

Nutrición Hospitalaria



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En la parte inferior se especificará el número total de palabras del cuerpo del artículo (excluyendo la carta de presentación, el resumen, agradecimientos, referencias bibliográficas, tablas y figuras).

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Encabezando nueva página se incluirá la traducción al inglés del resumen y las palabras clave, con idéntica estructuración. En caso de no incluirse, la traducción será realizada por la propia revista.

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Se deben citar aquellas referencias bibliográficas estrictamente necesarias teniendo en cuenta criterios de pertinencia y relevancia.

En la metodología, se especificará el diseño, la población a estudio, los métodos estadísticos empleados, los procedimientos y las normas éticas seguidas en caso de ser necesarias.

1.7 Anexos

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A critical, updated overview of the role of sugar in our diet

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A healthy diet should meet individual nutritional needs and also incorporate cultural and gastronomic values that make it enjoyable. However, various studies show that nutritional imbalances are the main cause of the premature development of most chronic or degenerative disease that currently affect today's society. Changes that have taken place in the population's lifestyle habits have caused an increase in the prevalence of many chronic illnesses such as obesity, diabetes and metabolic syndrome which have ultimately led to a rise in cardiovascular morbidity. Evolving demographic and lifestyle trends, particularly when it comes to the incorporation of unhealthy diets and the absence of physical activity, are behind all this. Fortunately, despite all this, it is preventable.

Balance, weighting and variety continue to be the cornerstones of a healthy diet. The simplification and categorisation of foods into 'good and bad' is not appropriate, and it would be more accurate to talk about adequate or inadequate diets, according to the variety of foods they contain and the overall calorie intake in relation to physical activity. It's therefore a matter of educating and training people with the aim of encouraging suitable decision making about their diet and nutrition. Moderation should be applied to those foods that may provide excessive calories and/or fat, particularly, if they are poor quality. As far as sugar is concerned, for decades it has been a widely held opinion that it has been responsible for a multitude of ills that humans have to face, and has even been considered a poison. It is a statement which is meaningless from a scientific and critical point of view, and yet, the reality is that this concept has many faithful followers, including in the scientific and health sectors which inevitably creates a great deal of confusion among the general public, who don't know what to believe.

The objective of this paper is to review the most debated aspects of this food. To do this and with the intention of providing greater clarity on various issues, a methodology for studying the literature related to SWOT analysis, detailing the internal issues (Weaknesses, Strengths) and their external issues (Threats and Opportunities) as well as making final recommendations.

Confusing terminology

Carbohydrates are the world's main source of food energy, particularly in developing countries. Carbohydrates should provide between 50 and 55% of total dietary energy and should also be evaluated for their potential energy, their sweetness and their high fibre content¹. The European Food Safety Authority (EFSA) has recently advised that carbohydrate intake should range from 45 to 60% of energy for both adults and healthy children over one year of age².

Carbohydrates present in the diet may be in the form of complex molecules (polymers or polysaccharides) or more simple molecules, commonly referred to as sugars; these can be classified as monomers (monosaccharides) or dimers (disaccharides). Of all the dietary sugars, the most important from a nutritional point of view are: glucose, fructose, galactose, maltose, lactose, sucrose and trehalose.

Sucrose is the sugar par excellence. It consists of one fructose and another of glucose linked via a glycosidic bond. It is extracted industrially from sugar cane and sugar beet. It is also used to sweeten foods, to improve the sour and bitter taste of many of them and to preserve them by increasing osmotic pressure, which inhibits the growth of many microorganisms. In addition to this, there are other less well known functions of sugar such as modifying the melting and freezing points or as a method of naturally colouring food.

There are many terms used to describe sugars and their components: sugar(s), total sugars, total available glucose, free sugars, added sugars, refined sugar(s), simple sugars, intrinsic and extrinsic sugars, non-milk extrinsic sugars and caloric sweeteners.

The existence of numerous terms to define sugars and their indiscriminate use in different countries, substantially hampers the comparison of intake studies that have currently been published. For this very reason, when it comes to quantifying daily sugar consumption, individuals should only take added sugars into consideration, such as sucrose (white and brown sugar), saccharin, aspartame and other substitutes, excluding added sugar as an ingredient in precooked/processed foods.

Sugar and artificial sweetener consumption

In our country the sugar consumption patterns in foods have remained constant during recent years, whilst the consumption of soft drinks has tended to increase. The National Survey of Spanish Dietary Intake (ENIDE, 2010-11) estimates that 20% of calorie intake comes from carbohydrates known as sugars³. This consumption pattern is characterised by the high consumption of processed foods, with changes that affect fat and sugar content marking a notable shift away from the traditional Mediterranean diet⁴.

In this respect, there has been an exponential growth both in energy-rich foods and those that are supposedly low-calorie. Industry pressure plays a pivotal role in these food consumption patterns. The term 'sweetener' refers to those food additives which are able to mimic the sweetness of sugar and which usually provide less energy. Some are natural extracts whilst others are synthetic; in the case of the latter they are also known as artificial sweeteners. The use of non-caloric sweeteners, as a substitute for all or part of the sugar content in food and drink, has experienced its biggest growth over the past 35 years. However, the substitution of sugar with artificial sweeteners represents an insignificant calorie saving with regards to total daily calories, and can generate a 'false sense of security' that encourages counterproductive behaviour, such as the excessive consumption of 'low calorie' products and other foods with a high fat content. Following their appearance and in response to the interest in clarifying their beneficial effects (faced with different conditions such as obesity, diabetes, tooth decay, etc.) a multitude of studies have been carried out that, instead of supporting this hypothesis, seem to prove that their use is ineffective.

Therefore, today further research is needed to provide convincing evidence of their long-term effectiveness, as well as the absence of potential negative effects from their continued use. The general population, including many health professionals, often lacks correct knowledge on the particular characteristics of the different sweeteners available in the market, such as advising on and/or choosing a particular sweetener based on its properties⁵.

Not all calories are equal

In addition to the sweet flavour, sugars add a wide variety of favourable qualities to foods, such as their antimicrobial action, taste, smell and textures, as well as viscosity and consistency, which generate satiety. Although the sensation of fullness is greater for proteins, it is higher for sugar and carbohydrates than fats which are also the diet's greatest source of calories. By using the glycaemic index (GI) we can categorise foods based on the postprandial plasma glucose response to sucrose compared with a reference food

(glycaemic index = 100). Sucrose has a medium glycaemic index (≈ 65)⁶.

Studies on food induced thermogenesis showed that energy dissipated in the form of heat is lower following the digestion of fats ($\approx 7\%$), than after digesting carbohydrates ($\approx 12\%$) and proteins ($\approx 22\%$).

Furthermore, isotopic studies have confirmed the absence of significant 'de novo' lipogenesis in high carbohydrate diets. Glucose is stored in the liver and muscles in the form of glycogen. Excess glucose that is not used as immediate source of energy or for the synthesis of glycogen can be converted, via de novo lipogenesis, into fat which is stored in the adipocytes. However, this conversion is energetically costly. Astrup and Raben calculate that 68% more energy is needed to increase body fat by 1kg with carbohydrates than with fats, which confirms that 'it is difficult to increase body fat in subjects of normal body weight, especially through the overconsumption of carbohydrates'.

In contrast to what happens with fat consumption, where a strong correlation has been shown between excessive consumption and the increased risk of becoming overweight and/or obese; the relationship between the consumption of sugars included in foods or added to them and an increase in weight gain is less clear. Various cross-sectional studies have concluded that there is no association, or even that there is a negative association, between sugar consumption and weight gain.

However, there is widespread debate over whether increased sugar consumption via sugary drinks may have a more significant impact on body mass index (BMI). The CARMEN study (Carbohydrate Ratio Management in European National diets) compared the effects on body weight, and the lipid profile of isocaloric diets with high sugar and polysaccharide contents, in relation to diets with a high fat content over a period of 6 months. The results showed that both high sugar and high polysaccharide diets led to a significant reduction in body weight of 1.6 kg and 2.4 kg respectively, when compared with isocaloric diets with a higher fat content⁷.

Studies carried out in different countries, such as Australia and the UK, have observed how in spite of an increase in non-caloric drinks at the expense of sugary drinks, whether they contain sucrose, fructose or high fructose syrup, there has not been an associated reduction in the incidence of obesity. This phenomenon is called the 'Australian paradox' and suggests that there is no association between sugar consumption and the appearance of obesity in the general population⁸.

The importance of sucrose consumption at different times of day, during physical exercise and at different stages of life

Carbohydrates are important for our bodies to function properly and particularly for the brain, as brain

cells need a constant supply of glucose from the blood stream in order to maintain their integrity and functionality (140 g/day). When sucrose is included in the diet, in a balanced manner, it has important properties because it facilitates the rapid supply of glucose to the brain and muscles; it is an essential carbohydrate for cognitive function and physical activity. The consumption of a food or drink containing sucrose is associated with an improvement in mental agility, memory, reaction time, attention and the ability to solve mathematical problems, as well as a reduction in the feeling of tiredness, in young and elderly healthy people, and also in Alzheimer patients.

Sports drinks that contain sugar, minerals and water prevent dehydration, the depletion of glycogen reserves and delay the onset of tiredness and fatigue, as sugar, primarily, provides a direct supply of glucose to muscles and this prolongs exercise time. There is evidence that a high carbohydrate diet and the consumption of carbohydrates before and during exercise is beneficial, due to the increase in concentrations of hepatic glycogen and the maintenance of blood sugar concentrations.

The idea that sucrose may cause addiction in a similar way to 'abused drugs' and should therefore be included among addictive substances, is an opinion of little scientific basis. Available experimental evidence does not support the hypothesis that sugar and other highly palatable foods cause addiction. It is important to note that sugars are part of pleasurable foods and do not cause dependence⁹.

Stereotypes used in relation to sucrose consumption

The natural sugar content of foods cannot be distinguished from added sugar, although it is true that this added sugar mainly provides energy and no other essential nutrients. Furthermore, in the context of diet, sugar is not consumed in isolation and helps to make the diet more varied and appetising, enabling foods to be included that would perhaps otherwise not be consumed, thus contributing indirectly to the intake of other nutrients.

Interested in finding out about the possible relationship between high sugar intake and the decrease in micronutrient intake, we noted that the diet's nutrient density might be influenced by factors such as the high levels of sugar added to food. It seems that this nutritional dilution, which is produced by adding sugar to food, is in general, not very significant and is often offset by the fortification in micronutrients that we usually can find in many sugary products. Following a detailed analysis of the studies published on this subject, it has been found that there is no clear evidence of the hypothetical micronutrient dilution that could occur by adding sugar to the diet.

On the other hand, given that the addition of sugar to the diet doesn't seem to highlight any remarkable advantages from the point of view of micronutrient intake; it seems reasonable to promote the moderate consumption of sugary foods and drinks, to avoid them becoming an extra important energy source.

Sucrose consumption and illnesses

In developed countries tooth decay has ceased to be a problem due to the widespread use of adequate oral hygiene, exposure to fluoride and regular visits to the dentist. Sweet foods should be consumed with main meals (meals, snacks..) and after that the teeth should be brushed.

In relation to the nutritional recommendations, based on the evidence for treating and preventing diabetes, the total amount of carbohydrates consumed is more important than the type. Nowadays diabetics can consume sucrose (sugar) and foods that contain sugar as long as they are eaten as part of a healthy diet and there is appropriate medical supervision. Sugar consumption does not cause the onset of diabetes, however, becoming obese, an unbalanced diet and a lack of exercise have a lot to do with this condition¹⁰.

Although there is a clear relationship between fat intake and weight gain, the role of carbohydrates and, more specifically, the role of sucrose in developing obesity is more controversial. A huge part of this controversy is attributed to the growing demand for sugary drinks and the possible dietary calorie intake associated with their consumption. In spite of the publication of numerous studies and communications on this subject in recent years, there are still many uncertainties about the role that high sugar diets play in the rising incidence and prevalence of obesity. In the dietary treatment of obesity the key objective is to reduce energy intake, whereas macronutrient distribution is less important. Once the desired weight has been reached, diets that limit fats and that are high in carbohydrates are the preferred method to maintain the weight lost. Although low carbohydrate diets may exercise a pathophysiological mechanism in short term weight loss, it is important to point out that the long term effects of continuing to follow these diets are not known.

There is insufficient evidence available on the association between monosaccharide intake (glucose and fructose), as well as disaccharides intake (sucrose) and different types of cancer. In certain cases there is clear evidence of no association.

Consumption recommendations: numerous recommendations and a lack of consensus

Some mathematical models, which are decidedly pessimistic, point to the possibility that we are at a time

when the life expectancy curve, which has always been rising in industrialised countries in recent years, has for the first time reversed its upward trend and is levelling out. If we want our strategy to succeed we should move away from any repressive, prohibitive temptation, that undermines our creativity, lifestyles and the recreational role that food plays in our society. On the other hand, what is clear is that we can't move forward without considering that there are no 'guilty' foods, and as Paracelsus said before me (a long time before me) even poison depends on the amount consumed.

Recommendations by international organisations such as the FAO/WHO recommend that simple carbohydrate consumption (sugars) should be lower than 10% of the diet's energy value, acknowledging that this figure is 'controversial'. Although public health strategies should be developed in order to reduce the excessive consumption of sugary drinks, as part of a healthy lifestyle and the best nutritional advice for the general population could be to eat a balanced and varied diet with foods and nutrients that come from different sources, combining this diet with exercise and physical activity.

The Mediterranean diet is currently the eating pattern that should be promoted or revived amongst the Spanish people. This diet is characterised by its low calorie density, as it is rich in fruit, vegetable, pulses, fish and olive oil, which allows sugar to play a role in energy levels and the palatability of this diet¹¹.

References

1. FAO/WHO (Food and Agriculture Organization / World Health Organization), 1998 Carbohydrates in human nutrition. (FAO Food and Nutrition Paper - 66) Rome: FAO.
2. EFSA Panel on Dietetic Products, Nutrition, and Allergies (NDA); Scientific Opinion on Dietary Reference Values for carbohydrates and dietary fibre. *EFSA Journal* 2010; 8 (3): 1462 [77 pp.]. doi:10.2903/j.efsa.2010.1462. Available online: www.efsa.europa.eu
3. Valoración de la Dieta Española de acuerdo al Panel de Consumo Alimentario. Ministerio de Agricultura Pesca y Alimentación (MAPA)/Fundación Española de la Nutrición (FEN). Madrid: Ministerio de Medio Ambiente y Medio Rural y Marino, 2008. Visitado en: http://www.magrama.gob.es/es/alimentacion/temas/consumo-y-comercializacion-y-distribucion-alimentaria/valoracion_panel_tcm7-7983.pdf
4. Agencia Española de Seguridad Alimentaria y Nutrición (AESAN), 2011. Encuesta Nacional de Ingesta Dietética Española 2011. http://www.aesan.msc.es/AESAN/docs/docs/notas_prensa/Presentacion_ENIDE.pdf
5. Wiebe N, Padwal R, Field C, Marks S, Jacobs R, Tonelli M. A systematic review on the effect of sweeteners on glycemic response and clinically relevant outcomes. *BMC Med* 2011; 9: 123.
6. Atkinson FS, Foster-Powell K, Brand-Miller JC. International Tables of Glycemic Index and Glycemic Load Values: 2008. *Diabetes Care* 2008; 31: 2281-3.
7. Saris WH, Astrup A, Prentice AM, Zunft HJ, Formiguera X, Verboeket-van de Venne WP et al. Randomized controlled trial of changes in dietary carbohydrate/fat ratio and simple vs. complex carbohydrates on body weight and blood lipids: the CARMEN study. The Carbohydrate Ratio Management in European National diets. *J Obes Relat Metab Disord* 2000; 24: 1310-8.
8. Barclay A, Brand-Miller J. The Australian Paradox: A Substantial Decline in Sugars Intake over the Same Timeframe that Overweight and Obesity Have Increased. *Nutrients* 2011; 3: 491-504.
9. Gearhardt Ashley N, Grilo CM, DiLeone RJ, Brownell KD, Potenza MN. Can food be addictive? Public health and policy implications. *Addiction* 2011; 106 (7): 1208-11.
10. American Diabetes Association. Standards of medical care in diabetes—2013. *Diabetes Care* 2013; 36 (Suppl. 1): S11-66.
11. Sociedad Española de Nutrición Comunitaria (SENC). Objetivos nutricionales para la población española. *Rev Esp Nutr Comunitaria* 2011; 17 (4): 178-199.

Food as a source of mono- and disaccharides; biochemical and metabolic aspects

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Abstract

Carbohydrates are important and necessary components of human diet. Although they primarily play an energetic function, they also have structural and functional roles. According to the European Food Safety Authority, carbohydrate intake should range between 45 and 60 percent of the energy in adults and children older than one year of age. An important part of carbohydrates available in foods are mono and disaccharides, commonly referred to as sugars. Dietary sources of sugars include fruits, fruit juices, vegetables, milk and milk products, and foods containing added sucrose and starch hydrolysates. Despite their importance in daily life, there is currently no clear and adequate terminology on the various types of carbohydrates, particularly sugars. Nor are there available sugar intake recommendations or food composition tables. Without these recommendations or reference values, dietary unbalances might occur, which subsequently may end in the premature onset of most chronic or degenerative diseases of our society. The aims of the present work are: to classify dietary carbohydrates, to define the biochemical and common terms for sugars, to explain their nutritional value and their metabolism as well as their food sources and to carry out a SWOT (Strengths, Weaknesses, Opportunities, Threats) analysis about the nomenclature and dietary intakes of sugars.

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Key words: Carbohydrates. Sugars. Food intake. Food composition tables.

LOS ALIMENTOS COMO FUENTE DE MONO Y DISACÁRIDOS; ASPECTOS BIOQUÍMICOS Y METABÓLICOS

Resumen

Los hidratos de carbono constituyen una parte importante y necesaria en la alimentación humana. Aunque desempeñan una función primordialmente energética, también tienen funciones estructurales y funcionales. Según la Agencia Europea para la Seguridad Alimentaria, la ingesta de hidratos de carbono debe oscilar entre un 45 y 60% de la energía en adultos y niños mayores de un año. Una parte importante de los hidratos de carbono disponibles en los alimentos la componen los mono y disacáridos, comúnmente denominados azúcares. Las principales fuentes dietéticas de azúcares son las frutas, los zumos de fruta, algunos productos vegetales, la leche y los productos lácteos, y los alimentos que contengan sacarosa añadida e hidrolizados de almidón. A pesar de ser fundamentales en nuestra vida diaria, no existe una terminología adecuada y clara sobre los diversos tipos de hidratos de carbono, y de forma muy especial de los azúcares. Tampoco en lo referente a las recomendaciones de ingesta y contenido en los alimentos. Sin recomendaciones ni valores de referencia, pueden producirse desajustes alimentarios, que pueden asociarse con la aparición precoz de la mayor parte de las enfermedades crónicas o degenerativas en nuestra sociedad. Los objetivos de este trabajo son: clasificar los hidratos de carbono presentes en los alimentos, establecer definiciones claras sobre todos los términos bioquímicos y comunes relacionados con los azúcares, explicar su valor nutricional y describir su metabolismo, así como las fuentes alimentarias que contienen tanto mono como disacáridos y, finalmente, realizar un análisis DAFO (Debilidades, Amenazas, Fortalezas y Oportunidades) sobre la nomenclatura y las ingestas de azúcares.

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Palabras clave: Hidratos de carbono. Azúcares. Ingesta de alimentos. Tablas de composición de alimentos.

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List of abbreviations

1,3-BPG: 1,3-bisphosphoglycerate.
ADP: Adenosine diphosphate.
AMP: Adenosine monophosphate.
ATP: Adenosine triphosphate.
CO₂: Carbon dioxide.
SWOT: Strengths, Weaknesses, Opportunities and Threats.
DHAP: Dihydroxyacetone phosphate.
EFSA: European Food Safety Authority.
FIP: Fructose-1-phosphate.
FAO/WHO: Food and Agriculture Organisation of the United Nations and the World Health Organisation.
GA: Glyceraldehyde.
GAP: Glyceraldehyde-3-phosphate.
GAPDH: Glyceraldehyde-3-phosphate dehydrogenase.
GLUT: Glucose Transporters.
GOT: Glutamate-oxaloacetate transaminase.
GTP: Guanosine triphosphate.
HK: Hexokinase. GI: Glycaemic Index.
K⁺: Potassium.
Mg⁺⁺: Magnesium.
NAD⁺: Oxidised nicotinamide adenine dinucleotide.
NADH: Reduced nicotinamide adenine dinucleotide.
PEP: Phosphoenolpyruvate.
PEPCK: Phosphoenolpyruvate carboxykinase.
PFK-1: Phosphofructokinase-1.
P_i: Inorganic phosphate.
TIM or TPA: Triose phosphate isomerase.
UTP: Uridine triphosphate.

Introduction

The health of individuals is determined by genetic and environmental factors, of which the most important is diet. An adequate diet should satisfy individual daily nutritional requirements, incorporating cultural and gastronomic values, and personal satisfaction. Recent studies in the field of nutrition show that dietary imbalances are the main cause of the premature development of the majority of chronic and degenerative diseases in today's society. Therefore, the possibility of making better eating patterns part of our lifestyle habits and reducing the overall risk factors in the general population, is in our hands.

Carbohydrates are the world's main source of food energy, particularly in developing countries. Carbohydrates should provide between 50 and 55% of total dietary energy and should also be evaluated for their potential energy, their sweetness and their high fibre content^{1,2}. The European Food Safety Authority (EFSA) has recently advised that carbohydrate intake should range from 45 to 60% for both adults and healthy children over one year of age³.

Carbohydrates are found in most cereals and tubers, as well as legumes, fruit and vegetables, and contribute to the

texture and flavour of these foods. They are directed to and absorbed in the small intestine and, to a lesser degree, some of them are partially fermented in the large intestine.

A significant proportion of dietary carbohydrates are mono- and disaccharides, commonly referred to as sugars. The main dietary sources of sugar are fruit, fruit juices, certain vegetables, milk and certain dairy products and foods which have added sucrose or starch hydrolysates (for example, glucose syrup or with high levels of fructose) such as carbonated drinks, pastries, sweets and confectionary¹⁻³.

The aims of this study are: to classify dietary carbohydrates, to define clearly all the biochemical and common terms for sugars, to describe their nutritional value and their metabolism, as well as food sources which contain both mono- and disaccharides, and finally to carry out a SWOT analysis (Strengths, Weaknesses, Opportunities and Threats) on the nomenclature and intake of sugars.

TERMINOLOGY AND CLASSIFICATION OF CARBOHYDRATES

Dietary carbohydrates can be in the form of complex molecules (polymers or polysaccharides) or more simple molecules, commonly referred to as sugars, monomers (monosaccharides) or dimers (disaccharides). Of all the dietary sugars, the most important from a nutritional point of view are: glucose, fructose, galactose, maltose, lactose, sucrose and trehalose. There are many different classifications in the literature, but some of them can confuse consumers. Table I, below, details the classification of carbohydrates from the Food and Agriculture Organisation of the United Nations and the World Health Organisation (FAO/WHO) amended by the EFSA^{1,3}. The latter has chosen to classify carbohydrates in two categories, according to how they raise blood sugar, called glycaemic carbohydrates and dietary fibre. The first category includes sugars, maltodextrins, starches and glycogen. Dietary fibre includes all dietary components which are not hydrolyzed in the small intestine, at least for the most part, i.e. non-amyloid polysaccharides (celluloses, hemicelluloses, pectins and hydrocolloids —gums, mucilages and glucans—), resistant oligosaccharides (fructooligosaccharides and galactooligosaccharides and other resistant oligosaccharides), type IV resistant starches and lignin (Table I)³.

Classification of carbohydrates

Monosaccharides

Glucose

D-glucose is a reducing sugar that circulates freely in the blood of all mammals. It is absorbed by all cells

Table I <i>Classification of carbohydrates by the FAO/WHO amended by the EFSA³</i>				
<i>Class (DP)</i>	<i>Subgroup</i>	<i>Components</i>	<i>Monomers</i>	<i>Digestibility*</i>
<i>Sugars (1.2)</i>	Monosaccharides	Glucose Galactose Fructose		+ + +
	Disaccharides	Sucrose Lactose Trehalose Maltose	Glucose, Fructose Glucose Galactose Glucose Glucose	+ ± + +
<i>Oligosaccharides (3-9)</i>	Maltooligosaccharides	Maltodextrins	Glucose	+
	Other oligosaccharides	α-Galactosidases (GOS) Fructooligosaccharides (FOS) Polydextroses Resistant dextrins	Galactose, Fructose Fructose, Glucose Glucose Glucose	– – – –
<i>Polyalcohols</i>	Maltitol, Sorbitol Xylitol, Lactitol			+ or –
<i>Polysaccharides (< 9)</i>	Starches	Amylose Amylopectin Modified starches Resistant starches Insulin	Glucose Glucose Glucose Glucose Fructose	± ± – – –
	Other polysaccharides	Cellulose Hemicellulose Pectins Other hydrocolloids (gums, mucilages, β glucans)	Glucose Variable Uronic acids Variable	– – – –
<i>Related substances</i>	Lignin			

DP: Degree of polymerization.

* Digestibility in the small intestine, + digestible, ± mainly digestible, + or – partially digestible, - non-digestible.

by means of specific transporters. Glucose is found in most fruits and many vegetables. It is an abundant reserve polymer in animals (glycogen) and in plants (starch). Most glucose is present as a non-digestible polymer (cellulose). Glucose can be produced through starch hydrolysis by enzymes. Also, some glucose can be isomerised to fructose with the use of glucose isomerase. Both glucose and mixtures of glucose and fructose in the form of syrups can be added to various foods, in particular sweets, confectionary and pastries, as well as soft drinks and other products, like sweeteners^{2,4}.

Fructose

It is the sugar which has the most sweetening power. It is absorbed passively, more slowly than glucose. It is abundant in fruit. There are fructose polymers, both natural (inulin) and synthetic (fructooligosaccharides), but these compounds don't contribute significantly to the sweetness of foods and are poorly digested, so they behave like soluble fibre^{2,4}.

Galactose

Galactose forms part of glycolipids and glycoproteins of cell membranes, especially neurons. It is synthesised by the mammary glands in mammals to produce lactose, therefore, the majority of dietary galactose comes from consuming lactose in milk. It is absorbed in the intestines together with glucose using the same transporter^{2,4}.

Disaccharides

Sucrose

It is the sugar par excellence. It consists of one fructose and one glucose molecule linked by a glycosidic bond. It is hydrolysed in the intestine by the action of the sucrase-isomaltase enzyme complex. It is a non-reducing sugar which is very soluble in water and crystallizes easily. It is extracted industrially from sugar cane and sugar beet. It is also used to sweeten foods, to improve the sour and bitter taste of many of them and to

preserve them by increasing osmotic pressure, which prevents the growth of many microorganisms^{2,4}.

Maltose

It is a reducing sugar composed of two glucose molecules linked by a glycosidic bond, present in some fruits where it accounts for 15% of the total sugars. Its sweetness is 50% when compared to sucrose. It is hydrolysed in the intestine by the action of maltase. Maltose forms part of maltodextrins and glucose syrups, and is an ingredient used in many foods as a source of energy. It is produced industrially through the hydrolysis of rice or corn starch^{2,4}.

Lactose

It is the sugar in milk and is formed by the union of two galactose and glucose molecules linked by a glycosidic bond. It is hydrolysed by the action of lactase, an enzyme whose activity decreases from 2 or 3 years of age in most humans. It is extracted, in purified form, from cow's milk and whey, it has a low solubility in water and its sweetness is only 40% relative to sucrose. In infants and young children, lactose not only provides energy, but also aids the development of gut microbiota (bifidobacteria and other lactic acid bacteria), increases the bioavailability of calcium and other mineral elements and provides galactose which is directly usable for developing the nervous system^{2,4}.

Polysaccharides

Starch is abundant in the plant world and it is the substance that we refer to when, in terms of nutrition, we are talking about 'complex polysaccharides'. It is a polysaccharide consisting of glucose bonds in position α 1-4 and branches in position α 1-6. The partial hydrolysis of starch leads to the industrial production of dextrins or maltodextrins, which are formed by glucose units of varying size with some branches. Its sweetening power depends on the degree of hydrolysis. Only dextrins that have an increased reducing power (a degree of dextrose equivalent to 25-45) contribute, to some extent, to the sweetness of foods.

Glycogen only exists in the animal world (liver and muscle) and, as with starch, it does not contribute to the sweet flavour of foods, as well as being a polysaccharide consisting of glucose bonds in position α 1-4 and abundant branches in position α 1-6.

Cellulose and *hemicellulose* form part of the cell wall of all vegetables. They are polysaccharides formed by glucose bonds in position β 1-4.

Pectins are part of the middle lamella of plant cell walls. They are polysaccharides of galacturonic acid in position 1-4, with the carboxyl groups which are often methoxylated. They are plentiful, especially in fruit.

Gums and *mucilages* are especially found in seeds and pulses. They are complex polysaccharides in terms of the type of saccharide component, branching and degree of polymerisation. All of these polysaccharides have the common feature that they are not broken down by digestive enzymes and make up a large part of what is known as dietary fibre^{2,4}.

Carbohydrate terminology

The two main categories of digestible carbohydrates, and therefore glycaemic, are sugars and starch. Both, in general, present problems when it comes to being defined and characterised, causing complications when their daily intake and the impact they have on health need to be examined^{3,5}.

Sugars

There are many terms used to describe sugars and their components, like for example: sugar(s), total sugars, total available glucose, free sugars, added sugars, refined sugar(s), simple sugars, intrinsic and extrinsic sugars, non-milk extrinsic sugars and caloric sweeteners.

The existence of many of these different terms and their use in various countries has meant that it's not possible to compare the different intake studies currently being carried out. In the same way, the possibilities of comparing food intakes and making recommendations about them, and establishing the relationship between food consumption and risk factors, is limited.

The different terms and their current definition or general meaning are shown in table II⁴.

Starch

The term starch refers to the total starch present in food. However, starch can be subdivided by the degree and extent to which they are digestible. Resistant starches are not digestible in the small intestine and, therefore, should not be considered as digestible carbohydrates, but so far, there is no explicit separation of the intake measurements that were conducted on the populations. There are three types of starch in the diet, those which are digested quickly that we can find in recently cooked foods and foods that are rich in starch, those that are poorly digested which are found in pasta and cereals, and finally resistant starches⁵.

Table II <i>A general listing of the different terms used to describe sugars⁴</i>	
<i>Term</i>	<i>Description</i>
<i>Total sugars</i>	All mono- and disaccharides in food, whether they are natural and/or added during their production.
<i>Added sugars</i>	Sugars and syrups which are added to food during production or preparation. In accordance with the US Department of Agriculture, only mono- and disaccharides. On the other hand, the US Economic Research Service includes oligosaccharides derived from corn syrup in their definition. Other: all refined sugars (for example, sucrose, maltose, lactose, glucose and dextrin) used as ingredients in processed foods.
<i>Free sugars</i>	Traditional: any sugar in food which is not combined, including lactose. Recent: all monosaccharides and disaccharides added to food by the manufacturer and consumer, plus sugars naturally found in honey, syrups and fruit juices.
<i>Refined sugars</i>	For most European countries sucrose, fructose, glucose, hydrolysed starch (glucose syrup, high fructose corn syrup) and other isolated sugar preparations, such as the components of foods used during the manufacture and preparation of foods.
<i>Non-milk extrinsic sugars</i>	Total sugars, except for lactose in milk and milk products, and sugars present in the cellular structures of fruit and vegetables.
<i>Sugar (without 's')</i>	It has many meanings, in certain cases it only refers to sucrose, while in others it includes 'all monosaccharides and disaccharides; or 'any free monosaccharide or disaccharide in food'.
<i>Caloric sweeteners</i>	Sweeteners which are consumed directly as well as an ingredient in food, such as sucrose, from refined sugar candy and sugar beet, honey, dextrose, edible sugars and commercial sweeteners, as well as oligosaccharides.

The nutritional value of carbohydrates

Carbohydrates are the cheapest and most important source of energy. Even though they are not essential in nature as they can be synthesized by the body, they should form part of our diet and should make up 45-60% of energy intake³. Their considerable importance rests on the fact that they are the primary energy source for all countries; 50% in developed countries and 90% in developing countries. The energy contribution of carbohydrates is 4.1 kcal/g. Although, at first glance one might think that the sole function of carbohydrates is as an energy source for human and animal metabolism, sugars have other significant structural functions in living creatures, such as important components of membrane antigens and proteins secreted by many cells.

Complex carbohydrates are part of foods like cereals or their derivatives, such as bread, pasta, corn tortillas, etc., as well as pulses, which also contain many other extremely important nutrients for daily consumption like fibre, vitamins, proteins and minerals. On the other hand, refined sugars themselves don't contribute to the value of other nutrients, but they supply large amounts of energy to the diet without helping meet the daily nutritional demands of other nutrients. However, the fact remains that simple sugars, in the context of a moderate and balanced diet, contribute to the intake of other nutrients by making them more appetising. Carbohydrates are also found in high volume foods and those with a more complex structure, they slow down the digestive process and make the absorption of

glucose a slow and gradual process, preventing postprandial hyperglycaemia. However, simple sugars are absorbed rapidly and cause hyperglycaemia^{1,3}.

The concept of glycaemic index (GI)

Glycaemic index is defined as the incremental area under the blood glucose response curve of a 50g portion of carbohydrates absorbable from a test food, expressed in a response rate for the same quantity of glucose ingested by the same subject³. There are research studies which describe how meals that contain low GI foods reduce both postprandial blood sugar and insulin response. Some epidemiological studies indicate that a low GI diet is associated with a reduced risk of developing type II diabetes in humans. Clinical trials on normal, diabetic and hyperlipidaemic subjects show that low GI diets reduce average blood sugar concentrations, insulin secretion and serum triglycerides in individuals with hypertriglyceridaemia.

Therefore low GI foods raise the amount of carbohydrates that enter the colon and increase fermentation and the production of short chain fatty acids. Table III shows the GI values of certain foods⁶.

CARBOHYDRATE METABOLISM

The glucose used by the tissues comes from starch, sucrose and lactose in the diet and from the body's

stores of liver and muscle glycogen, or from hepatic or renal synthesis, via gluconeogenic precursors such as the carbon skeleton of certain aminoacids, glycerol and lactate; these sources enable the concentration of blood sugar to be maintained within the appropriate ranges.

The balance between oxidation, biosynthesis and glucose storage depends on the hormonal and nutritional state of the cell, tissue and organism. The predominant metabolic pathways of glucose vary in different types of cell depending on physiological demand. Thus, the liver plays a fundamental role in glucose homeostasis in the body. Glucose can be completely oxidised by the hepatocytes to obtain energy and can be stored in the form of glycogen or supply carbons for fatty acid and aminoacid synthesis.

The heart and skeletal muscles can completely oxidise glucose or store it in the form of glycogen. Glucose can be partially broken down in the fatty

tissues to provide glycerol, necessary for triglyceride synthesis, or oxidised completely and supply two units of carbon (acetyl- CoA) for fatty acid synthesis.

The brain depends on a continuous supply of glucose, which it can oxidise completely until it is CO₂ and water. On the other hand, erythrocytes have a limited capacity for oxidising glucose as they don't have mitochondria, but energy production depends entirely on such metabolic fuel partially oxidising it to lactate via glycolysis. Other specialised cells, such as corneal cells, the lens, the retina, leucocytes, testicular cells and renal medulla cells, are predominantly glycolytic⁵.

Most mammalian cells capture glucose, as well as other sugars and polyalcohols, through membrane transport proteins which are known as glucose transporters (GLUT *Glucose Transporters*). So far thirteen members of this family are known, which are characterised by

Table III
The glycaemic index of 62 common foods^a

<i>Foods high in carbohydrates</i>		<i>Breakfast cereals</i>		<i>Fruit and their derivatives</i>		<i>Vegetables</i>	
White bread, wheat	75 ± 2	Cereals	61 ± 6	Apple	36 ± 2	Boiled potato	78 ± 4
Brown bread	74 ± 2	Wheat crackers/biscuits	69 ± 2	Orange	43 ± 3	Freshly chopped potato	87 ± 3
Special grain bread	53 ± 2	Oatmeal	55 ± 2	Banana	51 ± 3	Fried potato	63 ± 5
Unleavened bread, wheat	70 ± 5	Rolled oats	79 ± 3	Pineapple	59 ± 8	Boiled carrot	39 ± 4
Roti, wheat	62 ± 3	Rice porridge	78 ± 9	Mango	51 ± 5	Boiled sweet potato	63 ± 6
Chapatti	52 ± 4	Millet	67 ± 5	Watermelon	76 ± 4	Boiled pumpkin	64 ± 7
Corn tortilla	46 ± 4	Muesli	57 ± 2	Dates	42 ± 4	Plantain	55 ± 6
Cooked white rice	73 ± 4			Peach	43 ± 5	Boiled taro	53 ± 2
Cooked brown rice	68 ± 4			Strawberry jam	49 ± 3	Vegetable soup	48 ± 5
Barley	28 ± 2			Apple juice	41 ± 2		
Sweet corn	52 ± 5			Orange juice	50 ± 2		
Spaghetti (white flour)	49 ± 2						
Spaghetti, serving	48 ± 5						
Rice noodles	53 ± 7						
Japanese noodles (Udon)	55 ± 7						
Couscous	65 ± 4						
<i>Dairy products and alternatives</i>		<i>Pulses</i>		<i>Snack products</i>		<i>Sugars</i>	
Whole milk	39 ± 3	Chickpeas	28 ± 9	Chocolate	40 ± 3	Fructose	15 ± 4
Skimmed milk	37 ± 4	Kidney beans	24 ± 4	Popcorn	65 ± 5	Sucrose	65 ± 4
Ice cream	51 ± 3	Lentils	32 ± 5	Crisps	56 ± 3	Glucose	103 ± 3
fruit yoghurt	41 ± 2	Soya	16 ± 1	Soft drinks	59 ± 3	Honey	61 ± 3
Soya milk	34 ± 4			Rice cakes	87 ± 2		
Rice milk	86 ± 7						

Data expressed in g/100g or g/100ml in mean ± standard error of mean, adapted from Atkinson et al. 2008 *Diabetes Care* 31: 2281-3.

their twelve transmembrane fragments and a series of aminoacids that are well-preserved in the different species, which are implicated directly in their function.

The various GLUT isoforms differ in their tissue location, their kinetic properties and whether they are dependent or not on insulin. In fact, glucose absorption is regulated by the expression and location of the various GLUT in different cells and in different metabolic states. GLUT2, 3 and 4 are good examples to illustrate the regulation of glucose absorption by these type of transporters. So, GLUT3 is the main glucose transporter in the brain and it has a K_m (1 mM) far below normal blood sugar levels (4-8 mM), which indicates that it constantly transports glucose into the cells which express it.

On the other hand, GLUT2 has a high K_m (15-20 mM) therefore the cells which express it only absorb glucose when blood sugar is elevated. This transporter is expressed, among others, in the intestinal and pancreatic β cells in which glucose entry is a sign that blood sugar levels are elevated and that the necessary mechanisms for insulin release should be triggered (adenosine triphosphate (ATP) via the breakdown of glucose with the consequent inhibition of the K^+ -ATP channel, activating calcium entry and as a result the release of insulin from endosomes into the blood). Finally, GLUT4 is a transporter which is expressed in the muscles and fatty tissue. The location of this transporter in the cell and, therefore its activity, depends on blood insulin levels, as the latter is needed for the receptor, which is normally found in the intracellular vesicles, is transported to the plasma membrane⁷.

Glycolysis

Glycolysis is the central pathway of glucose catabolism. It breaks down glucose with a dual purpose: to obtain energy in the form of ATP and to supply precursors for the biosynthesis of cell components. Glycolysis occurs in all mammal cells and is the exclusive or almost exclusive source of energy for certain cells and tissues, such as erythrocytes, the renal medulla, the brain and the testicles.

Glycolysis takes place entirely in the cytoplasm and during the process one glucose molecule is divided into two pyruvate molecules. This pathway can be separated into two phases: the preparatory phase, in which glucose is converted to two triose phosphate molecules and the pay off phase, where two triose molecules are converted to two pyruvate molecules and ATP and NADH are obtained (reduced nicotinamide dinucleotide) (Fig. 1).

Preparatory phase

During this phase glucose is modified to produce fructose-1,6-bisphosphate which is divided to form

two triose phosphates with ATP consumption. The preparatory phase of glycolysis can be divided into the following stages (Fig. 1):

- a) *Glucose phosphorylation.* During this irreversible reaction glucose is phosphorylated by a kinase at the expense of ATP to become glucose-6-phosphate. The kinase which catalyses glucose phosphorylation in all cells is hexokinase (HK). Like all kinases, it needs ATP and magnesium (Mg^{++}). HK is not specific for glucose and can therefore phosphorylate other sugars, although it does have a high affinity for glucose (K_m 100 mM).
- b) *Conversion of glucose-6-phosphate to fructose-6-phosphate.* In the following reaction, catalysed by phosphohexose isomerase (phosphoglucose isomerase), glucose-6-phosphate is converted to fructose-6-phosphate. It is the first reversible stage of the pathway. Phosphohexase isomerase also needs Mg^{++} as a cofactor and is specific for glucose-6 phosphate and fructose-6-phosphate.
- c) *Formation of fructose-1,6-bisphosphate.* Fructose-6-phosphate is phosphorylated, at the expense of ATP and Mg^{++} , to be converted to fructose-1,6-bisphosphate by another kinase, the phosphofructokinase-1 (PFK-1). It is called PFK-1 to distinguish it from phosphofructokinase-2 which catalyses the production of fructose-2,6-bisphosphate from fructose-6-phosphate.
- d) *The breakdown of fructose-1,6-bisphosphate.* Fructose-1,6-bisphosphate is divided into two trioses, glyceraldehyde-3-phosphate (GAP) and dihydroxyacetone phosphate (DHAP). This reaction is catalysed by fructose-1,6-bisphosphate aldolase, usually known simply as aldolase.
- e) *Interconversion of the triose phosphates.* Only one of the trioses, GAP, can be broken down through glycolysis, therefore two trioses are isomerised to GAP in a reaction catalysed by triose phosphate isomerase (TIM).

Pay-off phase

In the pay-off phase two GAP molecules are converted to a pyruvate and the energy from the broken down glucose is preserved in the form of ATP and reducing power in the form of NADH. This phase is divided into the following stages:

- a) *Oxidation of glyceraldehyde-3-phosphate.* GAP is converted to 1,3-bisphosphoglycerate (1,3-BPG) in a reaction catalysed by glyceraldehyde-3-phosphate dehydrogenase (GAPDH). This enzyme requires inorganic phosphate (P_i) and NAD^+ as cofactors.

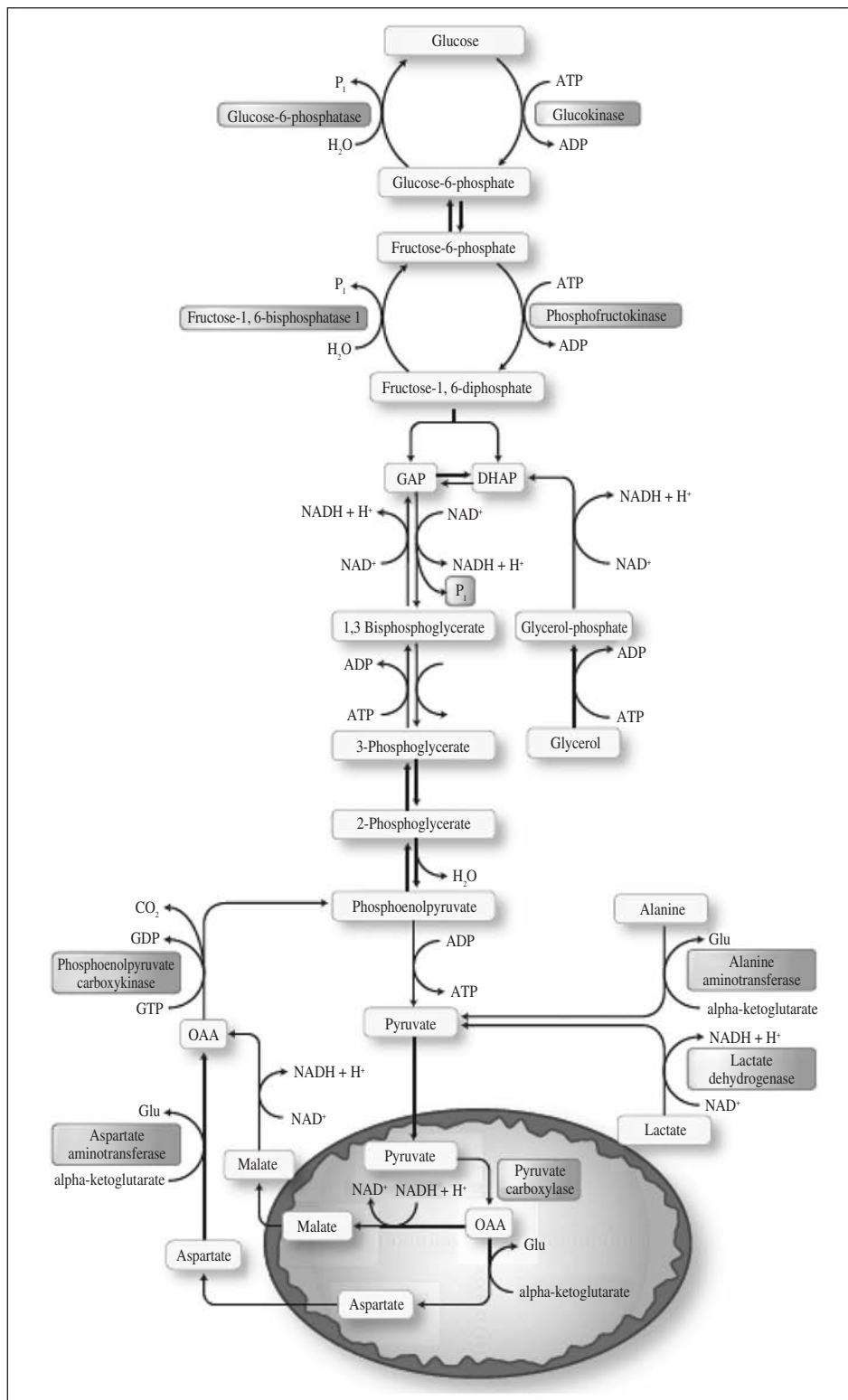


Fig. 1.—Overall diagram of glycolysis and gluconeogenesis. DHAP: dihydroxyacetone phosphate; GAP: glyceraldehyde-3 phosphate; OAA: oxaloacetate. Taken from Gil, A. Tratado de Nutrición, 2.^a Ed. Vol. I, Editorial Panamericana, Madrid, 2010.

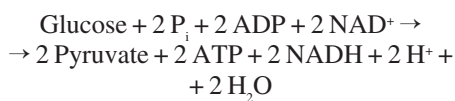
- b) The formation of ATP from 1,3-bisphosphoglycerate. In the following reaction, catalysed by phosphoglycerate kinase, 1,3-BPG is converted to 3-phosphoglycerate and ATP is synthesised. It is a substrate level phosphorylation reaction, during which 1,3-bisphosphoglycerate gives up

its phosphate, which is energy-rich, to adenosine diphosphate (ADP). This is a reaction which is reversible in the cell and requires Mg^{++} as a cofactor.

- c) Conversion of 3-phosphoglycerate to 2-phosphoglycerate. 3-phosphoglycerate is isomerised, in a

reversible way, to 2-phosphoglycerate mutase which requires Mg^{++} as a cofactor. The reaction occurs in two stages. During the first of them the enzyme, phosphorylated into a histidine residue, transfers phosphate to the C_2 hydroxyl of 3-phosphoglycerate, forming 2,3-bisphosphoglycerate. In the following stage 2,3-bisphosphoglycerate transfers phosphate to the enzyme C_3 and releases the phosphorylated enzyme and the 2-phosphoglycerate.

- d) *Phosphoenolpyruvate formation.* The 2-phosphoglycerate is dehydrated and forms phosphoenolpyruvate (PEP), which is an 'energy-rich' enol phosphate, in a reversible reaction catalysed by enolase.
- e) *The synthesis of pyruvate* PEP transfers its phosphate to ADP in a reaction catalysed by pyruvate kinase, which requires Mg^{++} y K^+ (potassium), in order to form pyruvate.
- f) *Glycolysis balance.* In the breakdown of glucose, via the glycolytic pathway, two pyruvate molecules, two molecules of ATP and two of NADH are obtained. Although four ATP molecules are obtained, two are consumed in the formation of fructose-1,6-bisphosphate. Therefore, the net balance of the reaction is:



Gluconeogenesis

Gluconeogenesis is the pathway through which glucose is generated from non-glucidic precursors. The importance of this pathway comes from the fact that certain tissues and organs (the central nervous system, renal medulla, lens, retina, testicles and erythrocytes) need a permanent supply of glucose (Fig. 1).

Phosphoenolpyruvate formation from pyruvate

The first stage of gluconeogenesis is the conversion of pyruvate to PEP. The glycolytic reaction is irreversible, given that it has a very negative standard free energy change and to reverse it, a detour would be needed using two enzymes from a different location: pyruvate carboxylase, which is located in the mitochondria, and phosphoenolpyruvate carboxykinase (PEPCK), which is cytosolic.

As a consequence, the pyruvate should initially be transported to the mitochondria where the pyruvate carboxylase will catalyse its conversion to oxaloacetate. This enzyme requires biotin, ATP and carbon dioxide (CO_2).

Oxaloacetate should leave the mitochondria. However, it doesn't have a transporter in the mitochondrial membrane, therefore it has to be converted to malate or aspartate so that it can be transported. In order to convert it to malate, oxaloacetate is reduced to mitochondrial malate dehydrogenase, using NADH as a reducer. Malate enters the cytosol and is oxidised by cytosolic malate dehydrogenase using NAD^+ as an acceptor and in this way, as well as oxaloacetate, NADH is obtained for the reduction which takes place during an earlier reaction catalysed by GAPDH.

Oxaloacetate can also be converted to aspartate by mitochondrial glutamate-oxaloacetate transaminase (GOT); the aspartate enters the mitochondria and because of cytosolic glutamate-oxaloacetate transaminase it converts to oxaloacetate.

Once it is in the cytosol, oxaloacetate is decarboxylated by PEPCK which gives rise to PEP. This enzyme requires Mg^{++} and guanosine triphosphate (GTP) as a phosphate donor.

Conversion of fructose-1,6-bisphosphate to fructose-6-phosphate

Fructose-6-phosphate is produced by a hydrolytic reaction, during which inorganic phosphate is released, catalysed by fructose-1,6-bisphosphatase which requires Mg^{++} as a cofactor. Fructose-1,6-bisphosphatase is the most important control point of the gluconeogenic pathway, it is activated by ATP and citrate and is inhibited by adenosine monophosphate (AMP) and fructose-2,6-bisphosphate.

Extraction of free glucose

The last stage of gluconeogenesis consists of the formation of free glucose from glucose-6-phosphate in a reaction catalysed by glucose-6-phosphatase, which, to be stable, has to be attached to a protein which in turn joins with Ca^{++} . Glucose-6-phosphate is generated in cytosol and should be transported to the lumen of the endoplasmic reticulum.

Metabolism of other monosaccharides

Fructose

Fructose is metabolised by means of its conversion to glycolytic pathway intermediaries. It is phosphorylated in most tissues by HK to fructose-6-phosphate which is a glycolytic intermediary. It follows a different pathway in the liver, it is phosphorylated to produce fructose-1-phosphate (F1P) in a reaction catalysed by ketohexokinase or fructokinase. Fructose-1-phosphate is divided by the action of aldolase B, to

form DHAP and glyceraldehyde (GA). To be able to metabolise GA, it has to be phosphorylated by triose kinase creating GAP, which enters the glycolytic pathway, along with dihydroxyacetone phosphate, at the triose phosphate level (Fig. 2).

Galactose

The metabolism of galactose takes place via the conversion of glucose. The first stage of its metabolism is the formation of galactose-1-phosphate, in a reaction catalysed by galactokinase. This enzyme is found in the red and white blood cells and the liver. The following stage consists of the formation of uridine diphosphate-galactose from galactose-1-phosphate and uridine diphosphate-glucose, in a reaction catalysed by galactose-1-phosphate uridyl transferase.

Uridine diphosphate-galactose is epimerised to uridine diphosphate-glucose, in a reaction catalysed by uridine diphosphate-galactose-4-epimerase whose coenzyme is NAD^+ . The enzyme catalyses the reaction in both directions and can also be used as a substrate of uridine diphosphate- N-acetylglucosamine or uridine diphosphate-N-acetylgalactosamine. The following stage is catalysed by uridine diphosphate glucose pyrophosphorylase, which facilitates not only the

production of glucose-1-phosphate from uridine diphosphate glucose but also the formation of uridine diphosphate glucose from uridine triphosphate (UTP) (Fig. 2).

Mannose

Mannose comes from the digestion of polysaccharides and glycoproteins, it is phosphorylated by HK to mannose-6- phosphate and is subsequently isomerised by phosphohexose isomerase, giving rise to fructose-6-phosphate which enters the glycolytic pathway (Fig. 2).

DIETARY SOURCES OF MONO- AND DISACCHARIDES

Generally speaking, the composition of mono- and disaccharides are considered in three food groups and then the content of each of the sugars from those food groups are examined individually. Unfortunately, the international data bases such as the US Department of Agriculture (<http://ndb.nal.usda.gov/ndb/search/list>) and the FAO (<http://www.fao.org/infoods/infoods/tablesand-databases/faoinfoods-databases/en/>) do not provide a detailed composition

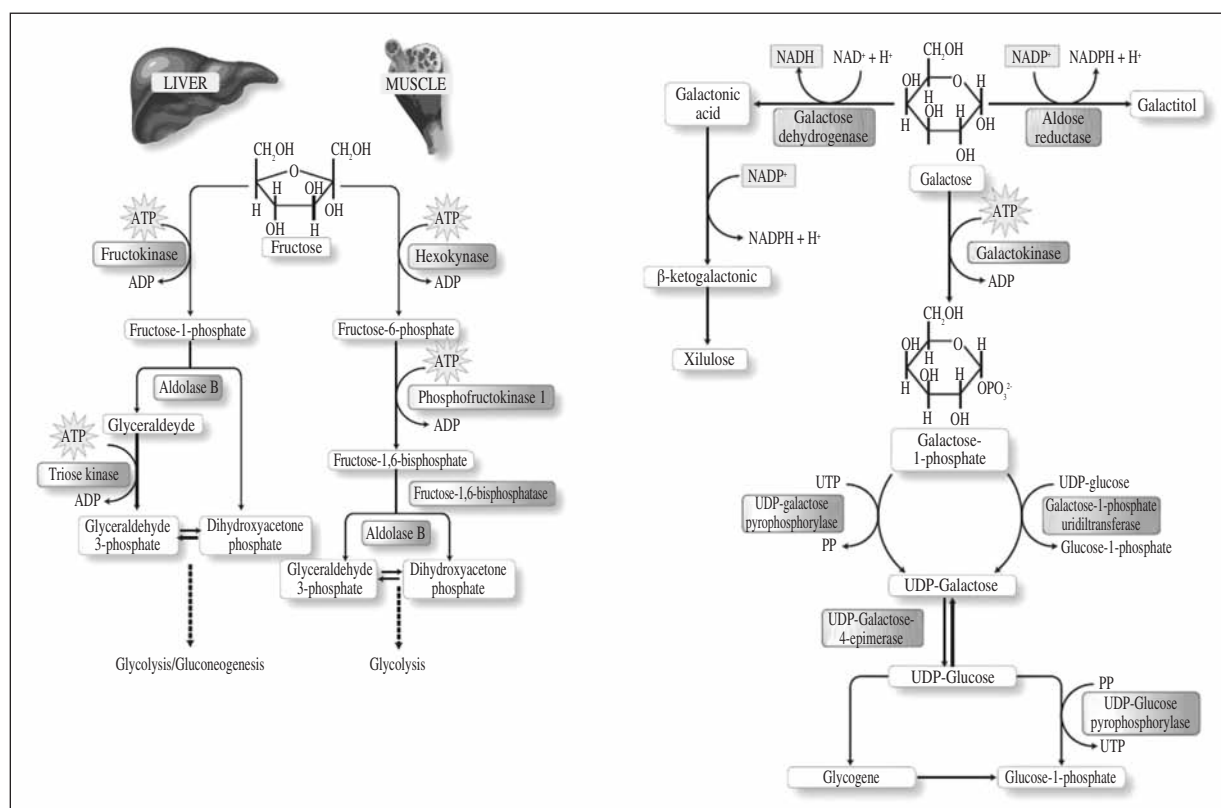


Fig. 2.—Reactions of fructose interconversions in the liver and muscle, and of mannose and galactose. Taken from Gil A. *Tratado de Nutrición*, 2.ª Ed. Vol. I, Editorial Panamericana, Madrid, 2010.

of the mono- and disaccharides in foods and are limited to only provide information on total carbohydrates 'by difference' in relation to the other nutrients, fibre and total sugars^{8,9}.

Milk and dairy products

Lactose is the main and sole carbohydrate in milk. The lactose content of milk depends on the species. Dehydrated dairy products vary in their average lactose composition, from 10% pp for evaporated milk up to 50% pp for powdered skimmed milk, and around 3 g for cream and 1.1 g per 100 g of butter¹⁰.

Lactose appears to have beneficial effects in the intestinal absorption of calcium. In people with lactose intolerance, milk consumption can produce symptoms of intestinal disorders, which to a greater or lesser degree, cause abdominal bloating, excessive intestinal gas, nausea, diarrhoea and abdominal cramps. People who don't tolerate milk well can substitute it for other dairy products, like cheese (since a large proportion of the lactose content is lost during the coagulation and maturing process), or fresh fermented products, such as yoghurt.

Cereals and their derivatives

The sugar and oligosaccharide content of cereals is low (1-3%) and is distributed between the germ, bran and endosperm. The principal sugar in all of them is sucrose, which can reach up to 1%; the average content of rice, oat and wheat flours is 0.13%, 0.25% and 0.56%, respectively. There are lower concentrations of glucose and fructose of 0.02-0.06% in rice, oat and wheat flours. Maltose is found in varying quantities depending on the degree of starch hydrolyses and, in the case of wheat flours, the content varies between 1.7 and 2.4. In cakes and pastries the energy value is very high, as they correspond to products that are rich in carbohydrates (37%-79%), especially starch. Furthermore, sucrose is added to many of them¹¹.

Sugars are a basic ingredient of biscuits, sweets and confectionary. In these products sucrose, glucose syrup, fructose and honey constitute 40% of the total carbohydrates¹¹.

Fruit, vegetables and honey

The main fruit sugars are sucrose, glucose and fructose. Whichever one is predominant depends on the type of fruit. So, drupes (plums, apricots, peaches, etc.) contain mainly sucrose, with the exception of cherries. As far as reducing sugars are concerned, the highest proportion is usually glucose. In pip fruits, called pomes (apples, quinces and pears) there is also glucose

and fructose, but in this case the proportion of fructose is higher and continues to increase, even after they have been harvested. Other fruits like grapes or figs don't contain sucrose and their main source of sugar is glucose¹².

D-glucose is found naturally in honey (31%)¹³; fruit, like grapes and cherries (around 7%), apples and peaches (1%), vegetables like onions (2%), tomatoes, carrots, cucumber, green beans, potatoes and sweet corn (1%)¹².

Fructose is found naturally in honey (38%)¹²; fruit, like grapes and cherries (around 7%), apples and pears (6%), strawberries (2%) and peaches (1%), vegetables like onions, tomatoes, carrots, cucumbers, green beans (1%) and potatoes and sweet corn (0.3%)¹². Sucrose is found in both fruit and vegetables such as peaches (7%), apples (4%), grapes and pears (2%); vegetables and fruits like beetroot (6-20%), peas (5%), carrots (4%), sweet corn (12-17%) and potatoes (3%)¹².

Maltose is found in honey (7%), produced by transglycosylation reactions, and in variable proportions in fruits, vegetables and cereals by enzymatic starch hydrolysis, which these foods contain¹³.

SWOT ANALYSIS OF THE NOMENCLATURE, COMPOSITION OF FOODS AND SUGAR INTAKE

Strengths

Various international agencies have recently been concerned with providing a comprehensive overview of the terminology to be used for the different types of carbohydrate and, in particular, for sugars^{1,3}.

The intakes of different types of carbohydrate are being assessed systematically in several cohort studies which enables the current intakes of sugars and other carbohydrates to be established with a greater degree of safety.

Weaknesses

International food composition data bases do not provide detailed quantities of mono- and disaccharides in foods and are limited only to provide results for total carbohydrates 'by difference' in relation to the other nutrients, fibre and total sugars.

There are a number of major factors which limit the amount of comparable information on carbohydrate intake in adults, infants and small children. The first focuses on the limited number of studies conducted. The second, on the different approaches and varying results published in data bases on carbohydrates. The third is the large amount of terms used and finally the lack of information on global starch intakes.

Opportunities

The specific dietary recommendations for infants and children must be better addressed, on an individual basis, incorporating the possible interactions between diet and genes, which are crucial to understand the relationship that exists between diet and the risk of metabolic disease.

Future cognitive research should incorporate neurodevelopmental assessments, as well as measuring the general cognitive levels, associated with the intake of sugars and other carbohydrates. Prospective cohort studies are needed, covering infancy and childhood, so that the influence of consuming the different types of carbohydrates on obesity and diabetes can be established.

Threats

Based on the lack of information available on carbohydrate intake, particularly in infants and young children, the clearest threat is the growing and worrying relationship with the development of various illnesses, such as obesity, cognitive problems, insulin resistance and diabetes. It's necessary to evaluate and address each of the facts which help us to understand if there is such a relationship and which are the normal values necessary for a healthy diet; not for total carbohydrates but the values for their key components and, in particular, sugars.

References

1. WHO/FAO Joint expert consultation. Carbohydrates in human nutrition, 1998.
2. Gil A, Ramírez Tortosa MC. Azúcares y derivados. En: Aran-ceta J, editor. Guías alimentarias para la población española. Recomendaciones para una dieta saludable. Madrid: IM&C, S.A. y SENC; 2001, pp. 133-46.
3. EFSA Panel on Dietetic Products, Nutrition, and Allergies (NDA); Scientific Opinion on Dietary Reference Values for carbohydrates and dietary fibre. EFSA Journal 2010; 8(3):1462 [77 pp.]. doi:10.2903/j.efsa.2010.1462. Available online: www.efsa.europa.eu
4. Stephen A, Alles M, de Graaf C, Fleith M, Hadjilucas E, Isaacs E, Maffei C, Zeinstra G, Matthys C, Gil A. The role and requirements of digestible dietary carbohydrates in infants and toddlers. *Eur J Clin Nutr* 2012; 66: 765-79.
5. Sánchez de Medina Contreras F, Gil A. Funciones y metabolismo de los nutrientes. En: Gil A, editor. 2 ed. Tratado de Nutrición, Tomo I: Bases fisiológicas y bioquímicas de la nutrición. Madrid: Editorial Médica Panamericana; 2010, pp. 17-42.
6. Atkinson FS, Foster-Powell K, Brand-Miller JC. International Tables of Glycemic Index and Glycemic Load Values: 2008. *Diabetes Care* 2008; 31: 2281-3.
7. Martínez Agustín O, Suárez Ortega MD. Metabolismo de los hidratos de carbono. En: Gil A, editor. 2 ed. Tratado de Nutrición, Tomo I: Bases fisiológicas y bioquímicas de la nutrición. Madrid: Editorial Médica Panamericana; 2010, pp. 203-33.
8. USDA United States Department of Agriculture, National Agricultural Library, Nutrient Data Library <http://ndb.nal.usda.gov/ndb/search/list>
9. International Network of Food Data Systems (INFOODS), FAO/INFOODS Food Composition Databases <http://www.fao.org/infoods/infoods/tables-and-databases/faoinfoods-databases/en/>
10. Baró Rodríguez L, Lara Villoslada F, Corral Román E. Leche y derivados lácteos. En: Gil A, editor. 2 ed. Tratado de Nutrición, Tomo II: Composición y calidad nutritiva de los alimentos. Madrid: Editorial Médica Panamericana; 2010, pp. 1-26.
11. García-Villanova Ruiz B, Guerra Hernández EJ. Cereales y productos derivados. En: Gil A, editor. 2 ed. Tratado de Nutrición, Tomo II: Composición y calidad nutritiva de los alimentos. Madrid: Editorial Médica Panamericana; 2010, pp. 97-138.
12. Ruiz López MD, García-Villanova Ruiz B, Abellán P. Frutas y productos derivados. En: Gil A, editor. 2 ed. Tratado de Nutrición, Tomo II: Composición y calidad nutritiva de los alimentos. Madrid: Editorial Médica Panamericana; 2010, pp. 167-98.
13. Guerra Hernández EJ. Azúcares, miel y productos de confitería. En: Gil A, editor. 2 ed. Tratado de Nutrición, Tomo II: Composición y calidad nutritiva de los alimentos. Madrid: Editorial Médica Panamericana; 2010, pp. 221-48.

A current and global review of sweeteners; regulatory aspects

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Abstract

In this chapter we review the role and potential benefits of non-caloric sweeteners, as part of the diet. After appearing and interest in the beneficial effects attributed to them, face different situations and conditions (obesity, diabetes...), more and more numerous studies, show their ineffective use.

In conclusion, further research and results are needed to provide convincing evidence of their long-term effectiveness and the absence of negative effects from their use.

The interest of the chapter lies in examining the distinctive aspects of sweeteners compared with sugar, measured as the standard of comparison. We will focus then on the other substances that are commonly used to sweeten foods instead of sugar.

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Key words: Artificial sweeteners. Sugar and sugar substitutes. Nonnutritive sweetener. No caloric sweetener. Glycemic response.

UNA VISIÓN GLOBAL Y ACTUAL DE LOS EDULCORANTES; ASPECTOS DE REGULACIÓN

Resumen

En este capítulo revisamos el papel y los posibles riesgos/beneficios de los edulcorantes como parte de la alimentación. Tras su aparición e interés por los efectos beneficiosos atribuidos a los mismos, frente a diferentes situaciones y patologías (obesidad, diabetes, caries, etc.), cada vez son más numerosos, sin embargo, los estudios que parecen constatar la ineficacia de su uso. Por tanto, se requieren más investigaciones que aporten datos convincentes de su efectividad a largo plazo, así como de la ausencia de efectos negativos, derivados de su uso.

El interés del capítulo reside en examinar los aspectos distintivos de los edulcorantes frente al azúcar, considerándose ésta como patrón de comparación. Nos centraremos pues, en las otras sustancias que habitualmente se utilizan para edulcorar los alimentos en lugar del azúcar.

Nutr Hosp 2013; 28 (Supl. 4):17-31

Palabras clave: Edulcorantes artificiales. Azúcar y sustitutos del azúcar. Edulcorantes no nutritivos. Edulcorantes acalóricos. Respuesta glucémica.

Abbreviations

ACS: The American Cancer Society.
ADA: The American Diabetes Association.
AHA: The American Heart Association.
APM: Aspartame.
mRNA: Messenger RNA.
DM: Diabetes Mellitus.
HIS: High-intensity sweeteners.

MS: Member states.
EFSA: European Food Safety Authority.
FFQ: Food-frequency questionnaire.
FOS: Fructooligosaccharides.
GIP: Glucose-dependent insulintropic peptide.
GLP: Glucagon-like peptides.
GRAS: Generally recognised as safe.
ADI: Acceptable daily intake.
EDI: Estimated daily intake.
GI: Glycaemic Index.
BMI: Body mass index.
JECFA: Joint FAO/WHO Expert Committee on Food Additives.
NNS: Non-nutritive sweeteners.
WHO: World Health Organisation.
SCF: The EU Scientific Committee on Food.
EU: European Union.

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Introduction

The term ‘sweetener’ refers to those food additives which are able to mimic the sweetness of sugar and which usually provide less energy. Some are natural extracts whilst others are synthetic. In the case of the latter they are also known as artificial sweeteners. The use of non-caloric sweeteners, as a substitute for all or part of the sugar content in food and drink, has experienced its biggest growth over the past 35 years¹ and projected sales for 2014, according to a recently published systematic review, are expected to exceed one billion².

New eating patterns, characterised by the high consumption of processed foods, with changes involving their fat and sugar content, mark a notable shift away from the traditional Mediterranean diet. In this respect, there has been an exponential growth both in energy-rich foods and those that are supposedly low-calorie. Industry pressure plays a pivotal role in these food consumption patterns. It is therefore essential to clarify the effectiveness and safety of these substances so that consumers can be provided with clear information.

Considering 77% of all calories consumed in the USA, from 2005 until 2009, contain caloric sweeteners and there is a trend toward consuming non-caloric sweeteners, it’s vital to conduct extensive research and to take a strict regulatory approach on these issues. There are currently no conclusive data on the effects of sweeteners on crucial factors such as energy intake, appetite and their relationship with the sweet taste and, furthermore, the exact quantities of these sweeteners that foods contain are unknown. For this reason, it would be of great interest to quantify, as accurately as possible, the prevalence of consuming products containing non-caloric sweeteners. This article tries to summarise the current principal scientific and legislative findings on this issue with an eye to improving the rational use of these substances in our diet³.

The concept of health is very broad and its determinants encompass biological aspects, such as genetic characteristics, and other socio-economic and cultural aspects which, as a whole, determine an individual’s health status (Fig. 1). Over time, changes in disease patterns, probably associated with lifestyle changes in the general population, have led to an increase in the incidence of many chronic diseases such as obesity, type II diabetes, and metabolic syndrome, which ultimately result in an increase in cardiovascular morbidity and mortality. Interest in the potential role of sweeteners has grown, due to the need to find alternatives to prevent disease and maintain good health by following a healthy diet.

Obesity has become one of the biggest global health challenges of the 21st century. The increase in childhood and adolescent obesity is particularly

alarming given its association with metabolic diseases and their cardiovascular complications. The people of developing countries are experiencing rapid changes in their eating habits as well as increases in the rates of childhood obesity. The considerable increase in sugary drink consumption among adults and children in the USA and other countries is regarded as a potential contributor to the obesity pandemic. Recent evidence shows that sucrose consumption in drinks is approaching 15% of the American public’s daily caloric intake, accounting for up to 357 kcal per drink. All this has resulted in the development of regulatory strategies which limit the sale and, consequently, the consumption of these drinks.

Several randomised and controlled studies have been published in *The New England Journal of Medicine*⁴ which provides a basis on which to promote the development of health recommendations and government/political decisions aimed at limiting sugary drink consumption, particularly those which are served at a low cost and in over-sized portions, with a view to reversing the growth in childhood obesity. In these studies the use of sugar was limited, substituting it for lower-calorie sweeteners. Interventions of this kind, if proven safe and effective, could help to prevent young people from developing type II diabetes and its complications.

Besides the interest generated by their potential preventative role in the development of chronic metabolic disease, we could also highlight their effect on diseases of the oral cavity such as tooth decay. In particular, polyalcohols can reduce the risk of tooth decay. For example xylitol is considered to be cariostatic and helps to prevent tooth decay⁵.

Therefore, and from the perspective of sweetener consumption, survey data confirms that they are currently looking to use non-caloric sweeteners with a view to reducing the total caloric intake, promote weight loss and/or prevent the development of diseases like diabetes or tooth decay. However consumers are also concerned about the risk associated with their use, such as ‘artificial or natural’ elements and whether they pose a risk to health.

The estimated consumption of sweeteners is complex but it would appear that there are more than 6000 ready made products which contain them in the USA, mainly soft drinks. Information on nutrition labels is often incomplete, without any details on the exact amount. Data from NHANES 2007-2008 24-hour recalls and food frequency questionnaires show an increase in the American population’s consumption of sweeteners which, interestingly, is not associated with a reduction in sugary foods.

With regard to the legal aspects of the use of sweeteners at a European level, the European Parliament and Council Directive 94/35/CE of 30th June 19946 on all sweeteners for use in foodstuffs arose as the initial

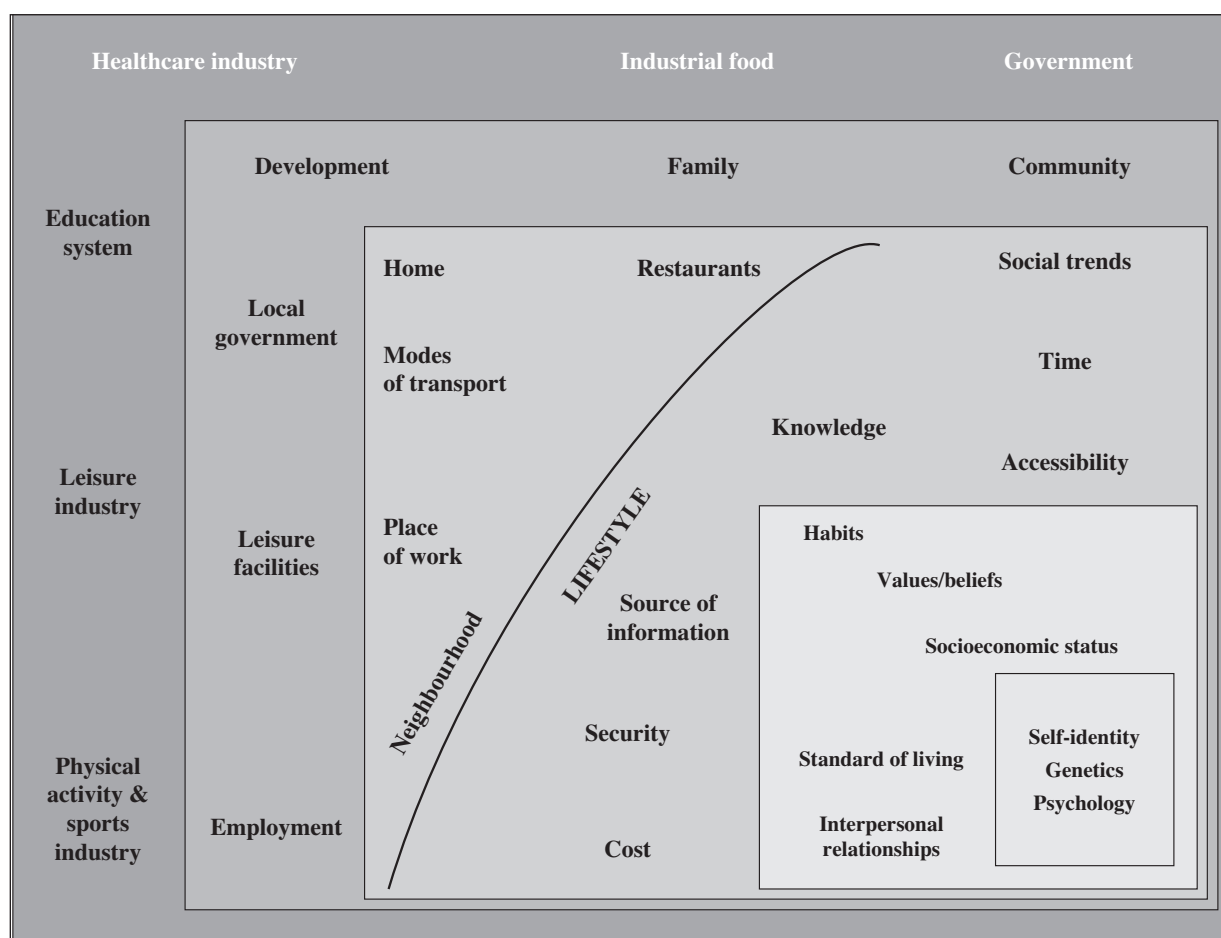


Fig. 1.—Global determinants of health: The multifactorial relationship between individuals and environmental factors, in relation to food choices and healthy behaviour. Amended by Anderson GH, Foreyt J, Sigman-Grant M, Allison DB. The use of low calorie sweeteners by adults: impact on weight management. *J Nutr* 2012; 142 (6): 1163S-9S.

governing regulation. It is a specific directive from the Framework Directive on food additives used as sweeteners. The articles of this law contain explanations and specific provisions for the use of sweeteners in food and drinks. The maximum usage levels for each of the low calorie sweeteners are set out in specific food categories in the Directive's annex. Over the years, this Directive has been amended three times to accommodate technological advances in the field of sweeteners. Later on, the European Parliament and Council adopted a regulatory framework (Regulation No.1333/2008) which, from January 2011, consolidated all the existing authorisations for sweeteners and food additives into a single legal text. At present, the following low calorie sweeteners are authorised in the European Union (EU): Acesulfame-K (E950), Aspartame (E951), Aspartame-Acesulfame salt (E962), Cyclamate (E952), Neohesperidin dihydrochalcone (E959), Saccharin (E954), Sucralose (E955), Thaumatin (E957) and Neotame (E961)⁷ (International Sweeteners Association at <http://www.info-edulcorants.org/es/recursos-profesionales/folleto-isa>).

Following the EFSA's favourable opinion use of stevia derivatives, steviol glycosides, were finally approved as a natural non-caloric sweetener throughout the European market. They can be used as food additives and thereby provide a healthy and natural alternative for sweetening foods, especially for diabetics or those who wish to stay in shape, for example: flavoured drinks or diet foods designed for weight control).

Annex II to Regulation (EC) No. 1333/2008 of the European Parliament and Council was amended with the introduction of the Commission's Regulation (EU) No. 1131/2011 of the 11th November 2011, with regards to steviol glycosides (E960) and limits on the use of sweeteners in different foods and drinks were established (soft drinks, fermented dairy products, flavoured ice creams, table sweeteners, diet products for weight control.)

The safety of sweeteners is evaluated by the national authorities, the EU Scientific Committee on Food (SCF) and the Joint FAO/WHO Expert Committee on Food Additives (JECFA). The SCF was responsible for

it from 1974 until 2003, the year in which it became the responsibility of the European Food Safety Authority (EFSA) (<http://efsa.europa.eu/>). Within the EFSA, the Scientific Panel on Food Additives and Nutrient Sources (ANS) is currently responsible for the regulation of these substances⁸.

Legal aspects need to be reviewed on a continual basis to update new scientific developments published on the safety or effective use of sweeteners. As they are very diverse molecules, there are numerous potential risk sources: interference with absorption, metabolism or the excretion of any intermediate metabolite, as well as any allergic reaction, accumulation in tissues, effects on normal intestinal flora, changes in blood sugar regulation, or interaction with other pharmaceuticals or drugs.

The European Food Safety Authority (EFSA) has recently produced a scientific evaluation of the safety of aspartame. In order to carry out this comprehensive risk assessment, the EFSA (<http://www.efsa.europa.eu/en/press/news/130108.htm>) has carried out a thorough review of the scientific literature on aspartame and its breakdown products, including new studies on humans. In this safety re-evaluation by the EFSA, it was concluded that aspartame does not present any risk of toxicity to consumers at current exposure levels. The current acceptable daily intake (ADI) is considered safe for the general population and consumer exposure is normally below the ADI. When establishing the ADI for aspartame the ANS commission also considered the results of long-term studies on phenylalanine, an aspartame metabolite, both in toxicity and carcinogenesis tests on animals and humans, specifically, the foetal development in mothers who consume this sweetener.

At the same time, in North America the US Food and Drug Administration (FDA) has been responsible for evaluating its safety since 1958 and seven sweeteners have been approved for use in the USA: Acesulfame K, Aspartame, Neotame, Saccharin, Stevia, Sucralose and Luo han guo.

The American FDA regulations also refer to the concept of estimated daily intake (EDI), which is a conservative estimate of the probable daily intake over a lifetime and the concentration of food additives in commonly eaten foods. Another important concept concerning consumer safety is GRAS (Generally recognised as safe), which implies that, although the potential risks aren't yet completely understood, experience through common use has not raised any problems. This is the accepted recognition to market stevia currently in the USA, pending further information in the future.

Information on the correct use of these substances comes from knowing the differences on the nutrition facts labels of commonly consumed products that contain sweeteners. The presence of sweeteners should be listed in the food's ingredients, along with calorie

content, fat or carbohydrates, in the nutritional labelling information.

However, with the exception of warnings about phenylalanine from aspartame or the amount of saccharin, this information is usually missing or incomplete. It opens up a major area for improvement in the field of sweetener use, to provide consumers with the best information in the future. Scientific research, although limited in humans according to the Evidence Analysis Library of the Academy of Dietetics and Nutrition (<http://www.adaevidence.com/files/Docs/NNSResourceDraft3.pdf>), shows that artificial sweeteners are safe to use for the general population, including pregnant women and children. Most studies have not found any adverse effects related to the consumption of sweeteners, even when they are consumed in large quantities. Special population groups, such as pregnant women, should limit their use even though they have been approved by the FDA, using them in moderation.

In this chapter we will review the main sweeteners, their metabolic effects and we will analyse their potential strengths, weaknesses, opportunities and threats (SWOT system).

Sweeteners: types and key characteristics

With regard to overall classification, given the large variety of the existing types, sweeteners can be grouped according to their calorie content (caloric or non-caloric), their source (artificial or natural) or even their chemical structure (Fig. 2). Naturally sourced sweeteners are not necessarily safer or more effective and, in this respect, there is a great deal of consumer misinformation about them. There are a wide variety of sweetening substances. In this chapter we will focus on the most common ones and those with existing scientific studies which are of interest. The current classification of the main sweeteners is presented in table I.

Sugars are carbohydrates and therefore contain 4 calories per gram. They are found naturally in many foods such as fruit, vegetables, cereals and milk. As with starches, if good oral hygiene is not maintained they can be harmful to teeth, although the most recent scientific literature also points out that the stickiness of food and the frequency with which these foods are consumed could have an impact on levels of tooth decay. Sucrose has a moderately high glycaemic index (GI). Other natural caloric sweeteners, like honey and maple syrup, are older, contain sugar, but they also have other nutritional qualities. Their glycaemic index is somewhat lower than sugar. Saccharins are included in the group of natural sweeteners, among which the most commonly used are sucrose, fructose, glucose (GI of 100 and a sweetness relative to sucrose of 0.5-1) and maltose (GI 105 and a sweetness relative to sucrose of

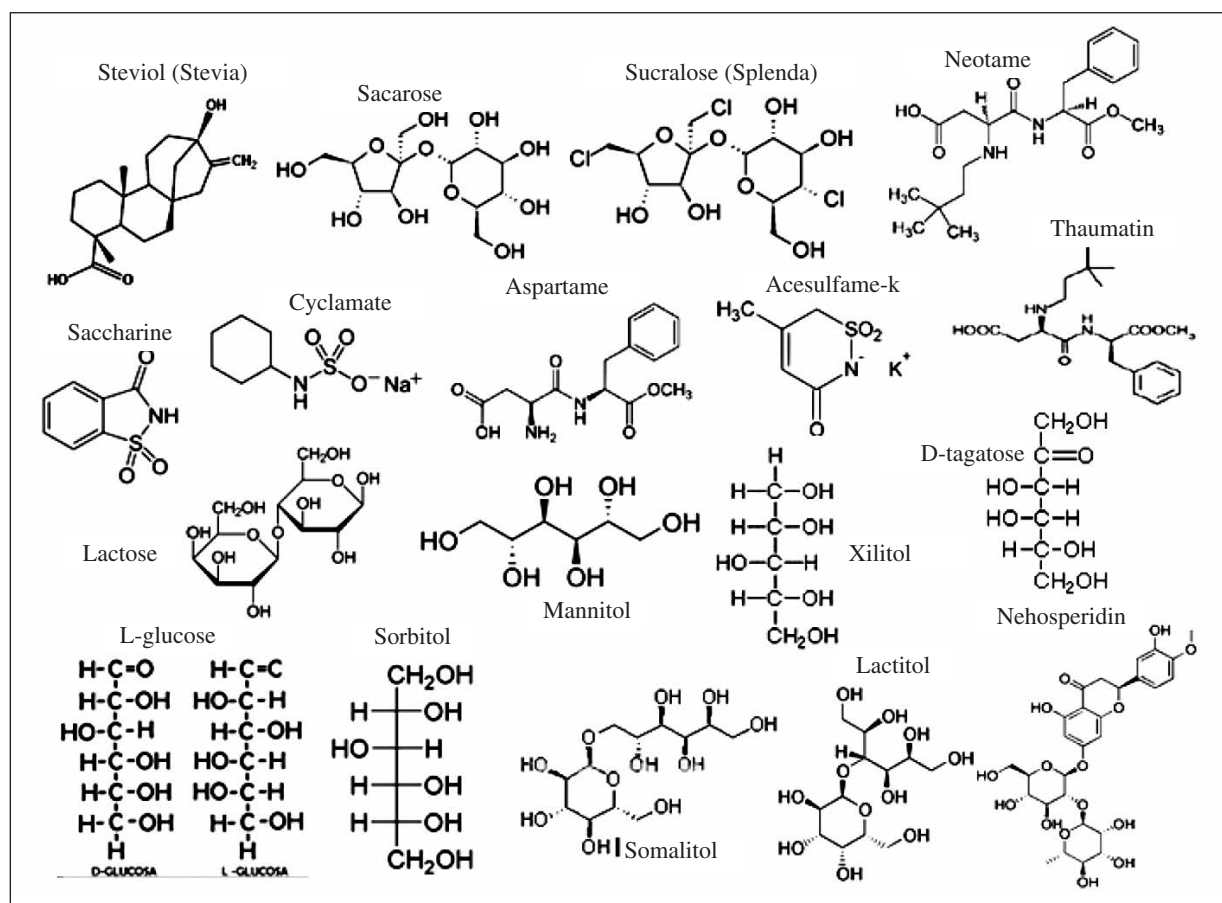


Fig. 2.–The chemical structure of sweeteners.

Table I Classification of sweeteners			
Caloric	Natural	Sugars	Sucrose, glucose, dextrose, fructose, lactose, maltose, galactose and trehalose, tagatose, Sucromalt*
		Natural caloric sweeteners	Honey, maple syrup, palm or coco sugar and sorghum syrup
	Artificial	Modified sugars	High fructose corn syrup, caramel, inverted sugar
		Sugar alcohols	Sorbitol, xylitol, mannitol, erythritol maltitol, isomaltulose, lactitol, glycerol
Non-caloric	Natural	Non-caloric sweeteners	Luo Han Guo, stevia, thaumatin, pentadin, monellin, brazzein
	Artificial	Sweeteners	Aspartame, sucralose, saccharin, neotame, acesulfame-K, cyclamate, neohesperidin DC, alitame, advantame

* Caloric value similar to fructose, although it really is an artificial oligosaccharide.

of 0.5). Fructose is typically used as a substitute for sucrose in diabetic patients and as a sweetener in the manufacture of many products labelled as 'suitable for diabetics'. However, recently it has been proven that diets high in fructose, especially if it is added to manufactured foods, can cause hyperinsulinaemia, hyper-

triglyceridaemia and insulin resistance, which was a determining factor in the recommendation that diabetics should limit its use. Its properties include a calorie content of 4 kcal/g, a GI of 23 and a sweetness relative to sugar of between 1 and 2. On the other hand, tagatose and trehalose have different calorie contents,

1.5 and 3.6 and a sweetness of 0.9 and 0.45 respectively.

Fructooligosaccharides (FOS) have half the calories per gram than sucrose or glucose with a sweetness relative to sucrose of 0.3-0.6. Inulin is a fructan with a degree of polymerisation of 20 to 60 fructose monomers and a documented prebiotic effect, which is found naturally in a native Andean tuber, the yacon (12.5g/100g), and which had historically been grown in various Latin American countries. This tuber is mainly used as a sweetener and the possibility that it has nutraceutical properties due to its high content of various minerals, vitamin C and B group vitamins. Coco sugar is another traditional product which can be used as an alternative to sugar in diabetic patients as it is a food which is considered to be low GI. It consists of sucrose, aminoacids such as glutamine and stands out for its high mineral and group B vitamin content.

Alcohols derived from sugar are also carbohydrates which are produced naturally, although in small amounts, in plants and cereals. They generally contain less calories per gram than sugar and are not associated with tooth decay. Despite the fact that they are carbohydrates, the body can't fully metabolise them and, consequently, they tend to have less than 4 calories per gram and a very low glycaemic index. Some of the carbohydrates used as sweeteners (i.e. polydextrose or xylitol) have been proposed as ingre-

dients for functional foods useful for controlling intake because of their low energy content, which is due to their partial metabolism (1.5 to 3 kcal/g) and also the possible effects of some of them on appetite suppression, although the clinical of this is not yet known. A large number of these are increasingly used as sweeteners in 'sugar free' products. The chemical structure of these substances (Table II) cause them to have greater sweetening powers when they interact with taste receptors and a lower absorption by the digestive tract, consequently they have a lower usable calorie content than sugar. Limits on the amount consumed are related to their secondary gastrointestinal effects.

The manufacture of sugars modified by enzymatic starch conversion, which are frequently used in industrial cooking or in processed foods, gives rise to a blend of carbohydrates which are usually high in calories and have an elevated glycaemic index. One product regularly used in the industry which belongs to this group and has a nutritional value close to 4 kcal/g, typical in carbohydrates, is high fructose corn syrup with a relative sweetness of 1.

Sucromalt is an artificial oligosaccharide (http://www.aesan.msc.es/AESAN/docs/docs/cadena-alimentaria/tabla_decisiones_2013.pdf) which is converted from sucrose and maltose, to fructose and an glucose oligosaccharide with links at 1-3 and 1-6 alternatively. It has been extensively used in the design of

Table II
Description of sugar alcohols

<i>Sugar alcohols</i>	<i>Nomenclature</i>	<i>Nutritional value (kcal/g)</i>	<i>Sweetness, relative to sucrose</i>	<i>Maximum tolerable quantity without gastrointestinal symptoms (g/day)</i>	<i>Presence</i>	<i>GI</i>
- Erythritol	E968	0.2	0.75	At higher doses	In fruits and other fermented foods	1
- Hydrolysed hydrogenated starch (Lycasin) Polyglycitol Syrup	E964	≤ 3	0.4-0.9	–	Sports drinks (e.g.: powerade), ice cream	–
- Lactitol	E966	2	0.5	≥ 20	Sweets, biscuits, ice cream	3
- Maltitol	E965	2.1	1	30-50	Chewing gum, sweets jelly sweets	35
- Manitol	E421	1.6	0.7	10-20	Chewing gum*	2
- Sorbitol	E420	2.6	0.5-1	> 80	Chewing gum*	4
- Xilitol	E967	2.4	1	> 50	Chewing gum, breath mints, toothpaste and mouthwash	12

* Furthermore it also contains isomalt, aspartame, and acesulfame-K. They amount to 61.7g of polyalcohols/100 g.

low GI foods. Its nutritional value is similar to fructose and its sweetness relative to sucrose is 0.7.

There are also natural sweeteners (Stevia, Luo Han Guo, Thaumatin and Brazzein) whose calories are insignificant compared to the quantities usually used for sweetening purposes. These are not carbohydrates, therefore they don't have a glycaemic index. They are considered high-intensity sweeteners (HIS).

Stevia, is probably one of the sweeteners which has generated the most interest in scientific and informative forums over the past few years. It is used as a sugar substitute and it has a slow taste onset at the beginning and is longer lasting, although in high concentrations it can have a bitter taste similar to 'liquorice'. Although the word 'stevia' refers to the whole plant, only certain parts of the stevia leaf are sweet. These sweet components are known as steviol glycosides (an alcohol which can be naturally found in the plant). Furthermore, the term 'stevia' typically refers to a crude preparation (whether it's powder or liquid) made from the plant's leaves and these preparations contain a mixture of various components, not just those that give the leaf its sweet flavour.

Steviol glycosides are the sweet components of the stevia leaf and there are several types, although the most common are stevioside and rebaudioside A. Stevioside is the most common steviol glycoside in the stevia leaf and has been widely studied. On the other hand, rebaudioside A is a better tasting steviol glycoside and is metabolised in the same way as a stevioside. These sweeteners are up to 480 times sweeter than sugar. Their leaves are naturally 15-30 times sweeter than sugar. It is a natural product which has a glycaemic index of zero and is therefore suitable for diabetics. It is heat-stable and suitable for cooking as well as suitable for use in processed foods. It has been used for centuries by the indigenous people of Paraguay, South America and also in Asia (Japan) since the 1970s. Its standardisation in the American market from 2008 onwards with GRAS recognition has proven complicated. The native plant contains proteins, fibre, iron, phosphorus, calcium, potassium, zinc and vitamin A, and its derivatives often provide varying amounts of the active compounds which is a major limitation of their use. Various studies have been conducted to examine their effects on weight, appetite or gut flora with results that are not entirely conclusive, therefore more studies will be needed in the future to clarify this matter.

The EFSA recommended ADI for stevia, or steviol glycosides, is consistent with the level adopted in the past by the Joint FAO/WHO Expert Committee on Food Additives, JECFA).

Luo Han Guo is a non-caloric high-intensity natural sweetener (300 times sweeter than sugar cane). It is extracted from monk fruit, from China, which has been used for hundreds of years. Its sweet-

ness comes from a substance called mogroside in the pulp of the fruit. One of the advantages it has over stevia is the lack of a bitter aftertaste which is characteristic of stevia. Like stevia, its glycaemic index is zero and it has recently been approved by the FDA for use in GRAS recognition for the additives of certain foods from 2010. It's still under evaluation in Europe⁵.

One subset of natural sweeteners which is still completely outside of the commercial market is 'sweet proteins'. In spite of the fact that seven sweet proteins have been identified (Thaumatin, monellin, mabinlin, pentadin, brazzein, curculin and miraculin) only two have been commercialised: Thaumatin and brazzein. All of these proteins have been extracted from plants which grow in rain forests. Sweet proteins tend to have lingering aftertastes, a characteristic which clearly distinguishes them from sugar. Thaumatin is the most advanced sweet protein regarding product development and its situation with the regulatory authorities.

'Artificial sweeteners' per se usually refer to the various existing compounds on the market which are characterised by the fact that they are non-caloric, they have no glycaemic effect whatsoever and they are high-intensity sweeteners: This group is of the most interest in the area of research, with the aim of proving their safety and providing firm data on their possible therapeutic effects on patients with diabetes or other specific health problems. At a consumer level interest in these products has increased significantly in the search for low-calorie products (Table III).

Saccharin continues to dominate the global market of HIS for levels of consumption, with millions of tonnes in 2010. Asia continues to be by far the world's biggest consumer of saccharin. Saccharin was the first artificial sweetener which was discovered more than 120 years ago. Like most artificial sweeteners, it was discovered by accident whilst looking for other unrelated substances. It is 300 times sweeter than sugar, but it has a slightly unpleasant metallic aftertaste. It has a glycaemic index of zero, it contains no calories and is suitable for diabetics. It doesn't tolerate high temperatures so it is not suitable for cooking. It blends well with other sweeteners, or even with a small amount of sugar as in some 'diet' or 'zero' drinks.

Cyclamate is the second oldest artificial sweetener in use today. It's the least powerful of this group, only 40 times stronger than sugar. For this reason, it's often blended with other sweeteners like saccharin. It is heat-stable and has a long shelf life which makes it suitable for cooking and food processing. It has a glycaemic index of zero and contains no calories. Like saccharin, it is also widely used in Asia. Its use is authorised in Europe and 50 other countries, but it has been prohibited in the USA since 1969 due to a reported associa-

Table III
Description of sugar alcohols

Sweetener	Nomenclature	Nutritional value (kcal/g)	Sweetness, relative to sucrose	ADI* (mg/kg weight/day)		Maximum amount of sweetener (mg/day) in a 70 kg subject	No. of drinks/ over = ADI for a 70 kg subject ***
				EU	FDA		
- Acesulfame-K	E950	0	200	0-9	15	630	16/13
- Aspartame	E951	4	160-220	0.40	50	2,800	15/70
- Cyclamate: Cyclamic acid and sodium and calcium salts	E-952	0	30	0-7	Not permitted	490	–
- Lu Han Guo or concentrated fruit extracts (mogroside)	Natural sweetener	0	150-250	Not permitted	Unspecified. Included in GRAS status	–	–
- Neohesperidin DC	E959	0	1,500	0-5	–	350	–
- Neotame	E961	0	8,000	0-2	18	140	Absent in carbonated drinks and not consumed in products
- Saccharin and its sodium, potassium and calcium salts	E954	0	300	0-5	Unspecified	350	44/9
- Stevia (steviol glycoside)	E960	0	300	0-4	4	280	16.5/31
- Sucrose (splenda)****	E955	0	600	0-15	5	1,050	15/95.5
- Thaumatin	E957	approx. 0	2,000-3,000	Unspecified or by JECFA	Not specified. Included in GRAS status	–	–

tion with developing bladder tumours in animal models and has not been reviewed since then.

Sucrose is a modified form of table sugar (sucrose) which has no calories and is 600 times sweeter than sugar. Its flavour is considerably different to table sugar and does not decompose when heated. It is commonly used all over the world, alone or with other sweeteners, and can be found in more than 4,500 foods and drinks.

Neohesperidin dihydrochalcone is a sweetener which is derived from the chemical modification of a substance found in bitter oranges. It is between 250 and 1,800 times sweeter than sucrose and has a longer lasting sweet flavour with a liquorice aftertaste. It has not been approved by the FDA, but it has been in Europe.

Aspartame is an artificial sweetener which is almost 200 times sweeter than sugar. It is a protein and as such, contains 4 calories per gram. However, it is so sweet that only a small amount is needed and so its calorific value is insignificant. It continues to be one of the most commonly used and well-known intense

sweeteners, thanks largely to its strong market position in the USA, its main producer, which consumes 60% of the global demand for this substance. It decomposes when heated and is therefore not suitable for cooking. It has almost completely replaced saccharin as the most commonly used sweetener in 'diet' drinks. There have been huge controversies over its safety although agency reports claim that it is safe for consumption. It is the biggest source of complaints to the FDA, more than any other product or medication.

Acesulfame-K is another compound 130-200 times sweeter than sucrose. It is not metabolised and is eliminated unchanged. It's frequently used in soft drinks, fruit nectars, table sweeteners, dairy products, oven-baked goods, toothpaste and pharmaceutical products. There is a combination of aspartame and acesulfame whose composition is 64%-36% respectively. It is known by the E number E-962, has an immeasurable nutritional value and its sweetness relative to sucrose is 350.

Neotame is a dipeptide derived from aspartame and has a sweetness 8,000 times higher than sugar.

Unlike aspartame it doesn't decompose when heated and therefore is suitable for cooking and for use in processed food. It has zero calories per portion and a glycaemic index of zero, which makes it suitable as part of a diabetic diet. It is not metabolised to phenylalanine and so it is safe for phenylketonuria patients. It is mainly used by food manufacturers, in blends with sucrose and other HES. Since it was introduced to Europe in 2010, its use has grown considerably.

Alitame is 2,000 times sweeter than sugar. It is a dipeptide made from aspartic acid and alanine. It is stable, doesn't have any calories and has a glycaemic index of zero. It has still not been approved in the USA but it has in Europe (E956).

There are many new sweeteners like advantame, a derivative of the same aminoacids as aspartame with vanillin, a component of vanilla. Compared to aspartame (about 200 times sweeter than sugar), advantame is between 20,000 and 40,000 times sweeter than sugar. It has been authorised in Australia and New Zealand, and is considered GRAS as a flavouring for non-alcoholic drinks, chewing gum and dairy products.

The health effects of sweetener consumption

The general and metabolic impact of using these substances, mainly added to food and drinks, can affect the quality of the end product (nutritional and organoleptic properties), energy consumption and body weight.

Before choosing one of these substances for its supposed metabolic effects it should be compared with sugar as a reference standard. However, in reality the current lack of knowledge on these possible effects is very significant, therefore it is difficult to support their use based on scientific evidence which is clearly full of contrasts².

In theoretical models these lower calorie substances, which have less of an effect on blood sugar, could have a beneficial impact on weight control or diabetes, however this correlation is unlikely.

Recent results obtained through short-term intervention models show that artificial sweeteners, especially in drinks, may be useful in reducing energy intake as well as body weight and reducing the risk of type II diabetes and cardiovascular disease, if it's compared with sugar consumption. But in order to confirm this specially designed long-term research is needed⁹.

A consensus among the organisations has recently been published (the American Diabetes Association - ADA and the American Heart Association - AHA) in order to clarify certain aspects of the effects on appetite and components of cardiometabolic

syndrome. There are significant limitations on the interpretation of research data due to inherent difficulties in the design due to isolated modifications of the diet's carbohydrate content but without changing fat or protein content, that is to say, in order to maintain the calorie content proteins or fats have to be increased and this can affect appetite. The majority of data involving humans comes from observational studies and certain randomised controlled trials on changing sweeteners in soft drinks. In many of these studies, the data on sweetener consumption in food frequency questionnaires (FFQ) on their exact composition in the products consumed is not well documented due to incomplete information on labels or industry references on the quantities contained in processed foods. On the other hand, experimental research on animals provides important data on the potential adverse effects or toxicity of sweeteners. However, extrapolation of these results to the general population has major limitations¹⁰.

Sweeteners and energy intake

A priori, it would be logical to think, from an energy intake point of view, that substitution of sugar with lower calorie sweeteners should reduce the total energy consumption. However, this subject is controversial given that scientific research exists with contradictory results. Furthermore, it should also be taken into account that in processed products, not only the sweetness provided by sugar needs replacing but also the physical or other technological properties of sugar. The result is that a product reformulated with less sugar is often higher in calories than the 'full sugar' version because the sugar has been replaced with other higher calorie nutrients, such as fat.

Some studies on humans have shown a short-term reduction in calorie intake resulting from only a partial compensation of the calories that are not ingested when compared with sucrose, mainly in soft drinks. However, there is also epidemiological data which connects the use of sweeteners with weight gain. It would appear that the dissociation between the sensation of sweetness and the reduced calorie content produced by sweeteners could cause an increase in appetite, giving rise to a higher energy consumption and weight gain. This operative conditioning theory (Pavlov's Model) has been demonstrated in animal models¹¹.

Observational studies have also described the association of sweetener use with an overall poorer quality diet due to the loss of healthy eating patterns which include fruit and vegetables. There are many associated confounding factors which make it difficult to draw clear conclusions, such as the fact that these low-calorie foods are frequently associated with other higher calorie foods, and individuals choose

them precisely in order to reduce their overall calorie intake.

Furthermore, it is known that, both in humans and animals, food consumption causes a thermogenic response in the cephalic phase of digestion. This response prepares the gastrointestinal tract for the arrival of nutrients. There is evidence in rodent models that the chronic use of sweeteners, such as saccharin, causes a reduction in this stimulus and slows down the thermic effect of food, and perhaps other aspects of metabolic equilibrium¹¹.

It has been proven that sweeteners can play an active role in the gastrointestinal tract by reacting with the sweet taste receptors (T1R family of receptors and a-gustducin), and mediating changes in peptide hormone responses such as glucagon-like peptides (GLP) in intestinal L cells. It has therefore been hypothesized that the concomitant intake of artificial sweeteners together with food or drinks containing sugar could enable faster absorption of sugar, as well as enhancing GLP-1 and insulin secretion, affecting weight, appetite and blood sugar¹².

Sweeteners and appetite regulation

The mechanisms that sweeteners can use to modulate appetite include:

a) *Cephalic Phase Stimulation*. In this respect, some studies maintain the hypothesis that failure to stimulate the cephalic phase response can increase the risk of obesity, conversely others claim that stimulation of cephalic phase responses, from ingesting of simply being exposed to sweet foods, can be problematic because it stimulates both appetite and food intake. Another proposed mechanism could be mediated by the direct effect of non-caloric sweeteners on insulin secretion and glucose metabolism¹³.

b) *Nutritional and osmotic effects*. It is known that the stomach produces appetite signals, mainly based on the volume it can or cannot cover, whereas in contrast, the bowel is more sensitive to signs of the presence of nutrients, even though this hypothesis does not appear to be fulfilled as strictly as the presence of osmo-receptors, at an intestinal level, and chemoreceptors, at a gastric level, imply. It has been proven that with gastric distension, whether it's due to the presence of nutrients or for another reason (gastric balloon), the feeling of fullness increases. Drinks which contain caloric sweeteners have more energy in terms of osmotic load which can be the same or even less than the load produced by non-caloric sweeteners, which means to say that with the same osmolarity the calorie content of non-caloric sweeteners is less; therefore gastric emptying doesn't just depend on osmolarity (chemoreceptors/osmoreceptors).

However, caloric sweeteners cause slower emptying regardless of the osmotic effects.

Activation of signals both in the gut and in the stomach, from the presence of nutrients, have a synergistic effect on satiety. It has been hypothesized that drinks containing non-caloric sweeteners can weaken this effect present in those which contain nutritive sweeteners, even though no clear data really exists on this matter¹³.

c) *Responses of gastrointestinal peptides*. Each macronutrient stimulates the release of peptides in the digestive tract with varying degrees of effectiveness. so, it has been proven that carbohydrates stimulate the secretion of GLP-1, which plays a significant role both as a satiety and incretin factor.

It is thought that non-caloric sweeteners don't permit such a release of peptides and therefore, in theory, this would mean a lesser feeling of satiety and would cause an increase in energy intake.

Some more recent evidence shows that there are receptors, with properties similar to the sweet receptors located on the tongue, in the gastrointestinal tract that stimulate the release of GLP-1, which could give non-caloric sweeteners a role in regulating these incretin systems.

d) *Palatability*. Another of the major benefits of using non-caloric sweeteners, as part of the diet, is to improve the organoleptic properties of the food in question, thus enabling improved acceptance both of the foods themselves and reduced calorie meals in which any food of this type is used, compared with its original higher calorie version and which may contain sugar as such, which undoubtedly contributes to optimal organoleptic properties. This might be a huge advantage in overweight, obese or diabetic patients with a view to improving adherence to treatment regimes and changes in nutritional habits. The hypothesis is whether the degree of food palatability affects appetite sensation, but following numerous studies there is still no conclusive evidence on this matter.

e) *Changes in gut microbiota*. It has been proven that changes in bacterial populations, which make up the gut microbiota, can contribute to the low-grade chronic inflammatory process which is observed in some obese patients and which seems to promote weight gain at the expense of fat mass, as well as actively contributing to the development of the comorbidities typically associated with obesity, such as insulin resistance¹⁴.

Aspartame releases a methanol molecule, which is metabolised into a formaldehyde molecule, a highly reactive substance which is classified as carcinogenic. However, the amounts of these dangerous substances that are ingested, are usually well below the levels of risk. Therefore, it is not unusual for very small amounts of sweeteners to alter intestinal flora, as they act as the first line of defence in the gut and are therefore in direct

contact with the sweetener and its metabolic compounds. During low-calorie diets for weight control the use of sweeteners like aspartame can change the optimal functioning of gut microbiota¹⁵.

f) *Overcompensation*. Studies show that saving/withdrawing energy by substituting foods with non-caloric sweeteners could subsequently lead to overcompensation of food intake later on, which may even exceed the energy deficit induced by the sweetener and therefore cause a positive energy balance.

g) *Loss of signal fidelity*. Certain sensory properties of food influence the metabolic response required for each product. So, if the sensory input of sweetness by non-caloric sweeteners leads to an inaccurate or inconsistent prediction, energy regulation could be affected and lead to a positive energy balance due to excessive intake caused by these signals.

h) *Activation of reward systems*. It's possible that the improved palatability of sweetened products could play a role in stimulating food reward.

i) *Learning through the positive reinforcement of sweet flavours*. It refers to the possibility that repeated exposure to non-caloric sweetener can perpetuate a preference for sweet products in the diet, including those sweetened with caloric sweeteners¹⁰.

Sweeteners and their effect on body weight

For many years, weight management has been one of the main reasons for the extensive use of sweeteners as part of a regular diet. However, from 1986 onwards doubts surfaced over the possible effects on weight gain, according to the results of surveys conducted by the American Cancer Society (ACS)¹³.

Furthermore, in many instances an increase in use has not been accompanied by a reduction in nutritive sweeteners, which they are intended to replace with the aim of reducing calorie intake, consequently intake remained unchanged. The extent to which foods are chosen where nutritive sweeteners have been replaced by other non-caloric sweeteners should also not be overlooked, as, in the majority of cases, this leads to an increase in fat and protein content which may be trying to compensate for the calorie deficit caused by the food containing non-caloric sweeteners.

Changes in appetite regulation are responsible for changes in energy intake and therefore managing body weight. So, all the mechanisms of appetite regulation mentioned in the previous section may be involved in weight gain.

Changes in neural response mechanisms have been proposed as a possible explanation for the weight gain associated with sweetener use. It is known that the act of eating and the satisfaction derived from it, is the result of sensory stimulation from foods after ingestion. In humans, when a food is swallowed the taste,

which is detected by receptors found in the oral cavity, ascends via the thalamus and reaches the area of the anterior insula frontal operculum as well as the frontal orbital cortex. Similarly, the amygdalae also make connections, via the taste pathway, at every level. Last but not least, the role played by the mesolimbic dopaminergic system is discussed, as it is responsible for recognising the stimuli and the pleasure/satisfaction sensation following the ingestion of food.

Following studies on rats, it has been demonstrated that the hypothalamus mediates the postprandial effect on the food reward system, given its different functions in the secretion of various peptides which regulate energy, osmotic equilibrium and behaviour in the presence of food. We increasingly have more evidence which shows that artificial sweeteners don't activate the food reward cascade in the same way as natural sweeteners, as it appears that the lack of calories suppresses the post-ingestive component. Moreover, the gustatory branch activation mechanism also differs in each case.

The sweet taste of non-caloric sweeteners may boost the appetite and dependency on such flavours, and there is a high correlation between the repeated exposure to a flavour and the degree of preference for it. Research in this field, but on the reduction of fat and salt in the diet, showed how with reduced exposure, the group's preference for these products diminished, so a possible theory has been put forward that the presentation of unsweetened diets could be one of the keys to reducing sugar consumption and consequently reverse the obesity epidemic¹⁶.

These aforementioned hypotheses which have already been brought to light in previous studies, are also reflected in the research carried out by the American Academy of Nutrition and Dietetics³, where it was observed that the sweeter a product is, the higher the consumption of food or drink will be. For the test subjects, the effect on appetite, caused by the repeated exposure to sweeteners, is due to an interruption of the hormonal and neurobehavioral pathways responsible for controlling hunger and satiety.

With regard to the risks of cardiovascular disease associated with weight gain, the prospective observational studies which exist to date only allow the identification of casual associations, but they are not in any way determinants; in many cases reverse causality is very plausible for some of the significant associations observed¹⁰.

Sweeteners and diabetes

The potential benefits attributed to non-nutritive sweeteners for diabetics are the reduction in calories and carbohydrates which improve weight control and blood sugar respectively.

Various studies have shown that the use of non-caloric sweeteners does not seem to affect blood sugar or plasma lipid levels in adults with diabetes; it has not been researched sufficiently in children¹⁷. Diabetics should take into account total carbohydrate intake in order to improve blood sugar control. It has been suggested that blood sugar and weight control can be improved by using non-caloric sweeteners better than with sugary foods.

Two transporters are involved in the control of glucose absorption through the gut wall, the sodium-glucose linked transporter (SGLT1) which has an active role as a transporter in the apical membrane, and the facilitated glucose transporter (GLUT2), present both in the basolateral and apical membrane. The cells responsible for their absorption are enterocytes. Sugars as well as low calorie sweeteners that may be present in the diet, increase SGLT1 mRNA, protein expression and the absorption capacity for glucose; furthermore, given the relationship between SGLT1 activity and the insertion of GLUT2 in the apical membrane, T1R3 stimulation (sweet taste receptor subunit), also promotes a greater insertion of GLUT2.

Enteroendocrine cells communicate with enterocytes by producing signals which are detected by the latter, increasing SGLT1 expression. These incretin signals include glucose-dependent insulintropic polypeptides (GIP) and GLP-1, which have numerous effects on glucose metabolism, including the stimulation of insulin release, inhibition of glucagon secretion, reduced gastric emptying and an increase in the feeling of fullness. As for the other mechanisms that have been described, the data available comes from in vitro and other short term studies, as well as studies on animals, which implies significant limitations on the extrapolation of results to human studies¹⁸.

The effects of specific sweeteners on postprandial glycaemia, insulin and blood lipids have also been studied. As a result, following the comparison of a diet rich in sucrose versus another which, in contrast, contains non-caloric sweeteners, there is a significant increase in both postprandial blood glucose and postprandial insulinaemia, and blood lipid levels in a slightly overweight but healthy population, for the group with a diet rich in sucrose¹⁹.

If we look at the latest American Diabetes Association (ADA 2013) recommendations²⁰, with a B level of evidence, it states that in the case of type II Diabetes Mellitus, patients should limit their consumption of sugary drinks without specifying the appropriate number. There are no specific ADA recommendations that include other sources of sweeteners, apart from in soft drinks, where decreased consumption is recommended. We can also add that there are no recommendations on limiting the use of other sources of sweeteners.

Information on sweetener use should be transmitted clearly during diabetic education sessions. Accurate proven information, based on the best scientific evidence available, is necessary, so that relevant decisions and recommendations concerning their consumption can be made. In this respect, it is essential to disprove the myths which frequently surrounds this issue as well as combat the disinformation/misinformation that we come across on the internet and in the media on a daily basis. Research on non-caloric sweeteners gathered by regulatory agencies (FDA) contributes to their safe use and their potential benefits in controlling blood sugar.

Sweeteners and tooth decay

A cavity is formed by the localised destruction of hard dental tissue by acidic material which comes from the fermentation processes carried out by certain pathogenic bacteria, cariogenic bacteria, from fermentable carbohydrates present in the diet. Other factors which contribute to the development of tooth decay are microbiological changes in bacterial flora, saliva composition and its pH buffering capacity, the type of sugary foods consumed and the frequency of their consumption, and the quality and regularity of oral hygiene. Among the large group of sweeteners and according to the health claims that they help prevent cavities, the sugar alcohols erythritol, D-tagatose, sucralose, and isomaltulose, have been approved for consumption⁵.

A global view of sweetener use: SWOT Analysis

Once all the matters relating to the general aspects of sweetener use and their possible metabolic effects on the body have been addressed, an overview of their use will be given, in accordance with the SWOT analysis system (Fig. 3).

Strengths

- Growing interest and the discovery of nutraceutical products with sweetening properties.
- The supposed beneficial effects of sweetener use as a whole, on metabolism, in different diseases (Obesity, diabetes, tooth decay) carving out a niche for them in the food industry.
- Non-caloric sweeteners provide sweetness without the extra calories or in the worst case scenarios, never as high as sugar.
- Large potential market demand associating them with a more balanced diet, although according to international organisation recommendations such as the FAO/WHO, the consumption of simple carbohydrates (sugars) below 10% of the total

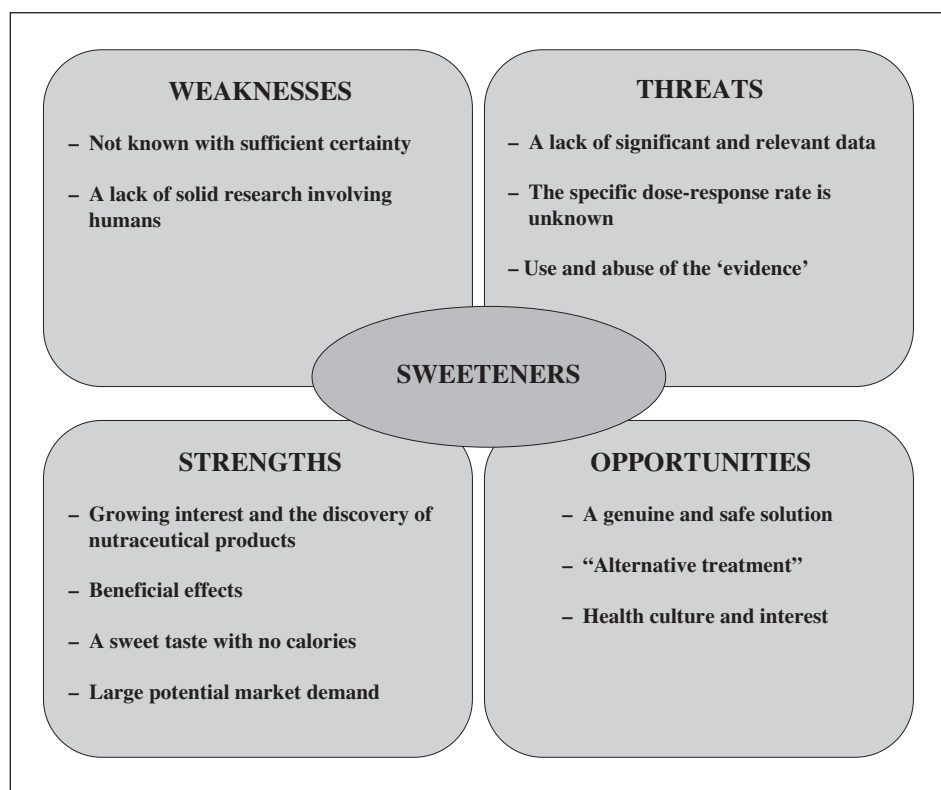


Fig. 3.—SWOT analysis on the use of sweeteners.

dietary energy is acceptable as part of a healthy balanced diet.

- They appear to help limit refined sugar consumption in the diet

Weaknesses

- The general population, including many health professionals, often lack correct knowledge on the particular characteristics of the different sweeteners available in the market, such as advising on and/or choosing a particular sweetener based on its properties.
- Even though the production-demand binomial is gradually becoming more evident in society, in the search for ‘possible solutions’ for improving health, there is actually a real lack of sound research on humans which confirms their potential benefits.

Opportunities

- They could eventually be a genuine and safe solution, taking into account an increase in chronic illnesses in society today (diabetes, obesity).
- They could become an ‘alternative treatment’, for the prevention and favourable evolution/ management of certain illnesses.

- As the years go by, the culture and interest in looking after the body and achieving optimum health grows. Included here are any criteria which could be a way of achieving this (i.e.: physical activity, specific diets, consumption of diet foods).

Threats

- A lack of significant and relevant scientific data.
- The specific dose-response rate, which clarifies the metabolic effects of their use, is unknown.
- Derived from previous ideas, use and abuse of ‘evidence’ to date in order to recommend for use.

Recommendations

Considering the existing disputes over their potential benefits and the importance and cost to public health at the moment because of the high incidence of chronic diseases (in particular obesity and its long-term consequences), sweeteners could be an alternative strategy in dietary treatment, as well as both a primary and secondary preventative measure in the treatment of obesity and its associated conditions. However, and despite the need for higher quality clinical research and while this takes places, once again moderation and the false myth of a ‘miracle food’ should not be consigned

to healthy structured eating patterns which tend towards calorie balance through a variety of natural healthy foods, that are adapted to our customs without forgetting to combine this with a healthy active lifestyle.

We need to avoid indiscriminate use of them¹³, as their potential interest as a tool for preventing excess weight or diabetes, even in the healthy population who want to take care of their health, has not been proven with evidence that supports their beneficial effects over the alternative standard caloric sweeteners².

In any case, the American Academy of Nutrition and Dietetics' position that any individual can use them safely is true, but only if they form part of a diet based on dietary recommendations and reference intakes for the general population, without forgetting, on the other hand, personal preferences and health objectives. Irrespective of non-caloric sweetener use in the diet, it is essential to control the total energy intake and increase the degree of physical activity in order to maintain body weight.

Recommendations from scientific societies (ADA, AHA) confirm that sugar alcohols and non-nutritive sweeteners are safe if daily dietary intake is within the levels established by the regulatory agencies (FDA, AESAN)⁵.

Conclusions

So far, the existing evidence on the benefits of using non-caloric sweeteners as part of the population's regular diet and nutrition, is lacking in long-term results which are of significant scientific relevance, and the majority are epidemiological studies.

There are plenty of results on their effects and benefits from studies on animals, but not so many on humans due to bias and limitations on the interpretation and extrapolation of population data. On the other hand, it is necessary to determine the exact dose-response rate, which explains the metabolic effects of their use.

Likewise, and even though sugar consumption can be limited in patients with metabolic disorders, there is no evidence that recommendations on the use of sweeteners has been sufficiently scientifically proven to recommend the supposed long-term benefits of their use.

In 2009 the AHA concluded that limiting added sugars is a core strategy for maintaining optimum nutrition and a healthy weight. Likewise and for its part, the ADA has included monitoring carbohydrate consumption (which includes limiting added sugars) in their clinical practice recommendations, as a key strategy¹⁰.

Finally, it should be noted that all non-caloric sweeteners approved for use are deemed to be safe, within permitted usage levels. Intake estimates are difficult to

assess, if you also take into consideration that, in the majority of cases, food products contain a mixture of them which makes them even more difficult to calculate. It is essential that future studies on their consumption include a sufficient number of subjects, consumers in the 95th percentile, and even other groups that may have an above average intake (e.g. diabetics) or groups with particular issues (pregnant women or children).

References

1. Anderson GH, Foreyt J, Sigman-Grant M, Allison DB. The use of low-calorie sweeteners by adults: impact on weight management. *J Nutr* 2012; 142 (6): 1163S-9S.
2. Wiebe N, Padwal R, Field C, Marks S, Jacobs R, Tonelli M. A systematic review on the effect of sweeteners on glycemic response and clinically relevant outcomes. *BMC Med* 2011; 9: 123.
3. Ng SW, Slining MM, Popkin BM. Use of caloric and noncaloric sweeteners in US consumer packaged foods, 2005-2009. *J Acad Nutr Diet* 2012; 112 (11): 1828-34.
4. Caprio S. Calories from soft drinks—do they matter? *N Engl J Med* 2012; 367 (15): 1462-3.
5. Fitch C, Keim KS; Academy of Nutrition and Dietetics. Position of the Academy of Nutrition and Dietetics: Use of Nutritive and Nonnutritive Sweeteners. *J Acad Nutr Diet* 2012; 112: 739-58.
6. Directiva 94/35/CE del Parlamento Europeo y del Consejo de 30 de Junio de 1994. Diario Oficial de las Comunidades Europeas, 10.09.94, N° L 237/3.
7. Andrew Renwick et al. Edulcorantes bajos en calorías: funciones y beneficios (monografía). International Sweeteners Association, pp. 1-31.
8. Mortensen A. Sweeteners permitted in the European Union, Safety aspects. *Scandinavian Journal of Food and Nutrition* 2006; 50 (3): 104-16.
9. Raben A, Richelsen B. Artificial sweeteners: a place in the field of functional foods? Focus on obesity and related metabolic disorders. *Curr Opin Clin Nutr Metab Care* 2012; 15 (6): 597-604.
10. Gardner C, Wylie-Rosett J, Gidding SS, Steffen LM, Johnson RK, Reader D, Lichtenstein AH; American Heart Association Nutrition Committee of the Council on Nutrition, Physical Activity and Metabolism, Council on Arteriosclerosis, Thrombosis and Vascular Biology, Council on Cardiovascular Disease in the Young; American Diabetes Association. Nonnutritive sweeteners: current use and health perspectives: a scientific statement from the American Heart Association and the American Diabetes Association. *Diabetes Care* 2012; 35 (8): 1798-808.
11. Swithers SE, Martin AA, Davidson TL. High-intensity sweeteners and energy balance. *Physiol Behav* 2010; 100 (1): 55-62.
12. Brown RJ, de Banate MA, Rother KI. Artificial sweeteners: a systematic review of metabolic effects in youth. *Int J Pediatr Obes* 2010; 5 (4): 305-12.
13. Mattes RD, Popkin BM. Nonnutritive sweetener consumption in humans: effects on appetite and food intake and their putative mechanisms. *Am J Clin Nutr* 2009; 89 (1): 1-14.
14. Pepino MY, Bourne C. Non-nutritive sweeteners, energy balance, and glucose homeostasis. *Curr Opin Clin Nutr Metab Care* 2011; 14 (4): 391-5.
15. Wu GD, Chen J, Hoffmann C, Bittinger K, Chen YY, Keilbaugh SA, Bewtra M, Knights D, Walters WA, Knight R, Sinha R, Gilroy E, Gupta K, Baldassano R, Nessel L, Li H, Bushman FD, Lewis JD. Linking long-term dietary patterns with gut microbial enterotypes. *Science* 2011; 334 (6052): 105-8.
16. Yang Q. Gain weight by "going diet?" Artificial sweeteners and the neurobiology of sugar cravings: Neuroscience 2010. *Yale J Biol Med* 2010; 83 (2): 101-8.

17. Scientific Opinion on the substantiation of health claims related to intense sweeteners and contribution to the maintenance or achievement of a normal body weight (ID 1136, 1444, 4299), reduction of post-prandial glycaemic responses (ID 4298), maintenance of normal blood glucose concentrations (ID 1221, 4298), and maintenance of tooth mineralisation by decreasing tooth demineralisation (ID 1134, 1167, 1283) pursuant to Article 13(1) of Regulation (EC) No 1924/2006. *EFSA Journal* 2011; 9 (6): 2229 [26 pp.].
18. Renwick AG, Molinary SV. Sweet-taste receptors, low-energy sweeteners, glucose absorption and insulin release. *Br J Nutr* 2010; 104 (10): 1415-20.
19. Raben A, Møller BK, Flint A, Vasilaris TH, Christina Møller A, Juul Holst J, Astrup A. Increased postprandial glycaemia, insulinemia, and lipidemia after 10 weeks' sucrose-rich diet compared to an artificially sweetened diet: a randomised controlled trial. *Food Nutr Res* 2011; 55.
20. American Diabetes Association. Standards of medical care in diabetes—2013. *Diabetes Care* 2013; 36 (Suppl. 1): S11-66.

Consumption patterns and recommended intakes of sugar

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Abstract

Sugars are sweet-flavoured carbohydrates that provide energy to the body. The adult brain uses about 140 g of glucose per day, amount which can represent up to 50 of the total number of carbohydrates consumed.

In our country the sugar in food consumption pattern remains constant, while the consumption of soft drinks has increased in the past four years. According to the national survey of dietary intake of Spain (ENIDE) (AESAN, 2011) 20% carbohydrate intake comes from the so-called sugar (sugar, chocolate and derivatives, non-dairy beverages and miscellaneous) Sugar consumption has been associated with various pathologies (diabetes, obesity, tooth decay, cardiovascular disease) but these relationships are not consistent enough.

Food information through nutritional labeling, including sugars present in food, pretend to protect the consumer health and to guarantee their right to information so they can make their own decisions with criterion.

In view of different appraisals and existing studies, and above all, in the absence of a solid scientific evidence that concrete data on which make recommendations, the best nutritional advice for the general population could be a diet varied and balanced with food and nutrients from different sources, combining such a diet with exercise and physical activity.

More specifically in terms of moderate consumption of sugar in the previous context of varied and balanced diet is perfectly compatible.

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Key words: *Consumption. Frequency. Recommendations. Labelling.*

PATRÓN DE CONSUMO E INGESTAS RECOMENDADAS DE AZÚCAR

Resumen

Los azúcares son hidratos de carbono con sabor dulce que proveen energía al organismo. El cerebro adulto utiliza aproximadamente 140 g de glucosa al día, cantidad que puede representar hasta el 50% del total de hidratos de carbono que se consumen.

En nuestro país el patrón de consumo de azúcar en alimentos permanece constante, mientras que el consumo de bebidas refrescantes presenta un aumento en los últimos cuatro años. Según la Encuesta Nacional de Ingesta Dietética de España (ENIDE) (AESAN, 2011) un 20% de la ingesta de los hidratos de carbono proviene de los denominados azúcares (azúcar, chocolate y derivados, bebidas no lácteas y miscelánea). Se ha asociado el consumo de azúcar con diversas patologías (diabetes, obesidad, caries, cardiovasculares) si bien estas relaciones no presentan consistencia en las evidencias encontradas. La información alimentaria a través del etiquetado nutricional, incluida la relativa a los azúcares presentes en los alimentos, facilitada al consumidor persigue proteger la salud de los mismos y garantizar su derecho a la información para que puedan tomar decisiones con criterio.

A la vista de las distintas valoraciones y estudios existentes y sobre todo, en ausencia de una evidencia científica sólida que arroje datos concretos sobre los que realizar recomendaciones, el mejor consejo nutricional para la población general podría ser llevar una dieta variada y equilibrada con alimentos y nutrientes procedentes de diversas fuentes, combinando dicha dieta con el ejercicio y la actividad física. De manera más concreta, un consumo moderado de azúcar (< 10% de la energía total) en el anterior contexto de dieta variada y equilibrada, es compatible.

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Palabras clave: *Consumo. Frecuencia. Recomendaciones. Etiquetado.*

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Abbreviations

AESAN: Spanish Agency for Food Safety and Nutrition.

EC: European Community.

EEC: European Economic Community.

DRI: Daily Recommended Intake

EAR: Estimated Average Requirement.

ENIDE: Spanish National Survey on Dietary Intake.

FAO: Food and Agriculture Organisation of the United Nations

FAOSTAT: Statistics Division of the Food and Agriculture Organisation of the United Nations.

FEN: Spanish Nutrition Foundation.

FSA: *Food Standards Agency*.

IOM: Institute of Medicine of the National Academies.

MAPA: The Spanish Ministry of Agriculture, Fisheries and Food.

WHO: World Health Organisation.

RDI: Recommended Daily Intake.

SENC: Spanish Society of Community Nutrition.

AAGR: Average Annual Growth Rate.

EU: European Union.

EU-27: European Union of 27 states.

UL: Tolerable Upper Intake Level.

Introduction

The term sugars has traditionally been used to refer to mono- and disaccharides¹. Sugars are used as sweeteners, because of their sweet taste, to make food and drinks palatable, to preserve foods and to bestow certain characteristics to foods, such as viscosity, texture, body and the ability to add flavours or a brown colour to them.

Although the main sources of sugar are sugar cane and sugar beet, other sugars are obtained from plants rich in starch, such as high fructose corn syrup, produced in the USA, and sugars produced in Japan from potatoes.

Sugar cane has been known to man for more than 2,500 years. Originally from Bengal and Southern China, it emerged in Europe as a result of the conquest of Persia by Alexander the Great. The Arabs introduced the plant to the Iberian Peninsula, initially reserving it for use as a condiment for flavouring stews along with salt and pepper. Later on, apothecaries reserved this condiment for preparing formulas and remedies, and soon its use became widespread, replacing honey in many cases.

The first milestone in the history of sugar beet was marked by the German scientist Andreas Marggraf in 1747, who demonstrated that the sweet-tasting crystals obtained from beet juice were the same as those from sugar cane. Sugar beet did not receive attention until a blockade of the French trade-lines during the

Napoleonic wars. In 1806, cane sugar had virtually disappeared from European shops. In 1811, French scientists presented Napoleon with two loaves of beet sugar, impressed he ordered that 32,000 hectares of sugar beet be planted and contributed to the establishment of factories.

Nowadays cane and beet sugar are produced in more than 130 countries. Global cane sugar production is two and a half times higher than beet sugar. As far as the consumption per country is concerned, India, China Brazil, the USA and Russia stand out as they represent 45.2% of global consumption for the 2010/11 sugar cycle. India and Russia showed the greatest increases in demand 8.5% and 3.3%, respectively, for that cycle.

There are six sugar-consuming regions in the world. The first of these, is found in Asia (comprised of 36 countries), with a deficit (between production and consumption) of 6.3 million tonnes and an annual consumption of 14.9kg per capita. The region with the second highest consumption comprises the 12 countries of the former Soviet Union, with a deficit of 4.8 million tonnes. In third place is North Africa with a sugar consumption deficit of 3.9 million tonnes. In fourth and fifth place are North America and Europe with a deficit of 2.8 and 2.4 million tonnes, respectively. In North America the deficit region is comprised of two countries, Mexico and the USA, with a population of 422 million inhabitants, who maintain a high sugar intake, compared to their production levels, in per capita terms the sugar consumption deficit in this region increases to 2.8 million tonnes².

The world's main sugar exporters are: Brazil, Thailand, Australia, the 27 states of the European Union (EU-27) and Guatemala, which represent 79.4% of total exports for the 2010/11 cycle. For its part, during this period Brazil exported 67.2% of its produce while Thailand and Mexico exported close to 25%.

The world's main sugar importers (by population size and/or economy) that consume more than they can produce are the USA, India, Indonesia, Russia and China, who, as a whole, purchase 29.6% of global imports. Of these countries, the USA took 6.3 of the total during 2010/11, followed by the EU with 6.0% and Indonesia with 5.8%.

For more than ten years, there has been a growth trend in global sugar production, with a recorded Average Annual Growth Rate (AAGR) of 2.3% (between the 2000/01 and 2010/11 cycles). World sugar consumption trends have remained stable from the 2000/01 to the 2010/11 cycle, showing an AAGR of 2.0% for this period. The recorded global consumption during the last sugar cycle was 158.6 million tonnes. Global sugar consumption for the 2009/10 and 2010/11 cycles increase by 2.4%, lower than recorded global production.

As a food, in our country sugar is consumed in roughly the following proportions: 30% domestic consumption and the remaining 70% industrial consumption. In total terms, Spain consumes around 1,300,000 tonnes of sugar per year, of which 300,000 are imported.

In Spain daily sugar intake per capita is 29.8g. However, this 'intake' refers to the purchase of sugar for use at home, and therefore does not include sugars contained in foods (it is based on 'purchased' not 'ingested' sugars). In this analysis, sugar consumption was greater in populations with higher socioeconomic status, in areas where there were less than 100,000 inhabitants, home workers and the elderly (> 65 years)³.

According to the National Survey of Spanish Dietary Intake (ENIDE) (AESAN, 2011) 20% of carbohydrate intake comes from so-called sugars (sugar, chocolate and its derivatives, non-dairy and miscellaneous beverages)^{4,5}.

Nutritional information for consumers

Information is one of the best methods of involving consumers so that they can understand and deal with the nutrient content of food and drinks.

*Regulation (EC) no. 1924/2006 of the European Parliament and of the Council, of 20th December 2006*⁶, on nutrition and health claims made on foods came into force to bring order to a situation which had actually been consolidating itself: the marketing of an increasing number of products with nutritional messages and healthy properties in the labelling and advertising. To some extent, the new Community Regulation has sought to address the absence of legal references in the advertising and labelling of those foods which, in addition to nourishing us, have specific scientifically proven health benefits.

The purpose of this Regulation is intended to guarantee a high level of consumer protection, by introducing scientific assessment as a necessary prerequisite to be able to make nutritional or health claims, maintaining the general principles of Community legislation in terms of the labelling, presentation and advertising of foodstuffs, generally prohibiting the use of information which could mislead the purchaser or attribute preventative or curative properties to foods.

In the same vein, it's necessary, as a general condition, that the average consumer understand the beneficial effects as they are expressed in the declaration. It's important to encourage consumers to carefully read the labels of products in their shopping baskets.

This standard applies to nutritional declarations and health claims made on any kind of commercial

communication (labelling, presentation and advertising) for foods which are delivered as such to the end consumer, intended for supply to restaurants, hospitals, schools and mass caterers.

It also affects the following forms: packaged foods, non-prepackaged foodstuffs (fresh produce, fruit, vegetables, bread) on sale to the end customer or caterer and products packaged at the point of sale at the purchaser's request or pre-packaged with a view to immediate sale.

The nutritional claims and conditions regarding sugars are laid down in the Annex of this Regulation and are defined as follows:

'Low sugar'

A claim that a food is low in sugar, and any claim likely to have the same meaning for the consumer, may only be made where the product contains no more than 5 g of sugar per 100 g for solids or more than 2.5 g of sugar per 100 ml for liquids.

'Sugar free'

A claim that a food is sugar free, and any claim likely to have the same meaning for the consumer, may only be made where the product contains no more than 0.5 g of sugar per 100 g or 100 ml.

'With no added sugars'

A claim stating that sugars have not been added to a food and any claim likely to have the same meaning for the consumer may only be made where the product does not contain any added mono- or disaccharides or any other food used for its sweetening properties. If sugars are naturally present in the food, the following indication should also appear on the label: "*Contains naturally occurring sugars*".

Regulation (EU) no 1169/2011, of 25th October on the provision of food information to consumers, with the aim of pursuing a higher level of health protection for consumers and to guarantee their right to information, was published in 2011 so that they could make informed decisions and make safer use of food, with particular regard to health, economic, environmental, social and ethical considerations⁷. The text has reinforced and updated two areas of legislation on labelling: general food products, covered by Directive 2000/13/EC, and nutritional labelling, the objective of Directive 90/496/EEC.

As far as compulsory nutritional labelling is concerned, the elements which are mandatory are: energy, fats, saturated fats, carbohydrates, sugars, proteins and salt, which should all be presented in the

same visual field. Information must be declared per 100 g or per 100 ml, to enable the products to be compared; the declaration may also include, on a voluntary basis, the amounts per portion consumed. This information can be voluntarily supplemented with the values of other nutrients such as: monounsaturated and polyunsaturated fatty acids, polyalcohols, starch, dietary fibre, vitamins and minerals.

In this regulation sugars mean “*all mono- and disaccharides present in foods, excluding polyalcohols*”. For those foods which contain sugar or added sugars and a sweetener or sweeteners authorised under Regulation (EC) no. 1333/2008, the following additional wording should appear on the label: “*with sugar(s) and sweetener(s) that accompany the name of the food. It also states, in reference to ingredient names, that the food category which corresponds to “all types of sugars” should be called “sugars”*”.

The energy value and the amounts of nutrients can be expressed, as appropriate, as a percentage of the

reference intakes that are referred to in Annex III as 2,000 kcal, 260 g of carbohydrates and 90 g of sugars (Table I).

From 13th December 2016, according to Regulation (EU) no. 1169/2011 nutritional labels will be mandatory, irrespective of whether or not foodstuffs carry nutritional declarations or health claims.

The new regulation also allows the energy value and the amounts of nutrients to be specified using other forms of expression (pictograms or symbols), provided that they meet certain criteria, such as they can be understood by consumers and they don't create unnecessary barriers for the free movement of goods.

Weaknesses

Assuming the data from the Ministry of Agriculture and Food refer to the declaration of purchases in and out of the home (hotels and catering) and not consumption, and having this data as the sole reference, it can be said that added sugar consumption in Spain has evolved over the past few years (2005-12) and there has been a decrease of around 0.5kg per person per year, which even though it was very pronounced between 2005 and 2008, has stabilised during the past five years at approximately 4 kg/persona/year, as seen in figure 1⁷.

As far as soft and fizzy drinks are concerned (Fig. 2), they behave differently. It is generally observed that the consumption of this kind of drink is increasing. In 2011 consumption was 46.5 litres per capita, which represents an increase of 1.9% compared with the previous year and 21.3% compared with 2004, the consumption in 2005 was 40.5 litres per person per year⁸.

The FAO (FSTAT) Food Balance Sheet shows the potential availability for human consumption which

Table I <i>Reference intakes for energy and selected nutrients, other than vitamins and minerals (adult)⁶</i>	
<i>Nutrient</i>	<i>Reference intake</i>
Energy (kcal)	2,000
Total fats (g)	70
Saturated fatty acids (g)	20
Carbohydrates (g)	260
Sugars (g)	90
Proteins (g)	50
Salt (g)	6

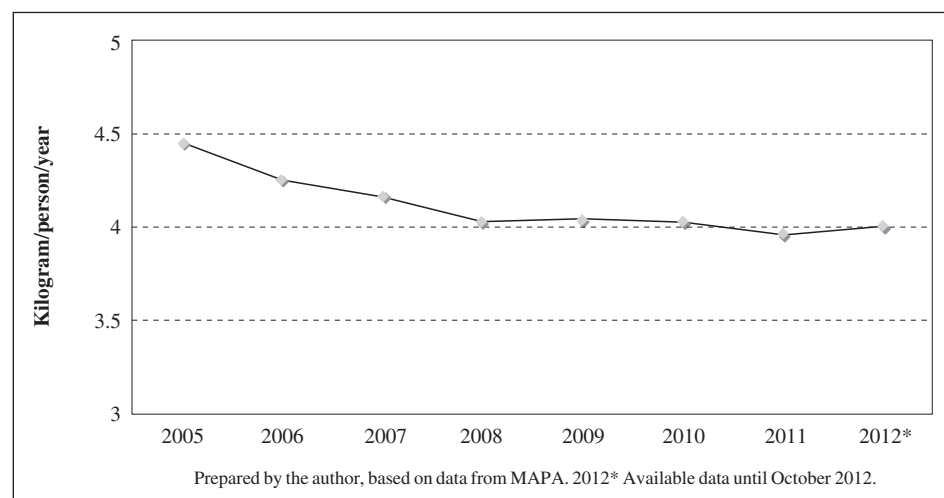


Fig. 1.—The evolution of sugar consumption in Spanish households 2005-2012⁷.

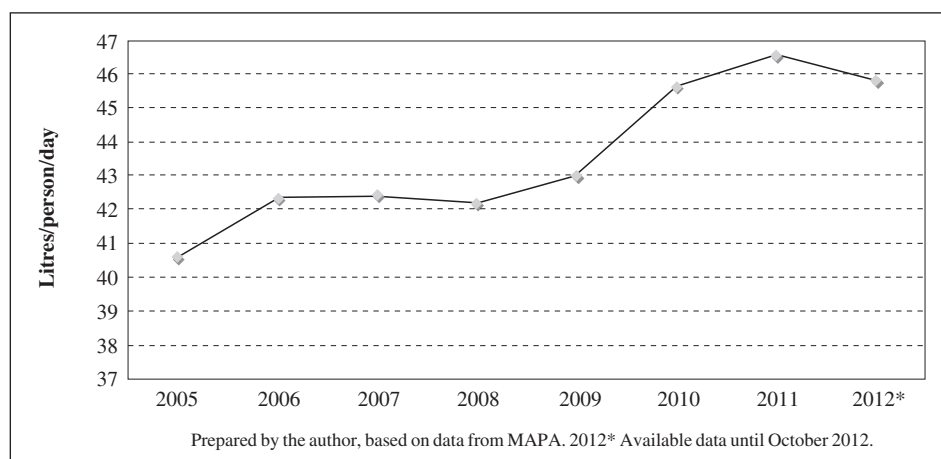


Fig. 2.—The evolution of soft and fizzy drink consumption in Spanish households 2005-2012*.

corresponds with supply sources. It's advisable to draw attention to this information on sugar availability, a figure that tends to be higher than the actual consumption, because it doesn't take sugars that are intended for non-food use, into consideration or the volume of sugars, declared in FAOSTAT data, which are exported out of the country, in the form of ingredients in processed foods. From this data we can see that for the period of 2000- 2009 there has been a decrease in availability from 32.9 kg/person per year to 25.5 kg/person per year (Fig. 3). Recent data implies availability of around 70g per person per day, which would mean 11.2% of the total energy for a diet of 2,500 kilocalories⁹.

From a nutritional point of view, sugars are not essential nutrients, as glucose can be synthesised by the body, however they are significant within the framework of a healthy diet. The lower limit of dietary carbohydrates compatible with life is apparently zero (but a minimum of 120 g per day is

advised), provided that adequate amounts of proteins and fat are consumed so that the body can synthesize it 'de novo' from them. There are traditional populations (Masai) that ingested a high fat, high protein diet containing only a minimal amount of carbohydrate for extended periods of time, and in some cases (Alaska and Greenland Natives, Inuits, and the indigenous people of the Pampas) for a lifetime after infancy¹⁰.

Other circumstances that could be considered a weakness with regard to their consumption, is the so-called glycaemic index. This concept, devised by Jenkins in 1981, is an approach to quantify the glycaemic response of a food that contains the same amount of carbohydrates as a reference food. The glycaemic index of sucrose is intermediate. It has been proposed that a high GI diet leads to an increase in serum glucose and insulin and this could cause certain pathologies. Even though this theory has not yet been proven.

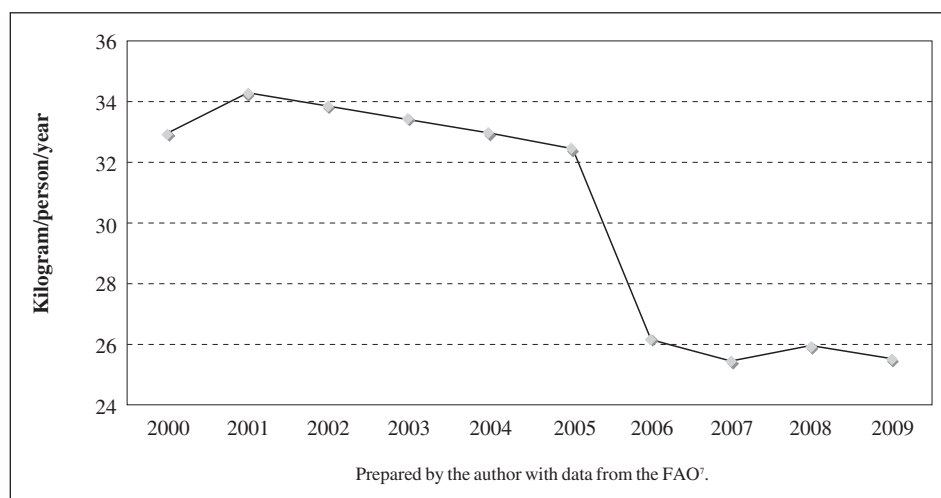


Fig. 3.—Food availability of sugar in Spain for the period 2000-2009 according to the FAO food balance sheets⁷.

Threats

Sugars have been considered a nutritional element whose abuse could lead to the displacement of micronutrients in the diet¹¹.

Despite the fact that there is currently no definitive data, increased sugar consumption can be associated with having various medical disorders¹², a relationship which was discussed in detail in another article in this very magazine.

In spite of everything, some publications and media propose that a surcharge be introduced through special taxes as a way of reducing the excessive consumption of certain sugary foods and drinks (like sugary fizzy drinks, other drinks sweetened with sugar such as juices, sports drinks, chocolate milk and sugary cereals)¹³.

Canada and some European countries have introduced additional taxes on certain sweetened foods. However Denmark, for example, who had already taken this step for high-fat products, recently withdrew this surcharge, basing their decision on protectionist arguments such as the increase in food prices that had resulted in a reduction in sales within their territory and a rise in food purchases from other countries, resulting in job losses. Most countries choose to promote nutritional education.

Strengths

However, the consumption of sugars also has its advantages that should not be overlooked. Their pleasant flavour encourages the consumption of other foods. The sweet flavour is associated with pleasure. Nutritionally, sugars are a major source of energy for human beings, with a nutritional value of 3.7-4 kcal/g depending on the compound, however, compared to other nutrients (lipids) or related substances (alcohol) they provide less energy.

Carbohydrates are important for our bodies to function, the brain needs an almost constant supply of glucose from the blood stream. It is estimated that an adult brain uses approximately 140g of glucose per day; an amount that could represent up to 50% of the total carbohydrates consumed. The cells of the central nervous system need glucose as a metabolic fuel, although they can adapt to use fat derivatives. It is also needed by those cells that depend on anaerobic glycolysis, such as erythrocytes, white blood cells and renal medulla cells.

Opportunities

In a healthy or balanced diet all kinds of carbohydrates should be represented, mono- and disaccharides

or simple sugars (10%) as well as complex sugars or polysaccharides (90%). Simple sugars enable normal blood sugar levels to be achieved rapidly which, in relatively healthy individuals and normal concentrations in the diet, helps to prevent hypoglycaemia and satisfies immediate energy requirements. On the contrary, polysaccharides, such as starch, enable blood sugars to gradually be stabilised without causing spikes in it.

Recommendations

The WHO/FAO report: *Diet, nutrition and the prevention of chronic illnesses* of 2003 states that the following ration for *total carbohydrates* is 55%-75%, the percentage of total energy available after fat and protein consumption has been taken into consideration, hence the breadth of the range, and in reference to *free sugars*, defined as all mono- and disaccharides added to foods by the manufacturer, cook or consumer, plus sugars naturally found in honey, syrups and fruit juices, would be less than 10% of total energy¹⁴. However, the same document, recognises that a figure of 10% of total dietary intake is 'controversial'.

In an annex to this report, the situation regarding the scientific evidence between 'free sugars' and certain illnesses is summarised, and confirms what the FAO had previously said, in that there is no convincing correlation between sugar intake and a series of non-communicable chronic illnesses including obesity, type 2 diabetes, cardiovascular disease, cancer and osteoporosis, pointing out instead a convincing relationship between free sugars and tooth decay, in the same way that this correlation exists with starch intake.

In 2005, the report *Dietary Reference Intakes for Energy, Carbohydrate, Fibre, Fat, Fatty Acids, Cholesterol, Protein and Amino Acids (Macronutrients)* of the *National Academy of Sciences* established¹⁰ that the recommended daily intake (RDI) for adults and children over 1 year of age is 100g of carbohydrates per day as an estimated average requirement (EAR) and 130g/day as a reference daily intake (RDI).

The DRI committee concluded that there was insufficient evidence to establish a tolerable upper intake level (UL) for total or added sugars (IOM, 2005). However, it was suggested that sugar intake level should be a maximum of 25% of total energy. This suggestion was based on data which shows that people who were on diets that were above this level of sugars were more likely to have poor intakes of certain macronutrients. It is also recognised that hypertriglyceridemia can occur with increases in total sugar intake (intrinsic and added), so the total sugar intake should be moderate in terms of added sugar intake as sugars

present in milk, dairy products and fruit are also regularly consumed¹⁵.

In 2006 the American Heart Association recommended that the consumption of foods and drinks with added sugars should be minimised. In 2009 a proposal was put forward to set an upper limit (UL) for added sugar intake in the daily diet of the American population. It was established that women should not consume more than 100 kilocalories of added sugar per day (25 grams), and for men no more than 150 calories (around 37.5 grams). This level is proportional to the daily energy required for a person to maintain a healthy weight.¹⁶

The Spanish people's nutritional goals established by the Spanish Society of Community Nutrition (SENC)¹⁷, reflect the existence of a major controversy over whether the energy percentage from sugars should be measured and suggest that, rather than measuring the intake limit for sugars and sugary foods, qualitative guidelines should be issued. In line with the etiopathogenesis of tooth decay, it was decided that sweets and dessert consumption should be limited to four or less times a day (intermediate objective) and three times a day as a final objective, suggesting as a final objective that they should not exceed 6% of total energy intake.

In reference to soft drinks, SENC and the Nutrition and Physical Activity Observatory (2008) subscribed to the 'Zaragoza Declaration' on water, health and hydration which considered five groups of drinks and placed *carbonated and non-carbonated soft drinks, sweetened with sugar or fructose* in group 4. In the healthy hydration pyramid (SENC) they were only recommended for consumption once a week.

The publication of the 2011 ESFA document on 'The Opinion on Dietary Reference Values for Carbohydrates' states that, with the current level of evidence, it's not possible to establish a threshold for the dietary reference intakes for sugars in the context of preventing a range of conditions, with specific reference to obesity.

Conclusions

In view of the different assessments and existing studies, and above all, in the absence of solid scientific evidence that provides concrete data on which recommendations to make, the best nutritional advice for the general population would be to eat a balanced and varied diet with foods and nutrients that come from different sources, combining this diet with exercise and physical activity. In this context moderate consumption of sugar could be considered.

References

1. FAO/WHO (Food and Agriculture Organization/World Health Organization), 1998 Carbohydrates in human nutrition. (FAO Food and Nutrition Paper-66) Rome: FAO.
2. Secretaría de Economía. Dirección de industrias básicas. EE UU de México. 2012. Análisis de situación económica, tecnológica y de política comercial del sector de edulcorantes en México. Visitado en: http://www.economia.gob.mx/files/comunidad_negocios/industria_comercio/Analisis_Sectorial_Mercado_Edulcorantes.pdf
3. Valoración de la Dieta Española de acuerdo al Panel de Consumo Alimentario. Ministerio de Agricultura Pesca y Alimentación (MAPA) / Fundación Española de la Nutrición (FEN). Madrid: Ministerio de Medio Ambiente y Medio Rural y Marino, 2008. Visitado en: http://www.magrama.gob.es/es/alimentacion/temas/consumo-y-comercializacion-y-distribucion-alimentaria/valoracion_panel_tcm7-7983.pdf
4. Agencia Española de Seguridad Alimentaria y Nutrición (AESAN), 2011. Encuesta Nacional de Ingesta Dietética Española 2011. http://www.aesan.msc.es/AESAN/docs/docs/notas_prensa/Presentacion_ENIDE.pdf
5. Agencia Española de Seguridad Alimentaria y Nutrición (AESAN), Evaluación Nutricional de la Dieta Española I. Energía y Macronutrientes sobre datos de la Encuesta Nacional de Ingesta Dietética (ENIDE) http://www.aesan.msc.es/AESAN/docs/docs/evaluacion_riesgos/estudios_evaluacion_nutricional/valoracion_nutricional_enide_macronutrientes.pdf
6. Reglamento (CE) n° 1924/2006 del Parlamento Europeo y del Consejo, de 20 de diciembre de 2006. 30.12. 2006 L404. Visitado en: <http://eur-lex.europa.eu/LexUriServ/LexUriServ.do?uri=OJ:L:2006:404:0009:0025:ES:PDF>
7. Reglamento (UE) N° 1169/2011 del Parlamento Europeo y del Consejo de 25 de octubre de 2011 sobre la información alimentaria facilitada al consumidor y por el que se modifican los Reglamentos (CE) n° 1924/2006 y (CE) n° 1925/2006 del Parlamento Europeo y del Consejo, y por el que se derogan la Directiva 87/250/CEE de la Comisión, la Directiva 90/496/CEE del Consejo, la Directiva 1999/10/CE de la Comisión, la Directiva 2000/13/CE del Parlamento Europeo y del Consejo, las Directivas 2002/67/CE, y 2008/5/CE de la Comisión, y el Reglamento (CE) n° 608/2004 de la Comisión.
8. Consumo alimentario en España mes a mes Año 2001 a 2012. Visitado en: <http://www.magrama.gob.es/es/alimentacion/temas/consumo-y-comercializacion-y-distribucion-alimentaria/panel-de-consumo-alimentario/ultimos-datos/>
9. Organización de las Naciones Unidas para la Alimentación y la Agricultura (FAO). Hojas de balance alimentario, 2009. Visitado en: <http://faostat.fao.org/site/368/default.aspx#ancor>
10. Food National Board, 2005. *Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids* (2002/2005). A Report of the Panel on Macronutrients, Subcommittees on Upper Reference Levels of Nutrients and Interpretation and Uses of Dietary Reference Intakes, and the Standing Committee on the Scientific Evaluation of Dietary Reference Intakes. <http://www.nap.edu/openbook.php?isbn=0309085373>
11. Farris RP, Nicklas TA, Myers L, Berenson GS. Nutrient intake and food group consumption of 10-year-olds by sugar intake level: The Bogalusa Heart Study. *J Am Coll Nutr* 1998; 17: 579-85.
12. Johnson RJ, Segal MS, Sautin Y, Nakagawa T, Feig DI, Kang DH, Gersch MS, Benner S, Sánchez-Lozada LG. 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* 2007; 86 (4): 899-906.
13. The toxic truth about sugar. Lustig RH, Schmidt LA, Brindis CD. *Nature* 2012; 482: 27-9, doi:10.1038/482027.

14. OMS/FAO: Dieta, nutrición y prevención de enfermedades crónicas de 2003. Informe Técnico. OMS, Ginebra, 2003. Visitado en: http://whqlibdoc.who.int/trs/WHO_TRS_916_spa.pdf
15. Raben A, Vasilaras TH, Moller AC, Astrup A. Sucrose compared with artificial sweeteners: different effects on ad libitum food intake and body weight after 10 wk of supplementation in overweight subjects. *Am J Clin Nutr* 2002; 76: 721-9.
16. Johnson RK, Appel LJ, Brands M, Howard BV, Lefevre M, Lustig RH, Sacks F, Steffen LM, Wylie-Rosett J and on behalf of the American Heart Association Nutrition Committee of the council on Nutrition, Physical Activity, and Metabolism and the Council on Epidemiology and Prevention. Dietary sugars intake and cardiovascular health: a scientific statement from the American Heart Association. *Circulation* published online Aug 24, 2009: DOI: 10.1161/CIRCULATIONAHA.109.192627.
17. Objetivos poblacionales para la población española. Consenso de la Sociedad Española de Nutrición Comunitaria, 2011. *Rev Esp Nutr Comunitaria* 2011; 17 (4): 178-99.

Sugar at different stages in life; from childhood to old age

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Abstract

The chapter reviews and updates the role of the different types of sugar along the life cycle, mainly during infancy and aging, but also for physiological situations such as pregnancy and breastfeeding. Moreover, several examples from infancy to the elderly illustrate that a moderate consumption may be considered as adequate within the context of a healthy diet and active life.

In addition, the importance of sugar to provide palatability to the diet is also revised and attempted. The consequences of low sugar consumption (e.g. glucose) are also evaluated, but also the potential hazard effects of high and prolonged intakes at the different ages or pregnancy, as well as its association with risk factors for chronic diseases.

Finally, recommendations are given for adequate consumption to serve as tools for a benefit/risk evaluation at individual level and for public health strategies.

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Key words: *Carbohydrates. Sugars. Infancy. Adolescence. Aging. Pregnancy.*

EL AZÚCAR EN LOS DISTINTOS CICLOS DE LA VIDA; DESDE LA INFANCIA HASTA LA VEJEZ

Resumen

Se revisa la importancia que tienen los diferentes tipos de azúcares consumidos a través de la dieta y en diferentes fuentes alimentarias en las etapas de la vida y situaciones fisiológicas especiales, y como el consumo moderado de azúcar es compatible con una dieta equilibrada y estilos de vida activos.

Se describe también su función en el disfrute y placer de comer, como uno de los pilares básicos también de una alimentación equilibrada. Al mismo tiempo, se evalúan los riesgos del consumo muy insuficiente, principalmente en lo referido a un bajo aporte de glucosa en etapas críticas de la vida, así como las potenciales consecuencias negativas sobre el estado de salud en el ciclo vital cuando la ingesta es excesiva, y la asociación con factores de riesgo en enfermedades crónico-degenerativas, o con complicaciones en el embarazo.

Finalmente, se aportan recomendaciones para un correcto consumo desde el punto de vista nutricional que constituyan, finalmente, herramientas válidas para una evaluación beneficio/riesgo a nivel individual o desde el punto de vista de la salud pública.

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Palabras clave: *Hidratos de carbono. Azúcares. Infancia. Adolescencia. Envejecimiento. Embarazo. Gestación.*

Abbreviations

FAO: Food and Agriculture Organisation of the United Nations

WHO: World Health Organisation

EFSA: European Food Safety Authority.

IOTF: International Obesity Task Force

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Introduction

Barb Stuckey states in her book, "*Taste, What You're Missing*", that *the sweet taste resonates loudly in our subconscious and, what is more, that it's very difficult for someone to try sugar and say that it doesn't taste good*. This statement is valid for all age groups and physiological circumstances, and two important considerations should be made which are very valid for the specific topic that will be tackled: Good nutrition should be based on an inseparable binomial, health and pleasure, with logical exceptions, such as artificial feeding in hospital or very high dependency; secondly, we will require, at different stages in life, sufficient information and tools that will enable us to assess the benefits versus the risks of the regular consumption of

certain foods and drinks, to ultimately achieve a balanced, healthy and pleasurable diet.

The main function of sugar is to provide our body with the energy that it needs so that the different organs, such as the brain and muscles, function; an essential function throughout life and its physiological circumstances. The brain alone is responsible for 20% of energy consumed from glucose, although it is also a essential source of energy for all of the body's tissues. If it falls the body starts to suffer from certain disorders: weakness, tremors, mental dullness and even fainting (hypoglycaemia). Such critical and anabolic ages of cognitive expansion, i.e. infancy or adolescence, serves as an example: one of the most common mistakes when it comes to food is avoiding or not eating enough breakfast, when in fact it is the most important meal of the day. Breakfast should provide the energy required to start our daily activities, as at this time our blood sugar is at its lowest. Scientific evidence shows that breakfast should provide a quarter of daily calories and nutrients. For this reason moderate sugar consumption should be included, along with other foods, not only for its energy input but also because it sweetens and brings flavour to foods which make them easier to eat, as well as drinks which at this point transport essential nutrients. This previous statement is equally valid for population groups which are proportionally higher in our country, the elderly, pregnant or breastfeeding women, or sportspeople who require more energy. Throughout this chapter other examples are illustrated, while, as with other dietary components, what may be beneficial if we follow nutritional recommendations, can clearly become harmful when consumption is excessive or lifestyles are inappropriate.

Sugar consumption is particularly important because it allows glycogen stores to be increased and replenished, both in the muscles and the liver. Whether the work is physical or intellectual, sugar consumption is still advisable in adulthood. It is a food that provides energy which is rapidly assimilated by the body, enabling a fast recovery for people who have a physically demanding working day, although the actual reality is that jobs and our general lifestyles are becoming more sedentary, a situation which involves the need to adjust the total energy intake to energy expenditure. Therefore it's essential to provide consumers with the necessary tools to better understand the nutritional aspects of the foods they eat by means of correct food labelling, so that by scrutinising them they can choose the products which are more suitable. It is very common for women to follow some kind of low calorie diet. In this case it's very important to know the benefits of sugar consumption for a successful diet. It's high level of palatability contributes to the success of any weight loss regime, because it encourages the consumption of key foods that are part of any balanced diet. Most weight loss

diets, for example, no matter how well they are planned from a nutritional point of view, fail shortly after they have started because they are not appetising as the importance of one of the main senses, taste, is often left out. Also the pleasure of eating is particularly important in the elderly population as their sense of taste and smell declines, and requires a higher concentration of sugar to detect the same sensation of sweetness, provided that this does not involve an increase in the risk of conditions such as cardiovascular disease, type 2 diabetes or excess body weight, which is also reflected in this chapter. In this respect, sugar consumption for this population group increases satisfaction when it comes to eating. In short, it's a matter of bringing this pleasure back to the elderly so that they can enjoy the great benefits of eating, which contributes to making their lives more pleasant. These sensory problems also affect an individual's nutritional status, which may lead to a reduction in food and energy intake. This is where sugar plays an important role, boosting the consumption of other foods like yoghurts, milk, fruit, etc., and of course the nutrients and bioactive components they contain.

Due to all the above, below is a summarised but detailed *road map* of the role of sugar, its benefits and risks, throughout the different stages of life as well as in certain special physiological situations and predisposing risk factors for certain disorders associated with food, without ever forgetting that the individual should be free to make decisions about their own diet; self-reliance and willpower should be maintained until the eating process has finished.

Weaknesses

General

The exact daily requirements for carbohydrates in human nutrition are not currently well-defined. Nevertheless, the recommendations and objectives in our Western countries indicate that they should provide between 55 and 60% of total dietary energy, recommendations which are not currently being met in Spain, unlike the situation in our society only a generation ago.

Foods with a high glycaemic index, such a particular type of cakes and pastries, *snacks* or certain drinks, are rapidly digested, their glucose reaches the blood stream in a short space of time, causes spikes in blood sugar and requires larger quantities of insulin.

Nutritional density of the diet

One of the most talked-about effects of the excessive consumption of sucrose, is the reduced consumption of other nutrients, especially macronutrients, which at

Table I
The main features of SWOT analysis (Strengths, Weaknesses, Opportunities, Threats) during the different stages of life

WEAKNESSES

- During pregnancy an excessive increase in sugars in the diet could predispose women to excessive pregnancy weight gain and an overgrowth of the foetal placenta. This situation could also be linked to the development of gestational diabetes.
- Similarly, it could also contribute to higher incidences of tooth decay.
- During childhood and adolescence an excessive consumption of sugars in the diet could displace the intake of other nutrients as well as increasing body weight and the incidence of tooth decay and possible hyperactivity disorders.
- During adulthood, the excessive consumption of sugars could displace the intake of other nutrients, lead to excess weight or obesity, increase insulin resistance, increase the risk of cardiovascular disease, as well as tooth decay.
- In the elderly, an increase in sugars in the diet could lead to nutritional imbalances, obesity and an increased risk of tooth decay or developing diabetes.

STRENGTHS

- During pregnancy it produces an increase in glucose available to the foetus, which constitutes this sugar as an essential and preferential substrate.
- In newborns, as a result of pancreatic amylase deficiency, it is recommended that milk formulas for infants contain lactose as the main carbohydrate and the addition of glucose is also permitted as it can be hydrolysed on the enterocyte's borders.
- During sports activities, they enable blood sugar to be maintained during exercise and muscular glycogen to be replenished as quickly as possible following exercise.
- In the elderly they bring about drastic changes in the perception of basic tastes and in particular sustain the sweet flavour, which is very important for maintaining appetite and the transportation of nutrients that are essential at this stage in life.
- The moderate consumption of sugar, in the context of a balance diet and active lifestyle, is a component which makes healthy food more enjoyable.

THREATS

- The perception that carbohydrates are fattening, harmful or non-essential has meant that their contribution to the diet's energy content ('calorie profile') decreases when purchasing power increases, and vice versa. Against this background, polysaccharide consumption has decreased the most, whereas proportionally sugars like sucrose or fats and proteins have increased.
- Changes in the consumption of fresh foods in favour of processed foods with high quantities of added sugars.
- A reduction in adherence to the Mediterranean diet.
- The vulnerability of the youngest members of society to advertising messages.

OPPORTUNITIES

- Nutritional education campaigns to raise awareness and to circulate the message that the moderate consumption of sugar is compatible with a nutritionally balanced diet containing the required micronutrients and dietary fibre.
- With regard to preventing tooth decay, effective preventive measures have to be put into place and not just stopping the consumption of sucrose and other sugars, as it has been observed that, with appropriate oral hygiene, dietary composition has a minimal effect on the incidence of tooth decay.
- The use of low calorie or non-caloric sweeteners as a substitute for added sugars to reduce the total energy intake, in the event of excessive consumption, particularly in overweight or sedentary individuals.

certain stages in life lead to nutritional imbalances that could compromise health. This is due to the fact that sugar mainly provides energy and therefore some authors have suggested that its high consumption could displace other nutrients in the diet, resulting in a nutritionally unbalanced diet¹. However, this view had not been verified by the few research studies that have been carried out using suitable methodology², and have demonstrated that both the intake of energy and of other nutrients are above the recommendations when the various increases in sugar intake during childhood and adolescence are assessed, therefore, they admit that it is difficult to determine the dose of sugar that could jeopardise children's health and the claims could only be justified, where effects were mentioned, for the highest levels of consumption.

During pregnancy, the mother's diet, especially the type and content of carbohydrates, affects both the mother and the foetus, since it had been proposed that foods containing simple sugars, as is the case with sucrose (with a medium glycaemic index) can predispose them to excessive pregnancy weight gain and an overgrowth of the foetal placenta, that could even cause foetal macrosomia in newborns, which, in the majority of cases, is a result of maternal or gestational diabetes. On the other hand, low-GI diets do not reduce the incidence of macrosomia, but they do have a significant positive effect on the mothers' pregnancy weight gain and glucose intolerance³. Therefore, the excessive consumption of foods with an elevated simple sugar content are not recommended during pregnancy, with the exception of foods that

mainly contain fructose (fresh fruit that are ripe enough).

In the case of elderly people, the situation may be very different, as the moderate consumption of simple sugars could even help to stimulate the appetite and could be a valid tool, provided that they are consumed in moderation, for transporting different nutrients that are of great interest to this age group (i.e. calcium or Vitamin D which are consumed in sugared dairy products). Once again, which has been mentioned in the introduction to this chapter, from the scientific evidence it is too simplistic to consider the negative or positive effects of a nutrient in isolation and in a consistent manner for all age groups and physiological situations.

Excess weight and obesity

The prevalence of excess weight and obesity has increased over the past few years in the majority of so-called industrialised countries and those in transition. It is estimated that around 200 million children of school age are overweight and around 50 million are obese according to the IOTF (International Obesity Task Force), due to excessive energy intake from products that have a high energy content and a low nutrient density, but mainly due to the current inability to compensate by means of adequate energy expenditure.

Carbohydrates are macronutrients that provide our bodies with energy, therefore excessive consumption of them can result in the disorders and pathologies that have previously been mentioned, although there is no clear evidence that changing the proportion of total carbohydrates in the diet is the final deciding factor on energy intake. What there is growing evidence of is the excessive consumption of sugary drinks (which contain sucrose or a mixture of glucose and fructose), along with inactive lifestyles and an increase in total fat consumption, which is associated with increased energy consumption, weight gain and the development of metabolic and cardiovascular disorders⁴. Fortunately, there are a large amount of reformulated drinks currently available on the market, with a very low or virtually zero energy content, which together constitute more than 30% of the total market. What is more, a systematic review carried out by Malik et al.⁵ showed a positive association between the excessive consumption of sugary drinks and weight gain in children and adults. The most recent data available (2012) on the nutritional interpretation of the Spanish diet produced by the Spanish Nutrition Foundation, via the Food Consumption Panel, shows that for the group of non-alcoholic drinks, the contribution to the total energy consumed by the adult population is approximately 3%.

Furthermore, it has been observed that excessive pregnancy weight gain and the mother's eating habits

during pregnancy, especially the mother's consumption of high sugar foods or drinks, may have an impact, increasing her offspring's weight from the earliest stage of life⁶. However, it's a controversial issue, as an analysis⁷ that compares and contrasts obesity trends, both in children and adults, and sugar consumption in Australia over the past 30 years with data from the USA and the UK, revealed an inverse relationship between sugar consumption and the prevalence of obesity, i.e. there is an increase in this disease with a reduced consumption of refined sugar. So, a reduction in sugary drink consumption may not be an effective and adequate strategy for reducing obesity. Therefore, there is still no clear evidence that involves sugar directly in obesity, although it has to be taken into account that an excess of energy also comes from simple carbohydrates or any other energy-providing macronutrient and is already associated with a sedentary lifestyle, which would benefit this condition if it is not offset with adequate energy expenditure.

Insulin resistance and diabetes

Diabetes mellitus is a syndrome that is characterised by chronic hyperglycaemia which is accompanied by changes in carbohydrate, fat and protein metabolism. Insulin resistance can be modulated by dietary factors, and of these, carbohydrates are the nutrients which have the greatest impact on blood sugar. In general, at all stages of life, an intake equivalent to 50-60% of total energy consumed in the form of complex carbohydrates is recommended, leaving simple carbohydrates (monosaccharides and disaccharides) consumption to be reduced to those found in milk and dairy products (lactose) and fruit and vegetables (sucrose and fructose). However, the current reality of the Spanish diet shows a percentage that is well below recommendations, slightly higher than 40%. Nevertheless, the most recent recommendations for diabetic patients indicate that the amount of carbohydrates and monounsaturated fatty acids should account for between 60-70% of total calorific value. For this reason, the total amount of carbohydrates in a meal is considered more important than the type or source of food⁸.

Generally, there are no studies that directly link sugar consumption with diabetes, although a strong association has been observed between the excessive and prolonged consumption of sugary drinks and an increased risk of metabolic syndrome and type 2 diabetes⁹. On the other hand, it was observed that high rates of type 2 diabetes are connected to changes from more traditional diets to those that are more cariogenic and high in fat.

Meanwhile, during pregnancy, gestational diabetes mellitus can arise which is an intolerance to carbohydrates of varying severity, irrespective of the type of

treatment used to achieve metabolic control, whether it is diet or insulin, whether it persists or not once the pregnancy has finished^{4,10}. Therefore, gestational diabetes is actually 'prediabetes' and in most cases is the early stage of developing type 1 or 2 diabetes. So every woman diagnosed with gestational diabetes is a subsidiary of diabetic treatment¹¹. A pregnant diabetic's diet should be neither low calorie nor restrict carbohydrates. Only refined sugars and products that contain them in large quantities (cakes, sweets, jams and soft drinks) should be avoided or at least limited, in accordance with energy requirements at different stages in life and for different physiological circumstances.

Dental health

The prevalence of tooth decay in the Spanish population is approximately 36.7% at 6 years of age, increasing to 45% at 12 years of age and 54.8% in 15 year old adolescents, from the age of 35 tooth decay is widespread, affecting between 92 and 94% of the adult population, according to the results of the latest national epidemiological survey conducted by the General Council of Odontology and Stomatology in 2010¹⁴. Furthermore, today we know that tooth decay can even affect the elderly. In fact, the Spanish Oral Health Study (2010) has shown that in people over the age of 65%, 94% have an average of fourteen decayed teeth, which are not treated in the majority of cases. Nowadays we know that the prevalence of tooth decay is more related to age, social class and hygiene levels with fluoride.

The frequent consumption of simple carbohydrates is significantly associated with a higher risk of tooth decay¹⁵. Likewise, the consumption of sugary soft drinks is also linked to an increase in both the prevalence and incidence of this oral process¹⁶. However, this relationship is complex since it is not just sugars that contribute to the development of tooth decay, due to the fact that this process is also interconnected with the frequency of tooth cleaning, the use of fluoride, as well as saliva composition, so a simplistic and exclusive relationship associating tooth decay with sugar consumption is not scientifically admissible.

Hyperactivity

From the 1970s until the 1990s it was argued that sugars were involved in hyperactivity among young children, described as Attention Deficit Hyperactivity Disorder. It has been observed that the research studies that proposed there was a link between sugar and hyperactivity had problems with their methodology¹⁷. Therefore Wolraich et al.¹⁸ published a meta-analysis which concluded that sugar does not affect children's

behaviour, although it does not rule out the slight effect of sugar on a subset of children, with special characteristics or risk profiles.

Threats

In addition to their regulatory and structural role, carbohydrates are the main source of energy, as well as the cheapest and most abundant, at the same time as being the quickest and most easily obtainable by our metabolism. However, the widespread perception that carbohydrates are fattening, harmful and non-essential, which effectively means that, in general, their contribution to the diet's energy content ('calorie profile') decreases as purchasing power increases, and vice versa. This phenomenon has also been observed in Spain over recent years. In this inadequate calorific profile, it is complex carbohydrates that are reduced the most, while, proportionally, certain carbohydrates such as sucrose or fats and proteins are increased. For the nation as a whole, carbohydrate intake amounts to around 300 g/day, which represents 40-45% of total calories.

During pregnancy of the nutrients that the mother has to continuously provide to the foetus via the placenta, glucose is quantitatively the most important, followed by amino acids, so much so that the foetus' metabolism and development depend directly on the nutrients that come from the mother. As a result of the high amounts of glucose that the foetus needs from its mother, this can lead to the development of hypoglycaemia during the third trimester of pregnancy, especially when fasting. Furthermore, during this physiological circumstance changes in eating habit usually occur, cariogenic diets become more important in relation to different foods, which could result in a substrate for bacteria, although understandably for the duration of this physiological state, it could in any case be considered a transitory phenomenon of little relevance.

The development and industrialisation of our countries is leading to changes in eating habits, magnified by the economic crises that have been happening in recent years. The changes in consumption from fresh foods to very processed foods, with high quantities of added sugars, a reduction in adherence to the Mediterranean diet, the vulnerability of our youngest members of society to advertising messages, amongst other things, give rise to nutritional risk factors in the entire population.

Similarly, the incorporation of adults into the world of work, the stress caused by an unhealthy lifestyle, a reduction in physical exercise, possible scenarios that may lead to anxiety states or poor eating habits, are some of the most important factors that can lead to weight gain through the abuse of processed foods with a high percentage of calories, saturated fats or simple sugars.

Furthermore, the energy cost per nutrient in processed foods that can provide more energy, more total fat and simple carbohydrates, may be considered a threat, an even more in times of economic crisis such as the current one which is being experienced throughout Europe. These lifestyle could, on a long-term basis, cause unfavourable situations for adults through an increase in insulin resistance, an accumulation of glucose in the body's deposits or the presence of diabetes mellitus in the population (one of the most common illnesses, together with high blood pressure, at this age).

On the other hand, the aging process affects the metabolism of nutrients, so that the diet should be perfectly tailored to each elderly adult's situation. If we add to this the inability or unwillingness to engage in physical exercise, the presence of illnesses or polymedication, this can dramatically increase the onset of hyperglycaemia, changes in insulin resistance or the development of diabetes.

Strengths

In general carbohydrates are the main source of energy, which are easier to obtain and metabolise, and they are better value for money, which could be of interest in certain situations and for vulnerable or marginal groups of the population.

Carbohydrates can be stored and used when the body needs energy, turning them into glycogen in the liver and muscles, or converting them to fat. On the other hand, and by no means less important, they are the main source of energy for the nervous system and the blood cells, which is certainly an essential aspect of all stages and physical situations in life, but especially in pregnancy, for newborns, athletes or the elderly. From a cognitive point of view, it is considered that glucose can primarily improve the short-term storage and retrieval of information. Also in old age, with widespread memory loss, adequate glucose levels help to maintain it. And of course, a satisfactory performance in learning tasks, as is the case for children and adolescents, can be stimulated by adequate glucose levels.

Carbohydrates have an anti-ketogenic effect, at the same time as being protein-saving. Furthermore, monosaccharides can be produced from certain amino acids and glycerol; however, more than 100g/day of this nutrient is needed in order to avoid a high protein catabolic process, the possible increase in ketones or cation loss.

Due to Spain's location, the Mediterranean diet is the best available example of a traditional diet; a great deal of interest has also been generated both inside and outside its geographical area, thanks to different epidemiological studies which have shown that the Mediterranean diet is associated with lower mortality and lower rates of cardiovascular disease. In the first

few years of the new millennium data showed that Mediterranean countries obtained around 50% of their total dietary energy from carbohydrates. Despite the reduction of carbohydrate intake compared with the sixties in countries like Greece, Spain or Italy, the intake of simple sugars is still well below 10% of the total caloric value of the diet¹⁹. When more carbohydrates are consumed, less fats are eaten, and it must be considered that the energy value per gram of nutrients is less than half, an aspect which is also important in the design of adequate energy density diets.

Furthermore, sugar has properties that can have a positive effect on the diet, such as appetite suppression, i.e. it has a certain satiety potential. In addition, it is also well known that the presence of sugar in the preparation of recipes improves their palatability, which is very important in the elderly.

In relation to the strengths that sugar presents during the different stages of life, the following should be highlighted:

- During pregnancy it produces an increase in glucose availability for the foetus, which constitutes this sugar as an essential and preferential substrate.
- In newborns, as a result of pancreatic amylase deficiency, it is recommended that infant milk formulas don't contain starch, they should, on the other hand, contain lactose as the main carbohydrate and the addition of glucose is also allowed, as it can be hydrolysed on enterocyte's borders.
- During sports activities, they enable blood sugar to be maintained during exercise and muscular glycogen to be replenished as quickly as possible following exercise. Nowadays good sports nutrition practice does not make sense without an adequate supply of sugar, as a component that regulates exertion. Hence the wide range of *sports drinks* for different conditions and situations.
- In the elderly a drastic change in the perception of basic flavours occurs, and it is precisely the sweet taste that is maintained. This is of great importance for maintaining appetite and avoiding frequent involuntary weight loss in the elderly and malnutrition that is all too frequent in our Western countries.

Opportunities

Due to the fact that poor eating habits are leading to the consumption of excessive amounts of simple sugars, to the detriment of complex carbohydrates, it is a matter of priority that nutritional education campaigns be carried out to raise public awareness that the high consumption of these carbohydrates may seriously compromise health if they are not consumed in

an appropriate manner. However, in the same campaigns, the message should be conveyed that a moderate intake of sugar is compatible with a nutritionally balanced diet containing the required amounts of micronutrients and dietary fibre.

Nowadays the majority of diabetic treatments allow for the moderate inclusion of sucrose and other added sugars (30-50g/day) in the diet, provided that it is a balanced diet and good long-term metabolic control is maintained. Accordingly the Food and Agriculture Organisation of the United Nations (FAO) and the World Health Organisation (WHO) Recommend eating a wide range of foods rich in carbohydrates, mostly processed cereals, fruit and vegetables, as well as the moderate consumption of sugars²⁰.

Furthermore the WHO and FAO proposed the implementation of effective preventative measures to prevent tooth decay in children and not just stopping the consumption of sucrose and other sugars, as it has been observed that, with appropriate oral hygiene, dietary composition has a minimal effect on the incidence of tooth decay²¹.

Finally it should be mentioned that the American Heart Association and the American Diabetes Association²¹ suggest using low calorie or zero calorie sweeteners as a substitute for added sugars as it is an option for reducing total energy intake and also as these sweeteners don't increase blood sugar levels, they may be an option for diabetics. Although they also show that sweetener use should be considered in the context of overall diet when they are used to control body weight.

Recommendations

During the first months of life carbohydrates provide 40% of total energy from breast milk, with lactose being the main carbohydrate. This percentage gradually increases during the first year of life until they reach the range established for adults.

Although cultural and gastronomic patterns are usually difficult to change in adulthood, nutritional recommendations have been proposed that are easily achievable such as the use of low calorie or zero calorie sweeteners, limiting the consumption of cakes and pastries that are high in fat and simple sugars, sweetened drinks or certain sweets and desserts that may be high in simple sugars and that provide extra calories, taking part in regular physical exercise or eating a balanced diet.

The Committee of Experts from the European Food Safety Authority (EFSA)²² has proposed that total carbohydrates should provide between 45 and 60% of the total energy intake and <10% of the total energy intake from sugars for all age groups, except for children under the age of 2. On the other hand, the Spanish Society for Community Nutrition in 2011²³ decided not

to establish limits on the consumption of sugars and sugary foods, although they suggest that the total energy intake should not exceed 6%. Similarly, certain proposals have been made for quantifying sugar intake recommendations: 32-37g for a 2,000 kcal diet for men, and around 25g for women, for children it is calculated at about 12 g/d and approximately 25 g/d for adolescents, even though factors have to be considered such as a tendency to suffer with acne or other dermatological problems that might affect the previous recommendations.

It has also been proposed that sucrose should be avoided or limited when it is used as a sweetener, as well as in industrially prepared foods, that is consumed in excess. Longitudinal studies suggest that if infants become used to the sweet taste from an early age they will continue this preference during childhood and adolescence. However, there are no guidelines that mark or set the amount of sucrose or other sugars that should be consumed at this stage, therefore efforts should be made to limit the introduction of sweet foods or the sweetening of processed foods, in order not to establish unbalanced and cariogenic eating habits.

Conclusions

Carbohydrates are essential nutrients that should be part of the diet during the different stages and physiological situation in life, according to recommendations. The associated problems are determined by excessive consumption and not by their presence. Although polysaccharides should be found in the highest proportions, sugars are also compatible with a balanced healthy diet and they are particularly important during the stages in life that require a significant amount of glucose for the continual processes of attention and learning (infancy) or for appetite maintenance and so that other critically important nutrients can be transported, as is the case in elderly people, without forgetting their importance when practicing sport properly. Sugar, even in very moderate amounts, is a very important source of pleasure in the diet for certain age groups; it is a cornerstone to ensure proper nutrition.

Finally, it is essential to carry out short term nutritional balance studies, with mono- and disaccharides, that enable specific requirements to be established for the different population groups in Spain, without posing the risk of associated chronic degenerative illnesses.

References

1. Gibson S, Boyd A. Associations between added sugars and micronutrient intakes and status: further analysis of data from the National Diet and Nutrition Survey of Young People aged 4 to 18 years. *Br J Nutr* 2009; 101 (1): 100-7.

2. Stephen A, Alles M, de Graaf C, Fleith M, Hadjilucas E, Isaacs E, Maffeis C, Zeinstra G, Matthys C, Gil A. The role and requirements of digestible dietary carbohydrates in infants and toddlers. *Eur J Clin Nutr* 2012; 66 (7): 765-79.
3. Walsh JM, McGowan CA, Mahony R, Foley ME, McAuliffe FM. Low glycaemic index diet in pregnancy to prevent macrosomia (ROLO study): randomised control trial. *BMJ* 2012; 345: 345.
4. Tappy L, Lê KA. Metabolic effects of fructose and the worldwide increase in obesity. *Physiol Rev* 2010; 90 (1): 23-46.
5. Malik VS, Schulze MB, Hu FB. Intake of sugar-sweetened beverages and weight gain: a systematic review. *Am J Clin Nutr* 2006; 84 (2): 274-88.
6. Phelan S, Hart C, Phipps M, Abrams B, Schaffner A, Adams A, Wing R. Maternal behaviors during pregnancy impact offspring obesity risk. *Exp Diabetes Res* 2011; 985139.
7. Barclay AW, Brand-Miller J. The Australian paradox: a substantial decline in sugars intake over the same timeframe that overweight and obesity have increased. *Nutrients* 2011; 3 (4): 491-504.
8. Franz MJ, Bantle JP, Beebe CA, Brunzell JD, Chiasson JL, Garg A, Holzmeister LA, Hoogwerf B, Mayer-Davis E, Mooradian AD, Purnell JQ, Wheeler M, American Diabetes Association. Nutrition principles and recommendations in diabetes. *Diabetes Care* 2004; 27 (Suppl. 1): S36-46.
9. Malik VS, Popkin BM, Bray GA, Després JP, Willett WC, Hu FB. Sugar-sweetened beverages and risk of metabolic syndrome and type 2 diabetes: a meta-analysis. *Diabetes Care* 2010; 33 (11): 2477-83.
10. Metzger BE, Coustan DR. Summary and recommendations of the Fourth International Workshop-Conference on Gestational Diabetes Mellitus. The Organizing Committee. *Diabetes Care* 1998; 21 (Suppl. 2): B161-7.
11. Kjos SL, Buchanan TA. Gestational diabetes mellitus. *N Engl J Med* 1999; 341 (23): 1749-56.
12. Llodrá Calvo JC. Encuesta de Salud Oral en España 2010. *RCOE* 2012; 17 (1): 13-41.
13. Mobley C, Marshall TA, Milgrom P, Coldwell SE. The contribution of dietary factors to dental caries and disparities in caries. *Acad Pediatr* 2009; 9 (6): 410-4.
14. Lim S, Sohn W, Burt BA, Sandretto AM, Kolker JL, Marshall TA, Ismail AI. Cariogenicity of soft drinks, milk and fruit juice in low-income african-american children: a longitudinal study. *J Am Dent Assoc* 2008; 139 (7): 959-67.
15. Bellisle F. Effects of diet on behaviour and cognition in children. *Br J Nutr* 2004; 92 (Suppl. 2): S227-32.
16. Wolraich ML, Lindgren SD, Stumbo PJ, Stegink LD, Appelbaum MI, Kiritsy MC. Effects of diets high in sucrose or aspartame on the behavior and cognitive performance of children. *N Engl J Med* 1994; 330 (5): 301-7.
17. Schmidhuber J, Shetty P. The EU Diet – Evolution, Evaluation and Impacts of the CAP. Global Perspectives Studies Unit, FAO, 2009. Disponible en: <http://www.fao.org/ES/esd/Montreal-JS.pdf>
18. FAO-OMS. Estudio FAO Alimentación y nutrición. Los carbohidratos en la alimentación humana Informe Técnico N° 66, 1999. Disponible en: <http://www.fao.org/docrep/W8079E/W8079E00.htm>.
19. Gardner C, Wylie-Rosett J, Gidding SS, Steffen LM, Johnson RK, Reader D, AH; L, American Heart Association Nutrition Committee of the Council on Nutrition, Physical Activity and Metabolism, Council on Arteriosclerosis, Thrombosis and Vascular Biology, Young CoCDit, Association tAD. Nonnutritive sweeteners: current use and health perspectives: a scientific statement from the American Heart Association and the American Diabetes Association. *Circulation* 2012; 126 (4): 509-19.
20. EFSA Panel on dietetic products Nutrition and allergies. Scientific opinion on dietary reference values for carbohydrates and dietary fibre. *EFSA J* 2010; 8: 14-6.
21. Sociedad Española de Nutrición Comunitaria (SENC). Objetivos nutricionales para la población española. *Rev Esp Nutr Comunitaria* 2011; 17 (4): 178-99.

Sugar and physical exercise; the importance of sugar for athletes

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Abstract

Muscle glycogen, the predominant form of stored glucose in the body, and blood glucose are the main energy substrates for muscle contraction during exercise. Sucrose is an ideal substance for athletes to incorporate because it provides both glucose and fructose. Therefore, it is essential that athletes monitor their diet to maintain and increase muscle glycogen deposits, since they are a major limiting factor of prolonged exercise performance. Carbohydrate-rich diets are also recommended for endurance and ultra-endurance exercise, because they are associated with increased muscle glycogen stores, as well as delayed onset of fatigue. In addition, high carbohydrate diets and carbohydrate intake before and during exercise have shown to be beneficial due to increased concentrations of hepatic glycogen and maintenance of blood glucose. The effect of carbohydrate intake on athletic performance mainly depends on the characteristics of the exercise, the type and amount of carbohydrate ingested and the time of intake. A combination of these factors must be taken into account when analysing individual athletic performance.

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Key words: *Carbohydrate intake. Endurance sports. Strength sports. Performance. Training.*

EL AZÚCAR Y EL EJERCICIO FÍSICO; SU IMPORTANCIA EN LOS DEPORTISTAS

Resumen

El glucógeno muscular, principal almacén de glucosa en el organismo, y la glucemia sanguínea constituyen uno de los principales sustratos energéticos para la contracción muscular durante el ejercicio. El azúcar (sacarina) es un estupendo suplemento al suministrar tanto glucosa como fructosa. Por ello, es esencial que los deportistas cuiden su alimentación, para mantener y aumentar los depósitos de este combustible, ya que las reservas de glucógeno muscular constituyen un factor limitante de la capacidad para realizar ejercicio prolongado. Las dietas ricas en hidratos de carbono se han recomendado para el ejercicio de resistencia y ultra-resistencia debido a su relación con el aumento de las reservas musculares de glucógeno y la aparición tardía de la fatiga. Además de las dietas altas en carbohidratos, la ingesta de carbohidratos antes y durante el ejercicio, han demostrado ser beneficiosas debido al aumento de las concentraciones hepáticas de glucógeno y el mantenimiento de las concentraciones de glucosa en sangre. El efecto de la ingesta de carbohidratos sobre el rendimiento deportivo dependerá principalmente de las características del esfuerzo, del tipo y cantidad de carbohidratos ingeridos y del momento de la ingesta. La combinación de todos estos factores debe ser tenida en cuenta a la hora de analizar el rendimiento en las diferentes especialidades deportivas.

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Palabras clave: *Ingesta de carbohidratos. Deportes de resistencia. Deportes de fuerza. Rendimiento. Entrenamiento.*

Abbreviations

FFA: Plasma free fatty acids
ATP: Adenosine triphosphate.
HR: Heart rate Max.
HR: Maximum heart rate.
O₂: Oxygen.
TG: Triglycerides.
VCO₂: Carbon dioxide production.
VO₂: Oxygen consumption.
VO_{2max}: Maximum oxygen consumption.

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Energy metabolism of carbohydrates and their importance in different types of exercise

The sugar (sucrose) we consume in our diet is an important source of glucose for the body, as it is a disaccharide formed from one molecule of glucose and one of fructose. However, by extension, all carbohydrates are included under the term sugar. Among the different kinds of carbohydrate that are consumed monosaccharides (glucose, fructose and galactose) and disaccharides (maltose, sucrose and lactose) and glucose polymers (maltodextrin and starch) stand out. Their differences in osmolarity and structure have an impact on palatability, digestion, absorption, hormone release and the availability of glucose to be oxidised in the muscles¹. All the metabolic pathways of carbohydrates are reduced by the breakdown (catabolic pathways) of glucose (glycolysis) or glycogen (glycogenolysis), or the formation (anabolic pathways) of glucose (glycogenesis) or glycogen (glycogenosynthesis). Glucose is the only carbohydrate that circulates around the body and whose concentration can be measured in the blood (blood glucose). So all carbohydrates that are consumed in the diet are converted to glucose.

Muscle glycogen, the main form of stored glucose in the body, and blood glucose are the main energy substrates for muscle contraction during exercise, and they become progressively more important as exercise intensity increases. They are the most important substrates, as a quick source of energy for the body because their oxidation produces 6.3 moles of ATP (Adenosine triphosphate) per mole of oxygen (O_2) compared with 5.6 moles obtained from oxidising fats. One of the factors that may determine muscle fatigue is the depletion of carbohydrate reserves.

During low intensity aerobic activity (~30% of maximal oxygen consumption [VO_{2max}]) the total energy produced comes from 10-15% of carbohydrate oxidation. With an increase in intensity this percentage increases, and could reach 70-80% when VO_{2max} is ~85%, or even 100% during maximal or supra-maximal intensity activities². Besides exercise intensity, their use during exercise is influenced by various factors such as its duration (Fig. 1), the level of physical fitness, diet, gender, environmental conditions, etc. As most sports are carried out at intensities that are above 60-70% of VO_{2max} , carbohydrates from muscle glycogen and blood glucose are the main source of energy.

The role that carbohydrates play in energy metabolism during exercise highlights the importance of analysing adequate sugar intake for sports performance. The availability of carbohydrates during exercise, as well as the subsequent recovery of muscle glycogen deposits, plays a pivotal role in the performance of different sports. Skeletal muscle has a higher concentration of glycogen and is the tissue which has

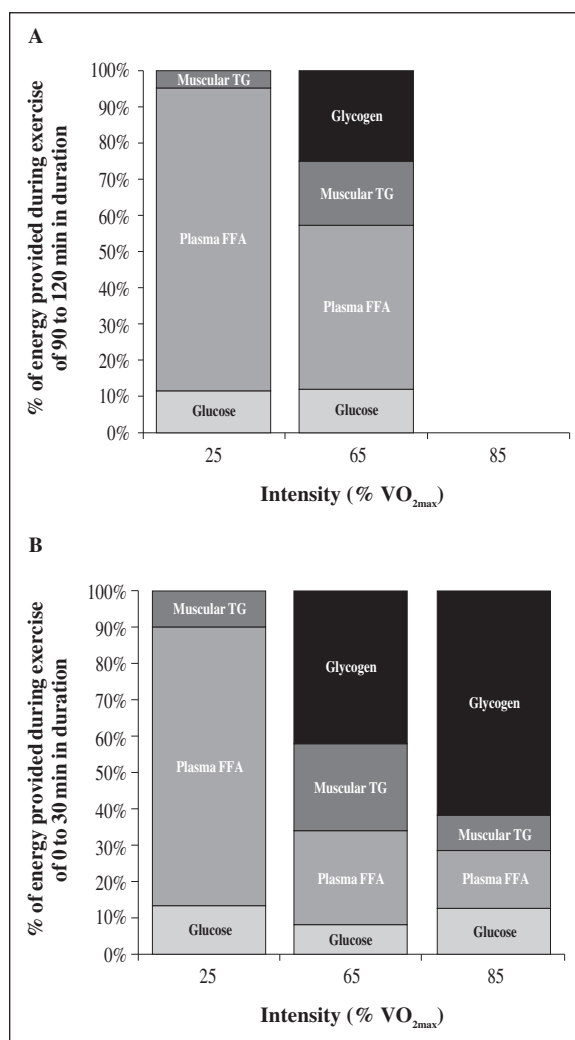


Fig. 1.—The effects of exercise intensity and duration in the use of metabolic substrates. The percentage of energy supplied by glucose, plasma free fatty acids (Plasma FFA), glycogen and muscular triglycerides (Muscular TG), during exercise from 90 to 120 min (A) and from 0 to 30 min (B) at intensities of 25, 65 and 85% of maximal oxygen consumption (VO_{2max}) (Adapted from¹³).

the largest deposits, as the liver (another glycogen store) only stores an eighth of the amount that muscle stores. For example, a 70 kg person with a muscle mass percentage of 45%, has 315 g of glycogen stored in their muscles, while there is about 80 g in the liver. The liver contains the glucose-6-phosphate enzyme which enables the dephosphorylation of glucose-6-phosphate and therefore supplies the rest of the organs and tissues with glucose. Liver function is vital during exercise to maintain blood sugar and supply the brain with glucose. For their part, muscles are able to use glycogen deposits independently. Therefore it's essential that athletes watch their diet so that they can maintain and increase these fuel deposits because muscle glycogen reserves are limiting factors in the capacity to perform prolonged exercise³.

A person can store around 1,500-2,000 kcal as blood glucose and glycogen. In the blood we only have 50 kcal of glucose for immediate use. Hepatic glycogen can provide around 250-300 kcal. Muscle glycogen in trained long distance runners is around 130 mmol·kg⁻¹, which is a higher value than those found in sedentary subjects or people who practice other sports that are shorter in duration. Because carbohydrates have limitations during exercise, including in cases where fats were are used as the main energy source, an athlete's diet should be rich in carbohydrates to cope with the high energy consumption and to maintain full glycogen stores¹.

Then, the role of sugar in different kinds of exercise will be briefly analysed, specifically glucose and glycogen: submaximal, maximal or supramaximal and intermittent.

Long term submaximal exercise

For this type of exercise, the higher the intensity the more muscular glycogen is used and the less energy is obtained from fat. However, the longer the durations the more fatty acids are used as a source of energy⁴ (Fig. 1). Muscles are metabolically independent thanks to glycogen stores, although these stores are not inexhaustible. That's why fat tissues and the liver have to provide the muscle fibres with fuel. This inter-relationship between tissues helps prevent the complete exhaustion of glycogen stores, because its concentration is the main limitation on the ability to perform prolonged exercise. Furthermore, when energy mainly comes from fats, mechanical performance is reduced, therefore, the coordination between muscles, the liver and fatty tissue is imperative^{3,4}.

Short term maximal or supra-maximal exercise

The high intensity of this type of exercise means that it can be performed for a long period of time. Moreover, the aerobic metabolic pathway is not able to supply energy at the speed that it's needed, therefore, from a quantitative point of view, anaerobic metabolism is more important in this type of exercise. In the phosphagen system glucose and glycogen are the main sources of energy. The contribution of muscle glycogen in short term maximal intensity exercise could be as follows: 20% in the first 30 seconds, 55% from 60 to 90 seconds and 70% from 120 to 180 seconds³.

Intermittent exercise

Combined periods of exercise and rest periods are known as intermittent exercise. These exercises are

very common in training and in many sporting activities. The fuel used during this type of exercise depends on the intensity, the duration of the exercise, the length of the rest period and the number of times the exercise is repeated, therefore the possibilities are endless. Focusing on glycogen, the four characteristics above determine the decrease in glycogen stores, whilst their replenishment (hepatic and muscular) depends entirely on diet.

Specific recommendations of sugar intake for athletes

Information on this matter is extensive and there are numerous studies that have examined the effectiveness of consuming different amounts of sugar. Tables I and II summarise the recommended carbohydrate intake guidelines for athletes.

Endurance sports

During endurance exercise muscle glycogen gradually decreases and, as we have previously mentioned, performance deteriorates. An effective way of improving endurance is to increase the amount of glycogen stored in the skeletal muscles and the liver before starting exercise⁵. The availability of carbohydrates, as a substrate for the muscles and central nervous system, becomes a factor that limits performance during prolonged submaximal (> 90 min) and intermittent high intensity exercise⁶.

Traditionally, diets rich in carbohydrates have been recommended for endurance and ultra-endurance training, because of the relationship between these diets, the increase in muscular glycogen stores and delayed onset fatigue. More recently diets high in

Table I
Recommended carbohydrate intakes in athletes.
Translated and amended from¹³

	<i>Intake recommendations</i>
<i>Daily requirements.</i> These recommendations should take the total individual energy expenditure, specific training needs and performance into consideration.	
Light or low intensity activities	3-5 g·kg ⁻¹ ·day ⁻¹
Moderate intensity exercise programme (~1 h·day ⁻¹)	5-7 g·kg ⁻¹ ·day ⁻¹
Moderate to high intensity exercise programme (1-3 h·day ⁻¹)	6-10 g·kg ⁻¹ ·day ⁻¹
High intensity exercise programme (4-5 h·day ⁻¹)	8-12 g·kg ⁻¹ ·day ⁻¹

Table II
Carbohydrate loading strategies. Translated and amended from¹³

		<i>Intake recommendations (in grammes of carbohydrates)</i>
Strategies aimed at promoting high carbohydrates availability that enables optimal performance in competition or important training sessions.		
Carbohydrate loading	Preparation for events < 90 min of exercise	7-12 g·kg ⁻¹ ·day ⁻¹ (daily requirements)
Carbohydrate loading	Preparation for events > 90 min of exercise	36-48 hours of 10-12 g·kg ⁻¹ ·day ⁻¹
Rapid loading	< 8 hours of recovery between two intense training	1-1.2 g·kg ⁻¹ ·h ⁻¹ during for the first 4 hours, followed by daily requirements
Intake before exercise	One hour before exercise	1-4 g·kg ⁻¹ consumed 1-4 hours before exercise
Intake during exercise	<45 min	Not necessary
Intake during high intensity exercise	45-75 min	Small amounts
Intake during endurance training	1-2.5 h	30-60 g·h ⁻¹
Intake during ultra endurance training	2.5-3 h	90g·h ⁻¹

carbohydrates and carbohydrate intake before and during exercise, have been shown to be beneficial due to an increase in hepatic glycogen concentrations and the maintenance of blood sugar concentrations⁷. The daily carbohydrate requirements for training and recovery are summarised in table I. To address the specific carbohydrate needs of athletes it is important to express them with regard to body weight. Various articles have suggested that a carbohydrate intake of 8 to 10 g·kg⁻¹·day⁻¹ is needed to replenish glycogen^{6,9}, a higher intake (10-13 g·kg⁻¹·day⁻¹) is needed for athletes whose sports disciplines generate a greater depletion of glycogen reserves⁶. In female athletes it appears that glycogen synthesis may increase during the luteal phase, therefore, the menstrual cycle is an important consideration when it comes to carbohydrate consumption in female endurance athletes^{6,7}.

It is vital for athletes to replenish glycogen reserves following exercise, with a view to providing enough energy for the next training session or competition. A high carbohydrate diet can be effective, on its own, at quickly replenishing glycogen stores, but there are a variety of strategies that can increase efficiency, such as adding proteins. Glycogen stores can be increased 1.5 times more than normal, for example, by consuming a high carbohydrate diet for 3 days prior to competition, after having followed a low carbohydrate diet for the 3 previous day (for a total period of 6 days before competition). Furthermore, if we take citric acid, which inhibits glycolysis, at the same time as following a high carbohydrate diet, glycogen stores increase even more because of the inhibiting effect they have on glycolysis⁵. In table II the strategies employed by athletes to increase and replenish glycogen stores are summarised.

Strength sports

Strength training, as a basic physical quality, has a major impact on nearly all of the homeostatic regulation systems of the body and also has significant metabolic consequences in terms of energy supply. Muscles do not differentiate between sporting activities, what they do differentiate between is the number of motor units that are recruited and the amount of time they are active (% in relation to maximal voluntary contraction), the rest are cultural differences that don't have any physiological implications. For example running a marathon is no more than a sequence of very small frequent muscle contractions at a moderate or low intensity and for a very long time, whereas running the 100 metres requires a very high proportion of muscle fibres available in a short space of time.

Strength training has a number of peculiarities that have a direct and important impact on the choice and use of different fuels. For activities that are longer than 120 seconds, total energy expenditure in strength training is less than aerobic activities because they are performed with an embedded rhythm. In this respect, for stand-alone exercises like the bench press or squats, anaerobic energy expenditure may be more than 30% of total energy¹⁰, in the case of circuit training this requirement is no more than 10%, as can be seen in figure 2. Carbohydrate requirements are important, in fact, when training intensity is high hypoglycaemias can be very common. However the total energy expenditure is not high. In figure 3 we can see that circuit training with weights does not require more than 35% of $\dot{V}O_{2max}$, whereas the required heart rate is higher than 90%.

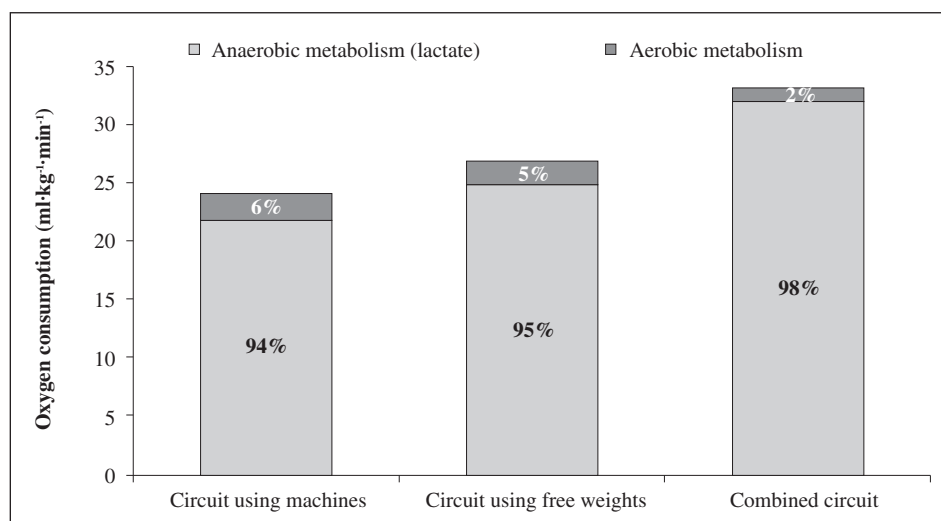


Fig. 2.—The proportion of energy provided and oxygen consumed during three rounds of circuits with weights, with 8 exercises without rest at 65% of intensity and for 54 min.

Although there are clear benefits to following a high sugar diet for endurance sports, the situation for strength training isn't as clear, as the small amount of energy that this kind of exercise usually needs is easily supplied by our energy system¹¹. What can often happen, and primarily due to the inadequate progression of training variables (amount and intensity), is that, due to the limited amount of energy, in the form of sugars in the blood, a very rapid demand for glucose (intense strength training) can exhaust these small reserves and this means that the liver is not able to supply glucose to the bloodstream as quickly, so the likelihood of hypoglycaemia occurring is very high.

The effects of sugar consumption on sports performance

The effect of carbohydrate intake on sports performance depends mainly on the kind of exertion (intensity, duration, etc), the type and amount of carbohydrates eaten and when they are consumed. The combination of all these factors should be taken into consideration when analysing performance in different sports. In the following points, the effects of consuming sugars before, during and after exercise will be explained, as well as the main features of low carbohydrate diets.

Carbohydrates should provide 55-60% of athletes total daily calorie intake. During periods of increased training this percentage should be increased to 65-70%¹⁻⁶. When they should be consumed will be discussed below.

Intake before exercise

The general recommendations for carbohydrate intake before exercise stipulate that dinner on the day

before competition should be high in carbohydrates (250-250 g), that the meal before (3-6 hours before) should include the intake of 200-350g and 60-30 minutes before competing 35-50 g of glucose, sucrose or glucose polymers should be consumed. The foods consumed should be low in fat, fibre and proteins, well tolerated, not in large portions and with a high or medium glycaemic index¹. On the other hand, certain studies indicate that the intake of glucose 30 or 45 minutes before exercise causes muscle fatigue more quickly than when it's not consumed (due to changes in glucose and insulin concentrations). However, if fructose is consumed plasma glucose and insulin concentrations do not change drastically before exercise¹².

The American College of Sports Medicine (ACSM) maintains that the amount of carbohydrates that enhances performance varies between 200 and 300g for meals 3-4 hours before exercise, at the rate of 30-60 g·h⁻¹ in intervals of 15-20 min (primarily in the form of glucose), for exercise that lasts more than an hour⁹. Furthermore, consumption of 0.15-0.25 g of protein·kg⁻¹, 3-4 hours before exercise, with a ratio of 3-4:1 (glucose: protein), can stimulate the synthesis of proteins during endurance exercise, but it has not been proven to improve performance⁹. Genton et al. propose the consumption of 1-4 g·kg⁻¹ 1 to 4 hours before exercise to increase carbohydrate availability during prolonged exercise sessions, and 0.5 to 1 g·kg⁻¹ during moderate intensity or intermittent exercise sessions > 1 h⁵.

When exercise is performed for a long period of time, such as a marathon, consuming carbohydrates immediately before or during exercise is an effective way of improving endurance. Under such conditions, it is desirable that athletes consume mono- or oligosaccharides, because they are quickly absorbed and transported to the peripheral tissues for use. On the other hand, carbohydrate consumption inhibits the breakdown of fats and stimulates insulin secretion. This

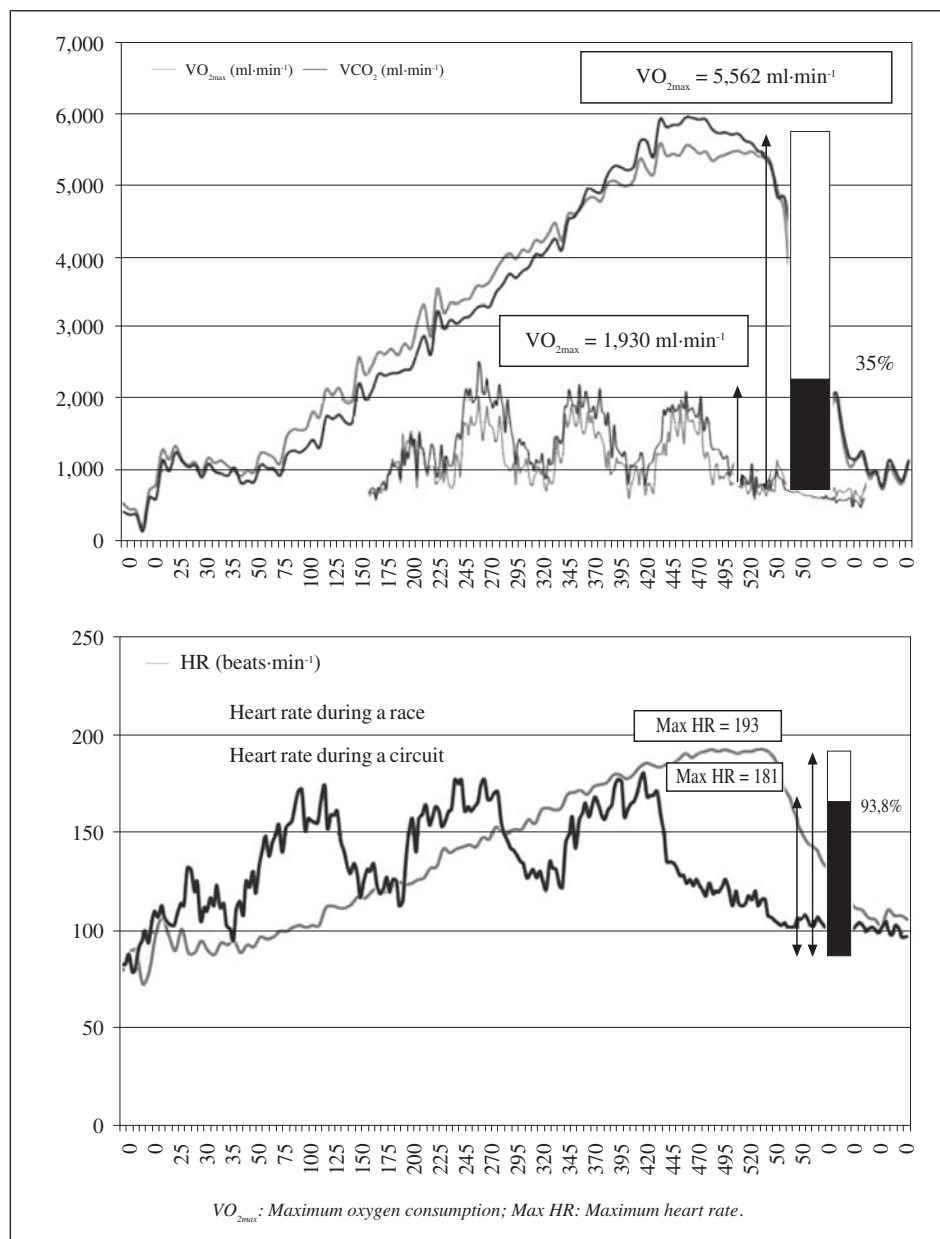


Fig. 3.—Oxygen consumption response (VO₂) and heart rate (HR) during a strength training circuit, compared with the response during stress test until exhaustion (Adapted from¹⁶).

leads to a deterioration in energy production through the metabolism of fats and accelerates glycolysis as a way of producing energy. As a result, muscle glycogen consumption increases. Therefore it's necessary to consume carbohydrates that don't inhibit fat metabolism. It has been suggested that supplements containing fructose stimulate insulin release less and it is unlikely that they inhibit lipolysis, instead of common carbohydrates such as glucose and sucrose. Moreover, the simultaneous consumption of citric acid and arginine can facilitate energy consumption from fats through the inhibition of glycolysis, delaying glycogen depletion. Therefore, consuming both together with carbohydrates that slowly stimulate insulin secretion, before or during

exercise, could be an effective way of improving energy metabolism and providing an optimal source of energy during prolonged exercise⁷.

Some studies show a reduction in muscle glycogen use when carbohydrates are consumed before and during exercise. Others have reported a reduction in hepatic glucose synthesis, the maintenance of normal blood sugar and high levels of glucose oxidation during the final stages of exercise, but not a reduction in glycogenolysis. However, high levels of circulating insulin reduce lipolysis and therefore reduce the contribution of muscle fat during exercise. Consequently, the amount of carbohydrates provided should be sufficient to cover energy demands from exercise and the energy lost from fat oxidation⁷⁻⁸.

A temporary reduction in blood sugar levels usually occurs when carbohydrates are consumed before exercise. This is probably due to an increase in glucose uptake in the plasma, as a result of increased insulin levels and the suppression of hepatic glucose synthesis. This imbalance causes a reduction in plasma glucose concentration that is subsequently offset by an increase in the intestinal absorption of glucose, with the aim of normalising plasma glucose levels. For the majority of individuals this reduction in blood sugar concentrations is temporary and of no functional significance¹².

Intake during exercise

High intensity and long duration endurance tests ($> 65\% \text{VO}_{2\text{max}}$) are characterised by a steady gradual decrease of glycogen in the active muscles. Although glycogen is not the only source of energy, it is necessary for maintaining intensity and it will be compensated by plasma glucose if it decreases, which is compensated by the liver (stored glycogen and the conversion of substrates like lactate or alanine in glucose). The reduction in plasma glucose, which occurs during prolonged exercise, is an indication that the liver cannot provide enough glucose once its glycogen stores are exhausted. Under these conditions, additional glucose may benefit performance⁸. Therefore, the aim of eating during exercise is to provide a readily available source of oxygen fuel, since the endogenous glycogen stores are exhausted⁷.

The maximum oxidation rate for exogenous carbohydrates during moderate intensity exercise is 0.8 to $1.0 \text{ g}\cdot\text{min}^{-1}$. This proportion is slightly less than 1 mJ of energy, whereas some forms of exercise need four times this amount. This suggests that there is the potential for supplementing with fat during exercise. Various studies have used medium-chain triglycerides as a source of additional fuel during exercise. Therefore, the consumption of both carbohydrates and fats together during exercise could prevent the decrease in fat metabolism that is observed when only carbohydrates are consumed¹³. The rate that limits the oxidation of ingested carbohydrates is due to its intestinal absorption, specifically, the type of transport mechanism. So, if glucose is consumed in combination with a glucide such as fructose, which is absorbed via a different transport mechanism, the total rate of carbohydrates consumed may be higher than $1.5 \text{ g}\cdot\text{min}^{-1}$. Following this, recommendations for glucose and fructose intake have been raised to $80\text{--}90 \text{ g}\cdot\text{h}^{-1}$, at a ratio of $2:1$. Furthermore, it has been shown that the time to exhaustion increases with the consumption of fructose and is dose-dependent¹². Improvements in performance are significantly higher when the subject receives larger amounts of fructose. The possible mechanism, by which fructose intake might spare muscle glycogen, involves its influence on plasma

lipids, as they enable the increased use of fats¹⁹. So, sugar (sucrose) becomes an excellent supplement to both glucose and fructose.

On the other hand, during tests of less than 60 min. in length recommendations suggest not giving any specific carbohydrates. However, consuming $300\text{--}500 \text{ ml}$ of drink with a carbohydrate concentration of $6\text{--}10\%$ every 15 min at a temperature of $8\text{--}12^\circ\text{C}$, could help preserve muscle glycogen and balance fluid loss, especially if the exercise is carried out during high temperatures. For events of between 1 to 3 hours it is recommended that $800\text{--}1,400 \text{ ml}\cdot\text{h}^{-1}$ of liquid are consumed, with a carbohydrate concentration of $6\text{--}8\%$ and a sodium concentration of $10\text{--}20 \text{ mmol}\cdot\text{l}^{-1}$. When the exercise duration exceeds 3 hours it is advisable to drink around $1,000 \text{ ml}\cdot\text{h}^{-1}$ of liquid with $23\text{--}30 \text{ mmol}\cdot\text{l}^{-1}$ of sodium.

Post exercise intake

After physical exertion of more than an hour, muscle glycogen stores may be empty, with a loss that could be around 90% . As a result, an exogenous supply of substrates is required to achieve the levels of glycogen prior to exercising. The full replenishment of glycogen stores following exercise takes between 24 and 48 hours , as the resynthesis rate is directly proportional to the amount of carbohydrates in the diet during the first 24 hours ¹³. The recovery of muscle and hepatic glycogen is a key objective of recovery between training sessions or competition events, particularly when the athlete engages in multiple training sessions during a condensed period of time^{6,8}. Previously it was thought that 48 hours of rest were needed to recover muscle and liver stores. Now it is accepted that, in the absence of severe muscle damage, glycogen reserves can normalise after 24 hours of reduced training and adequate fuel consumption^{7,8}.

The diet after each exercise session should contain sufficient carbohydrates to replenish glycogen reserves and to maximise subsequent performance (an average of 50 g of high carbohydrate foods for every 2 hours of exercise). The aim should be to consume a total of approximately 600 g of high carbohydrate foods with a high and moderate glycaemic index in a 24 hour period⁶. After intense exercise, muscle glycogen synthesis needs to recover about $100 \text{ mmol}\cdot\text{kg}^{-1}$, with a glycogen synthesis rate of $5 \text{ mmol}\cdot\text{kg}^{-1}\cdot\text{h}^{-1}$, requiring about 20 hours to recover (normalise) glycogen stores. Carbohydrate consumption during the first 2 hours after exercise allows a slightly faster rate of synthesis than normal ($7\text{--}8 \text{ mmol}\cdot\text{kg}^{-1}\cdot\text{h}^{-1}$). For this reason, athletes should consume enough carbohydrates following exercise as soon as possible, especially during the first hour after exercise due to the activation of the glycogen synthase enzyme by glycogen depletion, an increase in sensitivity to insulin and the perme-

ability of muscle cell membranes to glucose. Glycogen synthesis throughout the day is similar, whether the carbohydrates are consumed in large meals or as a series of small snacks, with no differences between consuming them in liquid or solid form, the only important thing is the total amount of carbohydrates consumed. Primarily, high carbohydrate foods should have a high glycaemic index (they increase muscle glycogen stores as much as possible), whereas those with a low glycaemic index should not constitute more than a third of recovery meals^{1,6,9}.

The recommendation, according to the type of activity, (grams of carbohydrates) are as follows (Table I and II)⁶:

- Immediate recovery after exercise (0-4h) 1.0-1.2 g·kg⁻¹·h⁻¹, every 2 hours.
- Daily recovery < 1 h·day⁻¹ from low intensity exercise: 5-7 g·kg⁻¹·day⁻¹.
- Daily recovery < 1 h·day⁻¹ from moderate to intense resistance training: 7-10 g·kg⁻¹·day⁻¹.
- Daily recovery < 4 h·day⁻¹ from moderate to very intense training: 10-12 g·kg⁻¹·day⁻¹.

During the first few hours meals with 70-80% carbohydrates should be eaten to avoid consuming lots of protein, fibre and fats, which besides suppressing hunger and limiting carbohydrate consumption, may cause gastrointestinal problems, in which case liquid preparations are preferred. At the same time, sports drinks, which fundamentally aim to create an anabolic environment, should cause an increase in blood sugar and consequently raise insulin, thereby enhancing the effects of different anabolic hormones to stimulate the synthesis of muscle and liver glycogen¹⁴.

Low carbohydrate diets

After addressing, at great length, the importance of consuming large quantities of carbohydrates for sports, the opposing alternative will be discussed: low sugar diets. It suggests that low carbohydrate diets that provide less than 50-150 g·day⁻¹, and their influence on sports performance has also been studied.

Low carbohydrate and high fat diets have been considered as a potential mechanism for improving performance in endurance exercises. However, amongst athletes these diets are perceived negatively when it comes to performance. The authors that proposed these diets suggest that this dietary practice provides large amounts of lipids as energy substrates to synthesise ATP. Low carbohydrate diets result in metabolic and hormonal adjustments that can improve the oxidation of fats and encourage muscle glycogen conservation during exercise. Like with endurance training adaptations, there is a shift towards increased fat oxidation as fuel, at rest or during exercise, which

may be due to a combination of an increase in oxidative enzymes, an increase in mitochondrial density, the increased storage and use of intramuscular triglycerides and the increased muscle uptake of plasma free fatty acids. This combination of mechanisms leads to a reduction in muscle glycogenolysis and carbohydrate oxidation and contributes to the increased use of free fatty acids during exercise¹⁴.

The low amount of glucose stored in the human body restricts the ability to maintain a high power output during prolonged endurance exercise. It had been argued that one of the consequences of a low carbohydrate diet can be a reduction in muscle glycogen content before exercise, particularly in untrained individuals, which could defeat the purpose of creating a glycogen sparing effect. Therefore, studies indicate that an increase in carbohydrate consumption tends to cause less disruption in sports performance compared to low carbohydrate diets^{14,15}.

Weaknesses

In general physical exercise has very specific energy and sugar demands. Therefore, metabolic activity during physical activity and training can cause problems with homeostasis in healthy people, and more so in at-risk populations, if the performance-based requirements are not met. In this way, the qualified professionals' ignorance and lack of consultation of these requirements could involve the implementation of a series of initiatives (eliminating foods and encouraging others) that could carry an unwarranted and unreasonable risk in many cases.

Threats

The difficulty in establishing the specific requirements for each physical activity, according to the intensity and volume of exercise, poses a major threat, as well as the proliferation of advertising campaigns or miracle diets that discredit the benefits of consuming sugar for sport. At present, the methods of quantifying physical activity help determine the energy requirements of each activity, although a number of confounding factors such as age or gender, could influence the accuracy of these measurements. Even so there is still a long way to go before specific sugar requirements can be quantified with real accuracy, for each person in every situation.

Strengths

This text takes a pedagogical approach to understanding general sugar requirements according to the type of physical exercise: endurance or strength.

Nowadays the requirements for many activities are known, as well as the significance or influence that adequate intake may have on performance. However, more work is needed in this line of research.

Opportunities

The demand to know the exact sugar requirements adapted to each person and situation creates work opportunities for research groups that are dedicated to studying specific blood sugar requirements. These groups are working on both healthy and populations with medical conditions. Certain lines of work will help improve the administering of insulin in diabetics, as well as its interaction with exercise.

Recommendations

All the carbohydrate intake recommendations have already been outlined throughout the text, however we should remember that it is important to assess the type of exercise performed, because sugar intake depends on these characteristics. In at-risk populations, the monitoring of blood sugar levels during exercise should be common practice.

Conclusions

The skeletal muscle and liver are the main stores of glycogen in the body. These stores, together with blood glucose, are the main source of energy for most athletes. Therefore, carbohydrate availability during exercise, as well as the subsequent recovery of muscle glycogen reserves, plays a pivotal role in the performance of different sports. The reduction in muscle glycogen levels (a substrate of the muscles and the central nervous system) becomes a factor that limits performance. There is evidence that consumption of a high carbohydrate diet, before and during exercise, is beneficial due to the increase in concentrations of hepatic glycogen and the maintenance of and blood sugar concentrations. Its effect on sporting performance depends primarily on the kind of the exercise, the type and amount of carbohydrates eaten and when

they are consumed. It is also important for athletes to replenish glycogen stores after exercise, with a view to providing enough energy for the next training session or competition, through a high carbohydrate diet with a high and moderate glycaemic index, enabling glycogen synthesis to be enhanced through the addition of protein. In conclusion, sugar (sucrose) becomes an excellent supplement to both glucose and fructose.

References

1. González-Gross M, Gutiérrez A, Mesa JL, Ruiz-Ruiz J, Castillo MJ. Nutrition in the sport practice: adaptation of the food guide pyramid to the characteristics of athletes diet. *Arch Latinoam Nutr* 2001; 51 (4): 321-31.
2. Jensen TE, Richter EA. Regulation of glucose and glycogen metabolism during and after exercise. *J Physiol* 2012; 590 (5): 1069-76.
3. Calderón FJ. Fisiología Humana. Aplicación a la actividad física. Madrid: Médica Panamericana, 2012.
4. Holloszy JO, Kohrt WM. Regulation of carbohydrate and fat metabolism during and after exercise. *Annu Rev Nutr* 1996; 16: 121-38.
5. Aoi W, Naito Y, Yoshikawa T. Exercise and functional foods. *Nutr J* 2006; 5: 15.
6. Burke LM, Kiens B, Ivy JL. Carbohydrates and fat for training and recovery. *J Sports Sci* 2004; 22 (1): 15-30.
7. Brown RC. Nutrition for optimal performance during exercise: carbohydrate and fat. 2002. *Curr Sports Med Rep* 1(4): 222-9.
8. Genton L, Melzer K, Pichard C. Energy and macronutrient requirements for physical fitness in exercising subjects. *Clin Nutr* 2010; 29 (4): 413-23.
9. Rodríguez NR, Di Marco NM, Langley S. American College of Sports Medicine position stand. Nutrition and athletic performance. *Med Sci Sports Exerc* 2009; 41 (3): 709-31.
10. Scott CB. Contribution of blood lactate to the energy expenditure of weight training. *J Strength Cond Res* 2006; 20 (2): 404-11.
11. Ainsworth BE, Haskell WL, Whitt MC, Irwin ML, Swartz AM, Strath SJ, et al. Compendium of physical activities: an update of activity codes and MET intensities. *Med Sci Sports Exerc* 2000; 32 (9): S498-504.
12. Craig BW. The influence of fructose feeding on physical performance. *Am J Clin Nutr* 1993; 58 (Suppl.): 815-819S.
13. Burke LM, Hawley JA, Wong SH, Jeukendrup AE. Carbohydrates for training and competition. *J Sports Sci* 2011; 29: S17-27.
14. Cook CM, Haub MD. Low-carbohydrate diets and performance. *Curr Sports Med Rep* 2007; 6 (4): 225-9.
15. Romijn JA, Coyle EF, Sidossis LS, Gastaldelli A, Horowitz JF, Endert E et al. Regulation of endogenous fat and carbohydrate metabolism in relation to exercise intensity and duration. *Am J Physiol* 1993; 265 (3 Pt 1): E380-91.
16. Morales M, Calderón FJ, Benito PJ, Lorenzo I. Fisiología del Ejercicio. In: Maroto Montero JM, Pablo Zarzosa CD, editores. Rehabilitación Cardiovascular. Madrid: Médica Panamericana: 229-252, 2011.

Energy density and the nutritional quality of diets based on their sugar content

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Abstract

Sugar content in foods cannot be distinguished from added sugar, although it is true that this added sugar brings mainly energy and no other essential nutrients. On the other hand, in the context of diet, sugar helps make it more varied and palatable allowing including foods that may otherwise not be consumed, thus indirectly contributing to the intake of other nutrients.

Having interest in knowing the possible relationship between a high intake of sugars and the decrease in micronutrients intake, we noted that the nutrient density of the diet might be influenced by factors such as the high presence of sugar added to food. It seems that this nutritional dilution produced by adding sugar to food is, in general, not very significant and, often, offset by the fortification in micronutrients that we usually can find in many sugary products. After a detailed analysis of the published studies on the subject, it has been found that there is no clear evidence of the hypothetical micronutrient dilution that would occur by adding sugar to the diet.

On the other hand, given that the addition of sugar to the diet doesn't seem to report any remarkable advantages from the point of view of the intake of micronutrients; It seems reasonable to promote a moderate consumption of foods and sugary drinks, so in that way, they become an important extra energy source.

It should also be borne in mind that the addition of sugar to the diet does not seem remarkable report any advantage in terms of intake of micronutrients. For this reason, it seems logical that the consumption of sugary food and sweet drinks will be moderate given the ease of consumption and the likelihood of their becoming a major source of energy.

Finally, it is concluded on the need for further research on the mechanisms underlying that, up to now showed no, possible displacement of micro-nutrients and other food components of the diet that could occur in cases of consumption of foods with a high sugar content, as well as their impact from a clinical point of view.

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Key words: *Sugar. Micronutrients. Energy density. Nutrients dilution.*

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LA DENSIDAD ENERGÉTICA Y LA CALIDAD NUTRICIONAL DE LA DIETA EN FUNCIÓN DE SU CONTENIDO EN AZÚCARES

Resumen

El azúcar contenido de forma natural en los alimentos no se puede distinguir del añadido, aunque lo cierto es que este azúcar de adición aporta fundamentalmente energía y no otros nutrientes esenciales. Por otro lado, en el contexto de la dieta, el azúcar contribuye a hacerla más variada y apetecible permitiendo incluir alimentos que quizá de otra manera no se consumirían, contribuyendo pues indirectamente a la ingesta de otros nutrientes.

Habiendo interés en conocer la posible relación entre una elevada ingesta de azúcares y la disminución de la de micronutrientes, se ha observado que la densidad nutricional de la dieta podría estar influida por factores como la presencia elevada de azúcar añadido en los alimentos. Parece ser que esta dilución nutricional producida al añadir azúcar a los alimentos es, en general, poco significativa y, a menudo, contrarrestada por la fortificación en micronutrientes que frecuentemente encontramos en numerosos productos azucarados.

Tras el análisis en detalle de los estudios publicados al respecto, se ha comprobado que no existe una evidencia clara de la hipotética dilución de micronutrientes que se produciría al añadir azúcares a la dieta.

Por otra parte, dado que la adición de azúcar a la dieta no parece reportar ninguna ventaja destacable desde el punto de vista de la ingesta de micronutrientes; parece razonable que se promueva un consumo moderado de alimentos y bebidas azucaradas, para de ese modo, evitar que se conviertan en una importante fuente energética extra.

Finalmente, se concluye sobre la necesidad de profundizar en la investigación acerca de los mecanismos subyacentes a ese, hasta ahora no demostrado, posible desplazamiento de micronutrientes y otros alimentos componentes de la dieta que podría ocurrir en casos de consumo de alimentos con un elevado contenido en azúcares, así como su repercusión desde un punto de vista clínico.

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Palabras clave: *Micronutrientes. Densidad energética. Dilución de nutrientes.*

Abbreviations

NT: Nutrition transition.

NMES: Non milk extrinsic sugars.

DRI: Dietary reference intakes.

EBRB: Energy balance-related behaviours.

DOiT: Dutch Obesity Intervention in teenagers.

FAO: Food and Agriculture Organisation of the United Nations.

Introduction

There is growing concern about the relationship between high sugar consumption and the possible decrease in macronutrient intake in the populations of developed and developing countries. However, the fact is that so far studies have not reached a clear conclusion on this matter.

The concern about a possible connection between sugar and public health is old, as confirmed by the various guidelines that have been issued on the dietary intake of added sugars and tooth decay¹, however the guidelines on limiting added sugars with the aim of preventing the dilution of micronutrient intake are unclear and often controversial.

If we consider what is really important for public health it is lifestyle, and within this, overall diet; we accept that, although sugar intake is important, it continues to be just one part of an individual's overall diet.

Which is precisely why countries that are concerned about excessive sugar consumption have undergone,

over the last few decades, huge lifestyle changes in their populations, especially when it comes to food. This change is known as nutrition transition (NT) and consists of a number of eating habit and lifestyle changes, associated with certain improvements in socioeconomic and health conditions (demographic and epidemiological transition) which far from leading to an improvement in health, seem to have a positive relationship with increases in excess weight and obesity rates, as well as certain chronic illnesses, such as cardiovascular disease and type II diabetes mellitus.

NT is broadly characterised by a reduction in consuming foods that are high in complex carbohydrates and fibre (bread, cereals, pasta, pulses, potatoes) in favour of those that contain sugar, dairy products and other products of animal origin. As a result, in these populations there has been an overall increase in total energy intake, animal protein and fats.

Since NT is worldwide phenomenon², Of the Western countries, Spain is a good example for illustrating its impact, as the social and economic changes in our country during the 20th century were very rapid (Fig. 1) and especially from the 1970s onwards³.

According to Popkin y Gordon-Larsen⁴, different regions or countries of the world would be (depending on their level of cultural and technological development) in one of the three final stages proposed by the authors in an attempt to summarise the characteristics that define the progression of NT. According to this proposal, Spain would currently be in phase⁴ of the process which is summarised in figure 2.

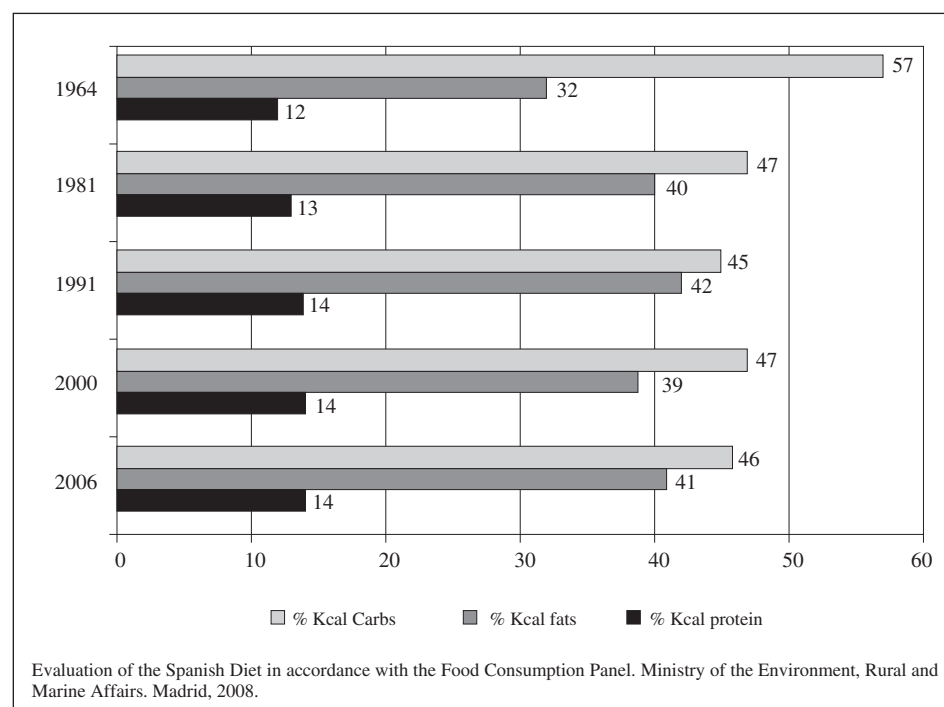


Fig. 1.—The evolution of calorie profiles in Spain (1964-2006). % of total energy from the immediate principles.

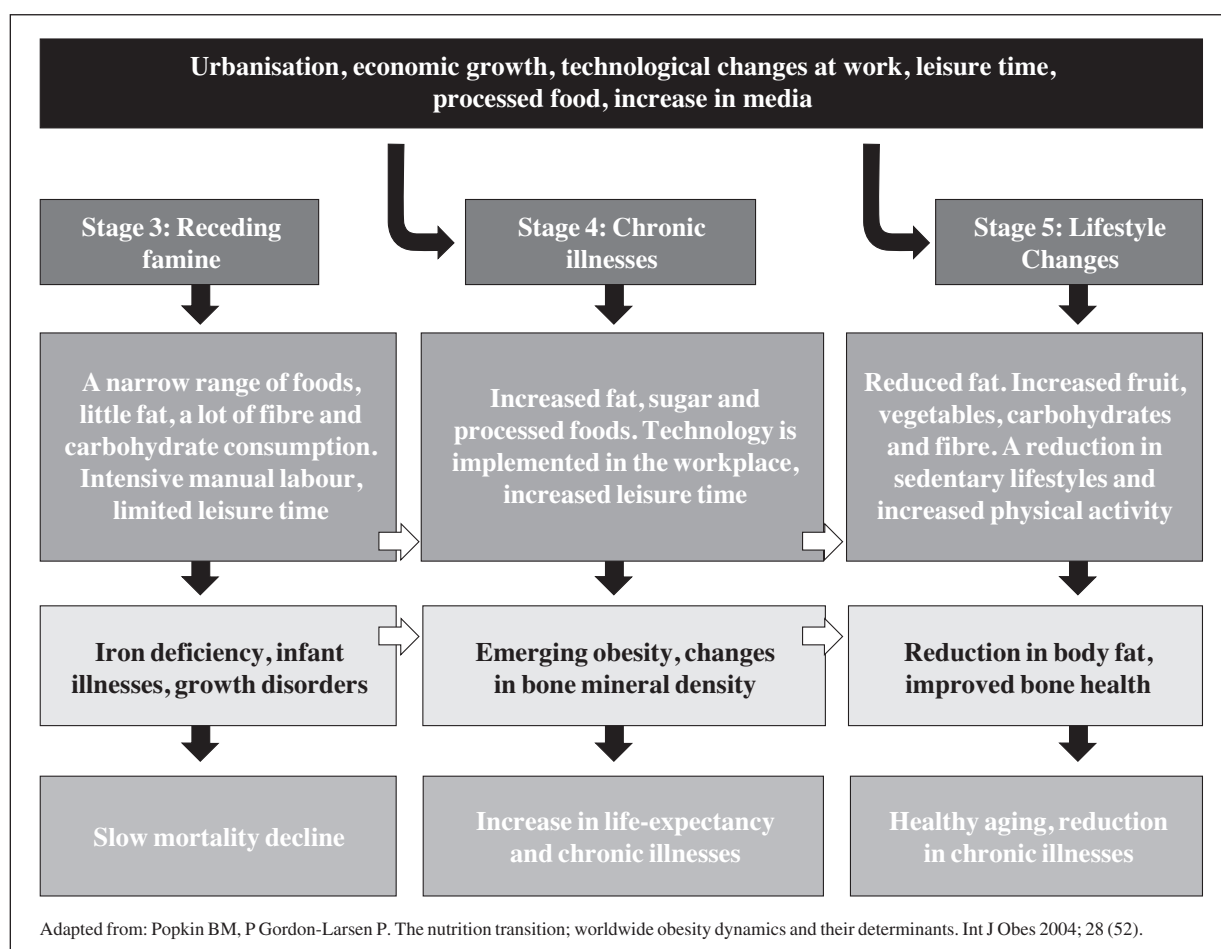


Fig. 2.—Stages of nutrition transition.

Definitions

Any review of the evidence on micronutrient dilution should refer to the inconsistencies that exist in scientific literature⁵ and that mainly stem from a variation in the definition of the term ‘sugar’ (as this term can include total sugars, added sugars or free sugars). This inaccurate categorisation could substantially affect the analysis of the relationship between sugar intake and micronutrients.

Sugar is an all-pervasive ingredient in the food industry and is also consumed as an ingredient that is naturally present in many foods. One of the main concerns is, precisely, to specify what is meant by ‘sugar consumption’. So, in the USA added sugars are defined as those sugars, sweeteners and syrups that are consumed as such or even used as ingredients in processed or prepared foods, therefore excluding sugars naturally found in honey and fruit.

In the UK, the term NMES is preferred (non milk extrinsic sugars), a category which includes all sugars that are not naturally found in milk or dairy products and which is effectively synonymous those that are usually called ‘free sugars’.

Sugar intake recommendations

In the USA, in the year 2000, when drafting the corresponding *Dietary Guidelines* it was recommended that the population choose foods and drinks that, on the whole, help moderate the population’s sugar intake.

The 1990 World Health Organisation guidelines, reiterated by the Committee of Experts on diet, nutrition and the prevention of chronic diseases, recommended that the consumption of free sugars should not exceed 10% of the total energy intake¹. However, the American Institute of Medicine concluded, in the 2002 dietary reference intake (DRI) edition, that there is insufficient evidence to enable the maximum intake of added sugars to be selected, as we don’t know the exact adverse effects on health associated with ‘excessive’ consumption. On the other hand, it has also been suggested in these DRI that the maximum intake from added sugars should be 25% of the total daily energy, given the growing concern about inadequate micronutrient intake but without being ‘interpreted as a recommendation for consumption’.

In short, from the overall data on dietary intake it could be concluded that, overall, it's possible that a proportion of the population consumes larger amounts of sugar than are recommended, which is reflected in the results of some of the reviewed studies, that place this consumption at about 15-17% of the total caloric value based on the population group studied.

Weaknesses

Without a doubt, sugar content which is naturally found in food, such as fruit, cannot be distinguished analytically from added sugar. Nutrient density, i.e. the intake of nutrients in relation to energy intake, is commonly used as an indicator for the quality of a diet. In spite of the fact that for certain populations it is imperative to achieve high nutrient density, as in young or sick people who for different reasons restrict their eating, we can't extend this recommendation to the whole population.

The nutritional consequences of added sugar consumption. The problem of nutrient density and dilution

The diet's nutrient density can be influenced by various factors, one of which is sugar added to foods. So, diets that include high intakes of added sugar are often characterised by an increase in total energy value, whilst decreasing nutrient density.

This has been noted in the case of adults and, above all, in young people. Studies carried out in South African, on an adult female population, have shown a reduction in nutrient intake as added sugar intake increases and the Lyhne study showed a general trend towards a reduction in nutrient density when the percentage of energy obtained from added sugars increased. This negative association is a lot narrower than those mentioned in previous studies.

All these results are in line with Gibson's results on British adults' diets, who observed that an increase in the energy percentage obtained from added sugars, increased total energy intake and slightly decreased micronutrient intake. The only micronutrient whose levels were not affected in this way was vitamin C.

It seems that this nutrition dilution which is produced by adding sugar to food is, in general, not very significant and is frequently offset by micronutrient fortification, which is often found in many sugary products. In this way, the possible negative association with added sugars and nutrient density can be offset through the fortification of foods. In fact, in the case of children and adolescents, fortified foods (for example, certain drinks, dairy products, cereals, etc.) contribute substantially to the total

intake of vitamins and minerals, at least in Germany and the USA.

In this respect, it is a good idea to take the World Sugar Research Organisation's declaration of principles into consideration⁵, whose report 'Sugar and micronutrient dilution' (issued in January 2012) states that 'It is often assumed that a diet high in sugars reduces the intake of essential nutrients (or micronutrient dilution). However, this is often a simplified view of a rather more complicated problem and for which there is insufficient scientific evidence. Additionally, a reduction in sugar intake may be unpredictable and have undesirable consequences for public health. For example, certain foods would be avoided that, even though they contain sugar, would be fortified or high in micronutrients per se.'

Energy consumption and obesity

Recent studies show a significant increase in daily energy intake in relation to increases in sugary drink consumption by children, adolescents and adults. However, the evidence regarding the positive association between the consumption of sugary drinks and obesity is inconsistent. This would seem logical as both excess weight and obesity are very complex situations, from a metabolic point of view, to be able to pinpoint the blame for their development on a single food or group of foods. As a result, the issue is open to debate as it is possible to find studies that highlight the likely relationship between sugary drink consumption and the risks of excess weight and obesity, meanwhile other authors show evidence to the contrary.

If we refer to this positive relationship, it should be taken into account that the development of obesity in the USA ran parallel to an increase in the consumption of added fructose, as a result of the introduction of high fructose corn syrup as a sweetener in drinks. Evidence on the causal relationship between high fructose intakes and metabolic disorders is quite clear and a recent study on American adults confirmed the link between fructose consumption and dyslipidaemia⁶. In this regard, high consumption of sugary drinks increases the risk of developing type 2 diabetes, as demonstrated by the Health Professional Follow-up Study⁷, an effect which also produces an increase in the risk of coronary heart disease according to the Nurse's Health Study (when more than two units of these drinks are consumed per day, the risk is 35% higher than in those subjects who did not consume them or who consumed them in very small quantities), an effect that could even be measured by an increase in different inflammatory markers.

We note here the recent Aeberli study⁸ which shows how adverse effects of cardiovascular risk markers occur (LDL, fasting blood glucose and C-reactive

protein or CRP) even following the consumption of low or moderate amounts of sugary drinks. This effect appears quickly and can be measured in healthy young men after 3 weeks of consumption, which gives these results special significance when it comes to providing dietary advice to young consumers.

The fact is that sugary drinks consumed with foods have doubled in recent years. In a well-known trial, it was found that increasing the size of sugary drinks involved a parallel increase in the energy consumed from solid foods. In this way, by increasing the size of sugary drinks, the energy consumed from foods ultimately increased by 10% in women and 26% in men.

Between 1970 and 2005, the sugars and sweeteners available for consumption in the USA increased on average by 76 calories a day, from 25 teaspoons (around 400 calories) to 29.8 teaspoons (476 calories), which corresponds to an increase of 19%. Bray et al pointed out that added sugar consumption increased from 235 calories per day per person in 1977 to 318 calories/day per person in 1994, drawing particular attention to the fact that high fructose syrups increased from 80 to 132 kcal per day per person during the same period.

A NAHNES study found that the average intake was 22.2 teaspoons of sugar per day (355 kcal) and between the ages of 14 and 18 is when the highest consumption occurs, equivalent to 34.3 teaspoons per day (549).

Added sugar intake in children's and adolescents' diets, according to the Donald study, represent between 10 and 13% of total daily energy and is similar to the values found in studies carried out in Germany (from 12 to 14% of total energy) and in Denmark (14% of energy). Sugar intake in Great Britain provides around 17% of daily energy and in the USA 16% of the energy for children between the ages of 2 and 5 years old, and 20% for young people between the ages of 12 and 19 years old. All the studies agree that sugar consumption is higher than the traditionally recommended limit (10% of total energy) in these countries.

Therefore, these results show a weak but general trend towards a reduction in the nutrient density of vitamins and minerals when there is an increase in calories from added sugars, as many authors have concluded. This effect is magnified when micronutrients are not fortified, which supports the hypothesis that fortified foods may behave like masking factors for the possible dilution of nutrients.

Minerals

As far as mineral intake is concerned, the results of the different studies published are inconsistent when it comes to the impact of a diet rich in added sugars on calcium, iron and magnesium intake. If we refer to iron intake, in most studies there is a correlation between

calcium levels and high sugar diets which is only seen in women. So, both low sugar and high sugar diets are associated with lower iron levels. Nevertheless, Gibson et al.⁹ did not find any association between sugar intake and iron status in aging adult populations, or the serum ferritin levels in men and women.

As far as zinc is concerned, the results are somewhat more consistent since numerous studies observed an inverse association between added sugar intake and zinc, in adults and it would seem also in children.

Vitamins

The results are inconsistent when it comes to the amounts of vitamins A, B₁, B₂, B₆, folates, vitamin C and vitamin E consumed, in the context of a high sugar diet. In the case of vitamin C and vitamin B₂, some authors indicate a positive association. However, it is important to bear in mind that the average intakes of these vitamins (A, B₁, B₂, B₆) in the general population normally exceeds the recommended amounts.

So, in the case of vitamin E, in the UK data that refers to intake in adults is indicative of higher than recommended intakes, although this data has not been confirmed by other researchers. On the other hand, when it comes to folate the overall folate intake is low, both in women and children, and independently from sugar intake. This could explain, at least in part, the gender differences that exist when reviewing what happens to folate when there is high added sugar consumption. In this case, there are more studies that show a clear connection between sugar consumption in women and a lower folate intake. For the same reason, this correlation does not occur in men⁹.

Given the proliferation of studies that used different methodologies, it is difficult to find conclusive data when it comes to the infant population, where, at any rate, there is an inverse association between added sugar intake and vitamin B₁. This reduction in consumption could reach 3% in children who obtain less than 8% of their daily calories from added sugars and is the same amount even if they consume more than 16%. Furthermore, in another study they found that children who consumed higher amounts of sugars were not even able to meet their daily recommended intakes of vitamin B₁.

As we have already observed in the case of iron, it seems there is a non-linear relationship between added sugar and vitamin intake. For example, women who eat added sugars in moderation consume more vitamin E than those who eat a lot or, on the contrary, very little sugar. In certain cases, there has been an increase in vitamin C intake. This is probably because the sugar consumed came from the addition of fruit juices. In short, it seems clear that there is no association between plasma vitamin C status and the addition of sugars to the diet.

Threats

Sugars added to foods as a percentage of energy intake tend to increase with age amongst young people; the opposite occurs when it comes to micronutrient intake, with lower intakes of some of them such as folates, vitamin C and calcium.

To clarify this data a little, as numerous as they are contradictory, maybe Gibson's statement is illustrative as it confirms that 'in the same vein, in England, it has been proven that⁹ a diet moderately high in added sugars (around 15% of total energy) does not appear to affect micronutrient intake, energy intake (and not its origin) is, in fact, the main determinant for the adequate intake of micronutrients.'

Although it is not the purpose of this chapter, the Intermap study included data related to blood pressure, sugar and sugary drink consumption in the UK and USA, in a sample of 2,696 people between the ages of 40 and 59 years old. Sugary drinks were directly linked to blood pressure¹⁰.

Strengths

It would be advisable to promote healthy eating to the population; a diet that incorporates moderate amounts of sugar in foods and specific situations. In this way, in order to develop measures that prevent unhealthy weight gain, changes should be made to the 'energy balance-related behaviours' (EBRB). So, consuming large quantities of sugary drinks, spending time watching TV or in front of the computer are behaviours that are associated with an increased risk of obesity.

In this sense, school is considered the ideal place to put measures in place that reduce the incidence and prevalence of childhood and adolescent obesity, as they offer the possibility of continuous, intensive contact with young people or children from all socio-economic and even ethnic backgrounds. However, the inadequate preventative measures that have so far been developed in this field, have sparked a debate, in various countries and on different occasions, over what would be the best and most effective strategies to deal with this.

The Dutch Obesity Intervention in Teenagers (DOiT) was a randomised study, developed in schools, which proved to be effective in reducing the consumption of sugary drinks among adolescents. It showed that interventions that try to reduce sugary drink consumption can be effective without changing the consumption of other drinks.

Of course, including the family and home environment in these strategies would increase their effectiveness in preventing obesity^{11,12}. Nevertheless, the mechanism and effect of parental involvement in obesity prevention programmes is still not clear.

As the effects of habits established at home show, we can mention the Harris study¹³, which found that when children ate breakfast containing high sugar products, it increased, in a similar way, total daily sugar intake and also reduced the overall nutritional quality of their breakfast.

On the other hand, it should be noted that the risk of obesity can be very different for different population groups, so strategies are needed that allow for the individual nature of each of these groups. In fact, the pupils' gender is one of elements which is regularly considered and there seems to be a correlation in that the measures are more effective in girls than boys, probably because in early adolescence girls and boys respond differently to different intervention strategies. This variable can be embedded in a very complex framework whatever the starting weight or socioeconomic background of the young people may be, for example, there hasn't even been sufficient evidence of consistent energy intake moderators related to different lifestyles.

In the Bjelland study¹⁴, which lasted eight months, it was confirmed that, amongst young people, gender was a result-related variable, in so far as it affected the response to educational interventions in such a way that the preventative measures seemed to work better for girls than for boys.

Finally, when it comes to the various opportunities to improve the diet and health of the population, it is of interest to mention Thornley's work¹⁵. It examined the possible association between asthma and the per capita sugar consumption of six and seven year old children. For this study, clinical data was collected on asthma between 1999 and 2004 in 53 countries and the sugar consumption per capita was obtained from the FAO's food balance sheets. Once the results had been analysed, a connection was observed between the severity of asthma symptoms and the per capita consumption of added sugars (in kilos per annum). In other words, a moderate ecological association was found between sugar consumption during the perinatal period and the subsequent risk of severe asthma symptoms in 6 or 7 year old children.

Opportunities

The link between diet and health opens up new prospects in areas which have certainly not had much emphasis placed on them in recent years. This occurs with the low consumption of sugary drinks and added sugars, which are associated significantly with a reduction in blood pressure. So, consuming moderate and adequate amounts of sugary soft drinks and added sugar could be a significant strategy for reducing blood pressure¹⁶. Nevertheless, the way in which high consumption of sugary drinks may be connected to the risk of hypertension is unclear. In fact, although this

link between high sugary soft drink consumption and increased blood pressure has previously been observed in tests animals, this has certainly not yet been proven in humans (which would be necessary where there is a 3 mmHG reduction in systolic pressure that could reduce the death rate from myocardial infarction by around 8% and from coronary heart disease by 5%).

Ultimately, as one might expect, a healthy and balanced diet contains natural sources of sugar, since monosaccharides, such as fructose, or disaccharides, like sucrose or lactose, are components of fruit, vegetables, dairy products and many cereals. Sugars, naturally found in foods or added to them, obviously emit a very pleasant flavour which allows certain foods and meals to be enjoyed. Furthermore, when sugar is added to certain nutrient-rich foods, such as milk, yoghurt or cereals, the quality of diets followed by children and adolescents may even improve, something that has also been observed in the case of flavoured milks which also have no negative effects on body mass index.

Conclusions

The general conclusion, obtained from the detailed analysis of studies published, is that there is no clear evidence that micronutrient dilution occurs when sugar is added to the diet. Adding sugar to the diet doesn't seem to have any remarkable advantages, in terms of micronutrient intake, either.

Sugar added to drinks can be a major source of energy, whose intake, due to its ease of consumption should be monitored.

Sugar, as an ingredient added to food and drinks, can satisfy the tastes of a significant proportion of the population, satisfying their appetite and allowing access to many foods that, without sugar, maybe more difficult to consume, and thus indirectly contributing to an increase in the consumption of other micronutrients.

It is therefore necessary to continue research into how certain foods, that have a high sugar content, could adversely affect micronutrient intake or even to what extent they can end up displacing other foods in the diet.

References

1. World Health Organization (2003) Diet, Nutrition and the Prevention of Chronic Diseases. Geneva: WHO.
2. Lomaglio, DB. Nutrition transition and the impact on growth and body composition in northwestern Argentina (NOA). *Nutr Clin Diet Hosp* 2012; 32 (3): 30-5.
3. Marrodán MD, Montero P, Cherkaoui M. Nutritional Transition in Spain during recent history. *Nutr Clin Diet Hosp* 2012; 32 (Suppl. 2): 55-64.
4. Popkin BM, P Gordon-Larsen P. The nutrition transition: worldwide obesity dynamics and their determinants. *Int J Obes* 2004; 28 (Suppl. 2).
5. World sugar research organization. Declaración de principios. Azúcar y dilución de micronutrientes (Accedido enero de 2012). <http://www.wsro.org/public/documents/position-statement-sugars-and-micronutrient-dilution-2012.pdf>
6. Welsh JA, Sharma A, Abramson JL, Vaccarino V, Gillespie C, Vos MB. Caloric sweetener consumption and dyslipidemia among US adults. *JAMA* 2010; 303: 1490-7.
7. De Koning L, Malik VS, Rimm EB, Willett WC, Hu FB. Sugar sweetened and artificially sweetened beverage consumption and risk of type 2 diabetes in men. *Am J Clin Nutr* 2011; 93: 1321-7.
8. Isabelle Aeberli, Philipp A Gerber, Michel Hochuli, Sibylle Kohler, Sarah R Haile, Ioanna Gouni-Berthold, Heiner K Berthold, Giatgen A Spinass, Kaspar Berneis. Low to moderate sugar-sweetened beverage consumption impairs glucose and lipid metabolism and promotes inflammation in healthy young men: a randomized controlled trial. *Am J Clin Nutr* 2011; 94: 479-85.
9. Gibson S. Dietary sugars and micronutrient dilution in normal adults aged 65 years and over. *Public Health Nutrition* 2001; 4 (6): 1235-44.
10. Brown I et al. Sugar sweetened beverage, sugar intake of individuals and their blood pressure: Intermap study. *Hypertension* 2011; 57 (4): 695-701.
11. Birch LL, Ventura AK. Preventing childhood obesity: what works? *Int J Obes (Lond)* 2009; 33 (Suppl. 1): 74-81.
12. Gruber KJ, Haldeman LA. Using the family to combat childhood and adult obesity. *Prev Chronic Dis* 2009; 6: 1-10.
13. Jennifer L. Harris, Marlene B. Schwartz, Amy Ustjanaskas, Punam Ohri-Vachaspati, Kelly D. Brownell. Effects of Serving High-Sugar Cereals on Children's Breakfast-Eating Behavior. *Pediatrics* 2011; 127: 71.
14. Bjelland et al. Changes in adolescents' intake of sugar sweetened beverages and sedentary behavior: Results at 8 month mid-way assessment of the HEIA study - a comprehensive, multi-component school-based randomized trial. *Int J of Behav Nutr and Phys Act* 2011; 8: 63.
15. Simon Thornleya, Alistair Stewart, Roger Marshalla, Rod Jackson. Per capita sugar consumption is associated with severe childhood asthma: an ecological study of 53 countries. *Primary Care Respiratory Journal* 2011; 20 (1): 75-8.
16. Chen L et al. Reducing consumption of sugar-sweetened beverages is associated with reduced blood pressure: a prospective study among U.S. adults. *Circulation* 2010; 121 (22): 2398-406.

Dental health; the relationship between tooth decay and food consumption

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Abstract

Although the reduction and prevalence of dental caries in many countries has been largely associated with the use of fluorine and improving dental hygiene, eating habits also play a role in the development of caries. Fermentable carbohydrates characteristics of the food, rate of consumption, food protectors, the quality and quantity of saliva indices that determine the remineralization of teeth are factors to be considered. All these elements are analyzed through the sociodemographic, behavioral, physical and biological environment directly or indirectly with diet and caries.

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Key words: Diet. Caries. Risk. Remineralization. Hygiene.

SALUD DENTAL; RELACIÓN ENTRE LA CARIES DENTAL Y EL CONSUMO DE ALIMENTOS

Resumen

A pesar de que la reducción de la incidencia y prevalencia de la caries dental en muchos países se relaciona en gran medida con el uso sistemático del flúor en las pastas dentífricas y la mejora de la higiene dental, se debe tener presente la importancia de los hábitos alimentarios en la prevención primaria y secundaria de la caries dental. En este sentido, destacan los carbohidratos fermentables, determinadas características de los alimentos, la frecuencia de consumo, distintos tipos de alimentos, algunos como factores protectores, la cantidad y la calidad de la saliva, en tanto que ello determina el índice de remineralización de los dientes etc. Todos estos elementos son analizados a través de los factores sociodemográficos, de comportamiento, físico-ambientales y biológicos relacionados directa o indirectamente con dieta y caries.

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Palabras clave: Dieta. Caries. Riesgo. Remineralización. Higiene.

Abbreviations

DMFT index: The rate of tooth decay resulting from the calculation of decayed teeth, those missing and filled due to tooth decay, for each permanent tooth.

DMF index: The rate of tooth decay resulting from decayed or filled teeth due to tooth decay, for each deciduous tooth.

CPI of sucrose; the Cariogenic Potential Index. The standard is sucrose with a value of 1.

CH: Carbohydrates.

HLA-DR: The HLA system (human leucocyte antigen, as it was initially only used to 'classify' leucocytes) consists of a cluster of genes located in the human chromosome 6. *Class II HLA genes*: They contain at least three loci, HLA-DR, HLA-DQ and HLA-DP, and all of them have corresponding alleles.

TCL: Total Cariogenic Load. It defines the individual net effects from assessing the risk factors (cariogenic potential, frequency of consumption, etc) together with the protective factors (intrinsic, hygiene, fluoride, sealants, etc).

Introduction

In general, the dietary factors in the aetiology of tooth decay are currently being played down, in favour of promoting hygiene and the adequate use fluoride. Since the widespread use of fluoride, some authors

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have even questioned the relationship between high sugar consumption and the incidence of tooth decay. However, it should be taken into account that in young children dental hygiene and the use of fluoride is often unsatisfactory and/or insufficient, and therefore, it is during this phase of dental development that dietary habits seem to be more important when it comes to preventing the appearance of tooth decay. Something similar occurs in the elderly, where there is a reduction in salivary secretion, which is secondary to the multi-medication and multiple pathologies that are associated with this age group¹⁻⁴.

Diet is not just important for general health, but also for oral health. If an appropriate diet is not followed, it's more likely that we will develop tooth decay and gum disease. This applies at any stage in life, for example, acquiring healthy eating habits is essential to prevent the development of tooth decay, amongst other things, in children. Similarly, pregnant women need a suitable diet so that their children develop normal teeth. Socio-economic factors and even obesity are identified as risk factors for developing tooth decay⁵.

In children there is a particular relationship risk between the presence of malnutrition, tooth decay and the timeline of its appearance. Malnutrition adversely influences craniofacial growth and development, and an adverse precedent that can have various consequences, including: changes in the quality and texture of certain tissues (bone, periodontal ligament and teeth). There is a significant connection between oral health and nutritional status (failure to thrive) which could determine higher rates of tooth decay, a higher prevalence of gingivitis in children and an increase in the frequency of malocclusions. There are enamel defects associated with endocrine protein malnutrition, hypocalcaemia, vitamin and mineral deficiencies, in the context of enteropathies (coeliac disease, non-specific diarrhoea). Dental enamel defects have also been reported secondary to an excess of, or poisoning by vitamin D, fluoride or other minerals⁶.

Morbid obesity or its associated conditions or comorbidities (gastro-oesophageal reflux, antidepressants, high blood pressure, etc), cause increased incidences of tooth decay, changes in periodontal indices, the need for dental prostheses and monitoring of salivary flow (quantity and quality).

Tooth decay is one of the most prevalent infectious disease in humans and one of the main public health issues on a global scale. It is a disease where the teeth's hard tissues are modified and eventually dissolved, a process where localised destruction of these tissues occurs due to the action of bacteria that are involved. Molecular decomposition occurs in the teeth's hard tissues through a histochemical and bacterial process that ends in the progressive decalcification and dissolution of inorganic materials and the disintegration of its organic matrix. The formation of dental cavities starts in the form of small demineralised areas on the

enamel's sub-surface, which is able to progress through the dentine and reach the dental pulp, producing a chalky lesion on the surface of the enamel. If treatment is not carried out to remineralise the initial lesion, it could progress and turn into a cavity¹.

Demineralisation is the disorganisation of mineralised dental tissues due to the action of bacterial metabolism products and as a result of biochemical interchanges which take place in three phases: saliva, plaque bacteria and enamel.

The causes of tooth decay are multifactorial, although there are three key factors which will be added to in time: the host, microorganisms and diet. Environmental factors include, among other things, the presence or absence of health services and oral health programmes, socio-economic class, stress, ethnicity, culture, bio-dental engineering factors (biomechanical, biochemical and bioelectrical). The risk of tooth decay is due to socio-demographic, behavioural, physical/environmental and biological risk factors.

Diet plays a pivotal role in the development of tooth decay, particularly in people at risk. Usually, a combination of the high consumption of fermentable carbohydrates and the failure to include fluoride is associated with the increased incidence of tooth decay, however, there is no reason why it should occur in developed societies that have adequate exposure to fluoride and a history of low levels of tooth decay. Although there is no direct relationship between protein-energy malnutrition and tooth decay, vitamin (A, D), calcium and phosphorus deficiencies can cause changes in tooth development and delayed eruption. In protein-energy malnutrition, which is so prevalent in developing countries, a reduction in saliva immunoglobulin A has been detected, which could increase susceptibility to tooth decay (mucosal immunity has an impact via a reduction in secretory IgA).¹

Nevertheless, there are many epidemiological studies that correlate sugar consumption with the prevalence of tooth decay and which show a clear association between the frequency of consumption, eating between meals and developing tooth decay. Furthermore, there are a variety of food characteristics that may have an influence on their cariogenic potential, such as sucrose concentration, consistency, oral rinsing, the combination of foods, sequence and frequency of ingestion and food pH^{1,7}.

Foods are a chemical mixture of organic and inorganic substances that provide the human body with the nutrients necessary for its maintenance, growth and the development of its functions. Carbohydrates are currently seen as the cornerstone of a healthy balanced diet, followed by fats, whose consumption has decreased to prevent cardiovascular disease, and finally proteins. The current methods for preparing high carbohydrate foods have a great impact on their physical and chemical structure. The carbohydrates found in foods are primarily: monosaccharides

(glucose, fructose and galactose), disaccharides (sucrose = glucose + fructose, maltose = glucose + glucose, lactose = glucose + galactose), oligosaccharides (3 to 8 glucose molecules) and polysaccharides (starch). The cooking and preparation of foods affects the carbohydrate composition of foods and has an influence on its cariogenic potential⁸.

The frequency of consuming cariogenic foods, particularly between meals, has a strong relationship with the risk of tooth decay, because it favours changes in pH and lengthens the oral rinse time which increases the likelihood of enamel demineralisation. With regard to consistency and oral rinse there are various studies which have observed that certain foods, even those with a high sugar content, may have a greater solubility and are eliminated more quickly from the mouth, whereas foods that are high in starch (bread, cereals, potatoes) may increase acid production and are eliminated more slowly from the mouth.

Epidemiological studies show that breast milk and breast feeding helps physical and nutritional development in children, and has psychological, social, economic and environmental advantages, at the same time as significantly reducing the risk of developing a large number of acute and chronic illnesses. So, breast feeding, and as such, breast milk itself, is not cariogenic. However, various studies have shown that, in combination with other carbohydrates or administered more frequently at night or on demand, it is associated with early tooth decay. Tooth decay that develops as soon as the tooth erupts, on smooth surfaces, and which progresses quickly until it has had an extensive destructive impact on the teeth. For this same reason, the frequent use of bottles of juice or carbohydrates should be avoided. They can be filled with water, for example, using them more for soothing than feeding^{1,8-10}.

Tooth decay in preschool children is due to a combination of many factors, including the colonisation of cariogenic bacteria on teeth, the type of foods consumed as well as the frequency of the cariogenic bacteria's exposure to these foods, and sensitive teeth. The risk of developing tooth decay is greater if sugars are consumed very often and are in a form that means the food stays in the mouth for long periods of time. Sucrose is the most cariogenic sugar as it can form glucan, a substance that enables bacteria to adhere better to the teeth and that diffuses plaque acid and buffers. The frequent, elevated consumption of drinks that have been sweetened with sugar and the absence of normal tooth brushing are considered to be the factors that most associated with developing tooth decay.

It is advisable to avoid snacking between meals and to limit sugar consumption for during meal times, when saliva flow is greater and enables quick oral rinsing. This rinse period depends on the consistency of foods and the solubility of particles, as well as other individual characteristics such as chewing, the amount

and qualities of the saliva, etc. It is very important to limit the frequency of cariogenic carbohydrate consumption outside of meals.

So, foods that contain between 15 and 20% of sugars, especially sucrose, are the most cariogenic, particularly if they are eaten between meals. There are other carbohydrates such as fructose, that have greater sweetening properties than complete sucrose, but are less cariogenic. Similarly, Xylitol is not cariogenic as it is not used by bacteria to produce acid, and can even have an anti-caries effect as it increases saliva flow, raises the pH and reduces *Streptococcus mutans* levels because it interferes with its metabolism.

Furthermore, there are different foods that can have cariostatic effects. In animal studies it was observed that foods that contain high levels of fat, protein, calcium and fluoride can protect against tooth decay. Fats cover the tooth, reducing sugar and plaque retention, and may have toxic effects on bacteria. Proteins increase saliva's buffering capacity and have a protective effect on enamel. Fats and proteins jointly raise pH following the consumption of carbohydrates. Another type of food that has this protective profile are those that, through chewing, stimulate saliva flow and thereby buffer pH acid and stimulate the remineralisation of enamel¹.

Sugar free chewing gums use non-caloric sweeteners that can help prevent tooth decay. The sweet taste and chewing stimulates saliva flow, which contributes to the prevention of tooth decay. These chewing gums can contain minerals such as calcium, phosphate and fluoride to improve the teeth's remineralisation process. Some studies have reported that sugar free chewing gums, that are consumed after a meal, accelerate the cleaning of food debris and reduce the rate of developing tooth decay in children and adolescents.

It is important that adolescents reduce their elevated and frequent consumption of sugary drinks, as they are a factor that is particularly associated with tooth decay.

Since diet is a factor that determines the development of tooth decay, adequate information needs to be provided to patients. It's also worth remembering that an increase in sugar doesn't just involve an increased risk of tooth decay but also a growing risk of obesity, and therefore a greater disposition for adults to suffer illnesses such as diabetes, cardiovascular (hypertension, cholesterol), respiratory (apnoea, asthma), orthopaedic (fractures) and liver diseases^{11,12}.

Establishing dental care in children, even during pregnancy and afterwards in newborns, is one of the most suitable strategies for preventing tooth decay, including dietary recommendations and instructions on how to practice proper dental hygiene once the first milk teeth have erupted. The predisposition to developing tooth decay varies among individuals and between different teeth in the same mouth. The shape of the jaw and oral cavity, the structure of the teeth and the quantity and quality of saliva are important in deter-

mining whether certain teeth are more predisposed than others.

On the other hand, it's necessary to put systems in place to promote health using health education and information as the key, with specific dental programmes and programmes or strategies that involve a multidisciplinary team to pass on both dental and general healthy habits. Highlighting, in this sense, the existing education programmes for pregnant women, the oral health guidelines directed towards staff who work in nurseries and educational centres, the prescription of sugar free medications and the appropriate, simple and uniform labelling of foods by food companies⁸.

Weaknesses

The lack of resources and capabilities, *the lack of awareness, motivation and resistance to change*; in terms of finding primary (avoiding the appearance of new caries) and secondary prevention strategies (avoiding the progression of existing caries and/or eliminating them where possible). The age and history of cavities are the main factors around which the others are centred.

Age affects the structure of teeth, as evidence of changes in tooth eruption and its effects shows, and because from a certain age it makes dental hygiene difficult/ poor. There are three major periods in life when the risks of tooth decay reach their peak: between 5 and 8 years old, which affects the milk teeth and the first permanent molars; the period between 11 and 13 years, which affects the permanent teeth and between 55 and 65, when root cavities are more common.

The enamel of a recently erupted teeth is more vulnerable, 5-8 year olds (first molars) and 11-13 year olds (second molars) and susceptibility increases in the pits and fissures due to cleaning difficulties. Cleaning is more difficult until the teeth's occlusal plane is established and provided that occlusion is correct. Initial lesions may appear in the posterior areas of permanent molars before they have straightened and after they have erupted (vestibular inclination of the upper and lower lingual molars).

There is an increased prevalence of tooth decay in the maxillary central incisors: the incisive papilla is located near the mesiopalatal surface of these teeth and retains more plaque. It is the same for the vestibular fossae of the lower molars and the palatal fossa of the upper molars.

Widespread tooth decay can be found (large, destructive, unrestricted, in uncommon places) in milk teeth during the first year of life due to breastfeeding, medicines and sweetened pacifiers. Mothers with tooth decay contaminate the milk teeth of their children with streptococcus mutans, in particular orally (pacifiers, spoons for trying food, kisses, mouth to mouth contact, etc.).

Table I
DMFT index prevalence levels
in 12 year olds (WHO)

<i>DMFT</i>	<i>Prevalence levels</i>
0.0-1.1	Very low
1.2-2.6	Low
2.7-4.4	Moderate
4.5-8.5	High
+ 6.6	Very high

Pit and fissure tooth decay is more frequently associated with tooth decay in mixed dentition. Children whose primary teeth are free from tooth decay tend to have decay-free mixed dentition. Children who have proximal caries in their primary teeth tend to develop new lesions on the smooth surfaces of their mixed dentition.

Occlusal surface tooth decay is more common in permanent teeth and root decay is more common in adults, accompanied by gingival recession.

Tooth decay is usually measured using the DMFT index from 12 years of age (decayed teeth + cavity filling + missing teeth due to decay per tooth) and the DMF index is used for deciduous teeth (Table I).

Threats

High risks and environmental changes are the most serious threats.

Socio-economic status has a negative correlation with the risk of developing tooth decay; the higher the economic status, the lower the incidence and prevalence of tooth decay. Increased immigration and unemployment give rise to an increased risk of developing tooth decay. There is usually a less positive attitude towards dental health and healthy eating, mainly due to the socio-cultural and economic costs involved. A decrease in the use of dental products and the reduced attendance of dental appointments, as well as an increase in dysfunctional families, causes tooth decay.

Culture and religion are also factors that should be considered, as well as cultural levels, longer breast-feeding or increased dietary fat consumption.

Geographical location, in addition to affecting cultural and religious aspects, determines socio-demographic characteristics, such as sugar availability or the concentration of fluoride and other minerals in water and/or soil. For example, it seems that the presence of selenium and cadmium promotes tooth decay. In hot countries, rates of tooth decay are lower, due to the sun and its positive influence on calcium and phosphate metabolism, with the intervention of vitamin D.

Oral hygiene and eating habits are probably the most important¹³.

Plaque bacteria are a prerequisite for the initiation of tooth decay, it's mechanical and/or chemical removal helps control the risks associated with eating habits, and therefore oral health. A large number of microorganism in the mouth, particularly in hard-to-reach-areas; the production of different acids during bacterial metabolism with the ability to dissolve mineral salts which are part of the tooth's structure, retaining factors (open cavities, overflowing tooth fillings, fixed or removable prostheses, orthodontic appliances, gingival recession, etc) are part of a series of threats that healthy teeth are regularly exposed to. The frequency and correct brushing of teeth, the use of additional hygiene devices such as dental floss or interproximal brushes are also determining factors. The frequency of brushing and being an immigrant have a significant association with the incidence and prevalence of tooth decay¹.

The truth is that *diet* plays an important role in the development of tooth decay (closely linked to the frequent consumption of carbohydrates and high cariogenic activity) and the fact is that frequency is more important than the amount consumed; there are authors who claim that sugar is not a causal factor in the etiopathogenic process of tooth decay, but it is a risk modifier. We found a statistically significant relationship ($P < 0.05$) between the colonisation of streptococcus mutans and feeding bottles containing sugary drinks⁶. The stickiness and retention of food, in the hard and soft tissues, depends on the type of foods consumed. Fats in food reduce the oral retention period; liquid foods are eliminated a lot faster than solid foods. The food retention parameters and acid formation are important in the development of tooth decay, but they are not a good epidemiological indicator of tooth decay. Therefore, it is difficult to relate the cariogenicity of foods to an individual physical property, such as its buffering capacity or acid production. The physical form, the consistency and the frequency of consumption, as well as the sugar content (particularly sucrose, which is the standard), are major determinants of the cariogenic potential of foods. Sucrose and starch are found in many products we eat that are not related to their presence (examples are fruit, milk, bread, cereals and even vegetables). The key to eating properly is not to avoid these products, but to know how to eat the right amounts of them and at the right times of day (avoiding snacking between meals). It's not just what you eat, but when you eat that makes a big difference to dental health^{1,6,8}.

The characteristics of foods that have an increased potential for causing tooth decay are: texture (the consistency of foods), taste, carbohydrate content and composition (direct, indirect or 'hidden', cariogenic potential (CPI of Sucrose = 1), prolonged retention

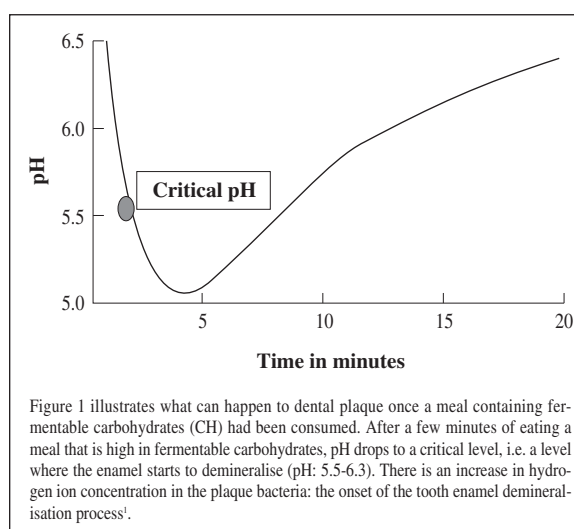


Figure 1 illustrates what can happen to dental plaque once a meal containing fermentable carbohydrates (CH) had been consumed. After a few minutes of eating a meal that is high in fermentable carbohydrates, pH drops to a critical level, i.e. a level where the enamel starts to demineralise (pH: 5.5-6.3). There is an increase in hydrogen ion concentration in the plaque bacteria: the onset of the tooth enamel demineralisation process¹.

Fig. 1.—Stephan Curve (prepared by the author).

(rinsing or clearance), intake during or between meals, protective factors (cheese, phosphates), consumption frequency (Critical pH: (5.2-5.5) that is expressed graphically by the Stephan curve, which relates the frequency of meals to the exposure time¹ (Fig. 1).

In addition to carbohydrates, there is also a connection with: nutritional deficiencies, protein, vitamin and mineral deficiencies, a diet that reduces saliva secretion and composition, hypoplasia caused by nutritional deficiencies and widespread tooth decay.

Controlling high sucrose diets, the frequency of tooth brushing and social class are predictors for developing tooth decay. Regular brushing (2 times/day) with fluoride toothpaste may have a bigger impact in young people than restricting sugary foods.¹

Physical and environmental factors such as previous experience of tooth decay (which we have also commented on under weaknesses), the areas at risk and the arch form should also be taken into consideration.

In this section, with regards to tooth decay, we highlighted that: primary teeth, a previous history of treated or untreated tooth decay, numerous unmonitored initial caries lesions, the presence of initial caries lesions or white spots, rare cases of tooth decay in previous groups, more than three new lesions per year, poor or non sealing of specified pits and fissures are good risk indicators for the future development of tooth decay

We have highlighted *areas at risk and the dental arch form*: premature dental extractions, multiple restorations, positioning of teeth in the arch (overcrowding, malocclusions,...), the composition of dental tissues and enamel maturation with fluoride uptake and carbonate, magnesium and sodium release, the tooth's surface texture (cracks and/or flakes, developmental abnormalities, wear on the tooth's surface (for example, prosthesis retainers).

The locations where tooth decay occurs most often in deciduous teeth are: the pits and fissures, the occlusal surfaces: in molars (primarily occlusal-buccal); buccal in canines and mesial in the incisors. The most affected areas are usually in the lower posterior molars, the upper posterior molars, the upper and lower anterior incisors. In permanent teeth the most common locations are the occlusal surface (buccal and lingual), especially the molars and subsequently the premolars.

Tooth decay in the pits and fissures is relatively easy to prevent with the use of sealants, which once they have been 'filled' prevent nutrients from entering and creating a bacterial biofilm. If the fissures are in the shape of a 'U', 'Y' and 'YK' there is an increased risk of tooth decay. Those that are in the shape of a 'V' and 'Y' pose less of a risk.

A white spot represents the first, clinically visible, stage of enamel demineralisation and is the main warning sign for putting a comprehensive strategy in place to prevent tooth decay, by promoting a healthy, balanced diet as well as monitoring dental hygiene and using topical fluoride treatments, and assistance with monitoring from the dentist (Table II).

One of the most important factors in developing tooth decay, along with diet and time, are microorganisms, as without their presence in the mouth there would be no tooth decay. The most frequently involved microorganisms are: *Streptococcus mutans* (associated with the onset of tooth decay: occlusal and smooth-surface) and *Lactobacillus* spp. (it appears once the lesion has been established, particularly in undercut areas and root caries). Other lactobacilli that should be considered are *casei* and *acidophilus*. An increase in salivary enzymes has been noted in individuals with poor oral hygiene and is associated with an increase in tooth decay. Once these microorganisms have been counted, we consider *S. Mutans* levels to be high if there are more than 1,000,000,000 colony-forming units per millimetre, and for *lactobacillus* if it is higher than 100,000 colony-forming units per millimetre in saliva.

Salivary secretion is essential due to the different functions it performs such as mechanical sweeping and

oral rinsing, along with the muscles and soft tissues of the mouth, antimicrobial action (children and adults with immunological disorders are more susceptible to tooth decay), its buffering effect, its viscosity and its effect on reducing enamel solubility. Secretion may be spontaneous (primarily from the submandibular and sublingual glands) and/or stimulated by chewing paraffin for five minutes (mainly from the glands previously mentioned plus the parotid gland). We talk about low levels of stimulated saliva when saliva production is lower than 0.7 millilitres per minute, and low levels without stimulation when it is lower than 0.25 millilitres per minute^{1,8}.

The sensation of a dry mouth is called xerostomia, and may or may not be accompanied by hyposalivation (reduced saliva production). Hyposalivation may be caused by systemic, local or medicine-induced disorders so, for example, we have highlighted certain medications: antipsychotics, antihistamines, diuretics, antihypertensives; anorexia, episodes of depression and their treatment, diabetes mellitus, etc.

Strengths

Included in the internal analysis are the strength of different capabilities, the natural advantages and the superior resources.

Historically *race* has been talked about, observing that pure ethnic groups suffer with tooth decay less often. Curiously, Afro-Americans have a higher incidence than Africans. Undoubtedly, *an individual's genetic predisposition* has an influence on tooth size, its crystal formation and immunity (natural immunity to tooth decay seems to be connected to HLA-DR locus activity located in chromosome 6 and in individuals that are decay resistant: HLA-DR W6 generates activity in the helper T lymphocytes, increasing the number of antibody-forming cells. Certain HLA class II (DR) alleles are related to oral salivary microorganism populations such as *S. Mutans* and *Lactobacilli*.

The genetic factors aren't fully known yet. It is also due to cultural and socio-economic factors (dietary habits, hygiene, health and dental education).

Gender, referring to women, is also a protective factor. This is explained by the earlier eruption of permanent teeth in females, by better dental hygiene habits and greater concern about preventing tooth decay. Hormonal changes during the menstrual cycle, breastfeeding and pregnancy can alter saliva composition and encourage bacterial growth, especially if adequate teeth cleaning is forgotten and the consumption of sugary foods is increased, and therefore encourages tooth decay.

The possibility of using *public and private resources* with professionals that have a high level of

Table II <i>Characteristics for diagnosing white spots</i> <i>(prepared by the author)</i>	
Visibility	Opaque when dry
Location	Close to the gingival, sulcus symmetrical or asymmetrical
Affected teeth	Primary molars both dentitions
Shape	Kidney or oval-shaped
Related to plaque bacteria	Yes
Related to fluoride consumption	No

scientific knowledge (awareness campaigns, free or low cost treatment, etc). The existence of collaborative agreements, between the various entities, particularly for the benefit of children, should also be considered¹¹⁻¹³.

Opportunities

The most significant opportunities are the autonomous communities' oral health programmes and new technologies. The Universities' and Vocational Dental Training Centres' most competitive prices should also be added.

Teacher training programmes for compulsory health education are an alternative that should be considered.

The quality and quantity of dental products available in pharmacies is also a factor that should be considered when it comes to home programmes for protecting against tooth decay.

The most available information via new communication methods is an alternative to misinformation. It is also worth highlighting the information programmes which take place in the professional associations of doctors, dentists, hygienists, nutrition experts and pharmacists, as well as professional scientific societies and bodies^{1,14}. In figure 2 an education feedback loop is displayed.

Recommendations

We recommend the following ten guidelines:

1. Watch what you eat: time and frequency.
2. Reduce sucrose consumption to less than 50 mg/day.
3. Reduce the number of exposures to or opportunities for consuming sucrose and sugary products (moments).
4. Avoid snacking between meals and reduce the consumption of sticky or viscous foods. Avoid, as much as possible, acid-producing foods (crisps, chips, chocolate milk, filled biscuits, sweet dried fruits, dates, etc).
5. Substitute sucrose for non cariogenic sweeteners, as required when there is a high risk of tooth decay, particularly between meals. Promote the use of xylitol in chewing gums and sweets.
6. Visit the dentist at least twice a year so that the risks can be assessed and an individual early diagnosis can be made. Avoid dental extractions.
7. Use products that are suitable for dental hygiene. Use toothpaste, mouthwashes and dental gels that contain fluoride.
8. The sealing and remodelling of the tooth profile. Removing barriers to dental hygiene and fermentable carbohydrates retention.

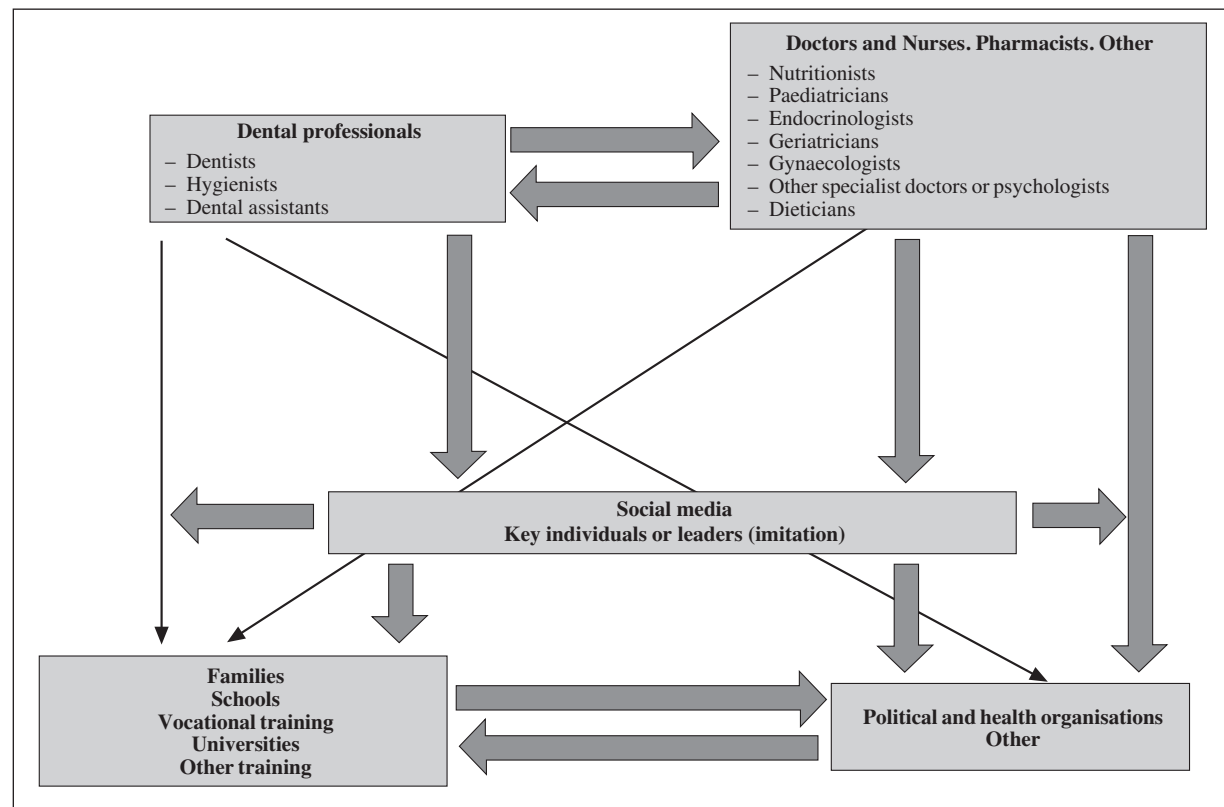


Fig. 2.—Dietary/tooth decay educational feedback loop (prepared by the author).

9. Improve oral health education at home and in schools. Paediatricians, educators, parents, GPs and nutrition experts should pay more attention to dental health.

10. Dental health is essential for general health: physical and mental.

Conclusions

1. The full cariogenic potential of a food is influenced by:

- Fermentable carbohydrate content (acid-forming potential).
- Components in food or the diet that may have cariostatic properties or a food's ability to remain in the mouth. Oral rinsing times (clearance) may be extended due to retentive factors in the teeth, a low saliva secretion rate, highly viscous saliva or low muscle activity.
- Eating patterns. An increase in chewing resistance and the presence of fat in foods, an increase in clearance speed.
- The sequence and frequency of consumption are closely linked to incidences of tooth decay, as the consumption of sugars between meals poses the highest risk of tooth decay and eating patterns are more important than the frequency.

2. The Total Cariogenic Load (TCL) defines individually the net effects from assessing the risk factors (cariogenic potential, frequency of consumption, etc) together with the protective factors (intrinsic, hygiene, fluoride, sealants, etc).

3. Due to the existing relationship between diet and oral health we need to teach our patients and the general population about the importance of proper eating habits and provide nutritional and dental health advice on this subject.

References

1. González Sanz A, González Nieto B, González Nieto E. 2012. Nutrición, dieta y salud oral. En Castaño A, Ribas B. Odontología preventiva y comunitaria. La odontología social, un deber, una necesidad, un reto. Sevilla: Fundación Odontología Social. 155-69.
2. Marinho VC, Higgins JP, Logan S, Sheiham A. 2003. Topical fluoride (toothpastes, mouthrinses, gels or varnishes) for preventing dental caries in children and adolescents. *Cochrane Database Syst Rev* (4): CD002782.
3. Greig V, Conway DI. 2012. Fluoride varnish was effective at reducing caries on high caries risk school children in rural Brazil. *Evid Based Dent* 13 (3): 78-9.
4. Autio-Gold J. 2008. The role of chlorhexidine in caries prevention. *Oper Dent* 33 (6): 710-6.
5. Hooley M, Skouteris H, Millar L. 2012. The relationship between childhood weight, dental caries and eating practices in children aged 4-8 years in Australia, 2004-2008. *Pediatr Obes* 7 (6): 461-70.
6. Lamas M, González A, Barbería E, Garcia Godoy F. *Am J Dent* 2003; 16 Spec No: 9-12.
7. Mobley C, Marshall TA, Milgrom P, Coldwell SE. The contribution of dietary factors to dental caries and disparities in caries. *Acad Pediatr* 2009; 9 (6): 410-4.
8. González Sanz AM. 2004 ¿Son todos los carbohidratos cariogénicos? *Nutr Clín* 2004; 24 (4): 27-31.
9. Lueangpianamut J, Chatrchaiwiwatana S, Muktabant B, Inthahit W. 2012. Relationship between dental caries status, nutritional status, snack foods, and sugar-sweetened beverages consumption among primary schoolchildren grade 4-6 in Nongbua Khamsaen school, Na Klang district, Nongbua Lampoo Province, Thailand. *J Med Assoc Thai* 2012; 95 (8): 1090-7.
10. Tinanoff N, Palmer CA. Dietary determinants of dental caries and dietary recommendations for preschool children. *Refuat Hapeh Vehashinayim* 2003; 20 (2): 8-23.
11. Steyn NP, Temple NJ. Evidence to support a food-based dietary guideline on sugar consumption in South Africa. *BMC Public Health* 2012; 12: 502.
12. Palacios C, Joshupura K, Willett W. Nutrition and health: guidelines for dental practitioners. *Oral Dis* 2009; 15 (6): 369-81.
13. Levine RS, Nugent ZJ, Rudolf MC, Sahota P. 2007. Dietary patterns, toothbrushing habits and caries experience of schoolchildren in West Yorkshire, England. *Community Dent Health* 2007; 24 (2): 82-7.
14. Sarmadi R, Gahnberg L, Gabre P. Clinicians' preventive strategies for children and adolescents identified as at high risk of developing caries. *Int J Paediatr Dent* 2011; 21 (3): 167-74.

Sugar and diabetes; international recommendations

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Abstract

Nutrition in the diabetic patient is not just a mere nutrient but his treatment is based. In fact, international scientific societies have called "medical nutrition therapy" to give it the emphasis it deserves. Nutritional recommendations of scientific societies have been changing in recent years with evidence-based medicine. Regarding the consumption of sugar, most believe it does not affect metabolic control if it is replaced by other carbohydrates, but does not indicate a specific amount.

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Key words: *Diabetes. Sugar. Nutritional recommendations.*

AZUCAR Y DIABETES; RECOMENDACIONES INTERNACIONALES

Resumen

La nutrición en el paciente diabético no es sólo un del mero aporte de nutrientes sino que es la base su tratamiento. De hecho, las sociedades científicas internacionales lo han denominado "tratamiento médico nutricional" para darle el énfasis que se merece. Las recomendaciones nutricionales de las sociedades científicas han ido cambiando en los últimos años con la medicina basada en la evidencia. Respecto al consumo de azúcar, la mayoría considera que no afecta el control metabólico si éste se sustituye por otros hidratos de carbono, pero no se indica una cantidad concreta.

Nutr Hosp 2013; 28 (Supl. 4):72-80

Palabras clave: *Diabetes. Azúcar. Recomendaciones nutricionales.*

Abbreviations

DM: Diabetes mellitus.
DM 1: Diabetes mellitus type 1.
DM2: Diabetes mellitus type 2.
IBG: Impaired basal glucose.
GIT: Glucose intolerance (GIT).
GIX: Glucose index. ADA: American diabetes association.
MNT: Medical nutrition therapy.
HbA1c: Glycated haemoglobin.
CH: Carbohydrates.
IG: Glycaemic index.
SoFAS: (solid fats and added sugars).

Introduction

Diabetes mellitus is one of the most prevalent endocrinological processes in the general population.

It is currently estimated that 366 million people suffer from DM (8.3%). 95% of these cases are mellitus type 2 (DM2). In 2003, it is estimated that 552 million people will suffer from this chronic illness (9.9%)¹.

In two decades, the number of adults with DM in developed countries will have increased by 20%, but the increase will take place especially in developing countries, where the predicted figure is 69%. This spectacular change is related to the ageing population, and more specifically the current obesity epidemic².

In Spain, the most recent figures³ show that the prevalence of DM is at 13.8%, although approximately half of these patients (6.0%) do not know they have DM. In terms of pre-diabetic conditions, impaired basal glucose (IBG) is at 3.4%, and glucose intolerance (GIT) is at 9.2%. 2.2% have both conditions.

The objectives of nutritional treatment of DM, apart from its aetiology, are the following:

1. Reach and maintain:
 - a) Plasma levels of glucose within normal limits, or as near to them as feasibly possible, while observing safety.
 - b) A lipids profile that reduces the risk of cardiovascular illness, while observing safety.

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- c) Artery pressure levels within normal limits, or as close to them as feasibly possible, while observing safety.
 - d) An adequate weight for the prevention of obesity development, or treatment of existing obesity, especially the visceral presence type.
2. Prevent or at least slow down the development of chronic complications of DM by modifying nutrient intake and lifestyle.
 3. Meeting individual nutritional needs, taking the patient's personal and cultural preferences into account, so that recommendations are followed in the long term.
 4. Improve general health, by promoting healthier food options and adequate levels of physical activity.

The nutritional approach in patients with DM is much more than just providing nutrients, because it forms the basis of their general treatment. The nutritional treatment is included within a physical exercise program, and goes alongside the pharmacological treatment. The American Diabetes Association (ADA)⁴, The Canadian Diabetes Association⁵, The American Dietetic Association⁶ and the American Association of Clinical Endocrinologists⁷ have called it "Medical Nutrition Therapy" (MNT) to give it the importance it requires. MNT is not just intervening in the control of glucose; it affects the whole metabolic system, and furthermore, it prevents the appearance and development of diabetes and related problems.

Review of recommendations made by various scientific associations

In spite of international recognition of the importance of MNT, it is not yet fully established. Recommendations made by scientific associations have changed substantially from the publication of the first ones by the European Society for the Study of Diabetes⁸ up to the latest recommendations presented by Diabetes UK⁹ and by the German Nutrition¹⁰ in the light of evidence based medicine.

In this review we will look at the most important and recent recommendations in terms of carbohydrate (CH) intake. The DAFO method will be used for the case of sugar consumption.

Table I shows the recommendations that carry most evidence from the five most important scientific societies, grouped in terms of category for ease of comparison.

The first thing to notice is that MNT does not appear in the European Society's recommendations from 2004. However, the ADA's "position statement"¹¹ does appear for the first time. From then on, all the societies have adopted it, and they now recommend it to a higher level than the ADA⁴ itself.

The diet is the fundamental basis for the treatment of DM, because it can reduce glycated haemoglobin (HbA1C) by up to 2%. In spite of that, there is no special diet for diabetic patients.

The current trend is to follow recommendations made for the general population such as a healthy diet as recommended by the Canadian guidelines⁵. The factor that makes DM different is that individualisation is key. Every individual diabetic patient has specific needs in terms of calories, according to their age, sex, weight, levels of physical activity etc.

The distribution of macronutrients depends on lipid profile, renal function, timetable, lifestyle, dosage of relevant glucose-lowering medication, and above all, personal, family and cultural preferences must always be considered.

Carbohydrates have historically been seen as the energetic substrate that has most impact on blood sugar. The total amount of carbohydrates taken in is the main factor in reactions after food consumption, although other factors exist such as the type of carbohydrate, the amount of fibre, the way of cooking, the ripeness of the fruit etc. Furthermore there are other factors apart from CHs that also influence blood sugar after food consumption, such as blood sugar before eating, distribution of macronutrients throughout the whole meal, glucose lowering treatments and the patient's insulin tolerance. Because of this, the majority of scientific associations recommend individual treatments, in conjunction with pharmaceutical treatment and based on diet in portions.

There is a lot of controversy surrounding the usefulness of slow-release CHs with a low glycaemic index (GI). The GI of foods is a concept that was developed in order to compare reactions after eating a fixed amount of foods rich in CH. This is defined as an increase in blood sugar with respect to fasting basal glucose in an area below the curve of two hours after eating a set amount of that food (usually equivalent to 50g of CH) divided by the response to a reference food (usually glucose or white bread).

The European Diabetes Association⁸ had already made an A grade recommendation in favour of foods rich in CHs, but with a low GI, in 2004. That recommendation was upheld by the UK Diabetes Association⁹ in 2011. However the American Diabetes Association⁴, the American Dietetic Association⁶, and the Canadian Diabetes Association⁵, have taken years to join with the European associations, and although they still have some misgivings, they concede that this type of diet may produce a modest benefit of control over blood sugar after food consumption, with a B grade recommendation.

Aside from these scientific arguments, all agree unanimously that a diet should provide CH in the form of fruit, cereal, pasta, pulses, vegetables and root vegetables (such as potatoes), which are all foods with low GI.

Table I
Nutritional recommendations from different diabetic societies

	<i>European Diabetic Association* (2004)</i>	<i>American Association of Clinical endocrinologists* (2007)</i>	<i>American Diabetic Association† (2008)</i>	<i>American Diabetics Association* (2010)</i>	<i>British Diabetics Association* (2011)</i>
<i>Nutritional Medical treatment</i>	This concept did not exist when these guides were written.	MNT is an essential component in any program for the treatment of DM (A).	Both for patients with DM and with pre-DM should receive MNT (B). Dietary advice should be sensitive to individual needs and their capacity to be able to change (E).	MNT reduces HbA1c between 0.25-2.9%, depending on the type and duration of the diabetes. Lipids, blood pressure, and weight also improve, less need for pharmaceuticals and a reduction in the risk of comorbidities progression (A).	MNT is effective in DM and at high risk of developing DM2, when integrated in educational and clinical care (A).
<i>Nutrients distribution</i>	It is not justified to recommend very low-carbohydrates diets in diabetes (B).	There's no specific diet for DM according to this Association (D). Fat intake < 30% and saturate fats < 10% of the total caloric consumption; increase fibre to > 15 g/ml kcal (A). Diet customization according to weight, drugs, meal preferences, life-style and lipidic profile (A).	Both low-carbohydrates diets and low-fat diets can be effective for reducing weight (A).	Must be based on the recommendations for a healthy diet. There is no ideal percentage for the energy distribution (A).	To achieve an optimal glycaemic control in DM2 it is important to manage the total amount of energy intake and not its composition in macronutrients (A).
<i>CH consumption</i>	The carbohydrate contribution depends on the metabolic characteristics of the patient (A).	The total CH contribution should be 45-65% of the total calories unless otherwise indicated (D).	Monitoring the intake of carbohydrates through the exchange method is a key strategy in order to maintain glycaemic control (A). De DM1 patients treated with fast action injected or pumped insulin should adjust their previously taken insulin doses to the amount of carbohydrates intake (A).	For patients with fixed doses of insulin or hypoglycaemic the CH intake should be the same every day (A). For patients that adjust their insulin doses with each meal, this will depend on the CH content (A). The total amount of contribution total (proteins and fats) should be considered.	For DM1 the CH content is the main nutritional factor for Glycaemic control (A). For DM1 that use multiple insulin doses or an insulin pump, it is beneficial to adjust the insulin doses to the quantity of CH ingested (A). In DM1 with fixed insulin doses, it is beneficial to ingest a daily fixed amount of CH (C). In DM2 that total quantity of HC consumed is a strong predictor of the total amount of CH ingested through exchanges and is the key strategy in order to achieve glycaemic control (A).
<i>Glycaemic Index GI</i>	Foods rich in CH but with a low GI are advisable (A).	No reference made.	The use of the glycaemic index can bring a small additional benefit, but higher than if we just consider the contribution of total carbohydrates (B).	There are contradictory results regarding its effectiveness due to the different definitions of high or low GI (B).	In DM2 diets with a low GI can reduce HbA1c > 0.5% (A).

Table I (cont.)
Nutritional recommendations from different diabetic societies

	European Diabetic Association* (2004)	American Association of Clinical endocrinologists ³ (2007)	American Diabetic Association ⁴ (2008)	American Diabetic Association ⁶ (2010)	British Diabetics Association ⁹ (2011)
<i>Fibre</i>	The diet of the DM patient, both type 1 and type 2, should include vegetables, pulses, fruits and wholegrain cereals. When CH intake is high, it is important to choose foods rich in fibre and with a low GI (A). It recommends the consumption of natural high fibre foods (A). The dietetic fibre should be >40g/day, half of which soluble. Benefits have been observed even with lower fibre contributions (A).	At least 25-50 g/day Of fibre should be consumed per day or 15-25 g/1,000 calories (A).	Individuals with a high risk of contracting DM2 should have a high fibre diet (14 g/1,000 kcal) and wholegrain foods (B). It is recommended that the dietary carbohydrates come from fruit, vegetables, wholegrain foods, pulses and skimmed milk (B). As for the general public, the consumption of high fibre foods is recommended, but no more than that of the general population (B).	There are no definitive conclusions regarding the effects of a diet rich in fibre and glycaemic control (A) but there are regarding total cholesterol (A). The intake of fibre recommended for patients with diabetes is similar to that of the general population (14 g/1,000 kcal) Diets with 45-50 g/day have improved their glycaemic control, but with doses more typical in real life (24 g/day) do not show any effects. It is recommended for people with DM to consume foods that contain 25-30 g of fibre a day (7-13 g soluble).	Diets with a low GI and a high fibre and wholegrain content protect against the apparition of type 2 diabetes (B).
<i>Non nutritional sweeteners</i>	There are no specific recommendations.	No reference made.	Polyalcohol and low calorie sweeteners are safe when consumed within the limits established by the (A).	Non nutritional sweeteners do not have an effect on glycaemic control. Although we should take into account that some products that contain these nonnutritive sweeteners also contain carbohydrates (C).	Sweeteners without calories are safe when consumed in moderation and can reduce HbA1c when taken as part of a low calorie diet. (Without being a recommendation).
<i>Consumption of sugar</i>	Moderate intake of sugar (50g/day) can be incorporated into the diet of patients with both type 1 and type 2 diabetes if the glycaemic control is satisfactory (A).	No reference made.	Foods that contain sugar can be substituted for other CH in the diet plan and if added, it must be taken into account in both hypoglycaemic pharmaceuticals and in the risk of increasing energy contribution (A).	The patient with diabetes can consume foods that contain sucrose, but always substituting them for others that are rich in carbohydrates. An intake of 10 to 35% of the total intake of this type of food does not alter glycaemic or lipid control when substituted for an isocaloric quantity of other carbohydrates (A).	In DM1, the consumption of sugar does not affect glycaemic control differently to other types of CH. Patients that consume a variety of sugars do not show a different glycaemic control if the total carbohydrate contribution is similar (Without constituting a recommendation). Fructose can reduce the post prandial blood sugar level, when substituting sugar (Without constituting a recommendation).

*In brackets the strength of recommendation.

With regard to fibre in diets, foods that are rich in fibre like fruit and vegetables are still recommended, especially wholegrain cereals. The recommendation for effective use of fibre for improvement of lipid metabolism and the glycaemic system is graded A or B depending on the scientific association. The negative aspects are their secondary effects on digestion, and their palatability means they cannot be used in large quantities. Generally, the consumption of foods that are rich in fibre is recommended to the general public, and there is no need to increase the amounts of fibre for diabetic patients.

Regarding non caloric sweeteners, most guides do not provide relevant information. They are simply considered to be safe in small quantities within the context of a low calorie (hypocaloric) diet.

Sugar consumption, using the DAFO model

Threats

Social and scientific environments can play a negative role in sugar consumption in the diets of patients with DM, as well as the competition of sweetening products aside from sugar.

Sugar consumption by diabetics has been roundly criticised, with its prohibition seen as fundamental to the treatment of diabetes. The popular definition of diabetes is “sugar in the blood”, so the connection between sugar and blood sugar plasma in diets is well recognised.

The first problem to be tackled is the diverse nature of the diabetic population in terms of the underlying understanding. In other words, DM2 has an important component of insulin resistance, whilst DM1 has to do more with insulinopenia (insulin deficiency). In the first case, the goal is to combat weight excess by reducing the total calorie intake, while the second case works by controlling CH intake and insulin deficiency.

Another important aspect is the lack of agreement between scientific associations in terms of recommendations on sugar consumption in diets amongst patients with DM, as well as the degree of evidence. In 2004, 50 g of sugar per day was seen as acceptable to the European Association⁸, with an A grade recommendation. The ADA was even more liberal with regard to sugar consumption, with the only limitation being its substitution for other foods that are rich in CH, with as much as 10-35% of total calorie intake per day according to the Dietetic Association⁶. On the other hand, the Canadian Association has a maximum limit of 10% with a B grade recommendation, and the American Association of Clinical Endocrinologists⁷ and UK Diabetes⁹ do not make explicit reference to this.

In the USA, following the publication of the Dietary Guides for the general population in 2010¹², there has

been a reduction in the consumption of drinks containing added sugars from 2000 to 2008, although energy drink consumption levels have stayed the same. The intake of these drinks may provide from 5 to 15% of the recommended total energy consumption¹³.

There are also artificial sweeteners such as fructose and polyols (low calorie sugar replacers / sugar alcohols). Fructose produces a weaker reaction in blood sugar after food consumption than sucrose, which is why it has been used so often in sweet foods, such as turrón (Spanish nougat) or cakes, for diabetic patients. Polyols also produce a weaker reaction in blood sugar, and they are often used in chewing gum and sugar-free sweets.

Lastly, the existence of a safe commercial alternative to artificial sweeteners has meant that diabetic patients use it in coffee instead of sugar. The ADA⁴ see them as a way of reducing CH consumption, and the North American Cancer Institute¹⁴ has not found any evidence that they increase the risk of cancer.

Weaknesses

Sugar alternatives that limit or reduce consumption amongst DM patients show the following characteristics (Fig. 1).

The GI of sugar is high, therefore historically its consumption has always been avoided in diets created for diabetic patients. In hospitals, the “sugar-free” term has always been used to describe low GI food diets.

The consumption of dietetic sugar produces a fast blood sugar reaction disproportionate to the amount of CHs taken in. This fact has been linked to states of endogenous hyperinsulinism (high internal levels of blood sugar), which stimulates the physiopathological process of DM2.

Sugar is always the first food that is removed from diets for slimming or losing weight. Obesity and DM2 are linked in the majority of cases, which is another reason for its restriction in diets for these patients. Sugar is usually added to other foods such as milk, juice or soft drinks, enriching them with calories that are easily absorbed and not very filling, so they are not usually included in records of how much has been consumed in one day.

There are some foods, such as industrially produced bread rolls, that like “sweet” foods are associated with sugar. However, there is a high amount of invisible fats in their makeup. These are fats that increase calorie concentration, because sugar alternatives only add 4kcal/g to the diet, whilst fats provide 9 kcal/g. Furthermore, these are usually saturated fats, with a very negative effect on lipid metabolism.

Finally, it must be mentioned that they are often associated with the appearance of cavities in the general population and also amongst DM patients. Although a clear link has not been demonstrated

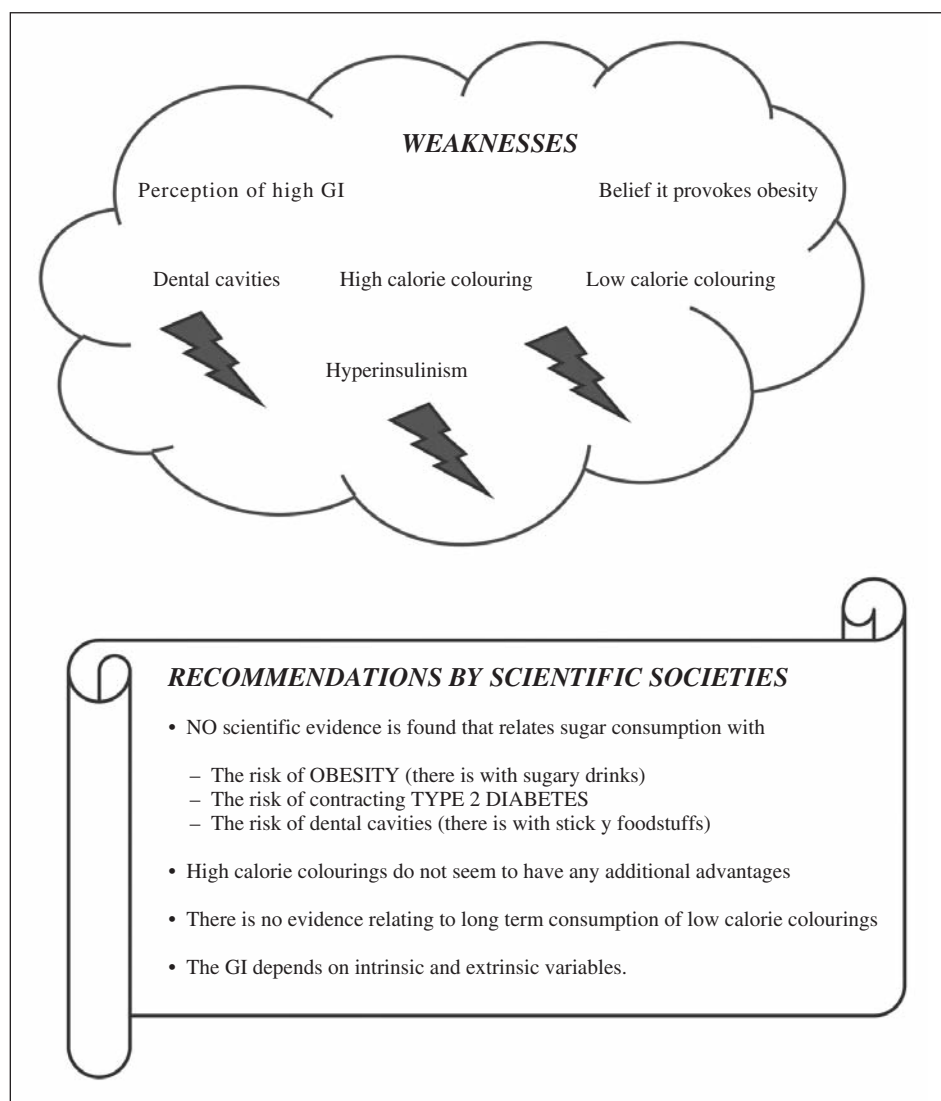


Fig. 1.—Strengths and weaknesses.

between sugar consumption and the appearance of cavities, it is true that foods that have a sticky consistency are related to bad dental habits.

Strengths

There are some strong points in favour of sugar consumption amongst patients with DM, due to the characteristics of this food.

The most important scientific societies have changed dietary recommendations based on evidence in order to help DM patients follow their diet correctly.

Sugar's main virtue is its quality as a sweetener, which has no comparison in other products, with calories or without. This means that other sweeteners are known as "sugar alternatives", but sugar still has public preference.

Sugar intake provides energy that is absorbed quickly and used easily by the nervous system so it is a

necessary food to overcome a spontaneous or a drug induced hypoglycaemia.

Sugar is added to other foods which are strongly related to social situations, such as having a coffee or having a drink. In many situations, DM patients consume it during social meetings, and need to remember their condition, ensuring that they do not have sugar, or they ask for a sweetener that does not contain calories. However it is quite common these days that a healthy population worried about weight consumes "diet" or "sugar free" food. Puddings such as cakes or tarts are consumed in special situations such as birthdays, and it is very difficult for DM patients to say no.

When a food is prohibited and removed from a diet, the patient longs for it, and this makes it harder for them to follow the diet recommended to them. When sugar consumption is permitted, DM patients adhere to the diet far better. In a Spanish study of 876 patients with DM by Muñoz Pareja et al.¹⁵ the parallels between

intake and the recommendations made by the European and American Diabetes Associations are appreciated. Only 3.4% follow the recommendations on fibre (by default), 10% follow the sugar recommendations (by excess) and 25.5% in terms of CHs (by default). It is noticeable that DM patients behave in the same way as people without DM, and adherence to the Mediterranean diet is only 57%. They consume large amounts of typical Mediterranean food, such as olive oil, fruit and vegetables, or fish, but they also have typical foods contained within a Western diet like meat products rich in saturated fats, and sugars instead of complex CHs. It is also worth pointing out that the best concordance with dietary recommendations is associated with better blood glucose control, especially within the Mediterranean diet.

It is important that children and teenagers with DM have the same diet as the rest of their family. Total prohibition of sweets and treats is very unlikely to be successful. It is better to accept that they can have these in moderation or within the context of special occasions, or mixed with other foods to reduce the GI.

Another special situation is Christmas sweets and *turrón* (Spanish Christmas nougat). “Diabetic *turrón*” is not recommended; it is better to have high quality *turrón*. Special food gives the patient a false sense of security which makes them eat larger amounts, so the end result is greater weight gain.

Opportunities

Nutritional recommendations by the different scientific associations, based on scientific evidence, state that sugar alternatives do not increase blood sugar any more than other CHs in isocaloric amounts (low in fat and carbohydrates) under certain conditions (Table I). Special attention is given to DM2 patients with obesity, so they calculate sugar intake according to how many calories it provides. With DM1 patients, sugar consumption is calculated within the context of total amount of CHs and its coordination with daily insulin intake and dosage. In general, patients with any kind of DM should have achieved a high level of knowledge on diabetes and good control of metabolism so that consumption of sugar alternatives does not have any negative effects.

The German Nutrition Society¹⁰ did not find any scientific evidence to suggest that sugar consumption is related to obesity in their review from 2012. However, it did find that it is probable that high consumption of sugary drinks is linked to a greater risk of obesity, depending on amounts. This is based on a meta-analysis by Mattes et al.¹⁶ which states that added consumption of 250 kcal (about 600 ml) of sugary drinks over the course of 3 to 12 weeks may lead to a weight gain of 0.2 kg. With regard to children and teenagers, there are several meta-analyses with contra-

dictory results, but the review by Mattes et al. shows that there is a link, especially amongst those who were obese or overweight beforehand.

None of the scientific associations found a link between sugar consumption and the risk of contracting DM2, and the German Nutrition Society¹⁰ found that there is probably no link between sugar consumption and the risk of DM2. However, this same review found that it is possible that sugar consumption is linked to high GI, based on the meta-analysis by Barclay et al.¹⁷

The power and variety of glucose lowering drugs available allows us to be free to consume sugar, as long as it does not increase the total calorie intake. There are oral drugs like repaglinide, and drugs administered by injection like insulin analogues, with a very fast initial effect, that allows us to control blood sugar after eating. The limitation is that the patient needs to know the amount of drug to take, depending on the amount of CH that they need to take in, and the calories that will need to be removed from other foods as a result of this intake.

Regarding GI, the total amount of CH consumed is usually the main factor that determines blood sugar response after eating, but the type of CH also affects this. There are several intrinsic and extrinsic variables that influence the effect of CH content in a food on blood sugar response.

The variables include the type of food consumed, the type of CH in the food, the method of cooking, the ripeness or maturity, and how much the food has been processed. In the case of sugar, the intrinsic variables do not provide a great variety of blood sugar responses, because it is a pure food, it only contains sucrose. With respect to processes, not many differences exist either in terms of effect on blood sugar, because the only types are white sugar and refined sugar, with fibre, or brown caramelised sugar.

The extrinsic values are the patient's blood sugar levels before eating, the distribution of macronutrients in the food that they consume, the availability and chemical makeup of the insulin administered, and the level of insulin tolerance. These variables are of special interest in this review because we can modify them so that sugar consumption does not have negative effects on the DM patient. We have already mentioned most of these in this paper (absence of obesity, controlled blood sugar before eating, and coordination with insulin treatments). One important aspect from a practical point of view is reducing sugar's GI when it is consumed with other foods of a low GI or after a meal, mixed with fats, proteins and other CHs. The most common example is sugar in coffee after lunch, which can be consumed almost without any effect on blood sugar after eating, whilst a coffee with sugar consumed mid-morning can have a direct effect on blood sugar.

All other sweeteners with calories such as fructose and polyols (low calorie sugar replacements / sugar alcohols) do not appear to bring any additional advan-

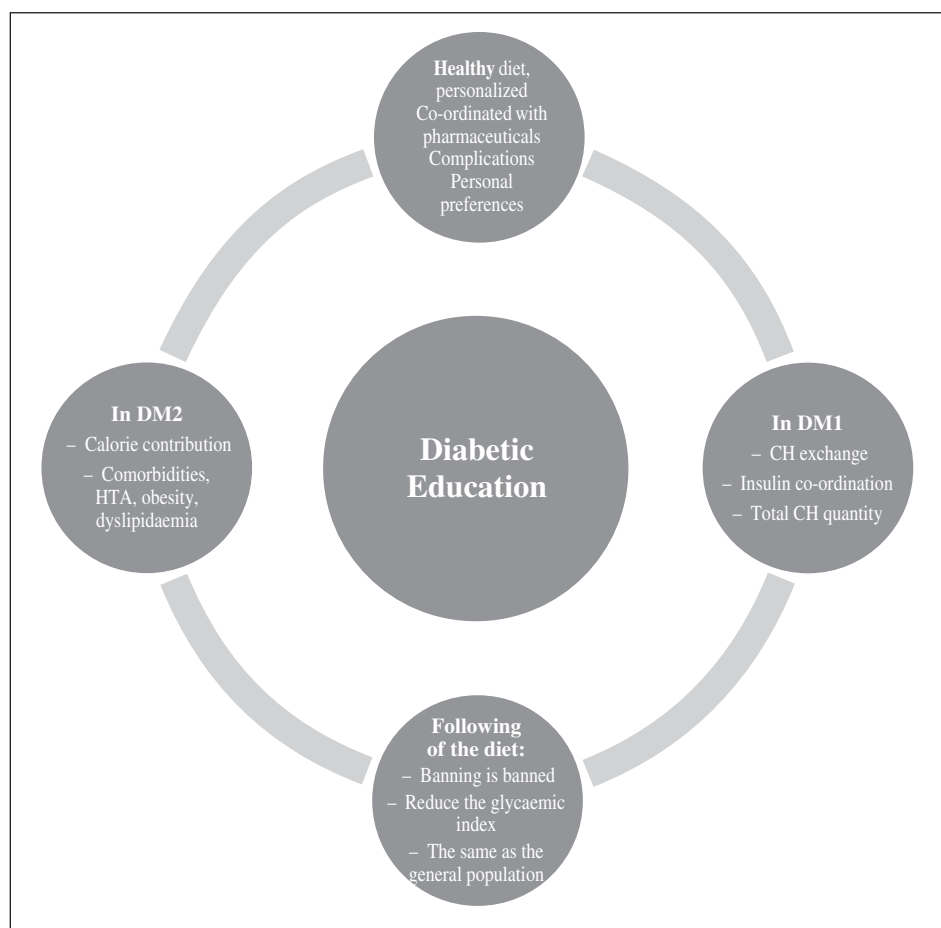


Fig. 2.—Diabetic education.

tages. In the case of fructose it is only advised to be consumed as a component of natural foods but not as a sweetener due to its side effects on lipid plasma. In a meta-analysis by Livesey and Taylor¹⁸ it was observed that the ingestion of more than 250 g a day of fructose produces an increase in the level of plasmatic triglycerides.

Sugar alcohols provide 2 kcal/g, so these could be of interest in low calorie diets, although they may cause diarrhea if too much is taken, especially with children.

Regarding no-calorie sweeteners, the consensus is that they do not damage health, but there is no evidence that they improve control of blood sugars in the long term.

Recommendations

Currently sugar consumption is not prohibited for any kind of DM, but it should be consumed under certain conditions:

- Its value in terms of calories must be considered so it can be substituted for another CH and therefore an increase in total daily calories can be avoided.

- Its consumption must be organised around routine and doses of insulin.
- The DM patient who is going to consume it must gain good control over blood sugar beforehand.
- There must not be any associated obesity.
- In order to reduce GI, it must be consumed after a meal, mixed with fats, proteins and other CHs.
- The patient must be well educated about diabetes so that they can manage and control these parameters (Fig. 2).

Regarding the amount of sugar allowed in a DM patient's diet, most societies neglect to give a specific figure. The only one that does is the European Association, which believes 50 g per day is appropriate. Other societies allow consumption, but do not give a specific amount.

Given the lack of specific recommendations, we can value what has been recommended to the general population. The American Institute of Medicine¹⁹ recommends that total sugar intake does not exceed 25% of total energy, and in that way it ensures that enough essential micronutrients are provided, which are often not present in foods with added sugar. The American Heart Association²⁰ limited the consumption of sugar to <100 kcal/day for women and <150 kcal/day for men.

In 2011 the World Health Organization²¹ limited the intake to <10% of the total energy amount and the United States government through its “Choose My Plate”²², advised against “SoFAS” food (solid fats and added sugar). They especially advised against drinks with added sugar, such as soft drinks, sports drinks or fruit juice and advised the consumption of fresh fruit, fresh fruit juice, water, skimmed milk and coffee or tea without sugar.

Conclusions

The consumption of sugar in a DM patient has been liberalized at the current time. This is due, in part to strong and versatile hypoglycaemic drugs. What's more, the scientific societies have published recommendations based on evidence that dispels some myths.

For its adequate consumption “diabetic education” plays a fundamental role. Patients should know the amount of calories, the ration exchange and how to reduce their GI.

References

1. International Diabetes Federation. Diabetes and impaired glucose tolerance: global burden: prevalence and projections, 2010 and 2030. International Diabetes Federation Website. <http://www.idf.org/diabetesatlas/5e/the-global-burden>. (9 April 2012, date last accessed).
2. Shaw JE, Sicree RA, Zimmet PZ. Global estimates of the prevalence of diabetes for 2010 and 2030. *Diabetes Res Clin Pract* 2010; 87: 4-14.
3. Soriguer F, Goday A, Bosch-Comas A, Bordiú E, Calle-Pascual A, Carmena R, Casamitjana R, Castaño L, Castell C, Catalá M, Delgado E, Franch J, Gaztambide S, Gibés J, Gomis R, Gutiérrez G, López-Alba A, Martínez-Larrad MT, Menéndez E, Mora-Peces I, Ortega E, Pascual-Manich G, Rojo-Martínez G, Serrano-Rios M, Valdés S, Vázquez JA, Vendrell J. Prevalence of diabetes mellitus and impaired glucose regulation in Spain: the Di@bet.es Study. *Diabetología* 2012; 55 (1): 88-93.
4. Nutrition Recommendations and interventions for Diabetes. A position statement of the American Diabetes Association. *Diabetes Care* 2008; 31 (Suppl. 1): S61-S78.
5. Canadian Diabetes Association 2008 Clinical Practice Guidelines for the Prevention and Management of Diabetes in Canada. *Canadian Journal of Diabetes* 2008; 32 (Suppl. 1): S40-S45.
6. Franz MJ, Powers MA, Leontos C, Holzmeister LA, Kulkarni K, Monk A, Wedel N, Gradwell E. The evidence for medical nutrition therapy for type 1 and type 2 diabetes in adults. *J Am Diet Assoc* 2010; 110: 1852-89.
7. Rodbard HW. AACE Diabetes Mellitus Clinical Practice Guidelines Task Force. American Association of Clinical Endocrinologists Medical Guidelines for Clinical Practice for the management of Diabetes Mellitus. *Endocr Pract* 2007; 13 (Suppl. 1): 47-9.
8. Mann JI, I. De Leeuw A, Hermansen K, Karamanos B, Karlström B, Katsilambros N, Riccardi G, Rivellese A, Rizkalla S, Slama G, Toeller M, Uusitupa M, Vessby B. and Diabetes and Nutrition Study Group (DNSG) of the European Association for the Study of Diabetes (EASD). Evidence-based nutritional approaches to the treatment and prevention of diabetes mellitus. *Nutr Metab Cardiovasc Dis* 2004; 14: 373-94.
9. Diabetes UK 2011. Evidence-based nutrition guidelines for the prevention and management of diabetes. www.diabetes.org.uk.
10. Hauner H, Bechthold A, Boeing H, Bronstrup A, Buyken A, Leschilk-Bonnet E, Linseisen J, Schulze M, Strohm D, Wolfram G. Evidence-Based Guideline of the German Nutrition Society: Carbohydrate intake and prevention of nutrition-related diseases. *Ann Nutr Metab* 2012; 60 (Suppl. 1): 1-58.
11. American Diabetes Association. Nutrition Principles and Recommendations in Diabetes. Position Statement. *Diabetes Care*. 2004; 27 (Suppl 1): S36-S46.
12. US Department of Agriculture. US dietary guidelines for Americans 2010. Available from: <http://www.cnpp.usda.gov/dietaryguidelines.htm>
13. Welsh JA, Sharma AJ, Grellinger L, Vos MB. Consumption of added sugars is decreasing in the United States. *Am J Clin Nutr* 2011; 94 (3): 726-34.
14. National Cancer Institute. National Cancer Institute fact sheet: Artificial sweeteners and cancer. <http://www.cancer.gov/cancertopics/factsheet/Risk/artificialsweeteners/> print. Updated 2009. Accessed November 21, 2011.
15. Muñoz-Pareja M, León-Muñoz LM, Guallar-Castillón P, Graciani A, López-García E et al. The Diet of Diabetic Patients in Spain in 2008-2010: Accordance with the Main Dietary Recommendations- A Cross-Sectional Study. *PLoS ONE* 2012; 7 (6): e39454. doi:10.1371/journal.pone.0039454.
16. Mattes RD, Shikany JM, Kaiser KA, and Allison DB: Nutritively sweetened beverage consumption and body weight: a systematic review and meta-analysis of randomized experiments. *Obes Rev* 2011; 12 (5): 346-65.
17. Barclay AW, Petocz P, McMillan-Price J, Flood VM, Prvan T, Mitchell P, Brand-Miller JC: Glycemic index, glycaemic load, and chronic disease risk – a meta analysis of observational studies. *Am J Clin Nutr* 2008; 87: 627-37.
18. Livesey G, Taylor R: Fructose consumption and consequences for glycation, plasma triacylglycerol, and body weight: meta-analyses and meta-regression models of intervention studies. *Am J Clin Nutr* 2008; 88: 1419-37.
19. Institute of Medicine, Food and Nutrition Board. Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein and Amino Acids. Washington, DC: National Academies Press; 2002/2005.
20. Johnson RK, Appel LJ, Brands M, Howard BV, Lefevre M, Lustig RH, Sacks F, Steffen LM, Wylie-Rosett J; American Heart Association Nutrition Committee of the Council on Nutrition, Physical Activity, and Metabolism and the Council on Epidemiology and Prevention. Dietary sugars intake and cardiovascular health. A scientific statement from the American Heart Association. *Circulation* 2009; 120 (11): 1011-20.
21. World Health Organization. Global strategy on diet physical activity and health. http://www.who.int/dietphysicalactivity/strategy/eb11344/strategy_english_web.pdf. Updated 2002. Accessed April 16, 2011.
22. US Department of Agriculture. Inside the pyramid, discretionary calories. What are “added sugars”? <http://www.choosemyplate.gov/foodgroups/empty-calories-sugars.html>. Accessed December 13, 2011.

Obesity and sugar; allies or enemies

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Abstract

In the last three decades, the prevalence of obesity in developed countries has reached epidemic proportions, and continues rising. Many factors have influence on the incidence of obesity, and with the decline of physical activity, overeating plays a role in the emergence of this public health problem. Although a clear relationship between fat intake and weight gain has been established, the role of carbohydrates and more specifically from sucrose and the development of obesity is more controversial. Much of this controversy is due to the growing demand for sweetened drinks and caloric increase posed by these in the diet. Despite multiple studies and communications on this subject in recent years, there are still many areas of uncertainty about the role played by diets rich in sugars over the increase in obesity in last years.

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Key words: *Obesity. Sugar. Sucrose.*

OBESIDAD Y AZUCAR; ALIADOS O ENEMIGOS

Resumen

En las últimas tres décadas, la prevalencia de obesidad en los países desarrollados ha alcanzado dimensiones epidémicas y continua en aumento ¹. Existen múltiples factores que influyen en la incidencia de obesidad, y junto con el descenso de actividad física, el exceso de ingesta, juega un papel preponderante en la aparición de este problema de salud pública ². Aunque existe una clara relación entre la ingesta de grasas y la ganancia de peso, el papel de los carbohidratos y más concretamente el de la sacarosa en el desarrollo de obesidad es más controvertido. Gran parte de esta controversia se debe a la creciente demanda de bebidas azucaradas y al posible incremento calórico en la dieta asociado a su consumo. A pesar de la publicación de múltiples estudios y comunicaciones a este respecto en los últimos años, siguen existiendo numerosas incógnitas acerca del papel que juegan las dietas ricas en azúcares en el incremento de incidencia y prevalencia de obesidad en los últimos años.

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Palabras clave: *Obesidad. Azúcar. Sacarosa.*

Abbreviations

DXA: Dual-energy x-ray absorptiometry.
GI: Glycaemic Index.
BMI: Body Mass Index.
NHANES: National Health & Nutrition Survey.
USDA: US Department of Agriculture.
FDA: Food and Drug Administration.
WHI: Waist Hip Index.

Introduction

Obesity

Obesity is defined as an excess of fat which, generally means a weight increase. However, we can find obesity without excess weight (normal weight obesity) and excess weight without an excess of body fat.

Therefore, for a correct obesity diagnostic it is necessary to quantify the total excess of body fat. The method that is considered as the "gold standard" for evaluating the composition of the body is by dual energy x-ray absorptiometry (DXA), a technique that quantifies total body fat via a calculation of the attenuation of rays of differing intensity, which pass through the patient's body. However, the reduced availability, high cost and exposure to DXA x rays determine the major limitations of its systematic use in regular clinical practices, meaning it is used mainly for investigative studies. In this sense, the body mass index, obtained via the (kg)/size (m²) weight formula is prob-

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ably the most universally accepted indicator for the definition of obesity, given its simplicity and its strong correlation with total adiposity. The body mass index (BMI) is a universal figure, which does not take into account the sex, or different ethnic groups in percentages of body fat. For this reason, obesity in Japan, is defined by $BMI \geq 25 \text{ kg/m}^2$, in China as $\geq 28 \text{ kg/m}^2$, while in Caucasians, as is our case, a BMI of between $25\text{-}30 \text{ kg/m}^2$ is considered as overweight.

Obesity can be classified in function of the differing BMI intervals, but also by the increase in the risk of mortality, by anatomical phenotypes or etiological criteria.

According to the World Health Organization (WHO), obesity is classified as class I for a BMI of between 30 and 34.9 kg/m^2 , class II for a BMI between 35 and 39.9 kg/m^2 , and class III for a $BMI \geq 40 \text{ kg/m}^2$. At the same time, class I obesity is associated with a “moderate risk”, class II with a “high risk” and class III as “very high risk” of mortality. The most common anatomic characterisation refers to the predominant localisation of excess fat both at a visceral level or subcutaneous tissue level. The relationship between waist / hip size known as the WHI (waist hip index), has been used for years to identify those subjects with a central level fat excess, which means, visceral, against peripheral obesity (i.e. subcutaneous). A WHI in men > 1.0 and 0.85 in women, indicates an excess of abdominal fat. In recent years however, the WHI has not been used as it was found to be more cumbersome and the waist measurement has taken its place due to the high correlation with abdominal fat and with the loss of health. As a matter of fact the SEEDO considers the determination of waist index in the obesity classification as it has been demonstrated that, within the same BMI, a risk waist ($> 102 \text{ cm}$ in men and $> 88 \text{ cm}$ in women) the morbidity and mortality associated with that BMI increases. It is known that visceral adiposity is correlated with a higher probability of developing the metabolic and cardiovascular complications classically associated with obesity, while subcutaneous fat, seems to be much more benign, and in some cases it even acts as protection against the development of metabolic complications.

From an etiological viewpoint, obesity can be classified as either primary or secondary. Obesity can in fact be iatrogenic, which is to say, secondary to pharmacological treatments, including some anti-psychotic, antidepressant, antiepileptic drugs and some steroids. Certain obesity phenotypes are characteristics of some pathologies, including polycystic ovary syndrome, Cushing syndrome, hypothyroidism, hypothalamus defects and growth hormone deficiency.

Alternatively, as a primary disorder, the etiology is quite complex to analyse. Even though its pathogenesis can be expressed in relatively simple thermodynamic terms, which is to say, the excessive storage of body fat as a result of a chronic positive energy balance (i.e.

excess of ingestion compared to use), the identification of the main causes of chronic energy imbalance continues to be a challenge as well as the fact that metabolic phenotypes, psychological and behaviour patterns that lead to obesity continue to be controversial. As a matter of fact, the excessive consumption of energy (or hyperphagia) is considered a characteristic highly prevalent in obese subjects. However, the connection of hyperphagia to the increase of real weight has turned out to be exceptionally difficult to document, very probably due to the fact that the measurement of energy consumption in the individuals already accounts for a real challenge, especially in obese people that tend to underestimate their food intake. Other aspects of food intake and their relationship with obesity, such as the diet make-up, the food's energy density, and the food intake figure, flavour preferences, life style and possible sub-phenotypes, have also been explored with somewhat contradictory results.

It is not surprising, therefore, that obesity related molecular biology is only partially understood. This is probably due to the heterogeneity of obesity and the fact that this is related to, the same as other complex illnesses, not by one single genetic mutation but by multiple allelic defects, which determine a higher susceptibility to environmental factors.

Despite obesity affecting a large proportion of the world's population, the incidence and prevalence estimates are not available for all countries, and the data available is not exact. In the United States the National Health and Nutrition Examination Survey (NHANES) in the last 50 years has provided data on the continual monitoring of the prevalence and incidence of obesity in a representative sample on a national level. This data shows the prevalence of obesity amongst adults (age ≥ 20 years), which started to increase notably after 1980. In 2007-08 (the set of most recent data available) obesity reached prevalence by age of 33.8% in total, corresponding to 32.2% in men and 35.5% in women. The highest prevalence is currently observed in black non-Hispanic race, followed by Hispanics and non-white Hispanics. Addressing the severity of obesity, grade 2 obesity has a global prevalence of 14.3% , while grade 3 reaches 5.7% . The racial distribution continues the same pattern as general obesity, except for grade 3, which is as frequent in Hispanics (5.5%) as amongst non white Hispanics (5.2%) (Fig. 1).

Amongst children and teenagers (age < 20 years), according to NHANES data of 2007-08, obesity reached a prevalence of 9.5% in girls and boys 0-2 years of age, while the prevalence for children from 2-19 years of age was 16.9% in both sexes. The prevalence of obesity in children by race / ethnicity is highest amongst Hispanics (12.5% for 0-2 years of age and 20.9% for 2-19 years of age), followed by black non Hispanics (10.3% for 0-2 years of age and 20.0% for 2-19 years of age), and white non Hispanic (8.7% of 0-2 years of age and 15.3% for 2-19 years of age)⁴.

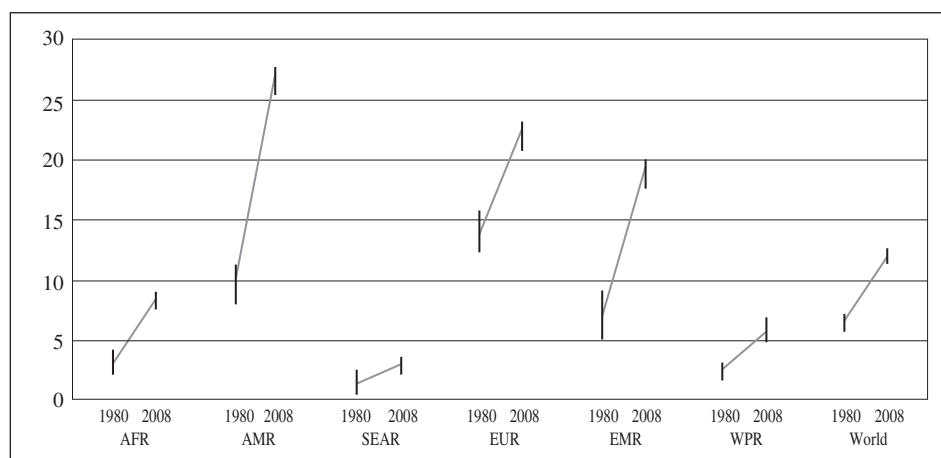


Fig. 1.—Prevalence of obesity data in the United States of America.

In the last 25 years obesity has also increased in Spain from 7% to 17% (Fig. 2). In Spain two of three men are overweight and one in six is obese.

The latest statistics show that this condition affects 10-27% of men and 38% of women in Europe. It is calculated that more than 200 million adults throughout the European Union could be overweight or are obese. More than half the population of Spain (over 18 years of age) is above what is considered a normal weight. 45.5% of men and 29.9% of women are overweight, while 17.3% of men and 14.7% of women are obese. 52.1% of males and 43.6% of females from 65 to 74 years of age are overweight while 23.9% of men and 27.4% of women suffer from obesity.

As for the juvenile population, the percentage of childhood obesity has increased by 35% in the last decade in our country.

According to the latest figures, 21.8% of Spanish children are overweight and an 8.2% are obese, and

nearly one in three children between the ages of 3 and 12 years are overweight.

Spain is the second country in the European Union, after Malta, with the highest percentage of obese or overweight children between 7 and 11 years old. Obesity has become an epidemic that every year affects 400,000 more children in Europe. 9% of Spanish school children suffer from obesity and 33% are overweight, compared to lower figures of 20% in France, Poland, Germany or Holland (Fig. 3).

As a consequence of the relationship between the excess of body fat and higher morbidity and mortality, and the rising epidemic of obesity, a high percentage of the population on a world level shows an increased risk of contracting certain illnesses and a higher risk of death by any cause⁵. In developed countries the increase of mortality associated with excessive body fat is 30-40% for coronary cardiac diseases, colon, breast and endome-

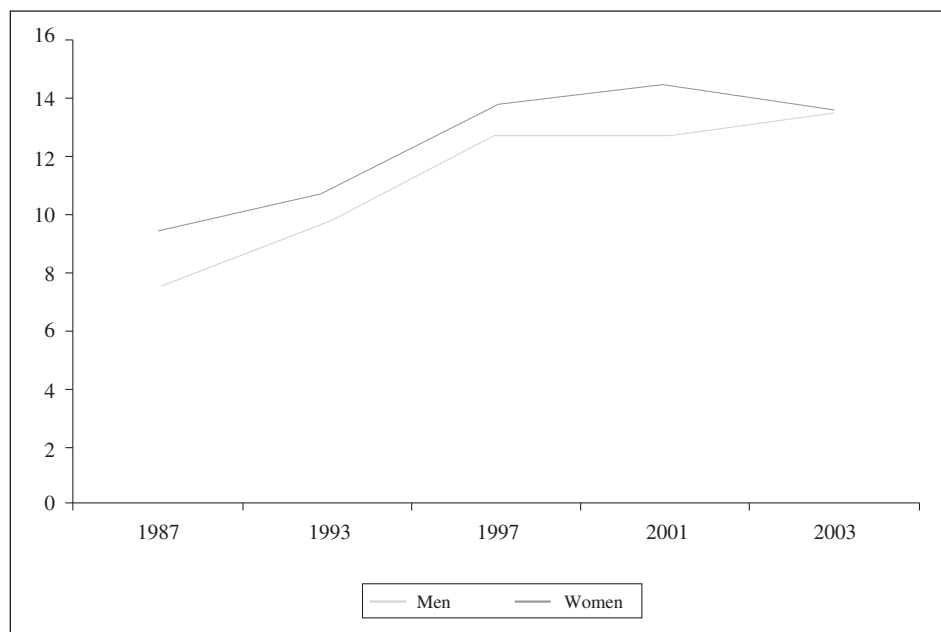


Fig. 2.—Prevalence of obesity in Spain from 1998 to 2003.

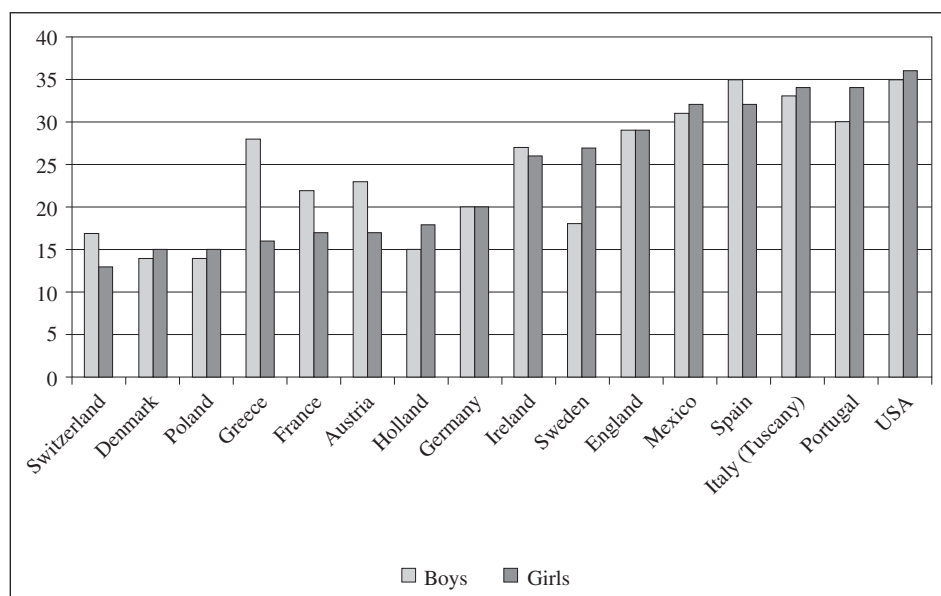


Fig. 3. —Prevalence of child obesity in European Union countries.

trial cancer, and in the majority of type 2 diabetes cases.

Even though the risks of morbidity and mortality are higher in obese people, the relationship with BMI, in terms of the range of obesity is not necessarily uniform for the illness (for example, cancer) subtypes of the genre, or race / ethnicity. In some cases, the data does not support a higher risk in obese people. For hip fractures, the observed risk was lower for body mass indexes in the region of 30 kg/m². In a similar fashion, the results of morbidity and mortality for obese patients with chronic renal illnesses during haemodialysis treatment, with cardiac insufficiency or peripheral arterial illnesses, seem to be better in obese patients than in those patients with a normal weight and especially in elderly patients over 75 years old. These observations have generated what is known as the “obesity survival paradox” According to some authors this paradox is explained by the fact that patients lose weight as the underlying illness progresses. Alternatively, this paradox has recently been proposed as a plausible explanation for two well established epidemiological observations: the shape of U (concave) of the relationship between BMI and the rate of mortality, as a result that people with an intermediate BMI (25-30 kg/m²) tend to live longer than people with a lower or higher BMI and the nadir of these curves tend to increase with age.

Sugar

With the term “sugars” we are referring to a group of compounds constituted by carbon, hydrogen and oxygen atoms that are classified in function of their chemical structure in monosaccharides, disaccharides and oligosaccharides. The monosaccharides contain

3- 7 carbon atoms per monomer, which can be absorbed by sugars. Glucose, fructose and galactose are the essential monosaccharides for the human diet, while mannose carries out a less important role. Disaccharides are compounds formed by two conjoined monosaccharides (2 monomers). Primary disaccharides in the human diet are sucrose (one glucose molecule and one fructose molecule), lactose (galactose and glucose) and trehalose (2 glucose molecules). Human beings possess enzymes which breakdown the disaccharide’s links for their later absorption and metabolism in the form of monosaccharides.

Sugars provide energy and a pleasant taste; this way, the ingestion of sugars seems to be influenced by two different brain systems: those associated with the regulation of food and the energy homeostasis, and those which are associated with the reward. During the last three decades, the existence of a series of neuromodulators which can act both as an orexigene and anorexigene involved both in the regulation of energy and in the circuits that measure their reward has been widely accepted.

In terms of sugar consumption, in Spain estimations rise up to 29 kg per capita annually. This consumption can be considered relatively high, if we compare with the world consumption per capita of 21 kg, with the geographical regions of Canary Islands, Galicia and Castilla y Leon showing a higher consumption. In global terms, Spain has a consumption of about 1,300,000 tonnes of sugar per year.

Obesity and sugar

The prevalence overweight and obesity has increased considerably throughout the world in the last three decades, and, although the genetic factors seem to

play an important role in the development of obesity, the dramatic increase in the incidences of obesity seems to suggest that the environmental factors and lifestyle changes could be contributing in an important way to the epidemic tendency of this condition. Both the reduction of physical activity and the increase in consumption of high calorie foods are factors that have been directly related to the development of excess weight and obesity. Currently, the role of sugar intake, and more precisely of sucrose, in the development of obesity is raising quite a lot of interest in the world scientific community.

Unlike what happens with the ingestion of fats, for which a high correlation between the excessive consumption of them and the increase in developing excess weight and/or obesity has been proved. The relationship between the consumption of sugars included in food or added to them and the increase in weight is not so evident. Different cross-sectional studies have concluded that there is no association or even a negative association between the consumption of sugars and weight gain. However, there does exist an ample debate regarding the possibility of the higher consumption of sugar through sugary drinks producing a more significant effect on BMI. In this regard, some authors support the hypothesis that drinks do not provide the same level of satiety as solid food and therefore, consumers do not adjust correctly their total intake to compensate for the extra energy consumed through sugary drinks.

DAFO analysis regarding the relationship between obesity and sugar

Weaknesses

- Human beings present an innate preference towards sugar, due to the fact that when in the uterus, the foetus is bathed in a “sweet liquid” and, later, from birth, children feed from milk, either maternal or artificial, which contains a high sugar content, so it is not surprising to find that in adult life there is a predominant preference for sugary foods⁵.
- Given that sugar is added frequently to food, the interpretations and discussions that surround the intake of sugar are complex due in great part to the large number of terms used to describe these ingredients, the lack of comparable data for the consumption of sugar in the diet, the lack of real data in the food composition databases and the almost exclusive provision of epidemiological studies as the principal knowledge base, with the limitations that this carries.
- As a matter of fact, when it comes to establishing a relationship between obesity and the consumption of sugar, one of the obstacles that we face is the difficulty to quantify precisely the quantity of sugar consumed by the population for various motives⁵:

- Even though an agreement exists regarding the chemical definitions of the different sugars, the terms sugar, sugars, added sugar, and caloric sweeteners are used without distinction in many sugar consumption studies, something which generates a great deal of confusion when it comes to establishing consumption statistics.
- When it comes to quantifying the daily sugar consumption, individuals tend to take into account only added sugar, such as sucrose (white and brown sugar), saccharine, aspartame and other substitutes, excluding added sugar as an ingredient in processed or pre-prepared foods.
- Individuals tend to selectively reflect a lower intake of foods rich in fat, carbohydrates and sugar.

Threats

- The composition of the denominated “low fat” food reduces the fat content at the price of increasing principally the sugar content. Despite the popularity and increase in consumption of this type of food, the incidence of obesity keeps rising, which would explain in part the increase in obesity.
- The excess of carbohydrates in the diet with respect to the caloric requirements of each individual, not through the conversion of surplus carbohydrates into fat but through lipogenesis, again, can produce an accumulation of body fat. There is a higher priority in the oxidation of carbohydrates against fat, but in the long term, this can result in a suppression of the oxidation of fat with the subsequent bodily deposits maintenance.
- Foods rich in high GI carbohydrates produce abrupt peaks in hypoglycaemia followed by the proportionate liberation of insulin, a situation which promotes the postprandial oxidation of the carbohydrate at the expense of the fat oxidation, inhibiting the lipolysis with the consequent reduction of the quantity of free chain fatty acids and the fat oxidation, all of which leads to an increase of body fat. Even though sucrose has a medium GI, some foods that contain it, such as breakfast cereals, sweets or pastries, tend to have an elevated GI and glycaemic load.
- The combination of frequent consumption of sugary drinks and the decrease in physical activity, is converted into a lowering of the metabolic demand for fat as an energy source, considerably increasing the risk of weight gain, especially among the younger population.
- When we compare diets with a high sucrose content with diets with a high polysaccharide content, an increase in total ingested calories of sucrose against polysaccharides in the region of 12% has been observed, which could explain the increase in consumption of sucrose in the form of sugary drinks.

- In studies that have compared the caloric ingestion and the increase in weight amongst subjects that consumed drinks sweetened with sucrose compared to others that drank drinks with artificial sweeteners, an increase of both calories consumed and the weight of those that drank drinks with sucrose, against those that drank drinks with artificial sweeteners⁶.

Strengths

- Sugar, apart from the sweet taste, brings a wide variety of favourable qualities to food, such as their antimicrobial action, taste, aroma and texture, as well as viscosity and consistency, which are generated endlessly. Even though the feeling of being full is higher for proteins than for sugars, and carbohydrates is higher than that of fats, which at the same time is the major source of calories in the diet⁷.

- The consumption of high fat diets produces a lowering in the intake of sugars and vice versa, a phenomenon known as the fat-sugar balance.

- In a cross-sectional study carried out on the population of New Zealand, it was observed that adults, who were overweight or obese, did not show a higher intake of sugar than people of a normal weight. Furthermore, obese children consumed less sugar than both children of a normal weight and overweight children.

- The intake of both sugars (both contained in foods and added) is not associated with the BMI.

- Studies regarding thermogenesis induced by food have shown that energy dissipated in the form of heat is lower after the intake of fat ($\approx 7\%$), than after the intake of carbohydrates ($\approx 12\%$) and proteins ($\approx 22\%$). Furthermore, isotopic studies have confirmed the significant absence of hepatic lipogenesis again in diets with high carbohydrate content.

Opportunities

- The US Department of Agriculture (USDA) and the FDA which establish the regulation of foodstuff and food ingredients, describe the different terms to name the different types of sugar (Table I). This proposal will allow in the future a higher rate of precision when quantifying the consumption of different types of sugars, in future research projects.

- The glycaemic index (GI) is a foodstuff classification based on the postprandial response of blood glucose, compared with a foodstuff with a (glycaemic index = 100) reference. Various studies have concluded that carbohydrates, with a high GI, increase satiety in the short term compared to carbohydrates with a lower GI. Sucrose shows an average GI of (≈ 65), which generates a higher level of satiety than other sugars such as fructose with a lower GI.

- In the CARMEN study (Carbohydrate Ratio Management in European National diets) the effect on bodyweight and lipid profile of isocaloric diets with a high sugar and polysaccharide content against diets with a high fat content were compared for a period of 6 months. The results showed that diets with a high content of both sugars and polysaccharides accompanied by a significant drop in body weight from between 1.6 kg and 2.4 kg respectively in comparison to isocaloric diets with a higher fat content level.⁸

- In an important number of epidemiological studies, an inverse relationship between the intake of sucrose and body weight or body mass index, as well as the intake of sucrose and total fat intake has been observed.

- There is little evidence to prove that the different sugars or carbohydrates have a negative effect on the control of body weight.

- In studies carried out in different countries such as Australia and the United Kingdom, despite the increase

Table I
Terms and definitions of sugars according to the USDA and the FDA

<i>Terms</i>	<i>Definition</i>
<i>Added sugar</i>	Consumed separately or used as ingredients in processed or pre-prepared foods (such as white sugar, brown sugar, raw sugar, corn syrup, solid corn syrup with high fructose corn syrup, malt syrup, maple syrup, syrup, fructose sweetener, liquid fructose, honey, molasses, anhydrous dextrose, dextrose and crystal. Can contain oligosaccharides.
<i>Sugars</i>	All monosaccharides and disaccharides (including natural sugars and sugars added to food or drink, such as sucrose, fructose, maltose, lactose, honey, syrup, corn syrup, syrup with a high content of corn fructose, molasses and concentrates fruit juice.) Oligosaccharides are not taken into account.
<i>Sugar</i>	Indicates sucrose on the list of declared ingredients.
<i>Caloric sweeteners</i>	Sweeteners consumed directly and as foodstuff ingredients, such as sucrose (refined sugar cane and beet) honey, dextrose, edible syrups and corn sweeteners (mainly corn syrup with a high fructose content). Contains oligosaccharides.

in the consumption of caloric drinks, with artificial sweeteners, in detriment to sugary drinks, either with sucrose, fructose or syrup with high fructose content, a lowering of the incidence or prevalence of obesity has not been observed. This phenomenon is known as the “Australian paradox” and suggests that there is no association between the consumption of sugar and the appearance of obesity in the population.⁹

- Glucose is stored in the liver and muscles in the form of glycogen. Excess glucose that is not used as an immediate source of energy or for the synthesis of glycogen can be converted through lipogenesis into fat again which is stored in the adipocytes. However, this is a costly conversion in terms of energy. Astrup and Raben calculated that 68% more energy is needed (155 in comparison to 42 MJ/kg) in order to increase body fat by 1kg through carbohydrates, and therefore “it is difficult to increase fat mass in subjects of a normal body weight, particularly through carbohydrate overeating”¹⁰.

Conclusions

- Current data suggests that, against what occurs with other macronutrients, in the case of carbohydrates, and more precisely, sugars, there is a lack of preciseness in the measurement, intake and availability. Due to this lack of clarity, the relative arguments over the health effects of sugar should be analysed carefully and always backed up by scientific evidence.

- There is little evidence to show that the different sugars or carbohydrates have negative effects on the control of body weight.

- The consumption of sugary drinks by itself does not seem to be related to the increase of the incidence of obesity in the population of the developed countries.

- In the short term, for the majority of individuals, the excess energy that comes from sugary drinks can be compensated by a reduction in the intake of successive meals. In the long term, the changes in body weight implicate physiological adaptations such as the sensation of hunger and of the metabolic rate, which tends to restore weight. Therefore, there is no clear evidence to show that the consumption of sugary drinks increases *per se* the caloric intake and causes obesity.

- Alternatively, the combination of the frequent consumption of sugary drinks together with a decrease

in physical activity brings an increase in the risk of weight gain.

- Even though the quantity of calories provided by the different macronutrients is not the same (carbohydrates 4 kcal/g; proteins 4 kcal/g and fats 9 kcal/g), from an energy stand point, the number of calories ingested by an individual is independent of where it came from, and it is fundamental in the maintenance of an adequate state of good health, to maintain a balance between the different macronutrients, as changing the distribution of these macronutrients that we all accept as healthy carries a risk of not maintaining a normal body weight.

References

1. Egger G, Swinburn B. An “ecological” approach to the obesity pandemic. *British Medical Journal* 1997; 315 (7106): 477-80.
2. Winsome Parnell, Noela Wilson, Donnell Alexander, Mark Wohlers, Micalla Williden, Joel Mann and Andrew Gray. Exploring the relationship between sugars and obesity. *Public Health Nutrition*/Volume 11/Issue 08/August 2008, p 860-866 DOI: 10.1017/S1368980007000948, Published online: 21 September 2007
3. WHO. Obesity: preventing and managing the global epidemic. 2000. Geneva, World Health Organization.
4. Flegal KM, Carroll MD, Ogden CL, Curtin LR. Prevalence and trends in obesity among US adults, 1999-2008. *JAMA* 2010; 303 (3): 235-41.
5. Katherine M, Brian K, Heather O, Barry I. Association of All-Cause Mortality With Overweight and Obesity Using Standard Body Mass Index Categories A Systematic Review and Meta-analysis. *JAMA* 2013; 309 (1): 71-82.
6. Raben A, Vasilaras TH, Moller AC, Astrup A. Sucrose compared with artificial sweeteners: different effects on ad libitum food intake and body weight after 10 wk of supplementation in overweight subjects. *American Journal of Clinical Nutrition* 2002; 76 (4): 721.
7. Sigman-Grant M Morita J. Defining and interpreting intakes of sugars. *Am J Clin Nutr* 2003; 78 (Suppl.): 815S-26S.
8. Saris WHM, Astrup A, Prentice AM et al. Randomized controlled trial of changes in dietary carbohydrate/fat ratio and simple vs complex carbohydrates on body weight and blood lipids. *Int J Obes Relat Metab Disord* 2000; 24: 1310-8.
9. Barclay AW, Brand-Miller J. The Australian Paradox: A Substantial Decline in Sugars Intake over the Same Timeframe that Overweight and Obesity Have Increased. *Nutrients* 2011; 3: 491-504.
10. Saris WHM Sugars, energy metabolism, and body weight control. *Am J Clin Nutr* 2003; 78 (Suppl.): 850S-7S.
11. EFSA Panel on Dietetic Products, Nutrition, and Allergies (NDA); Scientific Opinion on Dietary Reference Values for carbohydrates and dietary fibre. *EFSA Journal* 2010; 8 (3): 1462 [77 pp.]. doi:10.2903/j.efsa.2010.1462. Available online: www.efsa.europa.eu 1 © European Food Safety Authority, 2010.

Sugar and cardiovascular diseases

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Abstract

Cardiovascular diseases are the leading cause of death in the Spanish population and may be a relationship between the prevalence of these and excessive sugar consumption. In recent years, researchers have focused on the properties of these nutrients. Although there are many studies examining this association, the results are not unanimous. In any case there is sufficient basis for designing public health strategies in order to reduce the consumption of sugary drinks as part of a healthy lifestyle.

Therefore, the question we address is: sugar intake in abundant amounts, is associated with a higher risk of cardiovascular disease? We use as the focus of the discussion SAFO analysis model.

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Key words: Cardiovascular diseases. Sugar consumption. Sugary drinks. Healthy lifestyle.

AZÚCAR Y ENFERMEDADES CARDIOVASCULARES

Resumen

Las enfermedades cardiovasculares constituyen la principal causa de muerte en la población española y podría existir una relación entre la prevalencia de las mismas y el consumo excesivo de azúcar. En estos últimos años, los investigadores se han centrado en las propiedades de estos nutrientes. Aunque existen muchos estudios que analizan dicha asociación, los resultados no son unánimes. En cualquier caso, existe fundamento suficiente para diseñar estrategias de salud pública de cara a reducir el consumo de bebidas azucaradas, como parte de un estilo de vida saludable.

Por lo tanto, la cuestión que abordamos es: ¿la ingesta de azúcar, en cuantía abundante, se asocia un mayor riesgo de padecer enfermedad cardiovascular? Para ello utilizamos como eje de la discusión el modelo de análisis DAFO.

Nutr Hosp 2013; 28 (Supl. 4):88-94

Palabras clave: Enfermedades cardiovasculares. Consumo de azúcar. Bebidas azucaradas. Estilo de vida saludable.

Abbreviations

AMP: Adenosine monophosphate.

HBP: High blood pressure.

BMI: Body mass index.

MAPA: Arterial blood pressure monitoring.

WHO: World Health Organisation.

Introduction

The term sugar or sugars tends to be used to designate the different monosaccharides and or disaccharides which are characterised by having a sweet taste, but by extension its is used to make reference to practically all carbohydrates. 70% of the world's sugar is produced from sugar cane and the rest from beet. We know of the existence of sugar cane thanks to one of Alexander the Great's Admirals (356-323 b.c) named Nearco who mentions it in an expedition to India. From Roman times there are references towards elaborated sugar; for example, in the chronicles of the storming of the King of Persia's Palace by Emperor Flavio Heraclio Augusto's (575-641a.d.) troops in 627 a.d. sugar was part of the bounty, along with silks and varied spices. Nowadays, sugar is consumed by the majority of the population and is

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widely appreciated for its ability to make food more appetising¹.

During recent decades, the physiological properties of carbohydrates and sugars have not drawn excessive interest from the scientific community, who concentrated more on saturated fats, which until recently dominated the nutritional panorama. In recent years, however, researchers have concentrated on the properties of sugar, and in particular in fructose. This monosaccharide forms part (along with glucose) of sucrose, a disaccharide more commonly known as “table sugar”. Sucrose (fructose and glucose) is added to a large number of manufactured foods (yogurt, cereals, sauces, cakes, biscuits) and drinks (tea, coffee, soft drinks)².

In this chapter we analyse the relationship between sugar (glucose and fructose) and cardiovascular disease, to answer the following question: is the consumption of sugar, in large quantities, associated with a higher risk of suffering from a cardiovascular disease?

Current state of the problem

Cardiovascular disease is the main cause of death in the Spanish population. In our country the prevalence of the main cardiovascular risk factors is high^{3,4}. In the ERICE5 study (the aggregation of eight cross-sectional epidemiological studies, carried out in Spain between 1992 and 2001) the most common cardiovascular risk factors were high cholesterol (46,7%), high blood pressure (37,6%), tobacco consumption (32,2%), obesity (22,8%) y diabetes mellitus (6,2%). This prevalence of diabetes mellitus is in contrast to that obtained by the MAPA Group in the city of Madrid (10,6%)⁴ and with the most recent figure by Soriguer et al⁶ in Spain (13,8%; CI 95%, 12,8% a 14,7%), of which half did not know they suffered from diabetes (6,0% [CI 95%, 5,4% a 6,7%]).

The inhabitants of developed countries now consume more calories than in yesteryear. Obesity, defined as an excess of body fat, is the result of a positive energy balance, and is the most frequent form of malnutrition. The calorie consumption has increased on average from 150 to 300 calories per day. Almost 50% of this increase comes from liquid calories, in particular sugary drinks, manufactured in the main with fructose. The consumption of fructose is considered to contribute to the higher obesity rate in industrialised countries, as there is a temporary relationship, parallel and direct between their consumption and the increase in obesity. In some sections of the population of the United States, the intake of these drinks accounts for 15% of the daily-recommended calorie intake.

Alternatively, sugar is an essential ingredient in our diet. It provides a rapid and important energy source. It is part of oral hydration, so relevant for people who

practice sport. It can help the sick and elderly who have a lack of appetite to consume nutritionally desirable foods. Sugar and salt are great natural preservatives, which, during centuries and until the discovery of industrial cold preservation (fridges) were the only preservatives available¹. Furthermore, a very important aspect, related with the festive or emotional facet is the pleasure that it brings. We cannot envisage a party without a cake, a Christmas without nougat (*Turrones*) or a wedding without a cake.

However, it seems to be necessary to reflect upon the epidemic proportions that obesity is reaching, with the objective of establishing the most adequate therapeutic strategies and in this context, analyse the role of certain foods, as is the case of sugary drinks, as well as the evidence available to enable us to offer messages to the general public regarding moderation and restriction of their consumption.

So why is the intake of sugar, above the recommended quantities, (OMS: 10% of the total calories in the diet in the form of sugars), associated with a higher risk of suffering from a cardiovascular disease? This is the subject analysed in this chapter (Table I).

Strengths

In the last 25 years excess weight and obesity has increased notably, in relation to a positive energy balance, which is related to a clear drop in physical activity (especially notable in Spain) and with a less balanced diet with a lowering of carbohydrates and increase in fat consumed, with a total calorie intake lower to that of previous years.

The very high consumption of fructose, higher than current consumption levels, has been associated with an increase in the levels of triglycerides, visceral fat, blood pressure, resistance to the hypoglycaemic action of insulin and a drop in the levels of HDL-cholesterol. These variations taken alone or as a group have been associated with the increase in risk of suffering from some type of cardiovascular disease⁷. A large part of these effects of fructose is due to the fact that nearly 50% of the quantity absorbed is converted into fatty acids, as opposed to other carbohydrates, such as starch, which only 5% is converted into fatty acids.

Its excessive consumption has also been associated with high levels of urate serum. Fructose is converted in the liver into fructose-1 phosphate. The phosphorylation of the fructose also carries an increase of the synthesis of AMP, part of which can enter the degradation path of the purine nucleotides, which culminate in the synthesis of uric acid (Fig. 1). Diverse epidemiological studies have shown a significant relationship between uraemia and the appearance of a cardiovascular disease, high blood pressure (HBP), diabetes and a resistance to the hypoglycaemic action of insulin. The rise of uric acid levels in blood has been associated

Table I
*SWOT Analysis (Strengths, Weaknesses, Opportunities and Threats) overall application:
the intake of sugar, in large quantities, is associated with a higher risk of suffering from cardiovascular disease*

<i>Strengths</i>	<i>Weaknesses</i>
<p>Inherent elements (internal) in the binomial sugar and cardiovascular disease that strengthen this relationship:</p> <ul style="list-style-type: none"> • Possible deleterious effects of sugar: <ul style="list-style-type: none"> – Obesity³. – Reduction of HDL⁹. – Hyperuricemia¹¹. • Increase in birth weight³⁸. 	<p>Inherent elements in the binomial sugar and cardiovascular disease that weaken this relationship:</p> <ul style="list-style-type: none"> • CARMEN Study²². • The reduction of fat and slight increase in carbohydrates associated with weight loss²⁴. • Australian paradox²⁵.
<i>Threats</i>	<i>Opportunities</i>
<p>External elements for binomial sugar and cardiovascular disease that “threaten” this relationship:</p> <ul style="list-style-type: none"> • The genetic influence in the development of obesity²⁶. • The excessive consumption of fats associated with obesity²⁷. • Role of intestinal microbiota in obesity³⁰. • Being sedentary also influences obesity³¹. 	<p>External elements for binomial sugar and cardiovascular disease that offer the possibility to demonstrate a direct scientific relationship:</p> <ul style="list-style-type: none"> • Promoting education of healthy dietary habits (with the family, at school, healthcare professionals)²⁴.

independently, with the cardiovascular related morbidity, even though not all the epidemiological studies offer concurring results. In patients with HBP and diabetes this association is more intense than in the general population. It is estimated that in comparison with individuals with normal serum urate levels, patients with hyperuricemia have a medium risk of suffering ischemic cardio pathology or essential HBP 10 times higher. This pathological association can be explained by the endothelial dysfunction. The hyperuricemia cannot only be the cause of the endothelial damage, but a consequence of it. The hyperuricemia can be explained by an excessive production of urate caused by a higher influx of AMP (Fig. 1). But also, the increase in serum urate can be a consequence of a drop in its renal secretion related to the resistance to the hypoglycaemic effect of the insulin and/or the deterioration of renal function (nephroangiosclerosis). For some authors the sex of the subject can influence in the relationship between hyperuricemia and HBP, with a significant association, more intense in men than in women.

It is important to point out that a lot of literature regarding the consumption of high calorie drinks originating in North America, without the possibility of differentiating the results obtained relative to drinks sweetened with sugar to those that contain other sweeteners (such as high fructose corn syrup).

Recently three studies have been published that show the influence of sugary drinks (rich in fructose) on the development of obesity in children, teenagers and adults⁸⁻¹⁰ (Table II). These three studies formulate a common conclusion: excessive ingestion calories are possibly responsible.

Some studies have related the consumption of sugary drinks during pregnancy with the increased

weight of the baby when born. The temporary parallel tendency estimates that based on commercial sales figures, between the consumption of sucrose, in the country where the study took place (Norway), and the higher percentage of overweight babies, supports this hypothesis¹¹.

When examining the complete scientific evidence, which exists today, it does not allow us to demonstrate convincingly that the effects attributed to sugary drinks are exclusively due to those sugary drinks but can be associated with any high calorie drink¹².

Weaknesses

The claim that the intake of sugar, in large quantities and in all cases way above the current level of consumption, is associated with a higher risk of suffering a cardiovascular disease shows weaknesses; not all studies support this claim. This can be seen in the CARMEN¹³ report where fat was substituted by carbohydrates (simple or complex) and no detrimental effects on the lipid profile were shown. This study highlighted the importance of a low fat high carbohydrate diet to control obesity and its associated health problems.

Other studies are in line with the previous idea. A low fat diet with a higher carbohydrate content (of which some are sugar), in overweight subjects and with metabolic syndrome, can offer a slight loss in weight.

The large majority of epidemiological research, which has shown a positive relationship between the consumption of sugar and the prevalence of obesity, has been carried out in Europe and the United States. However, in other places it has not been possible to establish this relationship so clearly (i.e. – the

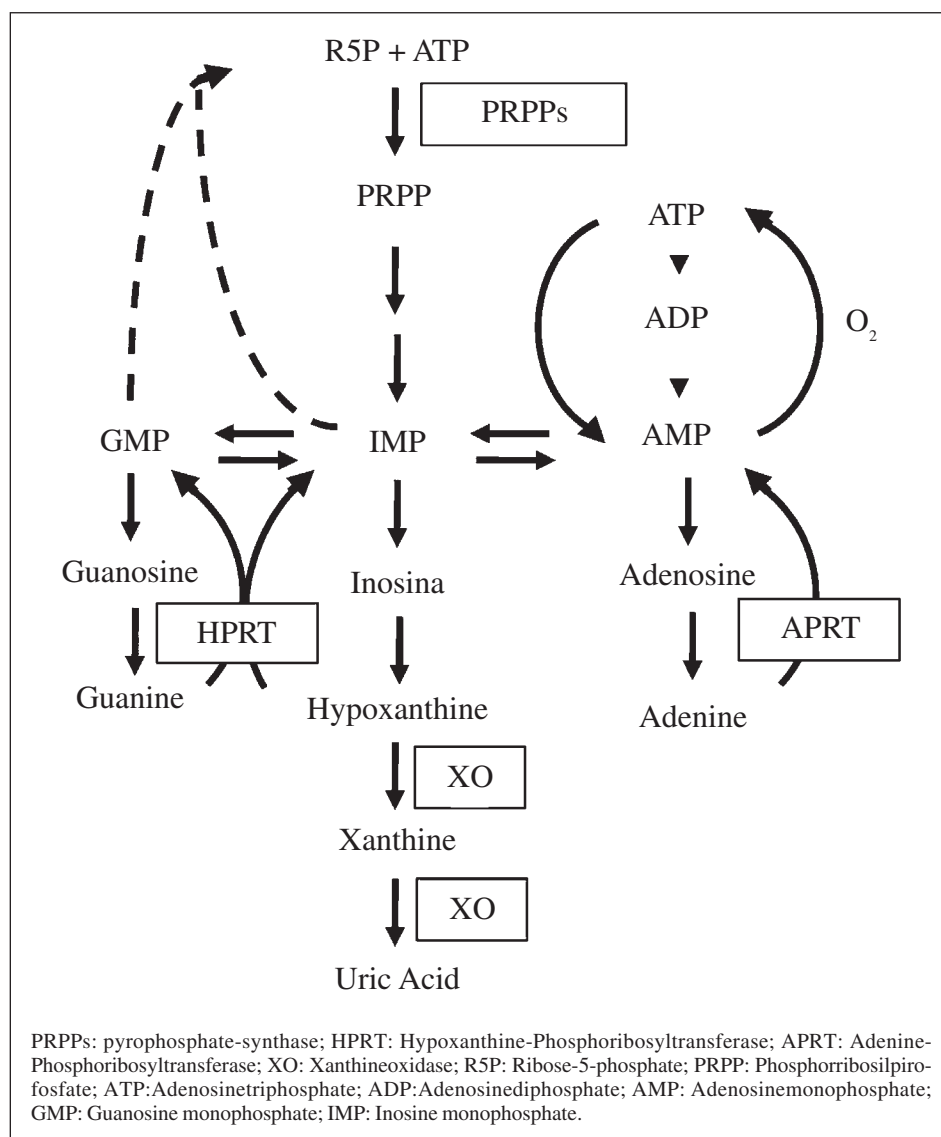


Fig. 1. —Synthesis and degradation of purine nucleotides. Fructose is phosphorylated to fructose-1 phosphate in the liver with the contest of ATP. The consumption of AMP that can enter into the degradation path of the purine nucleotides. This degradation path culminates in the synthesis of uric acid. In the presence of oxygen (O₂) the AMP is phosphorylated into ATP. In hypoxia situations the majority of the AMP enters into the degenerative path of the purine nucleotides.

Table II

Characteristics of three published studies¹³⁻¹⁵ regarding the relationship between the consumption of sugary drinks and obesity in different sections of the population. The first study shows a significant interaction between an important factor of our diet (sugary drinks) and the genetic predisposition towards obesity. In the following two studies, carried out on different age groups of the population, evidence was shown that the reduction of sugary drinks and diet education favoured the loss of weight

Study	Population objective	Design	Results
QJ et al. <i>N Engl J Med</i> 2012; 367: 1387-96	Adults (≥ 18 yrs) Interaction of sugary drinks with binomial genetic obesity	Cohort Prospective	Genetic-obesity relation modified due to consumption of sugary drinks
Janne C et al. <i>N Engl J Med</i> 2012; 367: 1397-406	from 4 to 11 years Replacement masked as sugar and weight gain	Cohort Prospective Double blind	Replacement of SD or non sugar reduces weight gain
Ebbeling et al. <i>N Engl J Med</i> 2012; 367: 1407-16	From 14 to 16 years Lifestyle-obesity relationship	Cohort Prospective	Lifestyle intervention reduction of body weight

SD: Sugary drinks.
All results shown were statistically significant.

Australian paradox)¹⁴. In Australia tendencies towards obesity and the consumption of sugar in the last 30 years have been put together and contrasts with what has been observed in the United Kingdom and the United States. The results confirm that in a set period, in Australia a substantial drop in the intake of sugars occurred, and during this same period obesity experienced a significant increase. In other words, the efforts made to reduce the consumption of sugar can reduce its consumption but cannot control the incidence of obesity.

Alternatively, the OMS report regarding Diet, Food and the Prevention of Chronic Diseases presented in 2003, does not show scientific evidence correlating the consumption of sugar with obesity. The same conclusion was reached by the *Institute of Medicine* in 2002, a conference of experts from the FAO and WHO in 1998, and a conference which took place in the EU in 2001. More recently, the European Food Safety Authority (EFSA) gave its opinion regarding the recommended daily carbohydrate quantities and highlighted that it was not possible to determine a top limit in the intake of sugar as there was not enough data to determine such a limit.

Threats

If excessive sugar consumption (above the recommended limits) poses a real damaging effect on health in such as it can be associated with an increase in the risk of developing obesity and suffering some form of cardiovascular disease, then why when after the consumption of similar levels of sugar some people gain weight and others not? The genetic predisposition, the different food patterns associated with the excessive consumption of sugar or the level of physical activity could explain this variability.

The genetic factors make a determining contribution to the risk of being obese. This influence oscillates between 40% and 70%¹⁵. Up to 32 loci have been identified as being heavily associated with the body mass index (BMI) in adults⁸. The evidence that these loci can contribute / determine the BMI in infancy and adolescence is always increasing. Even some loci could be affected by weight change during the lifespan. That is to say that the BMI of an adolescent tends to be similar in adulthood; obese and overweight adolescents are likely to be so too in adulthood. Not only that, studies also exist which show that certain genotypes can explain part of the variability which can be observed in the BMI and in the percentage or distribution of body fat. The understanding of the different mechanisms that determine the increase of weight during infancy and adolescence until reaching adulthood is important, from a clinical and preventative perspective. This knowledge could offer very useful information in relation to the possible extenuating effects (such as phys-

ical activity or personalised diets), which can have the capacity to modify the genetic protagonist.

Alternatively, the excess of fat is one of the main factors in the risk of developing resistance to insulin and obesity. Fats are not as abundant in nature as carbohydrates but, do in fact produce more than double the amount of energy. Furthermore, they are easy to store as an energy reserve for times of scarcity of carbohydrates. Despite their consumption having dropped in developed countries⁹, the main surveys in Spanish cities, and of almost all Western countries, highlight still, the excessive intake of fat in the general population of adults and children (above 40% in both), far from the established recommendations. This then makes us question the relationship between the excessive consumption of fat, still present in our population and obesity.

Another factor which can be a threat for the general claim (sugar-cardiovascular disease) is the changes in the intestinal flora. It has been claimed that substantial variations in the microbial intestinal community could constitute an environmental cause for being overweight and obesity. These changes can also happen as a cause of obesity, and in particular an unbalanced diet, which often is accompanied by excess weight. In animal experiments a high fat diet can induce changes in the intestinal flora, independently from the coexistence of obesity. In humans, obesity has been associated to a reduced diversity and change in the intestinal flora, but the differences observed are not homogenous between the different studies.

One very relevant factor that could question the sugar-cardiovascular disease relationship is a sedentary lifestyle. There are indicators which show clearly that our society is getting ever more sedentary. One of the most relevant indicators is the number of hours that we watch television, as is the number of television viewers. Another indicator that is growing quickly is the growing use of the Internet and “screens” in general. The majority of these indirect indicators of a sedentary lifestyle, and, of their growing tendency, reinforce the importance of a reduction of energy expenditure as a pathogenic element over which obesity is dependent.

Therefore, there are still a lot of uncertainties to determine which factor is the most influential for excess weight; elements other than sugar that contribute to obesity exist without doubt.

Opportunities

If the increase of sugar consumption above the recommended quantities is associated with an increase in the factors of vascular risk and of cardiovascular disease, then health professionals have at their disposal a great opportunity to change this occurrence. To overcome this problem effectively we should consider that

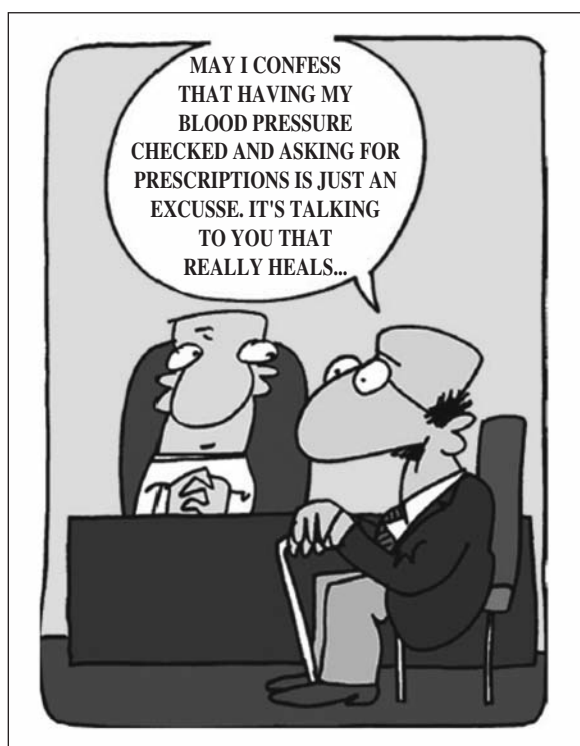


Fig. 2.—A cartoon by Summers which reflects how in the personalised services sector (i.e. – medical consultations) the level of satisfaction is in relation to the time spent listening, understanding and formulating recommendations.

any food, consumed to excess, could carry nutritional imbalances as well as impacting negatively on the state of health for which it is never advisable. We should know the current recommendations regarding the consumption of different groups of foods in the context of a balanced and healthy diet, in order to avoid possible changes which can lead to potential negative effects on your health.

Which external circumstances can reduce the excessive consumption of sugar in our society? Fundamentally, the empathy and education of dietary habits.

In the personalised services sector (i.e. - medical consultations) the level of satisfaction is in relation to time spent listening, understanding and formulating recommendations. This has been shown in multiple ways (Fig. 2).

At a family level, it is necessary to instill the basis of a balanced diet, promoting healthy eating, limiting non desirable or unhealthy ones (excessive consumption of sugar, fats, protein, pre-cooked foods, etc.) and promoting regular physical exercise.

At school, a fundamental objective is to instill dietary and lifestyle habits.

Until now, the campaigns that have taken place in the health education arena have been of an isolated Nature, with modest results. One of the possible reasons that can justify this failure is that the determining principles for which the population changes its



Fig. 3.—Sweets and dried fruits stand in a shopping centre. In the foreground one can see play trucks (white arrows) that contain these products (the trucks are used as packaging) Buying and consuming sweets is pleasant because it brings pleasure at any time of the day (i.e. an ice-cream, a summer product, now can be consumed all year round).

dietary habits and increases activity are not educational but environmental (Fig. 3). In other words there is a socio-economic and cultural background, which determine the different types of impact of the preventative campaigns. Publicity campaigns for sugary drinks and foods with a high sugar content, the presence of vending machines with high energy products in schools and colleges, the lack of areas to practice physical exercise in cities, or the ever rising price of fruit and vegetables are some of the limiting factors.

Recommendations

The key question is determining the proportion of sugar to be taken daily within the framework of a healthy and balanced diet.

With this objective in mind, the American Heart Association (AHA) recommends the reduction of energy intake from added sugars to 100-150 kcal/day, which expressed in grams of sugar daily is 25-37.5 (no more than six teaspoons per day)

In the same country, the Institute of Medicine recommends that up to a total of 25% of total consumed calories can come from sugar.

In any case there is large variety of substances such as sweeteners either high calorie or zero calorie, that in the context of a healthy lifestyle can be chosen by consumers in function of their state of health and personal preferences.

Conclusions

Even though the intake of sugar has dropped in recent years, in our country and in the majority of developed countries, in the context of nutrition aimed at,

above all, the lowering of fat and salt intake, in order to reduce the incidence of cardiovascular diseases, the possible excessive consumption of sugar has been related to the consumption of sugary drinks, which have a clear population based target, above all in the United States: children and adolescents (Fig. 3).

A clear drop in physical exercise accompanies the current obesity epidemic, with stress as pivotal pathogenic elements and a less balanced diet.

Even with all this we cannot “criminalise” sugar as it offers so many other benefits. Even though more studies are needed, there exists a sufficient basis to design strategies for public health with a view to reducing the excessive consumption of sugary drinks, as part of a healthy lifestyle. Maybe paying attention to other important aspects of our habits (playing sport, avoiding excessive animal fats, not ingesting toxins etc.) we can contribute in reducing the incidence of vascular and cardiovascular disease risk factors, and, without fear, look for the “right sugar to sweeten our lives”.

References

1. www.historiacocina.com/es/historia-del-azucar. (Consultado en 30 de Octubre de 2012).
2. Thornley S, Tayler R, Sikaris K. Sugar restriction: the evidence for a drug-free intervention to reduce cardiovascular disease risk. *Intern Med J* 2012; 42 (Suppl. 5): 46-58.
3. Castell MV, Martínez MÁ, Sanz J, García Puig J. Prevalencia, conocimiento y control de la hipertensión arterial en la población española. El estudio MADRIC. *Med Clin* 2010; 135: 671-2.
4. Rosado Martín J, Martínez López M^aA, Mantilla Morato T, Dujovne Kohan I, Palau Cuevas FJ, Torres Jiménez R, García Puig J, en representación del grupo MAPA-Madrid. Prevalencia de diabetes en una población adulta de Madrid (España). *Gaceta Sanit* 2011; 26: 243-50.
5. Gabriel R, Alonso M, Segura A, Tormo MJ, Artiago LM, Bane-gas JR et al. Prevalencia, distribución y variabilidad geográfica de los principales factores de riesgo cardiovascular en España. Análisis agrupado de datos individuales de estudios epidemiológicos poblacionales: estudio ERICE. *Rev Esp Cardiol* 2008; 61: 1030-40.
6. Soriguer F, Goday A, Bosch-Comsas A, Bordiú E, Calle Pascual A, Carmena R et al. Prevalence of diabetes mellitus and impaired glucose regulation in Spain: the Di@bet.es study. *Diabeteologia* 2012; 55: 88-93.
7. Torres Jiménez R, García Puig J. Disfunción endotelial e hiperruricemia: papel de la enzima Xantina oxidasa. *Rev Clin Esp* 2002; 202: 549-51.
8. Qibin Q, Audrey Y, Kang J, Jense MK, Curhan GC and Pasquale LR. Sugar-sweetened beverages and genetic risk of obesity. *N Engl J Med* 2012; 367: 1387-96.
9. Ruyter J, Olthof MR, Seidell JC and Katan MB. A trial of sugar-free or sugar-sweetened beverages and body weight in children. *N Engl J Med* 2012; 367: 1397-406.
10. beling CB, Feldman H and Chomitz VR. A Randomized trial of sugar-sweetened beverages and adolescent body weight. *N Engl J Med* 2012; 367: 1407-16.
11. Grundt JH, Nakling J, Eide GE, Markestad T. Possible relation between maternal consumption of added sugar and sugar-sweetened beverages and birth weight — time trends in a population. *BMC Public Health* 2012; 12: 901.
12. Fortuna JL. The obesity epidemic and food addiction: clinical similarities to drug dependence. *J Psychoactive Drugs* 2012; 44: 56-63.
13. Saris WH, Astrup A, Prentice AM, Zunft HJ, Formiguera X, Verboeket-van de Venne WP et al. Randomized controlled trial of changes in dietary carbohydrate/fat ratio and simple vs. complex carbohydrates on body weight and blood lipids: the CAR-MEN study. The Carbohydrate Ratio Management in European National diets. *J Obes Relat Metab Disord* 2000; 24: 1310-8.
14. Barclay AW, Brand-Miller J. The Australian paradox: a substantial decline in sugars intake over the same timeframe that overweight and obesity have increased. *Nutrients* 2011; 3: 491-504.
15. Bell CG, Walley AJ, Froguel P. The genetics of human obesity. *Nature Reviews Genetics* 2005; 6: 221-34.

The relationship between sucrose consumption and cancer; a review of the evidence

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Abstract

Objectives: The aim of this review is to summarize the evidence available about the association between sugar consumption, especially sucrose, and the risk of different types of cancer.

Methods: A systematic review was conducted of key reports, systematic reviews, meta-analysis as well as big prospective studies published after 2007 January 1 thru 2012 December 31 about the association between sugar consumption, especially sucrose, and the risk of cancer.

Results: Evidence of the association of the intake of mono- and disaccharides with different types of cancer is insufficient or there is evidence of lack of association. There is only possible evidence of a positive relation between the intake of monosaccharides (fructose and glucose) and pancreatic cancer. Evidence about the association between monosaccharides' intake and obesity is insufficient, as well as between the intake of sucrose or added sugars and the risk of obesity in adults and children. There is possible evidence of a positive association between glycemic index (GI) and colorectal cancer and that there is no association between GI and the risk of endometrial cancer, breast cancer and pancreatic cancer.

Conclusion: More research is needed. Cohort studies are especially required and randomized intervention trials would be desirable, although these are difficult in this field.

Nutr Hosp 2013; 28 (Supl. 4):95-105

Key words: Sugar. Sucrose. Disaccharides. Glycemic index. Glycemic load. Cancer. Obesity.

RELACIÓN ENTRE EL CONSUMO DE SACAROSA Y CÁNCER; UNA REVISIÓN DE LA EVIDENCIA

Resumen

Objetivos: El objetivo de esta revisión es resumir la evidencia disponible sobre la asociación entre el consumo de azúcar, principalmente sacarosa, y el riesgo de cáncer de distinta localización.

Métodos: Se ha realizado una revisión de los principales informes publicados, las revisiones sistemáticas, meta-análisis, así como grandes estudios epidemiológicos prospectivos publicados con fecha posterior al 1 de enero de 2007 hasta 31 de diciembre de 2012 sobre la asociación entre el consumo de azúcar, principalmente sacarosa, y el riesgo de cáncer.

Resultados: La evidencia de asociación entre la ingesta de mono y disacáridos con los distintos tipos de cáncer es insuficiente o existe evidencia de no asociación. Sólo existe evidencia posible de una relación positiva entre la ingesta de monosacáridos (fructosa y glucosa) y el riesgo de cáncer de páncreas. La evidencia sobre la asociación entre la ingesta de monosacáridos y el riesgo de obesidad es insuficiente, lo mismo que en relación con la ingesta de sacarosa o azúcar añadido y el riesgo de obesidad en adultos o en niños. Existe evidencia posible de una asociación positiva entre el índice glucémico (IG) y cáncer colorrectal y de que no hay asociación entre el IG y el riesgo de cáncer de endometrio, mama y páncreas.

Conclusiones: Es necesario seguir investigando. Son especialmente necesarios estudios de cohortes y serían deseables estudios aleatorizados de intervención, aunque son difíciles en este ámbito.

Nutr Hosp 2013; 28 (Supl. 4):95-105

Palabras clave: Azúcar. Sacarosa. Disacáridos. Índice glucémico. Carga glucémica. Cáncer. Obesidad.

Abbreviations

GL: Glycaemic Load. CI: Confidence Interval.

GI: Glycaemic Index.

IGF: Insulin like Growth Factor.

BMI: Body Mass Index.

NIH-AARP: National Institute of Health (previously American Association of Retired Persons).

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EPIC: European Prospective Investigation into Cancer and Nutrition.

OR: Oestrogen Receptor.

PR: Progestagen Receptor.

RR: Relative Risk.

WCRF-AICR: World Cancer Research Fund-American Institute for Cancer Research.

Introduction

Sugars form part of food, and they are used as sweeteners, and as preservatives and volume forming agents in some cases. They also contribute to improving the palatability of starches, fats and other ingredients. Sugars and syrups elaborated from sugar cane, beet and corn are used in many foods and processed drinks.

Up until the 16th century when sugar cane was first cultivated in large harvests, foods with added sugar were considered a luxury. Consumption of sugar beet, sugar cane and syrups greatly increased in industrialised countries in the 19th and 20th centuries, and it continues to grow all over the world, especially in poor countries. In many countries in the last few decades, sugar consumption represents a considerable part of energy intake.

Reports on sub nutrition often recommend a considerable amount of sugars and fats to their energetic density, which allows for a rapid weight gain, and because of the innate preference for sweet flavours, which favours energetic intake. On the other hand, reports on prevention of chronic illnesses usually recommend moderate consumption of sugars, among other reasons because there is evidence that sugars are the cause of tooth decay, although a combination of amount and frequency of sugar, exposure to fluoride and the stickiness of the food are better indicators of the risk of tooth decay than the amount of sugar consumed alone.

Intake of sugars, in the amounts usually consumed in industrialised nations, has become associated with obesity, and therefore, indirectly associated with illnesses related to obesity, although the evidence available in this sense is not sufficient. There are hypotheses stating that diets with high glycaemic response levels, and consequently hyperinsulinemia, may be implicated in the aetiology (study of cause or origination) of some types of cancer.

When we talk about sugars, we mean all sugars contained within a diet, which are mainly extrinsic, but we do not mean sucrose (commonly known as sugar) alone. The term “extrinsic sugars” also includes maltose, lactose, glucose and fructose contained within food and drink, including juices and milk, as well as honey and syrups, fructose-rich syrups, refined sugars used in the processing, preparation and cooking of foods, and at the table. Intrinsic foods are those that are present in natural form in foods such as fruit or in milk.

Sucrose is refined from sugar beet and sugar cane. Maltose and glucose are mostly refined from corn. The rich syrup in fructose includes a mix of glucose and fructose, usually in equal amounts, and currently it is used a lot in the production of foods and drinks, especially in the USA.

Sugars are simple carbohydrates that provide 3.75 kcal per gram. They are simple molecules like glucose, fructose, and galactose (monosaccharides) and two joined up molecules (disaccharides), like sucrose (fructose and glucose); lactose (glucose and galactose); or maltose (two molecules of glucose). The body metabolises the different sugars at different speeds. For example, fructose is absorbed and metabolised slower than glucose or sucrose. It is also somewhat sweeter than glucose or sucrose, and therefore, it can be substituted in lesser total amounts. Chemical sweeteners without calories produce a sweet flavour, but they are not sugars.

There are no dietary requirements for sugars. The World Health Organisation recommends that average sugar consumption should be less than 10 per cent of total energy intake¹.

The objective of this review is to summarise the available evidence about the links between sugar consumption, especially sucrose, and the risk of cancer of different parts of the body.

Methods

There has been a review of the main published reports, systematic reviews, meta-analyses and recent large-scale prospective epidemiological studies of the relationship between sugar consumption, especially sucrose, and the risk of cancer. The systematic review carried out by the World Cancer Research Foundation (WCRF) in collaboration with the American Institute for Cancer Research (AICR) was taken as a reference, and the report published in 2007 was based on it². This review brought together studies published up to December 2006. We have centred our strategy on searches for systematic reviews and meta-analyses published after the 1st of January 2007 until the 31st of December 2012. The bibliographic search was done on PubMed using the following key words in the search execution: (((“dietary carbohydrates/ adverse effects”[Mesh Terms] OR “dietary sucrose/ adverse effects”[Mesh Terms]) OR “glycaemic index” [MeSH Terms]) AND “humans” [MeSH Terms]) and “neoplasms”[MeSH Terms])). This method produced 226 publications, of which 110 had a publication after the 1st of January 2007. Special attention was given to meta-analyses and systematic reviews. 9 meta-analyses were identified along with 21 additional systematic reviews. The identification of studies was completed with the manual search and review of the bibliographical lists and preselected works, after an

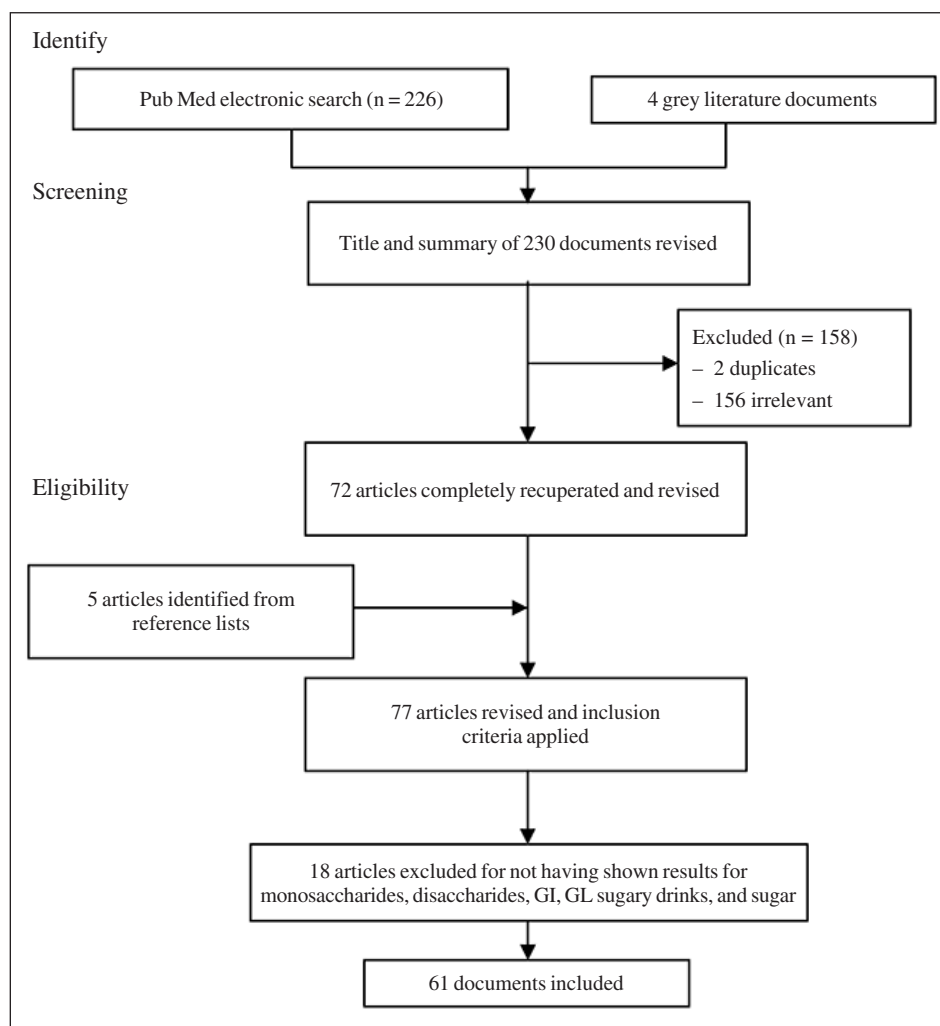


Fig. 1.—Identification, screening and selection for systematic revision process flow.

initial screening, following the PRISMA document. Only the studies carried out on human beings have been included, which analyse the relationship between consumption of total sugars, added sugars, monosaccharides, disaccharides, glycaemic index and the appearance of cancer. Secondary cancer prevention studies have not been included. After the exclusion of duplicates and work that did not meet the requirements for inclusion, there are 61 publications in this review (Fig. 1).

Weaknesses

One of the main difficulties when researching the relationship between sugar consumption and health is that it becomes very complicated to measure and evaluate the overall effect of sugars as possible modifiers of the risk of any illness, including cancer, in part due to inconsistency in terms of classifications of sugars. Sometimes “sugar” is identified as sucrose, traditionally the main sugar in the human diet, and some studies

research packaged sugar used domestically in families. However, in general, this type of sugar represents a relatively small proportion of all the types of sugar consumed, which is decreasing. Other studies include sugars contained in their natural form, in fruit and milk. Therefore, we need to study the methodology used in each study meticulously, in order to evaluate the results referred to. These methodological limitations complicate analysis of the evidence. It is very difficult to compare studies that use such different classifications for sugar.

Another possible inaccuracy in the studies could be the underestimation of real sugar intake, because added sugars are usually perceived as unhealthy foods, and therefore, studies of consumption based on information provided by participants might underestimate their consumption.

High sugar consumption is not an isolated feature of diet, but it usually forms part of consumption patterns that often go alongside low consumption of fruits, vegetables and legumes (green vegetables), in which carbohydrates could be indicative of a poor quality

diet, as well as high levels of endogenous insulin. Hyperinsulinemia (high levels of insulin in the blood) is usually found in instances of tolerance of adulterated glucose, which has been linked to a greater risk of cancer^{3,4}. It has been found that insulin acts as a catalyst for cancer *in vitro* (test tube) studies and in animals, possibly with an increase in insulin like growth factor insulin (IGF) IGF-1. IGF-1 inhibits apoptosis (programmed cell death); increases production of endothelial vascular growth factors and it has been linked to greater mitogenesis (mitoses induction) in cell lines of different types of cancer. High levels of IGF-1 have been observed in gastric (stomach) cancer patients when compared to healthy individuals².

Dietary demand for insulin is determined by the type of carbohydrates consumed, and by the combination of type and amount of carbohydrates. The glycaemic index (GI) and the glycaemic load (GL) are indicative of the absorption rate of carbohydrates in the diet and therefore they are measures of the demand for insulin, which in turn may be linked to IGF. The least favourable situation would be when foods with a high GI are consumed in large amounts (e.g. high quantities of refined cereals). However, other factors come into play in the secretion of insulin such as protein intake, and the addition of fats to a source of carbohydrates which may increase the insulin reaction without increasing glycaemia.

We currently have clearer evidence that excess weight and obesity increase the risk of certain types of cancer. In recent decades, excess weight and obesity rates in adults and children have increased considerably in most countries. There is evidence that foods and diets of high energy level content, especially highly processed foods and sweetened beverages, increase the risk of excess weight and obesity^{2,6}.

On the other hand, in most populations, people who live in urban industrialised areas participate in levels of physical activity that are below levels that the human body is adapted to. The increase in sedentary time and low levels of physical activity encourage excess weight.

Nutritional interventions add ethical, economic and logistical complications that make it difficult to carry out controlled experiments. Furthermore, unfeasibly long periods of exposure are usually necessary. For this reason, literary evidence in the field of nutrition is usually based mainly on long-term observational studies, and these lead recommendations classified as level C, and sometimes level B, in which there are some quality studies with minimal risk of bias. When nutrients that require less exposure time, it is also possible to achieve A level evidence.

Threats

Reports by experts, such as the one by the FAOOMS committee published in 2003¹ and others, did not

consider that high sugar consumption was linked to cancer. The WCRF report, based on studies published up to 2006, concluded that there is limited evidence to suggest that sugar is a cause of bowel cancer. This was also the conclusion of a review produced by a committee of experts for the FAO on the role of carbohydrates in human health from 2007⁵.

In the systematic review produced by Ruxton et al.⁷, which combines publications from between 1995 and 2006, 18 studies that analyse links between cancer risk and consumption of sucrose, sugar and foods that contain sugar were identified. The authors concluded that in spite of including studies that were more recent than previous reviews, the findings were still in line with previous reviews, with inconsistent evidence regarding the role of sugar intake in cancer development.

A review on which the production of Food Guides in Germany was based, using studies published up to December 2009 and meta-analyses published up to 2010, concluded that the evidence was insufficient in some cases, but it also pointed to evidence of a possible link between intake of monosaccharides and the risk of pancreatic cancer⁸.

The review of evidence that backs up the Australian Dietary Guidelines published in December 2011 includes studies published up to April 2009. The authors concluded that sucrose consumption was not linked to a greater risk of cancer, with C level evidence available.

GI and GL have been linked to bowel, breast, ovarian and uterine (lining of the uterus) cancers, and this suggests the hypothesis of a greater risk of gastric (stomach) cancer with high ingestion of carbohydrates^{2,6}.

Stomach Cancer

The hypothesis of a greater risk of stomach cancer with a high intake of carbohydrates has been suggested. High GIs and GLs can come as a result of high intake of refined carbohydrates, which usually comes accompanied with a low intake of vitamins, minerals, anti-oxidants and fibre, as well as a low consumption of legumes (green vegetables), fruit, vegetables and wholegrain cereals, food groups that have been inversely linked to the risk of stomach cancer. In some studies no links between GI and risk of stomach cancer have been found, which may suggest that it is not just the type but also the amount of carbohydrates that could be linked to the carcinogenesis of this form of cancer^{5,6}.

In the systematic review by the WCRF, no prospective or intervention studies were identified on the links between monosaccharides, disaccharides or sweetened beverages and the development of stomach cancer. Subsequently, there have been some panel studies and

investigations into this link, although the evidence is not sufficient^{7,8}.

Uterine cancer (cancer of the lining of the uterus)

Uterine cancer is linked to obesity, diabetes and polycystic ovary syndrome, all of which are linked to hyperinsulinemia (high levels of insulin in the blood). Diets with a high glycaemic index (e.g. with a high content of foods rich in refined carbohydrates and low in legumes (green vegetables), fruits, vegetables and wholegrain cereals) are characterised by a rapid absorption of their carbohydrate component in the consequent increase in glucose and insulin levels.

Carbohydrates have been directly linked to the risk of uterine cancer, mainly with refined carbohydrates^{9,10}, while foods with a wholegrain cereal base seem to play a protective role. In some studies a stronger link has been observed in women who are overweight, but it also seems that diets with a high GI tend to increase the risk of uterine cancer in women with a low body mass index (BMI). Although oestrogen is the main factor in the risk of uterine cancer, insulin can alter hormonal balance. Insulin has been seen to act as a cancer-inducing agent in test tube (in vitro) and in animal studies. It also has an affinity with receptors of IGF, which means IGF levels increase. It has been observed that IGF-1 stimulates mitogenesis (triggering of mitosis – process of encouraging a cell to commence cell division), in cell lines of uterine cancer. Another possible mechanism due to which a high glycaemic index may contribute to a risk of uterine cancer could be an increase in oxidative stress.

In the systematic review by the WCRF, a panel study is described which researched the links between sugar intake and uterine cancer, although this study was not related to risk in any way². Subsequently, two group studies have been identified that do not provide evidence of links with risk^{6,8}. The NIH-AARP Diet and Health Study cohorts found non-significant links that implied greater risk with fructose and sucrose intake¹¹.

Pancreatic cancer

In the systematic review by the WCRF form 2007, we are informed of a panel study that researched the relationship between fructose intake and pancreatic cancer. In this study there was no major significant risk from intake of fructose². Three more recent studies in the USA focused on this relationship and did not find a greater risk of pancreatic cancer with higher fructose intakes. In one of them, the NIH-AARP study, it was found that there was a greater risk from a high intake of glucose. On the other hand, in the study of Diet and Health in the Low Countries, intake of monosaccha-

rides and disaccharides was not linked to pancreatic cancer⁸.

The WCRF review looked at three panel studies on sucrose intake and none of these mentioned a link with pancreatic cancer². In studies published afterwards it was not seen that the amount of sugar consumed was a risk factor for pancreatic cancer. Nor did it mention a link with consumption of sweets or jams, or with the GI^{6,8}.

In one meta-analysis that carried out research into links between sweetened beverages consumption and pancreatic cancer, the analysis of 6 panel studies did not find evidence of change in risk of pancreatic cancer in participants who consumed sweetened beverages in comparison with those who did not (RR = 1.05; IC 95% 0.94; 1.17)¹². A study carried out in Sweden and another in China that analysed several categories found a positive link of risk between sweetened beverage intake and pancreatic cancer.

Meta-analysis of panel studies¹³ and another of group studies and cases and controls¹⁴ did not find links between GI, GL and the risk of pancreatic cancer. A later meta-analysis, which included a great number of studies and approximately 3000 cases, with therefore greater statistical power, found that only a few specific types of carbohydrates could increase the risk of pancreatic cancer. A link was found between insulin load and fructose intake, but not with the total intake of carbohydrates¹⁵.

The results of a meta-analysis published in 2012 do not support links between diets with a high glycaemic index, glycaemic load, total carbohydrate or sucrose intake and risk of pancreatic cancer. A greater risk was observed with fructose intake, although more studies are necessary to confirm and back up this finding, taking into account possible confounding factors¹⁶.

The data analysis of the Nurses Study and the Study of Health Professionals in the USA attempted to show that diets rich in foods that increase postprandial (after eating) insulin concentration do not increase overall risk of pancreatic cancer. However, in people with a BMI of less than 27, low physical activity and a greater presence of insulin are linked to a greater risk of pancreatic cancer, although not significant¹⁵.

Table I summarises the most relevant details of the most recent evidence of the relationship between sugar and risk of pancreatic cancer. There is some possible evidence of a positive link between monosaccharides intake and pancreatic cancer. There is also convincing evidence of causal links between body adiposity and pancreatic cancer; the evidence is probable with regards to abdominal fat.

Breast cancer

In the WCRF's systematic review of 2007, three panel studies on lactose, sucrose or fructose are described. No link was found between sucrose, lactose

Table I
Relationship between sugar intake, glycaemic index, glycaemic load and pancreatic cancer 2006

Author, year	Study description		Glycaemic index RR (IC 95%)	Glycaemic Load RR (IC 95%)	Sugars RR (IC 95%)
	n	sex			
Panel studies					
Larsson et al., 2006	77,797	M, F			1.69 (0.99-2.89) ^b
Nöthlings et al., 2007	162,150	M, F		1.10 (0.80-1.52)	1.28 (0.95-1.73) ^a 1.08 (0.81-1.44) ^b 1.23 (0.91-1.65) ^c 1.35 (1.02-1.80) ^d
Patel et al., 2007	124,907	M, F	0.92 (0.68-1.24)	1.01 (0.75-1.37)	
Bao et al., 2008	487,922	M, F			0.85 (0.68-1.06) ^b 1.12 (0.91-1.39) ^c
Heinen et al., 2008	120,852	M, F	0.87 (0.59-1.29)	0.85 (0.58-1.24)	0.78 (0.52-1.16) ^a
Jiao et al., 2009	482,362	M, F	1.09 (0.90-1.32)	0.95 (0.74-1.22)	1.10 (0.88-1.38) ^a 0.95 (0.78-1.16) ^c 1.29 (1.04-1.59) ^d 1.35 (1.10-1.67) ^e
Meinhold et al., 2010	109,175	M, F	1.08 (0.78-1.49)	1.45 (1.05-2.00)	1.37 (0.99-1.89) ^c 1.22 (0.87-1.71) ^d
	255,696 2,601 cases	M			0.98 (0.69-1.39) ^a 1.02 (0.75-1.38) ^b 0.96 (0.71-1.29) ^c 1.05 (0.76-1.46) ^d
Tasesvka et al., 2012 ¹¹	179,990 1,296 cases	F			1.05 (0.66-1.67) ^a 0.71 (0.47-1.08) ^b 0.85 (0.56-1.28) ^c 0.93 (0.60-1.44) ^d
Meta-analyses					
Gnardella et al., 2008 ¹⁴ Panels and case controls until Oct. 2007	12,790	M, F	1.11 (0.86-1.43)	1.00 (0.94-1.53)	
Barclay et al., 2008 ¹³ Panels until December 2007	349,982	M, F	1.03 (0.83-1.27)	1.02 (0.82-1.27)	
Mulholland et al., 2009 Panels and case controls until Jul. 2008	719,066	M, F	0.99 (0.83-1.19)	1.01 (0.86-1.19)	
Aune et al., 2012 ¹⁶ Panels and case controls until Oct. 2011	994,154	M, F	1.04 (0.93-1.17)	1.01 (0.88-1.15)	1.14 (0.96-1.35) ^a 1.02 (0.85-1.23) ^c 1.18 (1.01-1.37) ^d

M: Male; F: Female; RR: Relative Risk; IC: IC: Interval of confidence; ^a Total sugars; ^b added sugar; ^c sucrose; ^d total fructose; ^e glucose.

Referencias:

- Larsson SC, Bergkvist L, Wolk A. Consumption of sugar and sugar-sweetened foods and the risk of pancreatic cancer in a prospective study. *Am J Clin Nutr* 2006; 84: 1171-6.
- Nöthlings U, Murphy SP, Wilkens LR, Henderson BE, Kolonel LN. Dietary glycaemic load, added sugars, and carbohydrates as risk factors for pancreatic cancer: the Multiethnic Cohort Study. *Am J Clin Nutr* 2007; 86: 1495-501.
- Patel AV, McCullough ML, Pavluck AL, Jacobs EJ, Thun MJ, Calle EE. Glycemic load, glycemic index, and carbohydrate intake in relation to pancreatic cancer risk in a large US cohort. *Cancer Causes Control* 2007; 18: 287-94.
- Bao Y, Stolzenberg-Solomon R, Jiao L, Silverman DT, Subar AF, Park Y, Leitzmann MF, Hollenbeck A, Schatzkin A, Michaud DS. Added sugar and sugar-sweetened foods and beverages and the risk of pancreatic cancer in the National Institutes of Health-AARP Diet and Health Study. *Am J Clin Nutr* 2008; 88: 401-31.
- Heinen MM, Verhage BA, Lumey L, Brants HA, Goldbohm RA, van den Brandt PA. Glycemic load, glycemic index, and pancreatic cancer risk in the Netherlands Cohort Study. *Am J Clin Nutr* 2008; 87 (4): 970-7.
- Jiao L, Flood A, Subar AF, Hollenbeck AR, Schatzkin A, Stolzenberg-Solomon R. Glycemic index, carbohydrates, glycemic load, and the risk of pancreatic cancer in a prospective cohort study. *Cancer Epidemiol Biomarkers Prev* 2009; 18 (4): 1144-51.
- Meinhold CL, Dodd KW, Jiao L, Flood A, Shikany JM, Genkinger JM, Hayes RB, Stolzenberg-Solomon RZ. Available carbohydrates, glycemic load, and pancreatic cancer: is there a link? *Am J Epidemiol* 2010; 171 (11): 1174-82.
- Mulholland HG, Murray LJ, Cardwell CR, Cantwell MM. Glycemic index, glycemic load, and risk of digestive tract neoplasms: a systematic review and meta-analysis. *Am J Clin Nutr* 2009; 89 (2): 568-76.

or fructose intake and breast cancer². The Danish branch also investigated this link in the EPIC study, but no link was found in this case either. No panel studies were found on links with the consumption of sweetened beverages.

The meta-analysis carried out by Ruxton et. al in 2010⁷ looked at four studies on breast cancer, and three of these mentioned some level of links between risk and different estimators of sugar intake, although in a somewhat inconsistent way. Other authors found links with some sweet foods, but not with others. In a preceding systematic review a significant link was found between breast cancer and a high intake of cakes and biscuits, although they pointed out that apart from sugar, these foods also contain high quantities of fats, which may be a confounding factor. A systematic review of panel studies did not find a link with the GI or GL¹⁷.

In the Women’s Health Initiative cohort, no link was observed between GI, GL or carbohydrate intake and breast cancer, although a possible relationship is suggested between GL and carcinoma in situ (the early stages of cancer).

In the EPIC study, it was observed that a diet with high GL and carbohydrate intake is positively linked with a greater risk of breast cancer types that are receptors of negative oestrogen (OR-) and OR- / receptors of progesterone (PR-) in postmenopausal women¹⁸. An

Italian panel study mentioned this link in premenopausal women.

Diets that are rich in carbohydrates, especially those with high GI or GL, increase postprandial (after eating) concentrations of glucose and insulin. This metabolic change, as with diabetes, has been linked to lesser chances of survival in breast cancer^{19,20}.

The second WCRF-AICR report concluded that it seems probable that body fat may reduce the risk of breast cancer before the menopause, but there is insufficient evidence for a link to be established between intake of sucrose and other sugars, as well as sweetened foods and beverages, with premenopausal breast cancer. In postmenopausal women, however, there is convincing evidence that adds substance to the link between body adiposity and a greater risk of breast cancer, and it is likely that abdominal body fat gain (adiposity) and weight gain at adult age also implies a greater risk². There is not enough evidence either for linking breast cancer with sucrose intake, foods and soft drinks, or the glycaemic index (Fig. 2).

Colorectal cancer

The 2007 WCRF review looked at a panel study and 7 case-control studies researching the relationship

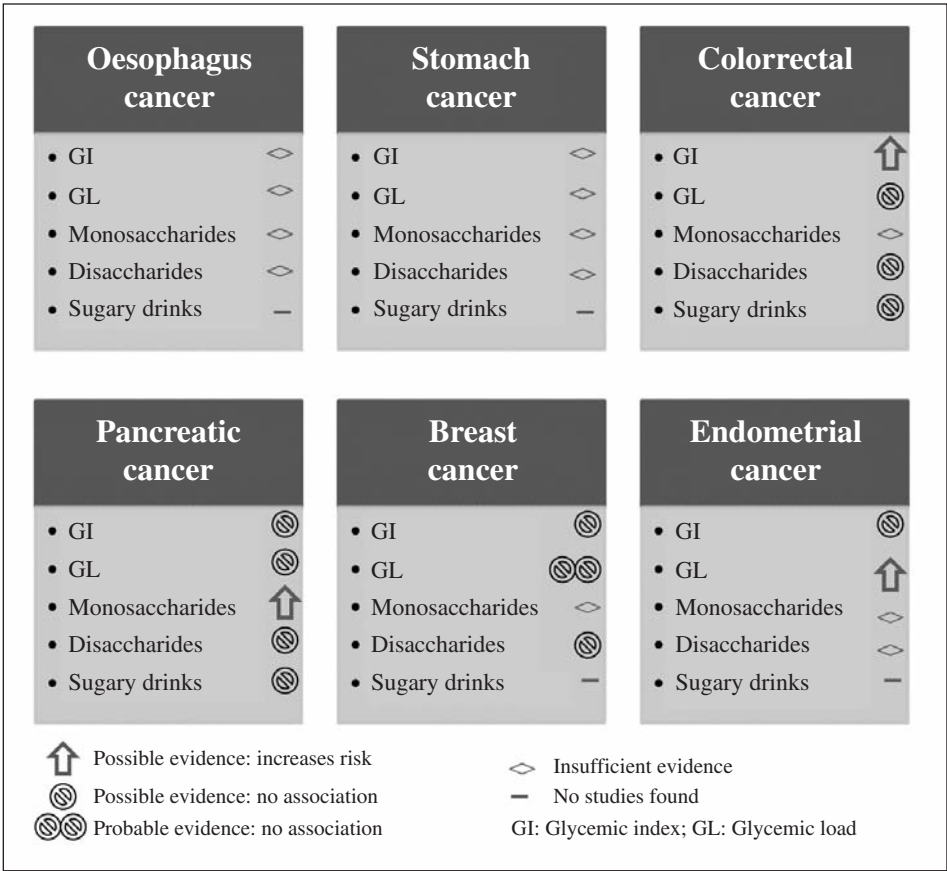


Fig. 2.—Summary of evidence for links between sugar intake, glycaemic index, glycaemic load and primary prevention of different forms of cancer.

between consumption of sugars as food and bowel cancer. Seven panel studies and 16 case-control studies on the relationship with sugars as nutrients, defined as sucrose or fructose. In the first group, looking at sugars as foods, the study panel stated that there was no link between adding sugar to cereals and cancer of the colon and rectum². In the case control studies a greater risk was observed with greater sugar consumption, statistically significant in two of them. However, the establishment of “sugar” as a food varied from one study to the next. The available evidence on the links between sucrose and bowel cancer is difficult to interpret; in any case there is limited evidence that suggests sugar might be a cause of bowel cancer^{2,8,21}.

Of the four panel studies on fructose intake, a positive link was found in one, and two on lactose intake did not find any links with risk. None of the three panel studies on disaccharide intake showed significant positive links with risk.

In subsequent panel studies that have not been included in this review such as the NIH-AARP Diet and Health Study¹¹ no links were found between sugar intake and cancer of the colon and rectum. A meta-analysis of case-control studies and panel studies published in 2008 suggested a direct overall link between GI, GL and bowel cancer¹⁴. The analysis of 13 panel studies that formed part of the Pooling Project did not find any links between sweetened beverage consumption and bowel cancer⁸.

According to the WCRF report, there is convincing evidence that body fat gain and abdominal fat gain increase the risk of bowel cancer. There is limited evidence to suggest that foods containing sugar increase the risk, and there is inconclusive limited evidence of the links with sucrose consumption or total carbohydrates. Subsequent reviews concluded that the evidence suggests a link with sugar, but the energy intake and the glycemic load could be confounding factors.

Meta-analyses published in 2012^{21,22} concluded that there is no consistent evidence, although they did observe a modest excess risk in case-control studies in which added sugars, GI and GL are linked to a greater risk of cancer of the colon, in spite of the effects on energy intake, obesity or diabetes, linked to greater risk of cancer of the colon.

Table II summarises the most relevant and current information from 2007 onwards on the relationship between sugar intake and bowel cancer.

Around 60 panel studies and 86 case-control studies have researched the link between body fat gain and bowel cancer, using the BMI as a gauge in most cases. The majority of panel studies showed a greater risk of cancer with greater body fat gain. The meta-analyses in panel studies demonstrated an increased risk of 15% for each 5 kg/m². Layered according to the location of the tumour, the magnitude of the increased risk is even greater for colon cancer than for rectum cancer.

There is a lot of consistent epidemiological evidence with a clear “dose and response” relationship, and plausible mechanical evidence. The evidence for greater body fat gain as a cause of bowel cancer is convincing. The risk of cancer is modified not just by obesity, but also by excess weight, and even by levels of body fat gain that could be considered to be within normal healthy weight limits.

Regarding obesity and excess weight, evidence for high consumption of drinks with added sugars, including sucrose and syrups rich in fructose, being linked to weight gain, excess weight and obesity is consistent, according to the WCRF report. This report classifies as probable evidence that high consumption of these products causes weight increase and obesity. However, other subsequently published meta-analyses state that the evidence of the importance of monosaccharides in obesity is not sufficient⁸, as is the evidence for the importance of sucrose or added sugar respectively to the risk of obesity in adults.

Panel studies and intervention studies show a greater risk of obesity in adults who consume sweetened beverages, with the level of evidence showing this to be probable. The conclusions of two out of four meta-analyses state that a greater consumption of sweetened beverages in children and teenagers is linked to a greater risk of obesity, whilst in another meta-analysis, no link was found. Subsequent panel studies confirm this greater risk. The most recent panel study concludes that the greatest risk of obesity is limited to individuals who already have a high BMI or excess weight, and the level of evidence is classed as possible.

Ovarian cancer and prostate cancer

In some panel studies it has been suggested that consumption in diets with high GL could be linked to a greater risk of ovarian cancer, although the evidence is limited and inconclusive^{2,8}.

The results of a case-control study in Australia suggested that diets with a high glycaemic load could increase the risk of ovarian cancer, especially in women who are overweight or obese. Panel studies have not found links between carbohydrate intake, GI or GL and the risk of prostate cancer.

Strengths

In recent years, the number of studies investigating links between sugar consumption and chronic illnesses like cancer has increased. Systematic reviews of the literature and meta-analyses have also been carried out attempting to answer this question. One of the most detailed critical reviews of the available evidence was carried out by the WCRF-AICR, which published its first report in 1997, and the second one in November

Table II
Relationship between sugar intake, glycaemic index and glycaemic load, and bowel cancer.
Summary of relevant evidence published since 2008

Author, year	Description studies		Glycaemic index RR (IC 95%)	Glycaemic load RR (IC 95%)	Sugar RR (IC 95%)
	n	sex			
Panel studies					
Weijenberg et al., 2008	120,852 1,811 cases	M, F	0.81 (0.61-1.08)	0.83 (0.64-1.08)	
Howarth et al., 2008	85,898 105,106	M F		1.15 (0.89-1.48) 0.75 (0.57- 0.97)	
Kabat el al., 2008	158,800 1,476 cases	F	1.10 (0.92–1.32)	1.11 (0.82-1.49)	1.16 (0.91-1.49) ^a
George et al., 2009	262,642 183,535	M F	1.16 (1.04-1.30) 1.16 (0.98-1.37)	0.88 (0.72-1.08) 0.87 (0.64-1.18)	
Li et al., 2011	73,061	F	1.09 (0.81-1.46)	0.94 (0.71-1.24)	
Tasesvka et al., 2012 ¹¹	255,696 2,601 cases	M			0.95 (0.83-1.09) ^a 1.02 (0.89-1.16) ^b 1.06 (0.93-1.21) ^c 0.99 (0.87-1.14) ^d
	179,990 1,296 cases	F			1.06 (0.87-1.29) ^a 0.99 (0.81-1.19) ^b 1.11 (0.92-1.33) ^c 1.05 (0.87-1.27) ^d
Meta-analysis					
Gnardella et al., 2008 ¹⁴ Panels and case controls until Oct. 2007	12,790 Panles + case controls	M, F	1.18 (1.05-1.34)	1.26 (1.11-1.44)	
Barclay et al., 2008 ¹³ Panels, until December 2007	349,982	M, F	1.10 (1.00-1.21)	1.08 (0.92-1.26)	
Mulholland et al., 2009 Panels and case controls until July 2008	719,066 20,330 cases	M, F	1.15 (0.99-1.34)	1.17 (0.98-1.39)	
Bosetti et al., 2009 Case-control and studies until 2007	10,000 1,225 cases	M, F	1.7	1.8 (1.5-2.2)	
Galeone et al., 2012 ²¹ Panels and case controls until 2012		M, F	1.17 (1.00-1.36)	1.01 (0.84-1.21)	1.25 (1.03-1.50) ^b
Aune et al., 2012 ²² Panels, cases and controls until Oct. 2011	994,154 12,382 cases	M, F	1.07 (0.99-1.16)	1.00 (0.91-1.10)	1.01 (0.87-1.16) ^c 1.05 (0.87-1.27) ^d

M: Men; W: Women; RR: Relative Risk; CI: Confidence interval

^aTotal sugars; ^bAdded sugar; ^cSucrose; ^dTotal fructose.

Referencias:

- Weijenberg MP, Mullie PF, Brants HA, Heinen MM, Goldbohm RA, van den Brandt PA. Dietary glycaemic load, glycaemic index and colorectal cancer risk: results from the Netherlands Cohort Study. *Int J Cancer* 2008; 122 (3): 620-9.
- Howarth NC, Murphy SP, Wilkens LR, Henderson BE, Kolonel LN. The association of glycaemic load and carbohydrate intake with colorectal cancer risk in the Multiethnic Cohort Study. *Am J Clin Nutr* 2008; 88 (4): 1074-82.
- Kabat GC, Shikany JM, Beresford SA, Caan B, Neuhauser ML, Tinker LF, Rohan TE. Dietary carbohydrate, glycaemic index, and glycaemic load in relation to colorectal cancer risk in the Women's Health Initiative. *Cancer Causes Control* 2008; 19 (10): 1291-8.
- George SM, Mayne ST, Leitzmann MF et al. Dietary glycaemic index, glycaemic load, and risk of cancer: a prospective cohort study. *Am J Epidemiol* 2009; 169: 462-72.
- Li HL, Yang G, Shu XO, Xiang YB, Chow WH, Ji BT, Zhang X, Cai H, Gao J, Gao YT, Zheng W. Dietary glycaemic load and risk of colorectal cancer in Chinese women. *Am J Clin Nutr* 2011; 93 (1): 101-7.
- Mulholland HG, Murray LJ, Cardwell CR, Cantwell MM. Glycaemic index, glycaemic load, and risk of digestive tract neoplasms: a systematic review and meta-analysis. *Am J Clin Nutr* 2009; 89 (2): 568-76.
- Bosetti C, Pelucchi C, La Vecchia C. Diet and cancer in Mediterranean countries: carbohydrates and fats. *Public Health Nutr* 2009; 12 (9A): 1595-600.

2007. This second report based its conclusions and recommendations on the analysis of evidence published up until the end of 2005, and then completed, subsequently, with studies published in 2006².

Since then, new evidence has been published, and other work groups have carried out systematic reviews and meta-analyses analysing the links between sugar consumption and some types of cancer^{6-8;12-14}.

The evidence is getting better all the time, and it is getting better in terms of quality. Systematic reviews have also improved, including evaluations of the quality of the methodologies used in the studies, and better control over confounding factors. The relationship between sugar intake and the risk of cancer is usually adjusted to the total energy intake, in such a way that sugars are valued as a proportion of total energy in a diet.

The most recent reviews are usually based on previous meta-analyses and systematic reviews, as well as on original publications that were not covered in existing reviews. Until now, the most recent meta-analyses on this topic cover publications up until October 2011, and they investigate the links between carbohydrate intake, GI, and GL and pancreatic cancer, or with bowel cancer.

However, there are still many areas that still need to be researched. Meta-analyses and panel studies are required.

Opportunities

Evidence for links between monosaccharide and disaccharide intake and the various forms of cancer is insufficient or there is evidence against links. There is only possible evidence for a positive relationship between monosaccharide intake and the risk of pancreatic cancer.

Although the evidence is insufficient, it has been suggested that added sugars, GI and GL could be linked to cancer of the colon, in spite of the effects on energy intake, obesity or diabetes. It has also been suggested in some panel-studies that consumption in diets with a high GL could be associated with a greater risk of ovarian cancer, and there is convincing evidence that abdominal fat gain increase the risk of bowel cancer and postmenopausal breast cancer.

High consumption of sweetened beverages increases the risk of obesity, with strong evidence for adults, and possible evidence for children.

There is evidence for the practice of physical activity as protection against cancer, and it helps to avoid excess weight and therefore cancers related to obesity have increased considerably since the 1990s.

When researching the links between carbohydrate intake and the appearance of chronic illness, one must take into account that the overall risk is determined by the interaction of lots of different food components, so

the importance of isolated components must not be overestimated.

Recommendations

More research is required. Panel studies are particularly important, and ideally randomised intervention studies are useful, although they are difficult to organise in this area.

Based on the evidence available, it would be ideal to keep body weight at a healthy level. Living an active life, avoiding sedentarism, practising regular physical exercise of moderate intensity, limiting consumption of food and drinks with high energetic density that favour weight gain, consuming a good amount of green foods with a full presence of fruit and vegetables, as with as regular consumption of wholegrain cereals are some measures that could help to maintain body weight.

Conclusions

Evidence in relation to intake of monosaccharides and the risk of cancer of the oesophagus, bowel cancer and breast cancer is insufficient. There is some possible evidence of links with a greater risk between monosaccharide intake and pancreatic cancer.

Regarding intake of disaccharides, the link to the risk of cancer of the oesophagus and uterine cancer is also insufficient. There is evidence that there is possibly no link between disaccharide intake and a risk of bowel cancer, breast cancer and pancreatic cancer.

There is also evidence that there is possibly no link between consumption of sweetened beverages and risk of bowel cancer and breast cancer.

Evidence of a link between GI and the risk of cancer of the oesophagus and stomach is insufficient. There is possible evidence for a positive link between GI and bowel cancer. There is evidence that there is possibly no link between GI and the risk of uterine cancer, breast cancer and pancreatic cancer.

The evidence of links between GL and the risk of cancer of the oesophagus is insufficient. In relation to uterine cancer, there is possible evidence of a positive risk with the GL. There is possible evidence that GL is not linked to risk of bowel cancer and pancreatic cancer. It is considered probable that there is no link between GL and breast cancer.

References

1. World Health Organization (WHO). Diet, nutrition and the prevention of chronic diseases. Geneva: WHO Technical Report Series 916, 2003.
2. World Cancer Research Fund/American Institute for Cancer Research. Food, Nutrition, Physical Activity, and the Preven-

- tion of Cancer: a Global Perspective. Washington DC: AICR, 2007.
3. Pisani P. Hyper-insulinaemia and cancer, meta-analyses of epidemiological studies. *Arch Physiol Biochem* 2008; 114: 63-70.
 4. Biddinger SB, Ludwig DS. The insulin-like growth factor axis: a potential link between glycemic index and cancer. *Am J Clin Nutr* 2005; 82: 277-8.
 5. Key TJ, Spencer EA. Carbohydrates and cancer: an overview of the epidemiological evidence. *Eur J Clin Nutr* 2007; 61 (Suppl. 1): S112-21.
 6. NHMRC. A review of the evidence to address targeted questions to inform the revision of the Australian Dietary Guidelines. Canberra (Au): National Health and Medical Research Council, 2011.
 7. Ruxton CHS, Gardner EJ, McNulty H M. Is Sugar Consumption Detrimental to Health? A Review of the Evidence 1995-2006. *Crit Rev Food Sci Nutr* 2010; 50: 1-19.
 8. Hauner H, Bechthold A, Boeing H, Brönstrup A, Buyken A, Leschik-Bonnet E, et al. Evidence-Based Guideline of the German Nutrition Society: Carbohydrate Intake and Prevention of Nutrition-Related Diseases. *Ann Nutr Metab* 2012; 60 (Suppl. 1): 1-58.
 9. Augustin LSA, Gallus S, Bosetti C, Levi F, Negri E, Franceschi S et al. Glycemic index and glycemic load in endometrial cancer. *Int J Cancer* 2003; 105: 404-7.
 10. Chatenoud L, La Vecchia C, Franceschi S, Tavani A, Jacobs DR Jr, Parpinel MT, Soler M, Negri E. Refined-cereal intake and risk of selected cancers in Italy. *Am J Clin Nutr* 1999; 70: 1107-10.
 11. Tasevska N, Jiao L, Cross AJ, Kipnis V, Subar AF, Hollenbeck A, et al. Sugars in diet and risk of cancer in the NIH-AARP Diet and Health Study. *Int J Cancer* 2012; 130 (1): 159-69.
 12. Gallus S, Turati F, Tavani A, Polesel J, Talamini R, Franceschi S, La Vecchia C. Soft drinks, sweetened beverages and risk of pancreatic cancer. *Cancer Causes Control* 2011; 22 (1): 33-9.
 13. Barclay AW, Petocz P, McMillan-Price J, Flood VM, Prvan T, Mitchell P, Brand-Miller JC. Glycemic index, glycemic load, and chronic disease risk—a metaanalysis of observational studies. *Am J Clin Nutr* 2008; 87: 627-37.
 14. Gnagnarella P, Gandini S, La Vecchia C, Maisonneuve P. Glycemic index, glycemic load, and cancer risk: a meta-analysis. *Am J Clin Nutr* 2008; 87: 1793-801.
 15. Bao Y, Nimptsch K, Wolpin BM, Michaud DS, Brand-Miller JC, Willett WC et al. Dietary insulin load, dietary insulin index, and risk of pancreatic cancer. *Am J Clin Nutr* 2011; 94: 862-8.
 16. Aune D, Chan DSM, Vieira AR, Navarro Rosenblatt DA, Vieira R, Greenwood DC et al. Dietary fructose, carbohydrates, glycemic indices and pancreatic cancer risk: a systematic review and meta-analysis of cohort studies. *Ann Oncol* 2012; 23: 2536-46.
 17. Mulholland HG, Murray LJ, Cardwell CR, Cantwell MM. Dietary glycemic index, glycemic load and breast cancer risk: a systematic review and meta-analysis. *Br J Cancer* 2008; 99: 1170-5.
 18. Romieu I, Ferrari P, Rinaldi S, Slimani N, Jenab M, Olsen A et al. Dietary glycemic index and glycemic load and breast cancer risk in the European Prospective Investigation into Cancer and Nutrition (EPIC). *Am J Clin Nutr* 2012; 96 (2): 345-55.
 19. Patterson R, Flatt S, Saquib N, Rock CL, Caan BJ, Parker BA et al. Medical comorbidities predict mortality in women with a history of early stage breast cancer. *Breast Cancer Res Treat* 2010; 122: 859-65.
 20. Erickson K, Patterson RE, Flatt SW, Natarajan L, Parker BA, Heath DD, Laughlin GA, Saquib N, Rock CL, Pierce JP. Clinically defined type 2 diabetes mellitus and prognosis in early-stage breast cancer. *J Clin Oncol* 2011; 29 (1): 54-60.
 21. Galeone C, Pelucchi C, La Vecchia C. Added sugar, glycemic index and load in colon cancer risk. *Curr Opin Clin Nutr Metab Care* 2012; 15 (4): 368-73.
 22. Aune D, Chan DS, Lau R, Vieira R, Greenwood DC, Kampman E, Norat T. Carbohydrates, glycemic index, glycemic load, and colorectal cancer risk: a systematic review and meta-analysis of cohort studies. *Cancer Causes Control* 2012; 23 (4): 521-35.

The importance of sucrose for cognitive functions; knowledge and behaviour

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Abstract

Sucrose is not present in the internal milieu as such, so it is physically impossible that it may have a direct influence on cognitive functions, behaviour and knowledge. However, during the digestive process, disaccharides are released into monosaccharides, in the case of sucrose into glucose and fructose, which reach the liver via the portal vein. Finally, they go into bloodstream in the form of glucose and in some cases as very low-density lipoproteins (VLDL).

Brain needs almost exclusively a constant supply of glucose from the bloodstream. Adult brain requires about 140 g of glucose per day, which represents up to a 50% of total carbohydrates consumed daily in the diet.

The consumption of a food or beverage enriched with sucrose has been associated with improved mental alertness, memory, reaction time, attention and ability to solve mathematical problems, as well as a reduction in the feeling of fatigue, both in healthy individuals and patients with Alzheimer disease.

An adequate nutrition of brain contributes to structural and functional integrity of neurons. It has been shown that in major mental illnesses such as schizophrenia, depression and Alzheimer's disease, nutritional deficiencies at cellular level are implicated.

At present, several studies highlight the need to improve understanding of the processes involved in the deterioration of cognitive functions and mechanisms through which, the nutritive components of the diet, particularly the sucrose, may modulate such functions.

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Key words: *Sucrose. Cognitive functions. Knowledge. Behavior. Memory.*

IMPORTANCIA DE LA SACAROSA EN LAS FUNCIONES COGNITIVAS; CONOCIMIENTO Y COMPORTAMIENTO

Resumen

La sacarosa no se encuentra en el medio interno, por lo tanto, es materialmente imposible que pueda influir directamente sobre las funciones cognitivas, el comportamiento y el conocimiento. No obstante, durante el proceso digestivo, los disacáridos se escinden en los monosacáridos correspondientes, en el caso de sacarosa en glucosa y fructosa que, por la vía portal llegarán al hígado. Finalmente, salen al torrente sanguíneo en forma de glucosa y en algún caso, además, como lipoproteínas de muy baja densidad (VLDL).

El cerebro precisa casi exclusivamente un suministro constante de glucosa desde el torrente sanguíneo. El cerebro adulto utiliza aproximadamente 140 g de glucosa al día, cantidad que puede representar hasta el 50% del total de los carbohidratos que se consumen.

El consumo de una comida o bebida con sacarosa se ha asociado con una mejora de la agilidad mental, la memoria, el tiempo de reacción, la atención y la capacidad para resolver problemas matemáticos, así como con una reducción de la sensación de cansancio, tanto en individuos sanos como en enfermos de Alzheimer.

La adecuada nutrición del cerebro mantiene la integridad estructural y funcional de las neuronas. Se ha demostrado que en las enfermedades mentales mayores, como la esquizofrenia, depresión y demencia de Alzheimer, hay deficiencias nutricionales a nivel celular.

En el momento actual, los estudios realizados ponen de manifiesto la necesidad de profundizar en el conocimiento de los procesos implicados en el deterioro de las funciones cognitivas y en los mecanismos, a través de los cuales, los componentes nutritivos de la dieta, y particularmente la sacarosa, pueden modularlos.

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Palabras clave: *Sacarosa. Funciones cognitivas. Conocimiento. Memoria.*

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Introduction

Digestible carbohydrates, both slow absorption, mainly starch, as well as fast absorption, monosaccharides and disaccharides (glucose, fructose and galactose; and lactose, maltose and sucrose respectively) are present in natural form. Furthermore, the food industry adds some of these carbohydrates to food during processing and elaboration, as they carry out important roles. Simple carbohydrates have an energy value of (4 kcal/g) and a sweetening power. They offer very important organoleptic properties, improving the product's consistency and texture, or giving it colour and flavour after thermal treatment. Finally, it has been and continues to be used as a preservation method.

In the case of table sugar or sucrose, it has been said that it is different to that found in foods which contain it, which is a clear error, sucrose is a chemically pure substance which has only been separated from other components which exist in the plant from which it has been extracted, sugar or beet, and is,

therefore, identical to that which exists in natural form in fruit.

From a nutritional point of view, the only drawback that can be attributed to sucrose is that, as it is a chemically pure product it only provides energy and no other nutrients¹.

The idea of some "experts" that sucrose can provoke some form of addiction in the same way as illegal drugs and, therefore, should be included in the list of addictive substances², is an opinion that has little scientific proof. The experimental evidence does not support the claim that sugar and other highly palatable foods produce addiction³. It is important to qualify that sucrose forms part of foods that produce pleasure but not dependence⁴.

Other effects that have been attributed to sucrose have been that they produce hyperactivity and aggression in children. However, different writers and a report by FAO/OM Sydicate that as well as not provoking such effects, they can, in fact have a sedentary effect⁵. Finally, Benton (2007)⁶, in his review of the effects of diet on behaviour, stated that sucrose had no negative effects on behaviour.

Table I
Classification of dementias in function of their aetiology

<i>Aetiology</i>	<i>Type of changes</i>
<i>Of degenerative origin</i>	Alzheimer Lewy diffuse bodies dementia Frontotemporal dementia Pick's disease Huntington's disease Dementia associated with Parkinson's Progressive supranuclear paralysis
<i>Vascular dementia</i>	Multi-infarct dementia Small blood vessels (lacunar, micro infarcts, leukoencephalopathy (Binswanger disease) Strategic infarcts Haemorrhages Hypoxia, hypoperfusion
<i>Of infectious origin</i>	Neurosyphilis Associated to AIDS Creutzfeldt disease Lyme disease Herpes virus encephalitis
<i>Of a metabolic or nutritional origin</i>	Hiccups or hyperthyroidism Hiccups and hyperparathyroidism, adrenal pituitary Kidney failure Hepatic failure Wilson's disease Vitamin B12 deficit Folic acid deficit Pellagra
<i>Of a toxic origin</i>	Associated with alcohol: alcohol dementia, Korsakoff and Marchiafava-Bignani disease Other toxic substances: aluminium, arsenic, bismuth, lead, etc.
<i>Of a neoplastic origin</i>	Primary and metastatic brain tumours Limbic encephalitis Carcinomatous meningitis

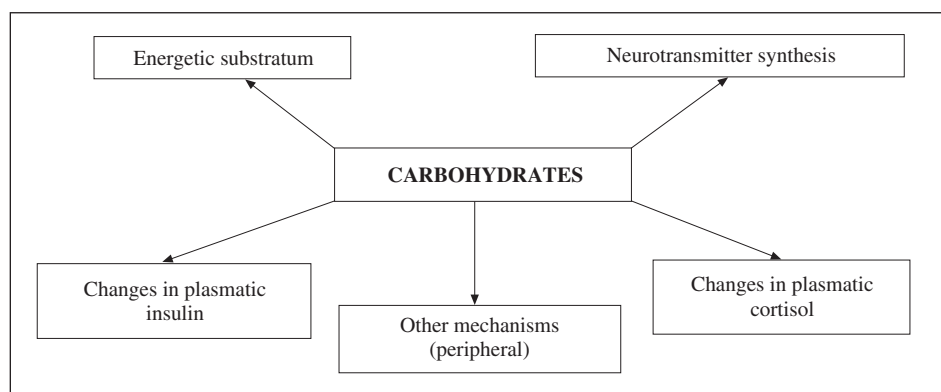


Fig. 1.—Carbohydrates action mechanisms on cognitive functions.

There is a growing worry for the increase in prevalence of changes associated with cognitive function. It is estimated that the number of cases of dementia will double every 20 years in developed countries, and will multiply by three in developing countries in the same time period. In the year 2040, it is calculated there will be more than 80 million people affected by these neurological illnesses of different origins (Table I)⁷.

The close relationship between the nutritional state and the function of the central nervous system evidences the importance of the diet models as a determining factor in the capacity of cognitive functions. So the deficiencies in numerous micronutrients show neurological manifestations, fats, and particularly saturated fats and their “trans” configuration, can negatively influence cognitive function, while carbohydrates, simple and complex could have the opposite effect.

In figure 1 it shows possible action mechanisms through which carbohydrates can act upon cognitive functions⁸.

Next we describe according to the DAFO analysis, some of the most impressive aspects and mechanisms, through which sucrose and other simple carbohydrates act upon cognitive functions.

Weaknesses

Sucrose is not found internally, and therefore, it is materially impossible for it to influence directly on cognitive functions, behaviour and knowledge. However, during the digestive process, disaccharides, such as sucrose, are absorbed in the enterocytes of the brush border of the small intestine, where, the corresponding disaccharides, in this case, the sucrose, break up the molecule splitting it into the corresponding monosaccharides, fructose and glucose, which reach the liver. The fructose will be converted into glucose if the person is hypoglycaemic, something not very likely if they have ingested sucrose, or triglycerides if hypoglycaemic or euglycaemic. The glucose will come from the liver, which, through systematic circulation,

will reach the different tissue. It is worth referring, once again, to the inadequacy of prescribing fructose to a diabetic as it is clear it does not increase blood sugar: fructose cannot be converted into glucose during hyperglycaemia, and continues on its way to being transformed into triglycerides (Fig. 2), and these leave the kidney in the form of very low density lipoproteins (VLDL), producing dyslipidaemia, which constitutes one of the side effects of diabetes.

The cells are dependent on and they all obtain their energy from glucose. Furthermore, some of them, in particular neurons, the only substrata that can obtain it is from this monosaccharide, a situation that it shares with red blood cells. This means to say that nerve cells need glucose to adequately carry out their functions; it is true though that, in extreme conditions they can obtain energy from other substrate such as lactate.

Direct diagnostics through imaging methods used to value the relationship between the ingestion of nutrients and cognitive functions are at the moment expensive and difficult to interpret. Furthermore, the evaluation of said relationship through indirect methods; based on the application of different tests, offer an interpretation of the result with notable limitations. At the same time, the easy application and interpretation biomarkers are insufficient and are low cost for this type of study⁹.

Currently, there is no effective therapy that reverses the symptoms caused by cognitive deterioration. Alternatively, the evidence of the effect of carbohydrates on cognitive functions is scarce and inconclusive.

Threats

There is a widely held opinion now for decades which suggests table sugar is responsible for a multitude of sins that threaten humans, even going as far as to consider it a poison, and was considered as much in some articles that referred to the four white poisons, and included sugar, salt, flour and milk. This is information that lacks any scientific and critical sense, apart from being completely false. But the reality is that this

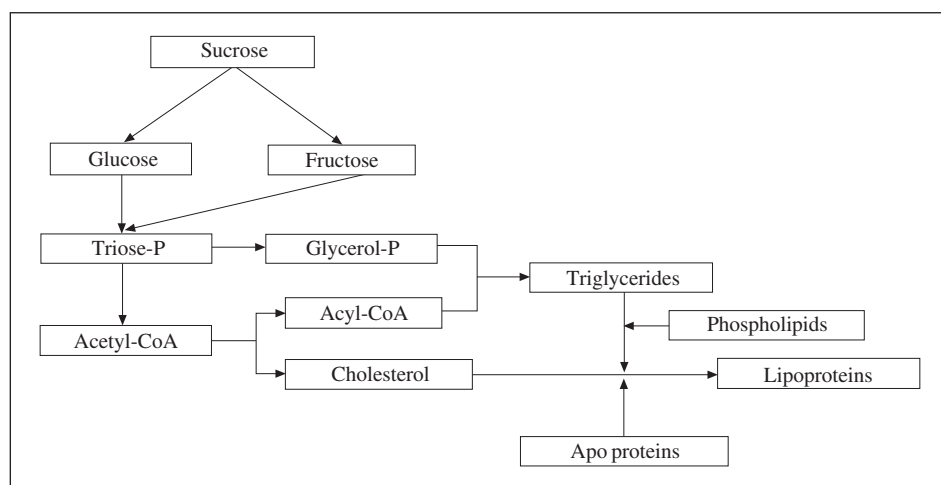


Fig. 2. —Metabolic fate of sucrose during a hypoglycaemic or euglycaemic episode.

idea is widely believed and generates great confusion amongst the general population.

All the energy that comes from ingested foods, solid or liquid, which exceeds an individual's needs, will be converted into triglycerides, which shall accumulate or be turned into fatty tissue deposits, independently of if the energy comes from carbohydrates, lipids or proteins. In a hypoglycaemic situation it is impossible for triglycerides to form, and what would increase in the bloodstream would be fatty acids, which would produce a serious dyslipidaemia with acidosis.

An incorrect control of the blood sugar has a close relationship with the deterioration of cognitive functions in diabetic patients. Furthermore, the presence of insulin and its receptors in different areas of the brain related with memory and learning (cortex and hippocampal), suggests that changes in concentration of said hormone can produce a deterioration of cognitive functions¹⁰.

The appearance of non evidence based and frequently confusing information related to beneficial aspects of sucrose on cognitive functions, can lead to an excessive consumption of this disaccharide which can be associated with the development of obesity, diabetes and metabolic syndrome, which can increase the risk of cognitive deterioration.

Taking part in physical activity, especially anaerobic, intense and or long duration, provokes fatigue as a consequence of depletion of glycogen in the liver and muscles, which can alter the cognitive state of the individual¹¹.

It has been suggested that certain components in foods amongst which carbohydrates and lipids are found, as well as the association among them, could be addictive. However, it has been pointed out that the addiction could be a phenotypic characteristic of obesity¹².

As a consequence of progressive ageing of the population and an increase in life expectancy, an increase in

the prevalence of neuro-degenerative illnesses is expected.

Studies of neuro-degenerative illnesses require long monitoring periods, making obtaining conclusive results difficult. Alternatively, the scientific bibliography shows inconsistencies and occasionally contradictory results of the effect that the ingestion of sucrose has over the cognitive functions. Furthermore, there is a great lack of knowledge regarding the processes and factors implicated in the recuperation or regeneration of the cognitive functions.

As in other fields, the field of neurosciences has produced a reduction in the resources allocated to research.

Strengths

The adequate nutrition of the brain helps maintain the structural and functional integrity of the neurons. It has been proved that in serious mental illnesses such as schizophrenia, depression and Alzheimer there exist nutritional deficiencies on a cellular level.

The consumption of food or drink with sucrose is associated with an improvement in mental agility, memory, reaction times, attention and the capacity to resolve mathematical problems, as well as a reduction in the sensation of tiredness, both in young and old healthy people and Alzheimer patients,

Other studies have shown that individuals that consume sugary drinks before and during the tests in a driving simulator made fewer errors in comparison with those who had only drunk water¹³.

The administration of oral sucrose solutions has been proved as a safe and effective treatment to combat sharp pains that cause some clinical procedures, both in healthy and sick children¹⁴.

Sports drinks that contain sugar, minerals and water, avoid dehydration, the depletion of glycogen reserves and slow down the appearance of fatigue, due to the sugar

that, in the first instance delivers glucose directly to the muscle and this lengthens the duration of the exercise¹⁵.

Not only is the ingestion of complex carbohydrates effective in promoting synthesis of muscular glycogen, as was initially believed, simple carbohydrates also produce similar increases in storage of it.

Recent advances in radio diagnostics, methodology applied to the knowledge of the organisation and function of the brain, have contributed to the design of studies that improve the understanding of the molecular make-up of the conduct⁹.

The prevention of the deterioration of the cognitive functions will contribute, without any doubt, to the promotion of autonomy, the mood and the quality of life of the elderly population.

Opportunities

In recent years, nutritional investigations have been centred on the long term evaluation of nutrients on cerebral functions, both in the promotion of neuron development and the prevention of age related cognitive deterioration.

Carbohydrates are important for the adequate functioning of the organism. The adult brain uses approximately 140 g of glucose per day; an amount, which can represent up to 50% of the total carbohydrates that are consumed. The brain requires a constant supply of glucose from the blood flow.

It is generally accepted that the ingestion of sucrose improves the short-term knowledge and memory while favouring concentration¹⁶.

The scientific advances in the knowledge of cognitive deterioration and of neurosciences in general, have without doubt, enabled the design and implementation of nutritional therapies that fight against the degenerative disorders derived from cerebral ageing.

Individuals that have a higher inclusion of the Mediterranean diet present a lower rate of Alzheimer. This coincides with the universally accepted idea that following this kind of diet brings high levels of general health and longer and healthier life¹⁷.

Due to the increase in the prevalence of neurological illnesses, and the high cost of treatment, any therapeutic intervention that slows down the increase of these disorders will have an enormous impact, not only on patients but also in the social and health care arena.

The growing interest of the food and pharmacological industries in the design of new products can have a function in the prevention and treatment of neurological disorders.

Recommendations

Sucrose has been throughout history and continues to be an important source of energy in the human diet.

The excessive consumption of disaccharides can replace other food in the diet and produce nutritional deficiencies and, therefore, have undesired consequences, issues that have been addressed in detail in previous chapters.

Sucrose, when included in a measured way in the diet, has important properties, it favours quick release glucose to the brain and muscles, being an essential carbohydrate for the development of cognitive functions and of physical activity. To avoid a rapid rise in blood sugar, which would implicate the formation and liberation of elevated quantities of insulin, the rest of the glucose necessary should be provided by starch. The organism does not differentiate, metabolically speaking, between glucose that comes from starch and that from sucrose.

What is truly important is to avoid hypoglycaemic situations, which would be responsible, amongst other undesired effects for glycosylation of proteins, which has also been addressed in previous chapters.

It is still premature to try and establish, in relation to cognitive functions, recommendations for the ingestion of sucrose for preventative and therapeutic ends. It is important to follow a balanced and varied diet, or as mentioned before, sticking to a Mediterranean diet, both for general health and to achieve a longer and healthier life.

Conclusions

The close relationship between the nutritional status and the function of the central nervous system evidences the importance of diet as one of the determining factors in cognitive function. In recent years the research into nutrition has been centred on the long-term effects of nutrients on cerebral functions. The adequate nutrition of the brain maintains the structural and functional integrity of the neurons. It has been proved that in serious mental illnesses such as schizophrenia, depression, and Alzheimer there exists nutritional deficiencies on a cellular level.

There is a growing concern for the increase in the prevalence of disorders associated with cognitive function. As a consequence of the progressive ageing of the population and the increase in life expectancy, an increase in the incidence of neuro-degenerative illnesses is expected. In fact, it is estimated that in the year 2040 there will be more than 80 million people affected by this type of illness.

Carbohydrates are important for the adequate functioning of the organism, and in particular for the brain, as the neurons, in order to maintain their integrity and functionality, need a constant source of glucose from the bloodstream (140 g/day).

It is generally accepted that the ingestion of sucrose improves, in the short term, knowledge and memory, which on a par favours concentration. Different

studies have shown that the consumption of food or drink that contains sucrose is associated with an increase in mental agility, memory, reaction times, the attention and ability to resolve mathematical problems, as well as a reduction in the sensation of tiredness, both in healthy individuals and Alzheimer patients. However, in scientific literature in consistencies and contradictory results can be found regarding the effects of the ingestion of sucrose on cognitive functions, for which it would be necessary to go deeper into knowing the effects of this disaccharide on the brain.

The prevention of the deterioration of cognitive functions will contribute, without any doubt, to the promotion of autonomy, mood and to the quality of life of the elderly population. Furthermore, due to the increasing prevalence of neurodegenerative illnesses and the high costs of treatment, any therapeutic intervention that slows the increase of these disorders shall have an enormous impact not only on the patients but also in the social and healthcare arena.

References

1. Grande F. El azúcar en la alimentación humana. Serie de divulgación nº 7. Madrid: Fundación Española de Nutrición (FEN). 1986.
2. Gearhardt, Ashley N; Grilo, Carlos M; DiLeone, Ralph J; Brownell, Kelly D; Potenza, Marc N. Can food be addictive? Public health and policy implications. *Addiction* 2011; 106 (7): 1208-11.
3. Ziauddeen H, Farooqi IS, Fletcher PC. Obesity and the brain: how convincing is the addiction model? *Nature Reviews Neuroscience* 2012; 13: 279-86.
4. Benton D. The plausibility of sugar addiction and its role in obesity and eating disorders. *Clinical Nutrition* 2010; 29: 288-303.
5. FAO/OMS. Reunión conjunta sobre los carbohidratos en la nutrición Humana. Roma: OMS. 1995.
6. Benton D. Review. The impact of diet on anti-social, violent and criminal behaviour. *Neuroscience and Behavioural Reviews* 2007; 31: 752-74.
7. Ferri CP, Prince M, Brayne C, Brodaty H, Fratiglioni L, Ganguli M, Hall K, Hasegawa K, Hendrie H, Huang Y, Jorm A, Mathers C, Menezes PR, Rimmer E, Sczufca M. and Alzheimer's Disease International. Global prevalence of dementia: a Delphi consensus study. *Lancet* 2005; 366: 2112-17.
8. Ooi C, Loke S, Yasiin Z, Hamid T. Hidratos de carbono para mejorar el rendimiento cognitivo de los adultos mayores que viven de forma independiente con función cognitiva normal o con deficiencia cognitiva leve. (Revision Cochrane traducida). Cochrane Database of Systematic Reviews 2011 Issue 4. Art. No.: CD007220. DOI: 10.1002/14651858.CD007220.
9. Silverman DHS, Alavi A. PET en la valoración de la función cognitiva normal y alterada. *Radiol Clin N Am* 2005; 43: 67-78.
10. Woods SC, Seeley RJ, Baskin DG, Schwartz MW. Insulin and the blood-brain barrier. *Curr Pharm Des* 2003; 9 (10): 795-800.
11. Rennie KL, Livingstone MB. Associations between dietary added sugar intake and micronutrient intake: a systematic review. *Br J Nutr* 2007; 97 (5): 832-41.
12. Corsica JA, Pelchat ML. Food addiction: true or false? *Curr Opin Gastroenterol* 2010; 26 (2): 165-9.
13. Sünram-Lea SI, Foster JK, Durlach P, Pérez C. Glucose facilitation of cognitive performance in healthy young adults: examination of the influence of fast-duration, time of day and pre-consumption plasma glucose levels. *Psychopharmacology (Berl)* 2001; 157 (1): 46-54.
14. Margaret Harrison DM. Oral sucrose for pain management in infants: Myths and misconceptions. *Journal of Neonatal Nursing* 2008; 14: 39-46.
15. Malik VS, Schulze MB, Hu FB. Intake of sugar-sweetened beverages and weight gain: a systematic review. *Am J Clin Nutr* 2006; 84 (2): 274-88.
16. Schmitt JAJ. Nutrition and cognition: meeting the challenge to obtain credible and evidence-based facts. *Nutrition Reviews* 2010; 68 (Suppl. S1): S2-S5.
17. Scarmeas N, Stern Y, Mayeux R, Manly JJ, Schupf N, Luchsinger JA. Mediterranean Diet and Mild Cognitive Impairment. *Arch Neurol* 2009; 66 (2): 216-25.