



SUCRES ET CONTRÔLE PONDÉRAL
Sugars and body weight control

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SUGARS: WHAT ARE WE TALKING ABOUT?

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Carbohydrates (starch, sugars, polyols) belong to a very heterogeneous range of substances: they differ and can be classified according to a large number of criteria, which causes confusion and difficulties as there is no obligatory correspondence between these different aspects. They are all carriers of energy intake from glucose, an essential substrate. The nature of the nutritional elements with which they are associated and the way in which they are consumed are important factors.

They can be differentiated according to their chemical (molecule type), physicochemical (textures), gustative (sweetening power), metabolic (uptake rate, insulin-secreting capacity, glycaemic index, etc.) characteristics, their source (naturally present or added) and regulations (ingredients or additives).

The term "sugars" (simple carbohydrates) should be reserved for mono and disaccharides, the most frequently consumed being glucose, fructose and sucrose (sugar in the singular form). Oligosaccharides (with a low sweetening power) have a similar metabolic fate. Naturally present in foods or added (by the consumer or manufacturer), they are the same molecules.

WHICH SUBSTANCES ARE USED/CONSUMED?

Sucrose, sugar (glucose-fructose, 50/50) essentially extracted from beetroot in France, remains in the lead (4 million tonnes produced, of which 2.2 million tonnes are sold in France). It represents approximately 75% of added sugars. 80% of sugar in food is used by the food industry and 20% by consumers. Its sweetening power is 100 (reference) and its glycaemic index (GI) is 68.

Glucose syrups obtained by means of wheat or corn starch hydrolysis are a mixture of glucose, oligosaccharides and polysaccharides with glucose chains of varying lengths. They are defined by a glucose content expressed as dextrose equivalent (DE) of at least 20% of dry content.

They represent approximately 25% of added sugars (in France). Their sweetening power varies between 30 and 55 depending on the formulation.

Fructose syrups (or isoglucose) are essentially used in beverages in the United States (HFCS, High Fructose Corn Syrup), but are very rarely, or not at all, used in Europe. They are obtained from glucose syrup, part of which has been converted to fructose. They contain 42% or 55% fructose, approximately 40% to 50 % glucose and the remainder in oligosaccharide form. Their sweetening power is 100, like that of sucrose.

Fructose (GI = 19), glucose (GI = 100), lactose (GI = 46) in their pure forms have quantitatively more limited uses in specific products.

Other sugars added by industry: honey, fruit juice concentrate (e.g. elderberry juice), "fruit sugars" (obtained from grape must), all consisting of glucose/fructose.

According to regulatory requirements, the presence of added sugars must be indicated on labels, but not necessarily their quantity and their type.

WHAT ARE SUGARS USED FOR?

While the use of sugars as "natural" sweeteners seems obvious in order to sweeten some foods, increase the sweetness in foods lacking in sweetness, or more or less mask the bitterness of others,

some of their functions used in industrial processes are not easy for consumers to identify.

"Sweeteners" play an important role in food technology: they can be alternatively used as texture substrates (raising of doughs in biscuit manufacture, crystallisation in chocolate manufacture, confectionery structure, etc.), natural preservatives (jams, syrups, candied fruit, etc.), fermentation substrates (chaptalisation), natural colorants (caramel, biscuit product colouring, Maillard reaction, etc.), filling agents (beverage and ice-cream body), precursors and flavouring substrates (baking products), flavour enhancers, etc., in the aim of producing stable products with an attractive taste. The question of whether it would be possible to replace them for an equivalent cost or quantitatively reduce them without altering the "recipes" remains open and needs to be considered for each individual product.

WHAT IS THEIR CONTRIBUTION TO THE ENERGY INTAKE?

Surveys such as INCA1 (1999) and INCA2 (2007) give approximations of the consumption of sugars (simple carbohydrates) in France and their contribution to energy intake.

In adults, their intake is on average approximately 100g/day at all ages, slightly less in women and adolescents, showing a slight rise in 8 years except in children and preadolescents (11-14 years). They contribute to approximately 17-20% of the energy intake in adults and 20-25% in children and adolescents.

The standard deviation is relatively broad and consumers of large quantities exist who generally consume large quantities of all types of nutrients.

The contribution of various types of foods to the total sugar intake varies according to the age group.

According to CREDOC, in children (3-15 years), juices and nectars supply 10.1%, soft drinks, fresh fruit, yogurts and fermented milks around 8% each, cakes and pastries, breakfast cereal, sugar-jam-syrup-honey, chocolate-chocolate bars around 6% each; these food categories contribute to one quarter of total energy intake (including 6.4% and 4.1% for cakes and pastries and breakfast cereal, respectively).

In adults, the main vectors appear in a different order! Sugar-jam-honey-syrup and fresh fruit account for approximately 16% each, followed by cakes and pastries 8.2%, yogurts and fermented milks 7%, soft drinks 5.5%, juices and nectars 4.5%, bread and rusks 4.2%, vegetables 4%; these foods contribute to 30% of energy intake (including 17% for bread).

IN CONCLUSION

The world of sugars is heterogeneous in many respects. They have a wide range of industrial uses, which go beyond sweetening the taste. Foods containing them are very varied in their composition and the sweetest are not necessarily the greatest contributors to the total energy intake. ■

DIFFERENTIAL METABOLIC EFFECTS OF SUGARS

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Polysaccharides, starch in particular, and some disaccharides are important components of our food. They are hydrolysed into monosaccharides by glycosidases located in the brush border of the intestine and are transported into the intestinal cells by transporters that are specific to each sugar. The sugars are then released into the blood by transport mechanisms. The major form in which sugars are transported in mammals is as glucose.

THE METABOLISM AND METABOLIC EFFECTS OF GLUCOSE

Glucose absorbed via the intestines is secreted into the portal vein and part of this glucose is taken up by the liver, thanks to a glucose transport mechanism called GLUT2. This is a high- K_m transporter, which allows it to quickly balance the glucose levels between the plasma and the intracellular environment. The glucose is then phosphorylated by glucokinase, another enzyme with a very high K_m , allowing it to phosphorylate the glucose into glucose-6-phosphate in proportion to the intracellular glucose concentration over a range of physiological concentrations (5 to 20 mM).

Glucose-6-phosphate can be used metabolically in several ways:

- it can be stored as glycogen
- it can be converted into pyruvate in the glycolytic pathway
- where the food supplies a very significant amount of glucose, it can be converted into fatty acids via the lipogenesis pathway
- finally, a small amount (10%) is metabolised via the pentose phosphate cycle to produce the NADPH required for lipogenesis.

The glucose that is not taken up by the liver is used by extra-hepatic tissues: brain (50%), muscles (30%), etc.

In the brain, glucose is transported into the neurons via a transport mechanism known as GLUT1 (low K_m) and phosphorylated by hexokinase, another enzyme with a very low K_m . In the brain, glucose is basically metabolised by glycolysis and then the Krebs cycle in order to produce energy (ATP).

In the muscles and adipocytes, glucose is transported via a transport mechanism known as GLUT4 (low K_m) and phosphorylated by hexokinase, another enzyme with a very low K_m . The specific feature of these tissues is that the glucose transporter is localised in the intracellular vesicles in the absence of insulin, meaning that it therefore does not take part in the transport of glucose through the membrane. When insulin is present, the GLUT4 vesicles are translocated to the plasma membrane and increase the glucose transport considerably.

In the adipocytes, the glucose is either converted into fatty acids (lipogenesis) and then stored as triglycerides, or oxidised into CO_2 in order to meet energy requirements. In the muscles, glucose can be stored as glycogen or converted into pyruvate via the glycolytic pathway and then oxidised in the Krebs cycle to produce ATP.

Aside from its roles as the energy substrate in the majority of tissues, it has been demonstrated in recent years that glucose is an important signal molecule. In short, glucose plays a key role in regulating the expression of genes coding for the enzymes involved in the regulation of glycolysis and lipogenesis via new transduction pathways that employ the transcription factor ChREBP (Carbohydrate Response Element Binding Protein). For more information on this topic, the reader is referred to a recent review of the subject (Postic et al, 2007).

This aspect will be discussed in particular during the oral presentation.

THE METABOLISM AND METABOLIC EFFECTS OF FRUCTOSE

Over recent years, there has been a significant change in the types of foods consumed. For example, the consumption of fructose has been hugely increased through sweetened drinks. Fructose has a sweeter taste than glucose or saccharose. It comes from fruit, but is also present in large quantities in sweetened drinks. This consumption has evolved in parallel with increases in obesity and diabetes.

Fructose is absorbed by the digestive tract using different mechanisms than glucose. Glucose stimulates the secretion of insulin, but fructose does not do this. Fructose is transported into the cell by a specific transporter, GLUT5, which is present in a limited number of cells. Once it is inside the cell, fructose is metabolised by the glycolytic pathway and can give glycerol-3-phosphate, which is used in the synthesis of triglycerides. Fructose is principally metabolised in the liver after having been phosphorylated at the carbon 1 position, a step that short-circuits the limiting step of glycolysis catalysed by phosphofructokinase. The metabolism of fructose thus favours lipogenesis and it is not surprising to observe that hyperlipidaemia is observed in people who consume large quantities of fructose. This aspect will be discussed during the oral presentation.

THE METABOLISM AND METABOLIC EFFECTS OF GALACTOSE

Galactose is also metabolised in the liver. Its metabolisation starts with phosphorylation into galactose-1-phosphate which can then be converted into glucose-1-phosphate and then glucose-6-phosphate, after which it follows the same metabolic pathway as glucose.

REFERENCE

Postic C, Dentin R, Denechaud PD, Girard J. (2007) ChREBP, a transcriptional regulator of glucose and lipid metabolism. *Annu Rev Nutr.* 27: 179-192 ■

SUGARS, MUSCULAR METABOLISM AND PHYSICAL EXERCISE

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Muscles basically get their energy from lipids and carbohydrates, in varying proportions. During exercise, the consumption of glucose by the muscles will be considerably increased. This is even greater when the muscular exercise is intense and/or prolonged. However, short-term efforts and/or moderately intense work do not require as many changes in nutrition.

The glucose oxidised during exercise comes from the glycogen reserves in the muscles and liver, and/or the carbohydrates ingested. In the muscle, utilisation of glycogen and uptake of glucose are affected by the glycogen concentration before the exercise starts.

The carbohydrates ingested during the exercise (in contrast to lipids) are rapidly available and oxidised during the course of the exercise. Blood sugar levels are kept constant thanks to neoglucogenesis in the liver coming into play, but may be slightly depressed during prolonged exercise. An appropriate intake of carbohydrates during the exercise prevents an excessively significant reduction in the glycogen stores from occurring. It is also important to note that the rate of protein breakdown in the muscle is increased if the stocks of glycogen before the exercise are low.

Several studies have shown that performance can be improved by increasing the intramuscular levels of glycogen and by an intake of carbohydrates either before the effort or during it. This is thought to be due to the additional use of exogenous glucose, which would allow the effort being made to be maintained for longer. It is however not recommended to ingest carbohydrates during the hour before the exercise, in order to limit the risks of hypoglycaemia following on from insulin secretion. Moreover, it has been shown that the ingestion of carbohydrates before exercise could inhibit lipid oxidation during the first phase of the exercise, which is compensated by an increase in the proper breakdown of glycogen to maintain the production of ATP.

The ideal is therefore to have significant stores of muscular glycogen before the effort, and to supply carbohydrates during the effort, as a function of the intensity of that effort.

BEFORE THE EXERCISE

It is therefore necessary to determine what means of nutrition allow the stores of glycogen in the muscles to be increased, particularly by a specific regimen before the exercise. It is possible to improve the levels of muscular glycogen by increasing the energy supply in the form of carbohydrates during the days preceding the exercise; these are referred to as “overloading” regimes that may or may not involve depletion beforehand, varying in the duration or the type and amount of carbohydrates ingested. On the other hand, fatigue during prolonged exercise is correlated to the depletion of glycogen and the strategies that can be implemented to economise on muscular glycogen allow stamina to be increased. The point at which fatigue arises can also be delayed by providing substantial reserves of glycogen at the start of the effort. The effects of nutritional intervention on the utilisation of glycogen by the muscle have been evaluated in terms of the glycogen concentrations in the muscles.

If the physical exercise goes on for more than 45 minutes, an intake of carbohydrates increases performance, probably by maintaining the glucose plasma concentrations at a steady level and by increased rates of carbo-

hydrate oxidation. Knowing about the origins of the sugars that are oxidised during the exercise (endogenous or exogenous) is indispensable if we are to understand these phenomena. It has been possible to achieve this thanks to sophisticated techniques involving double isotopic marking. Of these methods, tracing using ^{13}C has allowed a description to be made of the contribution due to oxidation of exogenous glucose in supplying energy during prolonged exercise. This becomes even more interesting as it becomes possible to quantify the amounts of energy supplied by the carbohydrates ingested or by other endogenous substrates respectively. So, studies have shown that the ingestion of carbohydrates by trained subjects during exercise reduces the oxidation of lipids and increases the oxidation of carbohydrates, as well as the rates of appearance and clearance of glucose in the bloodstream. The oxidation of exogenous glucose and its contribution to the supply of energy are increased as the levels of intake increase.

A substantial proportion of the energy during prolonged exercise is therefore supplied by the oxidation of glucose ingested before or during the exercise. For levels of supply at less than 1g/minute, the fraction oxidised varies from 50 to 100%. When the supply is increased to 3g/minute, oxidation flattens off at a plateau just a little higher than 1g/minute and the contribution of the oxidation of exogenous glucose to the energy supply can reach about 30%.

DURING THE EXERCISE

The quantity and quality of the sugars supplied during muscular exercise have been discussed. Fructose is widely used in sport drinks, particularly as studies have shown that fructose intake coupled with glucose intake produces an increase in the oxidation of exogenous carbohydrates (by about 50%) and an improvement in performance in comparison with an intake of glucose alone. This could be due to reduced competition during absorption, as glucose and fructose switch on different transport mechanisms in the intestines. A larger amount absorbed could increase the bioavailability of exogenous carbohydrates in the plasma and thereby explain the more elevated rates of carbohydrate oxidation when the two carbohydrates are combined. However, the metabolism of fructose is very different from that of glucose and could have negative long-term effects since it is relatively poorly tolerated in digestive terms. Saccharose and maltose seem to be oxidised in the same way as glucose in the blood. The involvement of glucose polymers in this context is open to discussion.

AFTER THE EXERCISE ...

After the effort and during the recuperation phases, the reserves of glycogen in the muscles and liver have to be rebuilt. In fact, where glycogen has been depleted, performance in subsequent exercises is significantly reduced. Numerous studies have concentrated on methods for rebuilding the stores of glycogen as quickly as possible, because it is difficult to replenish them within 24 hours. Regular consumption of large quantities of carbohydrates allows the breakdown of these stores to be restricted. The replenishment of the glycogen reserves after depletion caused by effort will be influenced by the bioavailability of the substrates for neoglucogenesis, by insulin blood levels, by the residual levels of glycogen stores, and also by the kinds of combinations of nutrients ingested.

Specific recommendations can be given to diabetics, particularly for type 1 diabetes. The crucial thing in these subjects is in fact to avoid hypoglycaemia, since exogenous insulin is not subject to the usual control mechanisms and is not reduced in response to the exercise.

This knowledge can also result in nutritional recommendations for sportsmen and women. These do however vary, depending on the type of exercise: duration, intensity, the fitness and the type of the subjects, whether trained or not, their age, their gender and their genetic makeup.

SUGARS AND BODY WEIGHT: A REVIEW OF EPIDEMIOLOGICAL DATA

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It is often suggested in the public press and in guidance from health authorities that high consumption of sugars should be avoided as these are involved in causing obesity and other disorders. However, the data on which such suggestions are based are limited and there are a number of reasons why it remains difficult to determine the relationship between sugars intake and weight control. This paper will outline some of the existing research using population-based data and describe some of the reasons why conclusions are still elusive.

SOME DIFFICULTIES IN DETERMINING THE RELATIONSHIP BETWEEN SUGARS INTAKE AND HEALTH

Of the reasons why there are difficulties in determining the relationship between sugars intakes and health re:

- *Confused terminology* – there are many different terms used to describe sugars in the diet. These vary from country to country in their usage and the same terms have different inclusions from one country to another, and even from one study to another in the same country. Studies are therefore difficult to compare and to combine together into meta analyses.
- *Carbohydrate analysis problems* – with a long history of analysing carbohydrate “by difference”, many countries have little information about the carbohydrate content of food by direct analysis. As a result, there is very limited information on sugars values in the food composition tables of many countries.
- *Dietary intake data are limited* – because of the lack of information on sugars in food composition tables, there is very limited data on sugars intakes. Only certain countries which have traditionally used directly analysed carbohydrate values, including those for sugars, have individual intake information. This has created a paucity of information on sugars intakes around the world and makes international comparisons and trends very difficult to study.
- *Food balance data are used* – with the very limited information on sugars intakes from individual surveys and studies, food balance data have been used to examine trends over time. However, these data represent food available for consumption, not that actually consumed, and there are major differences between these two terms, and the size of these differences has not remained constant over time. Hence interpretations from food balance data can be very misleading for interpretation in relation to incidence and should not be used.
- *Use of intakes of foods as a proxy for sugars intakes* – because of limited sugars intake data, many investigators have instead carried our research on intakes of specific foods, such as soft drinks and other sugar containing 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 sugars-containing foods have increased, but there is also evidence that intakes of other sugars-containing foods have decreased over the same period. Research on specific foods tells us only about these foods—it can not be extrapolated to total intake of any nutrient contained in those foods.
- *Sugars come from many foods* – many foods containing sugars are excellent sources of other nutrients.

For example, sugars-containing dairy foods, such as yoghurts and ice cream, are high sources of calcium, and breakfast cereals are high sources of many vitamins and minerals and because they are consumed with milk, also lead to high intakes of calcium. Hence different sugars containing foods have different impacts on health.

RESEARCH USING INDIVIDUAL DIETARY SURVEYS AND EPIDEMIOLOGICAL STUDIES

In order to resolve these problems, more sugars information is needed in food composition databases worldwide. These then need to be incorporated into individual dietary surveys and epidemiological studies where body weight is being measured.

Where data are available, trends in intake of total sugars show little change in intake over the last 20 years, although there have been changes in food sources. In those countries where there are fairly complete sugars intake data, such as the United Kingdom, Ireland, Australia and New Zealand, there is no evidence of a link between sugars intake and body weight in cross sectional analyses, with some studies showing no relationship and other showing 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 sugars consumption to prevent body weight gain are therefore contrary to the evidence provided by current epidemiological research. More longitudinal evidence is needed to confirm or refute the lack of relationship between sugars and obesity, and until results are available showing a positive relationship, recommendations to reduce obesity through reduction in sugars intake should be made with caution. ■

GLYCEMIC INDEX, INSULINEMIC INDEX AND BODY WEIGHT CONTROL

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It is commonly considered that postprandial blood glucose and insulin concentrations influence appetite regulation and, hence, that low glycemic 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 unclear.

USING ACCURATE, PRECISE AND CONSISTENT TERMINOLOGY

One important issue is that the term “glycemic index” has been used in the literature to mean many different things some of which may influence body weight and some of which may not. Using accurate, precise and consistent terminology is important here (as in any other field) if progress is to be made. The GI is defined as the $100 \times F/G$ where F is the incremental area under the curve (AUC) elicited by a 50 g available carbohydrate portion of a test food and G is the AUC elicited by 50g glucose taken by the same subjects. Two of the implications of this definition are: 1) 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 2) GI only applies to high carbohydrate foods. To obtain a quantitative index of how much a food will raise blood glucose one needs to know both the amount of food consumed and its GI; this is popularly assessed as glycemic load (GL): $GL = g \times GI/100$ where g is the grams of available carbohydrate consumed and GI is the GI of the food. Because it is quantitative GL is thought by some to be more useful than GI. However, GL can be altered by changing either the amount of carbohydrate or its GI (or both) and these 2 maneuvers may not have the same effects on body weight. Five recent studies lasting 10 weeks or more have examined the effect of low glycemic, low calorie diets on body fat in overweight and obese subjects. In 3 studies GL was reduced by reducing GI with no reduction in carbohydrate intake; in 3 studies GL was reduced by reducing both GI and carbohydrate intake; and one study GL was reduced by a modest reduction in carbohydrate (60 to 40% of energy) with no change in GI. All of these diets tended to reduce body fat compared to the control, but the most consistent effects were seen with low GI as opposed to low carbohydrate.

LOW GLYCEMIC DIETS AND WEIGHT MANAGEMENT

Clinical trials suggest that low GI diets may enhance weight loss, but the evidence is not clear. Part of the reason for this may be that there are many factors in foods which may cause them to have a low GI, but not all of them may have the same effects on body weight management. The 2 major mechanisms leading to a low GI are the nature of the monosaccharide absorbed and the rate of absorption. Fructose has a GI of approximately 20; it elicits glucose and insulin responses about 20% those of glucose. Thus, the GI of sucrose, being made up of equal proportions of glucose and fructose, has a GI of about 60, which is the mean of the GI values of glucose and fructose. However, preloads of fructose do not elicit feelings of satiety and do not reduce short-term food intake nearly as much as an equivalent amount of glucose. Thus, although incorporating fructose into a diet is an

easy and very effective way of reducing its GI, it may not be effective in eliciting weight reduction. The GI values of starchy foods are directly related to their rate of digestion *in vitro*. However, there are many factors in foods which affect the rate of starch digestion and absorption such as starch structure (amylose vs amylopectin), the degree of gelatinization, chemical or enzymatic modifications, particle size and the presence of dietary fiber or other components. These factors may have different effects on appetite and body weight control which may also confound the effects of differences in postprandial glucose and insulin *per se*.

Low GI or low II diets may influence body weight short-term (i.e. meal-to-meal) or long-term (i.e. adaptation over weeks or months) mechanisms. The most common mechanism ascribed to low GI/II foods is that they increase short-term satiety and reduce short-term food intake. However, the data are not consistent and there are confounding factors present in virtually every study. Ludwig recently cited 16 studies as evidence that low GI foods reduce appetite, but it is not possible to attribute this to the reduced glucose or insulin *per se* because of confounding factors such as differences in fiber, volume, protein or the amount of chewing required. In addition, there are several different hypotheses relating to glucose and insulin responses which are not consistently supported by the evidence. Sometimes the reduction in the peak glucose and insulin is considered to be important, sometimes it is the lack of undershoot (related to dynamic falls in glucose) and sometimes it is the prolonged absorption. The timing of the effects on satiety (early, late, continuous) do not correspond consistently with the timing of the glucose/insulin effects. In addition, the relationship between glucose/insulin responses and satiety varies for different types of foods, which strongly suggests that factors other than postprandial glucose and insulin are more important determinants of short-term satiety responses.

LOW GLYCEMIC FOODS ON SHORT TERM SATIETY AND FOOD INTAKE

The factors regulating food intake are very complex and include numerous hormones secreted by the gut (eg. GLP-1, PYY, CCK), the pancreas (eg. insulin) and adipose tissue (eg. leptin). It has long been debated as to whether hyperinsulinemia is a cause or an effect of obesity. This debate has not been resolved. Evidence that hyperinsulinemia is a physiological adaptation to obesity which helps to limit further weight gain includes a recent study of ours suggesting that hyperinsulinemic subjects have less food intake in response to a preload of glucose than those with low insulin. Also, insulin resistant, hyperinsulinemic adults gain less weight than insulin sensitive adults. However, in favor of insulin being a cause of obesity is evidence that insulin resistant children gain more weight than insulin sensitive children. In addition, suppressing insulin responses with octreotide elicits weight loss in obese subjects, an effect associated with reduced energy intake the latter of which was suggested to be due to increased leptin sensitivity. 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 a potential effect of colonic fermentation on hormones which regulate energy balance.

It is concluded that low GI foods may assist in weight management. The effects of low GI foods on short-term satiety and food intake are not likely to be due solely to reduced glucose or insulin responses, but to other factors; nevertheless these may contribute to long-term reductions in food intake. A reduced rate of starch digestion leads to small reductions in energy absorption and may have direct and indirect effects on gut, pancreatic and adipose hormones which regulate energy intake and energy expenditure. ■

SUGAR FROM SOLID VERSUS LIQUID FOODS : DOES IT MAKE A DIFFERENCE IN TERMS OF ENERGY BALANCE ?

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Reports that humans are incapable of detecting calories presented in liquid form have been used to blame caloric beverages for the global obesity epidemic. However, the scientific evidence on this point is inconsistent. In some experimental studies, solids were more satiating than liquids, in other studies, soups and liquid formula diets were more satiating than solids, leading to more effective weight loss. In a series of experiments, we tested the impact of liquid sugar calories on hunger and satiety. The first study compared the effects of liquid cola and solid cookies on hunger, thirst, satiety, desire to eat, and energy intakes at the next meal. Participants (16 men; 16 women) consumed equicaloric amounts (300 kcal) of either cola (24 oz) or raspberry cookies (87g). Lunch was served either 20 minutes after the snack or 2 hours later. Both cola and cookies spoiled appetite when consumed 20 min before lunch. It made no difference whether the calories were liquid or solid. The second study compared the effects of cola, orange juice and 1% milk, with sparkling water as zero-calorie control. The three caloric beverages differed from water, but not from each other in their satiating power. In other words, the satiating value of cola, juice and low fat milk (all have energy density of 0.4 kcal/g) was exactly the same. All had the same satiating power. On the other hand, consumption of beverages before lunch did not lead to reduced energy intakes at lunch – the total amount of energy was higher in the beverage than in the water condition. Later studies comparing yogurts with fruit-based or dairy-based beverages showed that yogurts had more satiating power. In particular, drinkable low-energy-density yogurts enriched with fiber behaved more like high energy-density yogurts. This may have been due to the yogurts' viscosity or their higher protein or fiber content. In clinical studies, liquid formula diets, containing sugar, as well as some protein and fiber, were associated with high satiety and improved weight loss. There is no merit to the idea that sugar calories provided in liquid form invariably lead to weight gain. Caloric liquid formula diets are one way to promote weight loss. Behavior, not sugar chemistry, is responsible for weight loss or weight gain. ■

ROLE OF SWEETNESS IN APPETITE CONTROL

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SWEETNESS, A POTENT PSYCHOBIOLOGICAL PHENOMENON

Sweetness is a potent psychobiological phenomenon. The importance comes about because the sweet taste, in nature, is normally associated with the presence of energy and therefore humans (and other animals) are likely to be strongly attracted to sweetness in foods and drinks. The sweet taste is also associated with a potent pleasure sensation. The hedonic properties of sweetness means that it embodies strong reward potential with the capacity to reinforce its own consumption and behaviour associated with consumption. For this reason it can be expected that sweetness will exert positive and distinctive effects on eating behaviour, food selection and other aspects of appetite control. It is likely that sweetness is likely to have a 'facilitative' or 'permissive' effect on eating behaviour.

Although all sensory features of foods exert marked effects on eating, sweetness may have a privileged position among the taste sensations. Sweetness can confer biological meaning and it can be argued that humans have a genetic preference for sweetness. This could have arisen because sweet receptors are innate and there is a universal association, in nature, between sweetness and energy yielding (useful) properties of foods. For this reason it is arguable that sweetness may have qualitatively distinct attributes of pleasure – because of the unique role of sweetness in nature. However sweetness can be conferred through different types of molecules which may have distinguishable properties.

SWEETNESS PER SE VS SWEETNESS PLUS ENERGY

In studying sweetness, one key issue is to identify the action of sweetness per se from the effects of sweetness plus energy (usually glucose, sucrose, fructose). Sweet taste signalling suggests that the actions of sweetness and energy can be dissociated anatomically. Experimental studies on sweetness in humans can be carried out with reference to the satiety cascade (Blundell, Hill and Rogers, 1987). The distinction between satiation (processes occurring whilst eating is in progress) and satiety (processes occurring after eating has terminated) is important for the interpretation of the effects of sweetness on appetite. Most experiments have studied the effect of sweetness on satiety. In this context the distinction between 'additive' and 'substitutive' strategies is important in unravelling the effects of sweetness per se. (Rogers and Blundell, 1989). Several studies have examined the effects on appetite of sweetness with and without energy. The physiological system in a state of need (energy deficit) is particularly sensitive to these effects (King, Appleton and Blundell, 1999).

Considering general satiety, sweet foods appear to exert a weaker effect than non-sweet or savoury foods (de Graaf et al, 1993). For so-called sensory specific satiety, the absence of equipotentiality among sensory qualities reveals that sweetness exerts quantitatively different effects on the willingness to consume different types of foods. This action may be mediated via the unequal action of sweetness on the separable components of the hedonic response – liking and wanting (Finlayson, King and Blundell 2007).

There is considerable individual variability in the consumption of sweet materials with high

consumers conforming to the description of a high sweet phenotype (Appleton, Rogers and Blundell, 2001; Appleton and Blundell, 2007). In addition sweetness may produce different effects when it is combined with other food materials such as fat; indeed the sweet-fat combination is a very potent sensory-nutritional matrix which leads to noticeably different responses according to gender (Drewnowski et al, 1992) and body mass (Macdiarmid et al, 1998). Sweet high fat foods are also highly preferred and selected in young normal weight women with a measurable tendency toward binge eating (Arlotti et al, FENS 2007). This means that there is considerable heterogeneity in the impact of sweetness on human behaviour.

There are powerful psychological and behavioural responses to sweetness which cannot be captured in a single summary statement. ■

ACQUIRING TASTES IN FOOD: THE CASE OF THE SWEET TOOTH

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Numerous observers have noted how children, the elderly and a majority of adults are attracted by sweet tastes. The preference for sugar, as other food preference, is partly acquired. When is this preference acquired, and what are the decisive factors? Those are the questions we shall attempt to answer. Where possible, we shall make comparisons with the acquisition of preferences for other tastes.

THE CHILD'S SWEET TOOTH

Unlike other tastes, the new-born infant has a liking for the taste of sugar which it expresses by signs of seeming satisfaction even before it has ever ingested sugar and gained from its energy content. This liking for sweet tastes can be seen as a positive adjustment mechanism as human milk has a high lactose content. It is used in paediatrics as the analgesic effect of sweet stimuli soothes the infant undergoing minor treatment. Although the liking for sweet tastes at birth diminishes to a certain degree during early childhood, it remains the most prevalent. The OPALINE study (Observatoire des Préférences ALimentaires du Nourrisson et de l'Enfant), which our team is working on, looks in particular at the development of the appreciation of different tastes from 3 to 20 months of age. First results confirm the appreciation of sweet taste at the age of 3 months and highlight amongst others a greater liking for salty tastes at 12 months, but at 20 months sweet taste remains the most appreciated.

THE ACQUISITION OF THE PREFERENCE FOR SUGAR

Beyond the inborn liking for sugar, the taste for sweet foods is acquired after repeated exposure. Various studies demonstrate that exposure to sugar in the early years of life generally tends to uphold the innate preference for this taste. But the effect of such exposure during childhood on the later developments of a more marked preference for sweet foods is not known. Certain longitudinal studies observe however that teenagers who, just like children, appreciate high sugar contents have a more moderate liking for sugar once they reach adulthood. The strong liking of children and teenagers for sugar may be explained by their high energy requirements while they are growing. The large number of sweet foods and beverages on the market give them easy satisfaction.

Although there is no knowledge of the long term impact of repeated exposure to sweet foods and beverages, it is clear that a liking for sharp or bitter tastes, far less present at birth, is far more difficult to encourage through repeated exposure than the liking for sweet foods which is reinforced after the first intakes. The need for high energy content is a decisive factor in the determination of food preferences. It is less decisive in the case of sharp or bitter foods, less associated with high energy content. Thus, adding sugar to bitter vegetables will make them more palatable to children, as the unpleasant taste will be disguised and/or the energy content increased.

Parents tend to believe they should curb their children's taste for sugar. Some parents avoid giving their children too many sweets or sweet beverages which they consider habit-forming. Being very strict can be counterproductive: for instance, banning certain sweet foods may only make them

more attractive. Despite this, sweet foods are often used as a reward or served at parties, thus strengthening children's positive perception of them.

A VARYING TASTE FOR SUGAR

Liking sugar goes back to childhood, but some children are more keen than others. Recent progress in molecular biology leads to better understanding of sugar detection mechanisms : the sweet taste receptor (a dimer with two transmembrane proteins) has been pinpointed. Differences in this receptor could lead to differences in perception between individuals. Furthermore, other genes could also explain subtle variations in the preference for sweet foods.

THE FUTURE OF THE SWEET TOOTH

For thousands of years man lived in an environment where sugar sources were scarce. In the past century his natural appetite for sweet foods has been increasingly satisfied thanks to the cultivation of sugar cane and sugar beet. Besides, artificial sweeteners have been developed which have a very sweet taste but less energy content than natural sugar. The impact of the presence of these ingredients in foods and beverages on a possible reinforcement of the appreciation of sweet taste is not yet known.

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ADDICTION TO SWEETNESS : IS THERE ANY TRUTH IN THIS ?

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The general public is occasionally told that sugar can induce an addiction. This statement is in fact far more complex than it may seem. But before we can decide whether or not it is true, let us consider what an addiction entails, after which we shall explain what “sugar” actually means.

WHAT IS ADDICTION?

In some scientific publications, the word addiction is used to describe 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 both irresponsible and irrational, given the sacrifices that addicts make, and the serious risks they take, to procure their drug of choice. In recent years, the concept of addiction has been extended to uncontrollable craving for palatable foods.

In this context, it is quite amazing to realise there is no authorised scientific or medical definition of addiction. The Diagnostic and Statistical Manual of Mental Disorders (DSM IV) does not provide a specific definition of addiction. Instead, the Manual provides diagnostic criteria for substance-dependence disorders, specifically those related to drugs, alcohol, nicotine and caffeine. The definition may include, though not necessarily, a physical dependence revealed by « tolerance » and « withdrawal », and describes a compulsive habit likely to seriously affect family and social obligations. The word addiction is absent from psychiatric nosography. During this conference, we will see whether, and to what extent, sugar can induce one or more of the criteria referring to physiological or psychological dependence.

WHAT DO WE CALL SUGAR ?

Whilst this question may seem trivial, the implications are actually more far reaching. We need to fully understand this substance that may trigger dependence. What the general public calls sugar is actually saccharose, a simple carbohydrate with a sweet taste. There are other sweet tasting simple carbohydrates (fructose, glucose); there are also other sweet tasting substances which are not carbohydrates; and there are carbohydrates that do not taste sweet. Which of these many substances might induce any of the dependence-related signs ? A great number of studies have been devoted to sugar, to sugars and to sweetness. All of them indicate that in some individuals, we find a pattern of compulsive use in line with the DSM IV definition, though it doesn't apply to sugar nor to sweetness but rather to palatable foods which often contain more lipids than sugars, though many of them are both fatty and sweet.

SUGAR, SWEETNESS AND HOW THE BRAIN WORKS

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 in drug-addicts. There are thus links between neural mechanisms involved in the attraction for foods (but not for sugar or sweetness specifically) and drug dependence. It seems that drugs leading to addiction exploit neural mechanisms whose main function is to mediate survival-related behaviours, in particular those that are linked to the attraction for foods.

SWEET TASTE AND THE FOOD PATTERN CONTROL

It is now a fact that, as early as birth, any sweet tasting stimulus perceived by a new born baby is met with approval. Later in life, the sweet taste will evolve and fit in every individual's hierarchy of food likes and dislikes. To most people, sweetness goes back to the pleasure linked to food without inducing any form of physical or psychological dependence. How far sugar or sweetness contribute to compulsive eating behaviours, as observed in bulimia nervosa or with some patients suffering from severe obesity, still needs to be established.

To say that sugar can induce an addiction is nonsensical since there is no clear definition of addiction. It is however, justified to ask whether sweetness, while enhancing the palatability of some foods, could contribute in seriously disrupting uptake patterns and thus jeopardise social or family obligations; and to wonder who might be the target (people suffering from bulimia, obesity, etc.), in which circumstances it may occur and what mechanisms are involved. ■

SUGAR: FROM IDEALISATION TO OSTRACISM

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The status of sugar, and by sugar we mean, for the purpose of this study, sucrose only, has long been controversial: is it a medicine, a seasoning, a food? Its nutritional value has been diversely appreciated. Its relation to pleasure and over-indulgence has even nourished theological debate. But the link between sugar consumption and overweight was not examined until the very end of the XVIIIth century.

Through scientific research and social representations the XIXth century first raises the contemporary issue of sugar and overweight. I propose to examine today's question in the light of that historical perspective.

It is during the XIXth century that technical progress generated increased sugar production; scientific discoveries lead to the inception of food chemistry; headway in medicine and physiology pave the way to a new food science and the first major research on plumpness and obesity.

Handling such notions is no easy task for historians: the definition and the measurement of excess weight have always been a moving target even in the XIXth and XXth centuries. Besides, research into the etiology of weight gain make a clear distinction between endogenous causes – where diet is not an issue – and exogenous causes which point the finger to overeating which in turn leads to what Brillat-Savarin called « waist-line obesity » among « big eaters ». In this case only, the link between overeating and excess weight is proven. However it is overall diet rather than the consumption of certain 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 1890-1930 on «How and what to eat ? » show that sugar alone, no more than other foods, cannot be held responsible for weight gain. The energy and nutritional content of sugar is confirmed and its consumption recommended to the working classes and only leads to overweight if taken in excessive quantities in association with overly rich diets.

However, to answer the question at hand one should also discuss the status of sugar in social representations and ideologies.

In the 1910s-1920s there is violent criticism of the « fat take-over » : the image of the « rich man eating too much » is replaced by the more contemporary image of the « poor man eating too much ».

The advent of the industrial age brings about deep changes in food production, and in their wake cheaper food prices and mass consumption. It heralds the era of abundance, which the proponents of the XXth century ideology of vegetarianism and naturism believe will have pathogenic consequences. Now accessible to all, overeating, with its consequential health risks – mainly obesity and the disorders it causes– is henceforth seen as « a plague of civilisation », a mismanagement of wealth and abundance.

Sugar plays an essential role, both in these economic processes and in the construction of these representations. First because of its increased production and consumption (once sugar is extracted from beet, it is no longer a luxury product but a convenience); secondly, because of a growing

popular taste for « sweet treats », increasing pleasure is taken in consuming confectionaries and cakes produced by a booming industry ; and finally , in view of its image as an industrially produced food, denounced by the naturist ideology as a « devitalised » or « dead » product, deprived of nutritional qualities.

Against a cultural backdrop which denounces pleasure and excess, the «modern » ostracising of sugar, its status as main culprit in the propensity to be overweight therefore seems largely rooted in a stigmatisation of a civilisation of abundance, a culture where food is produced industrially and consumed massively. ■

PROFESSOR BERNARD GUY-GRAND'S CONCLUSIONS

We have come to the end of a most informative day, during which each individual has had the chance to make up their own minds whether or not sugars plays any specific role in the obesity epidemic. If only one thing were to be remembered from today however, it would be that due to the complexity of the problem, it is difficult to come to simple yes or no answer.

I will summarise the points which seem to me to be the most important ones for the debate and the potentially useless controversies they raise.

Added sugars are identical to those naturally present in food. Their metabolism is the same. This first criticism of sugars is not necessarily well-founded, except perhaps in regard to the fructose-glucose ratio of certain added sugars, mainly in the United States, but not in France or Europe.

As J. Girard explained, it may also be claimed that fructose has particular metabolic characteristics. In small quantities it is useful; consumed in excess however, it fosters lipogenesis. But what is “excessive”? Nobody knows. What should be remembered is that that fructose has a positive effect on inflammatory conditions which play a role in the genesis of cardiovascular diseases or obesity.

As M. Laville reminded us, it can be inferred from historical trends that sugar is an energy food; all serious experts agree on that subject. Personally I am all in favour of a sugary fizzy drink, provided if taken while playing a tennis match, not while watching one on television.

As T. Wolever explained, the glycemic index, even though the mechanisms through which it intervenes remain unclear, does have certain virtues, at least in maintaining a stable weight, and possibly – but not necessarily – in weight loss.

I am talking here in terms of public health, not in terms of any obesity treatment, which is completely different area, one in which energy deficits completely change the data concerning the metabolic situation. The glycemic index is certainly of interest, but all of us have learned that the physiological effects of the vector and the food as a whole do not necessarily allow its effects to be predictable. Therefore, this might be a notion which, while it should perhaps not go unchallenged, should not be questioned on the basis of false notions, particularly on the basis that the glycemic index of a complex food can only be established if it is tested.

A. Stephen has demonstrated that, regarding epidemiology, she would find it impossible to say whether sugar plays a role or not, or better said, that she can offer no argument that might currently allow us to state that the consumption of sugars plays a role in obesity, in weight gain. There are as many positive arguments as there are negative ones, and a series of studies with results that contradict each other. But the same type of conclusion also applies to fat. It is impossible to imagine that a single food, or category of food, should be “guiltier” than any other, since the true case is the overall caloric intake.

The question of sugary drinks is quite a different one indeed. We understand that sugary drinks are neither more or less satiating than others, as was brilliantly demonstrated by A. Drewnowski. The question revolves around the quantity of calories ingested, the energy density, and water content. Sugary drinks, in themselves, do not have undersatiating qualities. Nevertheless, without there being any specific effect linked to their sugar content, the appetite for sugar, whose homeostatic roles J. Blundell recalled, is a powerful stimulus for its own consumption, and may thus increase the caloric ration. At fault here is not necessarily a

failure of the satiety related to the nature of the food. It may be due to a failure in energy homeostasis regulation. Indeed, J. Blundell reminded us that hedonism participates in a sort of homeostasis which is social or psychological, not metabolic.

S. Nicklaus has endeavoured to demonstrate how unwise it is to begin banning substances altogether. Those in charge of prevention policies do not see it in this light! They never stated that eating sugar was unnecessary, that this was the enemy. They merely said that an individual wanted to save a few calories, they might find them through food with either higher sugar contents or higher fat contents. This is simple common sense. On the other hand, in the public's mind – and this is where one calls on the lessons of history – sugar thereafter becomes a kind of protest. We are no longer living in the days of yesteryear, when sugar was the food of the rich, the food of kings. When the child becomes the child king, they eat sugar. It can be said, but one should take care not to confuse everything. Surely, the biological, the physiological, and psychological concepts, along with the social, moral, and historical concepts, form a cultural corpus which evolves through time in a cyclical fashion. J. Csergo has provided us with recurrent examples.

F. Bellisle gave a documented presentation which challenged the notion of addiction to sugar. A person does not become bulimic because they have eaten sugar. Bulimia has another dimension. In any case, a bulimic person will fill their internal void with whatever is at hand.

As for me, I would not hesitate to state my own position, and that is that sugars need not be ostracised and that the struggle against obesity goes far beyond nutrition. How can the impact of a single food on obesity be assessed if one does not take into consideration the energy expenditure of the person consuming it? This goes far beyond nutrition as we understand it: carbohydrates, lipids, proteins, nutrients, vitamins... There remain entire fields which have not been the subject of specific research. I am thinking, for example, of the insufficiency of the omega 6/omega 3 ratio in food, which has nothing to do with calories but do not have the same adipogenic potential. The coming years will see the rise of a new field, that of food toxics, additives, pesticide residues, which we know are controlled and studied. They are not toxic in the classic sense, they do not kill us, but these molecules are certainly not inert and can change the metabolism. Far beyond classic nutrition, this is an issue of society.

I am not overly optimistic regarding the evolution of the obesity epidemic. It is a global social problem which appeared in the middle of the 20th Century. It would seem to me that it would be difficult to bring about any in-depth change in the evolution of the world, at least over anything short of decades. If we could only stabilise the problem, that would already be cause enough for some contentment. There remains much to do, but nothing will be achieved by if we set one side against the other, launching invectives, responding with anathemas. There is progress to make on all sides; on the medical side, by ceasing to damage patients with restrictive diets imposed for no reason; on the agro-food side, by modifying what can be modified in the food on offer; progress also regarding agricultural policies. Nor should individual responsibility be forgotten: couldn't television advertisements for food products directed to children be forbidden? But focusing on such advertisements while overlooking the failure of the family unit and the lack of awareness-raising among children, that becomes a major problem. All of us are players, so what is needed is an overall policy: the policy of the city, the policy of urbanism, assuredly, all of this is as important as the food's sugar contents.

May humility and perspective impede overly hasty judgments. ■

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