten of 181 (5.5%) met the standard for “low sugar”. In the diets of children under 8, breakfast cereals rank only behind candy, cookies, ice cream and sugared drinks as a source of dietary sugar. However, while those other foods are obviously desserts, breakfast cereal masquerades as a healthy food, helped along with vast marketing budgets. Adding whole grains may improve the healthy image, but it doesn’t reduce the sugar content.

While cereal has been declining in popularity since the mid 1990s, it still managed $10 billion in sales in 2013, down from $13.9 billion in 2000. Declining birth rates
means fewer children, a key demographic. The rapidly growing populations of aging baby boomers are less likely consumers. Increased ethnic populations have their own traditional breakfast foods. Recently popular diets including the low carbohydrate, gluten free diet, Paleo, and whole foods diets all shun the breakfast cereal. A simple rule to follow is this. Don’t eat sugared breakfast cereal. If you must, eat cereals with less than 4 grams of sugar per serving or less.

Many breakfast items from the bakery are also thinly disguised desserts. Examples include muffins, cakes, Danishes, and banana bread. Not only do they contain significant amounts of refined carbohydrates, they are often sweetened with sugars and jams. Bread often contains sugar, and is eaten with sugary jams and jellies. Peanut butter often contains added sugars as well. Made traditionally from just peanuts, they are often mixed with honey or other sweeteners.

Traditional and Greek yogurts are nutritious foods. However, commercial yogurts are thinly disguised desserts with large amounts of sugar and fruit flavorings. A serving of Yoplait fruit yogurt contains 27 grams (almost 7 teaspoons of sugar). Oatmeal is another traditional and healthy food. Whole oats and steel cut oats require long cooking times because they contain significant amounts of fibre that requires heat and time to break down. This too has been corrupted into the thinly disguised dessert known as instant oatmeal. Heavy processing and refining allows instant cooking, and large amounts of sugar and flavors are added. Most of the nutritional content was lost long ago. Quaker’s flavored instant oatmeal may contain 13 grams of sugar per serving (3 ¼ teaspoons of sugar). Instant cream of wheat experiences the same problems. A single serving has 16 grams of sugar (4 teaspoons of sugar). With rolled oats and dried fruit, granola and granola bars attempt to disguise themselves as healthy. They are often heavily sugared and contain extra chocolate chips or marshmallows.
So, what to eat for breakfast? If you are not hungry – then don’t eat anything at all. It is perfectly acceptable to break your fast at noon with a piece of grilled salmon with a side salad. Technically, this will be your ‘break fast’. There is nothing inherently wrong with eating breakfast in the morning either. It is just like any other meal. However, in the morning rush, there is a tendency to reach for convenient prepackaged, heavily processed, and heavily sugared foods. Eat whole, unprocessed foods at all meals, including breakfast. If you find that you do not have time to eat – then don’t. Again – simplify your life. The notion that there is something mystical about eating immediately upon waking is perplexing.

Eggs, previously shunned due to concerns about dietary cholesterol, can be enjoyed in a variety of ways – scrambled, over easy, sunny side up, hard-boiled, soft boiled, poached etc. Egg whites are high in protein, and the yolk contains many vitamins and minerals, including choline and selenium. **Eggs are particularly good sources of lutein and zeaxanthin**, antioxidants that may help protect against eye problems like macular degeneration and cataracts. The cholesterol in eggs may actually help cholesterol profile by **changing particles to the larger, less atherogenic particles**. Indeed, large epidemiologic studies have **failed to link increased egg consumption to increased heart disease**. Most of all, eat eggs because they are delicious, whole, unprocessed foods.

**Snacks**

The healthy snack is one of the greatest weight loss deceptions. The ‘grazing is healthy’ myth has attained legendary status. Constant stimulation of insulin eventually leads to insulin resistance. Why people ever thought ‘grazing’ was healthy, I don’t know for sure. This advice is directly opposite of virtually all food traditions. Even as recently as the 1960’s most people still ate three meals per day. Snacking was met with an icy glare. “You’ll ruin your dinner”. Grandma, of course was right. Snacking just makes you fat.

This idea that ‘grazing’ is beneficial likely started with diabetics taking insulin. Normally, the body constantly adjusts insulin for the foods eaten. Artificially injecting insulin created the problem of volatile blood sugars. Diabetics need to match the food to the insulin injections, rather than the other way around. By eating constant smaller meals, blood sugars could more easily be matched to the insulin injection regimen.

Somehow, without anybody really noticing, it was decided that if diabetics ate like this, then everybody else should as well. No trials or studies were done. There was no scientific debate. Eventually, healthy
'grazing' became engrained into nutritional lore. Food companies, of course, could not be more thrilled. They encouraged the extra meal at every turn. Cha ching. Cha ching. It was all cash in the bank for them. The problem was that people were eating too much. The solution was not to eat all the time.

Snacks are often little more than thinly disguised desserts. Most contain prodigious amounts of refined flour and sugar. These pre-packaged convenience foods have taken over the supermarket shelves. Cookies, muffins, pudding, Jello, fruit roll ups/fruit leather, chocolate bars, cereal bars, granola bars and biscuits are all best avoided. Rice cakes advertising themselves as low fat, compensate for lack of taste with sugar. Canned or processed fruit conceal buckets of sugar behind the healthy image of the fruit. A serving of Mott’s Applesauce contains 21g (5 ½ teaspoons of sugar). A serving of Dole canned peaches contains 18 grams (4 ½ teaspoons of sugar).

Are snacks necessary? No. Simply ask yourself this question. Are you really hungry, or just bored? Keep snacks completely out of sight. Out of sight, out of mind. If you have a snack habit, then it is best to try to replace that habit loop with one that is less destructive to your health. Perhaps a cup of green tea in the afternoon should be your new habit. There’s a simple answer to the question of what to eat at snack time. Don’t eat snacks. Period. Simplify your life.
Beverages – Chapter 18

The sugar-sweetened beverage is one of the leading sources of added sugars. This includes all soda pop, sugar sweetened teas, fruit juice, fruit punch, vitamin water, smoothies, shakes, lemonade, chocolate or flavored milk, iced coffee drinks and energy drinks. Hot drinks such as hot chocolate, moccachino, caffé mocha, and sweetened coffee and tea would also be included. Trendy alcoholic drinks add significant amounts of sugar. This includes “Hard’ lemonade, flavored wine coolers, ‘cider’ beers, as well as more traditional drinks such as Bailey’s Irish Cream, margaritas, daiquiris, pina coladas, dessert wines, ice wines, sweet Sherries, and liqueurs. Artificially sweetened drinks, as noted previously are not better than their regular counterparts.

So what is left to drink? The best drink is really just plain or sparkling water. Slices of lemon, orange or cucumber are a refreshing addition. Several traditional and delicious drinks are also available.

Coffee

Legend has it that coffee was discovered by an Ethiopian goatherd that astutely noticed that his goats were extra frisky after eating the red fruit of the coffee tree. After reporting it to a local monastery, monks turned the fruits into a brewed drink to help them stay awake during long hours of prayer. From these legendary beginnings, coffee would spread first through Arab traders to Europe and then America. Today, it is enjoyed almost worldwide with local variations in brewing and drinking methods.

Due to the high caffeine content, it is sometimes considered an unhealthy habit. However, recent research has come to the opposite conclusion. Coffee is a major source of antioxidants. It is also a rich source of magnesium and lignans which are suggested to benefit many different areas of human health.

Coffee appears to have a protective role against type 2 Diabetes. In a 2009 review, each additional daily cup of coffee lowered the risk of diabetes by 7%, even up to six cups per day. The European Prospective Investigation into Cancer and Nutrition (EPIC) sub study estimated that drinking at least 3 cups of tea or coffee daily reduced
the risk of diabetes by 42%. The Singapore Chinese Health Study showed a 30% reduction in risk for more than 4 cups per day of coffee. This protection is evident even in decaffeinated coffees, suggesting that much of the benefit derives from antioxidants.

Coffee appears to reduce total mortality as well. A large 2012 analysis of the AARP Diet and Health Study found total mortality reduced by 10-15% in those drinking six cups of coffee. Other large-scale studies such as the Nurses’ Health Study and Health Professionals Follow Up Study found that most major causes of death, including heart disease were reduced. Coffee may guard against the neurologic diseases Alzheimer’s, Parkinson’s disease, liver cirrhosis and liver cancer.

Store beans in an airtight container away from excessive moisture, heat and light. Flavor is lost quickly after grinding, so investing in a reliable grinder is worthwhile. Grind beans immediately before brewing. On hot days, iced coffee is simple and inexpensive to make. Simply brew a pot of regular coffee and cool in the refrigerator overnight. Cinnamon, coconut oil, vanilla extract, almond extract and cream may be used to flavor coffee without changing its healthy nature. Avoid adding sugars or sweeteners.

**Tea**

Legend holds that the Emperor of China discovered tea in 2737 BC. He had been enjoying a cup of boiled water when leaves from a nearby tree blew into his drink. As the water changed color, the emperor sipped the brew and was surprised by the pleasant taste. For centuries afterwards, royalty enjoyed tea for its medicinal and religious purposes. Tea spread to Japan in the 6th century, where green tea would be developed and popularized. Tea did not reach English shores until the 17th century, where it gained popularity among the aristocratic class.

After water, tea is the most popular beverage in the world. There are several basic tea varieties. Black tea is the most common, making up almost 75% of global consumption. Harvested leaves are fully fermented giving it the characteristic black color. Black tea tends to be
higher in caffeine. Oolong tea is ‘semi-fermented’ meaning that it undergoes a shorter period of fermentation. Green tea is a ‘non-fermented’ tea. The freshly harvested leaves immediately undergo a steaming process to stop fermentation, giving it a much more delicate and floral taste. Green tea is naturally much lower in caffeine than coffee, which makes this drink ideal for those who are sensitive to its stimulant effects.

Green tea owes much of its beneficial effects due to large concentrations of a group of powerful antioxidants called catechins, notably one called EGCG (epigallocatechin-3-gallate). Fermentation changes the catechins to a variety of theaflavins, making green tea a richer source than black or oolong teas. The theaflavins in black tea may also have beneficial health effects, although different from those of green tea. The antioxidant potential of green tea and black tea are comparable. Polyphenols in the tea are also believed to boost metabolism, which may aid in ‘fat burning’. Many health benefits have been ascribed to green tea consumption, including increased fat oxidation during exercise, increased resting energy expenditure (34), lower risk of various types of cancer.

A meta-analysis of studies confirms that green tea helps with weight loss, although the benefit is rather modest in the range of 1-2 kg. Catechins may play a role in inhibiting carbohydrate digestive enzymes resulting in lower glucose levels, and protecting the pancreatic beta cell. The Singapore Chinese Health study showed that drinking more than one cup of black tea per day reduced the risk of diabetes by 14%. Other studies demonstrated an 18% decrease in the risk of Type 2 Diabetes for those drinking 3-4 cups per day.

All teas may be enjoyed both as hot or cold beverages. There are infinite varieties of tea available to suit any taste. Flavors can be added with the addition of lemon peel, orange peel, cinnamon, cardamom, vanilla pods, mint and ginger may all be added to any type of tea.

Herbal teas are infusions of herbs, spices or other plant matter in hot water. They make excellent drinks without added sugars, and can be enjoyed hot or cold. These are not true teas since they do not contain tea leaves. The varieties are endless. Some popular varieties include mint, chamomile, ginger, lavender, lemon balm, hibiscus, and rosehip teas. The addition of cinnamon or other spices can enhance the flavor.

**Bone Broth**

Virtually every culture’s culinary traditions include the nutritious and delicious bone broth. Animal bones are simmered with the addition of
vegetables, herbs and spices for flavoring. The long simmering time (4-48 hours) releases most of the minerals, gelatin and nutrients. The addition of a small amount of vinegar during cooking helps leach some of the stored minerals. Bone broths are very high in amino acids such as proline, arginine and glycine as well as minerals such as calcium, magnesium and phosphorus.

Animal bones are often available at ethnic grocery stores and fairly inexpensive. They are also very convenient, requiring little preparation time. They can be made in large batches and frozen. Upon cooling, broth may congeal like Jello due to the high gelatin content. Most commercially prepared broths have nothing in common with the homemade variety. Prepackaged broths often rely on artificial flavors and MSG to provide taste. The minerals, nutrients and gelatin are not present in many canned broths.
Wheat – Chapter 19

Wheat is one of the most vilified foods in the nutritional world. From gluten concerns to obesity, the poor fellow doesn’t have a friend to call his own. Yet wheat, along with rice and corn, is one of the most ancient domesticated foods in existence. The original Paleo – if you will. How can wheat possibly be so bad?

Somewhere around 3000 BC the people around the area of modern day Syria began to cultivate the ancestors of wheat – the emmer and einkorn varieties. Having a semi-stable source of food improved survival odds tremendously. Soon, the farmers had spread across the globe bringing wheat along with, later, domesticated animals.

The next major improvement in agriculture came with the application of fertilizers to increase yield. First, guano, the nitrogen and phosphorus rich droppings of penguins and seabirds were applied with great effect. With the advent of nitrogen processing, chemical fertilizers were soon making their mark. This kept agricultural production high enough to feed the world. For a time.

Nevertheless, by the 1950s there were Malthusian concerns of worldwide famine. In Mexico, Norman Borlaug, who would later win the Nobel Peace Prize in 1970, began to experiment with higher yielding varieties of wheat. One of his accomplishments was to increase the seed head size. However, there was a problem. The large head would tend to flop over on the stalk.

The solution was to shorten the stalk of the wheat. This became known as dwarf and semi-dwarf wheat and had the advantage that it would not buckle as well as faster maturation. No time was wasted growing the stalk which was not edible anyway. Within years, 95% of wheat was of the Borlaug variety and the yield increased by 6 fold. India, facing mass starvation in 1965, ordered tonnes of the new seed and farmers began to plant dwarf wheat. Wheat harvests quickly tripled and India became self-sufficient in food. This was the Green Revolution and Norman Borlaug was the Father.
But where Dr. Borlaug bred naturally occurring strains, successors quickly turned to new technology to enhance mutations. This was the atomic age after all. Using X-rays and thermal neutrons, these new Genetically Modified (GMO) crops were born. Later, scientists would discover how to target specific genes for inclusion into new genomes. Wheat is a relative laggard, with corn, rice and soybeans taking the lead.

The wheat varieties of today are not the same as those 50 years ago. The new varieties of wheat were not tested in any safety lab. They were merely assumed to be safe. But the Broadbalk Wheat Experiment is clear evidence that the nutritional content has changed significantly as documented in the paper "Evidence of decreasing mineral density in wheat grain over the last 160 years".

The red line depicts the introduction of dwarf wheat. Even as grain yields skyrocket, the micronutrients contained in the wheat grain plummet. Does this matter? I don’t actually know, but it sure can’t be good.

Has the wheat changed over the last 50 years? Hard to say but there has certainly been an increase in celiac disease. Gluten causes damage to the small intestine in susceptible patients. Dr. Murray of the Mayo Clinic compared blood samples from Air Force men 50 years and found that the prevalence of celiac disease has quadrupled. Could this be a result of the changes in wheat itself? Hard to say, but interesting to think about.

The other major change in wheat is the method of processing. Wheat berries were traditionally ground by large millstones powered by animals or humans. This has been replaced by the modern flour mill which is better at removing everything. The bran, middlings, germ

**Broadbalk Wheat Experiment**

![Broadbalk Wheat Experiment graphs](image-url)
and oils are removed leaving the pure white starch. Most of the vitamins, proteins and fats are removed. This is modern white flour in all its evil beauty. Modern milling is able to grind flour to such a fine dust that absorption into the body is extremely rapid.

Starches are composed of hundreds of units of sugars all linked together. 75% of the starch is organized in occasionally branched chains called amylopectin. The rest comes as unbranched chains called amylose. There are several classes of amylopectin. Legumes are particularly rich in amylopectin C. This is very poorly digested. As the undigested carbohydrate moves towards the colon, gut flora produces gas causing the familiar ‘tooting’ of the bean eater. While beans and legumes are very high in carbohydrates, much of it is not absorbed, Beano notwithstanding.

Amylopectin B is found in bananas and potatoes. This is intermediate in absorption. The most easily digested is Amylopectin A found in – you guessed it – wheat. The upshot is that wheat is converted to glucose more efficiently than virtually any other food. This is recognized in the Glycemic Index where the effects of the different amylopectins is evident.

There are also persistent concerns that the gluten in wheat produces exorphins. While other foods may have gluten, wheat is the the major source in our diets by a factor of 100. Digestion of this gluten may yield morphine like substances that can cross the blood brain barrier that many are concerned are addictive. While evidence in the medical literature is sparse, anecdotal evidence is not. Many people admit to being ‘addicted’ to bread and pasta. Comfort foods are also typically flour based – cookies, cakes, macaroni and cheese. While this does not prove anything, it is certainly worth noting.
China provides an interesting insight into a traditional rice based diet that has introduced wheat. Exhaustive data were compiled by T. Colin Campbell in The China Study. **Wheat is the strongest positive predictor of body weight.** As wheat intake increases, so does Body Mass Index. There was also a strong association with coronary disease and wheat intake.

So let’s see. Modern wheat is a problem because

1. Lower nutritional value
2. Processing removes most of fibre and vitamins
3. Modern milling speeds digestion therefore increasing glycemic effect
4. High in amylopectin A
5. May be addictive

Please, sir, can I have some more?

Not all carbohydrates lead to obesity. However, refined grains such as flour clearly do. This has been known since the time of William Banting.

The next step in weight loss?

Reduce refined grains, particularly wheat.
The Great Carbohydrate Debate – Chapter 20

In the ongoing effort to lose weight, the uncontroversial first step is to reduce added sugars. The next step is to reduce refined carbohydrates. There are many diets that advocate increasing carbohydrate intake instead. And some of them do indeed work.

Controversy surrounds the humble carbohydrate. Is it good or bad? From the mid 1950’s to the 2000’s, they were the good guys, the heroes. Low in fat, they were supposed to be our salvation from the phony epidemic of heart disease. The Atkins onslaught of the late 1990s recast them in the role of dietary villain. Many advocates avoid all carbohydrates. What, even vegetables and fruits? Yes, even vegetables and fruits. So, are carbohydrates good or bad?

Insulin and insulin resistance drive obesity. Refined carbohydrates such as white sugar and white flour cause the greatest increase in insulin. These foods are quite fattening. This does not mean that all carbohydrates are similarly bad. There is a substantial difference between ‘good’ carbohydrates (whole fruits and vegetables) and ‘bad’ (sugar and flour). Kale and broccoli will not make you fat no matter how much you eat. But eating even modest amounts of sugar can certainly cause weight gain. Yet both are carbohydrates – so what is the difference? How do we distinguish the two?

Dr. David Jenkins of the University of Toronto began to tackle this problem in 1981 with the Glycemic Index (GI). Foods were ranked according to their tendency to raise glucose. Since dietary protein and fat did not raise the blood glucose appreciably, they were essentially excluded from the GI. It was used exclusively to measure carbohydrate-containing foods. In these foods, there is a close correlation between the GI and insulin stimulating effect.

The GI uses identical 50-gram portions of carbohydrate. For example, you might take foods such as carrots, watermelon, apples, bread, pancakes, a candy bar, and oatmeal. You measure each portion to contain 50 grams of carbohydrate and then measure the effect on blood glucose. Foods are compared against the reference standard, glucose, which was assigned a value of 100.

However, a standard serving of food may not contain 50 grams of carbohydrate. For example, watermelon has a very high glycemic index of 72, but contains only 5% carbohydrate by weight. Most of the weight is water. So you would need to eat 1 kilogram (2.2 pounds!) of watermelon to get 50 g of carbohydrate. This is far in excess of a single serving. A corn tortilla, on the other hand, has a GI of 52. The
tortilla is 48% carbohydrate by weight, so you would only have to eat 104g of the tortilla to get 50g of carbohydrate. This is close to a standard serving. The Glycemic Load (GL) index attempts to correct this distortion by adjusting for serving size. Watermelon turns out to have a very low GL of 4, but the corn tortilla still has a high GL of 25.

Whether we classify carbohydrates by GI or GL, it becomes obvious that there is a clear distinction between refined carbohydrates and unrefined traditional foods. Western refined foods have a very high GI and GL. Traditional whole foods have low GL scores, despite containing similar amounts of carbohydrate. This is an essential distinguishing feature. Carbohydrates are not inherently fattening. The toxicity lies in the processing.

<table>
<thead>
<tr>
<th>Western refined foods</th>
<th>Glycemic index</th>
<th>Glycemic load</th>
<th>Unrefined traditional foods</th>
<th>Glycemic index</th>
<th>Glycemic load</th>
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<tbody>
<tr>
<td>Glucose</td>
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<td>Parsnips</td>
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<td>Rice cakes</td>
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<td>Table sugar (sucrose)</td>
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The glycemic reference is glucose with a glycemic index of 100 (Foster-Powell and Miller, 1995).

Refining significantly increases the glycemic index by purifying and concentrating the carbohydrate. Removal of fat, fibre and protein increases the speed of digestion. The other macronutrients slow down absorption of the carbohydrates. Furthermore, the refined carbohydrates are much easier to absorb. In the example of wheat, modern machine milling has almost completely replaced the traditional stone milling. This allows wheat to be pulverized into the very fine
white powder we know as flour. Cocaine users will know that very fine powders are absorbed into the bloodstream much faster than coarse grains. This allows for higher ‘highs’ – both for cocaine and for glucose. The refined wheat causes glucose to spike up. Insulin levels follow.

Secondly, refining encourages overconsumption. For example, a glass of orange juice may require 4 or 5 oranges. It is very easy to drink a glass of juice, but eating 5 oranges is not so easy. By removing everything other than the carbohydrate, we tend to over consume what is left. If we had to eat all the fibre and bulk associated with 5 oranges, we may think twice about it. The same applies to grains and vegetables. If we remove all the bran and fibre and fat from wheat and turn it into white flour, it is very easy to eat. Dr. Andreas Eenfeldt of www.dietdoctor.com compared identical 30g portions of carbohydrates in vegetables to bread. The bun contains almost pure carbohydrate in an easily digestible form. The pile of vegetables contains significant amounts of fibre and fat. One is highly refined, the other is not. One will cause obesity, the other will not.

The problem is one of balance. Our bodies have adapted to the balance of nutrients in natural food. By refining foods and only consuming a certain portion, the balance is entirely destroyed. People have been eating unrefined carbohydrates for thousands of years without obesity or diabetes. What has changed recently, is that we now predominantly eat refined grains as our carbohydrate of choice. In Western societies, wheat is often the grain of choice.
Fibre – Chapter 21

Fibre is the non-digestible part of food, usually a carbohydrate. A more formal definition is the “dietary constituents that are not enzymatically degraded to absorbable subunits in the stomach and small intestine”. These are generally plant foods, but occasional animal foods such as liver glycogen are included. Common types of fibre include cellulose, hemicellulose, pectins, beta-glucans, fructans, and gums. Whole grains, fruits, vegetables, legumes and nuts are all major sources of dietary fibre.

Fiber is classified as soluble or insoluble based on whether it is dissolvable in water. Beans, oat bran, avocado and berries are good sources of soluble fibre. Whole grains, wheat germ, beans, flax seeds, leafy vegetables and nuts are good sources of insoluble fibre. Fibre can also classified as fermentable or non-fermentable. Normal bacteria residing in the large intestine have the ability to ferment certain undigested fibre into the short-chain fatty acids acetate, butyrate and propionate. These fatty acids can be used as an energy source. They may also have other beneficial hormonal effects including the decreased output of glucose from the liver. Generally, soluble fiber is more fermentable than non-soluble.

Fibre has multiple purported mechanisms of health benefit but the importance of each is largely unknown. High fibre foods require more chewing, which may reduce food intake. Horace Fletcher (1849-1919) believed strongly that chewing every bite of food one hundred times would cure obesity and increase muscle strength. This helped him lose 40 pounds, and "Fletcherizing" became a popular weight loss method in the early 20th century.

Fibre may decrease palatability and thus reduce food intake. Children seem to naturally gravitate towards the white bread and white pasta, rather than the whole-wheat varieties. Fiber bulks up foods and decreases energy density. Soluble fibre absorbs water to form a gel, further increasing volume. This fills the stomach, which increases satiety. Stomach distention may signal a sensation of fullness or satiety through the vagus nerve. Increased bulk may lead to increased time for stomach emptying. This leads to a slower rise in blood
glucose and insulin after meals. In some studies, half the variance of the glucose response to starchy foods depends upon the fiber content.

In the intestines, soluble fibre may ‘trap’ nutrients in the gel formed. This blocks contact with the intestinal walls, reducing absorption of fats and proteins. The mixing of food and digestive enzymes is disrupted. Indeed, some studies have shown that increased fibre intake lowers fat and protein absorption. Increased unabsorbed macronutrients in the distal small bowel may result in further delaying of stomach contents. The mechanism has been called the ‘ileal brake’. Presumably, this is an adaptive feature to allow more complete absorption of nutrients farther upstream. Hormones involved in this ‘ileal brake’ may include GLP-1, peptide YY and neurotensin.

In the large intestine, the increased stool bulk reduces the transit time. The increased stools may lead to increased caloric excretion. On the flip side, fermentation in the colon may produce short-chain fatty acids. Roughly 40% of dietary fibre may be metabolized in this way. One study demonstrated that a low fiber diet resulted in 8% higher caloric absorption. In short, fibre may decrease food intake, slow down absorption in the stomach and small intestine, then exit quickly through the large intestines. All of these effects are potentially beneficial in the treatment of obesity.

Fibre intake has fallen considerably over the centuries. Fibre intake in Paleolithic diets is estimated to be 77-120 grams per day. Traditional diets are estimated to have 50g/day of dietary fibre. By contrast, modern American diets contain as little as 15 g/day. Indeed, even the US Dietary Guidelines only recommends 25-30 g/day. Removal of dietary fibre is a key component of the processing of foods. Improving the texture, taste and consumption of foods directly benefits the profits of food companies.

Fibre came to public attention in the 1970s. Physicians doing missionary work with native populations mostly in Africa noted a consistent pattern of disease. The Diseases of Civilization, including heart disease, obesity, diabetes, stroke, and cancer were rare amongst natives that followed a traditional diet and lifestyle. By contrast, in urban areas where native lifestyles became westernized, these diseases slowly appeared.
Some researchers 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. This was a significant advantage when traveling without refrigeration. Flour and sugar could be carried in great quantity over great distances. Through the 1960s and 1970s, debate raged back and forth. The pendulum finally swung in favor of lowering dietary fat, which necessitated an increase in carbohydrate consumption. Since most of the carbohydrates in the Western diet are refined, the goal of reducing fat was incompatible with lowering refined carbohydrates.

Thus, the carbohydrate hypothesis quickly fell into disfavor. But how to explain the Diseases of Civilization? Perhaps, it was not the carbohydrate but the lack of fibre that was the problem. Being compatible with the dietary fat hypothesis, this explanation quickly became accepted. Traditional unrefined foods that included carbohydrates tended to be very high in fibre. A missionary surgeon named Denis Burkitt became one of the leading proponents of the dietary fibre hypothesis. By 1977 the new Dietary Guidelines made the recommendation to “Eat foods with adequate starch and fiber”. With that, fibre was enshrined in the pantheon of conventional nutritional wisdom. Fibre was good for you. You should eat more fibre. But it was difficult to show exactly how it was good for you.

At first, it was believed that high fibre intake reduced colon cancer. The subsequent studies proved to be a bitter disappointment. The prospective Nurse’s Health Study followed 88,757 women over 16 years finding no significant benefit. A randomized study of high fibre intake failed to demonstrate any reduction in precancerous lesions called adenomas. The Nurses Health Study and found no benefit to cancer risk with high fibre. The huge Women’s Health Initiative study increased whole grains, fruits and vegetables, but failed to find a benefit for colon cancer, either.
If fibre wasn’t helpful in cancer, perhaps fibre might be beneficial in heart disease. The Diet and Re-infarction Trial (DART) study randomized 2033 men after their first heart attack to three different diets. 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) on the other hand, was beneficial. Dr. Ancel Keys had suspected this years ago from the Seven Countries Study. Recent trials such as the PREDIMED confirm the benefits of eating more natural fats such as nuts and olive oil. So eating more fat is beneficial. What about a high fibre diet? No benefit. Not only that, but increasing fibre may actually have increased the risk of dying!

But it was difficult to shake the feeling that somehow, fibre was good. Most correlation studies show lower BMI with higher fiber intake. This has also been found to be true in native Pima populations and native Canadians as well. More recently, the 10-year observational CARDIA study found that those eating the most fiber were least likely to gain weight. Short-term studies show that fibre increases satiety, reduces hunger, and decreases caloric intake. Randomized trials of fibre supplements show relatively modest weight loss effects with a mean weight loss is 1.3 – 1.9 kg (2.9 – 4.2 pounds) over a period of up to 12 months. Longer-term studies are not available.

When we consider the nutritional benefits of food, we think about the vitamins, minerals and nutrients they contain. We think about components in the food that nourish the body. Fibre is completely different. The key to understanding fibre’s effect is to realize that the benefit lies not as a nutrient, but as an anti-nutrient. Fibre has the ability to reduce absorption and digestion. Fibre subtracts rather than adds. In the case of sugars and insulin, this is good. Soluble fibre reduces absorption of carbohydrates, which in turn reduces blood glucose and insulin levels. In one study, type 2 diabetic patients were given liquid meals containing 55% carbohydrates with or without the addition of dietary fibre.

Fibre reduced both the glucose and the insulin peaks, despite consuming exactly the same amount of carbohydrates. Fibre acts as an anti-nutrient. Because insulin is the main driver of obesity and diabetes, reduction is beneficial. In essence, fibre acts as a sort of ‘antidote’ to the carbohydrate, which, in this analogy, is the ‘poison’. Carbohydrates, even sugar, are not literally poisonous, but comparison is useful to understand the effect of fibre.

It is no coincidence that virtually all plant foods, in their natural, unrefined state contains fibre. Mother Nature has pre-packaged the ‘antidote’ with the ‘poison’. Thus, traditional societies may follow diets
high in carbohydrate without evidence of obesity or Type 2 Diabetes. The Okinawans, for instance, base their diet upon the sweet potato, and consume an estimated 80% of their calories as carbohydrate. High fibre protects against obesity. Until recently, they were one of the longest-lived peoples on earth. The Kitavans of New Guinea followed a diet estimated to be close to 70% carbohydrate with no evidence of ill health. The one critical difference is that these carbohydrates are all unrefined. The toxicity lies in the processing.

Western diets are characterized by one defining feature. It is not the amounts of fat, salt, carbohydrates, or protein that distinguishes the Western diet from all other traditional diets in the world. It is the high levels of processing of foods. Typical Western diets are high in refined carbohydrates. Consider the traditional Asian markets, full of fresh meats and vegetables. Many Asian cultures buy fresh food daily so processing to extend shelf life is neither necessary nor welcome. By contrast, North American supermarkets have middle aisles filled with boxed, processed foods. Several more aisles are dedicated to processed frozen foods. North Americans will buy groceries for weeks or even months at a time. The large volume retailer Costco, for example, depends upon this.

Fibre and fat are key ingredients removed in the refining process. Fibre is removed to change the texture, and make food taste ‘better’. Natural fats are removed to extend shelf life since fats tend to go rancid with time. For example, white flour has virtually all the natural fibre and fat removed during processing. This exposes us to the full danger of the naked carbohydrate, which causes the ensuing high insulin levels. The ‘poison’ is ingested without the ‘antidote’. The protective effect of the fibre and fat are removed.
Where whole, unprocessed carbohydrates virtually always contain fibre, dietary proteins and fats contain almost no fibre. Our bodies have evolved to digest these foods without the need for fibre. The ‘antidote’ is unnecessary without the poison. Here again, Mother Nature has proven herself to be far wiser than us.

Natural foods have a balance of nutrients and fibre that we have evolved over millennia to consume. The problem is not with each specific component of the food, but the overall balance. For example, suppose we bake a cake with a balance of butter, eggs, flour and sugar. Now we decide to remove completely the flour and double the eggs instead. The cake tastes horrible. Eggs are not necessarily bad. Flour is not necessarily good, but the balance is off. The same holds true of carbohydrates. The entire package of unrefined carbohydrates, with fibre, fat, protein and carbohydrate is not necessarily bad. But removing everything except the carbohydrate may destroy the balance and make it harmful to human health.

Removing protein and fat may lead to overconsumption. There are natural satiety hormones (Peptide YY, cholecystokinin) that respond to protein and fat. Eating pure carbohydrate does not activate these systems and leads to overconsumption. For example, a glass of orange juice requires 4-5 oranges. It is difficult to eat 4-5 oranges, with all the associated pulp. However, by only drinking the carbohydrate portion, and discarding the rest, you may over-consume that carbohydrate. Another problem arises because the relatively pure carbohydrate results in increased speed of digestion. The rapid rise in blood glucose will result in the rapid rise in insulin.

*The toxicity lies not in the food, but in the processing.*

Nutritionism, where foods are considered based on their macronutrient content hid the dangers of refining for many years. Whole grains, vegetables as well as sugar were all considered similar because they happened to all be classified as carbohydrates. But the refined and unrefined carbohydrates were not alike.

**Fibre and Type 2 Diabetes**

Both obesity and Type 2 Diabetes are diseases caused by excessive insulin. Insulin resistance develops over time, with persistently high insulin. If fibre can protect against elevated insulin, then it should protect against Type 2 Diabetes. That is exactly what the studies show.
The Nurse’s Health Studies 1 and 2 monitored the dietary records of thousands of women over many decades. Overall, the risk of Type 2 Diabetes increases as the glycemic index increases. This is no surprise. This study was also able to confirm the protective effect of cereal fibre intake. Women who ate a high GI diet but also ate large amounts of cereal fibre are protected against Type 2 Diabetes. In essence, this diet is high in ‘poison’ but also high in ‘antidote’ at the same time. The two cancel each other out with no net effect. Women who ate a low GI diet (low ‘poison’) but also low fibre (low ‘antidote’) was also protected. Again the two cancel each other out.

But the deadly combination of a high GI diet (high ‘poison’) and a low level of fibre (low ‘antidote’) increase the risk of Type 2 Diabetes by a horrifying 75%. This is the exact effect of processing carbohydrates – increased glycemic index and decreased fibre.

The massive Health Professionals Follow-up studied 42,759 men over 6 years, with essentially the same results. A high GL and fibre diet confers no extra risk of type 2 Diabetes. A Low GL and low fibre diet also has no increased risk. But the diet high in glycemic load (poison) and low in fibre (antidote) increases the risk of disease by 217%. Yikes! The Insulin Resistance Atherosclerosis study confirms that fibre is an important protective factor against insulin resistance.

The Black Women’s Health Study demonstrated that a high glycemic index diet was associated with a 23% increased risk of Type 2 Diabetes. A high cereal fibre intake, by contrast was associated with an 18% lower risk of diabetes.

One of the key steps in weight loss is the addition of fibre. Even better, do not remove fibre from the natural foods that contain it.

The toxicity lies in the processing.

Carbohydrates in their natural, whole, unprocessed form,
with the exception of honey,
All diets fail. Long term dieting is sheer futility. After initial weight loss, the dreaded plateau appears, followed by the even more dreaded weight regain.

What do I mean? Weight loss follows the same basic curve so familiar to dieters the world over. Whether it is the Mediterranean, the Atkins, or even the old fashioned low fat, low calorie, all diets in the short term seem to produce weight loss. Sure, they differ by the amount – some a little more, some a little less. But they all seem to work.

However, by 6 months to 1 year, weight loss gradually plateaus followed by a relentless regain despite continued dietary compliance. This occurs regardless of the dietary strategy. In the 10 year Diabetes Prevention Program, for example there is a 7 kg weight loss after one year. The dreaded plateau, then weight regain, follows. So all diets fail. The question is why. Sure there are those that seem to maintain their weight loss for year, but we cannot ignore the majority that struggle with weight loss. Further, most academic research seems to indicate that most diets fail, whether it stops to work, or whether it is due to lack of compliance.

This happens because the body reacts to weight loss by trying to return to its original body set weight (BSW). This is the body weight ‘thermostat’, variously called the appestat, or the lipostat. Even the low carbohydrate diets, proven to have superior weight loss effects in the short term, show the same inexorable plateau and weight regain.
We hope the BSW will decrease over time, but this hoped-for reduction does not materialize. Why does the BSW still stay high even if we are eating all the ‘right’ things? This is because insulin levels stay elevated. Only half of the problem has been addressed. Two main things maintain high insulin levels. The first is the foods that we eat. This is what we usually change when we go on a diet. But we fail to address the other main concern. This is the longer-term problem of insulin resistance or ‘When to Eat’.

Insulin resistance maintains high insulin levels. High insulin maintains the high BSW. This inexorably erodes weight loss efforts. Hunger increases. Metabolism (TEE) relentlessly decreases until it falls below the level of energy intake. Body weight plateaus and then ruthlessly increases back to the original BSW, even as the proper diet is maintained. For some, changing ‘what we eat’ is clearly not enough. This is the problem we are all familiar with. All diets fail.

To succeed, we must break the insulin resistance cycle. But how? Consider the analogous problem of antibiotic resistance. If antibiotics are used at persistently high levels, resistance develops. How do you break antibiotic resistance? The knee-jerk reaction is to use even higher doses of antibiotics to ‘overcome’ the resistance. But this will only encourage the development of more resistance. The answer is the
exact opposite. The answer is to reduce use of antibiotics. Having periods of very low antibiotic usage breaks the resistance.

The same logic applies to insulin resistance. The body’s knee-jerk reaction to insulin resistance is to increase insulin levels. But this only creates more insulin resistance. The answer is the exact opposite. We must create recurrent periods of very low insulin levels, since resistance depends on persistent, high levels. But how can we create those very low levels of insulin?

So the forgotten question of weight loss is “When should we eat?” We don’t ignore the question of frequency anywhere else. Falling from a building 1000 feet off the ground once will likely kill us. But is this the same as falling from a 1-foot wall 1000 times? Absolutely not. Yet the total distance fallen is still 1000 feet.

The first step is to eliminate all snacking in between meals and at bedtime. This is hardly some far-fetched idea. We are simply turning back the clock to the 1960s. Eating snacks or eating at a place other than a table was actively discouraged. There was no eating in the car, or in front of the TV.

If all meals were eaten between 8am to 6 pm, this was enough to prevent obesity. So, even though they were eating white bread and pasta, there was minimal obesity compared to today. I mean – who ate whole wheat bread in the 1960’s? Nobody sold multigrain bread. Rye? Please. And don’t get me started on whole-wheat pasta – that didn’t even exist then. Ask for quinoa and people thought you were swearing in spanish.

All foods will increase insulin levels to some degree. Eating the proper foods will prevent high levels, but won’t do much to lower levels. Some foods that are better than others, but all foods still increase insulin. The key to prevention of resistance is to periodically sustain very low levels of insulin. If all foods raise insulin, then the only logical answer is the complete voluntary abstinence of food. The answer we are looking for is, in a word, fasting.

This post is not meant to be pessimistic despite the title (All Diets Fail), but actually optimistic (a certain strategy may fail, but you can use another one to achieve the same goal).

However, my point is this. Obesity is a multi-factorial disease, as I previously wrote about. This means, that there must be multiple targets in the pathway to obesity. A LCHF diet, for some people will be sufficient for weight loss. Many do extremely well. But we cannot ignore those for whom LCHF is not enough.
Let’s take an analogy of heart disease. Smoking, high blood pressure and family history all contribute to heart disease. So, simply stopping smoking is important but not the only strategy to use. We must also treat high blood pressure. This does not, in any way, mean that stopping smoking is incorrect. It is only incomplete.

In the same way, obesity has many facets, one of which is the diet. Treating only the diet is enough for some, but not enough for others. We cannot pretend that a LCHF will make 100% of people lean again. Let’s take a thought experiment, as one of my heroes Gary Taubes is fond of doing.

Suppose, I give you prednisone, the synthetic form of cortisol. It causes obesity. As you get fatter and fatter, I, as your physician advise you to go on a LCHF diet. Will it work? Of course not. Your diet was not the problem. Your problem was the excess cortisol I prescribed to you. The answer, of course is simple. Reduce the cortisol. You must treat the underlying cause of the obesity. You must understand the aetiology of obesity. Diet, in this case, is not enough.

In the same way, suppose your problem with obesity is the time dependent development of insulin resistance over two decades. That insulin resistance is the major pathway by which your insulin stays elevated. Now, I tell you to watch your diet. Will it work? Not likely. You have addressed the insulinogenic diet, but not the insulin resistance. Sure, changing you diet will lead to a lowered insulin resistance in 5 years, but that’s not good enough. (If it took 20 years to develop, doing the reverse will similarly require that amount of time).
Suppose your problem with obesity is due to excessive cortisol due to stress and sleep deprivation. Or chronic pain from fibromyalgia. Will a prudent diet help? A little bit. A good diet is not going to reduce your cortisol. If cortisol is the main pathway, that is what needs to be addressed. I have, in fact, patients in the Intensive Dietary Management Program who I cannot help because their problem is caused by, say anti-psychotic medication that stimulates insulin. Or fibromyalgia that stimulates cortisol. Unless I can deal with their underlying cause of obesity, I cannot treat it. But I certainly can understand it.

So, with regards to diet, refined carbohydrates stimulate insulin the most, and dietary fat the least. So the most logical dietary treatment is Low Carb, High Fat. Yes, I am still a firm believer in that. But sometimes, we need to move past that.
Why Can’t I Lose Weight? – Chapter 23

Why can’t I lose weight? I hear this question all the time. It’s usually followed by something like “my best friend SkinnyBitch used this internet diet and lost 30 pounds. Why can’t I?” The basic question is something like this. If their friend uses a LCHF diet to lose weight, why doesn’t it work for me? Or, if somebody uses a Paleo diet, why doesn’t it work for me?

The answer really lies in the multi-factorial nature of obesity. Let’s go back to our Hormonal Obesity Theory (HOT) diagram.

If you don’t remember all the parts to the aetiology of obesity, you may want to review my 6 part lecture series on YouTube or review the last 45 or so blog posts about the aetiology of obesity. Start with Calories part 1.

As you can see, insulin is the main driver of obesity. But there are many things that can increase or decrease insulin. Refined grains, carbohydrates, and animal proteins can all increase insulin levels. Cortisol is also a major player in stimulating insulin secretion. Fructose
increases insulin resistance directly which indirectly leads to increased insulin levels.

There are also factors that are not related to the ‘what we eat’ question. This is the key role played by insulin resistance. Insulin resistance both causes and is caused by high insulin levels – a classic vicious cycle. This accounts for the time-dependent nature of obesity. The fat get fatter. The longer you have obesity, the harder it is to get rid of it.

Why? Because the longer you go through the cycle of high insulin → insulin resistance → high insulin, the worse it gets. So long standing obesity is much harder to eradicate than recent obesity, as everybody already knew.

There are also a number of protective factors against obesity. The incretin effect, vinegar and fibre all help protect against the rise in insulin and play a protective role.

The key to understanding obesity is that many different things can contribute to the development and the treatment. Consider the analogy of your car not starting. There could be multiple problems. For example, the battery is dead, the car ran out of gas, or the spark plugs are worn out. So, if your problem is that the battery is dead, filling up with gas will not help. Neither will replacing the spark plugs. That seems kind of obvious.

But then, websites proliferate about how changing batteries is the cure for cars that don’t start. It is filled with testimonials of how people changed batteries and their cars effortlessly started. Other people attack the website saying that they changed batteries and nothing happened. Instead, they filled up with gas and the car started, so obviously, the key to starting the car is filling up on gas.

That’s exactly what happens in the sad sack, wacky world of weight loss. When you try to lose weight, people assume that there is only one problem for everybody. If your problem is insulin resistance, then reducing carbs may not be the best strategy (intermittent fasting may work better). If your problem is sleep deprivation/stress, then increasing fibre is not going to be too good.

Cutting sugar works well for those people whose problem is excessive sugar intake. They write books and websites about how sugar is the devil. Others think that is ridiculous and think that refined grains (wheat) is the real devil since they’ve done well reducing grains. Others think that stress relief is the major problem in weight loss. Others blame calories. They all ridicule each other and fill the internet
with testimonials. Worse, they all start bickering about how the real problem is carbs, or sugar, or wheat, or calories, or stress, or sleep deprivation, or fibre, or animal proteins etc.

You must understand that they can all be correct. Obesity is not a single problem. There is no single solution. A low sugar diet works amazingly for some, and not at all for others. Just as replacing the battery will work for some cars amazingly and not at all for others.

_So – why can’t I lose weight?_

Because you must be targeting the wrong pathway. You must find the particular problem that is causing your obesity and target that pathway.

**Example 1** – Let’s say that you suffer from a chronic pain syndrome, or fibromyalgia. This increases your stress level and your cortisol is chronically high. This leads to high glucose and insulin stimulation. This causes weight gain. Since cortisol is your problem, then you need to use treatments that target that pathway. This would include medical treatment for chronic pain, acupuncture, massage, meditation, yoga etc. This will help lower your cortisol level and treat your weight gain problems.

Reducing your carbohydrate or calorie intake will not be particularly successful. This is because these were not the problems increasing insulin levels in the first place.

**Example 2** – Suppose that you are chronically sleep deprived. We know that this situation increases cortisol levels and will lead to weight gain. Lowering the sugars in your diet will not be particularly successful, because that was not your problem. You need to either find a new job, or improve your sleep hygiene or just plain get more sleep.

**Example 3** – Suppose that your main problem is the insulin resistance cycle. This vicious cycle has developed over decades and insulin resistance is now the main stimulus to your high insulin levels. Reducing carbs may not be the most effective treatment. Why? Because your problem is the insulin resistance. Lowering carbs will lower insulin and reduce the vicious cycle, but this cycle has been running for decades.

So what do you need to do? Remember that the question of insulin resistance is mostly a question of ‘when to eat’. Therefore, to break resistance, you need a sustained period of low insulin. This means that fasting will be more effective here than simple carb restriction. You will also need time, because it took time to develop and will require time to resolve.
**Example 4** – Suppose your friend takes fibre supplements and loses weight. But it doesn’t work for you. Why? Because your friend’s problem may be excessive carbs and fibre will help reduce the insulin spikes associated with carb intake. However, if your problem is not that, then increased fibre won’t work so well.

**Example 5** – Why did the Asian Rice Eater of the 1990’s not develop obesity? At that time, China was eating extremely high carbohydrate (white rice) diets. But they also ate virtually no sugar, very little animal protein, high vinegar (pickled vegetables), high fibre, and no snacks. That means that all the other pathways to insulin were shut down as they were only eating rice. They ate lots of protective factors. They did not eat constantly, so did not develop insulin resistance. They had almost no sugar and therefore did not develop insulin resistance. The end result? No obesity.

**Example 6** – Suppose you eat a diet very high in unrefined starches, like sweet potato. At the same time you severely restrict animal protein. Will that work? It sure could. This increases carbs, yes, but decreases animal protein. You also significantly raise dietary fibre. This may be enough to sway the balance and decrease insulin which will lead to weight loss.

I should make clear that I am not anti-carbohydrate. That is only one piece of the puzzle – although refined grains and sugars tends to be a rather large part of the puzzle. They are certainly obesogenic, but there are other ways to compensate so that overall insulin does not increase. I’m anti-hyperinsulinemia. Insulin causes obesity. If carbs raise insulin then carbs will cause obesity. But you can certainly tweak your carbohydrate heavy diet to lower insulin. After all, the Kitavans ate a very high carbohydrate diet and still had insulin levels lower than 95% of the Swedish population. The Okinawans ate at diet of almost 70% sweet potato with no obesity at all. Of course, both populations still ate virtually no sugar, and no refined grains (wheat).

**Cortisol**
The number one mis-diagnosis we see in our Intensive Dietary Management program is chronic stress/cortisol confusion. We see people who come in and swear that they cannot lose weight. Then, upon taking a history, we find that some non-dietary factor is responsible for weight gain. For example, we may find somebody who is taking some antipsychotic medications. Some of them increase insulin. Or we’ll find pain syndromes. Or sleep deprivation.

Well, no wonder dietary changes didn’t make any difference. That wasn’t their problem. It was the cortisol/stress pathway all along.

While I wish I could write that we helped fix them, the unfortunate truth is that these problems are much harder to fix. While there are time tested ways of reducing stress and cortisol levels, most patients simply do not take our advice to look into mindfulness mediation/prayer/religion/yoga/acupuncture/massage. They came to a diet clinic and got advice to meditate. They look at me or Megan like we have two heads.

There is, however, good evidence that things such as mindfulness mediation can have an effect on weight loss.

At the University of California, San Francisco, a trial was done with mindfulness meditation and showed that they could reduce cortisol levels. This is no surprise since meditation has been done for thousands of years as a stress relieving method. This decrease in cortisol was closely paralleled by a decrease in abdominal fat.

The important thing to know about it is that meditation does not change the actual stressor. For example, suppose your boss is driving...
you crazy. You will be under a lot of stress. Meditation won’t change that one bit. What it will change is your body’s response to the stress. In the end, that is what is important. By decreasing the cortisol response, there is a decrease in abdominal fat. Your boss is still the jerk he was before. You have only changed your body’s response to this stressor.

One final thought about stress relief. It’s always a little amazing to me how far organized religion is ahead of the game. Think about the practices they preach. Prayer (similar to meditation). Belief in a higher power/ confession (stress relief). Weekly ceremonies, like mass (sense of community and continuity – important for stress relief). Small group session (friendship and sense of belonging – stress relief). Fasting. Yes, fasting. All of these practices that are so important for good health have been established thousands of years ago.