

ORIGINAL ARTICLE

Dietary carbohydrate quantity and quality in relation to obesity: A pooled analysis of three Finnish population-based studies

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Abstract

Background: The relationship between carbohydrate intake, dietary glycaemic index (GI) and load (GL), and obesity remains unsolved. Sugar intake and obesity represent a timely topic, but studies on sugar subcategories are scarce. We aimed to study whether total carbohydrate, sucrose, lactose, fibre, dietary GI, and GL are associated with obesity in 25–79-year-old Finns. **Methods:** Our pooled analysis included three cross-sectional population-based studies: the DILGOM Study ($n = 4842$), the Helsinki Birth Cohort Study ($n = 1979$), and the Health 2000 Survey ($n = 5521$). Diet was assessed by a validated food-frequency questionnaire, and anthropometric measurements were collected by standardised protocols. Pooled odds ratios (OR) and 95% confidence intervals (CI) were calculated using logistic regression analysis. **Results:** In the model, which included sex, age, education, smoking, physical activity, and energy intake, the likelihood of being obese (body mass index ≥ 30 kg/m²) appeared lower in the highest quartiles of total carbohydrate (OR 0.65; 95% CI 0.57–0.74; P for trend < 0.0001), sucrose (OR 0.53; 95% CI 0.47–0.61; $P < 0.0001$), and dietary GL (OR 0.64; 95% CI 0.56–0.73; $P < 0.0001$) compared to the lowest quartiles. In contrast, dietary GI did not associate with obesity. Fibre intake associated inversely with abdominal obesity (OR 0.80; 95% CI 0.71–0.90; $P < 0.001$). The inverse sucrose–obesity relationship appeared stronger in high fruit consumers compared to low fruit consumers (P for interaction 0.02). **Conclusions:** Although most of the studied carbohydrate exposures were associated with a diminished likelihood of being obese, prospective studies are needed to assess temporal relations to support causal inference.

Key Words: Dietary carbohydrate, sucrose, glycaemic index, glycaemic load, fibre, obesity, body mass index, waist circumference, meta-analysis, pooled analysis

Introduction

The rising prevalence of obesity globally is well documented. In a systematic analysis of health examination surveys and epidemiological studies, the highest body mass index (BMI) among high-income countries emerged in the USA, but the Nordic countries do not fall far behind [1]. In Finland, the prevalence of obesity (BMI ≥ 30 kg/m²) has risen between 1972 and 2012,

and has reached the level of 27% in men and 26% in women in some areas [2]. Obesity contributes to the disease burden of type 2 diabetes, cardiovascular disease, and several cancers, and thus, tackling the obesity epidemic requires effective strategies.

The role of carbohydrates in obesity development has been debated. Mechanistically, carbohydrates

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might, independent of their energy-providing nature, affect body weight and adiposity by altering postprandial metabolic processes such as nutrient storage and oxidation, and satiety regulation [3,4]. Thus the glycaemic index (GI), which quantifies the blood glucose-raising potential of carbohydrate-containing foods, and glycaemic load (GL), represent attractive attempts to describe carbohydrate quality. Despite extensive research, the relationship between overall dietary GI/GL and obesity remains unsolved. Studies conducted in 48,631 and 89,432 European Prospective into Cancer and Nutrition study participants, with 5.5- and 6.5-year follow-up times, have not consistently shown a relationship between GI and obesity measures [5,6]. Moreover, dietary GL is rarely associated with obesity in prospective studies involving European populations [6,7]. Overall, studies from Nordic countries, other than Denmark, are widely lacking.

Sugars as a carbohydrate subgroup have attracted growing interest in relation to obesity spurred by recently launched guidelines [8]. Recent meta-analyses of randomised controlled trials (RCT) and cohort studies have provided evidence that sugar-sweetened beverages (SSB) are obesity determinants [9,10] leading to the demand that SSB consumption should be reduced [11]. However, sugar subcategories (e.g. sucrose) in relation to obesity are rarely studied [9] and it is not settled whether high-sugar diets promote weight gain independent of energy intake [4]. Interestingly, inverse associations between self-reported sucrose intake and BMI in adults have been reported – mainly using data from the 1980s and 1990s [12]. It is not known whether this relation holds in different populations who are exposed to modern food environments.

Given the topicality of the carbohydrate–obesity discourse and the paucity of studies from the Nordic countries, we aimed to study carbohydrate quantity and quality in relation to obesity in Finnish adults by concentrating on nutrient-level exposures (total carbohydrate, sucrose, lactose, and fibre) and dietary GI and GL.

Methods

Subjects

The meta-analysis comprised three cross-sectional studies: the Dietary, Lifestyle, and Genetic determinants of Obesity and the Metabolic syndrome (DILGOM) Study, the clinical part of the Helsinki Birth Cohort Study (HBCS), and the Health 2000 Health Examination Survey (Health 2000 Survey), which all have been previously described in detail

[13–15]. Briefly, the DILGOM Study was implemented within the framework of the National FINRISK 2007 Study, which is the eighth population-based chronic disease risk factor monitoring survey in Finland [2]. The participants were a random sample drawn from the population register stratified according to geographical areas, sex, and 10-year age groups. In 2007, all FINRISK health examination participants were invited to a more detailed health examination (the DILGOM Study) concerning obesity and metabolic syndrome ($n = 5024$, 84% of the invited) [13]. The HBCS investigates the effect of early growth on the risk of non-communicable diseases in later life. The original cohort consists of subjects born at the Helsinki University Central Hospital between 1934 and 1944 [14]. Using random number tables, a sample of 2902 cohort members were derived and invited to a clinical examination during the years 2001–2004 ($n = 2003$, 69%). The Health 2000 Survey focuses on the health and functional capacity of Finns aged 30 years or over, and collects information on major public health problems, working, and functioning ability, and their determinants [15]. The baseline survey comprised a nationally representative sample ($n = 8028$) drawn from 80 health service districts. Of the invited, 85% participated in the health examinations.

The DILGOM Study, the HBCS, and the Health 2000 Survey were conducted according to the guidelines laid down in the Declaration of Helsinki, and all procedures involving human subjects were approved by the Ethics Committee of the Hospital District of Helsinki and Uusimaa. Written informed consent was obtained from all subjects.

Measurements

Demographic and anthropometric variables. All three studies used questionnaires to inquire about the participants' habitual leisure-time physical activity, smoking status, and educational attainment. Trained study nurses measured height, weight, and waist circumference (WC) using standardised protocols. BMI (kg/m^2) was computed as weight (kg) divided by squared height (m).

Diet. Diet was assessed using a comprehensive, regularly updated and validated semi-quantitative food-frequency questionnaire (FFQ) [16], which represents a standard method in nutritional epidemiology. The FFQ inquired into the average consumption of 130 items during the previous year, and included nine possible frequency categories ranging from never or seldom to six or more times per day. The portion size for each food item and mixed dish

was fixed and appeared on the FFQ as household or natural units (e.g. glass or slice). In the DILGOM Study and the HBCS, the participants completed the FFQ on paper at the study site, where a trained study nurse reviewed the FFQ. In the Health 2000 Survey, the FFQ was provided after the health examination on paper and the participants advised to complete it at home and mail it to the study centre. Trained nutritionists entered the data. The average daily energy and nutrient intakes, and dietary GI and GL values were calculated using in-house calculation software, and the National Food Composition Database (Fineli®), which includes GI values for all FFQ-food items [17]. In this study, sucrose represented the disaccharide sucrose, including both the intrinsic, naturally occurring, sucrose and the sucrose added to foods during preparation and processing.

Study population

From those participants who returned the FFQ to the study centre (DILGOM $n = 4996$, HBCS $n = 2003$, Health 2000 $n = 6373$) we excluded individuals older than 79 years ($n = 421$ Health 2000 participants), those who were pregnant ($n = 27$ DILGOM participants and $n = 34$ Health 2000 participants), and those who returned an incomplete FFQ (DILGOM $n = 74$, HBCS $n = 2$, Health 2000 $n = 375$). Furthermore, individuals with extremely low or high daily energy intakes were excluded. These cut-offs corresponded to 0.5% at both ends of the daily energy intake distributions for men and women in DILGOM ($n = 48$) and HBCS ($n = 20$), and in Health 2000 to the daily energy intake values < 600 and > 7000 kcal/d ($n = 18$). Further exclusions included missing information of the response variable (DILGOM $n = 5$, HBCS $n = 2$, Health 2000 $n = 4$). The final meta-analysis comprised 12,342 participants (DILGOM $n = 4842$, HBCS $n = 1979$, Health 2000 $n = 5521$).

Statistical analyses

All statistical analyses were performed using SAS version 9.2 (SAS Institute, Cary, NC, USA). Nutrient intakes, dietary GI and GL were log (natural)-transformed in order to satisfy the normality assumption and further energy-adjusted using the residual method. The exposure variables (total carbohydrate, sucrose, lactose, dietary fibre, dietary GI and GL) were divided into quartiles using study-specific cut-offs. Obesity was defined as BMI ≥ 30 kg/m² for both sexes, and elevated waist circumference as WC ≥ 102 cm for men and WC ≥ 88 cm for women. The modelling included the following a priori defined

confounding variables: sex and age (y, continuous variable), leisure-time physical activity (mild shortness of breath and perspiration < 1 time/wk, 1–3 times/wk, ≥ 4 times/wk, categorical variable), smoking status (non-smoker, former smoker, current smoker, categorical variable) education (total years of schooling, continuous), and total energy intake (kJ/d, continuous variable). To consider possible misreporting of energy intake, we calculated the ratio of reported energy intake to predicted basal metabolic rate and classified participants with this ratio ≤ 1.14 as energy under-reporters, as proposed by Goldberg et al. [18]. Possible effect modifiers included sex, and (when relevant) fat, protein, alcohol, fibre, and sucrose intakes (g/day) and fruit consumption (g/day). These were categorised into low- and high-intake groups using the study-specific median intakes as cut-off points.

Descriptive data were calculated separately for each study by BMI group. Logistic regression was used to calculate study-specific odds ratios (OR) and two-sided 95% confidence intervals (95% CI) for obesity by the exposure variable quartiles. The *P*-value for linear trend was determined with the Wald test using exposure quartile median values as continuous variables. The pooled OR and its 95% CI were estimated in a random-effects model by combining the study-specific log[OR]s, and by weighting them by the inverse of their variance [19]. The *P*-value for test of trend was based on a Wald test of the pooled estimates. Heterogeneity among the original study-specific ORs was tested using the *Q* statistics. The pooled *P*-value for test of interaction was obtained by the squared Wald statistic. *P*-value < 0.05 was considered statistically significant.

The logistic regression analysis comprised three models, the first of which included the exposure variable – sex and age. The second model also included leisure-time physical activity, smoking status, and education. The third, fully adjusted model further included total energy intake. We repeated the main analyses without energy under-reporters and changing the outcome variable to elevated WC. Furthermore, we examined possible interactions between the exposures and sex, between the exposures and other nutrients, and between sucrose and fruit consumption.

Results

The proportion of obese (BMI ≥ 30 kg/m²) participants varied between the studies from 21.3% (DILGOM) to 25.1% (HBCS) (Table I). Compared to the non-obese, the obese were on average older (DILGOM and Health 2000, $P < 0.0001$), more

Table I. Characteristics of the subjects by BMI group in the DILGOM Study, the HBCS, and the Health 2000 Survey.^a

	DILGOM (<i>n</i> = 4842)			HBCS (<i>n</i> = 1979)			Health 2000 Survey (<i>n</i> = 5521)		
	BMI < 30	BMI ≥ 30	<i>P</i> ^b	BMI < 30	BMI ≥ 30	<i>P</i> ^b	BMI < 30	BMI ≥ 30	<i>P</i> ^b
Number of subjects	3810	1032		1483	496		4289	1232	
Females (%)	53	58	0.002	52	59	0.01	53	57	0.037
Age (y)									
Mean	51.6	55.9	< 0.0001	61.5	61.6	0.98	49.6	54.1	< 0.0001
SD	13.6	12.3		2.9	3.0		12.8	12.1	
Exercise ≥ 4 times/wk (%)	29	21	< 0.0001	28	18	< 0.0001	27	27	0.76
Current smokers (%)	18	16	0.104	24	22	0.25	29	22	< 0.0001
Schooling years (y)									
Mean	12.9	11.6	< 0.0001	12.4	11.7	0.0001	11.9	10.4	< 0.0001
SD	4.0	3.7		3.7	3.5		4.0	3.7	
Waist circumference (cm)									
Mean	86.7	108.3	< 0.0001	90.7	110	< 0.0001	87.9	108.3	< 0.0001
SD	10.2	11.4		9.9	10.6		10.3	10.3	
Energy under-reporters ^c (%)	18	31	< 0.0001	26	42	< 0.0001	27	42	< 0.0001

^aBMI: body mass index (kg/m²); DILGOM: Dietary Lifestyle and Genetic Determinants of Obesity and the Metabolic Syndrome Study; HBCS: the Helsinki Birth Cohort Study; Health 2000 Survey: the Health 2000 Health Examination Survey.

^bDifferences between means were tested using the independent sample *t*-test for continuous variables, and chi-square test for binary background variables.

^cEnergy under-reporters were defined based on the Goldberg cut-off value (≤ 1.14) for the ratio of reported energy intake to predicted basal metabolic rate [18].

often females (all studies, $P < 0.05$), exercised less during leisure-time (DILGOM and HBCS, $P < 0.0001$), were less frequently current smokers (Health 2000, $P < 0.0001$), and were less educated (all studies, $P < 0.0001$). As expected, the WC was higher in the obese compared to the non-obese (all studies, $P < 0.0001$). Energy under-reporting was less common among the non-obese compared to the obese (all studies, $P < 0.0001$). Only a few differences in average daily energy and nutrient intakes, dietary GI and GL appeared between the non-obese and the obese (Table II, energy-adjusted intakes; see online Supplementary Table I for crude intakes).

The pooled likelihood of being obese was 35% lower in the highest total carbohydrate intake quartile compared to the lowest quartile (fully adjusted model (model 3); Table III). In model 3, sucrose and dietary GL were inversely associated with obesity risk (P for trends across quartiles < 0.0001). In contrast, the likelihood of being obese was 22% higher in the highest lactose intake quartile compared to the lowest quartile ($P = 0.001$, model 3). In model 3, no association emerged between dietary fibre or GI and obesity. Overall, the exclusion of energy under-reporters ($n = 3264$, 26%) did not change the results. Moreover, changing the outcome variable to elevated WC produced similar results as obtained in model 3 predicting overall obesity (data not shown). However, fibre intake was inversely associated with elevated WC (model 3, OR 0.80; 95% CI 0.71–0.90 for highest vs. lowest quartile; $P < 0.001$). Study-specific

results for model 3 and intake ranges across quartiles are provided in the online Supplementary Table II.

In general, sex did not modify the associations between carbohydrate exposures and obesity (data not shown). A statistically significant interaction emerged only for dietary GI (P for interaction 0.03): an inverse association between GI and obesity was evident in men (model 3; OR 0.76; 95% CI 0.62–0.93 for highest vs. lowest quartile; $P = 0.12$), but not in women (model 3; OR 1.02; 95% CI 0.85–1.22 for highest vs. lowest quartile; $P = 0.63$). Furthermore, the inverse association between GL and obesity appeared stronger in men (model 3; OR 0.55; 95% CI 0.45–0.67 for highest vs. lowest quartile; $P < 0.0001$) than in women (model 3; OR 0.72; 95% CI 0.61–0.85 for highest vs. lowest quartile; $P < 0.0001$) (P for interaction 0.04).

Study of the interactions between the main exposures and nutrients revealed only an interaction for fibre and alcohol (P for interaction 0.04). Dietary fibre was inversely associated with obesity in the high alcohol intake group ($n = 6172$; model 3; OR 0.79; 95% CI 0.64–0.96 for highest vs. lowest quartile; $P = 0.003$), while no association was found in the low alcohol intake group ($n = 6170$; model 3; OR 1.04; 95% CI 0.86–1.25 for highest vs. lowest quartile; $P = 0.67$). With regard to the strong inverse association between sucrose and obesity in the main analysis (model 3; OR 0.53; 95% CI 0.47–0.61), the data also revealed an interaction between sucrose and fruit intake (P for interaction 0.02). The inverse sucrose–obesity relationship emerged

Table II. Nutritional characteristics (mean and SD) of the subjects by BMI group in the DILGOM Study, the HBGS, and the Health 2000 Survey.^a

Characteristic ^b	DILGOM (n = 4842)				HBGS (n = 1979)				Health 2000 Survey (n = 5521)				P ^c
	BMI < 30		BMI ≥ 30		BMI < 30		BMI ≥ 30		BMI < 30		BMI ≥ 30		
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	
Energy (kJ/d)	10,452	3686	10,589	4124	9408	3357	9349	3610	9552	3265	9522	3528	0.78
Total carbohydrate (g/d)	279	35	275	35	244	35	238	36	235	30	232	32	0.004
Sucrose (g/d)	58.1	20.7	54.1	20.8	49.7	19.0	46.3	17.9	47.0	16.8	43.1	17.0	<0.0001
Lactose (g/d)	24.8	14.3	26.3	15.3	21.3	12.9	23.2	14.5	23.0	13.3	25.0	14.0	<0.0001
Dietary fibre (g/d)	29.5	9.0	29.8	9.1	25.9	8.4	25.6	8.5	23.8	7.2	24.3	7.2	0.036
Dietary glycaemic index (GI)	63.1	4.3	63.4	4.2	63.9	5.1	63.3	4.6	64.9	4.2	64.9	4.1	0.98
Dietary glycaemic load (GL)	173	24	172	23	153	21	149	23	155	22	153	23	0.018
Protein (g/d)	102	14	105	15	87.5	12.9	91.6	13.5	90.3	11.8	93.5	12.6	<0.0001
Fat (g/d)	82.7	13.1	83.5	13.1	77.0	12.6	79.4	12.8	88.2	11.9	88.7	12.6	0.18
Alcohol (ethanol; g/d)	7.4	10.5	7.0	11.3	9.7	13.1	7.8	11.7	5.5	9.0	4.8	8.6	0.009
Fruit consumption (g/d) ^d	275	234	259	234	282	265	262	239	213	205	216	211	0.63

^aSD: standard deviation; BMI: body mass index (kg/m²); DILGOM: Dietary Lifestyle and Genetic Determinants of Obesity and the Metabolic Syndrome Study; HBGS: the Helsinki Birth Cohort Study; Health 2000 Survey: the Health 2000 Health Examination Survey.

^bNutrient intakes, GI, and GL were energy-adjusted using the residual method.

^cDifferences between means were tested using the independent sample *t*-test.

^dUnadjusted crude intake (includes berries).

Table III. Pooled odds ratios of obesity for quartiles of carbohydrate intake measures, dietary glycaemic index and glycaemic load in Finnish adults.^a

	Quartile				<i>P</i> ^c	<i>P</i> ^d
	1 (Lowest)	2	3	4 (Highest)		
	OR (95% CI) ^b	OR (95% CI)	OR (95% CI)	OR (95% CI)		
Total carbohydrate (g/d)						
Model 1 ^e	1.00	0.82 (0.73, 0.92)	0.70 (0.62, 0.79)	0.65 (0.57, 0.73)	< 0.0001	0.74
Model 2 ^f	1.00	0.81 (0.72, 0.92)	0.70 (0.62, 0.79)	0.65 (0.57, 0.74)	< 0.0001	0.92
Model 3 ^g	1.00	0.81 (0.72, 0.92)	0.70 (0.62, 0.79)	0.65 (0.57, 0.74)	< 0.0001	0.92
Sucrose (g/d)						
Model 1	1.00	0.76 (0.68, 0.86)	0.61 (0.53, 0.72)	0.55 (0.48, 0.62)	< 0.0001	0.25
Model 2	1.00	0.76 (0.67, 0.85)	0.62 (0.52, 0.73)	0.54 (0.47, 0.61)	< 0.0001	0.13
Model 3	1.00	0.75 (0.67, 0.85)	0.61 (0.52, 0.72)	0.53 (0.47, 0.61)	< 0.0001	0.16
Lactose (g/d)						
Model 1	1.00	1.06 (0.92, 1.21)	1.16 (1.02, 1.31)	1.24 (1.10, 1.40)	< 0.001	0.93
Model 2	1.00	1.08 (0.95, 1.22)	1.12 (0.98, 1.27)	1.22 (1.08, 1.39)	0.001	0.91
Model 3	1.00	1.07 (0.94, 1.22)	1.11 (0.97, 1.26)	1.22 (1.08, 1.39)	0.001	0.89
Dietary fibre (g/d)						
Model 1	1.00	0.86 (0.76, 0.97)	0.82 (0.69, 0.97)	0.82 (0.71, 0.95)	0.005	0.34
Model 2	1.00	0.91 (0.80, 1.03)	0.86 (0.76, 0.98)	0.92 (0.81, 1.05)	0.21	0.62
Model 3	1.00	0.90 (0.79, 1.03)	0.86 (0.75, 0.98)	0.92 (0.80, 1.05)	0.20	0.61
Dietary glycaemic index						
Model 1	1.00	0.98 (0.87, 1.11)	0.94 (0.81, 1.08)	0.99 (0.87, 1.12)	0.71	0.44
Model 2	1.00	0.96 (0.85, 1.09)	0.90 (0.78, 1.03)	0.91 (0.80, 1.04)	0.10	0.56
Model 3	1.00	0.96 (0.85, 1.09)	0.90 (0.78, 1.04)	0.92 (0.81, 1.05)	0.15	0.48
Dietary glycaemic load						
Model 1	1.00	0.83 (0.73, 0.93)	0.75 (0.67, 0.85)	0.66 (0.58, 0.75)	< 0.0001	0.29
Model 2	1.00	0.81 (0.68, 0.96)	0.75 (0.66, 0.85)	0.64 (0.56, 0.73)	< 0.0001	0.34
Model 3	1.00	0.81 (0.69, 0.95)	0.75 (0.66, 0.85)	0.64 (0.56, 0.73)	< 0.0001	0.32

^aThe pooled analysis includes the Dietary Lifestyle and Genetic Determinants of Obesity and the Metabolic Syndrome Study (DILGOM), the Helsinki Birth Cohort Study (HBCS), and the Health 2000 Health Examination Survey (Health 2000 Survey). Obesity is defined as BMI ≥ 30 kg/m². Carbohydrate intake measures, dietary glycaemic index and glycaemic load are energy-adjusted using the residual method.

^b Pooled odds ratio (OR) and 95% confidence interval (CI).

^c Two-sided *P*-value (Wald test) for linear trend across quartiles using exposure quartile median values as continuous variables.

^d Two-sided *P*-value for heterogeneity between studies (Q statistic).

^e Model 1: adjusted for sex and age.

^f Model 2: Model 1 further adjusted for leisure-time physical activity (mild shortness of breath and perspiration < 1 time/wk, 1–3 times/wk, ≥ 4 times/wk), smoking status (non-smoker, ex-smoker, current smoker), and education (total years of schooling).

^g Model 3: Model 2 further adjusted for total energy intake (kJ/d).

stronger in the high fruit intake group ($n = 6172$; model 3; OR 0.45; 95% CI 0.37–0.55 for highest vs. lowest quartile; $P < 0.0001$) compared to the group with low fruit intake ($n = 6170$; model 3; OR 0.62; 95% CI 0.52–0.74 for highest vs. lowest quartile; $P < 0.0001$).

Discussion

This meta-analysis of three population-based cross-sectional studies including 12,342 adult Finns found inverse associations between total carbohydrate, sucrose, and dietary GL and obesity. GI and dietary fibre were not associated with obesity, while lactose intake was positively associated. The strengths of our meta-analysis include its high power and the population-based approach, which improves the

generalisability of the findings. All three studies included a standardised measurement of obesity, and utilised the regularly updated and validated FFQ. Although cross-sectional, our study is among the first to report large-scale findings on the relationship between carbohydrate exposures and obesity in a Nordic population.

The cross-sectional design prohibits conclusions regarding temporal relationships between the different carbohydrate measures and obesity. Residual confounding may remain, despite adjusting for important confounding factors. This is also due to imprecise measurement of the confounders. For example, physical activity – a central determinant of energy balance – is likely to be inadequately measured by questionnaires. Unfortunately, the studies

did not use objective physical activity measures (e.g. accelerometers). It is acknowledged that the assessment of diet is inherently inaccurate regardless of the applied method. BMI is considered a strong determinant of energy under-reporting and obese individuals tend to report fewer foods rich in sugar [20]. A selective misreporting of carbohydrate-containing foods might not be captured by the Goldberg cut-off [18]. Furthermore, reverse causation is likely to occur. Obese individuals may have the intention to lose weight or control weight gain through restricting carbohydrate or sucrose intake, which may result in lower dietary GI and GL. Although interactions between the studied carbohydrate exposures and selected nutrients were minimally evident here, studies on the food group level and comprehensive modelling with dietary confounders (e.g. substitution modelling) are needed in future analysis.

Finns derive carbohydrates mainly from cereals (e.g. starch, fibre) and potatoes (e.g. starch), while naturally occurring mono- and disaccharides are derived from milk (lactose), fruit, fruit juices and berries (e.g. sucrose). Sucrose also originates from sweets, SSBs, bakery products, and sweetened dairy products [21]. Overall, crude intake estimates of this study are higher than in other European countries as measured with FFQs or 24-hour dietary recalls [5–7,22]. This is in part attributable to the tendency of our FFQ to overestimate intakes [16], but also, true food culture differences exist. Higher GI and GL in Finns may result from the combined effects of local eating habits (e.g. high consumption of cereal products and potatoes) and high GI values, as measured and assigned for locally important foods (e.g. rye bread) [23].

Our finding that sucrose associated inversely with obesity is consistent with previous cross-sectional studies conducted in the 1990s [24]. Also, a recent report of the EPIC-Norfolk Study with 1734 adults followed for three years, showed an OR of 0.56 (95% CI 0.40, 0.77) for the sucrose–obesity relationship [12]. In contrast, the same study also showed a positive association between the urinary sucrose biomarker and obesity risk, thus questioning the reliability of measuring sucrose intake with self-reported methods. More research is needed to disentangle the reliability of the FFQ in measuring sucrose intake in Finns. In addition, despite representing added sugar, sucrose is also derived from fruits and berries, which are constituents of a healthy diet. In 2007, fruits, berries, and fruit and berry dishes accounted for one fourth of the sucrose intake in 25–64-year-old Finns and one third of the intake in 65–74-year-olds [21].

The observed stronger inverse sucrose–obesity relationship among the high fruit consumers compared with the low fruit consumers supports the hypothesis that the relation between sucrose and obesity is likely to be affected by food source. A Danish cohort of 42,696 adults followed for five years found an inverse association between carbohydrate intake from vegetables and fruits (women only) and WC change [25]. These results confirm our finding regarding high fibre intake and lower likelihood of elevated WC, and other results indicating that low fibre intake predicts higher adiposity [5]. Furthermore, certain population groups may benefit more from a high-fibre diet, as suggested by the observed interaction between fibre and alcohol intake.

Dietary GL is suggested to act as a surrogate for total carbohydrate intake [6]. This phenomenon was evident in our study, since both total carbohydrate and dietary GL associated inversely with obesity, and were highly correlated ($r = 0.81–0.88$ across studies, $P < 0.0001$). A high-carbohydrate, high-GL diet may reflect a generally healthy diet [26]. Subjects adhering to dietary recommendations may be more health-conscious overall, which again may associate with lower BMI. This phenomenon may be more pronounced in men compared to women. Similarly, the finding that high dietary GI compared to low dietary GI was associated with a lower likelihood of obesity in men suggests that their high-GI diets reflect aspects of a healthy diet. One contributor to inter-individual variation in dietary GI in the Finnish context includes rye bread. Finnish men are found to consume more rye (e.g. as rye bread) than women [21]. Moreover, the effect of a high GI value of rye bread may be balanced out in women consuming more low-GI foods such as fruits and may thus explain the observed GI–sex interaction.

Overall, the potential underlying reasons for discrepant results on dietary GI/GL and obesity in epidemiological studies [5–7,27] includes the disparity of carbohydrate sources and intake levels across populations, and different obesity outcome measures applied. For example, dietary GI was positively associated with WC change and change in visceral adiposity, but not BMI, in studies comprising 89,432 and 48,631 Europeans, respectively [5,6]. Furthermore, the GI of a diet alone might not predict whether it is healthy or not and should thus be interpreted in the overall dietary context [28]. From the statistical viewpoint, the range of dietary GI values in some populations may be too narrow to detect associations.

The positive association between lactose and obesity was unexpected. RCTs generally support a beneficial

effect of dairy in adiposity management [29]. Cohort studies addressing the association between dairy products and weight change are mixed, but no study has reported a positive association [30]. To our knowledge, no study has assessed the association between lactose intake and obesity. Our cross-sectional lactose finding may simply be due to chance and may represent effects in other dietary and lifestyle factors and their combinations, which are difficult to model comprehensively. More research is needed to untangle the role of lactose as a dairy constituent in relation to obesity.

In conclusion, in the Finnish dietary context, obese subjects are more likely to have low carbohydrate, low GL, and low sucrose intakes compared to non-obese subjects, independent of total energy intake. Since obesity develops during a relatively long time period, it remains to be elucidated how carbohydrate exposures associate with obesity longitudinally with diet and obesity measured repeatedly during the follow-up. Given that sucrose source is of importance, efforts are needed to produce standardised measures of added sucrose. Furthermore, sucrose-intake assessment needs to be improved.

Conflict of interest

None declared.

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Supplemental material

Online supplementary data is available at <http://sjp.sagepub.com/supplemental>.

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