Sickly Sweet

Summary

Sugars are simple, sweet-tasting carbohydrates. There are a number of different forms of sugar, each of which has a slightly different chemical structure. From the narrow point of view of where we get our energy, it does not matter much which sort of carbohydrates we eat: our body can break down food into glucose which is a simple sugar that is used in the body for fuel. But for other reasons, it is enormously important which sort of carbohydrates we eat, and in what form.

There are two main reasons to avoid eating refined sugars.

- First, eating refined sugars causes our bodies to produce excessive amounts of a hormone called *insulin* to manage the level of sugar in our blood. This leads in the short term to cycles in blood sugar, variable moods, snacking and over-eating, and it damages the immune system. Over time, excessive consumption of refined sugars leads to a collection of undesirable conditions, together known as *metabolic syndrome*, which include the build up of cholesterol and fats which clog our blood vessels, hormone imbalances and the inability to metabolize sugar; and these significantly increase our chances of suffering in later life from obesity, heart disease, cardiovascular disease, diabetes, and various hormone-induced cancers.
- Second, refined sugars are empty calories , in that they provide energy but are not accompanied by minerals and vitamins, and other essential nutrients that we also need in our diet, such as fibre, proteins, and unsaturated fats.

There are some circumstances in which it is desirable for an athlete to consume easily digestible carbohydrates, especially during and immediately after exercise, to restore levels of glucose in the blood, and in the body's energy stores in the liver and in muscles. But even then, it is preferable to eat foods such as banana or a bagel which contain easily digestible sugars, than to eat processed or refined sugars.

The structure of this note

This note is structured as follows:

- **types of sugar**
  an account of the different types of sugar, and how they are chemically different but related
- **how the body processes carbohydrates**
  the role of glucose as a fuel; and how the body deals with other carbohydrates by breaking them down into glucose;

- **the role of fatty acids**
  looks at cholesterol and triglycerides, which are produced in response to eating carbohydrates, excessive levels of which can cause cardiovascular disease
- **the role of insulin in managing blood sugar levels**
  insulin is a hormone used to control blood sugar; this explains its role in the metabolism of carbohydrates and fatty acids;
- **glycemic index as a measure of the impact of different foods**
  a measurement of how different foods impact on blood sugar, which is an indicator of how the body will react;
- **the insulin response to high glycemic foods**
  how the body responds to some foods containing accessible sugars by over-producing insulin
- **insulin resistance**
  why excessive insulin production can be damaging to health
- **carbohydrates and insulin resistance**
  summarizes the evidence on the link between diet and insulin resistance
- **carbohydrates and cardiovascular disease**
  summarizes the evidence on the link between carbohydrates and heart disease and stroke
- **insulin resistance and cardiovascular disease**
  discusses how insulin resistance is itself a cause of cardiovascular problems
- **metabolic syndrome**
  this is a collection of conditions (obesity, diabetes, heart disease) which occur together and is often the result of eating refined sugars
- **glycemic index is an imperfect indicator**
  GI is not a perfect indicator of whether a sugar is safe to eat: refined sugars with low GI are also a hazard to health
- **nutritional value of different sugars**
  looks at the reasons why sugars are “empty calories”; even “natural” sugars do not contain sufficient essential minerals and vitamins;
- **carbohydrates in the diet**
  summarizes the sources of carbohydrate in the diet, and how it has evolved towards more refined sugars;
- **implications for runners**
  looks at the implications for runners and other athletes
- **conclusions**
- **references**
  a series of key academic papers, with a short summary

Types of sugar

Discussion about sugar can be confused because the word means different things to different people. Among scientists [1], *sugars* are simple carbohydrates that taste sweet. They are split into two types, according to their chemical structure:

- **Monosaccharides**
  These are the simplest sugars; they each have the same number and types of atoms as each other, but they are arranged differently. Fructose, glucose and galactose are the most common monosaccharides which occur naturally in food.
- **Disaccharides**
  These more complex sugars are formed when two monosaccharide molecules join together, with the removal of one molecule of water. They are less sweet than monosaccharides. Sucrose – or table sugar – is a common disaccharide, made up of one glucose and one fructose molecule.

These chemical differences matter, because (as we shall see) they affect how the body reacts to the different sugars we eat. [2]

Table of common sugars

Carbohydrate	Type	Main source in food	Sweetness
Fructose	Monosaccharide	Primarily fruit also food additives	Sweetest monosaccharide
Glucose	Monosaccharide	Fruits, vegetables, grains	Medium sweet monosaccharide
Galactose	Monosaccharide	Milk	Least sweet monosaccharide
Sucrose	Disaccharide (fructose + glucose)	Sugar cane Table sugar	Sweetest disaccharide
Lactose	Disaccharide (glucose + galactose)	Diary products	Medium sweet disaccharide
Maltose	Disaccharide (glucose + glucose)	Germinating seeds Also added to processed foods	Least sweet disaccharide
Maltodextrin	Glucose polymer	Sports drinks Also added to processed foods	Less sweet than simple sugars

Carbohydrates

As well as sugars, our diet includes other, more complex carbohydrates. Starch, which is found in potatoes, bread, rice, pasta, grains and legumes, is made up of a complex chain – called a

polymer – of simple sugars. Other complex carbohydrates include fibre (cellulose), pectins and gums, which are found in fruits, vegetables, beans and whole-grain cereals.

How the body processes and stores carbohydrates

The human body cannot use most sugars or other carbohydrates directly as a source of energy – all carbohydrates must first be broken down into glucose. When you eat carbohydrates such as lactose or starch, enzymes in the saliva and the intestines break the bonds between the molecules, splitting the complex carbohydrates into simple sugars, which can be transported in the bloodstream. The other monosaccharides, fructose and galactose, must be converted into glucose by the liver before they can be used by the body for energy.

Just as plants usually store sugars as starch, which is a long chain polymer of glucose, humans store excess glucose by turning into a long chain polymer called *glycogen*, which is then stored in the liver and muscle tissues. Whatever the form of the original carbohydrate, it must be broken down into glucose before it can be used for energy or built up into glycogen and stored.

Cholesterol and triglycerides in our blood

Cholesterol and triglycerides are two forms of fat found in humans, and both are necessary for the human body to function. Cholesterol is necessary, among other things, for building cell membranes and for making several essential hormones. Triglycerides, which are chains of high-energy fatty acids, provide much of our energy. Both can come from the food we eat, or can be manufactured by the liver if necessary.

One of the jobs of the liver is to make sure that the body has access to the cholesterol and triglycerides it needs to function.

However, because fats are not soluble in water, they cannot be disolved in blood. So the liver packages cholesterol and triglycerides, along with special proteins, into tiny water-soluble packages called *lipoproteins*. In this form they are released into the blood and, through the blood circulation system, delivered to the cells of the body. The three major varieties of lipoproteins are called LDL (“low density lipoprotein”), HDL (“high density lipoprotein”) and VLDL (“very low density lipoprotein”).

The main function of HDL appears to be carrying excess cholesterol (and probably other chemicals) to the liver for re-use, or for excretion in the bile. Higher levels of HDL seem to protect against coronary artery disease, and so HDL is sometimes referred to as "good" cholesterol. LDL molecules, by contrast, carry mainly “bad” cholesterol; and VLDL molecules carry mainly triglycerides .

When LDL and VLDL levels are too high, they tend to stick the lining of the blood vessels, leading to hardening of the arteries, causing them to narrow, which eventually leads to heart attacks or stroke.

Elevated levels of VLDL in the blood is a condition known as *hypertriglyceridemia*. The exact consequences of this are not known for certain, as patients who have it almost always have other major risk factors for heart disease (mainly obesity, diabetes, and/or high blood pressure). However, the evidence suggests that elevated levels of triglycerides are a major risk factor for heart disease and stroke.

The role of insulin in stabilizing blood sugar levels

Insulin is a hormone used by the body to regulate the level of blood sugar. When the blood sugar level rises, the pancreas releases insulin into the blood-stream. The body responds to this increased level of insulin in a variety of ways. The most important is that, when there is a high level of insulin, the liver, muscle, fat and other tissues absorb more glucose and store it as glycogen.

When the body's glycogen stores are full, the presence of insulin causes the liver to make more triglycerides which are released into the bloodstream; and it causes the body's fatty tissues reduce the breakdown of triglycerides .

In summary, an increase in insulin will decreases the concentration of glucose in blood, by increasing the take-up of glucose by cells, and increasing the manufacture of glycogen, and then triglycerides, by the liver.

Note that the liver and cells only create triglycerides from glycogen once glycogen stores are full. That is why a high-carbohydrate diet will lead to greater production of triglycerides in sedentary people, whose glycogen stores are already full, than in athletes who use glycogen to provide energy in their sports.

Glycemic Index (GI)

In 1976 it was discovered[3] that foods with similar amounts of carbohydrate had differential effects on blood glucose . The glycemic index (GI) was created in 1981 to assist people with diabetes in choosing foods that do not rapidly raise blood sugar.[4] The index was intended to measure the speed with which the carbohydrates are broken down and absorbed into the blood stream, by observing actual blood glucose levels.[5]

It has been assumed that the speed with which carbohydrates are absorbed into the bloodstream was directly related to the complexity of the molecule (ie it was assumed that simple sugars increased blood sugar more quickly than starches). When GI measurements began, it turned out that the speed with which a carbohydrate is not especially closely related to the structure of the carbohydrate. For example, of the monosaccharides, the GIs for glucose, lactose and fructose are 100, 46, and 23 respectively. (This is important because, as we shall see later, fructose has a low GI but is no better for your health than refined sugars with a high GI.)

It turns out that the GI of a food does depend on nature of the carbohydrate (eg whether it is a simple sugar, or what kind of starch it is), but also on the nature of the food as a whole:

- The GI of complex carbohydrates depends whether and how the food has been processed, and how it has been cooked. The gut digests the same type of rice at different rates depending on how it has been milled.
- The food structure is important. Carbohydrates are absorbed less quickly if they are embedded within foods which take longer to digest (eg fructose embedded in the cells of fruit); and more quickly if they sugars added in a manufacturing process.
- Glucose is largely absorbed into the bloodstream in the small bowel, so the rate of glucose absorption depends in part on the overall rate of gastric emptying. This means that the absorption rate is also affected by what else is eaten at the same time – for example, the GI of a meal will be lower if it includes proteins and fats than if the same amount of the same carbohydrate is eaten on its own.

The *glycemic load* of a meal or diet is defined as the glycemic index of a food multiplied by the total carbohydrate content. It is therefore a way to quantify the impact of a food on levels of blood glucose. For example, a carrot has a high glycemic index, but because it contains relatively little carbohydrate, it has only a modest glycemic load.

The insulin response to high glycemic food

Throughout most of human evolution, humans did not have access to highly refined, simple sugars. The carbohydrates eaten by our ancestors tended to be complex carbohydrates such as grains, starches, or sugars embedded in complex foods (such as fructose in fruit). In other words, most food had a low glycemic index. After a meal, therefore, blood glucose levels would usually increase slowly, and for a sustained period as the food was digested.

The human insulin response to an increase in blood sugar has evolved to expect a moderate, sustained flow of sugars into the bloodstream. If a meal is eaten which creates a large increase in blood glucose, the body responds to that increase with a large increase in insulin to contain the rate of increase of blood glucose. The insulin causes the glucose to be absorbed into muscle and fat cells, and stimulates the liver to create glycogen and fatty acids, and the blood sugar level duly drops.

However, if the meal has a high glycemic load, the increase in blood glucose is large but short-term (compared to the same amount of carbohydrate eaten in low-glycemic form). Because humans have, through most of their history, eaten low-glycemic foods, the insulin reaction is calibrated on the expectation that any increase in blood glucose will be sustained. The body therefore over-reacts to the increase in blood glucose by producing more insulin than is needed. As a result, blood glucose falls too much, and within a few hours, the person feels hungry again – perhaps even dizzy and weak. If they respond to that hunger by eating a further meal containing high-glycemic sugars, they will set off a further cycle of spike and then trough in blood sugar and insulin.

This is a very damaging state of affairs. The swings in insulin levels are directly damaging to cells, increasing the risk of cancer and heart disease. The swings in blood sugar lead to over-eating[6] and obesity,[7] attention disorders,[8] hyperactivity and to a reduction in natural immunity.

More importantly, as we shall see in the next section, the excessive insulin levels caused by these cycles in blood sugar can cause very severe long term damage to the body.

Insulin resistance

The exaggerated peaks and troughs in blood sugar and insulin lead over time to a reduction in the body's ability to respond to insulin, and the development of a condition called *insulin resistance*. Insulin resistance is a reduction in the ability of the body to respond to insulin by disposing of glucose in the liver, muscles, and other tissues. Somewhere between a quarter and a half of all Americans now suffer from some level of insulin resistance.[9]

When insulin resistance sets in, the body does not respond sufficiently to the insulin, and so glucose levels in the blood stream remain elevated. The pancreas responds by generating larger and larger doses of insulin to try to maintain proper blood sugar levels. This overproduction of insulin is called *hyperinsulinemia*. As a result, people with moderate insulin resistance have higher than normal levels of insulin in their bloodstream, to maintain normal blood glucose levels.

Eventually, however, the pancreas is no longer able to produce enough insulin to maintain blood sugar levels at their normal level. The result is an increase in levels of blood glucose, which results in impaired tolerance to glucose, and eventually type-II diabetes.

Insulin resistance is a common feature of, and a contributing factor to, a number of common health problems, including type II diabetes, polycystic ovary disease, *hypertriglyceridemia*, high blood pressure, cardiovascular disease, sleep deprivation, certain hormone-sensitive cancers, and obesity.[10]

Carbohydrates and insulin resistance

Studies have not found a clear link in humans between diets that are high in carbohydrate and insulin resistance or diabetes.[11] (By contrast with human studies, there is considerable evidence from rats linking diets high in carbohydrates to insulin resistance.[12])

Indeed, some studies found a negative correlation between high carbohydrate diets and diabetes; but this evidence is complicated by the fact that lower carbohydrate diets are typically higher in fat; which is in turn correlated with obesity, which causes diabetes.[13]

However, there is considerable evidence which indicates that *high-glycemic* (as opposed to high-carbohydrate) diets are linked to insulin resistance. Studies have found that a history of

consumption of foods with a high glycemic load predicts the development of type II diabetes in women and men.[14] In one study, switching to a low glycemic diet reduced insulin resistance within 4 weeks.[15]

Evidence from dietary patterns and disease incidence suggest that insulin resistance is caused by a variety of factors, not just the body's response to processed sugars and glycemic load from the diet. Other contributory factors which tend to increase insulin resistance include obesity, increased consumption of saturated and total fat, decreased intake of fibre and lack of exercise.

Carbohydrates and cardiovascular disease

Studies have consistently found a link between a diet high in carbohydrates and elevated levels of triglycerides in the bloodstream.[16]

These studies do not distinguish between high-carbohydrate diets, and high GI diets. It may well be that the effect of high carbohydrate diets on blood lipids is mainly or entirely the effect of eating refined and processed sugars.

There is a very well established link between eating diets high in refined sugars, and high GI diets, and the accumulation of triglycerides in the blood (*hypertriglyceridemia*). [19] This increase is the result of both increased production of triglycerides by the liver, and impaired clearance of VLDL from the bloodstream.[20] If anything, the link is arguably even better established than the link between high GI diets and insulin resistance.[21]

These high levels of triglycerides in the blood are a major cause of cardiovascular disease. A recent report[22] showed that women who consumed diets with a high glycemic load had an increased risk of coronary heart disease; those with the highest glycemic load in their diet more than doubled their risk. This analysis sought to isolate the effect of high GI foods by taking account of differences in total energy intake, obesity and other major dietary and non-dietary risk factors for heart disease.

Several other studies have shown that the consumption of a diet with a high glycemic load is independently associated with an increased risk of developing obesity, type-II diabetes, cardiovascular disease, and certain cancers. These studies find that simple sugars, especially those containing fructose, are the worst offenders. Even modest changes in the ratio of simple to complex carbohydrate (*e.g.* from 40:60 to 60:40) can induce hypertriglyceridemia.[23]

Full circle: the link between insulin resistance and heart disease

So we have seen that there is good evidence of a link between high GI diets and insulin resistance, and between high GI diets and hypertriglyceridemia. But there is also a well-established statistical evidence of a direct link between insulin resistance and

hypertriglyceridemia. It seems that the excessive production of insulin is itself a cause of hypertriglyceridemia. However, we do not know enough about the impact on the liver of excessive insulin to know whether hypertriglyceridemia is the result of a failure of insulin to inhibit the secretion by the liver of VLDL-triglycerides (in other words, the liver itself suffers from insulin resistance), or whether it is the result of an impairment in the body's ability to remove VLDL-triglycerides from the blood.

Metabolic Syndrome

Metabolic Syndrome is also known as *Insulin Resistance Syndrome* or *Syndrome X*. The syndrome describes a collection of symptoms which are often found together, including:

- high blood pressure
- abdominal obesity
- insulin resistance and compensating over-production of insulin (*hyperinsulinemia*)
- high levels of triglycerides (*hypertriglyceridemia*)
- low levels of HDL or "good" lipoproteins
- low levels of antioxidant vitamins and DHEA (*dehydroepiandrosterone*)
- high cortisol levels
- depression

This combination of symptoms appears frequently to be the result of a combination of insulin resistance and hypertriglyceridemia, which are both independently caused by excessive consumption of high-glycemic foods and/or simple sugars.

Metabolic syndrome is a very dangerous condition. People with metabolic syndrome are four times more likely to have a heart attack or stroke that those who do not have it.[24]

Refined sugars may be dangerous even if they are low GI

We have seen that there is evidence linking diets with a high glycemic load to insulin resistance, obesity, heart disease and metabolic syndrome. But the direct impact of simple sugars on blood glucose, as measured by the glycemic index, appears to be only part of the story. The body also has to cope with other sugars entering the bloodstream, such as fructose, which has to be converted into glucose by the liver before it can be used for energy. Because glycemic index measures only glucose levels; and because fructose does not impact directly on blood glucose levels, it has a relatively low GI of 23.

Though it has a low GI, fructose is associated with increased obesity, which may result from its effects on hormones, particularly those related to hunger. Fructose also appears to lead to especially high triglyceride synthesis;[25] and this effect is independent of insulin resistance (since fructose does not increase blood glucose levels and does not stimulate the production of insulin).[26]

This suggests that, even though it has a low GI, fructose also presents a significant challenge to human health, and may make a significant contribution to metabolic syndrome, obesity and heart disease. So while we have good evidence suggesting that we should avoid high GI diets, it does not follow that refined sugars with low GI are safe.

The nutritional value of different sugars

In terms of total calories, processed sugars are an efficient way to increase your energy intake. But getting your energy from less refined, more natural sources, like fruits and grains, means you will also be getting other necessary nutrients, which are not found in refined sugar.

Because sugar does not contain vitamins or minerals, it is often called *empty calories*. For example, if you choose 200 calories of sugar, *e.g.* in a soft drink, instead of from a starchy food, *e.g.* in a bowl of muesli, you will not benefit from the fibre, vitamins and minerals that the food also contains.

In one study of children, a decrease in the intake of many essential nutrients was associated with increasing total sugar intake.[27] There are at least nine other studies[28] which show a decreased intake of at least one micro-nutrient where there is a higher sugar intake. In other words, the replacement of whole foods with high-sugar foods compromises adequate dietary vitamin and mineral intake from whole food sources.[29]

It is not the case that using more “natural” forms of sugar will cure the empty calorie problem. Sucrose is 99 per cent sugar, as most of the impurities have been removed. Less-refined sugars, such as cane juice crystals and maple syrup, are still about 90 percent sugar. While these contain marginally more minerals than sucrose, the quantities are so small that you would have to eat huge quantities of these less refined sugars to eat meaningful amounts of valuable minerals.

Carbohydrates in the diet

In the U.S, people have eaten about the same amount of carbohydrates – an average of about half a kilogram, or 1lb, a day – for about the last hundred years, and possibly longer. But instead of whole grains and vegetables, people are now getting more of those carbohydrates in the form of processed grains and sugars.

Food	Energy (kcal)	Total carbohydrates	Sugars (g)	Starch (g)	Fibre (g)	Glycemic Index
Brown rice	141	32.1	0.5	31.6	0.8	76

White rice (steamed)	141	30.9		30.8	0.1	97
Spaghetti, wholemeal	113	23.2	1.3	21.9	3.5	37
Spaghetti, white	104	22.2	0.5	21.7	1.2	41
Baked potato	77	18	0.7	17.3	1.4	85
Wholemeal bread	217	42	2.8	39.3	5	69
White bread	219	46.1	3.4	42.7	1.9	70
Lentils red	100	17.5	0.8	16.2	1.9	26
Peas	79	10	1.2	7.6	4.5	48
Sweetcorn	122	26.6	9.6	16.6	1.4	55
Banana	95	23.2	20.9	2.3	1.1	55
Raisins	272	69.3	67.3	0	2	64

The biggest change is the increase in consumption of “high fructose corn syrup”, which is widely added to food and now provides 20 percent of energy from carbohydrates. High fructose corn syrup has a chemical structure similar to sucrose: it is a disaccharide composed of one glucose and one fructose molecule. (Despite the name, this sweetener is no higher in fructose than sucrose.) High-fructose corn syrup is used extensively in soft drinks, baked goods such as cakes and muffins, sauces, prepared desserts, and other processed foods such as syrups

added to coffees. While the average consumption per person of refined cane and beet sugars has decreased by 35% over the thirty years to 2000, the consumption of corn sweeteners has increased by 277%.[31] High-fructose corn syrup consumption has increased 40-fold from less than 0.5 grams daily per person in 1970 to 53.9 grams daily per person in 2003.[32]

Part of the reason for the recent increase seems to be that the food industry is responding to a fad for low carbohydrate diets which count only “net” carbs (eg The Atkins Diet) and low glycemic index diets (eg The South Beach Diet). As consumers seek low carbohydrate or low glycemic foods, the industry is increasingly sweetening foods with articifical, low-glycemic index sweeteners, particularly fructose, which do not count as carbohydrates in these diets. They do, however, contribute to total calorie intake, and these refined sugars may be just as dangerous for human health as other refined sugars.

Looking for sugar in the ingredients list

In 2000, the intake of added sugars for the average American was 2.5 times that of the dietary guidelines, and almost one-half of the total came from high-fructose corn syrup.[33] About 20 percent of calories from carbohydrates – or 10 percent of total calories – now come from corn syrups. As well as corn syrups, a variety of processed and refined sugars are added to diets. Sugar might also be listed on ingredient panels as: corn syrup, corn sweetner, honey, glucose, fructose, levulose (a technical name for fructose), dextrose, lactose, maltose, malt syrup, molasses, raw sugar, sucrose, white grape juice concentrate, fruit juice concentrate, apple syrup, invert sugar, syrup and maltodextrin.

Implications for runners

Runners are at considerably lower risk than the population as a whole for conditions such as coronary heart disease, obesity, diabetes and cancers.[34]

However, while runners are less likely to suffer from diseases related to obesity, we are not immune from a number of the conditions that are caused by poor diet. For example, increased exercise does not eliminate the build up of plaque in blood vessels resulting from excessive intake of refined sugars and saturated fats.

Athletes also need a higher level of minerals and vitamins, and other nutrients, in their food than do sedentary people because they sweat more destroy more blood cells, and place greater demands on their bodies. It is important that, when eating the increased number of calories needed to support an active lifestyle, those calories are accompanied by the requisite higher levels of nutrients found in unrefined and unprocessed foods. Athletes should get their carbohydrates from fresh fruit and vegetables, whole grains, pulses and foods high in fibre.

The mechanism by which simple carbohydrates increase the level of fatty acids in the bloodstream is significantly more pronounced when glycogen stores are full, as the liver preferentially stores surplus glucose in the form of glycogen. During and immediately after

exercise, therefore, the body is able to absorb simple carbohydrates and store them as glycogen more readily.

Simple, readily accessible (typically high-glycemic) carbohydrates are a good choice of food during and immediately after exercise, because this is when the body adapts by increasing the overall size of glycogen stores in response to depletion during exercise. But even then, it is preferable to avoid refined sugars if possible, as these are stripped of their other beneficial ingredients. Choose instead bagels, bread, fruit and vegetables, preferably organically grown so that they include as many of the natural nutrients as possible.

Conclusions

The increase in the diet of refined sugars has coincided with an epidemic level increase in obesity, diabetes, cardiovascular disease and cancers. The evidence suggests that the vary large increase in the amount of refined and added sugars in our diet may, through its impact on insulin production and on the accumulation of plaque in the blood vessels, play an important role in this epidemic.

There is a growing body of scientific evidence to suggest that the nature of the sugars eaten, rather than high levels of carbohydrate, is an important determinant of the impact of sugars on our health. However, the glycemic index is also an imperfect measure of the impact on our bodies. Some refined sugars do not lead to an increase in blood glucose, but they appear to share the characteristics of other refined sugars in causing the build up of blood fats, obesity and heart disease.

While it is unlikely that sugar in moderation is dangerous to health, the normal western diet can no longer be described as containing a moderate amount of sugar. Very large quantities of refined sugars are being added to our food by the food industry, and sugar has replaced a large proportion of the other forms of carbohydrate in our diet. Many factors other contribute to obesity and heart disease, including lack of exercise, excessive consumption of saturated fats, and eating too much in general. The evidence is that it is also important how much sugar we eat, and in what form.

Given the current state of scientific knowledge, the best advice appears to be to take active steps to avoid refined sugars, preferring instead whole grain, unrefined, high-fibre carbohydrates. [35]

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January 2005

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Footnote 2

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[http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=7958495&itool=iconabstr]

Footnote 13

Ginsberg H, Olefsky JM, Kimmerling G, Crapo P, Reaven GM. (1976). Induction of hypertriglyceridemia by a low-fat diet. J Clin Endocrinol Metab 1976;42:729–35.
[Abstract:
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=1262445&itool=iconabstr]
Swapping to a low fat, high carb diet may increase triglyceride levels.

Reaven GM. (1997). Do high carbohydrate diets prevent the development or attenuate the manifestations (or both) of syndrome X? A viewpoint strongly against. Curr Opin Lipidol 1997; 8:23–7.
[Abstract:
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=retrieve&db=pubmed&list_uids=9127707&dopt=Abstract]
[*Argues that low fat/high carbohydrate diets may not help with the treatment of syndrome X; because it is simpler to better to switch from saturated to unsaturated fats.]*

Footnote 14

Daly M. Sugars, insulin sensitivity, and the postprandial state. (2003). Am J Clin Nutr 2003;78(suppl):865S–72S.
[Full text: http://www.ajcn.org/cgi/content/full/78/4/865S]
A history of consumption of foods with a high-glycemic load predicts the development of type II diabetes in women and men.

Frost G, Wilding J, Beecham J. (1994) Dietary advice based on the glycaemic index improves dietary profile and metabolic control in type 2 diabetic patients. Diabet Med. 1994 May;11(4):397-401.
[Astract:
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_ui

ds=8088113&itool=iconabstr]
High glycemic load associated with type II diabetes.

Schulze MB, Manson JE, Ludwig DS, Colditz GA, Stampfer MJ, Willett WC, Hu FB. (2004) Sugar-sweetened beverages, weight gain, and incidence of type 2 diabetes in young and middle-aged women. *JAMA*.
[Abstract: http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=15328324&itool=iconabstr]
Finds an association between consumption of sugar sweetened drinks (except fruit juices) and increased risk of type-II diabetes, possibly because of the large amounts of rapidly absorbed sugars.

Salmeron J, Ascherio A, Rimm E, et al. Dietary fiber, glycemic load, and risk of NIDDM in men. Diabetes Care 1997;20:545–50.
[Abstract: http://care.diabetesjournals.org/cgi/content/abstract/20/4/545]
Large-scale, observational studies indicate that the long-term consumption of a diet with a high glycemic load is a significant independent predictor of the risk of developing type 2 diabetes

Salmeron J, Manson J, Stampfer M, Colditz G, Wing A, Willett W. Dietary fiber, glycemic load, and risk of non-insulin-dependent diabetes mellitus in women. JAMA 1997;277:472–7.
[Abstract: http://jama.ama-assn.org/cgi/content/abstract/277/6/472]
Large-scale, observational studies indicate that the long-term consumption of a diet with a high glycemic load is a significant independent predictor of the risk of developing type 2 diabetes

Raben A, Holst JJ, Madsen J, Astrup A. (2001). Diurnal metabolic profiles after 14 d of an ad libitum high-starch, high-sucrose, or high-fat diet in normal-weight never-obese and postobese women. Am J Clin Nutr 2001;73:177–89.
[Full text: http://www.ajcn.org/cgi/content/full/73/2/177]
Finds that sucrose has no more effect than starch on insulin and lipids

Footnote 15

Frost G, Keogh B, Smith D, Akinsanya K, Leeds A. (1996) The effect of low-glycemic carbohydrate on insulin and glucose response in vivo and in vitro in patients with coronary heart disease. Metabolism 1996;45:669–72.
[Abstract: http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=retrieve&db=pubmed&list_uids=8637438&dopt=Abstract]
A low glycemic diet can reduce insulin resistance within four weeks.

Footnote 16

Ginsberg H, Olefsky JM, Kimmerling G, Crapo P, Reaven GM. (1976). Induction of hypertriglyceridemia by a low-fat diet. J Clin Endocrinol Metab 1976;42:729–35.

[Abstract: http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=1262445&itool=iconabstr]
Swapping to a low fat, high carb diet may increase triglyceride levels.

Katan MB, Grundy SM, Willett WC. (1997). Should a low-fat, high-carbohydrate diet be recommended for everyone? Beyond low-fat diets. N Engl J Med 1997;337:563–6.
[Full text (subscription): http://content.nejm.org/cgi/reprint/337/8/562.pdf]
Low fat diets can lead to excessive carbohydrate consumption. It would be better to replace saturated fats with unsaturated fats.

Ullmann D, Connor WE, Hatcher LF, Connor SL, Flavell DP. (1991) Will a high-carbohydrate, low-fat diet lower plasma lipids and lipoproteins without producing hypertriglyceridemia? Arterioscler Thromb 1991;11:1059–67.
[Abstract: http://atvb.ahajournals.org/cgi/content/abstract/11/4/1059]
Finds that moving gradually to higher carbohydrate diet does not lead to elevated levels of fatty acids.

Footnote 19

Parks EJ, Hellerstein MK. (2000). Carbohydrate-induced hypertriacylglycerolemia: historical perspective and review of biological mechanisms. Am J Clin Nutr 2000;71:412–33.
[Full text: http://www.ajcn.org/cgi/content/full/71/2/412]
*A very useful summary of the evidence linking carbohydrate consumption to elevated levels of fatty acids in the blood. ****

Frayn KN, Kingman SM. (1995). Dietary sugars and lipid metabolism in humans. Am J Clin Nutr 1995;62 (suppl):250S–61S.
[Abstract: http://www.ajcn.org/cgi/ijlink?linkType=ABST&journalCode=ajcn&resid=62/1/250S]
Increased triacylglycerol following eating sugars caused by both increases in production of VLDL, and reduced clearance.

Hudgins LC, Hellerstein MK, Seidman CE, Neese RA, Tremaroli JD, Hirsch J. (2000). Relationship between carbohydrate-induced hypertriglyceridemia and fatty acid synthesis in lean and obese subjects. J Lipid Res 2000;41:595–604.
[Full text: http://www.jlr.org/cgi/content/full/41/4/595]
Eating simple sugars increases the level of fatty acids in the blood.

Hudgins LC, Seidman CE, Diakun J, Hirsch J. Human. (1998). Fatty acid synthesis is reduced after the substitution of dietary starch for sugar. Am J Clin Nutr 1998;67:631–9.
[Full text: http://www.ajcn.org/cgi/reprint/67/4/631]
Replacing sugars with starch in the diet reduces the build up of fatty acids

Liu G, Coulston A, Hollenbeck C, Reaven G. (1984). The effect of sucrose content in high and low carbohydrate diets on plasma glucose, insulin, and lipid responses in hypertriglyceridemic

humans. J Clin Endocrinol Metab 1984;59:636–42.
[Abstract: http://jcem.endojournals.org/cgi/content/abstract/59/4/636]
While high carbohydrate diets elevate plasma glucose, insulin and fatty acids; small variations in sucrose levels are significant determinants.

Vidon C, Boucher P, Cachefo A, Peroni O, Diraison F, Beylot M. (2001). Effects of isoenergetic high-carbohydrate compared with high-fat diets on human cholesterol synthesis and expression of key regulatory genes of cholesterol metabolism. Am J Clin Nutr 2001;73:878–84.
[Full text: http://www.ajcn.org/cgi/content/full/73/5/878]
Finds that the impact of a high carbohydrate diet only occurs if it consists mainly of simple sugars.

Daly ME, Vale C, Walker M, Littlefield A, Alberti KG, Mathers JC. (1998) Acute effects on insulin sensitivity and diurnal metabolic profiles of a high-sucrose compared with a high-starch diet. Am J Clin Nutr 1998;67:1186–96.
[Full text: http://www.ajcn.org/cgi/reprint/67/6/1186]
Sucrose causes greater build up of fatty acids in the blood than starch

Ginsberg H, Olefsky JM, Kimmerling G, Crapo P, Reaven GM. (1976). Induction of hypertriglyceridemia by a low-fat diet. J Clin Endocrinol Metab 1976;42:729–35.
[Abstract: http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=1262445&itool=iconabstr]
Swapping to a low fat, high carb diet may increase triglyceride levels.

Albrink MJ, Ullrich IH. (1986). Interaction of dietary sucrose and fiber on serum lipids in healthy young men fed high carbohydrate diets. Am J Clin Nutr 1986;43:419–28.
[Abstract: http://www.ajcn.org/cgi/content/abstract/43/3/419]
High sucrose diets can cause a build up of fatty acids in the blood.

Footnote 20

Mittendorfer B, Sidossis LS. (2001). Mechanism for the increase in plasma triacylglycerol concentrations after consumption of short-term, high-carbohydrate diets. Am J Clin Nutr 2001;73:892–9.
[Full text: http://www.ajcn.org/cgi/content/full/73/5/892]
Looks at the mechanism by which a high carb diet increases levels of fatty acids in the blood.

Parks EJ, Krauss RM, Christiansen MP, Neese RA, Hellerstein MK. (1999). Effects of a low-fat, high-carbohydrate diet on VLDL-triglyceride assembly, production, and clearance. J Clin Invest 1999;104:1087–96.
[Abstract: http://www.jci.org/cgi/content/abstract/104/8/1087]
A summary of the mechanism by which high carbohydrate diets increase levels of fatty acids.

Footnote 21

Hellerstein MK. (2002). Carbohydrate-induced hypertriglyceridemia: modifying factors and implications for cardiovascular risk. Curr Opin Lipidol 2002;13:33–40. ***
[Abstract: http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=retrieve&db=pubmed&list_uids=11790961&dopt=Abstract]
Simple sugars (especially those containing fructose) are a more important cause of hypertriglyceridemia than are complex carbohydrates (starches). This effect is better established than the effect of simple sugars on blood glucose and insulin resistance.

Footnote 22

Liu S, Willett WC, Stampfer MJ, et al. (2000). A prospective study of dietary glycemic load, carbohydrate intake, and risk of coronary heart disease in US women. Am J Clin Nutr 2000;71:1455–61.
[Full text: http://www.ajcn.org/cgi/content/full/71/6/1455]
Epidemiologic data suggest that a high dietary glycemic load from refined carbohydrates increases the risk of coronary heart disease, independent of other known coronary disease risk factors.

Footnote 23

J. Wylie-Rosett, C. Segal-Isaacson, and A. Segal-Isaacson. (2004) Carbohydrates and Increases in Obesity: Does the Type of Carbohydrate Make a Difference? Obes. Res., November 1, 2004; 12(suppl_2): 124S - 129S.
[Abstract: http://www.obesityresearch.org/cgi/content/abstract/12/suppl_2/124S]
[Full text (subscription): http://www.obesityresearch.org/cgi/content/full/12/suppl_2/124S]
A useful summary which argues that the link between high glycemic index diets and obesity is well established; but we need more evidence on the impact of other refined sugars such as fructose, which is an important determinant too. It is better to eat non-starchy vegetables and higher-fibre legumes.

Foster-Powell K, Holt SH, Brand-Miller JC. (2002). International table of glycemic index and glycemic load values. Am J Clin Nutr. 2002 Jul;76(1):5-56
[Full text: http://www.ajcn.org/cgi/pmidlookup?view=full&pmid=12081815]
A set of tables of GI for different foods. Discusses impact of GI on health. Describes reasons for variation in measurements of GI.

Fried SK, Rao SP. (2003). Sugars, hypertriglyceridemia, and cardiovascular disease. Am J Clin Nutr 2003;78(suppl):873S–80S.
[Full text: http://www.ajcn.org/cgi/content/full/78/4/873S]
There is no evidence linking high carb diet to heart disease; but there is evidence that high-glycemic diets are associated with higher triacylglycerol and greater risk of coronary heart disease.

Footnote 24

Lakka HM, Laaksonen DE, Lakka TA, Niskanen LK, Kumpusalo E, Tuomilehto J, Salonen JT. The metabolic syndrome and total and cardiovascular disease mortality in middle-aged men. JAMA. 2002 Dec 4;288(21):2709-16.
[Abstract: http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12460094&dopt=Abstract]

Footnote 25

Jeppesen J, Chen YI, Zhou MY, Schaaf P, Coulston A, Reaven GM.(1995). Postprandial triglyceride and retinyl ester responses to oral fat: effects of fructose. Am J Clin Nutr 1995;61:787–91.
[Abstract: http://www.ajcn.org/cgi/content/abstract/61/4/787]
Fructose increases accumulation of fatty acids in the blood.

Footnote 26

Thresher JS, Podolin DA, Wei Y, Mazzeo RS, Pagliassotti MJ. (2000) Comparison of the effects of sucrose and fructose on insulin action and glucose tolerance. Am J Physiol Regul Integr Comp Physiol 2000;279:R1334–40.
[Full text: http://ajpregu.physiology.org/cgi/content/full/279/4/R1334]
Suggests that fructose is a primary mediator of insulin resistance.

Footnote 27

Farris RP, Nicklas TA, Myers L, et al. (1998). Nutrient intake and food group consumption of 10-year-olds by sugar intake level: the Bogalusa Heart Study. *J Am Coll Nutr.* 1998; 17: 579–585.
[Full text: http://www.jacn.org/cgi/content/full/17/6/579]
Children who get more of their calories from sugar lack essential nutrients.

Footnote 28

United States Department of Agriculture (2005) *Nutrition and Your Health: Dietary Guidelines for Americans.*
[http://www.health.gov/dietaryguidelines/dga2005/report/HTML/D5_Carbs.htm]

Footnote 29

Howard, BV, Wylie-Rosett J. (2002). Sugar and cardiovascular disease: a statement for healthcare professionals from the Committee on Nutrition of the Council on Nutrition, Physical

Activity, and Metabolism of the American Heart Association. Circulation 2002;106:523–7.
[Full text: http://circ.ahajournals.org/cgi/reprint/106/4/523]

Footnote 30

Leeds, A, Brand Miller J, Foster-Powell K, Colagiuri S. The Glucose Revolution (2000). (London: Hodder & Stoughton).
Diet book advocating low GI diet.

Footnote 31

Putnam, J, Allshouse, J, Kantor, LS. (2002). U.S. per capita food supply trends: more calories, refined carbohydrates, and fats *Food Rev.* 25,2-15
Examination of trends in US food consumption.

Footnote 32

Economic Research Service (2004). USDA Briefing Room: Sugar and sweetener: data tables. Table 52: high fructose corn syrup: estimated number of per capita calories consumed daily, by calendar year.
[Full text: http://ers.usda.gov/Briefing/Sugar/Data/data.htm]
Shows a substantial increase in the role of fructose as an additive to processed foods.

Footnote 33

Putnam, J, Allshouse, J, Kantor, LS. (2002). U.S. per capita food supply trends: more calories, refined carbohydrates, and fats *Food Rev.* 25,2-15
Examination of trends in US food consumption.

Footnote 34

Noakes, T. (2001) The Lore of Running. Oxford University Press.

Footnote 35

Murphy SP, Johnson RK. (2003). The scientific basis of recent US guidance on sugars intake. Am J Clin Nutr 2003;78(suppl):827S–33S.
[Full text: http://www.ajcn.org/cgi/content/full/78/4/827S]
An overall, and somewhat inconclusive, survey of the impact of sugars.