See corresponding editorial on page 895.

Fructose intake is a predictor of LDL particle size in overweight schoolchildren[1–3]

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ABSTRACT

Background: High amounts of dietary fructose may contribute to dyslipidemia in adults, but there are few data in children. Childhood adiposity is associated with smaller LDL particle size, but the dietary predictors of LDL size in overweight children have not been studied.

Objectives: We aimed to determine whether LDL particle size is associated with dietary factors and specifically with fructose intake in normal-weight and overweight children.

Design: In a cross-sectional study of normal-weight and overweight 6–14 y-old Swiss children ($n = 74$), dietary intakes were assessed by using two 24-h-recalls and a 1-d dietary record. Body mass index (BMI) and waist-hip ratio (WHR) were measured, and plasma lipid profile and LDL particle size were determined.

Results: Compared with the normal-weight group, overweight children had significantly higher plasma triacylglycerol concentrations, lower HDL-cholesterol concentrations, and smaller LDL particle size ($P < 0.05$). LDL particle size was inversely correlated to BMI SD scores and WHR ($P = 0.007$). Although there were no significant differences in total fructose intake, the overweight children consumed a significantly ($P < 0.05$) higher percentage of fructose from sweets and sweetened drinks than did the normal-weight children. After control for adiposity, the only dietary factor that was a significant predictor of LDL particle size was total fructose intake ($P = 0.024$).

Conclusions: In school-age children, greater total and central adiposity are associated with smaller LDL particle size and lower HDL cholesterol. Overweight children consume more fructose from sweets and sweetened drinks than do normal-weight children, and higher fructose intake predicts smaller LDL particle size. *Am J Clin Nutr* 2007;86:1174–8.

KEY WORDS Fructose, children, diet, LDL particle size, overweight

INTRODUCTION

In the United States, the commercial use of fructose as a sweetener has increased sharply since the 1970s. High-fructose corn syrup (HFCS), a sweetener that is widely used in soft drinks, bakery products, jams, and other daily food items, consists of 45% free, unbound glucose and 55% free, unbound fructose. In comparison, sucrose, a disaccharide, consists of one molecule glucose bound to one molecule of fructose, and it is cleaved during digestion. It has been suggested that high intakes of free fructose (not derived from sucrose) may contribute to hypertension, dyslipidemia, and obesity (1–3). High intakes of fructose can be converted to fat via de novo lipogenesis (3, 4). In animal studies, feeding large amounts of free fructose results in hyperlipidemia, hypertension, and fatty liver (1, 5, 6). Human studies also showed that short-term feeding of high amounts of fructose (up to 25% of total daily energy) leads to dyslipidemia and insulin resistance in liver and adipose tissue (7, 8).

HFCS is not a commonly used sweetener in Western Europe. In Switzerland, the origin of most free dietary fructose is fruit and vegetables, and overall fructose intakes are lower than those in the United States. Little is known about the metabolic effects of low-to-moderate levels of free fructose intake. Moreover, there are few data on the potential adverse effects of fructose consumption in children.

Smaller LDL particle size is associated with the metabolic syndrome and may be an early-onset risk factor for atherosclerosis and type 2 diabetes (9–11). Although obesity and central adiposity in adults and older children are associated with smaller LDL particle size (12–15), there are few data in younger, prepubertal children. Moreover, the dietary determinants of LDL particle size in children have not been studied. Therefore, the aim of the present study was to investigate the relations between dietary intakes—specifically, fructose consumption—and obesity, distribution of body fat, plasma lipids, and LDL particle size in a cohort of Swiss schoolchildren.

SUBJECTS AND METHODS

Subjects

The subjects recruited for this study were 6–14-y-old children ($n = 74$) living in the German-speaking part of Switzerland. Data

[1] From the Human Nutrition Laboratory, Institute of Food Science and Nutrition, ETH Zurich, Zurich, Switzerland (IA and MBZ); the Clinic for Endocrinology and Diabetes, University Hospital Zurich, Zurich, Switzerland (RL, GAS, and KB); the Children's Hospital of Eastern Switzerland, St Gallen, Switzerland (Dl'A); and the Child Development Center, University Children's Hospital, Zurich, Switzerland (LM).

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on subclinical inflammation in this cohort of children were previously reported (16). The children were a convenience sample of volunteers recruited through letters to primary schools and pediatric clinics. The aim was to compare a group of normal-weight children with a group of overweight children. Children recruited from the pediatric clinics were otherwise healthy, overweight children presenting to the clinic for the first time for weight-loss counseling.

Written informed consent was obtained from the parents, and oral informed assent was obtained from the children. Ethical approval for the study was obtained from the Ethics Committee of the Swiss Federal Institute of Technology in Zürich.

Methods

For the dietary assessment, each child was interviewed twice for a 24-h record, and each child filled in one 1-d weighed food record. Each child was visited at the family home 3 times within 3 wk by the same trained interviewer. The 24-h recalls were done at the first and second visits; at the second visit, the interviewer also gave instructions and guidelines for the use of the 1-d weighed food record. At the third visit, the 1-d food record was carefully reviewed with the parent and the child, and an appointment was scheduled at the hospital clinic, at the parents' convenience, usually within 1–2 wk of the dietary assessment. Volumes and portion sizes for the 24-h recalls were estimated by using measuring cups and spoons, photographs of food portions, and graduated food samples of cheese and bread. The combination of 24-h recalls and a food record provides a good overview of a child's habitual diet; this approach has been validated in children as young as 8 y old (17).

The physical activity of the children was assessed with the use of a questionnaire, which asked about the amount of time spent in organized sports, watching television, and playing computer games. On the day of the blood sampling, the children presented to the hospital clinic in the morning after a 12-h overnight fast. A blood sample (12 mL) was taken by venipuncture. Height was measured to the nearest 0.5 cm with the use of a metal measuring tape (Personcheck #44 444; Medizintechnik KaWe, Kirchner & Wilhelm, Asperg, Germany), and weight was measured to the nearest 100 g with the use of a digital balance (BF 18; Beurer, Ulm, Germany). Waist and hip circumferences were measured by using a nonstretchable measuring tape. The measurements of waist and hip circumferences and skinfold thicknesses were done by 2 experienced observers (IA and IH). After a 15-min rest, supine resting blood pressure was measured by auscultation.

Laboratory analysis

Blood samples were centrifuged for 15 min at 20 °C and at 2500 RPM (Omnifuge 2.0RS; Heraeus Sepatech GmbH, Osterode, Germany). Total, HDL, and LDL cholesterol and triacylglycerols were measured in fresh serum on a Hitachi 917 analyzer (Combination Triacylglycerol GPO-PAP and HDL+ 2nd-generation kits; Roche, Basel, Switzerland). Serum was stored at −20 °C for later analysis. Nondenaturing polyacrylamide gradient gel electrophoresis (GGE) of plasma was performed at 10–14 °C in 2–16% polyacrylamide gradient gels. Gels were subjected to electrophoresis for 24 h at 125 V in tris borate buffer (pH 8.3) as described elsewhere (18, 19). Gels were fixed and stained for lipids in a solution containing Oil Red O (Sigma Chemical, St Louis, MO) in 60% ethanol at 55 °C. Gels were placed on a light source and photographed by using Luminescent Image Analyzer (LAS-3000; Fujifilm, Tokyo, Japan) detection with transmitted white light. Using public domain software from the National Institutes of Health (IMAGE; version 1.62; National Institutes of Health, Bethesa, MD), we measured the migration distance for each absorbance peak and calculated the molecular diameter corresponding to each peak from a calibration curve generated from the migration distance of size standards of known diameter. These standards include carboxylated latex beads (Duke Scientific, Palo Alto, CA), thyroglobulin, and apoferritin (HMW Std; Pharmacia, Piscataway, NJ), which have a molecular diameter of 380, 170, and 122 Å, respectively, and lipoprotein calibrators of previously determined particle size.

Statistical analysis

The data for the 3 d of dietary assessment were carefully checked and entered into EBISPRO for WINDOWS software (version 4.0; J Erhardt, University of Hohenheim, Stuttgart, Germany) by the lead interviewer (IA). This system translates the amount of food eaten into individual nutrients. The program is based on the German Food and Nutrition Database (BLS 2.3; Federal Health Department, Berlin, Germany); for foods specific to Switzerland, it incorporates values of >700 foods from the Swiss Food Composition Database (20). Energy and nutrient data were averaged across the 3 d to obtain mean daily energy and nutrient intakes for each child.

The body mass index (BMI; in kg/m^2) of the children was calculated. BMI SD scores (BMI-SDS) were calculated (BMI-SDS = individual BMI value – reference mean BMI value divided by SD to scale the data for comparison across ages and sex) and used as age- and sex-independent values in the analysis. Age- and sex-specific criteria from the Centers for Disease Control and Prevention (CDC; 21) were used to classify children as normal-weight, above the 85th percentile, or above the 95th percentile. Although the CDC uses the terms "at risk for overweight" for children between the 85th and the 95th percentiles and "overweight" for children above the 95th percentile, in the present study, we used the terms "overweight" for the former and "obese" for the latter. This wording is consistent with that in a previous study in which these criteria were validated in Swiss children at the same age (22).

Statistical analyses were performed by using SPSS for WINDOWS statistical software (version 13.0; SPSS Inc, Chicago, IL). Nonnormally distributed variables were log transformed for comparisons. One-way analysis of variance (ANOVA) with a post hoc Bonferroni test was used to compare means. Pearson correlations were used to investigate the relation between measures of adiposity and LDL particle size. To analyze the associations of dietary factors and metabolic parameters with LDL particle size, multivariate regressions were done. All equations were checked for confounding factors, such as adiposity, age, pubertal stage, and sex, and those factors were added as covariates when necessary.

RESULTS

The anthropometric data and the blood lipid variables of the children, by body weight classification, are shown in **Table 1**. Compared with the normal-weight children, overweight children had significantly higher plasma triacylglycerol concentrations, lower HDL cholesterol concentrations, and smaller LDL particle

TABLE 1
Anthropometric measurements and lipid variables in 6–14-y-old Swiss children by weight classification[1]

	Normal-weight children ($n = 31$)	Overweight children ($n = 43$)
Age (y)	10.1 ± 2.0[1]	10.1 ± 1.9
M/F	19/12	21/22
Height (m)	1.40 ± 0.12	1.48 ± 0.12[2]
Weight (kg)	31.9 ± 9.1	51.5 ± 14.2[2]
BMI (kg/m^2)	15.9 ± 1.9	23.4 ± 3.2[2]
Body fat (%)	19.6 ± 4.9	36.1 ± 6.5[2]
Waist-hip ratio	0.79 ± 0.03	0.86 ± 0.06[2]
Total cholesterol (mmol/L)	4.12 ± 0.76	4.20 ± 0.85
HDL cholesterol (mmol/L)	1.60 (1.10–2.20)[3]	1.30 (0.90–2.30)[2]
LDL cholesterol (mmol/L)	2.20 (1.30–3.70)	2.60 (1.20–4.30)
Triacylglycerol (mmol/L)	0.50 (0.30–1.00)	0.80 (0.30–1.98)[2]
LDL particle size	290.14 ± 5.71	285.68 ± 7.96[2]

[1] $\bar{x}$ ± SD (all such values).
[2] Significantly different from normal-weight children, $P < 0.05$ (independent-samples t test).
[3] Median; range in parentheses (all such values).

size ($P < 0.05$). Although LDL-cholesterol concentrations were ≈20% higher in the overweight children than in the normal-weight children, that difference was not statistically significant. In the continuous analysis, there were strong associations between LDL particle size and BMI-SDS, an index of overall adiposity ($r = -0.313$, $P < 0.007$), and WHR, a measure of central adiposity ($r = -0.444$; $P < 0.001$).

Dietary intakes of macronutrients by weight classification are shown in **Table 2**. There was no significant difference between groups in total energy or fat intake, but protein intake was significantly ($P < 0.05$) greater in overweight than in normal-weight children. There were no significant differences in total carbohydrate, glucose, or sucrose intake, but overweight children had a significantly ($P < 0.05$) lower intake of fiber than did normal-weight children. There were no significant differences in total fructose intake, and median intakes were low (1.5–2.0 g/d), but the highest intakes were ≈10 times the lowest intakes. However, the sources of fructose were different in the 2 groups: overweight children consumed significantly less fructose, as a percentage of total fructose, from fruit and vegetables and significantly more fructose from sweets and sweetened drinks ($P < 0.05$ for both).

In the multivariate regressions, the addition of BMI-SDS, age, or sex (or age and sex) did not provide predictions; therefore, the results shown in **Tables 3** and **4** are those obtained after control for WHR only (except for the regression on HDL in Table 4, which was also controlled for sex). Table 3 shows the effects of multivariate regressions of dietary factors on LDL particle size. The only nutrient intakes that were a significant predictor of LDL particle size were total fructose ($P = 0.024$) and g fructose/1000 kcal ($P = 0.042$). There were nonsignificant trends toward an association with fructose from sweets and drinks ($P = 0.074$) and with fructose from fruit and vegetables ($P = 0.081$). All of these associations were negative, which indicated that a higher fructose intake was associated with smaller LDL particles, independent of adiposity. Time spent in watching television and playing computer games was significantly correlated to WHR (Pearson correlation coefficient: 0.407; $P < 0.001$). However, when that

TABLE 2
Daily macronutrient intakes of 6–14-y-old Swiss children by weight classification

	Normal-weight children ($n = 31$)	Overweight children ($n = 43$)
Energy (kcal)	1871.8 ± 469.5[1]	1849.0 ± 336.6
Fat (g)	76.82 ± 21.34	74.5 ± 18.18
Fat (% of energy)	36.23 ± 4.28	35.51 ± 4.75
Protein (g)	58.53 ± 14.83	66.00 ± 12.20[2]
Protein (% of energy)	12.74 ± 2.19	14.65 ± 2.37[2]
Carbohydrates (g)	235.88 ± 65.89	228.16 ± 48.13
Carbohydrates (% of energy)	50.90 ± 4.71	49.95 ± 4.61
Fiber (g)	18.11 ± 4.61	15.97 ± 4.47[2]
Fructose (g)	1.99 (0.12–12.3)[3]	1.62 (0.15–11.38)
Glucose (g)	1.71 (0.15–10.78)	1.44 (0.23–6.94)
Sucrose (g)	19.59 (3.65–129.76)	17.94 (0.23–73.12)
Fructose from fruit and vegetables (% of total fructose)	58.1 ± 31.4	41.9 ± 31.4[2]
Fructose from fruit and vegetables (g)	0.9 (0–10.5)	0.5 (0.00–11.20)
Fructose from sweets and drinks (% of total fructose)	23.4 ± 26.0	40.0 ± 31.7[2]
Fructose from sweets and drinks (g)	0.3 (0–2.4)	0.5 (0–5.8)

[1] $\bar{x}$ ± SD (all such values).
[2] Significantly different from the normal-weight group, $P < 0.05$ (independent-samples t test).
[3] Median; range in parentheses (all such values).

variable was introduced as a covariate into the regression of fructose intake on LDL particle size, it was not a significant predictor ($P = 0.456$, $\beta = -0.086$).

As shown in Table 4, fructose intake did not significantly predict any other measured lipid variable. In the regressions

TABLE 3
Multivariate regressions of macronutrient dietary factors on LDL particle size, after control for waist-hip ratio, in 6–14-y-old normal-weight and overweight Swiss children[1]

	LDL size[2]	
	β[3]	P
Fructose intake	−0.245	0.024
g Fructose/1000 kcal	−0.223	0.042
g Fructose from fruit and vegetables	−0.199	0.081
g Fructose from sweets and drinks	−0.228	0.074
Glucose intake	−0.187	0.083
Sucrose intake	−0.025	0.817
Carbohydrate intake	−0.134	0.214
Fiber intake	−0.062	0.570
Energy intake	−0.109	0.314
Fat intake	−0.014	0.896
SFA	0.037	0.734
MUFA	−0.038	0.728
PUFA	−0.047	0.677
Protein intake	−0.179	0.101

[1] $n = 74$. SFA, saturated fatty acids; MUFA, monounsaturated fatty acids; PUFA, polyunsaturated fatty acids.
[2] Controlled for waist-hip ratio.
[3] Standardized coefficient.

TABLE 4
Multivariate regressions of fructose and LDL size on other lipid variables after control for waist-hip ratio, in 6–14-y-old normal-weight and overweight Swiss children[1]

	Fructose intake		LDL size	
	β[3]	p	β	p
LDL size[1]	−0.245	0.024	—	—
HDL cholesterol[4]	−0.048	0.660	0.485	<0.001
LDL cholesterol[1]	−0.034	0.780	−0.113	0.307
Triacylglycerols[1]	0.014	0.901	−0.344	0.003
Total cholesterol[1]	−0.079	0.518	−0.015	0.893

[1] $n = 74$.
[2] Standardized coefficient.
[3] Controlled for waist-hip ratio.
[4] Controlled for waist-hip ratio and sex.

investigating the relations between the other measured lipid variables and LDL particle size, HDL cholesterol correlated positively with LDL size ($P = 0.001$, adjusted for WHR), there was an inverse association between plasma triacylglycerols and LDL size ($P = 0.003$, adjusted for WHR), and there was no association between LDL particle size and total or LDL cholesterol.

DISCUSSION

The median fructose consumption in the study children was 1.9 g/d (range: 0.1–12.2 g/d), which corresponded to ≈0.5% of daily energy intake. This is ≈5% of the mean intakes in US adults, who derive ≈9% of their daily energy from fructose (8). Although the median intake in the present study was low, the highest intake was ≈10 times the lowest intake; children with the highest intakes were consuming >10 g/d. In the present study, overweight children consumed a significantly higher percentage of fructose originating from sweets and sweetened drinks and a significantly lower percentage of fructose originating from fruit and vegetables than did normal-weight children. To our knowledge, these are the first published data on fructose consumption in normal-weight and overweight children in Europe.

High intakes of fructose have been implicated in the development of dyslipidemia and obesity (2, 23). High intakes of dietary fructose induce hyperlipidemia in rodents (1), but studies on the effects of fructose intake on plasma lipids in adults are equivocal. Several studies have shown the effects of a high fructose intake on plasma lipids—in particular, the triacylglycerol concentrations increased (7, 24); however, other studies did not show such effects (8, 25). In our study, compared with normal-weight children, overweight children had significantly lower HDL-cholesterol concentrations and smaller LDL particle size and significantly greater plasma triacylglycerol concentrations. This finding is consistent with 2 previous studies in obese older children and adolescents (26, 27). However, there was no association between fructose consumption and HDL, LDL, or total cholesterol or triacylglycerols. We are aware of no previous studies on fructose intake and dyslipidemia in children.

The dietary determinants of LDL particle size in adults and children are uncertain. Several studies have shown that short-term high-carbohydrate diets lead to a reduction in LDL particle size (28–31), but those studies investigated the total carbohydrate intake, not the intakes of specific types of carbohydrates. The major new finding of the present study is that higher intakes of dietary fructose predict smaller LDL particle size in young children, independent of adiposity. This association was found despite overall fructose intakes that were lower than those found in countries where HFCS is widely used as an added sweetener. Although the total free fructose intake predicted smaller LDL particle size, there was no significant relation between the intake of fructose derived from fruit and vegetables (intrinsic sources) or the amount added to sweets and sweetened drinks and LDL particle size. Thus, we could not distinguish whether a particular source of dietary fructose was responsible for an observed relation with LDL particle size.

Fructose intakes were correlated with LDL particle size but not with adiposity or any other lipid variable in the children in the present study, which suggests that one of the earliest metabolic effects of higher fructose intake may be modulation of LDL particle size. This finding is consistent with a previous study in adults with coronary artery disease (32), in which changes in LDL particle size appeared to precede other dyslipidemic changes.

The present study had some limitations. Because it was cross-sectional, the directionality of the reported associations cannot be established, and the sample size may have been insufficient to detect weak associations. However, studies in young children on the determinants of LDL particle size are valuable because the findings are usually not confounded by chronic disease or smoking and alcohol consumption. Moreover, dietary assessment, which is always challenging, is particularly so in children, because their ability to record or remember their diet and their knowledge of food and food preparation are limited (33, 34). We used a rigorous method that required 3 home visits and combined 24-h recalls and weighed food records—2 complementary assessment tools (17)

The pattern B phenotype (a predominance of small, dense LDL) is a common finding in obese adults, and this phenotype is linked to a greater incidence of cardiovascular disease (11). The present study shows that LDL particle size in overweight children is smaller than that in normal-weight children. However, the mean LDL particle size of 285.68 Å observed in the overweight children still exceeds the 264 Å that is considered the lower limit of large, buoyant LDL particle size for adults. However, the smaller LDL particle size in the overweight children may represent an early, subtle shift toward the dyslipidemic pattern of small, dense LDL particles often found in obese adults.

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The authors' responsibilities were as follows—All authors: contributed to the study design; IA, MBZ, RL, GAS, and DA: performed the fieldwork and the data collection; IA, KB, MBZ, and GAS: supervised the laboratory analysis and completed the data analysis. IA, KB, and LM: conducted the statistical analysis; IA, MZ, and KB: wrote the first draft of the manuscript; and all authors: edited the manuscript. None of the authors had a personal or financial conflict of interest.

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Erratum

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On page 1174, footnote 2 should read as follows: Supported by the Swiss National Science Foundation (research grant 3200BO-105258; to KB), the Swiss Ministry of Health (Bern, Switzerland), the Swiss Diabetes Foundation (Steinhausen, Switzerland), and the ETH Zürich (Zürich, Switzerland).

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Erratum

Owen CG, Whincup PH, Kaye SJ, et al. Does initial breastfeeding lead to lower blood cholesterol in adult life? A quantitative review of the evidence. Am J Clin Nutr 2008;88:305–14.

In the right-hand column on page 306, the penultimate sentence under "Systematic review process" should read as follows: (*See* Table S1 under "Supplemental data" in the current online issue.).

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