

Food and Nutrition Communication

July 2008



SUGAR

Are we addicted to sugar?

Growing evidence that fructose, especially high fructose corn syrup, can make you fat and damage your health.

After fat, sugar is now considered by public opinion as another main culprit in the worldwide spread of obesity which has developed over the last twenty years. This simple, white powder is perceived by many people to be almost as dangerous to health as a true addictive drug. What is the scientific evidence for this?

Nutrition experts recently met in Paris for a conference on sugars and weight control, organized by the Benjamin Delessert Institute. Many of their findings are reported in this edition, as is a new milestone in Nestlé research, new reported effects of excessive fructose consumption, and the essential points of the Nestlé Policy on sugars.



Sugars – What are we really talking about ?

Carbohydrates (starches, sugars and polyols) belong to a classification that is something of a mixed-bag, generally regarded as those macronutrients that are **neither** proteins, **nor** fats. They can be classified according to many different criteria, such as their chemical characteristics, (the nature of the molecule), their physio-chemical particularities such as texture, their sweetening power or taste aspects, their speed of absorption, capacity to provoke insulin secretion and their origin. The word sugars should really be reserved for mono and disaccharides of which the most frequently consumed are glucose, fructose and sucrose. Oligosaccharides, which have low sweetening power, are also metabolised in a similar way to the three others.

Sucrose (or saccharose) – table sugar extracted from sugar beet – is 50/50 glucose-fructose, and is the principal sugar in Europe representing 75% of added sugars. The production is used at 80% by industry, 20% by consumers. Its sweetening power is used as the reference (100%) against which other sugars are measured.

Cane sugar is by far the most important source of sugar in the world (75–80%); beet-sugar is only 20–25% (primarily in Europe and the USA)

Glucose syrup obtained by hydrolysis of corn or wheat starch, is a mixture of glucose, oligosaccharides, and medium and long chain polysaccharides. The sweetening power of corn syrups is between 30 and 55% that of sucrose.

Fructose syrups (isoglucose or high fructose corn syrups) are used mostly for beverages in the United States. They contain between 42 and 55% fructose, 40 to 50% glucose, and the rest as oligosaccharides. Their sweetening power is 100%, the same as sucrose. More on these later.

Pure fructose, lactose and glucose are used for specific products and in limited quantities.

Other sweeteners used by industry are honey and concentrated fruit juices, all constituted of glucose and fructose.

Sugars are widely used in food technology to obtain certain textures (in biscuits, cakes, chocolate and confectionery). They are also used in fermentation, as natural colorants (caramel), to preserve jams, cordials, syrups and fruits, and to enhance taste - all of these functions are used to produce the best stability and palatability for the consumer.

It has been “guesstimated” that in France, for example, the consumption of sugars is about 100g per day per person, or about 17–20% of the energy intake in adults and 20–25% in children and adolescents. (The statistical spread around the average is high).

Sugar and Weight – Background to Epidemiological Data

The media and certain health authorities often warn that sugars should be avoided because they may cause obesity. However, according to some experts, the data on which these warnings are based are limited, and there are a number of reasons why it remains difficult to establish a relationship between sugar intake and weight gain. Among them are:

Confused terminology – there are many different terms used to describe sugars in the diet. They vary from country to country and the same terms include different categories from one country to another - and even from one study to another in the same country. Therefore it is difficult to compile studies in meta-analyses which could help to see the bigger picture.

Carbohydrate analysis problems – with a long history of analyzing carbohydrates by difference (i.e. whatever was neither protein nor fat was carbohydrate) many countries have little direct analysis of carbohydrate or, “sugars” content in food composition tables.

Dietary intake data limited – because of the lack of information on sugars in food composition tables, there is limited data on actual intake.

Food balance data unsuited – because of the unsatisfactory nature of the basic analytic data mentioned above, researchers have resorted to using food balance sheets to examine trends. These data represent food *available* for consumption rather than food *actually consumed*, and without provisions for wastage etc. Interpretations from such data are therefore of limited value.

Use of intakes of foods as a proxy for sugar intakes – because of limited sugar intake data, research has been conducted on intakes of specific foods, such as sweetened beverages. However sugars are derived from many foods in the diet and trends in intake of one type of food may not indicate changing intakes of the nutrients themselves. There is clear evidence that intakes of certain sugar-containing foods have increased, but there is also evidence that intakes of other sugar-containing foods have decreased over the same period. Research on specific foods tells us only about those foods – it can not be extrapolated to total intake of any particular nutrient contained in those foods.

Sugars come from many foods – many foods containing sugars are excellent sources of other essential nutrients. Dairy foods containing the milk sugar lactose, such as yoghurt and ice cream, are good sources of calcium. Breakfast cereals are sources of many vitamins and minerals – eaten together with milk they also promote higher dietary intakes of calcium.





More information on sugar is needed in food composition data bases worldwide. These then could be incorporated into individual dietary surveys and epidemiological studies where body weight is being measured.

From the little data that **is** available, trends in intake of total sugars show little change in intake of the last 20 years, although there have been changes in food sources. As Dr. Alison Stephen, Head of Population Nutrition Research at MRC, Cambridge, points out, in those countries where there are fairly complete sugar intake data, such as the UK, Ireland, Australia and New Zealand, there is no evidence of a link between sugar intake and body weight in cross-sectional analyses, with some studies showing no relationship and others a negative relationship. While a small number of studies have shown a positive relationship between soft drinks and obesity, most **epidemiological** research suggests no relationship between soft drink consumption and obesity, even when studied longitudinally. Messages to reduce sugar consumption to prevent body weight gain, although seemingly plausible, are therefore contrary to the evidence provided by current epidemiological research. More longitudinal evidence is needed based on accurate data, to confirm or refute the relationship between sugar and obesity. Until results appear showing a positive relationship, recommendations to reduce obesity solely by reducing sugar intake, according to Dr Stephen, should be made with caution.

In a search for healthy carbohydrates, the attention of nutritionists and people trying to manage their weight has turned from simple sugars to starches, especially slowly absorbed complex starches which are thought to be of metabolic benefit on account of their lower Glycaemic Index. This “low GI diet” became popular after the decline from favour of the Atkins diet.



Glycaemic Index, Insulinemic Index: too many other factors involved for this to be a useful guide for slimmers.

The term “glycaemic index” has been used to mean many different things, some of which may influence body weight and some of which may not. It is commonly believed that blood glucose levels and insulin concentrations after meals influence appetite regulation, and therefore low glycaemic index (GI) and/or low “insulinemic index” (II) diets have a role to play in weight management.

However the role of low GI/II diets for body weight control is still unclear. The theoretical implications are that:

- a) GI is a qualitative indicator of the blood glucose raising ability of the available carbohydrate in a food, and is independent of the amount of food consumed, and
- b) GI only applies to high carbohydrate foods.

To obtain a quantitative index of how much food will raise blood glucose one needs to know both the amount of food consumed and its GI. This is generally known as the “glycaemic load” (GL). Because it is quantitative, GL is proposed as more useful than GI as an indicator. However, GL can be altered by changing either the amount of carbohydrate or its GI, or both, and these two operations may not have the same effect on body weight.

Clinical trials suggest that low GI diets may enhance weight loss, but the evidence is not clear. Too many other factors may intervene in free living conditions for this measurement to be used as a reliable diet calculation tool.

Other potential long-term mechanisms for an effect of low GI foods on body weight regulation are related to increased **colonic fermentation** and include a reduced efficiency of energy absorption, or an effect of colonic fermentation on hormones which regulate energy balance.

The effects on short-term satiety are not likely to be due solely to reduced glucose or insulin responses but to other factors. It can be said that a reduced rate of starch digestion leads to small reductions in energy absorption.

Nestlé researchers discover a new link between gut microbiota and glucose control

U.S. researchers recently showed a link between body weight status and gut microflora. Nestlé researchers then made another discovery linking the composition of bacteria in the gut with blood glucose control. Studies at the Nestlé Research Centre demonstrate that **modulating gut microbiota** improves the regulation of glycaemic control and **reverses the insulin resistance** that occurs with obesity. Obese, diabetic animals were given antibiotics to modify their gut microflora. The benefit of the modulated microbiota was shown by significantly enhanced oral glucose tolerance, insulin sensitivity, restored hepatic glycogen storage and a lesser accumulation of fat around the liver. Additionally, results revealed that the modified gut microbiota influenced whole body glucose homeostasis, independent of food intake or obesity. Gut microbial communities have been shown to play a critical role in the development of innate immunity, production of essential vitamins, and other biological processes. Nestlé took this knowledge a step further to determine that the presence or absence of specific bacteria in the gut may modulate the systemic inflammation which contributes to insulin resistance and obesity.

The next questions for Nestlé Research to answer are whether there is a gut microbiota profile that lowers the risk of obesity and diabetes development in human beings, and if so, can the microbiota be modulated accordingly, with food, to improve metabolic regulation and glucose control.



Nestlé will continue to work in this area to use the potential of gut microbiota regulation as an effective therapeutic strategy for managing type 2 diabetes in human beings. *The full article is published in the Journal of Medicinal Chemistry is available on the FASEB Journal website.*

Sugar from Solid vs. Liquid Foods – Does it make a difference ?

It has been widely reported that sugar ingested through drinks enter the digestive system too quickly for the caloric impact to be measured in terms of satiety, thereby adding to the body weight problem.

Scientific evidence on this point however is inconsistent. In some studies solids are found to be more satiating than liquids, in others, (for example soups), the contrary was found to be true.

Recent trials have been carried out on solid food and beverage intakes (equal calories) at 20 minute intervals before meals, to test their satiating properties -solid vs. liquid. The solid food (biscuits) and drinks of same caloric value had the same satiating power, and reduced appetite for a meal served 20 minutes after the snack.

A second trial tested different sweet drinks with the same satiating power, cola, orange juice and 1% fat milk, against a control drink of sparkling water (0 calories) this time two hours before the lunch. Neither the calorie-containing drinks nor the water reduced the appetite for lunch or moderated the number of calories ingested at that lunch, but of course the total calorie intake, (lunch + caloric drink) was higher than in the lunch + water group.

Viscosity has a lot to do with satiating effects. Drinkable yoghourts, for example, on account of their fibre or protein content, may be more satiating than regular sweet drinks. According to the author of the study, *“In the end, it is behaviour, not sugar chemistry that is responsible for weight loss or gain.”*

Sugars and Physical Exercise

Muscles mainly use energy from glucose derived from carbohydrates and fats. Glucose, which is oxidised during physical exercise comes from stores in the muscles and liver. During exercise, the consumption of muscle glucose increases, but short or moderately intense exercise does not require a change in food intake. Muscular performance can be enhanced by pre-loading of carbohydrates before or during a more intensive sports event. Fructose is often provided during sports activities, but its metabolism is different from that of glucose, and may cause digestive problems. If exercise lasts more than 45 minutes, intake of sugars at that time may increase the performance by maintaining constant the concentration of plasma glucose, instead of forcing the body to switch into fat-burning mode, which would involve a temporary drop in performance level. After exercise, it is important to re-establish liver and muscle glycogen levels in order to ensure satisfactory performance at successive events. Type 1 diabetic persons must avoid hypoglycaemia from physical effort, because exogenous insulin does not undergo the usual control mechanisms and does not diminish in response to exercise.

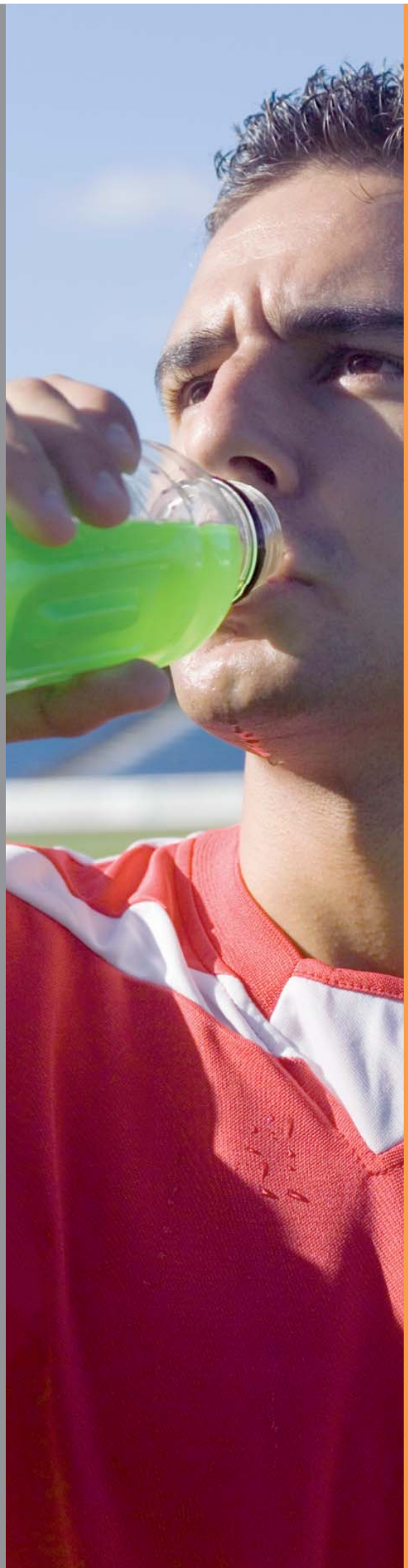
The Role of Sweet Taste in Appetite Control

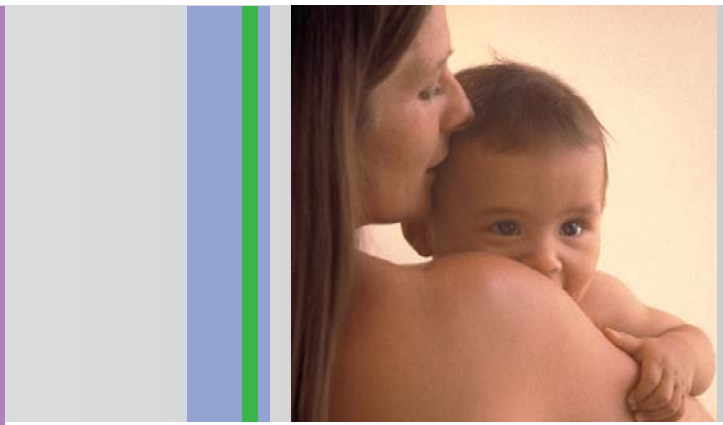
Sweetness is a potent psycho-biological phenomenon. It is associated with energy and a potent pleasure sensation. All sensory features of foods exert effects on eating, but sweetness may have a privileged position among the taste sensations. Sweet taste receptors are innate, and the pleasurable sensations are readily seen in the facial expressions of new-born babies given sweet solutions as opposed to plain water.



A key point to identify is the action of sweet taste *per se* from the effects of sweet taste plus energy (usually glucose, sucrose, or fructose). Sweet taste signalling suggests that the actions of sweet taste and energy can be dissociated. Experimental studies on sweet taste in human beings can be carried out with reference to the satiety cascade described by Blundell, Hill and Rogers 1987. The "satiety cascade" describes a series of behavioural and physiological events that occur following food intake and that inhibit further eating until the return of hunger signals. Satiety is a crucial element of the psycho-physiological mechanisms that allow adequate energy regulation and body weight control. Functional foods can affect satiety by acting at various moments of the satiety cascade. The distinction between satiation (process occurring whilst eating is in progress) and satiety (process occurring after eating has terminated) is important for the interpretation of the effects of sweet taste on appetite. Sweet foods appear to exert a weaker effect on satiety than non-sweet or savoury foods. Several studies have examined the effects on appetite of sweet taste with and without energy.

There is considerable individual variability in the consumption of sweet foods and high consumers can be said to conform to a certain high-sweet phenotype. In addition, sweet taste may produce different effects when combined with other food materials such as fat. Indeed, sweet and creamy fat combinations are a very potent sensory/nutritional mix which lead to very different responses according to gender and body mass. Sweet, high-fat foods are also much preferred and selected in normal weight women with a tendency to binge eating. Liking sweets and wanting sweets seem to have the same meaning for some people, who eat beyond hunger until the whole packet has gone, so there is a demonstrable pattern in the impact of sweet taste in particular on human behaviour, itself a highly complex domain.





Acquiring tastes in Food: The Sweet Tooth

Many observers have noted how children, the elderly, and a majority of adults are attracted by sweet tastes. The taste is partly innate, partly acquired.

The new born baby likes sugar and shows signs of pleasure even before it has ever ingested sugar and gained from its energy content. This liking for sweet tastes may be a positive adjustment mechanism as human milk has a high lactose content. Sugar is used in paediatrics for its analgesic effect during minor treatments. Although the liking for sugar tends to decrease during early childhood, it remains the most prevalent taste preference. The French study OPALINE (Observatoire des Préférences Alimentaires du Nourisson et de l'Enfant) confirms the liking for sweetness at 3 months, a new liking for salty tastes at 12 months, but at 20 months the sweet taste is still the best liked by babies.

Repeated exposure generally upholds the innate preference, but the effects of such exposure on later adult preferences are not well known. Some people retain it - some lose it. The strong liking of children and teenagers for sugar may simply reflect their high energy requirements during their growing years. It is far more difficult to encourage through repeated exposure a liking for sharp and bitter food tastes, less associated with high energy content.

Many parents believe children's desire for sugar should be curbed. However, too strict and severe an attitude can be counterproductive. By banning sugary foods: cakes, jellies, chocolates and ice-creams, traditionally served on special occasions such as birthday parties, parents can make these foods seem even more desirable to children, strengthening children's positive perception of them, and creating a greater desire to indulge and perhaps over-indulge in them, once the parental back is turned. The sweet tooth seems to be a habit developed in childhood.



Recent progress in molecular biology is leading to a better understanding of sugar detection mechanisms. The sweet taste receptor has now been identified. Differences in this receptor could lead to differences in perception between individuals. Other genes, too, could explain the subtle variations in the preference for sweet foods.

For thousands of years man lived in an environment where sugar sources were scarce. The cultivation of sugar cane and sugar beet has satisfied a natural desire for this food. Artificial and natural zero calorie sweeteners have also been developed with the idea of reducing overall calorie intake. The impact of these products in reinforcing the liking for sweet taste is not yet known.

Addiction to Sweetness: Is there any truth in this ?

One of the general definitions of the word "*addiction*" is a "*compulsive and repeated use of a substance that poses a threat to physical, social and/or economic health.*" Addictive behaviours have been described as irresponsible and irrational, given the risks taken by addicts to procure the drug of choice. In medical terms, diagnostic criteria for substance-abuse disorders often include physical dependence terms such as *tolerance levels* and *withdrawal symptoms*, and describe habits likely to seriously affect family and social obligations.

The concept has been extended via the media to the general public to include uncontrollable craving for highly palatable foods, especially those containing sugar, or fat and sugar combinations. But can we really claim that someone who simply has a sweet tooth is some sort of a sugar addict ? Perhaps the subject should be viewed from a slightly different angle.

The discovery of multiple neurotransmitters (endorphins, dopamine, etc) has inspired many theories concerning the neural substrate of sweet taste acceptance and drug addiction. More recent developments in the field of imaging have opened the way to new investigations of neural activity. For instance, a study using a PET scan revealed

similarities in brain responses in massively obese persons and drug addicts. There are therefore links between neural mechanisms involved in drug dependence and the attraction for foods **but not for sugar or sweet taste specifically**. It seems that drugs leading to addiction exploit neural mechanisms whose main function is to mediate **survival-related** behaviours such as the search for food.

While there is no intoxicating substance in sugar able to create a "substance-abuse situation" such as is clearly the case with cocaine, heroin, or crack, it is legitimate to examine whether sweet taste, while enhancing the palatability of some foods, could exacerbate conditions such as obesity, and clearly psychiatric conditions such as *bulimia nervosa*, in subjects already affected. Eating behaviours may be said to stem from individual psychological experience and personal coping strategies.

To the vast majority of people, sweet taste is simply a pleasure linked to food, and does not induce any form of physical or psychological dependence.

New evidence of potential negative effects of fructose on health since 2004

Digestion of Sugars – Glucose and Fructose

Polysaccharides, especially starches, make up a major part of our diets. They are broken down by enzymes into monosaccharides such as glucose in the villi of the intestinal walls, and carried by transporters specific to each sugar inside the intestinal cell to be delivered into the blood stream. The glucose absorbed at intestinal level is secreted into the portal vein and part of the glucose is captured by the liver. In fat cells, glucose is either transformed into fatty acids and stored in the form of fat (triglycerides), or oxidized into CO₂ to contribute to energy requirements. In the muscles, glucose can be stored in the form of glycogen or transformed into pyruvate and subsequently oxidized for energy production.



It has been observed that fructose seems to be absorbed through quite a different mechanism from that of glucose. Glucose also stimulates insulin secretion, whereas fructose does not. This is why up to a certain point fructose has been recommended for diabetics because its ingestion does not lead to rises and falls in blood sugar levels. Fructose follows the glycolic route, and may participate in the production of triglycerides. Fructose is transported into cells via a transporter known as GLUT5 which is present in only a limited number of cells. It is principally metabolised in the liver where it gets to work making fat.

By its specific metabolism, fructose facilitates fat production, and has therefore been accused of specifically contributing to obesity.

Some have pointed out that galactose, a milk sugar, is also metabolized in the liver, but galactose is converted into glucose and thereafter follows the same metabolic pathway as glucose. Research into the optimum ratio of fructose to glucose in the diet is being actively pursued. This ratio is important in understanding how fructose seems to disrupt metabolic pathways for glucose uptake.

Recent literature has highlighted the fact that fructose is unique in being the only sugar that increases blood uric acid concentrations, and evidence is accumulating that hyperuricemia is involved in many aspects of the Metabolic Syndrome.

Obesity and type 2 Diabetes are occurring at epidemic rates. The review *Nutrition and Metabolism* reports that the onset of these conditions corresponds to an important dietary change, the widespread use in the United States food industry of the sweetener high fructose corn syrup. A high flux of fructose to the liver, the main organ capable of metabolizing it, perturbs the glucose metabolism and glucose uptake pathways, and leads to a significantly enhanced rate of triglyceride synthesis. These metabolic disturbances appear to underlie the appearance of metabolic resistance characterised by deep dyslipidemia (*resulting from hepatic and intestinal overproduction of atherogenic lipoprotein particles*).



Diabetes

Global figures for type 2 diabetes are expected to reach 221 million cases in 2010. In the past, diets high in saturated fats were shown to induce insulin resistance and hyperlipidemia in humans and animals. Recent research suggests that a high intake of refined carbohydrates may also increase the risk of insulin resistance. In addition, diets specifically high in fructose have been shown to contribute to a metabolic disturbance in animal models resulting in weight gain, hyperlipidemia and hypertension.

Other literature has demonstrated that **high serum uric acid concentrations** are a strong and independent risk factor for diabetes, and that

High Fructose Corn Syrup

The process for making the high fructose corn syrup (HFCS) out of corn was developed in the 1960s. Because of its low cost and ready application in food production, especially in carbonated beverages, cured meats, condiments, preserves and bakery products, use of this syrup grew rapidly, from less than three million tons in 1980 to almost eight million tons in 1995. During the late 1990s, use of sucrose (table sugar) actually declined, as it was eclipsed by HFCS. Today Americans consume more HFCS than sucrose (table sugar).

High-fructose corn syrup is produced by processing white corn starch into clear syrup by a fairly complicated process. The corn starch is broken down by enzymes into a glucose and fructose mixture. A further process raises the level of fructose from about 40 to 90%. Three different enzymes are used in the process.

The potential role of high fructose corn syrup in the development of the metabolic syndrome, diabetes, and kidney disease was not known in the 1960s when this ingredient was being developed. Just as margarine was considered harmless for many years before the identification of partially hydrogenated fats or trans fatty acids as potentially detrimental to long-term cardiovascular health, so the use of this ingredient may soon need to be reconsidered.

HFCS is used primarily in the USA, where corn is subsidised. It is not very much used in Europe because the price of HFCS outside the US is very similar to that of sugar – and sugar gives a better sweet taste.

fructose-induced hyperuricemia can cause insulin resistance. By causing endothelial dysfunction, hyperuricemia has also been linked to cardiovascular and kidney disease. New evidence on the effects of fructose on organ-specific insulin resistance has been published in the *American Journal of Clinical Nutrition* (See references, Johnson).

Gout

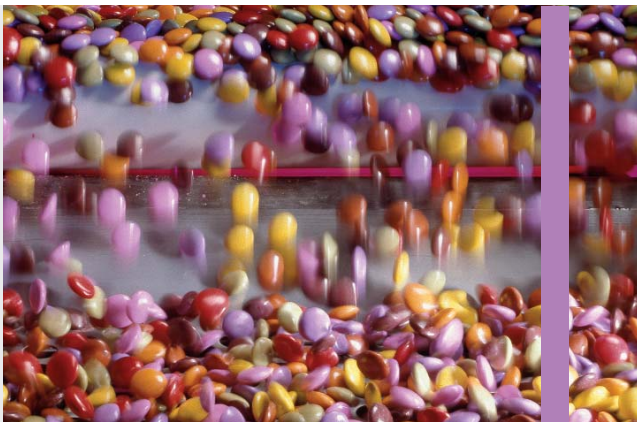
Uric acid is the main degradation product of purine that is present in a number of foods and beverages, such as anchovies, liver and beer. High blood uric acid concentrations are included in a 2002 definition of the Metabolic Syndrome. When chronically elevated, uric acid can cause the development of inflammatory arthritis, or gout. In a 12-year prospective study of more than 46,000 men, the relative risk of gout was 30% higher in people who drink 5 to 6 sweetened soft drinks per week, and 85% higher in people who drank 2 or more HFCS-sweetened soft drinks a day. High fructose fruit and fruit juice consumption were also associated with a risk of gout, whereas "Diet" soft drinks i.e. those containing artificial sweeteners, were not.

Overweight children

Recent literature and observation from Switzerland shows that overweight children consume more fructose from sweets and sweetened drinks than do normal-weight children. Higher fructose intake also predicts smaller LDL particle size. Professor G. Bray, an opinion leader in obesity research has singled out the use of high fructose corn syrup in foods and beverages as being one of the causes of the current obesity epidemic in the USA. He recommends that the relation of fructose to health should be re-evaluated.

Cardiovascular Health

Some of the literature on increased consumption of high fructose corn syrup indicates that it may also have detrimental effects on a number of biomarkers of cardiovascular health e.g. increasing



serum triglyceride and plasma insulin concentrations. Consequently this ingredient should be considered as an added sugar, and as such, used sparingly.

Nestlé Products Containing Sugars

Nestlé is committed to improving food products with due regard to food safety, nutrition standards, regulatory requirements and health concerns of consumers. Amongst the general population there is a widely held belief that table sugar (sucrose), is more dangerous to health than the sugar found in fruit (fructose) which is perceived as “natural” and therefore healthy. As we have seen above, this view is not fully supported by scientific evidence. Nestlé nevertheless appreciates the concern of consumers about excessive overall sugar intake. In response, Nestlé has formulated a policy, available on the company intranet site, with clear and specific instructions regarding all categories of our products. The policy is based on peer-reviewed scientific evidence but also taking into account consumers’ views. The goal is to reduce, wherever possible or appropriate, the total amount of sugar in our products in order to help individuals reduce their daily intake of total sugar in general and that of fructose in particular. The Nestlé Policy on the Level of Sugar in Food Products sets up limits for fructose in the product portfolio. The policy is regularly reviewed and updated in the light of sound scientific knowledge.

Nestlé policy is to reduce the level of sugars in certain products over a period of five years 2007–2012. Emphasis for sugar reduction is given to products that make a significant contribution to a person’s total dietary sugar intake, such as complete meals, snacks or drinks, and products that are intended primarily for consumption by children, such as breakfast cereals. In the first phase Nestlé will reduce the level of sugar in products by at least 16% by 2012.


Since January 2004, Nestlé has reduced the use of sugar in its product portfolio by more than 290,000 metric tonnes.

Sweeteners will be used in products to reduce sugar while retaining sweet taste only in products where this makes sense, such as calorie-controlled dairy desserts, without compromising regulatory issues or safety standards. Special products such as infant formula and baby foods are strictly regulated by national or regional authorities, professional organizations or arbitrated by the standards set by the Codex Alimentarius committee. These requirements take priority.

Sugar in its Sociological Perspective

The status of sugar has long been controversial: is it a medicine, a seasoning, or a food? Its nutritional value has been diversely appreciated. Its relation to pleasure and over-indulgence has even been the subject of theological debate. However, the link between sugar and overweight was not examined until the end of the eighteenth century. During the nineteenth century, technical progress generated increased sugar production while advances in medicine and physiology led to a new understanding of food science and the first investigative work on overweight and obesity.

The definition and measurement of excess weight has always been a moving target and therefore difficult to examine historically. Research made a clear distinction between endogenous causes where diet did not seem to be considered an issue, and exogenous causes which point the finger at overeating, leading to what the epicurean chef Brillat-Savarin called “l’obésité du ventre” or “fat paunch” among big eaters. In this case, only the link between overeating and excess weight is given as fact, but it is the overall diet rather than the consumption of specific foods which is quoted as being responsible for the increase in fatty tissue. Research by physiologists as well as medical literature for the general public between 1890 and 1930 showed that sugar alone could not, (any more than any other food taken in isolation) be held responsible for weight gain. In fact, medical authorities even promoted sugar consumption among the working classes.



The status of sugar in social representations and ideologies however, moves from one extreme to another, or, as the contemporary historian Julia Csergo observes, “from idealisation to ostracism”.

In the 1920s a social perception of the rich man as a big eater, begins to give way to the contemporary vision of the poor man as a big eater.

The industrial age brought about profound changes in food production and availability of food products. The pathological consequences of the age of abundance are denounced by the growing vegetarian and naturist movements of the twentieth century. Obesity due to over-indulgence was seen as poor management of the new wealth. Sugar took central place both in the economic process and the consolidation of these representations. Firstly because of the considerable increase in sugar production and consumption, (with the extraction of sugar from beet, sugar changed from a luxury to an everyday product). Secondly, with this increased supply came a new popularity of cakes and pastries, and an industrial boom in this type of product soon followed. Thirdly, because of its status as an “industrial” product, sugar was accused by the naturist ideology of being a “devalitized” or “dead” food, devoid of nutritive qualities. This ideology subsists, and indeed thrives, in the “bio” or “organic” movements of today.

Against this backdrop characterised by the denunciation of pleasure and self-indulgence, the modern day ostracism of sugar and its perceived role as being responsible for overweight and obesity, appears at least partially founded on a certain cultural stigmatisation of abundant, industrialised food production, and mass consumption.

To conclude:

Literature abounds on the internet condemning the consumption of all refined carbohydrates in the most vehement terms. One author even claims 146 potential health risks attributable to sugar, from loss of hair colour to both drowsiness and hyperactivity. Sorting out the sound science from the hype can be tricky. However new findings are adding scientific credibility to the popular recommendation to reduce significantly the total refined sugar we consume, if this consumption is excessive. With sugars, as with other nutrients, moderate intake is the key, especially if a person is already suffering from one of the conditions mentioned in this publication. The brain and the muscles need a lot of energy in the form of glucose to function normally, but the body does not require fructose specifically at all. The body is quite capable of converting carbohydrates from other healthy sources such as vegetables and whole grains into glucose. So who knows? It may not be long before the regulatory authorities recommend that a whole range of other foods including our favourite pure fruit juices should join the-top of-the-pyramid food category, i.e. foods only to be indulged in as an occasional treat. Let us hope at least we are the healthier for it.



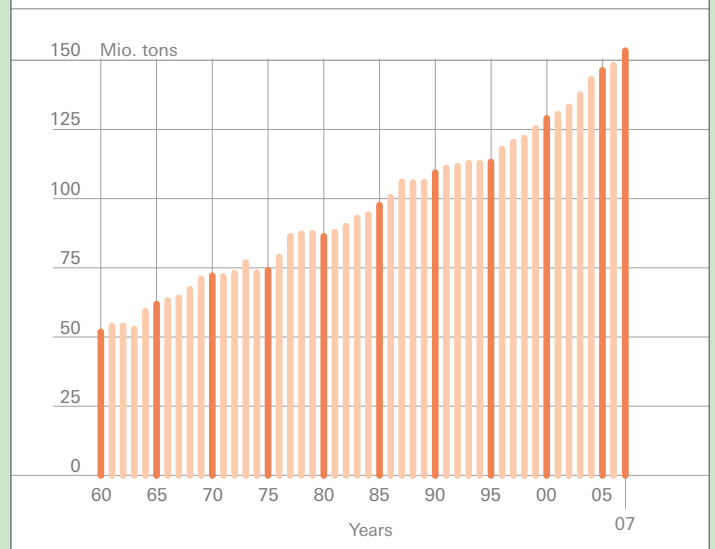
Some facts and figures

Sugar was discovered in India in the 5th century BC, and first enjoyed as a food in Polynesia. Alexander the Great introduced sugar as a sweetener into Mediterranean countries during the 4th century BC, and its popularity spread to North Africa and Southern Spain. Sugar arrived in England around 1100, brought back by returning Crusaders. It remained a rare delicacy for several hundred years, and in the 1500s a one-hundred-ton shipload of sugar was worth about a million pounds Sterling at today's prices. Venice became the hub of the world sugar trade in the 16th century, but Antwerp was the centre for sugar refining. It was Christopher Columbus who took sugar cane cuttings from the Canary Islands to the West Indies, where they thrived in the hot climate.

Imported cane sugar dominated world sugar trade until the 19th century, when continental Europe began to produce beet sugar, to challenge the monopoly of cane sugar.

More than 100 countries produce sugar, 78% of which is made from sugar cane grown primarily in the tropical and sub-tropical zones of the southern hemisphere, and the balance from sugar beet which is grown mainly in the temperate zones of the northern hemisphere. Generally, the costs of producing sugar from sugar cane are lower than those in respect of processing sugar beets.

World sugar consumption



Global sugar consumption continues to increase by about 2% p.a. Almost 154 million tons in 2007.

Recommendations for Sugar Intake

The WHO recommends that added sugars represent no more than 10% of total dietary energy, compared to the 25% recommended by the Institute of Medicine of the National Academies in the USA; this apparent contradiction is because the former relates to dental health, and the latter to avoidance of vitamin and mineral deficiencies.

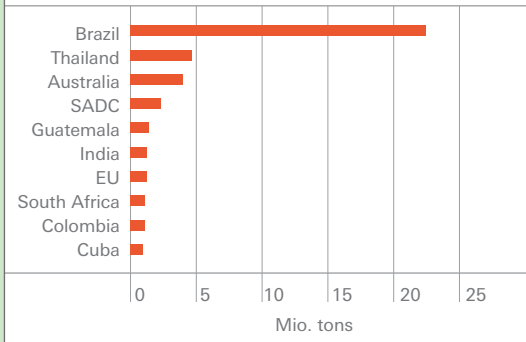
The WHO focuses on regions where there is no fluoridization of water, milk or salt, and where fluoride is not available in dental products. The Institute of Medicine's recommendation is based on the decreased intake of some micronutrients in American subpopulations exceeding this 25% level.

Refs: Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein and Amino Acids. Institute of Medicine of the National Academies, USA (2002).

*http://www.who.int/nutrition/topics/dietnutrition_and_chronicdiseases/en/print.html;
http://www.who.int/oral_health/media/en/orh_report03_en.pdf*

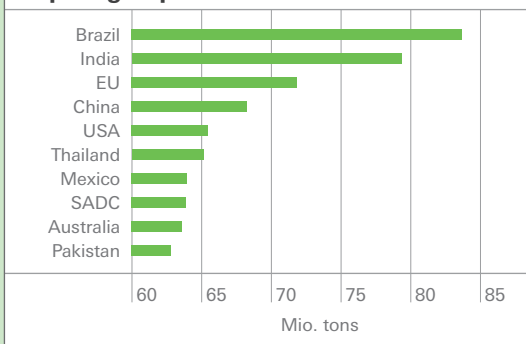


Top sugar exporters (2006/07 est)



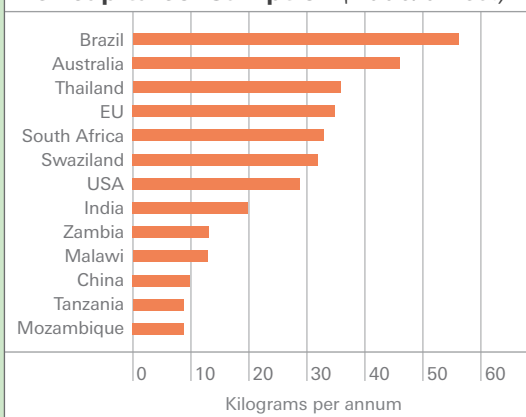
Currently, 69% of the world's sugar is consumed in the countries of origin. The rest is traded on world markets.

Top sugar producers



Global sugar production in 2006/07 is estimated at 161 mio. tons, 80% of which is produced by the world's top ten sugar producers.

Per capita consumption (2006/07 est)



Long-term potential for consumption growth, particularly in Southern Africa remains positive. Consumption growth in China has also increased

Conference participants, Paris, 12. 02. 2008

Pr. B. Guy-Grand, (Ancien Chef du Service de Nutrition, Hôtel Dieu, Paris, Vice-président de l'Institut Français pour la Nutrition) – Les Sucres: de quoi parle-t-on ?

Pr. Jean Girard, Institut Cochin, Dpt. d'Endocrinologie-Métabolisme-Cancer. Effets Métaboliques Différentiels des Sucres

Pr Martine Laville, CR Nutrition Humaine Rhône Alpes, Fac de Médecine RTH Laennec, Lyon. France. Sucres, métabolisme musculaire et exercice physique.

Dr Alison M Stephen, MRC Human Nutrition Research, University of Cambridge. UK Sugars and Body Weight: A Review of Epidemiological Data.

Pr. Tom Wolever, Dpts of Nutritional Sciences and Medicine, Univ of Toronto. Glycaemic Index, Insulinemic Index and Body Weight Control.

Pr. Adam Drewnowski, Center for Public Health Nutrition University of Washington, Seattle. Sugar from Solid versus Liquid Foods: Does it make a Difference in terms of Energy Balance?

Pr. John Blundell, Chair of Psychobiology, University of Leeds, UK. Role of Sweetness in Appetite Control

Dr Sophie Nicklaus, INRA, Dijon, France Acquisition des préférences alimentaires: le cas du goût sucré

Mme France Bellisle, INRA et CRNH Bobigny. Addiction au goût sucré: vrai ou faux débat?

Mme Julia Csergo, Maitre de conférences Histoire contemporaine, Univ Lumière Lyon 2. Le Sucre: de l'Idéalisation à l'Ostracisme.



References and Further Reading

The Nestlé Policy on the Level of Sugar in Food Products

Acheson, K.J. Green, J.H. (2004) Fructose and human health: its relevance to Nestlé.

Binnert, C. (2008) Update on fructose and concerns for human health.

Cirillo, P.W. Sato, et al. (2006) Uric Acid, the metabolic syndrome, and renal disease. *J Am Soc Nephrol* 17(12 suppl3):S165-8

Rho, Y. Woo, H.J.H. et al., (2008) Association between serum uric acid and the Adult treatment Panel III-defined metabolic syndrome: results from a single hospital database. *Metabolism* 57(1):71-6

Choi HK, Curhan G, (2008) soft drinks, fructose consumption and the risk of gout in men: prospective cohort study. *BMJ*

Dehghan, AM van Hoek, M et al, (2008) High serum uric acid as a novel risk factor for type 2 diabetes. *Diabetes Care* 31(2):361-2

Khosla U. et al., (2005) Hyperuricemia induces endothelial dysfunction. *Kidney Int.* 67(5): 1739-42

Johnson R et al., (2007) Potential role of sugar (fructose) in the epidemic of hypertension, obesity and the metabolic syndrome, diabetes, kidney disease, and cardiovascular disease. *Am J Clin Nutr* 86(4):899-906

Aeberli I.M. Zimmermann B et al., (2007) Fructose intake is a predictor of LDL particle size in overweight schoolchildren. *Am J Clin Nutr* 86(4):1174-8

Bray, GA (2007) How bad is fructose? *Am J Clin Nutr* 86(4):895-6

Basciano HK, Federico L, Adeli Khosrow., (2005) Fructose, insulin resistance, and metabolic dyslipidemia. *Nutr Metab (Lond)* 2(1):5

Membrez M, Blancher F, Jaquet M, Bibiloni R, Cani P, Burcelin R, Corthesy I, Macé K, Chou C.J. Gut microbiota modulation with norfloxacin and ampicillin enhances glucose tolerance in mice. *FASEB Journal*. 22, 2008. Published online March 7, 2008 ahead of print. doi:10.1096/fj.07-102723

Statistics: ilovosugar.com/worldofsugar/internationalSugarStats.htm
www.tateandlyle.com



Good Food, Good Life

This periodic bulletin on nutrition and health topics is written and edited in the Corporate Wellness Unit.

Concept, Writing and Editing

Veronica Roy

Review

Dr. D. Barclay

Design

Christian Stuker with Amy Becraft, Lausanne

Images

Alamy, Corbis, Getty

Printing

Presses Centrales S.A., Lausanne

Download previous editions of Food and Nutrition Communication:

- <http://www.nestle.com/MediaCenter/MediaLibrary/Documents/Documents.htm?GUID=9FC11A98-C49A-449C-A28F-CF9048BF1482>, or
- [www.research.nestle.com/Science in Action](http://www.research.nestle.com/Science_in_Action)