Perspective

THE MAGAZINE ON SUGAR AND NUTRITION NO. 1
SEPTEMBER 2006 : SPECIAL ISSUE:

CURRENT SUGAR ISSUES

Foreword

Modern lifestyle is a very topical issue indeed. Many people consume more calories than they burn, and this results in overweight and related diseases such as diabetes and cardiovascular disorders. For this reason, focus is increasingly turning to what we should eat and drink in order to stay fit and healthy, and also to who is responsible for what we consume.

A question of food culture?

We are rarely happy with restrictions – and it is no different when it comes to food. We want to be free to choose, and we do not like to have to say 'no thanks' to food. This is not very convenient in a world where many children and adults have grown up with a food culture in which the culinary experiences of healthy eating have such low priority.

In our opinion, increased appreciation of taste will give a fresh and alternative view of meals as both palatable and healthy. The experience is important because, ultimately, we are responsible for what we eat. That is why the solution for some children and adults who get more than the recommended amount of sugar is not necessarily just to avoid sweet taste, but also to promote individual appreciation of taste – for instance through varied taste experiences that challenge the taste buds.

Shared responsibility for knowledge and information

As a food producer, we share the responsibility of giving consumers the knowledge, information and, not least, inspiration that provide a real possibility to make informed choices and, in so doing, assume responsibility for adopting a healthier lifestyle.
Furthermore, we want to help to concentrate focus on key health concerns in society. We can do this by providing information on the nutritional aspects of sugar and in that way contribute to a balanced debate. That is why we have produced this special edition of Perspective – a 'mini-report' in which we have asked leading scientists and experts in the Nordic Countries to present the latest knowledge about the significance of sugar in a range of health-related areas.

The report mainly reflects the nutrition debate in the Nordic Countries, and addresses the current Nordic Nutrition Recommendations. The report is aimed at those who provide consultancy and advice on health. It supplements the more scientific reports that have been published in recent years. With a total of 10 different topics, we cover a lot of ground, but many of the topics naturally require further research before they can be said to have received a full scientific examination.

Other initiatives

Besides this special edition, Perspective – the magazine on sugar and nutrition is published two to three times a year. The magazine can be downloaded from www.danisco.com. There is also an online version, www.perspektiv.nu in Swedish and Danish, where we publish summaries of research news.

You can read more about Danisco Sugar's activities and views on various nutritional matters in our official health policy at www.danisco.com

We are always at your service for questions, dialogue and debate on sugar and health.

Danisco Sugar

■ *Perspective current sugar issues September 2006.* ■ ***Edition:*** *17,000 in Denmark, 30,000 in Sweden, 5,000 in Finland and 3,000 in the United Kingdom.*
■ ***Publisher:*** *Danisco Sugar, Langebrogade 1, 1001 Copenhagen K, Denmark. Tel.: + 45 32 66 25 00, Fax: + 45 32 66 21 50.* ■ ***Editorial Committee:*** *Marketing Manager Angela Everbäck (editor in chief), Scientific Adviser Ingrid Salomonsson, Product Specialist Kyllikki Kilpi, Nutrition Communication Manager Anne-Mette Nielsen, Danisco Sugar. GCI Mannov.* ■ ***Graphical production:*** *Katrine Boelsgaard.* ■ ***Photographer:*** *Christina Bull.* ■ ***Print:*** *Salogruppen A/S.*
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■ ***E-mail: nutrition.dk@danisco.com. Also visit our web site: www.danisco.com.***

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Sugar consumption

By Ingrid Salomonsson, Scientific Adviser, Danisco Sugar, Malmö, Sweden.

There are several factors that make it difficult to assess sugar consumption and to make comparisons between countries. Firstly, sugar is defined differently, so what is actually included in various studies is not always clearly evident. Secondly, although supply statistics give an estimate of sugar consumption, actual sugar intake is difficult to determine on the basis of existing dietary study methods because sugar is found in products of which consumption is often underestimated. Finally, some sugar is also used for purposes other than foods, while some becomes food waste.

Throughout the world, carbohydrates are an important dietary component and have always been the biggest source of energy. According to the Nordic Nutrition Recommendations (NNR 2004), 50-60% of our energy intake should come from carbohydrates, but sucrose and other refined sugars should not exceed 10% of the total energy intake. This applies in particular to persons with a low energy requirement. For persons with a daily energy requirement of 2,000 kcal, this is equivalent to around 50 g of sugar a day. See article on page 8. In the media it is often said that the consumption of new sugars such as glucose syrup, fructose or other fructose-based products is increasing at the expense of ordinary sugar and that new sugars are not included in the statistics. But what exactly is included under the terms 'added sugars' or 'refined sugars'?

Refined sugars

The term 'added sugars' covers all refined sugars, i.e. energy-giving carbohydrates that provide sweetness but do not generally contribute to the vitamin and mineral requirement. The majority of the added (refined) sugars in the diet comprises 'ordinary sugar' – sucrose from sugar beet or sugar cane – but syrup, invert sugar (equal parts of glucose and fructose), fructose, glucose, glucose syrup, glucose-fructose syrup (isoglucose) and honey are also found in this group.

Brown sugar is also regarded as added sugar. It contains some minerals, mainly potassium and magnesium but the amounts are so low that it may not be declared as it does not make any significant contribution to the daily requirement. Added sugars does not include naturally occurring mono- and disaccharides, which are found in e.g. fruit, juice and milk.

The terms 'added sugars' and 'refined sugars' are used in the Nordic Nutrition Recommendations. As well as the above-mentioned sugars, the sugar recommendations contained in WHO Report 916 also include 'free sugars', i.e. naturally occurring sugar in fruit drinks. Some foreign studies on sugar in the diet include lactose. And some studies also give the total sugar content, i.e. both natural and added sugars, which makes it difficult to assess the percentage of added sugars. The difference in calculation methods makes it difficult to compare the actual sugar intake of different countries.

Determination of sugar content in foods

Another difficulty in calculating sugar intake is determining the actual sugar content in foods and distinguishing between the contents of added and natural sugars. In most foods, the sugar content is calculated on the basis of the added quantity rather than analysis of the sugar content in the end-product.

The calculations can lead to an overestimation of the actual intake because some sugar is broken down in acid products such as drinks and preserves/marmalade. Some also ferments in bread baking. Various analysis methods can be used to determine the individual sugars and total sugar content in foods. In purely chemical terms, there is no difference between natural and added sugar. Ordinary analysis methods are unable to distinguish whether the sugars come from natural sugar in fruit and berries or from added sugar.

How much sugar do we eat?

In the Nordic countries, there are various sources of information on food consumption and eating habits:

- Supply statistics on food consumption per inhabitant
- Data from household budget studies
- Dietary studies

Supply statistics – consumption per inhabitant

Supply statistics are often used to indicate how much sugar is available on average per inhabitant per annum. The amount is obtained by adjusting production data for the sugar content of imported and exported foods respectively. Sugar in imported and exported foods is calculated using a model giving the sugar content for each individual product group indicated by a customs number/item number that is common in the EU. However, the sugar content is stated specifically for each Member State. Furthermore, the amount that is used for animal feed or for other purposes, e.g. the manufacture of pharmaceutical products, is deducted. Consumption is expressed in kilograms per inhabitant per annum, which means that the total consumption for a year is divided by the number of inhabitants. This calculation gives the amount that is available for consumption per inhabitant. However, the statistic is not a measure of how much sugar is eaten; for this, dietary studies are required.

Household budget studies

Household budget studies provide information on various households' expenditure on foods and possibly quantities purchased. They may also compare food consumption in different types of household and in relation to different socio-economic or regional classifications. However, the studies do not provide information on the distribution of consumption between household

members or on cooking methods and waste. It is also difficult to compare households directly.

Dietary studies

Dietary studies provide information on an individual's food and nutrition intake. There are several different methods. Diet recording, where the participant keeps a continuous record of food intake, is one of the most frequently used and best known methods. The advantages of diet recording are that it does not rely on memory and that, through weighing, a precise measure of actual intake is obtained. On the other hand, there is a risk that the participant may consciously or unconsciously change their diet or under- and overreport selected foods. For example, it is well known that foods that are regarded as being "unhealthy" such as cakes, sweets and soft drinks are underreported. The tendency to underreport is especially marked among overweight persons[1-3]. Thus, there are also dietary studies where the participant describes their diet after intake. Here, one of the problems can be that people cannot remember precisely what, and how much, they have eaten. The method places great demands on the interviewer. A future measure of sugar consumption could be breakdown products in urine[4].

Sugar in the Nordic countries

What is the estimated sugar consumption and intake? Although we have figures for both sugar intake and sugar consumption in the Nordic countries, it is difficult to assess the actual intake. For many decades, sugar consumption per inhabitant, i.e. the available consumption quantity, has been relatively constant. In the last few years, however, it has been declining. The difference is that the consumption pattern has changed so that we now get more sugar from soft drinks and sweets, whereas previously we got sugar from desserts, cakes and biscuits. Today, the majority of sugar (approx. 80%) comes from manufactured foods, whereas 40 years ago the percentage of sugar in finished products only made up around 35%. Current consumption is around 40 kg per person per annum. See Figure 1. But the actual intake is estimated at less than 30 kg per person per annum, partly due to waste in the food chain. However, we must be aware that these are average values. Consumption is not evenly distributed. Some groups, especially among children and young people, have a higher sugar intake than others. See Table 1.

Waste in the food chain

In order to get an overview of how much sugar we are actually consuming, we need to take account of waste and of sugar used in other ways. There can be waste in several parts of the food chain – industry, shops and homes. Sugar waste in the food industry is relatively low, possibly a few per cent of the sugar that is bought. Waste in shops is due in part to the fact that bread, cakes, ice cream, desserts and other sugar-containing products are thrown away if they are not sold before their sell-by date. Other waste results from the throwing away of leftover food in restaurants, cafés and private homes. There are no Swedish figures for waste, but according to Danish studies the total average waste for the entire food supply amounts to 20% of the energy in the food that is available for consumption[5]. Sugar waste is probably higher because of sugar's roles as a substrate in the fermentation process in bread-baking and wine production and as a preservative in pickle that is thrown away. An American study indicates waste of approx. 30% of the produced quantity[6].

Differences in calculations

So there is a big difference in estimated sugar intake assessed on the basis of dietary studies and supply statistics[7]. For example, the Danish dietary study in 2000/01[8] indicated an average self-reported sugar intake of approx. 50 g per day, while the quantity that was available in 2001 according to the supply statistics was roughly double that at 97 grams per day[9].
According to national dietary studies, sugar intake measured as a percentage of energy intake has also been relatively constant in recent decades. Neither sugar intake nor sugar consumption has increased. This is based on average values. However, there may have been a shift between the age groups in the population.

Labelling of sweetened foods

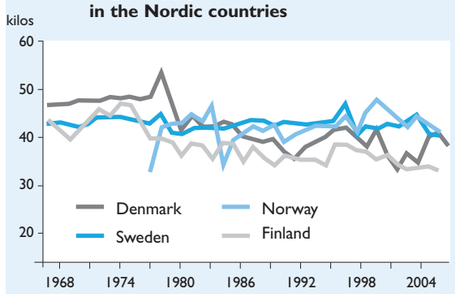
In order to distinguish light/unsweetened alternatives from other products, it has become common to clearly label sugar content. As well as nutrition declarations, there are symbols and statements such as 'unsweetened' and 'low sugar content', but a reduced sugar content does not necessarily mean that the energy content in the product has been reduced. Often the energy content is the same or higher in sugar-reduced products. See article on page 12. Today we have a large selection of products with varying content of added sugar or other sugars. This is good because it gives consumers freedom of choice. But at the same time, it is becoming more difficult to carry out dietary studies. In order to make it easier for consumers, foods should be labelled with the total added sugar content. As things are today, it is difficult for a consumer to assess the proportion of natural or added sugar in mixed foods.

Conclusion

Supply statistics show that sugar consumption has been relatively constant for several decades with a declining trend in the last few years. Dietary studies estimate lower sugar consumption than supply statistics. Food waste explains much of the difference between the supply statistics and the dietary studies, but the actual sugar intake per inhabitant per annum is probably somewhere between the supply statistics and the dietary studies because sugar is found in products that are to some extent underreported in dietary studies.

Figure 1

Sugar supply/consumption per inhabitant in the Nordic countries



The graph is based on figures from 'Food consumption in the Nordic countries 1965-1998, National annual per capita statistics', Nordic Council of Ministers 2001. Figures after 1999 are provided by the Swedish Board of Agriculture, Statistics Denmark, Statistics Norway and Statistics Finland.

Table 1

Intake of sugar, syrup and honey. Given as % of total energy intake (E %)

	Denmark	Finland	Norway	Sweden
	2000/01[8]	2002[10] og 1989/92[11]	1997[12] og 2000[13]	1997/98[14] og 1989[15]
Men	9	9,1	10,2	8,5
Women	10	10,8	9,7	9
Children	14	9-10	16-18	11-15

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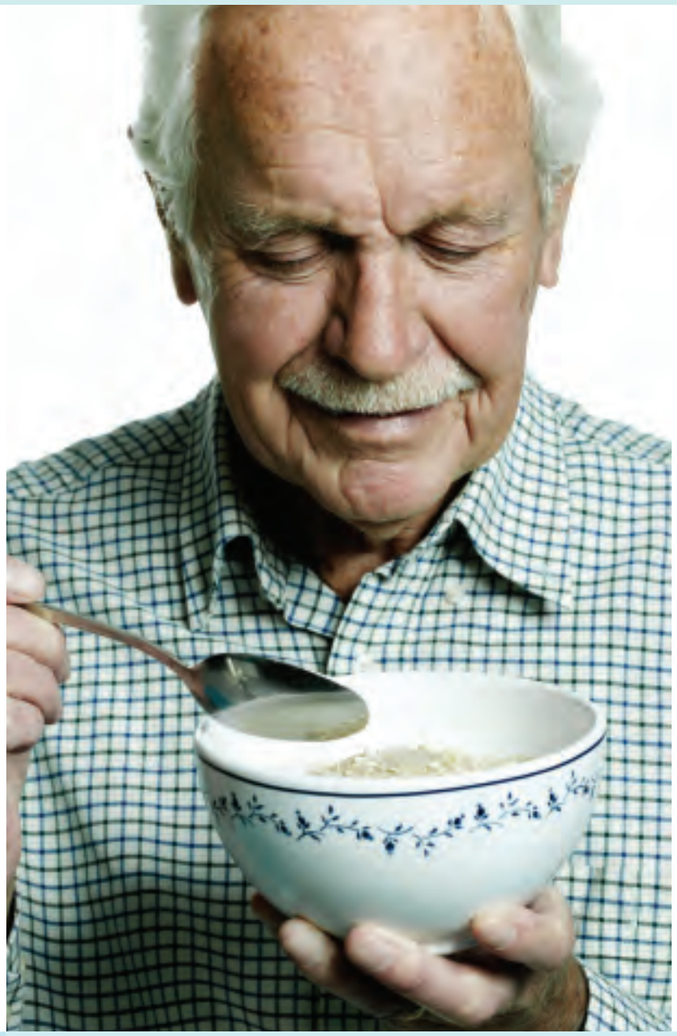
Carbohydrates according to the nutrition recommendations



By Nils-Georg Asp, MD, PhD, Professor of industrial nutrition science, Lund University, Managing Director, SNF, Swedish Nutrition Foundation, Lund, Sweden.

Carbohydrates account for the largest energy share in most diets and they constitute the preferred energy source for the cells in the body. When carbohydrates are available, they are primarily burnt in all cells. On carbohydrate depletion, the cells of most organs are able to switch to burning fatty acids and ketone bodies. However, brain cells and other nerve cells are always dependent on the supply of glucose from the blood. Consequently, a certain blood glucose level must always be maintained. According to Nordic and international recommendations, the carbohydrate and dietary fibre intake should be high, whereas added, purified sugars should be limited to 10% of the energy intake.

Chemical classification

Traditionally, carbohydrates are chemically classified according to molecular size into sugars, oligosaccharides and polysaccharides. The sugars include monosaccharides (simple sugars) and disaccharides (consisting of two monosaccharide units). The most common monosaccharides in food are glucose (grape sugar) and fructose (fruit sugar) that occur naturally in fruits and berries and in lower concentrations in vegetables. Free galactose occurs in small amounts and mainly in fermented milk products.

Sucrose ('sugar') is a disaccharide consisting of glucose and fructose. Sucrose occurs naturally in fruits, berries, vegetables and root crops, but in our food most of it comes from added sugar, i.e. purified (refined) sugar from sugar beets or sugar cane. Lactose (milk sugar) is the other quantitatively important disaccharide and occurs in milk and milk products. Lactose consists of glucose and galactose. Maltose is a disaccharide consisting of two glucose units resulting from enzymatic breakdown of starch.

Sugar alcohols such as sorbitol, xylitol, mannitol, etc., are also regarded as sugars. They occur naturally in fruits and berries and are used as sweeteners and bulking agents in tooth-friendly confectionery products, for example.

Oligosaccharides consist of 3-9 monosaccharides with different monomers and different branching. Fructo-oligosaccharides consisting of fructose and a glucose unit occur naturally in chicory, artichokes and other plants together with inulin, which consists of larger fructose polymers. Galacto-oligosaccharides consisting of galactose and glucose occur in peas, beans and other pulses. Malto-oligosaccharides may be breakdown products of starch, but also synthetically produced glucose polymers such as polydextrose and other resistant malto-dextrins.

Polysaccharides consist of a minimum 10 monosaccharide units and are divided into starch and non-starch polysaccharides (NSP). Starches are pure glucose polymers of two main types: amylose and amylopectin. Amylose consists of unbranched glucose chains forming a helix structure, whereas amylopectin has branched chains.

Cellulose is a dominant non-starch polysaccharide – it is an unbranched glucose polymer. This type of binding results in "straight" molecules easily forming long fibrils. Hemicellulose, pectin and plant gum are other groups of non-starch polysaccharides with a very variable structure of different monomers (glucose, mannose, galactose, uronic acids, etc.), different molecule sizes and branching, and varying physico-chemical properties (water binding, solubility, charge).

Physiological and nutritional classification

The many physiological and nutritional properties of carbohydrates do not comply with the above chemical classification. It was long thought that in general starches were nutritionally superior than sugars – the argument being that the breakdown and uptake of larger molecules ought to be more difficult, thus raising blood sugar levels more slowly. This resulted in the 1970s launch of the "complex carbohydrates" concept in the US. We now know that the nutritional properties of carbohydrates do not comply with the chemical classification.

Carbohydrates cleaved and absorbed in the small intestine produce blood sugar and provide all body cells with carbohydrates, mainly in the form of glucose. They are consequently called digestible or glycemic carbohydrates. The unabsorbed carbohydrates, i.e. dietary fibre, are transported to the colon, providing nourishment for the intestinal bacteria. Access to carbohydrate substrate benefits bifidobacteria and lactobacilli growth in the intestinal flora. In general, dominance by these bacteria is considered characteristic of a favourable and healthy intestinal flora. Non-digestible carbohydrates promoting beneficial intestinal bacteria are called prebiotics (as distinct from probiotics which means living microorganisms supplied through food or medicine that confer health benefits to the consumer).

Metabolism of blood-sugar increasing carbohydrates

Starches are the quantitatively most important type of glycemic carbohydrates followed by sucrose and lactose. Starch degradation starts in the oral cavity through amylase in the saliva and is completed in the upper part of the small intestine. The disaccharides are cleaved by enzymes (disaccharidases) bound in the mucous membrane. Released monosaccharides are transported into the mucous cells by special protein transporters. Galactose and fructose are converted into glucose in the liver.

The liver stores up to a couple of hundred grams of carbohydrates in the form of glycogen generated when carbohydrates are absorbed after a meal. Glycogen is a glucose polymer similar to amylopectin. The majority of the absorbed carbohydrates pass through the liver into the

peripheral circulation. The increased blood glucose level is the primary signal to the β–cells in the pancreas to secrete insulin, which is necessary for most of the cells in the body to take up glucose. Fructose can be taken up without insulin, which is important when given intravenously (directly in the blood). But, fructose in food is primarily metabolised to glucose in the liver, and only a small part reaches the peripheral circulation.

When glucose is available, it is primarily oxidised by all cells. The muscles store a few hundred grams in the form of glycogen. Between meals, the liver mainly uses glycogen to maintain blood glucose level. As these stores of glucose diminish, all the cells that are able to do so gradually switch to burning fatty acids. When there is no more glycogen, glucose is generated from amino acids through the breakdown of muscle protein in particular. This process is called gluconeogenesis.

Ketosis – the body's response to the absence of carbohydrates

In the absence of carbohydrates, the metabolism adapts further to reduce the need for gluconeogenesis. Breakdown products of fatty acids produce so-called ketone bodies that may be burnt in different cells, including brain cells, which gradually switch to burning ketone bodies. This is expedient in terms of saving proteins, especially if the supply is insufficient.

This switch in substance metabolism and concomitant elevation of keton body concentration in the blood is called ketosis. Ketone bodies are ten excreted in the urine. It may be seen as the body's defence against the absence of carbohydrates. The depletion of glycogen stores in the muscles results in functional deterioration, mainly in connection with acute swift movements such as running quickly, glycogen constituting the primary fuel. The absence of glycogen in the liver may result in reduced detoxification capacity. Carbohydrates are eliminated in Atkins diets, and the ketosis allegedly contributes to reducing the sensation of hunger. However, long-term risks cannot be ruled out in connection with protracted ketosis.

Sugar in Nordic nutrition recommendations

Since their introduction around 1970, Nordic and Swedish dietary recommendations have included the goal of reducing added refined sugars – i.e. added sugar – to 10% of the energy intake (10 E%).

As from the 1989 Swedish nutrition recommendations, the sugar recommendation has been aimed specifically at children and adults with a low energy demand (<8 MJ/day) – the same applies to the 1996 Nordic nutrition recommendations (NNR 1996). Even in connection with higher (normal) energy consumption it may be difficult to cover all nutrients and comply with the dietary fibre recommendation at a high sugar consumption, whereas physically active persons may have an intake exceeding 10 E%.

As the new Nordic nutrition recommendations specifically concern the planning of diets for heterogeneous groups, the recommendation concerning added refined sugars has generally been set to 10 E%. The primary rationale for the recommendation to reduce the intake of refined sugars is to ensure adequate intakes of iron and other essential nutrients as well as dietary fibre. It is aimed especially at children and adults with a low energy intake. At higher energy intakes, there is room for a higher sugar intake, which should be taken into account when assessing the diets.

Sugar in international dietary recommendations

In light of the above Nordic tradition to recommend limiting refined sugars and defining a limit compatible with a nutritionally balanced diet, it is hardly surprising that there were practically no reactions from the Nordic countries to the proposals of the WHO report to limit free sugars to 10 E%. American sugar producers reacted all the more strongly. According to the strategy document on which a compromise was gradually reached (WHO 2004), one goal is to limit sugar consumption, but the figure 10 E% has been removed. The 2002 US Food and Nutrition Board (FNB) report states that added sugar should not exceed 25 E%, a level that Nordic studies associate with a markedly lower nutrient intake. The main reason for the very high US figure is probably that many US products are enriched with minerals and vitamins. In conformity with the Nordic recommendations, the risk of excessive consumption of energy/calories from sweetened beverages is emphasised.

One innovation in the US recommendations is that for the first time carbohydrates have been awarded an RDA/RDI value (i.e. the recommended daily intake) of 130 g/day (175 g/day during pregnancy). As is the case with other nutrients, this value has been calculated on the basis of an estimated daily demand of 100 g/day for both adults and children based on estimates of the brain's energy demand. Thus, carbohydrates have obtained the status of at least 'semi-essential' nutrients, which is an expression of the body's physiological demand for a certain amount of carbohydrates.

Table 1

Recommendations concerning sugar and other refined or 'free' sugars in certain topical expert reports and nutrition recommendations

	NNR 1996	FNB, USA, 2002 'Macronutrient report'	WHO 2003 Technical Report Series (TRS) 916	NNR 2004
RDI/RDA For total carbohydrate		130 g/day (175 g/day during pregnancy)		
Limitations concerning added, refined sugars	Max. 10 E% for children and adults with low energy demand (<8 MJ/day)	Max. 25 E%	Max. 10 E% 'free sugars', including sugars occurring naturally in fruit juices	Max. 10 E%

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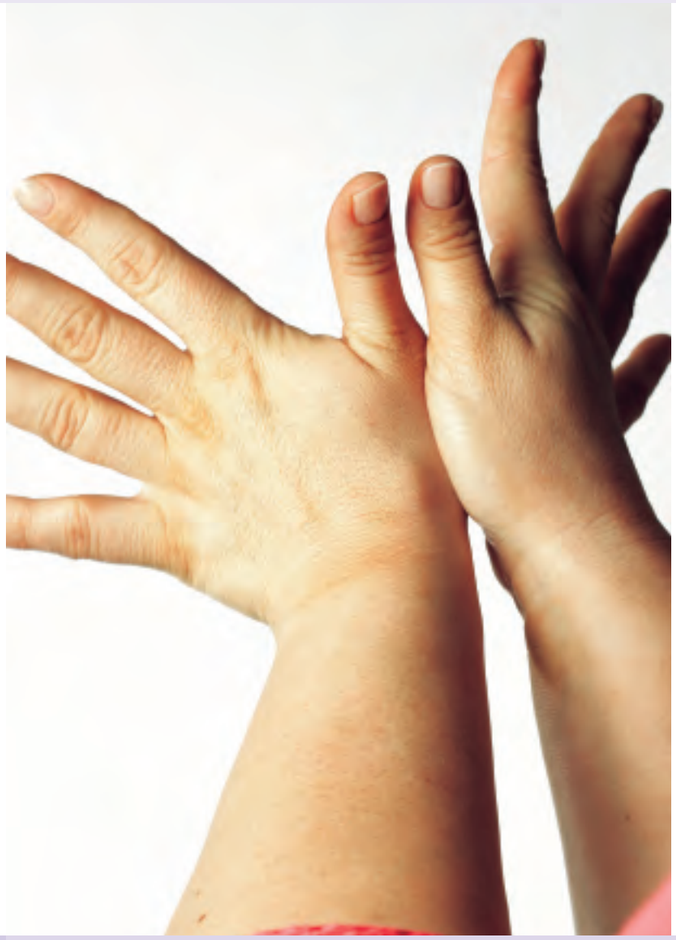
Sugar and overweight



By Arne Astrup, MD, Professor, Head, Department of Human Nutrition, Centre for Advanced Food Studies, Royal Veterinary and Agricultural University, Copenhagen, Denmark.

Differences in the ability of nutrients to fatten are partly due to differences in their ability to sate. It is less important for energy balance whether the nutrient is converted to fat. Sugar and other carbohydrates are only converted to fat to a very limited extent. High carbohydrate intake combined with an energy intake that exceeds the requirement can, however, inhibit fat burning and consequently lead to fat storage. Studies show that the most important thing for losing weight is to reduce dietary fat content. It is less important whether carbohydrates come from starch or sugar since they appear to sate equally well. On the other hand, there is much to suggest that the form in which sugar is consumed is important for its satiating effect. Sugar in solution has been shown to be significantly less satiating than sugar in solid form and can therefore increase total energy intake and create a positive energy balance. We still do not know whether it is important when the liquid sugar is consumed or whether a drink's carbonation plays a role. These conclusions are supported by both the Danish Nutrition Council and the WHO/FAO.

In an overview of the importance of sugar for overweight, it is important to differentiate feelings, intuition and politics from pure science. All calories fatten if you consume more than you burn, but when a special diet is recommended with a view to preventing overweight, it is in recognition of the fact that there are actually differences in the capacity of energy-giving nutrients to fatten. Physiologically, such differences are due to differences in the ability of nutrients:

- to sate, i.e. the ability to inhibit further food consumption after the meal has begun;
- to be absorbed from the gastrointestinal tract; and
- to stimulate heat production, i.e. a thermogenic effect.

Viewed in this physiological light, the question is whether sugar fattens more, less or the same as other carbohydrates, i.e. starch. Added to this is the question whether the form in which sugar is consumed (i.e. solid or in solution) can play an independent role.

Carbohydrate cannot be changed to fat

If we eat more than we burn, fat is stored regardless of whether we get the energy from excessively high amounts of carbohydrate, protein, fat or alcohol. Under normal circumstances, this is not due to the fact that carbohydrate, protein and alcohol are converted to fat (de novo lipogenesis)[1, 2, 3, 4], but to the fact that the burning of the energy-giving nutrients is adapted to what we eat and that carbohydrate, protein and alcohol have highest priority (Fig. 1), i.e. they are burnt before fat. If, for example, we eat an extra portion of carbohydrate and thus obtain a positive energy balance, the burning of carbohydrate is increased so that all carbohydrates are burnt. On the other hand, the burning of fat is reduced correspondingly and the fat that is not burnt is stored (Fig. 2). However, the fact that there is no quantitatively relevant conversion of carbohydrate to fat has been misunderstood and perceived as the impossibility of getting fat from eating carbohydrate and protein. But if we consume more energy than we burn, we will put on weight because protein, carbohydrate and alcohol inhibit fat burning. This means that the food's fat – instead of being burnt – is stored as fat deposits.

Tribal rituals can kick-start lipogenesis

Human beings do not normally convert carbohydrate to fat – it is not necessary because there is abundant fat in our diet. However, if we want to lose weight, it would be a huge advantage if carbohydrate were converted to fat since up to 25% of the energy that carbohydrate contains would be converted to heat instead of being stored as fat deposits.

If the conversion of carbohydrate to fat is to be increased so that it really has an effect, this would require a high energy intake where the energy from carbohydrate – and only from carbohydrate – would need to exceed the body's total energy needs for a period of time. The whole body's burning of carbohydrate would thus be covered and the body would be forced to start converting carbohydrate to fat simply to get rid of the excess carbohydrate. This experiment is carried out regularly in Africa, where, as part of a manhood test, boys take part in the Guru Walla overfeeding tradition in Cameroon[5]. They have to consume more than 29 MJ (7,000 kcal) of carbohydrate a day. This makes them put on 10 kg of fat over a 10-week period in which they only eat 4 kg of fat. Such a low-fat, carbohydrate-rich diet is difficult to eat in such large amounts over such a long period, but under extreme conditions it can be done (it is equivalent to e.g. a daily consumption of 17 l of cola).

Importance of fructose for obesity

In the USA, the use of isoglucose is widespread. Isoglucose has a higher content of fructose than sugar. It has therefore been discussed whether isoglucose contributes more to obesity than sugar. The background to this theory is that consumption, absorption and conversion of fructose differ from that of glucose, partly because the liver's conversion of fructose is quicker than its conversion of glucose. Furthermore, unlike glucose, fructose does not stimulate the excretion of insulin and leptin and so possibly provides less satiety because insulin and leptin are important appetite-regulating signal substances. The fructose content in isoglucose varies, but seldom exceeds 55%. It is therefore unlikely that it would have any practical importance given that sugar contains 50% fructose. Fructose and sugar contain roughly the same amount of energy[6].

Sugar affect on energy balance

Whether sugar fattens more than starch is therefore not determined by the degree of lipogenesis, but by how the sugar in the diet affects human energy balance. There is no basis for sugar having any significantly different thermogenic effect to starch, and sugar is absorbed just as well from the intestine as starch. It therefore remains to assess how sugar sates and affects our subconscious calorie consumption.

Population studies and mechanism studies

Population studies have repeatedly found that individuals

with a high sugar intake are slimmer than individuals with a low sugar intake. But this does not necessarily mean that sugar has a slimming effect. It may also be due to the fact that individuals with weight problems try to lose weight by cutting down on sugar, see article page 4. And it may also be due to underreporting of sugar intake. Experimental studies, known as 'mechanism studies', in which satiety and appetite are measured following the consumption of various meals containing sugar and starch, are not thought to suggest significant differences. The six-month-long CARMEN study, in which 300 overweight individuals were divided up according to starch-rich and sugar-rich diet, established that the most important factor in losing weight is to reduce dietary fat content. Whether the carbohydrate comes from starch or sugar[7] plays a minor role. The study was the largest and longest ever carried out.

Figure 1

When you are in energy balance

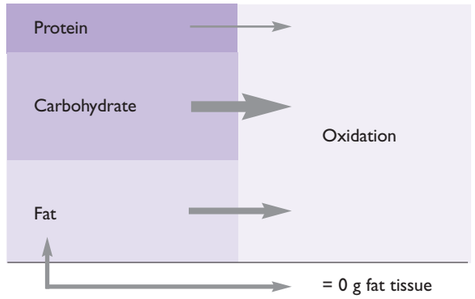
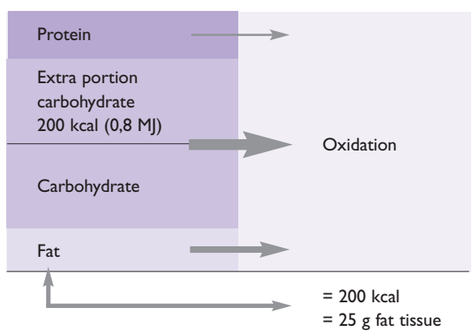


Figure 2

When you eat too much

Energy intake > energy consumption



Sugar in beverages

In 2001, an American population study was published which found that children who drank sugar-sweetened soft beverages had an increased risk of becoming obese[8]. However, no link could be found between the consumption of artificially sweetened soft beverages and the risk of obesity. More studies of a similar nature have been carried out since.

In a Danish study, a group of overweight subjects were divided up to receive a supplement of either sugar-sweetened or artificially sweetened beverages over a 10-week period[9]. The subjects were also asked to continue their usual diets and eat until they were sated. The trial was blinded so that the subjects did not know whether they were being given beverages that were sweetened with sugar or with artificial sweeteners. After 10 weeks, it emerged that the group that had been given artificial sweeteners had not consumed fewer calories and did not have any statistically certain loss of fat (0.5 kg). By contrast, the group that had been given the sugar-rich products had put on 1.3 kg of fat and increased their blood pressure. The study confirms that sugar in solutions can increase energy intake and thus create a positive energy balance. But we do not know why, just as we do not know if it plays any role whether the consumption takes place between or at mealtimes or whether or not the beverages are carbonated. The study also shows that the use of artificial sweeteners does not in itself cause weight loss, but we cannot draw any conclusions as to whether their use can clearly increase weight. From a biological viewpoint, human beings are not intended to consume beverages containing energy. On nature's part, water has been the natural drink, and it is only very recently in the history of human development that fruit juice and fermented beverages have entered into the equation. We might therefore imagine that sugar in solution would have a different effect on the gastrointestinal tract's appetite-regulating hormones (ghrelin, GLP-1, PPY, etc.) than sugar in solid food.

The importance of sugar for energy density

WHO Report 916 cites 'high intake of energy-dense micronutrient-poor foods'[10] as one of the causes of obesity. Further to the discussion on the importance of beverages for the development of obesity, it is therefore also relevant to assess the importance of sugar for the energy density of food and beverages. The assessment depends, however, on the energy density of the nutrient in the place of which the sugar is added. If sugar replaces starch (or other carbohydrates), the energy density is affected depending on how much water the starch binds in the given food. If fat is replaced with sugar, the energy density will be reduced, while the energy density will be increased if sugar is added instead of water. If sugar is replaced by sweeteners, the energy density can be either increased or reduced. Sweeteners have a greater sweetening ability per gram. The decisive factor for energy density is therefore which nutrient makes up the missing weight.

What do the authorities conclude?

The above conclusions are in accordance with those of other authorities such as the Danish Nutrition Council, which in its report entitled 'The impact of sugar on health'[11] concludes that "*Intervention studies in which a high intake of sugar is compared with a normal western diet have not shown any convincing difference in weight changes for the two study groups*" and that "*Sugar in beverages does not give the same degree of satiety, and a large intake of sugar-sweetened beverages can lead to a positive energy balance and weight gain in the long term*".

The American nutrition guidelines of 2002[12] conclude that the scientific literature cannot prove whether there is a causal link between increased sugar intake and the incidence of obesity: "*There is no clear and consistent association between increased intake of added sugars and BMI*". On the other hand, the new American dietary guidelines[13] suggest cutting back on sugar: "*Individuals who consume food or beverages high in added sugars tend to consume more calories than those who consume food or beverages low in added sugars; they also tend to consume lower amounts of micronutrients. Although more research is needed, available prospective studies show a positive association between the consumption of calorically sweetened beverages and weight gain. For this reason, decreased intake of such foods, especially beverages with caloric sweeteners, is recommended to reduce calorie intake and help achieve recommended nutrient intakes and weight control.*" The WHO/FAO report of 2002[10] on diet, nutrition and prevention of chronic diseases includes a chapter on diet and obesity which concludes that the results on the link between sugar in foods and obesity are not clear and do not permit any conclusion: "*Overall, the mixed results, especially amongst the few available trials, does not allow a judgment to be made about the sugar content of food and obesity*". On the other hand, it concludes that the documentation that sugar-sweetened soft beverages increase the risk of weight gain is clear and reasonably strong, and that it is especially relevant for populations with a high consumption: "*Overall, the evidence implicating a high intake of sugars-sweetened drinks in promoting weight gain was considered moderately strong*".

There is thus widespread agreement on the conclusions that can be drawn from the existing research on the importance of sugar for overweight. More long-term studies that can more fully reveal the importance are lacking, but, all the same, the studies carried out so far point unanimously to the fact that sugar in solutions sates poorly and contributes to a positive energy balance and weight gain.

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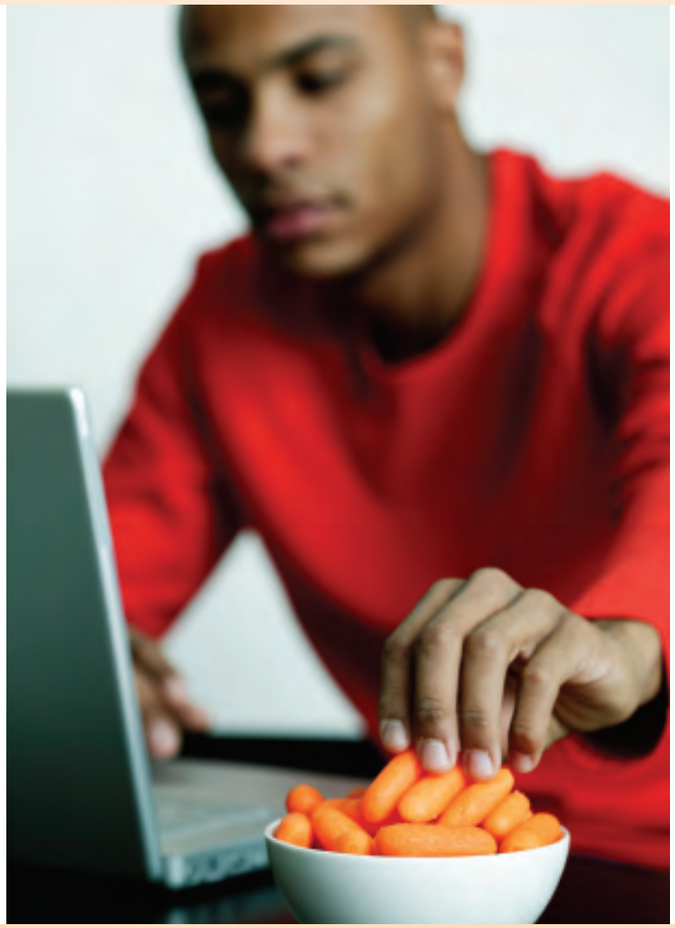
Sugar and diabetes



By Matti Uusitupa, MD, Professor, Department of Clinical Nutrition and Food and Health Research Center, University of Kuopio, Finland.

Sugar has been permitted in diabetic diets since the early 1980s. The latest European recommendations allow a daily intake of 50 grams of sugar (10% of total energy), preferably as part of mixed meals. Sugar-sweetened soft drinks and similar snacks, especially between meals, can cause unnecessarily high fluctuations in postprandial glucose levels, especially if glycemic control of a patient is poor. Fructose can be used as an alternative sweetener in diabetic diets because it has a low glycemic index. The recommended upper limit is 30 g/day because high doses may induce elevated serum triglycerides. Some observational studies link added sugar with the development of obesity and type 2 diabetes, but further controlled studies are needed to clarify the issue. Diabetic patients benefit from diets high in dietary fibre and low in high glycemic index foods. It is therefore recommended that people with diabetes follow the carbohydrate recommendations and consume large amounts of wholegrain food products, fruit and vegetables, regardless of diabetes type and body mass index (BMI).

The role of sugar in diabetic diets has significantly changed in the past few decades. As late as the start of the 1970s, the typical diet recommended for diabetic patients was relatively low in carbohydrates and added sugar. In clinical practice, the first line of advice for new diabetic patients was to avoid added sugar[1]. Since then, the diabetic diet has become more restrictive in terms of dietary fat and the attitude to the use of added sugar has also changed. This is due to the results of several controlled dietary studies carried out in the late 1970s and early 1980s. These showed that the addition to a diabetic diet of moderate amounts of sugar rather than comparable amounts of starch did not worsen either acute or long-term glycemic control and did not affect lipid values, provided that the metabolic control of diabetes was acceptable. In the late 1970s and early 1980s, interest in the effect of dietary fibres and glycemic index also became popular in nutritional research, which influenced the recommendations for diabetic diets. Recently, the epidemic in obesity and the derived increase in type 2 diabetes have revived interest in the role of sugar in the development of overweight, metabolic syndrome and type 2 diabetes.

Diabetic diet

There is no single diabetic diet because most diabetic patients are able to follow the dietary patterns recommended as healthy for the general population. In the 1980s, several randomised controlled studies with cross-over design showed no adverse effects on glycemic control or serum lipid values when diets containing small or moderate amounts of sucrose were compared to diets free of sucrose but with an equivalent amount of starch[2,3]. However, in some studies a high-sucrose diet has resulted in hypertriglyceridaemia in persons with metabolic syndrome[3,4]. Any precise recommendation for the upper limit for sugar content in diabetic diets is somewhat arbitrary since no comprehensive dose response studies are available.

European and American recommendations

There is some discrepancy between the American and European recommendations in respect of the content of sugar and fructose in diabetic diets, and the latest recommendations from the American Diabetes Association (ADA) do not give any precise quantitative upper limit for sugar content[2]. The ADA's recommendations stress that the total amount of carbohydrate in meals and snacks is more important than the type (carbohydrate source) and form. It is also recommended that terms such as 'sugars', 'starch' and 'fibre' should be used. In 2004, the ADA had not begun to use the term 'glycemic index'. Common to both the American and European recommendations is that whole grains, fruits and vegetables are preferred sources of carbohydrate. In the European recommendations, sugar intake has been restricted to 50 g/day or maximum 10% total energy. The European recommendations also use the term 'glycemic index' and recommend an ideal dietary fibre intake of 40 g/day[3], which is higher than in the Nordic Nutrition Recommendations, but which may give better blood sugar control.

Fructose and diabetes

It is well known that fructose has a low glycemic index, which means that it can be more suitable than sucrose for diabetic patients[4]. However, the use of added fructose is not recommended by the ADA because of the risk of an increase in the serum triglyceride content (hypertriglyceridaemic effect)[2,5]. In contrast, the European recommendations allow moderate amounts of fructose (up to 30 g/day) since this amount does not have any harmful effect on glucose, insulin or lipid metabolism[3].

General recommendation

To summarise, the use of sugar in diabetic diets is more liberal than previously and follows the recommendations for healthy people[6]. The carbohydrate composition, including added sugar, in meals should be planned according to other diabetes treatment. The aim is to prevent a significantly increased blood glucose level after meals. Insulin treatment and other medicine should therefore be individually tailored according to the carbohydrate content of a meal. The European recommendations also emphasise that a diet with a high content of dietary fibre and foods with a low glycemic index can contribute to controlling blood glucose fluctuations in the short term and to metabolic control in the long term.

Risk of type 1 diabetes

Several environmental and nutritional factors are associated with the development of type 1 diabetes, but firm evidence is still lacking regarding the significance of nutritional factors in the cause and development of type 1 diabetes. Increased linear growth in children and obesity may possibly increase the risk[7], and any nutritional factor that is involved in the increasing incidence of obesity in children and young people should therefore be taken seriously in this regard. It should also be noted that the increasing incidence of obesity in young people results in an increased incidence of type 2 diabetes even before adulthood is reached.

Risk of type 2 diabetes

The main risk factors for type 2 diabetes are obesity, in particular central obesity, sedentary lifestyle, diets rich in fat and saturated fatty acids, diets low in dietary fibre and diets with an abundance of high glycemic index foods[2,3]. Furthermore, three large-scale lifestyle intervention trials have shown that it is possible to substantially reduce the incidence of diabetes by changing lifestyle, i.e. through weight loss, increased physical activity and dietary change in line with the current recommendations[8,9,10]. In the Finnish Diabetes Prevention Study[9], a low energy density diet, rich in dietary fibre and the quality of fat may have contributed to the success of intervention, but it is almost impossible to analyse the independent effect of different nutrients and other dietary components (Lindström J et al, unpublished results).

Glycemic index and diabetes

Since many studies[3,11] have linked high glycemic index foods to the risk of diabetes, and since, on the other hand, low glycemic index foods, wholegrain food products and diets rich in dietary fibre have shown a protective effect in this regard, it is reasonable to assume that diets with a high sugar content may also be linked to the risk of diabetes, either directly through several mechanisms or indirectly, for example through the increased risk of obesity presumed to be caused by, among other things, high sucrose intake[12].

Conclusion

To summarise, high sugar consumption may induce obesity and, through this, may be linked to the risk of type 2 diabetes. Furthermore, because high sugar consumption can be a typical feature of fast-food habits and/or a high glycemic index, it also seems reasonable to restrict sugar intake in line with the current recommendations[6], even though the recommendation for the upper limit for sugar may be arbitrary. The recommendation for increased use of wholegrain food products, fruit and vegetables should be promoted, not only to prevent obesity and type 2 diabetes, but also to prevent other chronic diseases, i.e. cardiovascular diseases and some types of cancer.

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Sugar and cardiovascular disease

By Lars Ovesen, MD, Health Executive, Danish Heart Association, Copenhagen, Denmark.

Sugar intake has been studied for its effect on a number of risk markers for the development of coronary heart disease. High sugar intake increases the blood's triglyceride level. The effect varies and is greatest in overweight and insulin-resistant individuals respectively. High sugar intake also reduces HDL cholesterol, but total cholesterol and LDL cholesterol are not affected beyond the effects seen for complex carbohydrates. It is not certain whether sugar plays a role in the development of insulin resistance, and very few studies have investigated the effect on factors in the blood that are of importance for thrombosis. Regardless of the effect on individual risk markers, the few available epidemiological studies have not shown higher risk of coronary heart disease in the population groups with the highest sugar intake.

The Nordic Nutrition Recommendations prescribe that the population's energy percentage (E%) from fat should be reduced to 30, primarily by reducing the intake of saturated fat and increasing the intake of complex carbohydrates correspondingly. Replacing fat with carbohydrates would mean that total cholesterol and LDL cholesterol would be reduced, but would also increase triglyceride and reduce HDL cholesterol in the blood, both of which are independent risk factors for coronary heart disease. However, sugar differs from complex carbohydrates due to its content of fructose and may therefore be expected to have different metabolic effects to complex carbohydrates (which consist only of glucose). The following provides an update on the role of sugar in the risk of coronary heart disease, making reference to summaries published in recent years[1,2].

Sugar increases triglyceride and reduces HDL cholesterol

High dietary sugar content (in most studies the intake was higher than 20 E%) increases triglyceride during fasting and after consumption of a meal and reduces HDL cholesterol. Fructose is especially effective at stimulating fatty acid synthesis in the liver. Sugar therefore has a greater triglyceride-increasing effect than intake of an equivalent amount of complex carbohydrates. However, several studies suggest that the total intake of carbohydrates (in the form of complex fibre-rich carbohydrates) can be increased to the recommended level (50-60 E%) without the triglyceride level increasing or HDL cholesterol falling if the dietary sugar content is around the recommended maximum of 10 E%[1].

Nevertheless, it should be noted that the hypertriglyceridaemic effect of sugar varies significantly and is greater in overweight individuals than in normal-weight individuals and in individuals with insulin resistance and elevated fasting triglyceride. The effect is also supposedly modified by a large number of other factors, including dietary composition, e.g. its fat quality and fibre content, physical activity and genetic factors[3]. Sugar does not affect total cholesterol or LDL cholesterol differently to complex carbohydrates.

Insulin resistance increases the risk of heart disease

Elevated triglyceride is rarely seen alone, but typically as a component of what is known as the insulin resistance syndrome (the metabolic syndrome), which is also accompanied by reduced HDL cholesterol, reduced fibrinolysis, increased coagulation, dysfunction of the vascular endothelium, elevated blood pressure and abdominal fat distribution, all of which increase the risk of coronary heart disease.

Whether sugar plays a special role in the development of insulin resistance is uncertain. The few studies that have been carried out have not shown a higher risk of insulin resistance for high intake of sugar compared to other carbohydrates or fat[4,5]. A single study has reported that high intake of sugar-sweetened beverages is accompanied by increased incidence of the factors that are involved in the metabolic syndrome, including in normal-weight individuals[6].

Uncertain whether haemostasis is affected

Very few studies have investigated whether a sugar-rich diet is important for the risk of thrombosis. A Danish study found that a marker for coagulation (factor VII) was increased following the consumption of a sugar-rich diet compared to a diet with a high content of complex carbohydrates[7]. However, it is uncertain whether the factor VII level is a risk marker for coronary heart disease. On the other hand, a more reliable marker, the blood's content of fibrinogen, is not altered following the consumption of a sugar-rich diet.

Dietary glycemic load

No link has been found between sugar intake and the risk of coronary heart disease. By contrast, dietary glycemic load (the amount of carbohydrate-rich foods in the diet multiplied by their glycemic index) is a good predictor of the risk of coronary heart disease. The American Nurses Health Study showed that women who consumed a diet with the highest glycemic load had higher fasting triglyceride, lower HDL and twice the risk of coronary heart disease[8] compared to women who consumed a diet with the lowest glycemic load. The total intake of complex and simple carbohydrates and sugar was not associated with any risk.

Conclusion

A high sugar intake enhances the hypertriglyceridaemic effect of a recommended diet with a low-fat/high-carbohydrate content. Elevated triglyceride can increase the risk of coronary heart disease. A high sugar intake will also reduce HDL cholesterol. However, epidemiological studies have not shown a higher risk of heart disease in the population groups with the highest sugar intake.

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Food, eating habits and dental health

By Peter Lingström, DDS, PhD, Institute of Odontology, Göteborg University, Sweden.

There are many seemingly minor factors that can play a major role in determining the development of dental caries in individuals. Although fluoride has raised the threshold value at which caries develops, it is important to provide dietary advice to individuals at elevated risk of developing caries. This advice should include reduced intake frequency, lower total sugar intake, drinking water when thirsty between meals and at night, and using products sweetened with sugar substitutes. Another simple recommendation is to take a sugar-free chewing gum or lozenge immediately after meals if tooth-brushing is not possible. Individuals prone to caries should also take fluoride supplements and observe good oral hygiene. Today, it is less important what products we consume, and more important to observe suitable individual consumption patterns.

Consumption of food and drink is complex and varies greatly between individuals. Similarly, the effects of food intake on dental health show a large variation. Despite the increased range of sugary products available in the western world in recent decades, children's and young people's general dental health has improved dramatically during the same period. See Table 1. The average number of decayed and filled teeth (DFT) among 12 year-olds in Sweden was 1.1 in 2002, compared to 3.1 in 1985. Dental health has also improved among older age groups; for instance, elderly people keep more of their own teeth today. This improvement in dental health is ascribable to increased use of fluoride, primarily through regular use of fluoride toothpaste, and knowledge regarding good oral hygiene.

Nevertheless, many people still have significant problems with caries. In these individuals, links are frequently found with various caries-associated biological and social factors such as reduced saliva secretion, diseases or work situation. Research in recent years has also highlighted genetic differences that make certain people more prone than others to developing caries.

Over the years, researchers have tried to define the precise impact of diet on caries development. There is a general consensus today that the consumption of fermentable carbohydrates has been a key aetiological factor behind caries ever since prehistoric times. However, it is difficult to specify this factor's precise role in modern society in epidemiological and clinical data, and to determine a clear link between sugar intake and caries at population level. This is partly due to the large variations that occur in sugar intake. Our diet has become more complex. The consumption of pure sugar is progressively decreasing, while consumption of sugary drinks and foods is on the rise. Furthermore, the relationship between diet and caries has changed as a result of an increased use of fluoride.

Acid production – a central factor in caries development

Food in itself does not pose a risk to tooth substance. The risk of caries arises when food becomes one of the basic elements in a triad where the other two elements are the host organism – the tooth itself – and caries-inducing bacteria. The core of the caries process is the bacterial degradation of fermentable carbohydrates in the dental plaque. For many years, interest focused mainly on so-called mutans streptococci and lactobacilli, but today it is well-known that many other bacteria in the oral cavity are involved in this process. All these bacteria have the capacity to grow and act in an acid environment. The fermentable carbohydrates serve as an energy source for these bacteria. During consumption, they rapidly diffuse into the plaque and from there into the bacteria cell, where degradation occurs. Besides energy transformation, this metabolism also produces various organic acids as waste products, primarily lactic, acetic and propionic acid.

Acid production leads to the release of hydrogen ions, which alter the plaque's acidity level. When the pH level drops, the solubility of calcium and phosphate in the plaque fluid and saliva increases, thus disrupting the balance between the tooth and the surrounding liquid phase[1]. When the pH falls below the respective critical levels for enamel (pH 5.5-5.7) and dentine (pH 6.0-6.2), risk for damage of the enamel and dentine increases as demineralisation of the tooth surface occurs. The maximum pH reduction normally occurs 5-10 minutes after intake. After this, the saliva's cleansing and buffering system eliminates and neutralises the acids, and the pH level usually returns to normal within 60 minutes. There is strong individual variation in this process, which is primarily determined by the quantity and composition of the saliva[2]. If saliva production is low, and at night when the body is at rest, it takes considerably longer for the balance to be restored. The quantity and composition of the plaque also affects this process. This may explain why individuals with poor oral hygiene are at greater risk of developing caries in connection with a high sugar intake[3].

Different carbohydrates – different caries risks

Of the carbohydrates consumed, the oral bacteria are capable of metabolising the mono- and disaccharides fructose, glucose, lactose, sucrose and maltose, as well as the polysaccharide starch. However, sucrose plays a special role in the caries process[4]. Besides generating strong acid production, sucrose also acts as a substrate for so-called soluble and insoluble polysaccharides, which primarily favour plaque formation. Glucose and fructose cannot be utilised in this way, and thus have a lower plaque-inducing capacity. Lactose, present in milk and dairy products, is considered to have lower cariogenic potential. This is partly due to these products' content of calcium, phosphate and proteins, which have protective properties. However, if frequently subjected to lactose, the bacteria can learn to utilise it more efficiently, resulting in slightly increased acid production (adaptation)[5]. The cariogenic potential of starch has been found to vary greatly. This is primarily related to its botanical origin, and to the way the product is processed before consumption; a more highly processed product degrades

faster[6]. Studies have found a combination of sugar and starch to be more cariogenic than pure sucrose[7]. The starch's sticky quality causes the sugar to remain in the mouth for longer.

The role of intake frequency

The exact role a food plays in the caries process varies according to several food-related factors such as the quantity or concentration consumed, content of protective substances (e.g. proteins, calcium, phosphate, fluoride) and physical and chemical properties (liquid or solid, stickiness, solubility, buffering capacity). While some studies have attempted to rank foods from low to high risk in relation to caries, the most important factors for caries development lie with the individual. Intake frequency is the prime factor in this context, and can entirely determine whether or not caries develops. A high intake frequency means longer periods of demineralisation and only short periods when teeth have a chance to remineralise. Other key individual-related factors are consumption patterns, eating before going to sleep, or eating at night without subsequent toothbrushing. The individual's ability to eliminate food or drink from the oral cavity is another key factor, which is primarily related to saliva quantity[7].

A long-term increase in sugar consumption also alters the bacterial flora, which gradually causes an ecological shift towards more acid-resistant bacteria[8]. This results in higher overall acid production and increased risk of caries. Saliva is a major factor; reduced saliva secretion significantly prolongs the time it takes for food to be eliminated from the oral cavity. This is also what happens at night during sleep. Furthermore, low saliva production reduces the protection provided by the saliva's various neutralising systems and its antibacterial substances[9].

Dental erosion

The consumption of acid in solid foods and drinks means not only a risk of caries, but also of dental erosion[10]. Erosion is caused by the chemical effect of acid substances without bacterial involvement. A strong contributing factor in this context is the intake of acidic drinks. A dramatic increase in dental erosion has been observed in recent years, not least in children and teenagers. This is primarily ascribable to an increased consumption of acidic drinks. From this aspect, there is no difference between sugary and sugar-free drinks (so-called light products). As with caries, various food-related factors (for instance content of buffering substances) and individual-related factors may impact on the risk of dental erosion. Again, frequency and consumption patterns are prime individual-related factors. Individuals who keep the drink in their mouths for a long time are at greater risk than those who swallow it quickly[11]. In addition to diet, acid reflux from the stomach, frequent vomiting or an alkaline environment are other contributing factors.

New consumption patterns in modern society

Modern society has generated new consumption patterns where frequent intake of beverages during sedentary computer work is not unusual. Today, numerous energy drinks are available to athletes in connection with physical activity. Fast food and various disposable packagings, such as cups with re-sealable lids, now allow food and drink to be consumed in many different environments. Moreover, many people habitually drink soft drinks and other beverages while sitting at the computer. These consumption patterns can all contribute to a higher intake frequency. Furthermore, as a complement to traditional medical treatment, more and more people are being offered dietary advice which often recommends eating little and often. For people from other cultural backgrounds, moving to the Nordic countries may mean greater access to sugary products and difficulty in identifying healthy products with low sugar content on the market.

Table 1

	Percentage caries-free 6-years old		Percentage caries-free 5-years old	
	Finland	Sweden	Denmark	Norway
1985	40	45		
1987				56
1988	45			63
1990		60	63	
1991	52			
1992				63
1994	58			
1995		65	67	65
1996			67	68
1997	56		69	70
1998		72		69
1999			71	
2000	58	70	70	
2001		70	71	60
2002		69	70	
2003			71	64
2004			72	

	Percentage caries-free 12-years old			
	Finland	Sweden	Denmark	Norway
1985	15	22		19
1987				25
1988			33	
1990		40	46	
1991	30			
1992				36
1994	35			
1995		50	50	40
1996				43
1997	35		54	45
1998		62	55	46
1999			57	
2000	38		58	48
2001		61	60	46
2002		57	61	
2003			60	42
2004	42		60	

Sweden: Tandhälsan hos barn och ungdomar 1985-2002, Socialstyrelsen, Stockholm, juni 2003. Denmark: Sundhedsstyrelsens Centrale Odontologiske Register (SCOR) Norway: Statens helsetilsyn, Tannhelsetjenesten i Norge. Årsmeldinger 1994-2001. Oslo: Statens helsetilsyn; 1997-2003. Finland: Eeva Widström and Anne Hiiri, Themes from Finland, Oral Health care in Finland, Themes 111998, National Research and development centre for welfare and health (STAKES). Ministry of Social Affairs and Health.

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The role of sugar in physical activity and exercise

By Mikael Fogelholm, Sc.D., Director, the UKK Institute for Health Promotion Research, Tampere, Finland.

Carbohydrate is the preferred energy source for muscles and makes up 60-70% of total energy expenditure during activity of moderate or high intensity. Limited amounts of carbohydrate are stored as glycogen in the muscles and liver. These glycogen stores are therefore of vital importance for performance. Consequently, it is important to optimise the stores before, during and after physical activity – especially for long-duration physical activity of moderate or high intensity. The type of carbohydrates does not appear to be of major significance when it comes to loading the glycogen stores. However, if there is a need for rapid glycogen synthesis, it can be beneficial to choose high-glycaemic carbohydrates, immediately after physical activity, because this is when glycogen synthesis is at its highest level. Regardless of whether you are a jogger or an elite athlete, it is advisable to follow the general recommendations of maximum 10% energy from sugar. Nevertheless, during and immediately after days of extreme exertion (e.g. marathon competition) the intake of sugar may be higher.

During physical movement, work is done by voluntary contractions of skeletal muscles and energy expenditure increases above the resting level. Physical activity is considered 'light' when energy expenditure is 2-3 times the resting level. In 'moderate' activity, energy expenditure is 3-6 times and in 'intensive' activity more than 6 times the resting level. Elite athletes can use up to 20 times more energy in one hour than they would use while sleeping for the same period of time.

Musclar work always increases the body's total energy expenditure, but the muscles' choice of energy source depends on the intensity level[1]. Figure 1 illustrates the relative contribution of fat and carbohydrates in relation to work intensity. As the intensity increases, from light to above 30-40% of the body's maximum oxygen intake (VO_2max), i.e. a shift from light to moderate, the muscles start to use more intramuscular substrates (both fat and carbohydrates).

In particular, the use of muscle glycogen increases. When the intensity reaches 65-75% VO_2max, i.e. a shift to an intensive activity level, muscle glycogen becomes the primary energy source. This is presumably due to the fact that the burning of fat alone cannot meet the total energy requirement for intensive activity and that muscle glycogen is needed when activity level is intense[2].

In addition to fat and carbohydrates, the body can also use protein as an energy source[3]. In normal circumstances, the share of total energy expenditure from protein is less than 5%. But if muscle glycogen stores are empty, or if protein intake is much higher than the daily protein requirement, the contribution of protein may be much higher.

Important to ingest carbohydrates

Although the muscles' choice of energy source clearly depends on the intensity of physical activity, many other factors are also important. One such factor is the duration of the physical activity, particularly when the intensity is 40-80% VO_2max. At this intensity, the muscles eventually drain their own glycogen stores. After prolonged physical activity, the muscles begin to shift from a predominant use of carbohydrates (glycogen) to the predominant use of fat. Carbohydrates thus make up 60-70% of total energy expenditure at the beginning, but the proportion of carbohydrates decreases to 30-40% after 2-3 hours of physical activity. As the physical activity continues, the use of both muscle glycogen and fat (intramuscular triglycerides) decreases[1].

Glycogen stores are of major importance

The size of the body's glycogen stores is limited: the liver normally contains 100 g and muscle tissue about 400 g of glycogen[4]. As early as the 1960s it was shown that muscle glycogen depletion is the main cause of fatigue during aerobic, intensive and long-duration physical activity[5]. But although glycogen depletion plays an obvious role in fatigue, it is highly likely that other factors also contribute. For example, a concomitant rise in the brain serotonin level has been suggested as a cause of "central-nervous fatigue". Fatigue during anaerobic exercise (e.g. strength training) has origins other than glycogen depletion, and these kinds of sport are not dealt with in this article.

Glycogen depletion during endurance events is sometimes experienced as 'hitting the wall' – a sudden, complete feeling of total exhaustion. Normally, however, fatigue creeps in little by little. Physical activity is still possible with small glycogen stores, but the intensity must be reduced. Moreover, the subjective feeling of strain is exceptionally strong during physical activity with low glycogen stores. Besides endurance events, many ball games are also affected by muscle glycogen stores. It has been shown that the muscle glycogen stores are reduced during both football (soccer) and ice hockey. Both running speed and distance covered during these sports are reduced if muscle glycogen is low.

Optimisation of muscle glycogen

Muscle glycogen can be increased in the days preceding demanding physical activity[5]. Studies have shown that the consumption of about 450-500 g carbohydrates are needed per day to increase muscle glycogen to above normal for 4-5 days[4,6]. If less time is available to fill the stores, the daily carbohydrate intake should increase to about 8-10 g/kg body weight or 600-700 g/day, which is probably the upper useful limit. Elite athletes with a high energy intake are able to reach these levels by generous use of bread, other cereal products, pasta and sugar-containing foods and drinks. But it can be more difficult for recreational athletes, who could benefit from a starch-rich diet with extra sugar in the 3-4 days preceding intensive endurance activities such as a marathon.
The composition of the carbohydrates ingested does not seem to influence glycogen loading to a significant extent. Previously it was believed that foods with a low glycaemic index were better at increasing glycogen stores than foods that stimulated a higher blood sugar level, but this has not been confirmed[7]. As a rule of thumb, however, it can be said that the proportion of added sugar does not need to exceed 10% of the total

energy intake during normal training. The Nordic Nutrition Recommendations can therefore be followed.

Timing of meals

A carbohydrate-rich meal 3-4 hours before an activity can give the body additional carbohydrates. This can probably improve performance if the contribution of the glycogen stores during the activity would be insufficient. Many elite athletes do not consume foods with a high glycaemic index 1-2 hours before the activity. This may be due to the fact that blood glucose and insulin levels start high and fall rapidly at the beginning of an activity and some individuals can feel unwell and tired. However, studies have not been able to demonstrate that sugar intake immediately before an activity impairs performance.

During the activity

Intake of carbohydrates in connection with long-term, high-intensity exercise can significantly improve performance. This has been proved by a number of different studies in the last 30 years[8]. The body is able to absorb and use about 1 g carbohydrate per kg body weight per hour. Joggers who run the marathon should ingest at least 30-40 g carbohydrate per hour, while elite athletes should ideally ingest 60-70 g carbohydrates per hour[9].
Carbohydrate supply depends on two factors: the speed of gastric emptying and absorption from the intestine. The most important factor for speed of gastric emptying is the drink's energy density (carbohydrate g/100 g); the higher the energy density, the slower the emptying. In pure carbohydrate energy drinks, the energy density gets higher the more grams of carbohydrate are added per 100 g water. An increased concentration of the number of carbohydrate molecules (increased osmolality) will also reduce the speed of gastric emptying. If the carbohydrate concentration is high (more than 7-8%), it may therefore be beneficial for absorption speed to substitute part of the free glucose with maltodextrin.
Fructose is absorbed slower than glucose and the muscles cannot use fructose until it has been converted to glucose in the liver. Thus, if fructose is used in sports drinks, its concentration should be kept below 2%.
It is impossible to define one optimal sports drink for all situations. In very hot climates, where the fluid intake should be maximized, the carbohydrate concentration should be kept below 5%, even as low as 2-3%. Diluted drinks are also recommended for athletes, who easily get gastro-intestinal problems if gastric emptying is not fast enough. In more temperate climates, particularly in winter, a high intake of fluid is not as important and the carbohydrate concentration can be increased to 5-8%, or even as high as 10%. In addition to carbohydrate drinks, athletes can also ingest solid or semi-solid glucose or sugar products and drink pure water. This is common practice during ultra-long activity.

Recovery

Replenishment of the glycogen stores is one of the key components in recovery from competitions and from daily training. In some cases, it is necessary to recover within a few hours (e.g. between two daily training sessions) or within 24 hours (e.g. during the Tour de France or the ice hockey World Championships). Studies have shown that timing, the amount and type of feeding influence the replenishment of muscle glycogen stores.
The muscle cells' insulin sensitivity and the activity of the glycogen-storing enzyme, glycogen synthetase, are increased immediately after training. This physiological state is suitable for rapid synthesis of glycogen from blood glucose. The speed of glycogen synthesis is therefore maximal in the first 2 hours after long-duration training[10]. If rapid recovery is needed, this phase of rapid synthesis should be utilised by starting carbohydrate feeding as soon as possible after training. The speed of glycogen synthesis depends on the amount of carbohydrate intake. A plateau seems to be reached when taking in carbohydrate at the rate of 0.5 to 0.75 g/kg every hour. The rate of glycogen synthesis is not dependent on whether the carbohydrate is taken in less frequent, larger portions (e.g. 1 g/kg every 2 hours) or in more frequent, smaller portions (e.g. 0.25 g/kg every 30 minutes)[11].

The affect of type of carbohydrate is quite clear: carbohydrates with a high glycaemic index (glucose, sucrose, starch with high amylopectin content) are stored as glycogen much faster than carbohydrates with a low glycaemic index (fructose or starch with high amylose content)[7]. An interesting question is whether glycogen storage can be enhanced by adding protein or certain amino acids to carbohydrate drinks. Despite some positive findings, no clear results have been produced leading to a final conclusion. Among other things, this is because different proteins and amino acids have been used in the various studies. Further studies are therefore required.

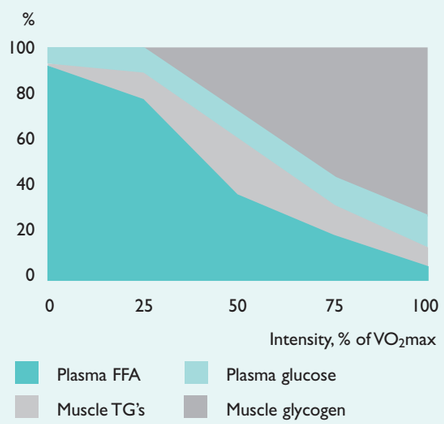
Table 1

Relationship between intensity level and energy consumption for physical activity

Intensity level	Energy consumption
Light	2-3 times higher than for inactivity
Moderate	3-6 times higher than for inactivity
Intensive	more than 6 times higher than for inactivity

Figure 1

The proportional whole body substrate oxidation rates when exercising with different intensities[1]



TG = triglycerides, FFA = free fatty acids

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The glycemic index in practice

By Mette Axelsen, PhD, Senior University Lecturer, Department of Metabolism and Cardiovascular Research, Sahlgrenska Academy, Göteborg University, Sweden.

The GI concept focuses on the quality of carbohydrates and has given us increased awareness of the role that the quality of carbohydrates plays in our health. However, there is uncertainty as to how GI can best be integrated into health messages. What happens when people change their diet? Replacing sugar with fat may lead to a worse diet in contrast to replacing sugar with whole grain products. GI can and should primarily be discussed within the scope of existing dietary recommendations. Comparisons should only be made within each individual food group. The GI method is under constant evaluation. More clinical and methodological studies are needed to give us a clearer picture of how the GI concept should be integrated into health messages in the future.

The Glycemic index (GI) is a concept that has attracted great interest. Several hundred scientific studies have been carried out, and a large number of popular scientific books have been written. GI is based on a new method that was launched in 1981[1]. It is at an embryonic stage with regard to both science and practical use. There is a real need to explain where GI stands, and where science and practice are headed.

Ranking of foods

The Glycemic index is a measurement of carbohydrate quality, and shows how quickly blood glucose rises after the intake of 50 g of available carbohydrate from carbohydrate-rich foods. As there is no single chemical property that determines how quickly blood sugar rises, GI is determined in vivo, i.e. using people as test tubes[2]. The speed of the rise in blood glucose is reflected in the area under the blood glucose concentration response curve for the first two hours after ingesting a test food.

The test individuals' glucose tolerance is also taken into consideration by dividing the individual's blood glucose level after ingesting the test food by the same person's blood glucose level after the ingestion of a glucose solution or white bread. Several internal and external factors have been identified as capable of modifying GI. See Table 1.

Effect on health risks

The effect of GI, i.e. how quickly blood glucose rises, on blood lipid disorders and glycemic control has been studied in clinical intervention studies. In a meta-analysis of these studies, it was concluded that total cholesterol was decreased through a low-GI diet, and that even LDL cholesterol tended to improve in people with Type 2 Diabetes[3].

However, a systematic overview article found that proof of these benefits is still inconclusive and longer studies are needed[4]. Similarly, experimental studies have not yet been able to provide evidence of weight reduction[5, 6]. On the other hand, a low-GI diet is often included in recommendations for diabetes treatment. A number of studies suggest that blood sugar control improves when carbohydrate foods with a low GI replace those with a high GI[3, 4, 7].

The importance of GI to future disease risk in initially healthy people has been researched in prospective epidemiological studies. In many, but not all studies, a self-selected diet with a low GI is associated with decreased risk of cardiovascular diseases[8, 9], Type 2 Diabetes[10, 11, 12], and certain types of cancer[13, 14, 15, 16]. The explanation for these findings is thought to lie in improved risk factor pattern, which has been found in the clinical studies. In addition, there are epidemiological connections between a low-GI diet and a high HDL[17], and between a low-GI diet and a low CRP level, representing decreased low-grade inflammation[18]. What cannot yet be dismissed, however, is the possibility that the protective effects linked to a low-GI diet may be caused by other beneficial factors than GI, such as whole grains, fibre, reduced energy density, and other protective dietary or lifestyle factors[19].

In many of the intervention studies, there were small differences in GI and/or short observation times. A limiting factor in the design of long term GI studies is that few or no low-GI varieties within the same food group are available for purchase. A good understanding of how to make use of GI in practice, and solid efforts to increase the range of low-GI foods available, are two challenges to be addressed in order to gain increased knowledge about GI and health in the future.

The GI concept is often confused with reduced total carbohydrate. If the carbohydrate intake is reduced, it is no longer a matter of the GI concept but a new concept known as 'low glycemic load' (amount of carbohydrate x GI/100). If carbohydrates are replaced with fat or protein, the advantages of a low-GI diet cannot be guaranteed. If a full dose of antibiotics kills an infection, this does not necessarily mean that a half dose will. Similarly, it cannot be concluded that the favourable effects of low-GI food will still be evident if combined with a low carbohydrate diet. Certain effects may be increased, but others may disappear.

Practical applications

The GI concept is important for understanding how the quality of carbohydrates affects our health. GI is also designed to facilitate the implementation of a low-GI diet by ranking various foods, from slow to fast options, within the framework of a high-carbohydrate diet. GI has been portrayed in the popular press as a general factor when prioritising between foods. In actual fact, no single nutritional aspect constitutes such a general health factor that it takes priority over all other aspects, and this also applies to GI. It goes against basic scientific principles to focus entirely on the quality of carbohydrate if the food/meal has an increased percentage of fat or otherwise is of a poor nutritional value.

The most important advice for avoiding incorrect use of GI:

- A comparison of GI values should only be made between foods in the same food group. For instance, bread should be compared with bread, cereals with cereals, rice with rice, etc.
- The GI values should only be used for foods containing 15-20 g of available carbohydrates per portion. This rules out less relevant sources of carbohydrates, such as vegetables, fruits, nuts and certain dairy products.

One argument voiced in discussions is that it is impossible to consistently follow a low-GI diet in practice, because there are too few low-GI options available. The selection in supermarkets, and particularly in cafés and restaurants, rarely includes low-GI options of the type described above (low-GI bread instead of high GI bread, etc.). This can easily result in a low energy intake (elimination diet).

Table 1

Factors that affect GI in food

Nutrient	Effect
Gel-forming fibre	Reduced GI
Non gel-forming fibre	Negligable effect on GI
High amylose starch	Reduced GI compared to amylopectin
High amylopectin starch	Raised GI compared to amylose
Added sugar	Negligable effect on GI in use as a flavour enhancer or in fermentation
Fructose or galactose	Negligable effect on GI
Fat	Reduced GI
Protein	Reduced GI
Water	Raised GI
Structure-related properties	
Granular structure (gelatinisation)	Raised GI in connection with increased gelatinising
Retention or induction of crystalline structures	Reduced GI
Structure	Raised GI in connection with finer grinding
Cellular structure (cell wall integrity)	Raised GI in connection with increased ripeness
Formation of interaction between large molecules	Reduced GI
Particle size	Reduced GI in increased particle size
Boiling	Raised GI in higher gelatinising
Mastication	Raised GI in connection with increased mastication
Organic acids	Reduced GI
Amylase inhibitors	Reduced GI
Low GI in previous meal	Reduced GI (second-meal effect)

The most important advice for avoiding an elimination diet:

- Choose other healthy products if there are no low-GI options, e.g. wholemeal bread, fruit and vegetables, and low-fat sandwich fillings.
- Ask for low-GI products, so that shops and restaurant owners, and ultimately producers, see that there is a demand and subsequently increase the selection available. As many factors affecting the GI value of food are documented today, there are tools to help producers design new low-GI products. See Table 1.

Standardising of GI

As with other methods, methodology regarding GI has taken time to mature. How many people should take part in a GI test to ensure a sufficiently small measurement error? How should test participants and surrounding factors be standardised? How should an extreme value from a test participant be dealt with? Are product-specific values required? At what point can it be concluded that a value applies to all products in its category, despite differences in brand, ripeness, cooking, etc.?

A Danish study has researched whether the GI values in international GI tables can be used to rank blood glucose levels resulting from various breakfasts[20]. Breakfasts with widely varying percentages of fibre, protein and fat were chosen. A total of 13 different breakfasts were studied, with the only common factor that they all contained 50 g of carbohydrate. When participants' blood glucose levels were studied after eating the breakfasts, and compared to the calculated values from the GI table, the GI value was found to have no link whatsoever to the measured blood glucose level. However, a high energy intake and a high protein and/or fat intake were linked to a low blood glucose level.

The Danish study[20] could be interpreted to prove that the tables lack any foundation whatsoever. However, the results are not surprising. The factors that affect a food's GI (Table 1) affect blood glucose level whether they are integrated into the food or have been added as an additional meal component (e.g. protein, fat, and/or water). As previously mentioned, it is recommended that GI only be applied in comparisons with products in the same category, so as not to disturb the nutritional value. The same applies to meals. If the energy, protein, carbohydrate or fat content is high, it is no longer just the carbohydrate quality that is being compared.

International GI tables

Even when GI is compared within the same food groups, international GI lists can be assumed to contain errors. As previously mentioned, the GI lists have gradually been developed since 1981, and many of the values were established before measurements had been standardised between the laboratories. To a certain extent, however, some of the values have been correctly calculated, and variations in the GI lists may be due to regional differences in raw materials or local cooking traditions. A value established in India, for instance, may be incorrect in another part of the world.

The international standardisation of the GI method must continue, and it is important for GI lists to be complemented with local foods, to avoid needing to refer to international values for raw materials and products that are different to those in the Nordic countries. More product-specific values would also be useful, so that a certain product, possibly in connection with a certain cooking method (boiling, frying, etc.) guarantees a low GI.

In Sweden, it is possible to apply for a Product-Specific Physiological Claim, which authorises the labelling and marketing of products on the basis of a low GI[21].

GI in nutritional recommendations

Healthy people are still recommended to eat a high proportion of carbohydrate, see article on page 8. In addition, high-starch foods with a low GI are also recommended, for example in WHO's dietary recommendations for optimal health. Further, the new Nordic nutritional recommendations for 2004 state that carbohydrate-rich foods with a low GI could have further health-related benefits in addition to the effects of a high dietary fibre content.

A high-carbohydrate diet is still the basis of European and American dietary recommendations for diabetics. In Europe, they include an evidence-based recommendation to primarily choose low-GI sources of starch. In addition, the low glycemic load concept is also specified here as a possible alternative to a high-carbohydrate diet in individualised treatment.

The changes that can be included in a low glycemic load diet are:

- A moderate increase in protein (25-30% of energy)
- An increased proportion of monounsaturated fat (35-40% of energy) and
- A low glycemic index.

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Sugar and addiction

By Anna Karin Lindroos, PhD, Clinical Nutrition Physiologist, Department of Body Composition and Metabolism, Sahlgrenska University Hospital, Göteborg, Sweden.

In recent years, the role sugar plays in our health has been extensively discussed in the media. Sugar is regarded as one of the causes of today's growing levels of obesity, and is sometimes even described as a drug that causes us to lose control and eat even more sweet foods. Like other pleasant-tasting foods, sugar can stimulate the brain's reward system. The mechanisms of these systems are in many ways similar to those that operate in response to drugs, alcohol, and narcotics, but there are differences. Natural rewards, such as good food, sex and intensive physical training, activate the same areas of the brain, releasing dopamine but addictive substances stimulate a much more intense reponse. Furthermore, sugar is an important source of energy, and its consumption is regulated by the same systems that regulate energy balance. Consequently, it is difficult to study sugar in isolation from other nutrients that provide energy, such as other carbohydrates, fat and protein. In order to study humans' difficulty in controlling their consumption of sweet foods, both the physiological processes that control human food intake and the far more complex psychological, cultural, and social factors that determine our attitudes and views regarding sugar and sweet foods must be taken into consideration.

Results of animal experiments

In animal models, addiction-like symptoms have been found following a high intake of pure sugar. For instance, sugar consumption by rats was gradually increased in an experiment and then the sugar solution was removed. The rats showed withdrawal systems and increased anxiety[1]. The rats' 'sugar addiction' could be explained by the sugar consumption triggering the rats' reward system through the release of an increased dose of dopamine[1]. However, it is highly unlikely that these animal experiments can provide any information at all about 'sugar addiction' in humans.

It would be a gross simplification to suggest that a single biological system could explain human sugar consumption[2]. Sugar is an important source of energy, and its intake is regulated by the same systems that regulate energy balance. Consequently, it is difficult to study sugar in isolation from other nutrients that provide energy, such as other carbohydrates, fat and protein. Furthermore, the physiological processes that control human food intake, and the far more complex psychological, cultural and social factors that control our attitudes and views regarding sugar and sweet foods must also be considered.

Is craving for sweets an addiction?

Addiction can be defined as a compulsive desire to consume a drug despite serious negative consequences. In addition to a compulsive need to consume a certain substance, the following elements are characteristic of addiction: difficulty controlling consumption of the substance, withdrawal systems, increased tolerance and continued consumption despite physical or psychological damage[3].

Like any other pleasant-tasting food, sugar can stimulate the brain's reward system. These mechanisms are in many ways similar to those found for drugs, alcohol and narcotics; but there are differences. Natural rewards, such as good food, sex and intensive physical training, activate the same areas of the brain, releasing dopamine.

Addictive substances stimulate a much more intense release of dopamine[4]. Furthermore, a lack of sugar triggers hunger, a physiological urge that is qualitatively different to the craving for drugs that a drug addict develops.

Many people also argue that the term 'sugar addiction' is incorrect, since it puts sugar in the same category as drugs such as alcohol and narcotics[4]. The term addiction is complex and includes various different elements.

'Addiction' to sugar or sweet foods can rather be regarded as a behavioural disorder similar, for instance, to 'sex addiction', or 'gambling addiction'[4]. Excessive sugar consumption can also be regarded as an expression of emotional and cognitive processes that are disturbed, but not necessarily to such an unhealthy extent as actual substance addiction[3].

Cravings for sugar?

Soft drinks and certain sweets consist almost entirely of sugar. Yet studies have shown that when people feel like eating something sweet, they primarily crave products such as chocolate, ice-cream and biscuits[6]. In practice, it appears that the craving is for a quick energy fix rather than for pure sugar.

Chocolate, ice-cream and biscuits are perceived primarily as sweet because sweetness is the most evident factor, while the fat content is mainly associated with the food's consistency. If the combination of fat and sugar is most strongly linked to the reward mechanism, it is misleading to focus on sugar alone.

Eating disorders and consumption of sweet food

An extreme craving for carbohydrates has been reported by people suffering from Seasonal Affective Disorder, premenstrual syndrome and bulimia nervosa[7,8,9]. However, in both laboratory studies and studies where the participants were required to write down everything they ate over a few days, it has been difficult to show that it is specifically carbohydrates that are over-eaten during binges. While the total consumption of protein, fat, and carbohydrates increases during binge periods, the proportional distribution between the energy-giving nutrients does not change to any significant degree[10]; if anything, the amount of fat in the diet increases during binge periods[11]. Bingeing is consequently not thought to be connected with the specific over-consumption of sugar.

Lack of data

There are currently no quantitative data on how common the problem of controlling sugar intake is. To gain increased understanding of the causes and scope of this problem, well-planned questionnaires or in-depth interviews are required to shed light on an individual's whole eating behaviour. It is also necessary to study the consumption of sweetened foods in relation to a person's overall diet, and to distinguish any problems from general eating disorders and other psychological disorders.

Summary and conclusion

The processes that determine peoples' food intake are highly complex and involve systems that govern both reward mechanisms and energy balance. Money, time, knowledge, advertising and access to various foods also affect what people choose to eat, just as our food intake is influenced by associative processes and learning. Young children quickly learn to associate sweets, ice-cream and biscuits with parties and special occasions. Consequently, it is hardly surprising that these foods are associated with comfort. Certain individuals experience particular difficulty controlling their intake of sweet foods. In order to offer these individuals adequate help, further research must be carried out into the complex mechanisms that govern food consumption. This research should take into account both physiological mechanisms and the environment in which people grow up and live.

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Sugar and hyperactivity

By Søren Dahlsgaard, PhD, Senior Registrar, Psychiatric Hospital for Children and Adolescents, Aarhus, Denmark.

It is widely believed that sugar intake influences behavioural problems and lack of concentration, especially in children. Studies in the 1970s suggested a possible link, but methodological problems make the conclusions unreliable. Subsequent studies with a stronger design have not been able to establish a link between sugar intake, behaviour and concentration – even in children characterised by their parents as "sugar-sensitive". The general conclusion is that there is no scientific basis for claiming that sugar intake causes hyperactivity or other behavioural problems in children.

The body and brain need a supply of energy to be able to function. We are supplied with energy by the fat, protein, carbohydrate and alcohol content of our diet. Some of these energy sources are metabolised in the body so that they can be used by muscles, bones and the brain. The brain is the only organ in the body that can use only the sugar molecule glucose as a fuel. A constant supply of glucose for the brain is vital for our cognitive and planning abilities and necessary for maintaining essential autonomous functions – in the most extreme cases, our survival depends on glucose. Numerous hormones and signal substances and complex mechanisms are involved in the sensitive balance of the brain's metabolism[1].

'Sugar flip'

Some parents describe their children becoming restless immediately after consuming sugar-containing beverages or foods such as fizzy pop or sweets. This applies to children who do not have problems with motor unrest or hyperactivity on a day-to-day basis as well as children who already have these problems. Many alternative practitioners and some doctors support the idea of a link between sugar and hyperactivity.

This gives rise to many different proposals for treatment – either with restrictive diets or, conversely, with dietary supplements. Many people also believe that children who are often subjected to these 'sugar flips' in early childhood have a higher risk of developing permanent attention difficulties, hyperactivity and impulsiveness. These difficulties, which were previously diagnosed in child and adolescent psychiatry under the DAMP spectrum (Deficits in Attention, Motor Control and Perception)[2], are now diagnosed as hyperkinetic disorder[3] or ADHD (Attention-Deficit Hyperactivity Disorder)[4].

The neurobiology behind hyperactivity

The causes of ADHD are still unknown. Studies of twins have shown that genetic factors are decisive in around 65-90% of the children that develop ADHD[5]. Using so-called SPECT brain scans, researchers at the start of the 1990s found that certain parts of the brain functioned differently in adults with ADHD than in other adults[6]. Energy conversion in the foremost frontal parts of the brain and the cerebral cortex was reduced in patients with ADHD. These SPECT scans involved the use of radioactively marked glucose, with the quantity of glucose reflecting metabolic activity. This made it possible to localise the areas of the greatest and the least activity in the brain. However, the results of these studies have been misunderstood by some people and incorrectly used as a compelling argument for the theory of an influence of sugar on hyperactivity.

Methodological problems

Many researchers have studied whether there is a scientific basis for a link between sugar intake and motor unrest. In the 1970s, when research on hyperactivity began, case studies were produced indicating that the behaviour of a hyperactive child could be improved by removing sugar from its diet[7, 8, 9]. However, these studies lacked control groups and were not blinded. In spite of the many methodological problems, which severely limit the scientific credibility of these early studies, they are still often cited. More recent, superior studies have shown completely different results. One explanation as to why some of the early studies found that a restrictive diet not containing sugar could reduce hyperactive behaviour might be that implementing such a diet requires a very strict routine and rigid structure on a daily basis. These are things that children with attention problems and hyperactivity derive great benefit from and principles that are used today in the educational treatment of ADHD.

The Wolraich study

At the end of the 1980s, Mahan used an experimental design to study a small group of children with ADHD who, according to their parents, became more hyperactive and aggressive after sugar intake[10]. However, in this blinded experimental study the researchers were not able to confirm any link comparable to that reported by the researchers in the 1970s.
In 1994 The New England Journal of Medicine published the most scientifically solid and best designed study in this area. The study by Wolraich investigated the possible link between both sucrose and the artificial sweetener aspartame and changes in child behaviour and cognitive performance for a range of parameters[11]. In a double-blinded study, Wolraich and his colleagues investigated both a group of normal preschool children and a group of school-age children who had previously been described by their parents as 'sugar-sensitive'. For a nine-week period the children were given the following diets one after another:

- a diet with a high sugar content
- a diet with a high aspartame content
- a diet with saccharin as a placebo sweetener.

The diets contained no other artificial additives, colours or preservatives. The children's behaviour and cognition before, during and after the diets were assessed on the

basis of information from both parents and teachers, focusing on attention, impulsiveness and executive functions. Objective, systematised measurements of motor coordination and motor unrest were also taken as well as blood samples with plasma concentration measurements of amino acids and glucose. Overall, the study found that there was no difference in the children's behaviour and intellectual capacity between the three diets. Among the 23 children who were considered by their parents to be 'sugar-sensitive', no significant differences were found in behaviour or cognition. Among the 25 normal preschool children, a significant difference was found for some parameters: behaviourally, the children fared slightly better on the high-sugar diet than on the aspartame and saccharin diets. Conversely, on the high-sugar diet they had slightly slower hand movements. The study also suggests that sugar may possibly have a beneficially calming effect rather than a hyperactive effect. Other studies have also found that sugar has a calming effect and reduces the activity level[12, 13].

No link

In 1995, in a meta-analysis comparing the results of 23 studies in this area, Wolraich et al[14] concluded that sugar – in spite the firm conviction of many parents – does not affect children's behaviour or cognition. However, the study also states that it cannot be fully excluded that some children might achieve a positive effect in behaviour by eliminating sugar from their diet. In general, it can therefore be concluded that, although many parents and alternative practitioners believe that sugar intake leads to hyperactivity or ADHD in children, there is no scientific basis for such a link.

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Glossary

Abdominal fat distribution
Body fat is concentrated around the abdomen. Abdominal obesity is associated with increased risk of complications such as high blood pressure, high blood fat, diabetes and cardiovascular disease. A waist measurement of 80-88 cm for women and 94-102 cm for men means an increased risk of complications – above that, the risk is greatly increased.

Aetiology
Cause(s) of a disease.

Alkaline
Basic, i.e. pH > 7.

Amylopectin
Main component of starch (approx. 80%). Unlike amylose, amylopectin is a branched molecule.

Amylose
Makes up approx. 20-25% of starch. Long, linear, spiral chains comprised of glucose units.

Anaerobic exercise
Short, intensive bursts of training lasting up to 1 minute where the intensity is so high that the muscles work without oxygen, instead getting their energy by breaking down glycogen and creatine phosphate. This produces lactic acid.

Autonomous functions
Self-regulating functions, i.e. outside the individual's control, such as blood circulation, breathing.

ß-cells
Cells in the pancreas that produce and store insulin.

Blinded studies
Studies in which the trialists and any researchers are not told which trialists receive which type of 'treatment' (e.g. a given diet) until the study is complete and the results are ready for processing.

Case-control studies
Method for retrospectively studying the link between exposure and disease. Persons with the problem to be studied as the 'case' are compared in terms of exposure with the 'control', i.e. persons who do not have the problem. For each 'case', one or more 'controls' are chosen. The case and control persons must be of the same age and gender, and often place of residence, occupation, etc., are also matched.

Cerebral cortex
Thin layer of brain tissue with many nerve cells.

Chicory
Root containing inulin (a dietary fibre that is a polymer of fructose). Can be used to obtain fructose and fructo-oligosacharides.

Coagulation
Generic term for a series of reactions that result in blood being converted from fluid to a jelly-like mass.

Cognition
Processes that take place in the brain when we receive, store, process and use information.

Coronary heart disease
Heart disease in the blood vessels leading to and from the heart.

Cross-over design
Study in which the trialists switch between two (or more) treatments that are being tested. Each trialist thus becomes his/her own control person.

Demineralisation
Loss of minerals from tooth enamel.

Dentin
Dental bone surrounding the root of a tooth and covered with enamel.

Dopamine
Signal substance between nerve cells in the brain that controls motor activity.

Dose-response study
Study of the variation in response as the administered amount of a substance is altered.

Energy density
Energy content per unit weight, expressed e.g. as kJ/100 g. High fat content gives high energy density. Low water content often gives high energy density.

Executive functions
Generic term for various functions controlled from the front part of the brain (frontal lobe). They act as coordinators for various types of information and are responsible for all purposeful behaviour, e.g. the ability to plan or to conceal (unwanted) actions and adaptability.

Fasting triglycerides
Amount of triglycerides (a form of fat) in the blood when fasting.

Fibrinogen
Soluble protein in the blood that forms part of the mechanism of coagulation, during which it is converted to insoluble fibrin.

Glucose tolerance
A measure of the body's ability to control blood glucose concentration. Reduced glucose tolerance is reflected in an increased blood glucose level when fasting, and especially, after a meal containing any source of carbohydrate. It is thought to be a preliminary to type 2 diabetes.

Glycaemic control
Control of blood glucose.

Glycemic load (GL)
GL is a measure that takes account of both the amount of available carbohydrates in a normal portion of food and how quickly the carbohydrate is absorbed.
GL = g carbohydrate in a normal portion x GI/100.

Glycogen
Stored form of carbohydrate in muscles and liver.

Granular structure
In plants, starch occurs in the form of so-called granules (grains) with varying shape and size depending on the plant.

Haemostasis
The physiologic process whereby bleeding is halted.

Helix structure
Spiral-shaped.

Hyperkinetic disorder
Behavioural disorders (usually in children) covers attention deficit, hyperactivity and impulsiveness.

Hypertriglyceridaemia
Increased triglycerides (fats) in the blood.

Insulin resistance
Reduced effect of insulin in the body.

Insulin sensitivity
The body's ability to respond appropriately to insulin.

Intervention study
Study in which the trialists are exposed to a change, usually in treatment (e.g. a drug) or prevention (e.g. diet).

Isoglucose
Viscous solution in which the starch is almost fully hydrolysed and 40-90% of the glucose is converted enzymatically to fructose (isomerisation). Isoglucose (fructose-glucose syrup) with 55% fructose is used in the USA in soft drinks. Isoglucose with 42% fructose is used in foods.

LDL cholesterol
LDL (low-density lipoprotein) transports cholesterol in the blood stream for use by cells. It is commonly referred to as "bad cholesterol" due to the link between high LDL levels and cardiovascular disease.

Leptin
One of several hormones that affect the feeling of satiety.

Lipid values
Content of fats, i.e. triglycerides, phospholipids and cholesterol, in the blood.

Maltodextrin
Partially hydrolysed starch in which the degree of breakdown is between starch and glucose syrup. Does not taste sweet. Used as a thickener in e.g. powdered soup.

Meta-analysis
Method for making a general evaluation of a number of studies by statistically combining their results.

Metabolic syndrome
Clustering of abdominal obesity, dyslipidaemia, high blood pressure and insulin resistance.

Plaque
Bacterial coating on the surface of teeth.

Polydextrose
Bulking agent that can be used together with high intensity sweeteners in foods.

Prospective study
Forward-looking observational study, i.e. collection of data on the trialists begins when they enter the study but no intervention is involved.

Serotonin
One of several brain signal substance that affect mood and feelings of well-being, satiety or hunger.

Serum triglycerides
Type of fats in the blood.

SPECT brain scanning
Form of brain scan using isotope-labelled substances.

Thermogenic effect of food
The increase in body temperature caused by food. Makes up approx. 10% of the body's total energy output.

Thrombosis risk
Risk of blood clots.

Triglyceride
Simple fats.

Vascular endothelium
Cells that line the inside of blood and lymph vessels.

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First you add knowledge...

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Langebrogade 1
1001 Copenhagen K
Denmark