

ORIGINAL ARTICLE

Sugar-sweetened beverages consumption in relation to changes in body fatness over 6 and 12 years among 9-year-old children: the European Youth Heart Study

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BACKGROUND/OBJECTIVES: In parallel with the obesity epidemic, consumption of sugar-sweetened beverages (SSB) has risen over the same period. Our aim was to investigate associations between the consumption of SSB in childhood and adolescence with subsequent changes in body fatness in early adulthood.

SUBJECTS/METHODS: A longitudinal study of 9-year-old children ($n = 283$) enrolled in the Danish part of the European Youth Heart Study with a 6-year and 12-year follow-up. Data were collected at ages 9, 15 and 21 years. Multivariate regression analyses with adjustment for potential confounders were used to evaluate the effect of SSB consumption at 9 and 15 years and change in SSB consumption from 9–15 years on subsequent change in body fatness until 21 years.

RESULTS: Subjects who consumed more than one serve of SSB daily at age 15 years had larger increases in body mass index (BMI) ($\beta = 0.92$, $P = 0.046$) and waist circumference (WC) ($\beta = 2.69$, $P = 0.04$) compared to non-consumers over the subsequent 6 years. In addition, subjects who increased their SSB consumption from age 9–15 years also had larger increases in BMI ($\beta = 0.91$, $P = 0.09$) and WC ($\beta = 2.72$, $P = 0.04$) from 15–21 years, compared to those who reported no change in consumption. No significant association was observed from 9–21 years.

CONCLUSION: This study provides new evidence that SSB consumption in adolescence and changes in SSB consumption from childhood to adolescence are both significant predictors of change in body fatness later in early adulthood.

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INTRODUCTION

In recent years, the consumption of sugar-sweetened beverages (SSB) has steadily increased across the globe.¹ Studies have shown that high SSB consumption is linked with obesity in children and adults^{2–4} as well as metabolic syndrome, type 2 diabetes and cardiovascular disease in adults.^{5–7} Recent evidence from randomized controlled trials supports a causal link between SSB consumption and obesity.^{8–10} Despite these studies, there are still many unanswered questions regarding the association between SSB consumption and obesity development such as the importance of age and life stage, the dose-response relationship and the biological mechanisms involved.^{11,12}

Several mechanisms have been proposed to explain the association between SSB and obesity. The most prevailing theory is that low satiety of liquid energy leads to an incomplete compensatory reduction in energy intake (EI) from other foods, which results in an overall increase in energy intake, positive energy balance and subsequent weight gain.^{13,14} Apart from total energy, the type of carbohydrate in SSB may also contribute to obesity. The rapidly absorbable carbohydrates (i.e., sucrose or high fructose corn syrup) used to sweeten SSB can contribute to a high glycaemic load, which has been associated with high insulin

response and increased risk of body fat and weight gain.¹⁵ Hepatic metabolism of fructose is also thought to promote body fat gain through an increase in *de-novo* lipogenesis.¹⁶ High SSB consumption may also be a marker of unhealthy dietary patterns that promote weight gain.

We recently examined the prospective associations between SSB consumption in childhood at age 9 y and subsequent 6 y change in body fatness in Danish children from the European Youth Heart Study, and found that SSB consumption was associated with body fatness, and further, that such association was mediated through EI and fasting insulin levels.¹⁷ However, the association between SSB consumption and body weight may vary with each life stage due to differences in physiological development, social and environmental factors. Indeed, most of the evidence is obtained from studies conducted in children or older adults. Few studies are available among older adolescents and young adults in a longitudinal study design with three time points, that is, measuring change of exposure and the subsequent change in outcome. The current study, a longitudinal design, used the same cohort of Danish children but with a third measurement point and a longer follow-up period. We examined whether SSB consumption in childhood (at age 9 years) and adolescence

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(at age 15 years) or change in consumption from childhood to adolescence were associated with subsequent change in body fatness until early adulthood (at age 21 years). Furthermore, the effects of potential mediators, total EI and insulin sensitivity/secretion, were also examined.

MATERIALS AND METHODS

The European Youth Heart Study (EYHS) is an international multicenter study designed to identify environmental, personal, lifestyle and physiological causes of early development of cardiovascular disease from childhood to adulthood. All study protocols complied with the Declaration of Helsinki and was approved by the scientific ethics committee of the local counties of Vejle and Funen, Demark (VF 20030067).¹⁸ This study includes data on 9 year-old children who participated in the Danish part of EYHS in 1997 with a 12-year follow-up. A total of twenty-eight schools in Odense, Denmark were asked to participate, of which twenty-five schools agreed. Schools were stratified according to school type, location and socioeconomic character of local area. A proportional, two-stage cluster sampling was used for sample selection of schools, and children were randomly selected within each school. Data collection was conducted throughout the school year to minimize seasonal effects, and daily order of testing was standardized to minimize between-test interaction.¹⁹

Subjects

In 1997, 590 children aged 9 years (3rd grade) took part in the baseline interview (Figure 1). The dropout rate from baseline to first follow-up interview in 2003 was 34.9%, resulting in 384 children. Of these, 237 children participated in the second follow-up in 2009 and a further 50 were excluded due to incomplete information on anthropometrics and dietary intake ($n = 25$) or who underreported EI ($n = 25$). A total of 187 children (98 girls and 89 boys) were included in the analysis that involved all three time points (Figure 1a). For analysis involving only two time points (9 and 21 years), the dropout rate from 9–21 years was 52.4% ($n = 281$). Children with incomplete information on anthropometrics and dietary intake ($n = 25$) or underreported EI ($n = 1$) were excluded, resulting in a final cohort of 283 children (158 girls and 125 boys) in the 9–21 years analysis (Figure 1b). Attrition analyses showed that the characteristics of participants and those who dropped out were essentially similar from baseline to first follow-up²⁰ and from first to second follow-up (Supplementary Table 1S).

Anthropometry

Anthropometry was assessed by standard anthropometric procedures at all three time points. Height was measured bare feet to the nearest 5 mm using a stadiometer. Weight was measured in light clothing to the nearest 0.1 kg using a beam balance scale. Body mass index (BMI) was calculated as weight in kilograms per square of height in meters. Waist circumference (WC) was measured twice with a metal anthropometric tape midway between the lower rib margin and the iliac crest, at the end of gentle expiration. The mean value of the two measurements was used for analysis. Skinfold thickness was measured with Harpenden fat calipers with two measurements taken at each site. The sum of four skinfolds ($\Sigma 4SF$) was obtained by adding the average skinfolds of the biceps, triceps, subscapular and suprailiac.²¹ BMI, WC and $\Sigma 4SF$ are used as indicators for total body fatness, visceral fat and subcutaneous fat, respectively.

Dietary intake

Dietary intake was collected using a 24 h recall face-to-face interview supplemented with a qualitative food record from the same day at baseline and the first follow-up.²² All children completed a qualitative food record at home, followed the next day by a 20–30 min recall interview. The interviews were conducted from Monday to Friday by the same interviewer. At baseline, the 9 year-olds were assisted by parents for obtaining valid estimate of food intake. During the interview, different-sized drinking glasses, plates, spoons and food pictures of most common foods and food groups in different portion sizes were used to estimate food quantities. This method has been shown as valid to assess dietary intake of children for the purpose of group comparison.²³ Food recalls were entered into the software program Dankost 3000 (Danish Catering Centre, Copenhagen, Denmark) for nutrient analysis using the Danish Food Composition Tables 2006.²⁴ SSB were defined as regular soft drinks, fruit drinks and cordials sweetened with caloric sweeteners but excluded 100% fruit juice, flavored milk, coffee and tea. Per capita consumption (oz per day) of SSB and 100% fruit juice were calculated.

Identification of underreporting

The Goldberg cut-off method was applied to identify under-reporters, based on the ratio of reported EI to basal metabolic rate.²⁵ Basal metabolic rate was estimated using the Schofield equation.²⁶ The lower 95th percentiles lower cut-off value of 0.9 was applied to all individuals.²⁵

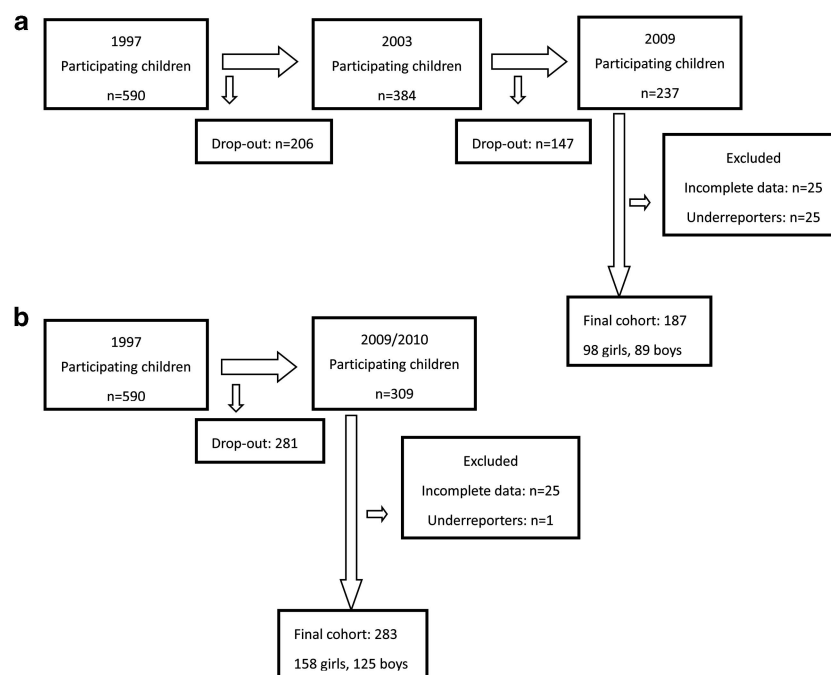


Figure 1. Flow chart showing participation of study population: (a) children participating at baseline, 1st follow-up and 2nd follow-up; (b) children participating at baseline and 2nd follow-up.

All under-reporters were excluded for analysis: one girl aged 9 years, and 19 girls and 6 boys aged 15 years.

Confounding factors

Physical activity was measured by both a computer-based questionnaire and validated accelerometer. Detailed study procedures have been published previously.¹⁹ Since a large number of participants failed to complete the accelerometer measurements, it was decided to use questionnaire data to maximize sample size. This categorized participants as physically inactive (reported 'no' or 'sometime exercise') or active (reported as 'regular exercise'). Comparison of questionnaire and accelerometer data revealed that all estimates for accelerometer data were in the same direction but no longer significant, likely due to less statistical power (Supplementary Table 2S). Pubertal status was assessed by trained personnel according to Tanner's five development stages.²⁷ Maternal education was used as the socioeconomic status indicator of choice²⁸ and was grouped as low (reported as elementary, high school or vocational education) or high (reported as short, medium or long-term tertiary education).

Blood samples

Blood samples were taken from the antecubital vein after an overnight fast and analysed in a WHO-certified laboratory at baseline (age 9 years) and at first follow-up (age 15 years). Fasting glucose level was measured by standard methods using Olympus autoanalyser (model AU600, Olympus Diagnostica GmbH, Hamburg, Germany). Fasting insulin level was measured by two-site enzyme immunoassay with either 125I or alkaline phosphate labels. HOMA was used to estimate insulin sensitivity ($HOMA-IR = (\text{fasting glucose (mmol/l)} \times \text{fasting insulin } (\mu\text{U/ml}))/22.5$) and β -cell function ($HOMA-\beta = (\text{fasting insulin } (\mu\text{U/ml}) \times 20)/(\text{fasting glucose (mmol/l)} / 3.5)$). These indices have been validated as surrogate markers of insulin sensitivity and secretion in healthy children.²⁹

Statistical methods

Descriptive analyses were undertaken for participants at baseline and follow-up. Multiple linear regression analyses were used to examine the associations between SSB consumption and changes in body fatness. Exposure variables were SSB consumption at ages 9 and 15 years as well as the change in SSB consumption from 9 to 15 years. Due to a high number of non-consumers, SSB consumption was categorized into three categories: non-consumers, ≤ 1 serve per day and > 1 serve per day (one serve = 12oz). Change in SSB consumption between ages 9 to 15 years was calculated by subtracting the intake at 9 years from intake at 15 years and was categorized into three categories: decrease (< 0), no change ($= 0$, mostly subjects who were non-consumers at both time points) or increase (> 0). Outcome variables were changes in BMI, WC and $\Sigma 4SF$ from 9 to 21 years (ΔBMI_{9-21} , ΔWC_{9-21} , $\Delta \Sigma 4SF_{9-21}$) and from 15 to 21 years (ΔBMI_{15-21} , ΔWC_{15-21} , $\Delta \Sigma 4SF_{15-21}$).

Model 1 included only exposure and outcome variables. Model 2 included potential confounders: age, gender, baseline BMI/WC/ $\Sigma 4SF$, physical activity, socioeconomic status and pubertal status. Baseline intake of SSB was also included as a confounder, when effects of change in SSB consumption on subsequent change in body fatness were estimated. Confounding factors that are theoretically important or associated with outcome variables at a P -value < 0.25 were included in the multiple regression models.³⁰ Model 3 included total energy intakes, as this may be a confounder if SSB is a marker of other weight-promoting dietary factors, or may be a part of the causal pathway linking SSB consumption and obesity. In order to examine the mediating effect of insulin sensitivity/secretion on the association, further adjustment for HOMA-IR and HOMA- β were performed separately as the correlation between these two indices was high ($r = 0.79$ at 9 years and $r = 0.54$ at 15 years, both $P < 0.001$). As similar results were found following adjustment for either HOMA-IR or HOMA- β , only the results for HOMA-IR are presented in the table (Model 4). Tests for linear trend were performed among SSB consumption categories using regression analysis. Additional sensitivity analyses were performed using (1) liquid sucrose content of SSB and (2) the inclusion of 100% fruit juice as part of SSB. Liquid sucrose content was defined as added sugar in SSB (1 serve = 40 g), as sucrose is the major added sugar source in Denmark. Statistical significance was set at $P < 0.05$ (two-tailed). All statistical analyses were performed in SPSS version 20.0 (IBM Corp, Armonk, NY, USA).

RESULTS

The distribution of baseline and follow-up characteristics according to the category of SSB consumption is presented in Table 1. At both baseline and follow-up, age, BMI, WC and $\Sigma 4SF$ were not significantly different among SSB categories. However, greater SSB consumption was associated with higher intakes of energy and greater % energy from total sugar, but lower % energy from protein ($P < 0.001$). Fasting glucose, fasting insulin, HOMA-IR and HOMA- β were not significantly different among SSB categories. The distribution of pubertal status, socioeconomic status and physical activity was not significantly different among SSB categories, except for gender at follow-up ($P = 0.003$). A higher proportion of boys than girls consumed SSB at first follow-up. Mean SSB consumption among SSB categories at baseline and follow-up as well as the extent of change in SSB from baseline to follow-up are illustrated in Figure 2.

There was no evidence of an association between SSB consumption at age 9 years and changes in BMI and WC until age 21 years in either unadjusted or adjusted models (Table 2). However, at age 15 years, participants who consumed more than one serve of SSB had a significantly greater increase in WC ($\beta = 2.69$, $P = 0.04$) and BMI ($\beta = 0.92$, $P = 0.046$) from 15–21 years relative to non-consumers after adjusting for potential confounders (Table 2). In addition, the overall trend analysis between SSB categories at age 15 years and change in body fatness measures from 15–21 years adjusted for all confounders revealed positive linear trend ($P = 0.08$ for ΔBMI and $P = 0.048$ for ΔWC , data not shown). Further adjustment for EI or HOMA indices attenuated these associations slightly ($P > 0.05$).

The analyses examining the association between change in SSB consumption from 9–15 years and subsequent changes in BMI and WC from 15–21 years revealed similar findings (Table 2). Compared to the group reporting no change in consumption, the group that increased SSB consumption from 9–15 years had a significantly higher increase in WC ($\beta = 2.72$, $P = 0.04$), and a non-significant higher increase in BMI ($\beta = 0.91$, $P = 0.09$) with adjustment for potential confounders. No evidence of a linear trend was found among the three categories of change in SSB consumption ($P = 0.29$ for ΔBMI and $P = 0.17$ for ΔWC , data not shown). Adjusting for EI or HOMA indices had little effect on the association between change in SSB consumption and changes in BMI or WC. The results for change in $\Sigma 4SF$ were in the same direction as BMI and WC but were not statistically significant.

Sensitivity analyses using sucrose content of SSB instead of SSB serves (12oz) as the exposure variable showed stronger associations with BMI and WC (Supplementary Table 3S). However, when sugary drinks, including both SSB and 100% fruit juice, were analysed as exposure variables, no significant association with change in body fatness was observed compared to SSB alone (Supplementary Table 4S).

DISCUSSION

Our study investigated the impact of SSB consumption on the development of body fatness from childhood (age 9 years) and adolescence (age 15 years) to early adulthood (age 21 years) in a cohort of Danish children. We found SSB consumption at age 15 years was associated with changes in body fatness from age 15 to 21 years. The 15-year-olds who consumed more than one daily serve of SSB (12oz) increased their subsequent 6-years BMI and WC by 0.92 kg/m^2 and 2.69 cm , respectively, compared to non-consumers. The possible role of SSB consumption in the development of body fatness was further illustrated by children who increased their SSB consumption between 9 and 15 years. They had a larger subsequent increase in body fatness from 15 to 21 years.

This is the one of the first studies to demonstrate a direct association between SSB consumption and change in body

Table 1. Characteristics of participants at baseline and follow-up according to SSB consumption^a

	1997 (n = 283)				2003 (n = 187)			
	Non-consumer (n = 134)	≤1 serve per day (n = 112)	> 1 serve per day (n = 37)	P-value ^b	Non-consumer (n = 94)	≤1 serve per day (n = 44)	> 1 serve per day (n = 49)	P-value ^b
<i>Continuous variable (Mean ± s.d.)</i>								
Age (years)	9.6 ± 0.4	9.6 ± 0.4	9.6 ± 0.3	0.12	15.7 ± 0.4	15.7 ± 0.3	15.8 ± 0.3	0.08
Height (cm)	139.3 ± 6.4	139.7 ± 6.4	139.4 ± 5.3	0.88	169.7 ± 7.9	171.5 ± 9.1	174.4 ± 10.2	0.01
Weight (kg)	33.8 ± 6.0	34.1 ± 6.1	33.6 ± 5.4	0.88	60.5 ± 9.6	63.0 ± 10.8	64.1 ± 12.0	0.12
Body mass index (kg/m ²)	17.3 ± 2.5	17.4 ± 2.2	17.2 ± 1.9	0.93	21.0 ± 2.9	21.4 ± 2.9	20.9 ± 2.6	0.66
Waist circumference (mm)	58.6 ± 5.9	58.9 ± 5.5	58.0 ± 4.1	0.69	73.3 ± 6.9	74.2 ± 6.1	75.0 ± 7.2	0.33
Sum of four skinfold (mm)	33.5 ± 12.1	38.5 ± 16.6	37.5 ± 18.7	0.32	37.7 ± 16.4	44.5 ± 19.4	43.0 ± 18.3	0.15
Total energy (MJ/d)	9.0 ± 2.0	9.4 ± 2.3	10.2 ± 2.6	<0.001	9.3 ± 3.0	9.9 ± 3.2	11.7 ± 3.9	<0.001
Protein (En%)	13.8 ± 2.6	12.8 ± 2.5	11.5 ± 2.6	<0.001	14.8 ± 2.7	13.1 ± 2.2	11.9 ± 2.9	<0.001
Fat (En%)	32.5 ± 6.7	31.7 ± 6.1	32.9 ± 6.0	0.48	27.5 ± 7.7	27.0 ± 8.0	27.4 ± 7.5	0.95
Carbohydrate (En%)	52.8 ± 6.9	54.6 ± 6.4	54.7 ± 5.8	0.06	56.8 ± 8.1	58.8 ± 8.2	59.5 ± 8.3	0.13
Total sugar (En%)	17.0 ± 5.7	21.6 ± 6.0	23.6 ± 5.0	<0.001	18.2 ± 7.4	21.4 ± 6.8	25.3 ± 9.6	<0.001
Fasting glucose (mmol/l)	5.1 ± 0.4	5.1 ± 0.4	5.1 ± 0.4	0.85	4.9 ± 0.3	5.1 ± 0.4	5.1 ± 0.4	0.65
Fasting insulin (pmol/l)	46.8 ± 22.6	46.2 ± 21.5	55.0 ± 19.5	0.13	58.9 ± 26.9	62.0 ± 26.8	58.7 ± 25.9	0.79
HOMA-IR	1.6 ± 0.8	1.5 ± 0.8	1.8 ± 0.7	0.17	1.9 ± 0.9	2.0 ± 0.9	1.9 ± 0.9	0.58
HOMA-β	83.6 ± 36.0	84.9 ± 38.0	99.6 ± 38.7	0.10	127.9 ± 65.7	119.6 ± 54.6	119.4 ± 111.5	0.77
<i>Categorical variable (%)</i>								
Gender								
Boys	44.8	42.9	45.9	0.93	36.2	52.3	65.3	0.003
Girls	55.2	57.1	54.1		63.8	47.7	34.7	
Pubertal stages								
Stage 1	82.7	84.7	89.2	0.65				
Stage 2	17.3	14.4	10.8					
Stage 3	0	0.9	0		4.3	9.1	2	0.08
Stage 4					85.1	84.1	4.1	
Stage 5					10.6	6.8	93.9	
Socioeconomic status								
Low	51.2	59.6	71.9	0.09	31.9	44.2	38.3	0.37
High	48.8	40.4	28.1		68.1	55.8	61.7	
Physical Activity								
Inactive	46.9	39.1	53.6	0.33	34	47.7	31.3	0.20
Active	53.1	60.9	46.4		66	52.3	68.8	

Abbreviation: En%, Percentage energy. ^aContinuous variables were expressed as mean ± standard deviation. Categorical variables were expressed as percentage of each category. ^bSignificance tested by ANOVA for continuous variables and Chi-Squared test for categorical variables.

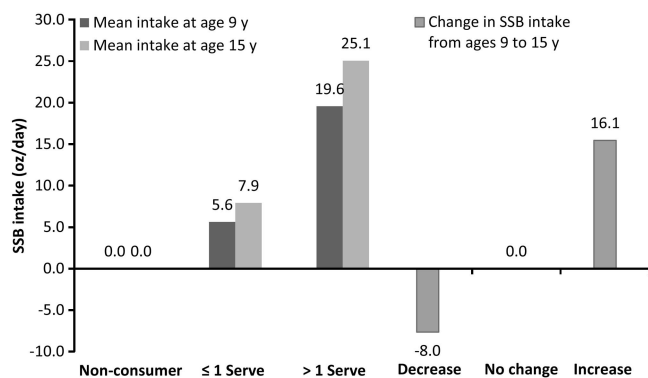


Figure 2. Mean SSB intake (oz per day, 1oz = 28 g) among each SSB category (non-consumer, ≤1 serve per day, >1 serve per day) at ages 9 and 15 years and mean change in SSB intake (oz per day) among changes in SSB from ages 9–15 years (decrease, no change, increase).

fatness from adolescence to early adulthood. One longitudinal British birth cohort study also found subjects who consumed two or more carbonated drinks per day at 16 years significantly predicted an increase in BMI z-score from ages 16–30 years with adjustments for gender, socioeconomic status and height.³¹ Most previous studies examining associations between SSB consumption and obesity were conducted during childhood or from childhood to adolescence.^{5,32,33} Soft drink consumption

predicted excess weight gain in early adolescence in a small cohort of Australian children.³² Similarly, in a study of US girls, greater SSB consumption (≥2 serves per day) at age 5 years was associated with a higher percentage body fat, WC and weight status from 5 to 15 years.³³

The current study is one of the first to investigate a longitudinal association between change in SSB consumption and subsequent change in body fatness from childhood to early adulthood. The study by Kral *et al.*,³⁴ found a positive association between an increase in energy consumed from SSB from ages 3 to 5 years and change in WC from ages 5 to 6 years. Two other studies investigated the association between change in SSB consumption and concordant change in weight. A large US study⁶ found a positive linear association between change in SSB consumption and concordant 1-year change in BMI in boys, but not girls, while a smaller US study³⁵ found no evidence of an association between 2-years change in SSB consumption and concordant change in BMI z-score among a group of children aged 3–5 years, although a positive trend was observed.

Although we found significant association for BMI and WC, no evidence of a significant association was found for the sum of four skinfolds (Σ4SF). This may be attributed to different types of adipose tissue being studied. Σ4SF was a crude measure of subcutaneous adipose tissues, whereas BMI and WC are indicators of total and visceral adipose tissues, respectively. Additionally, Σ4SF was calculated from the Σ4SF thickness measures and may be associated with higher measurement error in relative to direct measure of BMI and WC. Little research has been done to explore the effects of SSB on body fat distribution. One recent

Table 2. Regression analysis results for associations between daily consumption of SSB at ages 9 and 15 years, change in SSB consumption from 9 to 15 years and change in body fatness from 9–21 years and from 15–21 years

	None (n = 134)	ΔBMI_{9-21} (kg/m ²)				ΔWC_{9-21} (cm)				$\Delta\Sigma 4\text{SF}_{9-21}$ (mm)			
		≤ 1 serve per day (n = 112)		> 1 serve per day (n = 37)		≤ 1 serve per day (n = 112)		> 1 serve per day (n = 37)		≤ 1 serve per day (n = 112)		> 1 serve per day (n = 37)	
		$\beta \pm \text{SE}$	P-value	$\beta \pm \text{SE}$	P-value	$\beta \pm \text{SE}$	P-value	$\beta \pm \text{SE}$	P-value	$\beta \pm \text{SE}$	P-value	$\beta \pm \text{SE}$	P-value
SSB consumption (9y) (n = 283)													
^a Model 1	ref	0.50 ± 0.44	0.26	1.25 ± 0.64	0.15	0.68 ± 1.2	0.57	1.93 ± 1.73	0.27	0.22 ± 4.80	0.96	1.43 ± 3.26	0.66
^a Model 2	ref	0.53 ± 0.55	0.34	1.42 ± 0.68	0.29	0.98 ± 1.3	0.46	0.80 ± 2.02	0.69	0.76 ± 5.68	0.79	0.98 ± 3.63	0.79
	None (n = 94)	ΔBMI_{15-21} (kg/m ²)				ΔWC_{15-21} (cm)				$\Delta\Sigma 4\text{SF}_{15-21}$ (mm)			
		≤ 1 serve per day (n = 43)		> 1 serve per day (n = 50)		≤ 1 serve per day (n = 43)		> 1 serve per day (n = 50)		≤ 1 serve per day (n = 43)		> 1 serve per day (n = 50)	
		$\beta \pm \text{SE}$	P-value	$\beta \pm \text{SE}$	P-value	$\beta \pm \text{SE}$	P-value	$\beta \pm \text{SE}$	P-value	$\beta \pm \text{SE}$	P-value	$\beta \pm \text{SE}$	P-value
SSB consumption (15y) (n = 187)													
^b Model 1	ref	0.47 ± 0.56	0.41	1.10 ± 0.54	0.03	2.38 ± 1.47	0.11	3.40 ± 1.40	0.02	1.67 ± 3.49	0.63	2.85 ± 3.65	0.44
^b Model 2	ref	0.66 ± 0.56	0.24	0.92 ± 0.54	0.046	2.57 ± 1.49	0.09	2.69 ± 1.45	0.04	1.79 ± 3.85	0.64	3.20 ± 3.90	0.42
^b Model 3	ref	0.69 ± 0.57	0.23	0.97 ± 0.56	0.09	2.58 ± 1.50	0.09	2.72 ± 1.51	0.07	1.37 ± 3.94	0.73	2.95 ± 3.92	0.45
^b Model 4	ref	0.58 ± 0.56	0.30	0.85 ± 0.54	0.12	2.40 ± 1.49	0.11	2.66 ± 1.46	0.07	1.76 ± 3.88	0.65	3.09 ± 3.93	0.43
	NC (n = 45)	ΔBMI_{15-21} (kg/m ²)				ΔWC_{15-21} (cm)				$\Delta\Sigma 4\text{SF}_{15-21}$ (mm)			
		Decrease (n = 64)		Increase (n = 78)		Decrease (n = 64)		Increase (n = 78)		Decrease (n = 64)		Increase (n = 78)	
		$\beta \pm \text{SE}$	P-value	$\beta \pm \text{SE}$	P-value	$\beta \pm \text{SE}$	P-value	$\beta \pm \text{SE}$	P-value	$\beta \pm \text{SE}$	P-value	$\beta \pm \text{SE}$	P-value
Δ SSB consumption (9–15y) (n = 187)													
^c Model 1	ref	0.41 ± 0.58	0.48	1.05 ± 0.57	0.06	1.12 ± 1.53	0.47	3.31 ± 1.49	0.03	3.11 ± 3.71	0.40	3.89 ± 3.69	0.38
^c Model 2	ref	0.38 ± 0.59	0.51	0.91 ± 0.57	0.09	0.87 ± 1.55	0.57	2.72 ± 1.53	0.04	3.14 ± 3.98	0.43	3.54 ± 3.97	0.38
^c Model 3	ref	0.38 ± 0.61	0.53	1.00 ± 0.59	0.11	0.83 ± 1.61	0.75	3.25 ± 1.53	0.07	4.03 ± 4.09	0.33	4.60 ± 4.09	0.26
^c Model 4	ref	0.49 ± 0.65	0.45	0.98 ± 0.64	0.13	1.22 ± 1.72	0.48	3.05 ± 1.72	0.08	5.79 ± 4.44	0.19	5.31 ± 4.43	0.23

Abbreviations: SSB, sugar-sweetened beverages; β , regression coefficient; SE, standard error; Δ , change; ref, reference category; NC, no change; 1 serve of SSB = 12oz, 1oz = 28g. ^aModel 1: Crude model including SSB consumption at 9years (none, ≤ 1 serve, > 1 serve) and $\Delta\text{BMI}/\Delta\text{WC}/\Delta\Sigma 4\text{SF}$ only. Model 2: Adjusted for age, gender, BMI/WC/ $\Sigma 4\text{SF}$, socioeconomic status, pubertal status and physical activity at age 9years. ^bModel 1: Crude model including SSB consumption at 15years (none, ≤ 1 serve, > 1 serve) and $\Delta\text{BMI}/\Delta\text{WC}/\Delta\Sigma 4\text{SF}$ only. Model 2: Adjusted for age, gender, BMI/WC/ $\Sigma 4\text{SF}$, socioeconomic status, pubertal status and physical activity at age 15years. Model 3: Adjusted for all variables in model 2 and energy intake at age 15years. Model 4: Adjusted for all variables in model 2 and HOMA-IR at age 15years. ^cModel 1: Crude model including change in SSB consumption (decrease, no change, increase) and $\Delta\text{BMI}/\Delta\text{WC}/\Delta\Sigma 4\text{SF}$ only. Model 2: Adjusted for age at 15years, gender, BMI/WC/ $\Sigma 4\text{SF}$ at 15years, SSB consumption at 9years, socioeconomic status, pubertal status and physical activity at 15years. Model 3: Adjusted for all variables in model 2 and change in energy intake from ages 9–15years. Model 4: Adjusted for all variables in model 2 and change in HOMA-IR from ages 9–15years.

experimental study examined the association between sucrose-sweetened beverages intake and fat storage in adults and found significant associations for liver, skeletal muscle and visceral fat, but not for total fat mass.¹⁰

We did not find an association between SSB consumption at age 9 years and subsequent 12-years changes in BMI and WC. This lack of an association is likely to be caused by changes in dietary and lifestyle habits occurring over the rather long 12-years follow-up period, covering puberty and adolescence. Indeed, previous studies on tracking of diet from childhood to adulthood have suggested mixed results,^{36,37} and dietary intake in childhood may not be useful in relation to studying subsequent 12-year changes in body fatness. Only few previous studies have examined the long-term effects of SSB consumption on obesity. One study examined the association using a large cohort of Finnish children with a follow-up of 21 years, and did not find an association between the SSB consumption in childhood and BMI in adulthood.³⁸ However, they reported significant association between increase in SSB consumption from childhood to adulthood and BMI in adulthood in women. Another explanation for lack of significant associations may be the larger measurement error in the assessment of dietary intake among the 9 year-olds in comparison to the 15- and 21- year-olds, since parents in the present study

reported food intake for the 9-year-olds, but adolescents and young adults reported their own intakes. Additionally, SSB consumption at 15 years may be more relevant for adult body composition than consumption during childhood.

The present study examined the potential mediating effects of total EI and insulin sensitivity/secretion on the pathways linking SSB consumption and body fatness. It is assumed that energy from SSB consumption or incomplete compensatory reduction of EI from other sources as a result of energy consumed in liquid form have played a role in the associations.¹⁵ However, our findings suggest that total EI had a relatively minor effect on the association between SSB consumption and body fatness. These results are consistent with some other studies,^{5,33,39,40} but not all,⁶ and it has been suggested that the under-reporting of EI may be responsible.³³

High SSB consumption also contributes to a high glycaemic load and high glycaemic load diets have been associated with increased appetite and food intake as well as a high insulin response, which may in turn affect fuel partitioning and favour weight gain.⁴¹ We only found a minor effect of insulin sensitivity/secretion on the association between SSB consumption and change in body fatness. This is in contrast with our previous study¹⁷ and is likely to be the result of different life stages studied, that is, the transition from adolescence to early adulthood, rather

than from childhood to adolescence. Few studies have examined the role of insulin sensitivity/secretion on the association between SSB and obesity. An experimental study found SSB consumption induced a fast and dramatic increase in both blood glucose and insulin levels and the insulin response was linearly correlated to BMI.⁴² Future research is required to confirm the role of insulin sensitivity/secretion on the association between SSB consumption and body fatness.

Sensitivity analyses showed liquid sucrose was more closely related to changes in body fatness compared to the amount of SSB consumed. This is likely due to small difference in categorization of exposure variables, which may indicate that liquid sucrose is a more sensitive predictor of body fatness. Furthermore, inclusion of 100% fruit juice as part of SSB revealed a weakened effect, suggesting that 100% fruit juice may be less obesogenic than SSB.

Our study has several strengths. The longitudinal nature of our study allows us to examine the association between SSB consumption and body fatness across three life stages: childhood, adolescence and adulthood. We were able to control for many potential confounders such as socioeconomic status and physical activity. Furthermore, this study considered the pubertal status and gender in the analysis, which has been suggested to play a role in development of childhood and adolescent obesity.⁴³ Lastly, although BMI, WC and $\Sigma 4SF$ were crude measures of body fatness, they were measured objectively rather than self-reported, which eliminates the possibility of reporting bias.

There are also a number of limitations to our study. The small sample size of the current study may have hindered our ability to detect significant effects. The larger measurement error of skinfold measures compared to weight, height and WC measures contributed to lower power (<80%), and hence it cannot be excluded that we overlooked true associations for skinfold measures. Due to the observational nature, the study cannot infer causal relationships and the possibility of unmeasured residual confounding cannot be ruled out. Additionally, SSB consumption excluded contemporary beverages such as energy drinks and vitamin waters, but these were assumed to be minimal during the study period. The study results may also be affected by some methodological limitations. Dietary intake was collected by a single 24 h dietary recall, which is not a measure of habitual intake, but provides a valid estimate of mean intake at a group level.²³ Although we did exclude extreme under-reporters, the cut-off limit used is unable to identify all under-reporters. In addition, under-reporting of foods perceived to be unhealthy such as SSB is common, especially among overweight and obese individuals.⁴⁴ However, the fact that we were able to show many consistent and significant associations using the relatively crude estimate of SSB consumption from one single 24 h recall speaks in favour of the true associations being even stronger, and suggests that had we had access to a more precise estimate of intake, the associations would have been even stronger. A final methodological issue was the crude categorization of physical activity into only two groupings. This may have resulted in residual confounding, although sensitivity analysis using accelerometer data was conducted.

In conclusion, we found that adolescents who consumed more than one serve (12 oz) of SSB had higher body fatness as evidenced by BMI and WC in early adulthood. An increase in SSB consumption from childhood to adolescence was a predictor of change in body fatness until early adulthood. However, further high quality, long term longitudinal and intervention studies that examine the role of SSB consumption on body fatness as well as the potential mediating effects of EI and insulin sensitivity/secretion on this association are required.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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LBA was responsible for collection of data, except diet data for which BLH was responsible. NJO calculated the sugar fractions. BLH conceived the hypothesis for the study, MZ analysed data; BLH, MZ and AR interpreted data, wrote the manuscript and reviewed/edited the manuscript. All authors reviewed/edited the final manuscript. We thank all those involved with the European Youth Heart Study.

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