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Added sugar intake and metabolic syndrome in US adolescents: cross-sectional analysis of the National Health and Nutrition Examination Survey 2005–2012

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Abstract

Objective: To examine the association between added sugar intake and metabolic syndrome among adolescents.
Design: Dietary, serum biomarker, anthropometric and physical activity data from the US National Health and Nutrition Examination Survey cycles between 2005 and 2012 were analysed using multivariate logistic regression models. Added sugar intake in grams per day was estimated from two 24 h standardized dietary recalls and then separated into quintiles from lowest to highest consumption. Multivariate logistic regression analyses were adjusted for physical activity, age, BMI *Z*-score and energy intake, and their interactions with race were included.
Setting: Nationally representative sample, USA.
Subjects: US adolescents aged 12–19 years (*n* 1623).
Results: Added sugar was significantly associated with metabolic syndrome. The adjusted prevalence odds ratios for having metabolic syndrome comparing adolescents in the third, fourth and fifth quintiles *v.* those in the lowest quintile of added sugar were 5·3 (95 % CI 1·4, 20·6), 9·9 (95 % CI 1·9, 50·9) and 8·7 (95 % CI 1·4, 54·9), respectively.
Conclusions: Our findings suggest that higher added sugar intake, independent of total energy intake, physical activity or BMI *Z*-score, is associated with increased prevalence of metabolic syndrome in US adolescents. Further studies are needed to determine if reducing intake of added sugar may help US adolescents prevent or reverse metabolic syndrome.

Keywords
Metabolic syndrome
Adolescents
United States
Added sugar

Non-communicable chronic diseases, including type 2 diabetes, CVD, hypertension, dyslipidaemia and non-alcoholic fatty liver disease (NAFLD), are responsible for more deaths globally than infectious diseases[1], presenting a tremendous and growing public health problem. Metabolic syndrome (MetS) is the clustering of these anomalies in individual patients[2]. NAFLD represents a spectrum of liver disease – from fatty accumulation in the liver, to inflammation and progressive fibrosis[3] – and it is strongly associated with MetS in adolescents aged 12–19 years[4]. Paediatric MetS predicts adulthood MetS and type 2 diabetes[5], consequently providing a suitable window of opportunity for preventive action against the advancement of this growing public health problem.

Various aspects of MetS, such as type 2 diabetes and NAFLD, have increased in tandem with obesity, which has increased considerably over the past four decades. Among US adolescents the prevalence of obesity increased from 4·5 % in the 1960s to 20·3 % in 2012[6]. In addition, obesity has disproportionally affected certain minority groups; for instance, current obesity rates for African-American girls are 24·8 % and 28·9 % for Mexican-American boys[6]. While lifestyle interventions to induce weight loss are the current standard of care for reversing MetS[3], among adolescents they result in only modest weight reductions and most adolescents will continue to manifest the diseases of MetS into adulthood[7]. Similarly, it is unclear if weight reduction strategies during adolescence prevent co-morbidities in adulthood[8]. Lack of empirical evidence for MetS reduction via weight management alone argues the need to explore alternative interventions to prevent and treat MetS.

Added sugars (unlike naturally occurring sugars found in milk, fruits and vegetables) are sugars and syrups

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added to foods and beverages during processing or preparation[9]. Recently, total added sugar consumption in the USA has decreased, primarily due to a reduction in soda consumption; however, adolescents continue to have the greatest added sugar consumption among all age groups[10]. Recent studies suggest that excessive added sugar consumption contributes to the development of diabetes, dyslipidaemia and cardiovascular mortality, independent of energy intake or effects on adiposity[11–16]. However, none have looked at the impact of added sugar on the constellation of symptoms of MetS among adolescents. The objective of the present study was to examine the direct association of added sugar intake with MetS in a large, nationally representative, cross-sectional sample of US adolescents aged 12–19 years participating in the National Health and Nutrition Examination Surveys (NHANES) of 2005–2012. While added sugar likely has indirect effects on MetS through increased energy intake or obesity, we were specifically interested in investigating the independent association. In addition, given that obesity, and likely MetS, disproportionately affects adolescents of different racial/ethnic backgrounds, we report here the racial differences in risk of MetS among Mexican-Americans, non-Hispanic whites and non-Hispanic blacks.

Methods

Study protocols and population

The study protocols for the NHANES were approved by the National Center for Health Statistics Ethics Review Board[17]. Parents or caregivers of children gave their informed consent to participate and provide information about their children. Only unidentified public-domain data were used in the secondary data analysis conducted for the present study.

NHANES is a programme of studies designed to assess the health and nutrition status of children and adults. Each two-year cycle between 2005 and 2012 includes a nationally representative sample of adolescents, selected with a multistage complex design[18]. For the present analysis, only data from adolescents 12–19 years of age (n 4733) were analysed. Participants were classified as Mexican-American, other Latino, non-Hispanic white, non-Hispanic black, and other. Due to the absence of a standard waist circumference (WC) cut-off definition for MetS among US adolescents under 16 years old who are not European-American, African-American or Mexican-American, adolescents who self-identified as 'other Latinos' or 'other' were excluded from analyses (n 624)[19,20]. Pregnant adolescents were also excluded from analyses, as well as those who reported the use of steroids, growth hormone, sex hormones, blood glucose regulators, insulin or other antidiabetic agents (n 45). A subset of 3453 adolescents reported dietary information (from two 24 h dietary recalls). We further excluded adolescents who were not randomly selected by the NHANES sampling design to provide serum samples or who had missing values of fasting biomarkers or covariates (n 1711), those with very high (>20 920 kJ/d (>5000 kcal/d)) or low (<2092 kJ/d (<500 kcal/d)) daily energy intake (n 60), as well as those considered to have a BMI <5th percentile for age/sex (n 59). Sensitivity analyses were conducted to assess whether exclusions of low BMI or very high or low daily energy intakes influenced our results. These exclusions left a sample size between 1179 and 1623 for the associations between added sugar and each MetS biomarker.

Exposure

Dietary intake data were collected using two 24 h dietary recalls, following the US Department of Agriculture's (USDA) Automated Multiple Pass Method, and administered directly to the adolescent[18]. The first recall interview was conducted in person in the mobile examination centre and the second was conducted by telephone 3 to 10 d later[21]. A recall was deemed reliable if all relevant variables associated with the 24 h dietary recall contained a value[21]. The arithmetic mean of added sugar intake in grams per day was obtained by merging individual dietary recalls from NHANES with the USDA Food Patterns Equivalents Database (FPED)[22]. Our main exposure of interest was the resultant mean daily added sugar intake.

Outcomes

Blood was collected from participants; details of the laboratory assessment are available and described elsewhere[18]. Biochemical markers of MetS included fasting TAG, blood glucose and insulin, from which the homeostasis model assessment of insulin resistance (HOMA-IR) was calculated ([fasting blood glucose (mmol/l) × fasting insulin (mU/l)]/22·5)[23]. Non-fasting biomarkers included HDL cholesterol (HDL-C), uric acid (a marker of increased cardiovascular risk)[24] and alanine aminotransferase (ALT), a surrogate for NAFLD. Also included in our analyses was TAG:HDL-C, an index of insulin resistance[25–27]. Other indicators of MetS included WC and systolic and diastolic blood pressures. We used the International Diabetes Federation definitions for MetS for children (12–16 years old) and adults (≥16 years old)[19,20].

Covariates

Anthropometry

BMI was evaluated through measurements of weight and height, and subsequently transformed to standard age- and sex-specific Z-scores using the growth references of the Centers for Disease Control and Prevention of 2000[28]. BMI Z-score for age and sex was classified as underweight when less than −1·64, overweight when greater than +1·04 (85th percentile) and less than +1·64 (95th percentile), and obese when greater than or equal to

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+1·64 (95th percentile). Data were included and considered valid for *Z*-scores between −1·64 and +3·50 from the mean of the reference population[28].

Physical activity

The Physical Activity Questionnaire (PAQ) included questions related to daily activities; all participants answered their own questions. Respondents between 12 and 15 years old answered these questions during their physical examination at the mobile examination centre; respondents older than 16 years old answered these questions from home before the examination, using a computer-assisted personal interviewing system[21]. Starting in 2007, the NHANES physical activity questionnaire was changed to better quantify vigorous physical activity. The questions asked: 'Does your work involve, (or) do you do any moderate-intensity or vigorous-intensity activities that cause small or large increases in breathing or heart rate for at least 10 minutes continuously?'[21] For consistency across all study cycles, and similar to a recent study[13], we defined 'non-sedentary adolescents' as those who engaged in moderate or vigorous work or physical activity for at least 10 min once weekly.

Total energy intake

Estimated total energy intake in kilocalories per day was obtained from the mean of two individual 24 h dietary recalls following the USDA Automated Multiple Pass Method, administered directly to the adolescent[18].

Statistical analyses

All analyses were performed using the STATA statistical software package version 13·1 (2013). The 'svy' module was used to conduct multivariate logistic regression analysis adjusting estimates for the complex survey design, taking into account the expansion factor, strata and primary sampling unit parameters to ensure that the results were representative of the adolescent US population. Total energy and added sugar intake variables were divided into quintiles and analysed as ordinal categorical predictors. For risk of MetS and each individual MetS biomarker, as well as ALT and uric acid, we used multivariate logistic regression analyses to estimate adjusted prevalence odds ratios (POR) comparing each quintile of added sugar intake with the reference (first quintile). Multivariate linear regression models were also used to test for linear trends between ALT and uric acid levels and quintiles of added sugar. Because we were interested in examining the direct association between added sugar and each biomarker of interest, each linear and logistic regression model was adjusted for physical activity, age, BMI *Z*-score and energy intake. Interaction effects of potential moderators (race and sex) of the association between added sugar and MetS were examined and found significant ($P < 0·05$) for race (Mexican-Americans compared with non-Hispanic whites). Analyses were therefore stratified by race (Non-Hispanic whites and Mexican-Americans; for completeness non-Hispanic blacks were also reported).

Results

Sample characteristics

The estimated population prevalence of MetS among US adolescents between 2005 and 2012 was 5·4 % overall; 5·4 % in non-Hispanic whites, 3·3 % in non-Hispanic blacks and 16·4 % in Mexican-Americans, with the latter being significantly higher than the first two (Table 1). The mean age of our adolescent sample was 15·9 years, with 53·7 % being male and 46·3 % female (Table 1). The mean added sugar intake for the adolescent sample was 94·0 g/d, constituting roughly 17·9 % of mean energy intake. The estimated prevalence of MetS in the lowest added

Table 1 Descriptive characteristics of the sample of US adolescents (aged 12–19 years), National Health and Nutrition Examination Survey (NHANES) 2005–2012

Characteristic	NHANES 2005–2012	Expansion (thousands)	Mean added sugar (g/d)	MetS (%)
Participants, *n*	1623	20 738	94·0	5·4
Mean age (years)	15·9	20 738	94·0	5·4
Gender (%)				
Male (Ref.)	53·7	11 141	105·3	5·8
Female	46·3	9597	80·8*	4·7
Race/ethnicity (%)				
Non-Hispanic white (Ref.)	59·7	12 388	96·6	5·4
Non-Hispanic black	16·0	3327	93·1	3·3
Mexican-American	12·6	2601	87·7*	16·4*
Other race	11·7	2423	89·1	n/a
BMI (%)				
Underweight	3·5	750	96·0	n/a
Normal weight (Ref.)	64·7	13 727	90·1	0·4
Overweight	16·3	3454	90·4	6·9*
Obese	15·5	3281	91·8	57·4*,†

MetS, metabolic syndrome; Ref., reference category; n/a, not applicable.
*Statistically significant compared with reference category ($P < 0·05$).
†Statistically significant compared with overweight status (Wald test, $P < 0·05$).

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sugar quintile was 4·2 %, compared with 3·6 % in the second quintile, 4·2 % in the third quintile, 6·7 % in the fourth quintile and 8·4 % in the fifth quintile (Table 2).

Added sugar intake and metabolic syndrome

The adjusted POR of having MetS comparing adolescents in the second, third, fourth and fifth quintiles *v.* those in the lowest quintile of added sugar were 2·4 (95 % CI 0·6, 9·9), 5·3 (95 % CI 1·4, 20·6), 9·9 (95 % CI 1·9, 50·9) and 8·7 (95 % CI 1·4, 54·9), respectively (Table 3). Results from our sensitivity analyses in which adolescents with low BMI (<5th percentile) or very high (>20 920 kJ/d (>5000 kcal/d)) or low (<2092 kJ/d (<500 kcal/d)) daily energy intakes were included showed no effect on the adjusted POR for risk of MetS comparing adolescents in the second, third, fourth and fifth quintiles *v.* those in the lowest quintile of added sugar. POR results were 2·6 (95 % CI 0·6, 11·3), 5·3 (95 % CI 1·4, 20·4), 9·9 (95 % CI 1·9, 50·7) and 8·7 (95 % CI 1·4, 54·1), respectively. The adjusted POR for risk of elevated ALT (surrogate for NAFLD) were not significantly different across different added sugar quintiles. However, the adjusted POR for having elevated ALT was 3·0 (95 % CI 2·3, 3·9) for every 1-point increase in BMI *Z*-score (data not shown). Logistic regression models did not find an association between added sugar intake and elevated uric acid levels using previously used multiple cut-off values (5·5, 6·0 and 7·5 mg/dl)[29]. However, using a multivariate linear regression model, we found that for every unit increase in added sugar quintile, uric acid levels increased by 0·06 mg/dl ($P = 0·033$; Table 4).

When evaluating individual MetS components, we found that the adjusted POR for having elevated levels of TAG and insulin, and depressed levels of HDL-C, comparing adolescents in the third, fourth and fifth quintiles *v.* those in the lowest quintile of added sugar, increased significantly (Table 3). The adjusted POR for having elevated HOMA-IR was higher among adolescents in the second quintile (POR = 1·7; 95 % CI 1·0, 2·9), but not statistically higher among the highest three quintiles. In addition, the adjusted POR for WC increased in the third and fourth quintiles (POR = 2·0; 95 % CI 1·0, 4·1 and POR = 2·7; 95 % CI 1·1, 6·9, respectively). A strong positive association remained between BMI *Z*-score and the odds of MetS among all race/ethnic groups; the adjusted POR for the risk of MetS for every one unit increase in BMI *Z*-score was 48·1 (95 % CI 19·6, 117·9; data not shown).

Added sugar intake and metabolic syndrome by race/ethnicity

After stratifying by race, we were unable to examine the association between added sugar intake and risk for MetS due to small sample sizes. When evaluating individual MetS components among Mexican-American adolescents (Table 5), added sugar was associated with higher adjusted POR for elevated TAG in the fourth quintile (POR = 2·9; 95 % CI, 1·0, 8·2) and HOMA-IR in the second quintile (POR = 2·9; 95 % CI, 1·0, 8·8). We also performed a linear regression analysis, adjusted for total energy intake, BMI *Z*-score and physical activity, to test for trend between added sugar and ALT, and uric acid levels. We found no association between ALT and added sugar (Table 6). However, we observed an association between uric acid and added sugar, and after stratifying for race/ethnicity the associated persisted among Mexican-American adolescents (Table 4).

Lastly, among non-Hispanic white and black adolescents (Tables 7 and 8), added sugar was associated with increased odds of elevated TAG and insulin, although less consistently across quintiles. Added sugar was also associated with increased odds of elevated TAG:HDL-C among non-Hispanic whites. Among Mexican-Americans, the association was only marginally significant ($P = 0·06$–$0·1$ for third through fifth quintiles), and it was not present among non-Hispanic blacks.

Discussion

Many studies have examined the effects of added sugars on cardiometabolic health, but none have examined the risk for MetS among adolescents. Using nationally representative cross-sectional data on US adolescents (aged 12–19 years) participating in NHANES 2005–2012, our findings indicate that increased added sugar intake is an independent risk factor for MetS, but not for NAFLD. Similar to our findings, Zhang *et al.*[13] found added sugar intake to be positively associated with risk of dyslipidaemia, a component of MetS, across different race/ethnicities and sex among adolescents participating in NHANES 2005–2010. Furthermore, our findings agree with prior studies that suggest that BMI reduction should be a primary intervention for the prevention of MetS[3–5,19]. However, our findings also suggest that added sugar intake is associated with MetS, independent of total energy intake or BMI *Z*-score[2,20]. Recently, a study by Lustig *et al.* showed a marked improvement in cardiometabolic biomarkers among obese children with MetS after only 9 d of isoenergetic substitution of glucose for fructose, suggesting that the effect of added sugar on biomarkers was unrelated to its energy equivalence[16].

Individual MetS components that were associated with added sugar intake in the present adolescents include TAG, HDL-C, insulin and WC, although the last two were significant only for the third and fourth added sugar quintiles. Paradoxically, when stratified by race, HDL-C remained strongly and positively associated with added sugar consumption among non-Hispanic whites, but the statistical significance among non-Hispanic blacks or Mexican-Americans diminished. This difference appears to be due to loss in power from smaller subpopulations. Moreover, HOMA-IR was significantly higher only in the

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Table 2 Unadjusted prevalence of elevated biomarkers for metabolic syndrome (MetS) and non-alcoholic fatty liver disease across added sugar quintiles among US adolescents (aged 12–19 years), National Health and Nutrition Examination Survey 2005–2012

		Mean added sugar intake									
		Q1: 30·2 g/d		Q2: 58·3 g/d		Q3: 81·8 g/d		Q4: 112·7 g/d		Q5: 186·0 g/d	
MetS biomarker/ indicator	Sample size	%	95 % CI	%	95 % CI	%	95 % CI	%	95 % CI	%	95 % CI
TAG	1521	4·1	1·7, 6·5	4·7	1·8, 7·5	9·2*	5·6, 12·8	9·6*	4·2, 15·1	10·3*,†	5·5, 14·9
HDL-C	1623	10·1	6·0, 14·2	13·4	8·9, 17·9	11·5	7·6, 15·5	19·4*,‡	14·4, 24·4	23·9*,†,‡	18·9, 28·9
TAG:HDL-C	1521	8·2	5·0, 11·4	10·8	5·9, 15·6	13·8	9·5, 18·1	16·0*	9·6, 22·4	16·1*	10·7, 21·6
BG	1521	20·9	15·3, 26·7	18·6	14·2, 23·1	24·6	18·7, 30·6	20·6	14·1, 27·1	20·2	14·9, 25·5
Insulin	1503	22·8	16·0, 29·5	26·6	21·1, 32·1	30·3	22·6, 38·0	25·8	19·2, 32·2	18·7†,‡,§	14·3, 23·1
HOMA-IR	1503	15·6	10·4, 20·8	18·9	13·9, 23·9	17·5	12·6, 22·4	17·6	11·6, 23·7	15·7	11·6, 19·8
SBP	1588	3·8	0·7, 6·9	1·5	0·2, 2·7	4·4	1·3, 7·7	2·2	0·4, 4·0	3·7	1·0, 6·4
DBP	1588	0·1	0·0, 0·3	0·2	0·0, 0·7	0·4	0·0, 0·9	0·4	0·0, 1·0	0·8	0·0, 2·0
WC	1623	25·5	19·4, 31·7	23·6	19·6, 31·7	25·6	20·9, 35·9	28·4	20·9, 35·9	21·8	16·8, 26·9
Uric acid	1602	35·9	29·2, 42·6	33·1	26·3, 39·7	38·7	32·8, 44·6	43·6	34·3, 52·9	45·2†	37·7, 52·7
ALT	1568	14·8	8·5, 21·1	10·9	6·7, 15·3	14·6	8·6, 20·6	13·5	8·2, 18·7	14·7	9·3, 20·1
MetS	1179	4·2	0·3, 8·0	3·6	0·8, 6·3	4·2	1·5, 6·8	6·7†	2·9, 10·4	8·4†,‡	4·7, 12·1

HDL-C, HDL cholesterol; BG, blood glucose; HOMA-IR, homeostasis model assessment of insulin resistance; SBP, systolic blood pressure; DBP, diastolic blood pressure; WC, waist circumference; ALT, alanine aminotransferase.
Added sugar quintile ranges: Q1, 0–46 g/d; Q2, 47–69 g/d; Q3, 70–95 g/d; Q4, 96–133 g/d; Q5, 134–500 g/d.
*Statistically significant compared with Q1 ($P < 0·05$).
†Statistically significant compared with Q2 (Wald test, $P < 0·05$).
‡Statistically significant compared with Q3 (Wald test, $P < 0·05$).
§Statistically significant compared with Q4 (Wald test, $P < 0·05$).

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Table 3 Adjusted prevalence odds ratios (POR) of metabolic syndrome (MetS) biomarkers in relationship to intake of added sugar by quintile among US adolescents (aged 12–19 years), National Health and Nutrition Examination Survey 2005–2012: multivariate logistic regression

		Mean added sugar intake							
		Q1:	Q2: 58·3 g/d		Q3: 81·8 g/d		Q4: 112·7 g/d		Q5: 186 gd	
MetS biomarker/ indicator	Sample size	30·2 g/d	POR	95 % CI	POR	95 % CI	POR	95 % CI	POR	95 % CI
TAG	1521	Ref.	1·2	0·4, 3·2	2·6*,†	1·2, 5·6	2·7*	1·3, 5·9	3·7*	1·3, 10·9
HDL-C	1623	Ref.	1·6	0·7, 3·3	1·6	0·8, 3·5	3·2*,†,‡	1·6, 6·4	5·2*,†,‡	2·4, 11·6
TAG:HDL-C	1521	Ref.	1·5	0·8, 2·9	2·2*	1·2, 3·9	2·6*	1·3, 5·1	3·0*	1·3, 6·6
BG	1521	Ref.	0·9	0·6, 1·2	1·2	0·7, 1·9	0·9	0·6, 1·5	0·9	0·5, 1·7
Insulin	1503	Ref.	1·6	0·9, 2·7	2·1*	1·2, 3·5	1·7*	1·1, 2·7	1·2	0·7, 1·9
HOMA-IR	1503	Ref.	1·7*	1·0, 2·9	1·5	0·8, 2·9	1·6	0·7, 3·5	1·6	0·8, 3·0
SBP	1588	Ref.	0·6	0·2, 2·1	1·9	0·5, 7·1	0·7	0·1, 3·1	1·0	0·2, 4·9
DBP	1588	Ref.	3·3	0·2, 53·3	4·9	0·4, 57·9	2·1	0·1, 36·2	7·9	0·5, 118·6
WC	1623	Ref.	1·4	0·6, 3·2	2·0*	1·0, 4·1	2·7*	1·1, 6·9	2·3	0·8, 6·7
Uric acid	1602	Ref.	0·9	0·5, 1·7	1·3	0·8, 2·1	1·4	0·8, 2·6	1·4	0·8, 2·5
ALT	1610	Ref.	0·7	0·4, 1·3	1·0	0·5, 1·9	0·7	0·4, 1·2	0·7	0·4, 1·4
MetS	1179	Ref.	2·4	0·6, 9·9	5·3*	1·4, 20·6	9·9*,†	1·9, 50·9	8·7*	1·4, 54·9

HDL-C, HDL cholesterol; BG, blood glucose; HOMA-IR, homeostasis model assessment of insulin resistance; SBP, systolic blood pressure; DBP, diastolic blood pressure; WC, waist circumference; ALT, alanine aminotransferase; Ref., reference category.
Added sugar quintile ranges: Q1, 0–46 g/d; Q2, 47–69 g/d; Q3, 70–95 g/d; Q4, 96–133 g/d; Q5, 134–500 g/d.
*Statistically significant ($P < 0·05$) adjusted for physical activity, age, BMI *Z*-score and energy intake.
†Statistically significant compared with Q2 (Wald test, $P < 0·05$) adjusted for physical activity, age, BMI *Z*-score and energy intake.
‡Statistically significant compared with Q3 (Wald test, $P < 0·05$) adjusted for physical activity, age, BMI *Z*-score and energy intake.

Table 4 Association between usual intake of added sugar in grams per day and uric acid among US adolescents (aged 12–19 years), National Health and Nutrition Examination Survey 2005–2012

Biomarker	Sample size	*β* coefficient*	95 % CI	*P* value
Uric acid				
All adolescents	1396	0·06	0·01, 0·12	0·033
Non-Hispanic whites	441	0·07	−0·01, 0·14	0·102
Non-Hispanic blacks	462	−0·004	−0·09, 0·09	0·930
Mexican-Americans	493	0·12	0·03, 0·22	0·008

**β* coefficient for usual intake of added sugar in grams per day represents the change in uric acid associated with every unit increase in added sugar quintile. Linear regression model adjusted for physical activity, age, BMI *Z*-score and energy intake.

second quintile; while we anticipate a similar association among the highest three quintiles, the lack of significance may be a consequence of insufficient power to detect a difference due to small sample size. In the same way, our stratified results show that added sugar was associated with higher adjusted POR for HOMA-IR only in the second quintile among non-Hispanic black and Mexican-American adolescents, compared with their respective references. In the present study, HOMA-IR was calculated by fasting measures of blood glucose and insulin, thus our study design cannot capture the postprandial acute effects of added sugar intake on insulin resistance, as have been previously demonstrated[16]. A detailed analysis exploring postprandial effects is beyond the scope of the current study; however, differences in insulin resistance according to sugar consumption in racial subgroups are important and warrant further investigation. Lastly TAG:HDL-C also appeared to be associated with added sugar intake among non-Hispanic whites, but not among Mexican-Americans or non-Hispanic blacks. The differential association by race demonstrated in the present study has been seen in other studies examining TAG:HDL-C as an indicator of insulin resistance[27,30–32].

In contrast, added sugar intake did not appear to be associated with elevated levels of ALT (surrogate for NAFLD) for the aggregate adolescent population. We used a cut-off value of 25 U/l, representing the 95th percentile of ALT for non-overweight adolescents of US NHANES[33]. We also used 40 U/l as the cut-off value for ALT and similar results were observed. Prior studies have shown that genetics and sex seem to play a role in the risk for NAFLD, particularly among Mexican-origin male adolescents in the USA[34,35]. In our data, however, we found no association between added sugar intake and ALT levels. In addition, prior studies have shown positive associations between elevated uric acid levels and MetS, as well as with consumption of sugar-sweetened beverages[20,29,36]. As expected, in our multivariate linear regression model we found a positive association between added sugar

Table 5 Adjusted prevalence odds ratios (POR) of metabolic syndrome (MetS) biomarkers in relationship to intake of added sugar by quintile among US Mexican-American adolescents (aged 12–19 years), National Health and Nutrition Examination Survey 2005–2012: multivariate logistic regression

		Mean added sugar intake									
		Q1: 28·3 g/d	Q2: 54·5 g/d		Q3: 77·7 g/d		Q4: 104·0 g/d		Q5: 161·0 g/d	
MetS biomarker/ indicator	Sample size		POR	95 % CI	POR	95 % CI	POR	95 % CI	POR	95 % CI
TAG	470	Ref.	1·3	0·4, 4·3	2·3	0·6, 8·8	2·9*	1·0, 8·2	1·9	0·6, 6·2
HDL-C	496	Ref.	1·4	0·5, 3·7	1·6	0·5, 5·6	2·1	0·7, 6·4	3·1	0·8, 12·3
TAG:HDL-C	470	Ref.	1·3	0·6, 2·8	2·5	0·9, 7·3	2·2	0·8, 5·7	2·6	0·9, 7·0
BG	470	Ref.	0·7	0·4, 1·3	1·2	0·6, 2·4	1·0	0·4, 2·6	1·7	0·7, 4·3
Insulin	465	Ref.	2·2	0·8, 5·9	1·4	0·4, 4·3	2·4	0·8, 6·9	1·4	0·5, 3·6
HOMA-IR	465	Ref.	2·9*	1·0, 8·8	1·3	0·4, 4·2	2·7	0·9, 7·7	1·9	0·8, 4·5
WC	491	Ref.	3·4*	1·2, 9·4	3·1	0·9, 9·7	5·2*	1·0, 28·4	5·5*	1·3, 23·4
Uric acid	493	Ref.	1·1	0·5, 2·2	1·4	0·6, 3·3	1·9	0·8, 4·7	1·8	0·9, 3·8
ALT	474	Ref.	1·4	0·5, 4·6	0·9	0·3, 2·9	0·7	0·2, 2·1	0·9	0·3, 3·1

HDL-C, HDL cholesterol; BG, blood glucose; HOMA-IR, homeostasis model assessment of insulin resistance; WC, waist circumference; ALT, alanine aminotransferase; Ref., reference category.
Systolic and diastolic blood pressure and MetS had insufficient cell sizes to populate.
Added sugar quintile ranges: Q1, 0–44·3 g/d; Q2, 44·5–65·6 g/d; Q3, 65·9–90·4 g/d; Q4, 90·4–120·8 g/d; Q5, 120·9–321·3 g/d.
*Statistically significant ($P < .05$) adjusted for physical activity, age, BMI *Z*-score and energy intake.

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Table 6 Association between usual intake of added sugar in grams per day and alanine aminotransferase (ALT) among US adolescents (aged 12–19 years), National Health and Nutrition Examination Survey 2005–2012

Biomarker	Sample size	β coefficient*	95 % CI	P value
ALT				
All adolescents	1367	−0·16	−0·55, 0·20	0·351
Non-Hispanic whites	435	−0·39	−1·10, 0·30	0·258
Non-Hispanic blacks	457	−0·14	−0·55, 0·20	0·579
Mexican-Americans	475	−0·16	−0·65, 0·33	0·514

*β coefficient for usual intake of added sugar in grams per day represents the change in ALT associated with each unit increase in added sugar quintile. Linear regression model adjusted for physical activity, age, BMI *Z*-score and energy intake.

quintiles and uric acid levels, suggesting a dose–response relationship.

Our data also support the apparent dose–response effect of increased added sugar intake and the increased adjusted POR for MetS among adolescents in the highest quintiles of added sugar consumption (third, fourth and fifth quintiles). The fructose component of added sugar intake appears to be a direct cause of liver mitochondrial dysfunction and insulin resistance, which are hypothesized causes of MetS[37]. Fructose makes up at least 50 %, and possibly more[38], of added sugar intake. Physiologically, fructose undergoes first-pass metabolism in the liver. The entry of fructose into hepatocytes via the Glut5 transporter and its subsequent metabolism are insulin independent. Importantly, fructose bypasses two enzymes, glucokinase and phosphofructokinase, that normally route excess energy substrate away from the liver mitochondria. As a result, fructose is metabolized directly to fructose-1-phosphate, and subsequently to acetyl-CoA, which in the cytosol is then carboxylated to malonyl-CoA, which initiates the process of fatty acid synthesis[3,39,40]. Thus, in the glycogen-replete state, fructose increases the rate of *de novo* lipogenesis, generating intrahepatic lipid, inflammation and insulin resistance[41], consistent with some of our findings. At the same time, our data are consistent with the fact that there seems to be an inherent capacity of the human body to metabolize added sugar in low doses without a consequent effect on MetS biomarkers. For instance, among adolescents in the second quintile of added sugar consumption with a mean intake of 58 g/d, there were no differences compared with those in the lowest quintile of added sugar consumption.

Food disappearance data, which tend to overestimate food and nutrient intakes, have shown associations between added sugar consumption and poor cardiometabolic outcomes. A global econometric analysis using repeated cross-sectional data on diabetes and nutritional components of food found that each increment in added

Table 7 Adjusted prevalence odds ratios (POR) of metabolic syndrome (MetS) biomarkers in relationship to intake of added sugar by quintile among US non-Hispanic white adolescents (aged 12–19 years), National Health and Nutrition Examination Survey 2005–2012: multivariate logistic regression

		Mean added sugar intake									
		Q1: 32·5 g/d	Q2: 62·7 g/d		Q3: 87·8 g/d		Q4: 121·9 g/d		Q5: 200·8 g/d	
MetS biomarker/ indicator	Sample size		POR	95 % CI	POR	95 % CI	POR	95 % CI	POR	95 % CI
TAG	427	Ref.	0·2*	0·1, 0·8	2·1†	0·9, 5·2	1·8†	0·6, 5·4	2·4†	0·7, 8·4
HDL-C	447	Ref.	1·2	0·4, 3·8	2·9*	1·1, 7·8	5·4*,†	1·9, 15·1	6·8*,†,‡	2·2, 21·7
TAG:HDL-C	427	Ref.	0·6	0·2, 1·9	2·5*,†	1·3, 4·9	2·2†	0·7, 7·4	3·1*,‡	1·1, 8·7
BG	427	Ref.	0·9	0·5, 1·8	0·8	0·4, 2·3	1·0	0·4, 2·3	1·2	0·5, 3·2
Insulin	425	Ref.	1·3	0·5, 3·9	3·3*	1·6, 6·9	1·3‡	0·5, 3·0	1·3‡	0·5, 3·1
HOMA-IR	425	Ref.	1·6	0·5, 5·0	2·6	0·8, 8·1	1·9	0·8, 5·0	2·3	0·8, 6·6
WC	445	Ref.	0·9	0·2, 3·4	1·8	0·4, 7·5	2·3	0·5, 11·0	3·5	0·5, 24·6
Uric acid	442	Ref.	1·0	0·4, 2·9	1·7	0·7, 4·4	1·4	0·5, 3·6	1·5	0·5, 4·3
ALT	436	Ref.	1·1	0·4, 3·3	1·6	0·6, 4·1	0·6	0·3, 1·6	0·6	0·2, 1·8

HDL-C, HDL cholesterol; BG, blood glucose; HOMA-IR, homeostasis model assessment of insulin resistance; WC, waist circumference; ALT, alanine aminotransferase; Ref., reference category.
Systolic and diastolic blood pressure and MetS had insufficient cell sizes to populate.
Added sugar quintile ranges: Q1, 1·8–49·9 g/d; Q2, 50·2–74·9 g/d; Q3, 75·0–100·9 g/d; Q4, 100·9–147·0 g; Q5, 147·4–412·8 g/d.
*Statistically significant ($P<0·05$) adjusted for physical activity, age, BMI *Z*-score and energy intake.
†Statistically significant compared with Q2 (Wald test, $P<0·05$) adjusted for physical activity, age, BMI *Z*-score and energy intake.
‡Statistically significant compared with Q3 (Wald test, $P<0·05$) adjusted for physical activity, age, BMI *Z*-score and energy intake.

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Table 8 Adjusted prevalence odds ratios (POR) of metabolic syndrome (MetS) biomarkers in relationship to intake of added sugar by quintile among US non-Hispanic black adolescents (aged 12–19 years), National Health and Nutrition Examination Survey 2005–2012: multivariate logistic regression

		Mean added sugar intake									
		Q1: 32·7 g/d	Q2: 60·4 g/d		Q3: 84·7 g/d		Q4: 117·7 g/d		Q5: 190·7 g/d	
MetS biomarker/ indicator	Sample size		POR	95 % CI	POR	95 % CI	POR	95 % CI	POR	95 % CI
TAG	425	Ref.	19·0*	2·0, 184·2	13·6*	1·0, 182·6	10·2	0·6, 166·7	20·1*	1·4, 293·8
HDL-C	468	Ref.	1·2	0·4, 3·7	1·1	0·4, 3·1	1·5	0·5, 4·8	2·3	0·6, 8·2
TAG:HDL-C	425	Ref.	1·7	0·8, 3·5	0·8	0·3, 2·4	1·0	0·5, 2·2	0·8	0·3, 2·4
BG	425	Ref.	2·1	0·9, 5·2	1·8	0·6, 5·3	0·9	0·3, 3·2	0·7†,‡	0·2, 2·2
Insulin	417	Ref.	4·5*	2·0, 9·9	2·6*	1·1, 5·8	2·1	0·7, 6·4	2·1	0·7, 6·6
HOMA-IR	417	Ref.	3·3*	1·4, 7·6	2·5	0·9, 7·0	1·1	0·3, 3·8	1·2	0·3, 5·0
WC	460	Ref.	0·4	0·1, 2·0	0·8	0·3, 2·6	1·6	0·4, 5·6	1·3	0·3, 6·1
Uric Acid	462	Ref.	0·9	0·4, 1·7	1·1	0·4, 2·8	1·3	0·5, 3·6	1·2	0·5, 2·7
ALT	457	Ref.	1·7	0·8, 3·7	1·0	0·4, 2·8	0·8	0·3, 2·6	1·3	0·4, 3·8

HDL-C, HDL cholesterol; BG, blood glucose; HOMA-IR, homeostasis model assessment of insulin resistance; WC, waist circumference; ALT, alanine aminotransferase; Ref., reference category.
Systolic and diastolic blood pressure and MetS had insufficient cell sizes to populate.
Added sugar quintile ranges: Q1, 0–48·1 g/d; Q2, 48·1–72·4 g/d; Q3, 72·5–99·9 g/d; Q4, 100·0–137·9 g/d; Q5, 137·9–480·4 g/d.
*Statistically significant ($P < 0·05$) adjusted for physical activity, age, BMI *Z*-score and energy intake.
†Statistically significant compared with Q2 (Wald test, $P < 0·05$) adjusted for physical activity, age, BMI *Z*-score and energy intake.
‡Statistically significant compared with Q3 (Wald test, $P < 0·05$) adjusted for physical activity, age, BMI *Z*-score and energy intake.

sugar availability of 628 kJ (150 kcal)/person per d was associated with an elevenfold increase in prevalence of diabetes ($P < 0·001$), after controlling for obesity, poverty, urbanization, ageing and physical activity[12]. Likewise, in a study of US adults participating in NHANES 1988–1994, 1999–2004 and 2005–2010, adjusted hazard ratios for CVD mortality were 1·3 (95 % CI 1·09, 1·55) and 2·75 (95 % CI 1·4, 5·42), respectively, comparing participants who consumed 10–24·9 % or ≥25 % of daily energy from added sugar with participants who consumed <10 % of daily energy from added sugar[11]. A previous study among white, African-American and Mexican-American adolescents participating in NHANES during 1999–2004 found an association between the intake of sugar-sweetened beverages and markers of MetS (e.g. blood glucose, HDL-C, TAG, hypertension and WC)[42]. Importantly, our study looked specifically at added sugar from all foods in US adolescents.

In contrast, a study among adults (>20 years of age) participating in NHANES 1999–2006 found that total sugar consumption was not associated with MetS indicators (e.g. TAG, HDL-C, HbA_{1c}, uric acid, blood pressure, WC and BMI)[43]. However, in that study, the measure of total sugar did not distinguish between added sugars and endogenous sugars naturally occurring in fruits and vegetables. Distinguishing between added and natural sugars is important; the sugars inherent in fruits and vegetables do not result in insulin resistance in the same way added sugars do, consistent with our data. The slow rate of digestion of sugar in relation to the high fibre content and the high micronutrient and antioxidant content of fruits and vegetables may alter the intestinal microbiome[44], reduce monosaccharide absorption and protect against systemic insulin resistance[45]. Therefore, studies that group all sugar intakes together may show an attenuation of the association between intake of added sugar and MetS.

Lastly, even though Mexican-American adolescents had a reduced quantity of added sugar consumption compared with non-Hispanic whites, they had a higher prevalence of MetS. This observation is likely multifactorial and may include a greater prevalence of the *PNPLA3* gene, which is associated with higher liver fat content in the presence of dietary sugar[46], higher overweight and obesity rates[6], as well as higher abdominal obesity, consistent with prior findings among US adolescent and adult populations[47,48].

Our findings have a number of limitations. First, the cross-sectional design prevents us from inferring temporal sequence between added sugar intake and MetS biomarkers. Second, the dietary recall method is subject to social desirability bias and imprecision, particularly among overweight/obese adolescents[49], who tend to under-report food and beverage consumption. However, this independent differential misclassification of exposure would diminish the strength of the association between added sugar intake and MetS, thus in fact strengthening our confidence in the significance of our conservative results. Third, using dietary intake data from only two time points introduces an

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additional source of measurement error due to intra-individual variability (beyond misreporting) since it is 'usual' consumption that we would like to measure and believe could be associated with our physiological outcomes. The additional within-person random error persists even after we convert mean sugar consumption to consumption quintiles; i.e. some subjects may have consumed an unusual amount of added sugar on the days of measurement and thus their quintile based on the two-day average could differ from one based on usual consumption. The consequence of this measurement error brought by day-to-day variation in added sugar consumption is that our estimates of the associations between added sugar quintile and health outcome will be biased towards the null (conservative). Unfortunately, the same measurement error exists for one of our covariates (total energy intake) and the effect of this error on the association between sugar and health outcome is less clear. Fourth, because of the wide distribution of added sugar in foods and beverages, and limitations in the available dietary databases, the method of calculating added sugar in the diet could contribute error. Fifth, generalizability of our results is limited to non-Hispanic white, non-Hispanic black and Mexican-American adolescents. Sixth, subject exclusion was greatly affected by subjects who were not randomly selected by NHANES to provide serum samples (n 1711). In our analyses, the result of this diminished sample size is that it likely widened our confidence intervals, but it unlikely biased the trend of our results. And lastly, our imperfect physical activity classification may result in incomplete adjustment of the effects of physical activity on MetS.

The primary underlying causes of MetS have been suggested to be visceral adiposity and insulin resistance[20]. Roughly 80 % of obese adults (BMI >30·0 kg/m^2) manifest MetS components, including elevated TAG and blood glucose levels, large WC, hypertension and low HDL-C levels[41,50]. At the same time, up to 40 % of normal weight (BMI <25·0 kg/m^2) adults also manifest these same co-morbidities[41,50], likely due to a combination of environmental and lifestyle factors, as well as genetic variants. Since paediatric MetS predicts adulthood MetS and type 2 diabetes[5], it is imperative to prevent further metabolic derailment during childhood and adolescence. Our findings suggest that added sugar intake is directly associated with MetS among non-Hispanic white, non-Hispanic black and Mexican-American US adolescents, and independent of total energy intake, physical activity or BMI Z-score.

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References

1. United Nations General Assembly (2011) Political Declaration of the High-Level Meeting of the General Assembly on the Prevention and Control of Non-Communicable Diseases. http://www.un.org/ga/search/view_doc.asp?symbol=A/66/L.1 (accessed January 2015).
2. US Department of Health and Human Services, National Institutes of Health, National Heart, Lung, and Blood Institute (2015) What Is Metabolic Syndrome? http://www.nhlbi.nih.gov/health/health-topics/topics/ms/ (accessed April 2015).
3. Perito ER, Rodriguez LA & Lustig RH (2013) Dietary treatment of nonalcoholic steatohepatitis. *Curr Opin Gastroenterol* **29**, 170–176.
4. Graham RC, Burke A & Stettler N (2009) Ethnic and sex differences in the association between metabolic syndrome and suspected nonalcoholic fatty liver disease in a nationally representative sample of US adolescents. *J Pediatr Gastroenterol Nutr* **49**, 442–449.
5. Magnussen CG, Koskinen J, Chen W *et al.* (2010) Pediatric metabolic syndrome predicts adulthood metabolic syndrome, subclinical atherosclerosis, and type 2 diabetes mellitus but is no better than body mass index alone: the Bogalusa Heart Study and the Cardiovascular Risk in Young Finns Study. *Circulation* **122**, 1604–1611.
6. Fryar CD, Carroll MD, Ogden CL *et al.* (2012) Prevalence of obesity among children and adolescents: United States, trends 1963–1965 through 2009–2010. NCHS Health E-Stats. http://www.cdc.gov/nchs/data/hestat/obesity_child_11_12/obesity_child_11_12.htm (accessed January 2015).
7. Whitlock EP, O'Connor EA, Williams SB *et al.* (2008) *Effectiveness of Weight Management Programs in Children and Adolescents*. Rockville, MD: Agency for Healthcare Research and Quality (US).
8. Butryn ML, Wadden TA, Rukstalis MR *et al.* (2010) Maintenance of weight loss in adolescents: current status and future directions. *J Obes* **2010**, 789280.
9. Sigman-Grant M & Morita J (2003) Defining and interpreting intakes of sugars. *Am J Clin Nutr* **78**, issue 4, 815S–826S.
10. Welsh JA, Sharma AJ, Grellinger L *et al.* (2011) Consumption of added sugars is decreasing in the United States. *Am J Clin Nutr* **94**, 726–734.
11. Yang Q, Zhang Z, Gregg EW *et al.* (2014) Added sugar intake and cardiovascular diseases mortality among US adults. *JAMA Intern Med* **174**, 516–524.

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12. Basu S, Yoffe P, Hills N *et al.* (2013) The relationship of sugar to population-level diabetes prevalence: an econometric analysis of repeated cross-sectional data. *PLoS One* **8**, e57873.
13. Zhang Z, Gillespie C, Welsh JA *et al.* (2015) Usual intake of added sugars and lipid profiles among the US adolescents: National Health and Nutrition Examination Survey, 2005–2010. *J Adolesc Health* **56**, 352–359.
14. >Singh GM, Micha R, Khatibzadeh S *et al.* (2015) Estimated global, regional, and national disease burdens related to sugar-sweetened beverage consumption in 2010. *Circulation* **132**, 639–666.
15. Welsh JA, Sharma A, Cunningham SA *et al.* (2011) Consumption of added sugars and indicators of cardiovascular disease risk among US adolescents. *Circulation* **123**, 249–257.
16. Lustig RH, Mulligan K, Noworolski SM *et al.* (2016) Isocaloric fructose restriction and metabolic improvement in children with obesity and metabolic syndrome. *Obesity (Silver Spring)* **24**, 453–460.
17. Centers for Disease Control and Prevention (2012) National Health and Nutrition Examination Survey. NCHS Research Ethics Review Board Approval. http://www.cdc.gov/nchs/nhanes/irba98.htm (accessed April 2015).
18. National Center for Health Statistics (editor) (2013) *National Health and Nutrition Examination Survey: Analytic Guidelines, 1999–2010*. Hyattsville, MD: US Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Health Statistics.
19. Zimmet P, Alberti KG, Kaufman F *et al.* (2007) The metabolic syndrome in children and adolescents – an IDF consensus report. *Pediatr Diabetes* **8**, 299–306.
20. Alberti KG, Zimmet P & Shaw J (2006) Metabolic syndrome – a new world-wide definition. A Consensus Statement from the International Diabetes Federation. *Diabet Med* **23**, 469–480.
21. Centers for Disease Control and Prevention (2015) National Health and Nutrition Examination Survey. Home page. http://www.cdc.gov/nchs/nhanes.htm (accessed March 2015).
22. US Department of Agriculture, Agricultural Research Service (2014) Food Patterns Equivalents Database. http://www.ars.usda.gov/Services/docs.htm?docid=23869 (accessed January 2015).
23. Matthews DR, Hosker JP, Rudenski AS *et al.* (1985) Homeostasis model assessment: insulin resistance and β-cell function from fasting plasma glucose and insulin concentrations in man. *Diabetologia* **28**, 412–419.
24. Gagliardi AC, Miname MH & Santos RD (2009) Uric acid: a marker of increased cardiovascular risk. *Atherosclerosis* **202**, 11–17.
25. McLaughlin T, Reaven G, Abbasi F *et al.* (2005) Is there a simple way to identify insulin-resistant individuals at increased risk of cardiovascular disease? *Am J Cardiol* **96**, 399–404.
26. Armato J, Reaven G & Ruby R (2015) Triglyceride/high-density lipoprotein cholesterol concentration ratio identifies accentuated cardio-metabolic risk. *Endocr Pract* (Epublication ahead of print version).
27. Abbasi F, Brown BW, Lamendola C *et al.* (2002) Relationship between obesity, insulin resistance, and coronary heart disease risk. *J Am Coll Cardiol* **40**, 937–943.
28. Centers for Disease Control and Prevention (2002) *2000 CDC Growth Charts for the United States*. Hyattsville, MD: Public Health Service, Centers for Disease Control and Prevention, National Center for Health Statistics.
29. Ford ES, Li C, Cook S *et al.* (2007) Serum concentrations of uric acid and the metabolic syndrome among US children and adolescents. *Circulation* **115**, 2526–2532.
30. Sumner AE, Finley KB, Genovese DJ *et al.* (2005) Fasting triglyceride and the triglyceride–HDL cholesterol ratio are not markers of insulin resistance in African Americans. *Arch Intern Med* **165**, 1395–1400.
31. Sumner AE, Harman JL, Buxbaum SG *et al.* (2010) The triglyceride/high-density lipoprotein cholesterol ratio fails to predict insulin resistance in African-American women: an analysis of Jackson Heart Study. *Metab Syndr Relat Disord* **8**, 511–514.
32. Giannini C, Santoro N, Caprio S *et al.* (2011) The triglyceride-to-HDL cholesterol ratio association with insulin resistance in obese youths of different ethnic backgrounds. *Diabetes Care* **34**, 1869–1874.
33. Schwimmer JB, Dunn W, Norman GJ *et al.* (2010) SAFETY study: alanine aminotransferase cutoff values are set too high for reliable detection of pediatric chronic liver disease. *Gastroenterology* **138**, 1357–1364.e2.
34. Welsh JA, Karpen S & Vos MB (2013) Increasing prevalence of nonalcoholic fatty liver disease among United States adolescents, 1988–1994 to 2007–2010. *J Pediatr* **162**, 496–500.e1.
35. Schneider ALC, Lazo M, Selvin E *et al.* (2014) Racial differences in nonalcoholic fatty liver disease in the US population. *Obesity (Silver Spring)* **22**, 292–299.
36. Nguyen S, Choi HK, Lustig RH *et al.* (2009) Sugar-sweetened beverages, serum uric acid, and blood pressure in adolescents. *J Pediatr* **154**, 807–813.
37. Bremer AA, Mietus-Snyder M & Lustig RH (2012) Toward a unifying hypothesis of metabolic syndrome. *Pediatrics* **129**, 557–570.
38. Walker RW, Dumke KA & Goran MI (2014) Fructose content in popular beverages made with and without high-fructose corn syrup. *Nutrition* **30**, 928–935.
39. Havel PJ (2005) Dietary fructose: implications for dysregulation of energy homeostasis and lipid/carbohydrate metabolism. *Nutr Rev* **63**, 133–157.
40. Tappy L & Lê K-A (2010) Metabolic effects of fructose and the worldwide increase in obesity. *Physiol Rev* **90**, 23–46.
41. Lustig RH (2013) Fructose: it's 'alcohol without the buzz'. *Adv Nutr* **4**, 226–235.
42. Bremer AA, Auinger P & Byrd RS (2009) Relationship between insulin resistance-associated metabolic parameters and anthropometric measurements with sugar-sweetened beverage intake and physical activity levels in US adolescents: findings from the 1999–2004 National Health and Nutrition Examination Survey. *Arch Pediatr Adolesc Med* **163**, 328–335.
43. Sun SZ, Anderson GH, Flickinger BD *et al.* (2011) Fructose and non-fructose sugar intakes in the US population and their associations with indicators of metabolic syndrome. *Food Chem Toxicol* **49**, 2875–2882.
44. Zhang L, Li HT, Shen L *et al.* (2015) Effect of dietary resistant starch on prevention and treatment of obesity-related diseases and its possible mechanisms. *Biomed Environ Sci* **28**, 291–297.
45. Ludwig DS (2013) Examining the health effects of fructose. *JAMA* **310**, 33–34.
46. Davis JN, Lê K-A, Walker RW *et al.* (2010) Increased hepatic fat in overweight Hispanic youth influenced by interaction between genetic variation in PNPLA3 and high dietary carbohydrate and sugar consumption. *Am J Clin Nutr* **92**, 1522–1527.
47. Cook S, Weitzman M, Auinger P *et al.* (2003) Prevalence of a metabolic syndrome phenotype in adolescents: findings from the third national health and nutrition examination survey, 1988–1994. *Arch Pediatr Adolesc Med* **157**, 821–827.
48. Ervin RB (2009) Prevalence of metabolic syndrome among adults 20 years of age and over, by sex, age, race and ethnicity, and body mass index: United States. *Natl Health Stat Report* issue 13, 1–7.
49. Briefel RR, Sempos CT, McDowell MA *et al.* (1997) Dietary methods research in the third National Health and Nutrition Examination Survey: underreporting of energy intake. *Am J Clin Nutr* **65**, 4 Suppl., 1203S–1209S.
50. Voulgari C, Tentolouris N, Dilaveris P *et al.* (2011) Increased heart failure risk in normal-weight people with metabolic syndrome compared with metabolically healthy obese individuals. *J Am Coll Cardiol* **58**, 1343–1350.

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