Dietary sugars, metabolic effects and child health

Azahara Iris Rupérez[a,*], *María Isabel Mesana*[a,*], *and Luis Alberto Moreno*[a,b]

Purpose of review
To describe current findings on sugar intake in children worldwide, including sugar sources and their impact on child health focusing on cardiometabolic alterations usually associated to obesity.

Recent findings
In children less than 4 years, intakes of added sugars across countries ranged from 9.8 to 11.2% of total energy; in children 4–10 years, it ranged from less than 3–18%; and in adolescents, it ranged from 13.6 to 16.6%. For most countries, intakes of added sugars were greater than the recommended upper limit of 10% of total energy for children and adolescents and less or around 10% in infants. In most studies, soft drinks and fruit-based drinks accounted for the greatest proportion of the added sugars intake, followed by milk products and sweet bakery products. High added sugar intake has been associated with increased obesity risk and fat deposition in the liver, contributing to dyslipidemia, high blood pressure, insulin resistance and cardio-metabolic risk.

Summary
As a high added sugar intake is associated with cardio-metabolic conditions in children and adolescents, the current scenario supports the need for stronger targeted long-term policies that prevent the excessive sugar intake in young populations.

Keywords
cardiometabolic complications, children, dietary sugars, obesity

INTRODUCTION

There is an important amount of information on the effects of sugar consumption on obesity and its associated cardiometabolic risk [1▪▪]. Given the importance of the negative consequences of a high added sugar intake, the WHO recently recommended that, for avoiding obesity development, the consumption of free or added sugars should not exceed 10% of total daily energy intake [2]. However, the evidence base for the recommendation was weak and the recommendation was conditional. In spite of this, added sugars are still widely present in the diet of infants, children and adolescents [3]. The aim of this review was to gather the current available information on dietary sugar consumption in infants, children and adolescents and on its potential health effects, with a special focus on obesity and cardiometabolic risk.

DEFINITIONS OF DIETARY SUGARS

The term 'total sugars' is used to describe the monosaccharides, glucose, galactose, and fructose, as well as the disaccharides, sucrose, lactose, maltose, and trehalose. Total sugars include all sugars in a food or beverage from any source, including those naturally occurring and those added to foods. Added sugars are defined as all monosaccharides and disaccharides added to foods by the manufacturer, cook and consumer. The WHO also suggests using the term 'free sugars,' defined as added sugars along with sugars naturally present in honey, syrup and unsweetened fruit juices [2]. Extrinsic sugars are those not located within the cellular structure of food, and can be divided into milk sugars and non-milk extrinsic sugars (NMES), which is synonymous to free sugars. Recent publications recommend that the term 'free sugars' should be adopted worldwide,

[a]Growth, Exercise, Nutrition and Development (GENUD) Research Group, University of Zaragoza, Food and Agriculture Institute of Aragón (IA2), Health Research Institute of Aragón (ISS Aragón), Zaragoza and [b]CIBER Obesity and Nutrition Physiopathology (CIBEROBN). Madrid, Spain

Correspondence to Luis Alberto Moreno, MD, PhD, Facultad de Ciencias de la Salud, University of Zaragoza, C/Domingo Miral s/n, 50009, Zaragoza, Spain. Tel: +34 976 761000 x4457;
e-mail: lmoreno@unizar.es

*Azahara Iris Rupérez and María Isabel Mesana contributed equally to the writing of this article.

Curr Opin Clin Nutr Metab Care 2019, 22:206–216
DOI:10.1097/MCO.0000000000000553

KEY POINTS

- In infants, children and adolescents, added sugar intake equals or exceeds the 10% of total energy recommendation.
- Soft drinks, fruit-based drinks, milk products and sweet bakery products are the greatest contributors of added sugar intake.
- The excessive energy supply from added sugars and, specifically, from fructose, has been associated with obesity and its cardiometabolic consequences in children (insulin resistance, high blood pressure and dyslipidemia).
- The highest evidence on the deleterious health effects of added sugar intake derives from the consumption of sugar-sweetened beverages (SSB).
- The presence of added sugars in foods should be minimized in order to decrease added sugar consumption and its associated negative health effects.

instead of that of NMES [4]. Therefore, this review focuses mainly on added sugars/free sugars and their association with obesity and cardiometabolic complications in children and adolescents.

SUGAR INTAKE AND FOOD SOURCES

Several dietary surveys in both developed and developing countries have reported sugar intake and its food sources. In the present review, the most recent data collected from epidemiological dietary surveys, identified through searches on PubMed and Internet using the key words 'sugar intake' or 'diet' or 'added sugar intake' or 'free sugar intake,' were considered.

Data are presented as provided in the reports and publications and divided by age range. Values are reported for estimated intakes of energy, total sugars, added sugars, NMES, free sugars or sucrose, as available.

Thirty-one dietary surveys, of which 19 have been recently reviewed [5▪▪–8▪▪], providing data on total and/or added sugars or NMES or free sugars have been identified in 26 countries and two European multicenter studies. The identified surveys differed by several parameters, such as the age range of the considered populations, the dietary data collection methods or the year of the survey (Table 1). Only food and beverages categories that contributed significantly to sugar intakes were considered. In Table 2, sugar intake of children and adolescents of the different studies are presented, expressed as total sugar, added sugar, free sugar or sucrose, in grams and percentages of total energy (%E).

Intake of sugars across countries in infants and young children (<4 years)

For infants and young children, sugar consumption data are reported in 15 studies, including intakes of total sugars (11 studies), added sugars (3 studies), free sugars (4 studies) and sucrose (2 studies; Table 1). Total sugar intakes expressed as %E ranged from 25.2% in Australia to 28.5% in Sweden. Intakes of added sugars ranged from 9.8%E in Australia to 11.2%E in Iceland. Intake of free sugars ranged from 3.6%E to 11.5%E; both studies were conducted in Australia, but in infants (12–14 months) and young children (2–4 years), respectively. Intake of sucrose ranged from 11.2%E in Finland to 13.8%E in Sweden (Table 2).

Intake of sugars across countries in children (4–10 years)

For children aged 4–10 years, sugar consumption data are reported for 19 countries including intakes of total sugars (14 countries), added sugars (7 countries), free sugars (6 studies) and sucrose (4 studies) (Table 1). Total sugar intakes expressed as %E ranged from 17% in Italy to 27.7% in the Netherlands. Intakes of added sugars ranged from <3%E in China to 18%E in the Netherlands. Intakes of free sugars ranged from 9.8%E in Spain to 13.5%E in Australia. Intake of sucrose ranged from 11.5%E in Austria to 12.6%E in Sweden (Table 2).

Intake of sugars across countries in adolescents (11–18 years)

Sugars consumption data are reported for 21 studies including intakes of total sugars (14 studies), added sugars (12 studies), free sugars (4 studies) and sucrose (2 studies; Table 1). Total sugar intakes expressed as a %E ranged from 17.7% in Spain to 26.2% in the Netherlands. Intakes of added sugars ranged from 10.8%E in Ecuador to 17.8%E in Argentina. Intake of free sugars ranged from 9.6%E in Spain to 19%E in adolescents from the European HELENA study. Intake of sucrose ranged from 10.5%E in Austria to 12.3%E in Sweden (Table 2).

Contributors to intake of total sugars

Sweet products were major contributors to the intake of total sugars in all studies and age groups. The other important contributors were beverages and dairy products, but the ranking varied according to ages and studies. Within the beverages group, soft drinks and fruit juices were the main contributors. Within dairy products, milk and dairy beverages were the major contributors, especially in infants and children.

Table 1. Characteristics of surveys alphabetically ordered by country

Country	Reference	Survey	Year of survey	Sugar-intake information	Age range	Sample size[a]	Dietary data collection method
Australia	Devenish *et al.* [9▪]	Study of Mothers and Infants Life Events Affecting Oral Health	2013–2014	Total sugars Free sugars	12–14 m	828	1 × 24-h recall + 2-day record
Canada	Langlois and Garriguet [10]	Canadian Community Health Survey	2004	Total sugars	1 y and older	34 386	1 × 24-h recall
Europe (Greece, Germany, Belgium, France, Hungary, Italy, Sweden, Austria and Spain)	Mesana *et al.* [11▪]	HELENA (Healthy Lifestyle in Europe by Nutrition in Adolescence)	2006–2007	Total sugars	12.5–17.5 y	1630	2 × 24-h recalls
Europe (Belgium, Cyprus, Estonia, Germany, Hungary, Italy, Spain and Sweden)	Svensson *et al.* [12]	IDEFICS (Identification and prevention of dietary and lifestyle induced health effects in children and infants)	2007–2008	Total sugars Added sugars	2–9 y	9497	1 × 24-h recall
Finland	Kytälä *et al.* [13]	Type 1 Diabetes Prediction and Prevention	2003–2005	Total sugars Sucrose	1–6 y	2535	3-day record
France	ANSES, Ministère des Solidarités et de la Santé, Ministère de l'Agriculture et de l'Alimentation [14]	Étude individuelle nationale des consommations alimentaires 3 (INCA 3)	2014–2015	Total sugars	0–79 y	5855	3 × 24-h recall (15–79 y) 3-day diary (0–14 y)
Japan	Fujiwara *et al.* [15▪]		2013–2015	Total sugars	18 m to 69 y	2051	1–4-day record
Sweden	Enghardt *et al.* [16]	Riksmaten-barn Swedish children's dietary survey	2003	Total sugar Sucrose	8–9 y	889	4-day diary
UK	Roberts *et al.* [17]	National Diet and Nutrition Survey. Public Health England and the Food Standards Agency	2014/15 to 2015/16	Free sugars	1.5–64 y	2723	4 × 24-h recall
USA	Wang *et al.* [18▪]	NHANES 2009–2014	2009–2014	Added sugars Free sugars	0–5 y	3345	2 × 24-h recalls
USA	Bailey *et al.* [19▪]	NHANES 2011–2014	2009–2014	Added sugars	2 y-adults	16 806	2 × 24-h recall
Azaïs-Braesco *et al.* [5▪▪]							
Australia	Louie *et al.* [20]	Australian Health Survey	2007	Total sugars Added sugars Free sugars	2–16 y	4140	2 × 24-h recall
Austria	Elmadfa *et al.* [21]	Austrian nutrition report	2010–2012	Sucrose	7–14 y	1002	3-day diary (consecutive)
Ireland	Irish Universities Nutrition Alliance [22]	National Pre-School Nutrition Survey (NPNS)	2010–2011	Total sugars Nonmilk extrinsec sugar	1–4 y	500	4-day weighed food record
Italy	Sette *et al.* [23]	INRAN-SCAI survey	2005–2006	Total sugars	0.1–97.7 y	3323	3-day record
Netherlands	Sluik *et al.* [24]	Duth National Food Consumption Survey	2007–2010	Total sugars Added sugars Free sugars	7–69 y	3817	2 × 24-h recalls
Spain	Ruiz *et al.* [25]	ANIBES Study	2013	Total sugars Intrinsic sugars Free sugars	9–75 y	2009	3-day record

Table 1 (*Continued*)

Country	Reference	Survey	Year of survey	Sugar-intake information	Age range	Sample size[a]	Dietary data collection method
Rippin *et al.* [6▪▪]							
Belgium	De Ridder *et al.* [26]	Belgium national food consumption survey (BNFCS)	2014–2015	Total sugars	3–64 y	3146	2 × 24-hr recall
Denmark	Pedersen *et al.* [27]	Danish national survey of diet and physical activity (DANSDA)	2011-2013	Added sugar	4–75 y	3016	7-day diary
Germany	Mensink *et al.* [28]	EsKiMo - German Health Interview and Examination Survey for children and Adolescents	2003–2006	Total sugars	0–17 y	17641	3-day record
Iceland	Steingrimsdottir *et al.* [29]	National dietary survey	2010–2011	Added sugar	6 y	1312	2 × 24-h recall + FFQ. Telephone interview.
Norway	Hansen *et al.* [30]	UNGKOST 3	2015–2016	Added sugar	4–13 y	1721	4-day online diary + FFQ
Afeiche *et al.* [7▪▪]							
China	Zhang *et al.* [31]	China Health and Nutrition Survey	1989–2011	Total sugars Added sugars	4–13 y	1460	3 × 24-h recall
Mexico	Romero-Martínez *et al.* [32] Fisberg *et al.* [8▪▪]	Mexican National Health and Nutrition Survey	2012	Total sugars Added sugars	4–13 y	3985	1 × 24-h recall
Argentina	Ministerio de Salud de Argentina [33]	National Survey of Nutrition and Health–ENNyS	2004–2005	Total sugars Added sugars	6 m to 5 y and women 10–49 y	36354	1 × 24-h recall
Brasil	Instituto Brasileiro de Geografía e Estatística [34]	Household Budget Survey - POF	2008–2009	Total sugars Added sugars	≥ 10 y	34003	2 × 24-h recall
Chile	Ministerio de Salud de Chile [35]	National Food Consumption Survey – ENCA	2014	Total sugars Added sugars	≥2 y	4920	Quantitative FFQ + 1 × 24-h recall
Colombia	Fonseca *et al.* [36]	National Nutritional Situation Survey –ENSIN	2008–2010	Total sugars Added sugars	5–64 y	17897	FFQ
Ecuador	Freire *et al.* [37]	Ecuatorian National Health and Nutrition Survey - ENSANUT-ECU	2011–2013	Total sugars Added sugars	1–59 y	19932	1 × 24-h recall
Venezuela	Instituto Nacional de Estadística [38]	Encuesta de Seguimiento al Consumo de Alimentos – ESCA	2012–2014	Total sugars Added sugars	≥3 y	6316	Diet history + FFQ

[a]Sample size refers to the complete age range in the study. The included studies have the following characteristics: available in the public domain, summarized (no statistical analysis has been performed on these datasets, the results are purely descriptive), collected at the individual level and with no limit in the study sample size. FFQ, food frequency questionnaire; m, months; y, years.

Table 2. Sugar intake of infants, children and adolescents

Country	Reference	Age range	Total sugars		Added sugars		Free sugars		Sucrose	
			(g)	(%E)	(g)	(%E)	(g)	(%E)	(g)	(%E)
Australia	Devenish *et al.* [9▪]	12–14 m	65.3				8.8	3.6		
Canada	Langlois *et al.* [10]	1–8 y	83–172	24.1–27.1						
Europe	Mesana *et al.* [11▪]	12.5–17.5 y	137.5	23.6			110.1	19		
Europe	Swensson *et al.* [12]	2–9 y	97	26						
Finland	Kytälä *et al.* [13]	1–6 y	38–108	24.6–30.0					9.0–48.7	5.4–13.8
France	ANSES [14]	0–17 y	77.7–105.5	21.2–33.95						
Japan	Fujiwara *et al.* [15▪]	18 m to 14 y	46.1–68.7						18.2–34.0	
Sweden	Enghardt *et al.* [16]	8–9 y	116	25.6					57	12.6
UK	Roberts *et al.* [17]	1.5–18 y					32.6–62.4	11.3–14.4		
USA	Wang *et al.* [18▪]	0–5 y				10.1		13.9		
USA	Bailey *et al.* [19▪]	2–18 y			40–108	14.3–16.2				
Azaïs-Braesco V *et al.* 2017 [5▪▪]										
Australia	Louie *et al.* [20]	2 y and older	92.8–140.7	23.5–27.3	34.5–76.8	9.6–12.8	42.8–88.5	12.3–14.8		
Austria	Elmadfa *et al.* [21]	7–14 y								10.0–14.0
Ireland	Irish Universities Nutrition Alliance [22]	1–4 y	75.9				57.0			
Italy	Sette *et al.* [23]	3–10 y	86	17.0						
Netherlands	Sluik *et al.* [24]	7–18 y	125–153	24.4–28.0	75–100	15.1–18.4	89–113	17.6–20.7		
Spain	Ruiz *et al.* [25]	9–17 y		16.9–19.2				9.6–10.8		
Rippin HL *et al.* 2018 [6▪▪]										
Belgium	De Ridder *et al.* [26]	3–17 y		22.7–27.5						
Denmark	Pedersen *et al.* [27]	4–17 y			48–60					
Germany	Mensink *et al.* [28]	0–17 y	101–167	25.8–28.4						
Iceland	Steingrimsdottir *et al.* [29]	9 m to 3 y				11.2				
Norway	Hansen *et al.* [30]	4–13 y			49–69	11.8–12.9				
Afeiche MC *et al.* 2017 [7▪▪]										
China	Zhang *et al.* [31]	4–13 y	26		9	<3				
Mexico	Romero-Martínez *et al.* [32]	4–13 y	92		55	12				

Table 2 (*Continued*)

Country	Reference	Age range	Total sugars		Added sugars		Free sugars		Sucrose	
			(g)	(%E)	(g)	(%E)	(g)	(%E)	(g)	(%E)
Fisberg M *et al.* 2018 [8▪▪]										
Argentina	Ministerio de Salud de Argentina [33]	15–19 y	131.1	21.9	106.9	17.8				
Brasil	Instituto Brasileiro de Geografía e Estatística [34]	15–19 y	120.9	21.7	74.2	14.5				
Chile	Ministerio de Salud de Chile [35]	15–19 y	95.4	21.4	58.5	12.9				
Colombia	Fonseca *et al.* [36]	15–19 y	118.0	21.0	65.0	11.6				
Ecuador	Freire *et al.* [37]	15–19 y	105.7	18.8	59.6	10.8				
Venezuela	Instituto Nacional de Estadística [38]	15–19 y	104.8	20.3	72.2	14.1				

%E, percentage of total energy intake; m, months; y, years.

Contributors to intake of added, nonmilk extrinsic sugars and free sugars

The top food sources of added sugars were soft drinks, fruit-based drinks and sweetened milk-based beverages. In most studies, soft drinks and fruit-based drinks accounted for the greatest proportion of the added sugars intake, followed by milk products, sweet bakery products, sugar and spreads, chocolates and confectionary and cereals. For infants and young children, milk products and sweet bakery products contributed to the largest proportion of daily added sugar intake, but soft drinks and fruit-based drinks were the largest contributors of added sugar in older children and adolescents. When NMES or free sugars are considered rather than added sugars, fruit juices also became significant contributors.

In the last years, high-sugar-containing products oriented to infants and young children have been made available. Sucrose and glucose, which are sweeter than lactose (the sugar found in breast milk) have been added to some infant formulas. The habit of adding sugars to foods that are commonly perceived as healthy, such as yoghurt, milk or fruit, may impact negatively the adherence to a healthy dietary pattern [39,40]. Pureed, semi-liquid complementary foods packed in squeezable plastic pouches are also widely available, having a proportion of sugar up to 84–98% of its total energy content [41].

HEALTH EFFECTS

Starting as early as *in utero* and continuing into childhood and adolescence, the effects of a high sugar intake have been associated with higher obesity and cardiovascular disease risk [42▪▪] and with other adverse health consequences, such as dental caries. In this review, we present the most recent findings relative to the effect of added sugars on obesity, insulin resistance, hypertension and dyslipidemia.

Obesity

Although negative results have been found, there is abundant literature supporting a relationship between sugar consumption and obesity risk [42▪▪,43▪▪]. Among the sugar-containing foods and drinks, most evidence has been observed for sugar sweetened beverages (SSB) intake [44,45]. The most plausible explanation for this finding would be the smaller dietary compensation for liquid than solid foods, which have a higher satiating effect than liquids possibly because of their similarity with food

(ingested to overcome hunger) rather than with drinks (more frequently considered as thirst-quenching) [46].

Already *in utero*, free sugar consumption through SSB by pregnant women has been observed to increase the fat mass of their offspring at 6 years [47]. Similarly, a higher sugar content of breastmilk in the form of fructose has been associated with increased total body weight, fat mass, lean mass and bone mineral content of exclusively breastfed babies at 6 months of age [48]. Later in infancy, SSB consumption has been associated with later obesity in childhood as a high added sugar consumption as SSB in children of 1–5 years almost tripled their odds of having total and abdominal obesity at age 8–14 years [49]. In childhood and adolescence, SSB consumption has also been widely associated with greater body weight in cross-sectional and longitudinal studies [50,51].

Interventions aiming to decrease obesity though a decrease in sugar consumption have yielded positive results in children [52] and adolescents [53]. However, there are many factors affecting the effectiveness of these interventions, which should be carefully considered [54]. In particular, intervention studies aiming to reduce SSB consumption have been moderately successful in children and only little reductions have been observed in adolescents [55▪,56].

Insulin resistance

Although evidence is still developing, there is an association between added sugar intake and different markers of insulin resistance in children [43▪▪,57▪], with recent works supporting a positive association between SSB intake and blood glycemia in children [58▪]. Interestingly, some studies in children and adolescents show an association between added sugar consumption and insulin resistance only when the added sugars considered are exclusively of liquid source.

In addition, obesity and insulin resistance have shown stronger associations with each other as well as with blood pressure (BP), when accompanied by a high SSB consumption. Among children with a high SSB consumption, those with obesity showed higher HOMA-IR and SBP; those with impaired glucose tolerance showed higher SBP and waist circumference; with no differences observed in children with normal weight [59]. Similar findings were observed in adolescents, in which HOMA-IR increased along with added sugar intake only in those with the highest BMI [60].

Hypertension and dyslipidemia

Recent studies have shown a positive association between added sugar intake and blood pressure in children and adolescents [1▪▪], with negative findings in others [45]. In summary, there was an association between a high added sugar intake and low high-density lipoprotein cholesterol (HDL-C) levels and high triglyceride levels in children with, but most commonly in boys. SSB consumption at the age of 1 year has been associated with a higher cardiometabolic risk score (high body fat percentage, BP, insulin, and triglycerides and low HDL-C) at 6 years, but only in boys [61]. Added sugar consumption has been associated with increased triglycerides levels in children and adolescents 8–16.9 years [57▪,62]. Several studies in older children and adolescents have reported similar findings that relate SSB consumption with cardiometabolic markers, such as high HOMA-IR, BP or triglyceride levels and low HDL-C concentrations in different populations [57▪]. In contrast, consumption of added sugars lower than 10%E intake has been associated with an increase of HDL-C concentrations in female adolescents [1▪▪]. However, recent meta-analyses have shown that these findings do not have the sufficient consistency among available published results [63].

PHYSIOLOGIC MECHANISMS

The main and most plausible reason underlying the relationship between sugar consumption and cardiometabolic risk is its associated increased energy intake. However, fructose, in particular, has been proposed as one of the main actors in the orchestra of cardiometabolic complications originated by sugar consumption [64▪▪,65▪,66▪▪] (Fig. 1). Although this hypothesis is not free of controversy, associations have been observed while adjusting by body weight, indicating a potential direct relationship between fructose intake and cardiometabolic alterations.

After being ingested, fructose has an effect in the gastrointestinal tract. Its uptake by the enterocytes is increased when given in form of sucrose or in combination with glucose (1 : 1). Moreover, a facilitated absorption mechanism has been proposed in mice in which fructose stimulates the expression of the fructose transporter GLUT-5 [67▪▪]. Indeed, an increased fructose absorption has been associated with obesity and nonalcoholic fatty-liver disease (NAFLD) in children [Fig. 1]. When fructose reaches the liver, its metabolism is not subject to regulation and it is constantly degraded into glyceraldehyde-3-P, reducing adenosine triphosphate (ATP) levels,

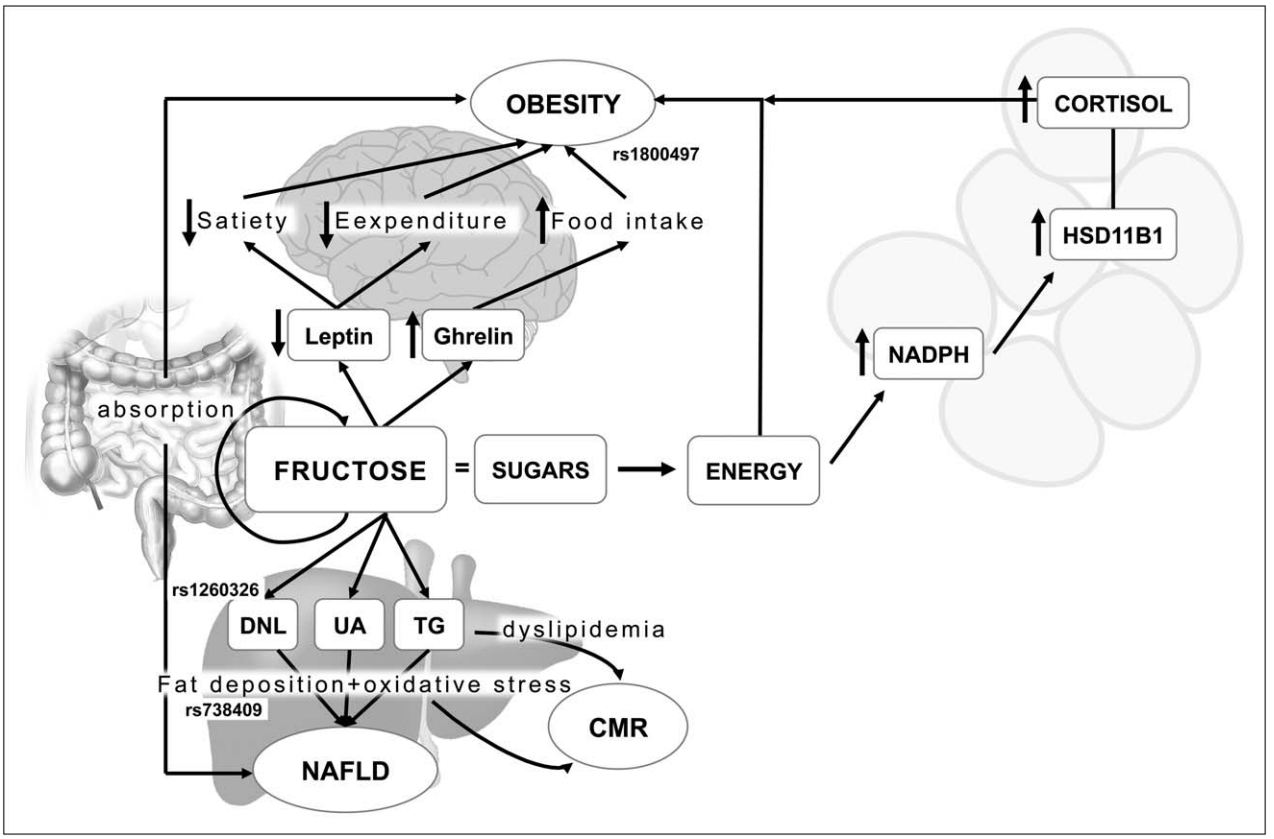

FIGURE 1. Potential mechanisms of the cardiometabolic effects of sugar intake. High sugar/fructose intake has an impact at different levels in the human body, such as the intestine, the liver, the central nervous system and adipose tissue. Relevant genetic variants affecting specific metabolic effects of sugar intake in children are indicated with their corresponding rs code. CMR, cardiometabolic risk; DNL, de novo lipogenesis; HSD11B1, 11β-hydroxysteroid dehydrogenase 1; NADPH, nicotinamide adenine dinucleotide phosphate; NAFLD, nonalcoholic fatty-liver disease; TG, triglycerides; UA, uric acid.

increasing uric acid production from adenosine monophosphate (AMP) and activating glycolysis [68▪]. All this favors an excessive production of lipids through de novo lipogenesis and triglyceride synthesis in the liver, which alters the lipid profile and promotes oxidative stress, contributing to cardiometabolic risk, NAFLD and atherosclerosis [64▪▪,69,70] (Fig. 1). Fructose's dyslipidemic effect has been observed in children; and fructose restriction has been observed to decrease DBP, triglyceride and insulin resistance [67▪▪]. Although uric acid has been proposed as a promoter of this vicious cycle by contributing to endogenous fructose synthesis [67▪▪], its implication in cardiometabolic risk is not fully clear [71].

The effect of fructose on insulin resistance has not yet been mechanistically investigated in children. However, studies conducted *in vitro* or in animal models have suggested the implication of several pathways, including an increased insulin secretion, an increased production of advanced glycation end-products (AGEs) [72▪], a reduction of endothelial NO, endoplasmic reticulum stress and an increased lactate release to the bloodstream that would be used by skeletal muscle instead of glucose, contributing to hyperglycemia [66▪▪].

Fructose also originates a lower leptin secretion and a higher ghrelin secretion than glucose in the adipose tissue and in the gastrointestinal tract, respectively [73]. Given leptin's anorexigenic and ghrelin's orexigenic roles, this situation leads to a decreased satiety and increased food intake. Furthermore, the lower leptin-circulating concentrations lead to lower energy expenditure in peripheral pathways activated by leptin-responsive neurons [74] (Fig. 1).

Another potential effect of the excess of energy supply of a high sugar intake is the activation of 11β-hydroxysteroid dehydrogenase 1 in adipocytes, which converts cortisone into cortisol, contributing to adipose tissue expansion and obesity (Fig. 1). This mechanism has been observed to be more efficiently triggered by fructose than glucose [75]. However, no

studies have been conducted in children to test this hypothesis so far.

Genetic variants can also affect the relationship between high sugar intake and cardiometabolic alterations [76] (Fig. 1). Children and adolescents carrying the GG genotype of the variant rs738409 in the patatin-like phospholipase domain-containing protein 3 (*PNPLA3*) gene have shown increased hepatic fat deposition associated with high sugar and SSB consumption. Adolescents carrying the TT genotype of the rs1260326 in the glucokinase regulatory protein (*GCKR*) gene – associated with increased glycolysis – showed higher *de novo* lipogenesis after receiving a liquid sugar load [77]. The AA genotype of the rs1800497 polymorphism in the ankyrin repeat and kinase domain-containing 1 (*ANKK1*) gene has been associated with higher sugar intake as well as with several measures of body fat in children [78]. Finally, the alpha-amylase 1 (*AMY1*) gene – involved in dietary starch and glycogen hydrolysis – known to be subject to copy number variation, has been associated with decreased BMI [79], although larger studies have failed to find the same association [80].

CONCLUSION

Infants have a natural strong preference for sweet taste and it has been observed that early introduction of added sugars in the diet of infants and toddlers may promote sweet taste preference [81,82]. As it was shown in this review, sugar intake in infants, children and adolescents worldwide often overpasses the maximum recommended 10%E from free sugars [2]. Efforts should be made towards the availability of nutritionally adequate foods and beverages for young populations. Targeted policies must be undertaken to reduce free sugar consumption to reduce the risk of obesity and cardiometabolic complications during childhood and adolescence.

Acknowledgements

The work of the authors is partially funded by the Centro de Investigación Biomédica en Red de Fisiopatología de la Obesidad y Nutrición (CIBERObn), Instituto de Salud Carlos III, Madrid, Spain (CB15/00043).

Financial support and sponsorship

None.

Conflicts of interest

There are no conflicts of interest.

REFERENCES AND RECOMMENDED READING

Papers of particular interest, published within the annual period of review, have been highlighted as:
- ■ of special interest
- ■■ of outstanding interest

1. ■■ Vos MB, Kaar JL, Welsh JA, *et al.,* American Heart Association Nutrition Committee of the Council on Lifestyle and Cardiometabolic Health; Council on Clinical Cardiology; Council on Cardiovascular Disease in the Young; Council on Cardiovascular and Stroke Nursing; Council on Epidemiology and Prevention; Council on Functional Genomics and Translational Biology; and Council on Hypertension. Added sugars and cardiovascular disease risk in children: a scientific statement from the American Heart Association. Circulation 2017; 135:e1017–e1034.

This review describes in a very detailed manner the association between added sugar intake and cardiovascular disease in children, which is mainly attributed to an increased energy intake, adiposity and dyslipidemia.

2. World Health Organization. Guideline: sugars intake for adults and children. Geneva: World Health Organization; 2015.
3. Newens KJ, Walton J. A review of sugar consumption from nationally representative dietary surveys across the world. J Hum Nutr Diet 2016; 29:225–240.
4. Swan GE, Powell NA, Knowles BL, *et al.* A definition of free sugars for the UK. Public Health Nutr 2018; 21:1636–1638.
5. ■■ Azais-Braesco V, Sluik D, Maillot M, *et al.* A review of total & added sugar intakes and dietary sources in Europe. Nutr J 2017; 16:6.

This review confirms that intakes of total and added sugars are high in many European countries, especially in children, and point to sweet products and beverages as the major contributors.

6. ■■ Rippin HL, Hutchinson J, Evans CEL, *et al.* National nutrition surveys in Europe: a review on the current status in the 53 countries of the WHO European region. Food Nutr Res 2018; 62.

This work shows that less than two-thirds of WHO Europe countries have nationally representative diet surveys, mainly collected post-2000. The main gaps lie in Central and Eastern European countries.

7. ■■ Afeiche MC, Koyratty BNS, Wang D, *et al.* Intakes and sources of total and added sugars among 4 to 13-year-old children in China, Mexico and the United States. Pediatr Obes 2018; 13:204–212.

The data highlight the heterogeneity of food patterns worldwide and the need for adapted country-specific public health recommendations on sugars.

8. ■■ Fisberg M, Kovalskys I, Gomez G, *et al.,* ELANS Study Group. Total and added sugar intake: assessment in eight Latin American Countries. Nutrients 2018; 10: pii: E389.

This study shows that Latin American adolescents show a high consumption of total and added sugar with some peculiarities in socio-demographic variables, which should be considered in each country's health intervention proposals.

9. ■ Devenish G, Ytterstad E, Begley A, *et al.* Intake, sources, and determinants of free sugars intake in Australian children aged 12–14 months. Matern Child Nutr 2018; e12692.

This article highlights the substantial contribution of infant foods to free sugar intakes and provide further evidence that dietary intakes are influenced by social determinants.

10. Langlois K, Garriguet D. Sugar consumption among Canadians of all ages. Health Rep 2011; 22:23–27.
11. ■ Mesana MI, Hilbig A, Androutsos O, *et al.* Dietary sources of sugars in adolescents' diet: the HELENA study. Eur J Nutr 2018; 57:629–641.

This work shows that the majority (94%) European adolescents exceed the 10% free sugars of daily energy intake, with a broad variety in contributing foods. Efforts shall be put to reduce the free sugar intake especially in families with a low educational level.

12. Svensson A, Larsson C, Eiben G, *et al.,* IDEFICS Consortium. European children's sugar intake on weekdays versus weekends: the IDEFICS study. Eur J Clin Nutr 2014; 68:822–828.
13. Kyttala P, Erkkola M, Kronberg-Kippila C, *et al.* Food consumption and nutrient intake in Finnish 1-6-year-old children. Public Health Nutr 2010; 13:947–956.
14. Agence nationale de sécurité sanitaire de l'alimentation, de l'environnement et du travail. Étude Individuelle Nationale des Consommations Alimentaires 3 (INCA3) (2014–2015). Rapport. Maisons-Alfort: AFSSA; 2017.
15. ■ Fujiwara A, Murakami K, Asakura K, *et al.* Estimation of starch and sugar intake in a japanese population based on a newly developed food composition database. Nutrients 2018; 10:E1474.

This article provides a foundation for future research, clarifying the starch and sugar intake in Japan. It shows that the top food sources of sugars for Japanese children were dairy products and confectionaries for total sugar.

16. Enghardt Barbieri H, Pearson M, Becker W. Riksmaten – barn 2003. Livsmedels-och näringsintag bland barn i Sverige. Uppsala: Livsmedelsverket; 2006.

17. Roberts C, Steer T, Maplethorpe N, *et al.* National diet and nutrition survey results from years 7 and 8 (combined) of the rolling programme (2014/2015 to 2015/2016). London: Public Health England; 2018.

18. ▪ Wang Y, Guglielmo D, Welsh JA. Consumption of sugars, saturated fat, and sodium among US children from infancy through preschool age, NHANES. Am J Clin Nutr 2018; 108:868–877.

This study demonstrates that the consumption of sugars, fats, and sodium in the USA exceeds recommended guidelines before children reach school age.

19. ▪ Bailey RL, Fulgoni VL, Cowan AE, Gaine PC. Sources of added sugars in young children, adolescents, and adults with low and high intakes of added sugars. Nutrients 2018; 10:; pii: E102.

This study shows that added sugars exceed the 10% guidelines across all age groups. Tailoring public health messages to reduce intakes of the identified food groups may be of utility in designing effective strategies to reduce added sugar intake in the United States.

20. Louie JC, Moshtaghian H, Rangan AM, *et al.* Intake and sources of added sugars among Australian children and adolescents. Eur J Nutr 2016; 55:2347–2355.

21. Elmadfa I. Österreichischer Ernährungsbericht 2012. Vienna: Bundesministeriums für Gesundheit; 2012.

22. Irish Universities Nutrition Alliance. National Pre-School Nutrition Survey. 2012.

23. Sette S, Le Donne C, Piccinelli R, *et al.* The third Italian National Food Consumption Survey, INRAN-SCAI 2005-06-part 1: nutrient intakes in Italy. Nutr Metab Cardiovasc Dis 2011; 21:922–932.

24. Sluik D, van Lee L, Engelen AI, Feskens EJ. Total, free, and added sugar consumption and adherence to guidelines: the Dutch National Food Consumption Survey 2007–2010. Nutrients 2016; 8:70.

25. Ruiz E, Rodriguez P, Valero T, *et al.* Dietary intake of individual (free and intrinsic) sugars and food sources in the Spanish population: findings from the ANIBES Study. Nutrients 2017; 9: pii: E275.

26. Karin DR, Thérésa L, Ost C, *et al.* Rapport 4: La consommation alimentaire. Résumé des principaux résultats. In: Teppers E, Tafforeau J, editors. Enquête de Consommation Alimentaire 2014–2015. Brussels: WIV-ISP; 2016.

27. Pedersen AN, Christensen T, Matthiessen J, *et al.* Danskernes kostvaner 2011–2013. Søborg: DTU Fødevareinstituttet, Danmarks Tekniske Universitet; 2015.

28. Mensink G, Heseker H, Richter A, *et al.* EsKiMo - German health interview and examination survey for children and adolescents. Bonn: Bundesministerium für Ernährung, Landwirtschaft und Verbraucherschutz; 2007.

29. Steingrimsdottir L, Valgeirsdottir H, Halldorsson TI, *et al.* National nutrition surveys and dietary changes in Iceland. Economic differences in healthy eating. Laeknabladid 2014; 100:659–664.

30. Hansen L, Myhre JB, Johansen AMW, *et al.* UNGKOST 3 Landsomfattende kostholdsundersøkelse blant elever i 4. -og 8. klasse i Norge, 2015. Oslo: Folkehelseninstituttet; 2015.

31. Zhang B, Zhai FY, Du SF, Popkin BM. The China health and nutrition survey, 1989–2011. Obes Rev 2014; 15(Suppl 1):2–7.

32. Romero-Martinez M, Shamah-Levy T, Franco-Nunez A, *et al.* National Health and Nutrition Survey 2012: design and coverage. Salud Publica Mex 2013; 55(Suppl 2):S332–S340.

33. Ministerio de Salud de Argentina. Alimentos Consumidos en Argentina. Resultados de la Encuesta Nacional de Nutrición y Salud -ENNyS 2004/5. Buenos Aires: Ministerio de Salud de Argentina; 2012.

34. Instituto Brasileiro de Geografia e Estatistica. Pesquisa de Orçamentos Familiares: 2008–2009. Rio de Janeiro: Antropometria e Estado Nutricional; 2010

35. Ministerio de Salud de Chile. Encuesta Nacional de Consumo Alimentario. Santiago de Chile: Ministerio de Salud de Chile; 2014.

36. Fonseca Z, Heredia A, Ocampo R, *et al.* Encuesta Nacional De La Situación Nutricional En Colombia 2010 - ENSIN. Bogotá: Instituto Colombiano de Bienestar Familiar; 2011.

37. Freire W, Ramírez-Luzuriaga M, Belmont P, *et al.* Tomo I: Encuesta Nacional de Salud y Nutrición de la población ecuatoriana de cero a 59 años. Quito: ENSANUT-ECU; 2014.

38. Venezuela. Instituto Nacional de Estadística. Encuesta de Seguimiento al Consumo de Alimentos (ESCA). Caracas: Venezuela. Instituto Nacional de Estadistica; 2014

39. Moore JB, Horti A, Fielding BA. Evaluation of the nutrient content of yogurts: a comprehensive survey of yogurt products in the major UK supermarkets. BMJ Open 2018; 8:e021387.

40. Dello Russo M, Ahrens W, De Henauw S, *et al.,* IDEFICS Consortium. The impact of adding sugars to milk and fruit on adiposity and diet quality in children: a cross-sectional and longitudinal analysis of the Identification and Prevention of Dietary- and Lifestyle-Induced Health Effects in Children and Infants (IDEFICS) Study. Nutrients 2018; 10: pii: E1350.

41. Koletzko B, Lehmann Hirsch N, Jewell JM, *et al.* Pureed fruit pouches for babies: child health under squeeze. J Pediatr Gastroenterol Nutr 2018; 67:561–563.

42. ▪▪ Fidler Mis N, Braegger C, Bronsky J, *et al.,* ESPGHAN Committee on Nutrition: sugar in infants, children and adolescents. Sugar in infants, children and adolescents: a position paper of the European Society for Paediatric Gastroenterology, Hepatology and Nutrition Committee on Nutrition. J Pediatr Gastroenterol Nutr 2017; 65:681–696.

This position paper reviews available terminology; current recommendations and intakes in children/adolescents; evidence on the development of sweet taste preference; evidence on health effects in childhood; and provides recommendations and practical points for the paediatric population.

43. ▪▪ Bleich SN, Vercammen KA. The negative impact of sugar-sweetened beverages on children's health: an update of the literature. BMC Obes 2018; 5:6.

This review gathers the available evidence of the effects of sugar-sweetened beverages on children's health, finding most evidence for overweight and obesity as well as dental caries, followed by insulin resistance and caffeine-related effects.

44. Welsh JA, Wang Y, Figueroa J, Brumme C. Sugar intake by type (added vs. naturally occurring) and physical form (liquid vs. solid) and its varying association with children's body weight, NHANES 2009–2014. Pediatr Obes 2018; 13:213–221.

45. Gui ZH, Zhu YN, Cai L, *et al.* Sugar-sweetened beverage consumption and risks of obesity and hypertension in chinese children and adolescents: a national cross-sectional analysis. Nutrients 2017; 9: pii: E1302.

46. Rogers PJ, Shahrokni R. A comparison of the satiety effects of a fruit smoothie, its fresh fruit equivalent and other drinks. Nutrients 2018; 10:; pii: E431.

47. Jen V, Erler NS, Tielemans MJ, *et al.* Mothers' intake of sugar-containing beverages during pregnancy and body composition of their children during childhood: the Generation R Study. Am J Clin Nutr 2017; 105:834–841.

48. Goran MI, Martin AA, Alderete TL, *et al.* Fructose in breast milk is positively associated with infant body composition at 6 months of age. Nutrients 2017; 9: pii: E146.

49. Cantoral A, Tellez-Rojo MM, Ettinger AS, *et al.* Early introduction and cumulative consumption of sugar-sweetened beverages during the preschool period and risk of obesity at 8–14 years of age. Pediatr Obes 2016; 11:68–74.

50. Marshall TA, Van Buren JM, Warren JJ, *et al.* Beverage consumption patterns at age 13 to 17 years are associated with weight, height, and body mass index at age 17 years. J Acad Nutr Diet 2017; 117:698–706.

51. Cafe ACC, Lopes CAO, Novais RLR, *et al.* Intake of sugar-sweetened beverages, milk and its association with body mass index in adolescence: a systematic review. Rev Paul Pediatr 2018; 36:91–99.

52. Franken SCM, Smit CR, Buijzen M. Promoting water consumption on a Caribbean island: an intervention using children's social networks at schools. Int J Environ Res Public Health 2018; 15: pii: E713.

53. Ochoa-Aviles A, Verstraeten R, Huybregts L, *et al.* A school-based intervention improved dietary intake outcomes and reduced waist circumference in adolescents: a cluster randomized controlled trial. Nutr J 2017; 16:79.

54. Lane H, Porter K, Estabrooks P, Zoellner J. A systematic review to assess sugar-sweetened beverage interventions for children and adolescents across the socioecological model. J Acad Nutr Diet 2016; 116: 1295.e6–1307.e6.

55. ▪ Vargas-Garcia EJ, Evans CEL, Prestwich A, *et al.* Interventions to reduce consumption of sugar-sweetened beverages or increase water intake: evidence from a systematic review and meta-analysis. Obes Rev 2017; 18:1350–1363.

This systematic review evaluated the effect of interventions, which aimed to reduce sugar-sweetened beverages (SSB) consumption or increased water intake, and to examine the impact of behavior change techniques (BCTs) in consumption patterns. They found that only small reductions in SSBs have been achieved by interventions targeting adolescents and adults.

56. Trude ACB, Surkan PJ, Cheskin LJ, Gittelsohn J. A multilevel, multicomponent childhood obesity prevention group-randomized controlled trial improves healthier food purchasing and reduces sweet-snack consumption among low-income African-American youth. Nutr J 2018; 17:96.

57. ▪ Deshpande G, Mapanga RF, Essop MF. Frequent sugar-sweetened beverage consumption and the onset of cardiometabolic diseases: cause for concern? J Endocr Soc 2017; 1:1372–1385.

This work brings together clinical and observational studies investigating the effect of sugar-sweetened beverages on cardiometabolic alterations, finding a lack of agreement among available results and proposing the need for well designed long-term clinical studies.

58. ▪ Seferidi P, Millett C, Laverty AA. Sweetened beverage intake in association to energy and sugar consumption and cardiometabolic markers in children. Pediatr Obes 2018; 13:195–203.

In this observational study, authors found an effect of both artificially sweetened and sugar-sweetened beverages on blood glucose levels.

59. Wang JW, Mark S, Henderson M, *et al.* Adiposity and glucose intolerance exacerbate components of metabolic syndrome in children consuming sugar-sweetened beverages: QUALITY cohort study. Pediatr Obes 2013; 8:284–293.

60. Welsh JA, Sharma A, Cunningham SA, Vos MB. Consumption of added sugars and indicators of cardiovascular disease risk among US adolescents. Circulation 2011; 123:249–257.

61. Leermakers ET, Felix JF, Jaddoe VW, *et al.* Sugar-containing beverage intake at the age of 1 year and cardiometabolic health at the age of 6 years: the Generation R Study. Int J Behav Nutr Phys Act 2015; 12:114.

62. Sanjeevi N, Lipsky LM, Nansel TR. Cardiovascular biomarkers in association with dietary intake in a longitudinal study of youth with type 1 diabetes. Nutrients 2018; 10:; pii: E1552.

63. Hilger-Kolb J, Bosle C, Motoc I, Hoffmann K. Associations between dietary factors and obesity-related biomarkers in healthy children and adolescents - a systematic review. Nutr J 2017; 16:85.

64. Mirtschink P, Jang C, Arany Z, Krek W. Fructose metabolism, cardiometabolic ■■ risk, and the epidemic of coronary artery disease. Eur Heart J 2018; 39:2497–2505.
This study summarizes the recent progress in the understanding of fructose metabolism and how it contributes to the development of systemic diseases that cooperatively promote the cardiometabolic syndrome and cardiovascular disease, helping shape preventive actions and therapeutic approaches.
65. Choo VL, Viguiliouk E, Blanco Mejia S, *et al.* Food sources of fructose- ■ containing sugars and glycaemic control: systematic review and meta-analysis of controlled intervention studies. BMJ 2018; 363:k4644.
This study unravels the association between fructose-containing sugars and glycaemic control by considering different food and drink sources. Adverse effects on fasting glucose and insulin are observed for fructose-containing sugars exceeding the energy supply of the diet, especially in the case of sugar-sweetened beverages.
66. Zhang DM, Jiao RQ, Kong LD. High dietary fructose: direct or indirect ■■ dangerous factors disturbing tissue and organ functions. Nutrients 2017; 9:; pii: E335.
In this work, the systemic and organ effects of fructose are reviewed, including potential mediatory molecules and giving detailed picture of the mechanisms involved.
67. Jensen T, Abdelmalek MF, Sullivan S, *et al.* Fructose and sugar: a major ■■ mediator of nonalcoholic fatty liver disease. J Hepatol 2018; 68:1063–1075.
Review of the available literature regarding the relationship between fructose and sugar consumption with nonalcoholic fatty liver disease, in children and adolescents.
68. Tappy L. Fructose metabolism and noncommunicable diseases: recent find- ■ ings and new research perspectives. Curr Opin Clin Nutr Metab Care 2018; 21:214–222.
This review highlights the existence of an endogenous fructose production that would further contribute to the adverse consequences of this monosacharide on human metabolism and health.
69. Caliceti C, Calabria D, Roda A, Cicero AFG. Fructose intake, serum uric acid, and cardiometabolic disorders: a critical review. Nutrients 2017; 9: pii: E395.
70. Chiu S, Mulligan K, Schwarz JM. Dietary carbohydrates and fatty liver disease: de novo lipogenesis. Curr Opin Clin Nutr Metab Care 2018; 21:277–282.
71. Bernardes N, Ayyappan P, De Angelis K, *et al.* Excessive consumption of fructose causes cardiometabolic dysfunctions through oxidative stress and inflammation. Can J Physiol Pharmacol 2017; 95:1078–1090.
72. Aragno M, Mastrocola R. Dietary sugars and endogenous formation of ■ advanced glycation endproducts: emerging mechanisms of disease. Nutrients 2017; 9: pii: E385.
This work gathers the available in-vitro, in-vivo and human results on the effect of advanced glycation endproduct formation on organ damage in liver, skeletal and cardial muscle and brain, in relation to their dietary sources, focusing on added sugars.
73. Hannou SA, Haslam DE, McKeown NM, Herman MA. Fructose metabolism and metabolic disease. J Clin Invest 2018; 128:545–555.
74. Belfort-DeAguiar R, Seo D. Food cues and obesity: overpowering hormones and energy balance regulation. Curr Obes Rep 2018; 7:122–129.
75. Legeza B, Marcolongo P, Gamberucci A, *et al.* Fructose, glucocorticoids and adipose tissue: implications for the metabolic syndrome. Nutrients 2017; 9: pii: E426.
76. Haslam DE, McKeown NM, Herman MA, *et al.* Interactions between genetics and sugar-sweetened beverage consumption on health outcomes: a review of gene-diet interaction studies. Front Endocrinol (Lausanne) 2017; 8:368.
77. Santoro N, Caprio S, Pierpont B, *et al.* Hepatic de novo lipogenesis in obese youth is modulated by a common variant in the GCKR gene. J Clin Endocrinol Metab 2015; 100:E1125–E1132.
78. Cardel MI, Lemas DJ, Lee AM, *et al.* Taq1a polymorphism (rs1800497) is associated with obesity-related outcomes and dietary intake in a multiethnic sample of children. Pediatr Obes 2018; 14:e12470.
79. Nakajima K. Low serum amylase and obesity, diabetes and metabolic syndrome: a novel interpretation. World J Diabetes 2016; 7:112–121.
80. Usher CL, Handsaker RE, Esko T, *et al.* Structural forms of the human amylase locus and their relationships to SNPs, haplotypes, and obesity. Nat Genet 2015; 47:921–925.
81. Forestell CA. Flavor perception and preference development in human infants. Ann Nutr Metab 2017; 70(Suppl 3):17–25.
82. Murray RD. Savoring sweet: sugars in infant and toddler feeding. Ann Nutr Metab 2017; 70(Suppl 3):38–46.