

What is so special about carbohydrates?

It's a sugary world!

Bread, potatoes for dinner, logs in the fireplace, fuel for your emotions, a page in your favorite novel, frosting on the cake, the green crest of the hill, an apple's dripping juice, coke, grass, the wooden boat, a horse's hay, the bee cube filled with honey, milk, a winner's driving energy, oak trees, rice for the world's masses. All based on sugar. Sweet sugar, rigid sugar, no taste sugar, flowing sugar, sugar crystals, sugar! One sugar in all of this. The sugar that flows in your blood. The sugar that drives your thoughts and pushes the sprinter faster and faster.

Glucose! Free or bound. Bound so that you can digest it. Bound so that you cannot digest it. Glucose! It covers the land. It powers the fish of the sea. All plants live of it. All animals survive on it. Glucose, probably the most common carbon substance in the world. Glucose, the foundation of life! Glucose, the core of cellulose and, therefore, of trees and flowers and grass. Glucose, that sugar bound in huge numbers in plant and animal starch (glycogen). Glucose, half of milk sugar and table sugar. Glucose, the basis for our genetic development through hundreds of thousands of years. One of the three simple digestible sugars and the only one in large quantities throughout millennia.

Are carbohydrates good food?

Eat low glycemic index food! LowCarb diets cure overweight! Eat fat! Watch out for sugar bombs! The daily press is full of articles advocating diets with special consideration taken to carbohydrates. Is there anything in these claims? Is bread really "bad food"? Are potatoes really safe? Do we need to eat carbohydrates at all?

The answers are really quite simple. Our genetic makeup seems to say that we should eat a good portion of carbohydrates daily. We must use carbohydrates to supply much of the energy needed to drive our bodies. In spite of this, we can survive quite well without consuming sugars and starch. For example, people survive for long periods just by eating

pemmican, a mixture of lean meat (proteins) and fat. Helmer Hansen gave some striking examples of this in his book about the "Gjøa" expedition under leadership of Roald Amundsen (Gjennom Isbaksen, Aschehoug, Oslo, 1941 p.26-27). He wrote (my translation) "they (the Eskimos) valued only meat. Coffee made them sick, candy and chocolate were too sweet for their taste. They did not like bread. They ate a little of our food after staying with us for a while, but their stomachs did not tolerate this. They were "cured" by eating meat so rotten that the dogs would not have it". Clearly, our genetic makeup plays an important role in our dietary choices! No single diet is correct for all people!

Never the less, all of us must have a stable supply of sugar in our blood. Mental activity is completely dependent on the availability of blood sugar. Neither fats nor proteins can be utilized by the brain! Strenuous muscle activity, so-called anaerobic work, uses only sugar as an energy source. So, if you choose not to eat carbohydrates your liver must make sugar from proteins. If you eat neither carbohydrates nor proteins, you will use your own body's proteins as a source of blood sugar. No sugar, no life!

So, which carbohydrates should we consume? This puzzle is also easy to answer. While we can choose among an almost endless variation in the protein or fatty components of our diet, the choice of carbohydrates is quite limited. Regardless of which food we choose, there are only three energy-giving carbohydrates (sugars) that we can make use of. Of these, only glucose is really adequate as an energy source.

Those three sugars are glucose, fructose, and galactose. Table sugar (sucrose) is merely a combination of glucose and fructose while milk sugar (lactose) is made up of glucose and galactose. Starch and glycogen (animal starch) are simply glucose polymers. Glucose is the only breakdown product of these huge molecules. NO OTHER CARBOHYDRATES CAN BE DIGESTED AND ABSORBED BY HUMANS! All the "others" wind up in the large intestine as energy sources for bacteria. If more than approximately five grams daily end up there the result is often "explosive" diarrhea. What happens if one eats more table sugar than can be absorbed? Ask a kid who has eaten too much candy! Eating more sugar than one can digest and take up in the small intestine leads to "stomach pain" when it is carried to the large intestine. The bacteria in the large intestine take over with production of gas as a result.

Another simplification: Most adults cannot digest milk sugar (lactose). For at least 80% of the world's adult population, milk must be avoided or fermented before consumption. Otherwise, "explosive" diarrhea! Lactose is a disaccharide composed of glucose and galactose. These are joined in such a way that a special enzyme is needed to split them. Most adults lack this digestive enzyme and cannot release galactose from lactose. Note that this is genetically determined. Most adults in northern European populations can digest lactose throughout life. Most Asian and African adults cannot. Galactose cannot be a major energy source for most of the world's mature population.

Another simplifying fact: Fructose has been a major dietary component for less than 200 years. Limited amounts of fructose are found in many fruits. Honey was a scarce but real treat in earlier years. It is super sweet, containing about 40% fructose and 30% glucose.

The sugar industry has made fructose a major source of energy today. Common sugar or sucrose is another sugar dimer, composed of glucose and fructose. Most of us utilize fructose quite well; too well in fact. Fructose metabolism in the liver is at least twice as rapid as that of glucose, leading to synthesis of fats and accumulation of these in the blood. Unlike glucose, fructose does not cause release of insulin from the pancreas. Now, insulin has many functions and one of these is to dampen appetite. That means that half of the sugar we eat gives no satiety signal to the brain. As we all know, we can drink a lot of soda pop without feeling full. Can it be that fructose is to blame for the rapid increase in overweight seen during the past twenty years? A new source of dietary fructose is "high fructose corn syrup". This is a sweetening product made from corn. The syrup is modified enzymatically such that 40-60% of the glucose content is converted to fructose. It is an inexpensive replacement for sucrose in ice cream and soda pop. In the USA, sucrose consumption has been reduced during the past decade or two, but the total sugar consumption has increased because of the use of high fructose corn syrup.

Which carbohydrate remains? Glucose, usually in its polymeric form, starch. That will say that only glucose or its "polymeric form" can normally fill our carbohydrate requirements. Starch is digested to glucose, rapidly absorbed in the small intestine and burned in most tissues. That which is not immediately used is stored as the glucose polymer glycogen in muscles and the liver. Glucose causes controlled release of insulin and its metabolism is very well controlled by this hormone.

What about that "glycemic index" business? Well, some carbohydrate-rich foods are quickly and easily digested and their glucose content is rapidly taken up. These have been defined as "high glycemic index" foods. White bread, corn flakes and soda pop are among these. Whole wheat bread, thick noodles and bran cereal preparations are examples of foods that are more slowly digested and taken up in the intestine. But note, they ARE taken up completely and give energy (and, if you do not use that energy, lead to overweight)! If this were not the case they would be sources of gastric distress!

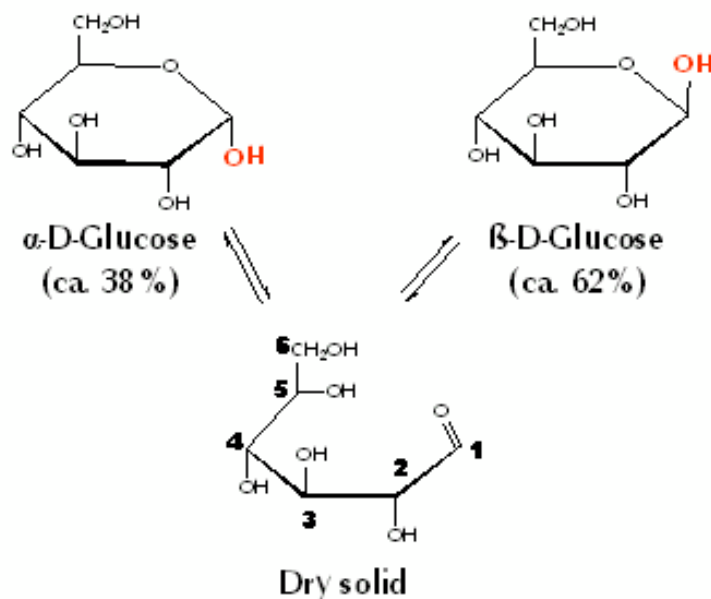
As you now understand, only three monosaccharides compose the total useful carbohydrate spectra, and glucose dominates as an energy source for most of us. Just what are the chemical difference between glucose, fructose and galactose?

The Monosaccharides

Glucose

The central actor in this game is glucose, known also as blood sugar and dextrose. Glucose is a "reducing sugar", that is, it is oxidized by ferric (Fe^{+++}) and cupric (Cu^{++}) ions yielding reduced metal ions. Glucose also

Mutarotation of glucose



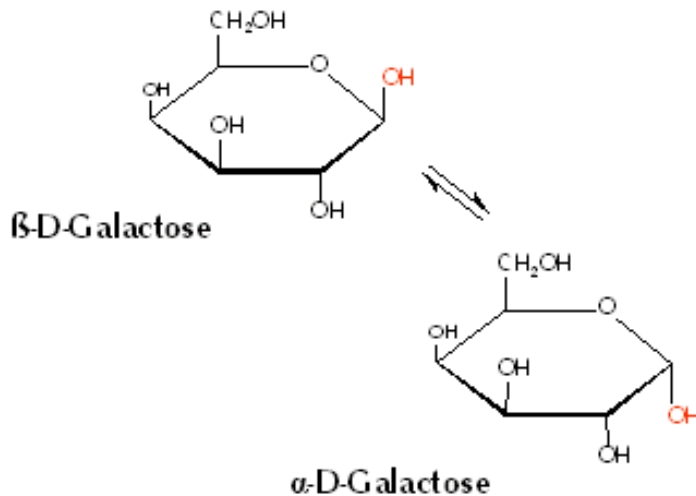
reacts with itself, leading to formation of a "6-ring" (5 carbon atoms and 1 oxygen atom) seen in the figure. The really interesting thing with the ring form is that there are two forms of these, α -D-Glucose and β -D-glucose. The first of these has the C-1 hydroxyl group on one side of the ring and the 6th carbon in a side chain on the other side. In the beta form of glucose, both of these are on the same side of the ring. Did I hear you say "so what"? Just wait and you will see that this makes a huge difference in digestibility of polysaccharides made from these compounds.

The carbonyl group in glucose also allows the sugar to interact with many other compounds. In the body this applies especially to various proteins. The level of glucosylated hemoglobin (HbA_{1c}) is used as a measure of blood glucose levels over time. Much of the pathology seen in diabetes is thought to arise from reaction of glucose with the body's proteins. [Click here for more information about the toxic effects of glucose.](#)

Galactose

The next of the three monosaccharides we should consider is galactose. This sugar is found in milk and milk products. Galactose is another "reducing sugar" and forms a "6-ring" when dissolved. Galactose is, in fact, almost identical to glucose, the only exception being the position of the hydroxyl group on carbon 4. Once again, you will soon see that this "minor" difference has large effects on metabolism and can lead to serious symptoms in some newborn babies. As with glucose, galactose undergoes mutarotation and has an alpha and a beta form. Only the beta form reacts with glucose to form the disaccharide lactose or milk sugar. Again, this has major effects on digestibility of lactose. We shall soon see that beta-linked disaccharides and polysaccharides are indigestible in humans. Lactose is the only exception to this rule. We have a specific enzyme, lactase, which catalyzes cleaving of lactose. While almost all new-born have this enzyme, most adults do not make lactase and are "milk intolerant". You will find more about this by clicking here.

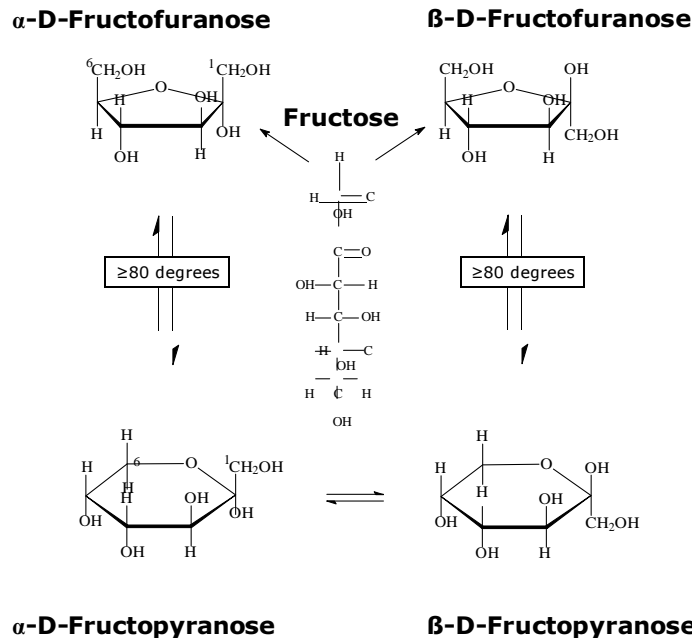
Mutarotation of galactose



Fructose

Fructose is the third of the common sugars found in our diet. As with glucose, it forms ring structures when dissolved. Because the carbonyl group is on carbon two, fructose forms a "5-ring" where 4 carbon atoms and one oxygen atom are involved in the ring structure. Carbons one and six are attached to the central ring and this affects both intestinal uptake and

Isomeric Forms of Fructose



metabolism of fructose. Fructose is a natural but limited component of our diet, being found in limited amounts in many types of fruits and berries. Honey contains about 40% fructose giving it a very sweet taste.

Until about 150 years ago fructose played a minor role in human nutrition. Fructose is one of the two sugars that are found in sucrose or "table sugar". It was first in the 1800s that sugar plantations in the West Indies began to produce so much sucrose that this disaccharide became an important part of the western diet. With the increase in "table sugar" or sucrose, came a huge increase in the amount of fructose in our diet. While the consumption of sugar per person was some few kilograms per year around 1850, the normal annual consumption in most western lands is now around 40-50 kilograms. During the last decade high fructose corn syrup (HFCS) has replaced a good bit of the sugar formerly used in soft drinks, ice cream and cakes. Monomeric fructose and glucose are now used in large quantities in the food industry.

While the use of "sugar", that is common table sugar or sucrose, has decreased in several western countries, total sugar consume has increased greatly. In the USA, sucrose consume is down to 33 kg per person per year. This figure is often presented as evidence for a major decrease in

"sugar" consumption. However, **total** sugar consumption (including glucose and fructose) is now approximately 60 kg per year. [Click here for more information](#). Fructose is not handled as other monosaccharides in our bodies. I will come back to this when I take up digestion of sucrose as well as under a discussion of the metabolism of monosaccharides.

Fructose is sweeter than table sugar (sucrose). For this reason it has been suggested by many "health experts" that one can cut down on the calorie intake by replacing sucrose with fructose. Since fructose is 30-40% sweeter than sucrose, one should be able to eat 30-40% less sugar and get the same sweetening effect. This sounds logical, but is quite wrong.

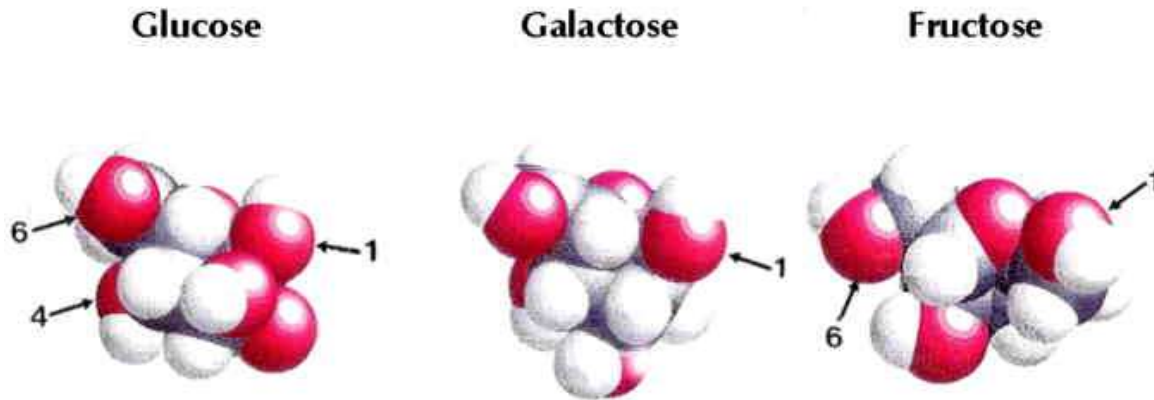
Firstly, fructose has a different metabolism than glucose (the other half of sucrose and the same as blood sugar). The enzyme required to initiate significant fructose metabolism (fructokinase) is only found in the liver. Fructose is not a direct energy source for other tissues. Fructose metabolism is not controlled. It goes very quickly forward unrelated to hepatic energy utilization. This results in an increased synthesis of lipids and increased serum lipoproteins. Fructose is substrate "number one" for gluconeogenesis and gives a rapid rise in serum glucose levels.

That increased sweetness of fructose is a feature of the 5-ring. Now, it just happens that fructose in solution and at higher temperatures (tea and coffee, baking etc.) goes over to a 6-ring form. More correctly, the alpha and beta furanose forms (5-ring) are in equilibrium with the pyranose (6-ring) forms. The latter dominate in solution and the equilibrium reaction speeds up as the temperature increases. The pyranose forms of fructose are not sweeter than table sugar and one must use just as much fructose as sucrose in warm drinks and baked goods to obtain if equal sweetness.

The message should be clear: increased fructose use, either from sucrose or the pure monosaccharide, is associated with increases in blood lipids (increased LDL and cholesterol and decreased levels of HDL) that are associated with cardiovascular disease. Fructose misses its sweetness when warmed and may not help to reduce caloric intake. Sorry, but fructose is not good for you! For a good discussion of fructose in relationship to the obesity epidemic, go to a recent article in the [American Journal of Clinical Nutrition by clicking here](#).

Let's look at some 3D models of these sugars.

Monosaccharides normally found in food



Here the red balls represent oxygen, the white hydrogen and the black, carbon. The black? Where are they? In the center of the sugar molecules! The ring structures are very compact. The hydroxyl groups, those with a white hydrogen on a red oxygen ball, dominate the surface of these molecules. Metabolic reactions involving large enzymes have to react with the easily reached hydroxyl groups. Look at glucose and galactose where the only difference is the position of the hydroxyl group on carbon 4. While the left side of galactose resembles that of glucose, something has happened at carbon 1. Here the hydroxyl group is turned upward and is more accessible to enzymatic attack. We shall see that metabolism starts at carbon 1.

Compare the 5-ring fructose with the other sugars. It is really larger than both of the others and the hydroxyl groups on both carbon 1 and 6 are relatively open to enzymatic attack. Because of these "little" differences, both the intestinal uptake and metabolism of fructose differ from that of the other monosaccharides.

Metabolic derivatives of fructose have that 5-ring form. Perhaps that follows the more reactive -OH groups in the furanose forms of the sugar.

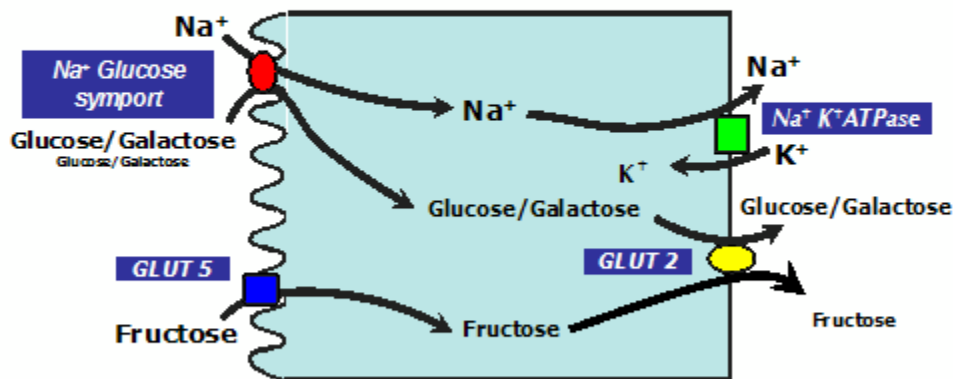
Adsorption of sugars in the small intestine

Adsorption of sugars in healthy persons occurs almost exclusively in the the small intestine. Adsorption is limited to the monosaccharides glucose, fructose and galactose. These "mono-sugars" are large molecules and can only cross cell membranes when a "carrier protein" is present. These are very specific and do not react with and transport other sugars. This topic is

very well discussed in most textbooks. I will take up just a few points here.

Note that uptake of glucose and galactose is through a Na^+ -coupled symport driven by ATP. This is an active process. Uptake of these sugars is not dependent on a concentration gradient over the intestinal wall. Glucose and

Intestinal Monosaccharide Transport



galactose are completely "sucked up" from the intestinal lumen and transferred over the mucosal cell to the intestinal circulation.

Fructose is not bound and moved by this active transporter. One of the glucose transport proteins (GLUT5) carries out this job. [\(You can learn more about these transporters by clicking here\)](#). GLUT5, just as the other members of the glucose transport protein family, is passive. It moves the sugar down a concentration gradient. That is, it requires that the fructose concentration in the intestinal lumen is higher than that in the mucosal cell. Transport from these cells to the circulation must also go "downhill". It is GLUT2, another glucose transport protein that carries fructose as well as glucose and galactose over the basolateral side of the intestinal cell. Why is the concentration of fructose in the portal blood so low that a concentration gradient is maintained during fructose uptake? The key to this is the liver's GLUT2 and fructokinase. These are very active and fructose is speedily removed from portal blood into the liver and trapped there as fructose-1-phosphate. This maintains the fructose concentration gradient over the intestinal cells in spite of the rapid absorption of the sugar.

I mentioned above that this localized uptake has to do with healthy persons. Intestinal inflammation reduces uptake; the enzymes responsible

for splitting of disaccharides are produced in the intestinal mucosal cells. Inflammation causes temporary loss of these and a reduced uptake of sugars. Leakage of mono and disaccharides over intestinal membranes can also take place when these are inflamed.

Other monosaccharides used as dietary supplements

There is no uptake mechanism for other monosaccharides in the small intestine. As we shall soon see, lacking or reduced uptake in the small intestine leads to further transport of sugars to the large intestine. There, bacteria overtake digestion, giving gas formation, pain and diarrhea when the transfer of sugar to the large intestine exceeds around 4-5 grams daily. This lack of small intestine uptake for some monosaccharides permits them to be used as "calorie-free" sweetening agents.

Sweet Sorbitol

Sorbitol is a sugar alcohol produced in our bodies by reduction of glucose. It is then oxidized and converted to fructose in some few tissues. Sorbitol is readily oxidized in the body and therefore should be a good source of energy. BUT, it is not taken up in the small intestine since no sorbitol carrier is found there. Ingested sorbitol gives food a sweet taste with only a slight contribution to the energy content of food. Around 1.5 kcal/g is taken up in the large intestine, mostly as two and three-carbon fragments. Sorbitol is used in chewing gum and some "drops" as a "non-fattening" sweetener. As long as one takes less than those 4-5 grams all is well. More can give "stomach pain" and diarrhea.

Ribose

Ribose is an essential part of our metabolic system, being found in phosphorylated form in, among other things, ATP. The diet supplement industry has presented ribose as a strength and energy giving stuff. Take a



Ribose

The most elemental source of energy in the body is ATP (Adenosine Triphosphate). This cellular energy is required to power your workouts and to recover from intense training. Your body must produce enough Ribose before it can build ATP, and that can take many days. D-RIBOSE enhances your body's ability to restore its ATP stores faster and more efficiently, for better recovery and muscle growth.

look at the next figure. In a way, the statement is correct, but the body builds all the ribose-5-phosphate it needs from glucose. Dietary ribose is completely unnecessary for ATP synthesis. And, ribose is not adsorbed in the small intestine! The label states that the daily dose should be around 4-5 grams, so the producer seems to know that this stuff is not taken up. It yields around 1.5 kcal/g as is the case with other material that is digested by colon bacteria.

A Norwegian weightlifter was disqualified at the Olympic Games in Australia a few years ago. He had taken ribose daily but stopped before the Olympics. He began to lose strength, he said, and began with ribose again. Ribose "worked" for him. But why? He was taken for doping with Nandrolon, an anabolic steroid. His ribose was found to be "contaminated" with the stuff. He has sued the producer for this. The bottom line: there is no need for dietary ribose and no intestinal uptake of this sugar!

The Disaccharides and their digestion.

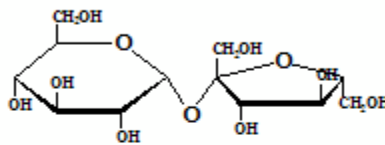
All good things come in "threes" when we study carbohydrates. First three monosaccharides, now three disaccharides, each synthesized from two of those simple sugars.

The BOND

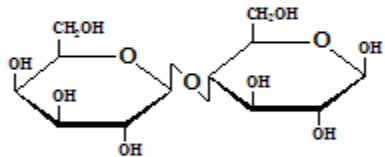
The first thing we must have straight is the nature of the bonds between the sugars in a disaccharide or polysaccharide. These ester bonds are formed through a dehydration of two hydroxyl groups, one on carbon 1 and the other on carbon 4 of either glucose or galactose, or carbon 2 of fructose. If the carbon-1 hydroxyl group is in the beta position (same side of the ring as

Digestible Disaccharides in Food

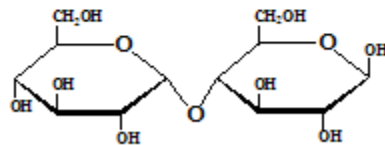
Sucrose
(Glucose-fructose)



Lactose
(Galactose-glucose)



Maltose
(Glucose-glucose)



carbon 6), the resulting bond is a beta 1-4 glucosidic bond. If the hydroxyl group was under the ring (an alpha hydroxyl group) the resulting bond is called an alpha 1-4 or alpha 1-2 glucosidic bond. None of the enzymes that cleave glucosidic bonds in our digestive tract can cleave beta 1-4 linkages. Well, there is ONE exception, lactase, which is specific for the beta 1-4 glucosidic bond in lactose, the sugar found in milk.

Sucrose

The disaccharide sucrose is composed of glucose and fructose joined by an alpha 1-2 bond. As mentioned above, sucrose is a relative "new-comer" in our diet. It is actually not especially sweet, and we use large amounts to get the taste we desire in breakfast cereals, soft drinks, cakes and sweets. Just imagine using the same amount of salt in a recipe! Sugar has become a major energy contributor. A year's consumption of around 50 kilograms of sucrose calculates out to about 500-600 kilocalories per day (our total requirement is around 2000-2500 kcal/day). The problem is that we use sugar as though it was a spice, while we pile on a lot of empty calories in the process! See the later chapter about weight regulation.

Sucrose is readily cleaved by sucrase, one of the enzymes produced by and bound to the mucosa cells of the small intestine (for details see Marks, Marks and Smith, Basic Medical Biochemistry or another of the popular and good text books covering medical biochemistry). An important point to remember here is that the activity of sucrase does not normally limit the rate of absorption of the monosaccharides that result from cleavage of sucrose (glucose and fructose). There is ample enzymatic activity to digest the amounts of sucrose usually consumed by adults. However, intestinal infections or inflammation may well give rise to a temporary loss of mucosa cells with a loss of enzyme activity. Any carbohydrate that reaches the large intestine will serve as a substrate for bacteria there. The resulting gas formation coupled with release of two and three-carbon products from bacterial metabolism can give pain and perhaps diarrhea. We see the same phenomena in children who eat and drink too many sweets. They simply take in more sucrose than they manage to digest. The resulting tummy ache is the result of bacterial gas formation in the large intestine.

Lactose

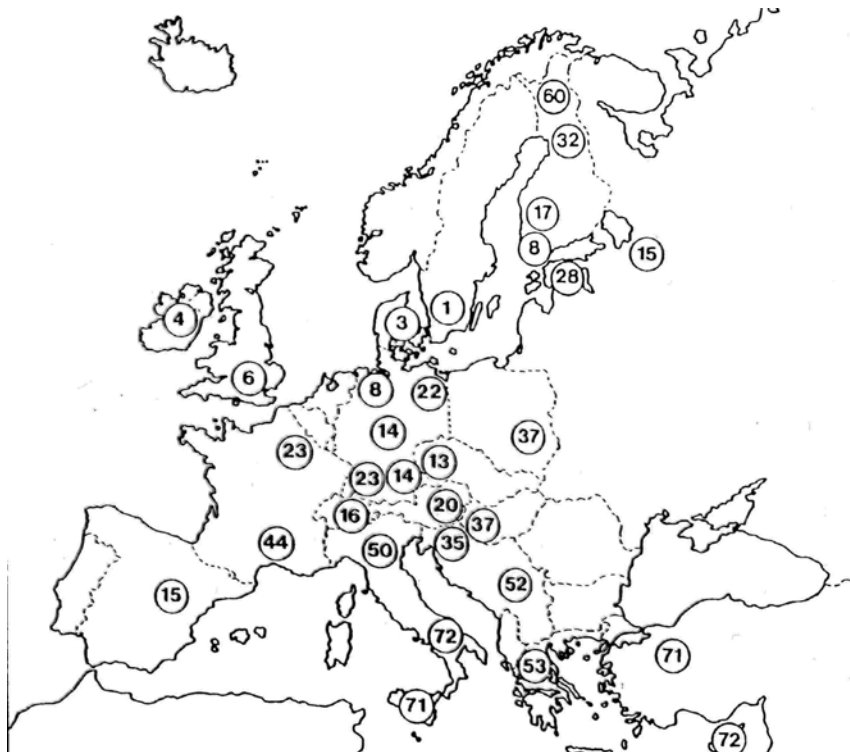
Lactose (or milk sugar) is a disaccharide composed of galactose and glucose. The enzyme responsible for synthesis of lactose is specific for the beta form of galactose. This results in formation of a beta 1-4 glucosidic bond between the two monosaccharides. Lactose is the only substance with such a bond that can be digested in the human intestine. This requires the presence of lactase, another of the enzymes involved in carbohydrate digestion that is produced and bound to the mucosal cell membrane. In contrast to sucrase and isomaltase, lactase production is restricted and can limit the digestion of lactose. Do you remember Mom who said "you mustn't drink milk when you have a tummy ache"? She "knew" that lactase production was reduced when the intestinal mucosa was enflamed. The result of drinking milk and other fresh dairy products in the absence of lactase is the transport of the sugar to the large intestine. Bacterial digestion of this leads to gas production. In addition will bacteria produce two and three carbon compounds that increase the osmotic pressure of the intestinal contents, thus retaining water. The result; "explosive diarrhea".

Lactase is usually found in the intestine at birth in all human races. In a global prospective one can state that production begins to decrease at the one to two-year stage and is all but lacking from the age of five years. Lactase-deficient adults can utilize milk products without discomfort if they are cultured with bacteria or yeast before ingestion. Culture milk, sour cream, yogurt and many cheeses are typical products in which microorganisms are allowed to digest lactose for us.

Lactase Persistence (adult milk tolerance).

Most adults do not produce intestinal lactase and do not tolerate milk. However, several isolated groups do continue to drink milk throughout life. These have one of several genetic mutations that allow sustained lactase synthesis. The map below shows the distribution of lactase in European adults (note that the figures represent lactose-intolerance or lack of lactase). Scandinavians (except Finns) tolerate milk as adults. The distribution of this characteristic throughout Europe largely follows trade and raiding routes which were common in Viking times.

Percent of the adult population who are lactose-intolerant.



Most of the people in Sweden, Denmark, England, Ireland, Norway (not shown but about 5% intolerant) and northern Germany have lactase activity as adults. In southern Europe the percent of the population that is lactose-intolerant is much larger. Spain was a frequently visited trade partner for Scandinavians in the Viking period. Here we find a relatively large part of the population with the "life-long lactase gene".

Most of the world's population is lactose-intolerant. Asians, many tribes in Africa, Jewish people and many others do not produce lactase as adults. However, many small and relatively well-defined groups do exhibit lactase persistence throughout life.

Lactase persistence is a result of one of several genetic mutations in the region of the lactase gene. A Finnish group reported a mutation in an enhancer-region close to the lactase gene which prevented it from being turned off after infancy. This seems to be the secret to adult lactase synthesis in Northern Europe. Exhaustive studies of lactase persistence in Africa by Sarah Tishkoff's group have revealed another mutation in the same gene region that also codes for continued synthesis of lactase. These studies are discussed in a recent news article in Nature entitled "How Africa learned to love the cow" (Nature 444, 994-996, 2006). [Click here if you have library connections.](#)

Espen



Meet Marianne and Espen. He is a happy Norwegian boy who is now about 9 years old. As a newborn he was not quite as happy. He was a typical colic-child who screamed and cried after every feeding and had an almost constant diarrhea. It was not until he was about 18 months old that it was discovered that he had a lactase-deficiency. Lactase deficiency is extremely rare in newborn, but it does occur. A lactose-free diet cured all of his

problems. Today he can drink one glass of milk without symptoms, but after two...

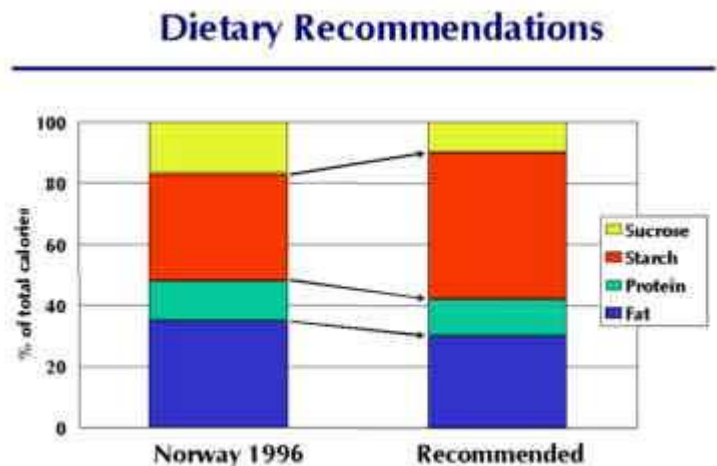
Maltose

Maltose and isomaltose are not major components of our diet but are formed from polysaccharides in the intestine through the action of amylase. These disaccharides are glucose dimers and are digested by maltase and isomaltase that are produced by and bound to the mucosal cell. The glucose formed is then readily adsorbed.

Polysaccharides

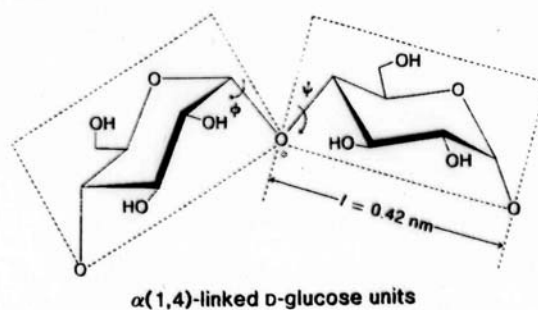
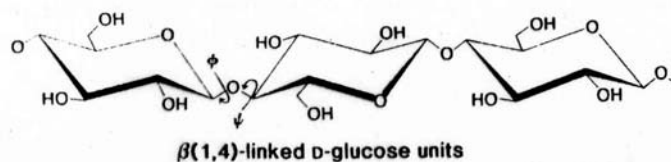
Sugar is stored in both plant and animal tissues in polymeric forms, that is, as starch and glycogen. Why go to the trouble of making (and breaking) these huge molecules? The answer lies in the fact that all water-soluble materials exert what we call osmotic pressure. They draw water from less concentrated solutions to more concentrated areas. If we loaded up our cells with sugar, water would be drawn from blood into our tissues, eventually rupturing the cells and thus destroying the tissues. To avoid this tragic fate we join sugar molecules together, forming huge polysaccharides. A glycogen or starch molecule containing 1000 sugar molecules exerts an osmotic pressure equal to 1/1000 of that of an equivalent number of free sugars. The polymerization process allows us to build up an active sugar reserve of about 90-100 grams in the liver and 300-400 grams in skeletal muscles. The liver glycogen buffers blood sugar and can replace this approximately five times. The glycogen stored in muscles serves only as an energy source in these tissues.

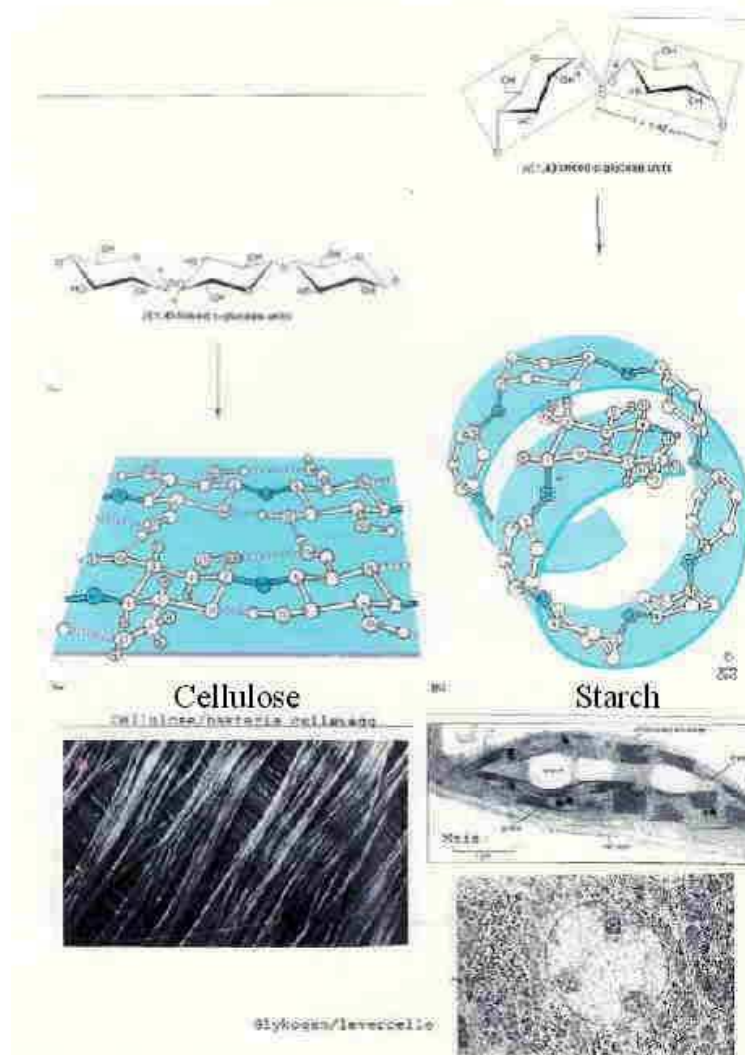
Polysaccharides are the basis of the agriculture-based diet. When we examine the recommendations from the Norwegian Council for Nutrition and most other official agencies, we see that about 55-60% of the daily energy supply should come from carbohydrates. Most should come from starch and not sugar. [Go to the section on fructose for an explanation of this.](#)



The nature of the polysaccharide components of food and the digestion of these are well documented in most of the available text books of nutrition or biochemistry. I will not take up this subject up here.

However, there is one area that I do wish to consider: the differing structures of the digestible and indigestible polysaccharides. We have seen that glucose units (the building stones of the polysaccharides in nature) can be connected with either alpha 1-4 or beta 1-4 bonds. This is dependent upon the specificity of the enzyme that catalyses formation of these polysaccharides. Some require glucose with an alpha hydroxyl group on carbon 1, others work only with beta C-1 glucose. The resulting glycosyl bonds (alpha 1-4 or beta 1-4) arrange the connected glycosyl groups at differing angles to each other. As you can see from the figure, beta 1-4 linked glycosyl units lie in a single plane while alpha 1-4 bonded glycosyl groups lie at a pronounced angle to each other. This "minor" difference results in entirely dissimilar 3-dimensional structures. Beta 1-4 linked glycosyl groups form sheets that pack together to form the rigid structure we know as cellulose. This is the major polysaccharide of grass, leaves and trees and is said to include around 50% of all biological carbon found on our planet. Digestion of cellulose is entirely dependent upon intestinal flora in herbivores. The anatomy of our digestive system precludes the presence and function of these organisms. Cellulose is, however, of importance in human nutrition in that fiber (good old indigestible cellulose) is an essential part of our diet, giving bulk to our food and promoting intestinal motility.



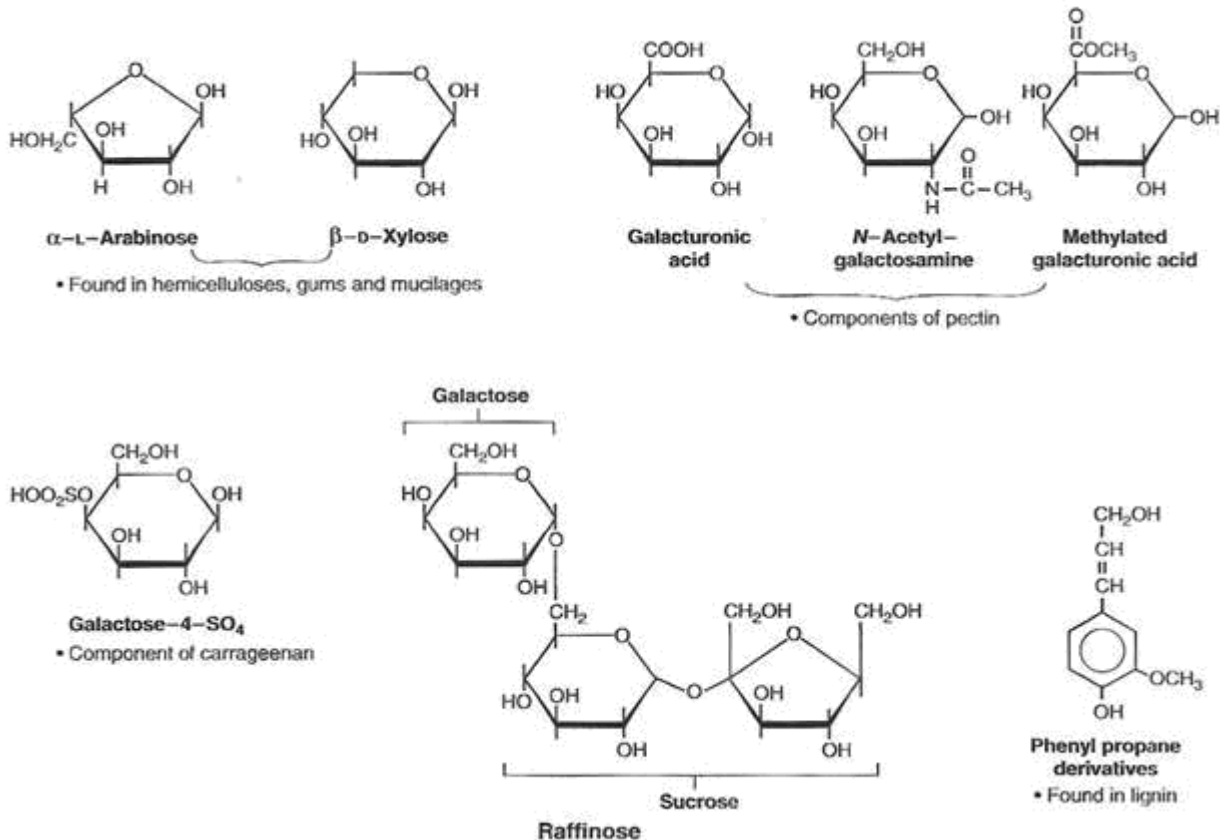


As you can see from the next figure, starch in plants or glycogen in us has a completely different structure. The α (1-4) glucosyl groups in these polysaccharide form structures with thousands of glucosyl groups bound together in a spiral. These are found packed together in granules. The granules bind water and the enzymes necessary for their synthesis and catabolism, providing a compact system for very rapid rates of synthesis and breakdown of starch and glycogen.

These two figures are modified from Geoffrey L. Zubay's excellent book, Biochemistry, 4. edition.

Soluble fiber

There are many other carbohydrates found in varying amounts in our diet, many of which can neither be digested or absorbed in the small intestine. These migrate further to the large intestine where they serve as substrates for the intestinal flora. We have all observed the effects of eating large portions of pea soup, baked beans and other legumes. These have a variety of mono-, di- and trisaccharides that cannot be digested in humans. They are sometimes called anti-nutrients because on their tendency to lead to gastrointestinal disturbances. Some of these are shown in the following figure, taken from Basic Medical Biochemistry, Marks, Marks and Smith. Here are a variety of mono, di and trisaccharides that are not digested by



our intestinal enzymes but are substrates for the bacterial flora of the large intestine. As in the case of lactose intolerance, the metabolism of these compounds leads to formation of gas and varying degrees of discomfort. Note especially pectin (found in apples) and raffinose (found in legumes-beans). Most of us have experienced the effects of these compounds.