Ad Libitum Intake of Low-Fat Diets Rich in Either Starchy Foods or Sucrose: Effects on Blood Lipids, Factor VII Coagulant Activity, and Fibrinogen

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People are advised to reduce their intake of saturated fat and replace it by carbohydrate to avoid coronary heart disease. It is unknown whether sucrose and starchy foods, two major sources of carbohydrates, have similar effects on cardiovascular risk markers if incorporated as a replacement for saturated fat into diets eaten ad libitum. We served 20 healthy, normal-weight women aged 21 to 52 years three strictly controlled diets ad libitum: FAT, high in total fat (46% of total energy [E%]) and saturated fat (21 E%); STARCH, high in total carbohydrates (59 E%) and low in sucrose (2.5 E%); and SUCROSE, high in total carbohydrates (59 E%) and sucrose (23.2 E%). The diets were eaten in randomized order for a period of 2 weeks. Blood lipids, factor VII coagulant activity (FVIIc), and fibrinogen concentrations were measured with subjects in the fasted state (9:45 AM) and the postabsorptive state (6:00 PM). STARCH was associated with lower total cholesterol (mean difference, 0.34 mmol/L; 95% confidence interval [CI], 0.18 to 0.50), low-density lipoprotein (LDL) cholesterol (0.25 mmol/L; 95% CI, 0.13 to 0.37), fasting triglycerides (0.15 mmol/L; 95% CI, 0.07 to 0.23), nonfasting triglycerides (0.44 mmol/L; 95% CI, 0.30 to 0.58), and nonfasting FVIIc (9.8%; 95% CI, 3.8 to 15.8) than SUCROSE. Compared with FAT, STARCH resulted in a desirable decrease of LDL cholesterol and nonfasting FVIIc. STARCH was also associated with a minor weight loss (0.7 kg) that was not found on the other 2 diets. We conclude that starchy foods with a natural content of dietary fiber can be recommended as substitutes for saturated fat in the dietary prevention of coronary heart disease. According to the present short-term findings in healthy females, substitution with sucrose is not advisable.

AUTHORITIES RECOMMEND that the high-fat diet consumed in many industrialized societies should be replaced with diets containing less fat—in particular, less saturated fat—to combat a number of health problems, not the least of which is cardiovascular disease. To maintain an adequate energy intake, the recommended reduction in the intake of saturated fat must be accompanied by an increased consumption of alternative energy sources. Starchy foods and sucrose could both be proposed as substitutes for saturated fat, but starchy foods may be preferable because, in contrast to sucrose, they most often contain other nutrients in addition to carbohydrate. Also, previous trials indicated that high-sucrose diets have an adverse impact on the blood lipid profile as compared with diets rich in starchy foods.[1-6] However, these results were obtained in trials comparing diets that were isoenergetic and may not apply to the real-life situation where diets are eaten ad libitum, because the satiating effect of sucrose and starchy foods may differ. If high-sucrose diets eaten ad libitum are associated with a better lipid profile than ad libitum diets rich in starchy foods, we would have to reassess what to recommend as a substitute for saturated fats.

We tried to elucidate this issue in the present study in which low-fat diets enriched with either sucrose or starchy foods, both consumed ad libitum, were compared. An ad libitum diet with a high saturated fat content was included in the comparisons. Besides blood lipids, we measured selected hemostatic risk markers of coronary heart disease, ie, factor VII coagulant (FVIIc) activity and fibrinogen.[7-10]

SUBJECTS AND METHODS

The study is described in detail in an earlier publication on the dietary effects on energy intake and energy balance.[11] It included 20 healthy, normal-weight women, 9 of whom were post-obese (ie, previously more than 10% overweight but normal-weight for at least 2 months prior to the study). Their mean age was 39 years (range, 21 to 52) and their body mass index was 23 kg/m^2 (range, 20.5 to 25.0 kg/m^2). The experimental protocol was approved by the Ethics Committee of Copenhagen and Frederiksberg, and written informed consent to participate was provided by all of the subjects. Three experimental diets—a diet rich in starchy foods (STARCH), a sucrose-rich diet (SUCROSE), and a high-fat diet (FAT)—were planned from common foods. The STARCH diet was composed in accordance with the Nordic Nutrition Recommendations, ie, with total fat contributing less than 30% of energy (E%) and saturated fatty acids less than 10 E% and a dietary fiber content greater than 3 g/MJ.[12] The SUCROSE diet was planned to resemble the STARCH diet as much as possible except for the inclusion of additional sucrose (approximately +20E%, or 120 g sucrose per 10 MJ) in the place of starchy foods (primarily bread, rice, pasta, and cereals). Consequently, the SUCROSE diet had less dietary fiber than the STARCH diet. The FAT diet was planned to have a fat content of 45 to 50 E% and a fatty acid composition comparable to the current Danish diet, ie, with a dominance of saturated fatty acids.[13] The 3 experimental diets were identical with respect to fish and marine n-3 polyunsaturated fatty acid content (a tuna sandwich was served twice and a cod dinner dish once per week). The habitual diet of the participants had a mean fat content of 33E%, a sucrose content of 9E%, and a fiber content of 2.4 g/MJ as assessed from the 7-day weighed food records.[11]

Each diet was consumed for a period of 14 days by each participant, with a washout period of 2 to 6 weeks on their habitual diet in between. The 3 diets were served in 4 of 6 possible orders (FAT-STARCH-SUCROSE (n = 5), SUCROSE-FAT-STARCH (n = 7), STARCH-FAT-SUCROSE (n = 6), and SUCROSE-STARCH-FAT (n = 2)). The participants collected the prepackaged diets at the department twice per week and were instructed to eat ad libitum at each meal (3 main meals

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and 1 snack meal per day). To make this possible, the diets were delivered in excess (15 to 18 MJ/d). Leftovers were returned to the department for recording, and then the actual dietary intake of each participant was calculated.

On day 15 of each experimental period, blood samples were collected at 9:45 AM after an overnight fast. Breakfast and lunch were consumed at 10:00 AM and 2:00 PM, respectively, in ad libitum amounts. The nutrient composition of these meals was similar to that of the diet eaten during the preceding 14-day experimental period. A final blood sample was collected at 6:00 PM.

Blood Sampling and Analyses

Blood was taken from an antecubital venous catheter into evacuated tubes either without additives (for lipid analyses) or citrated (for coagulation analyses) at ambient temperature. Tourniquets were not used during sampling. Blood for coagulation analyses was always taken as the second or third tube. Samples were centrifuged at 3,000× *g* for 15 minutes at 20°C, and the serum and plasma were stored at −80°C in plastic vials until analysis within 1 year after completion of the study. Samples from the same individual were always analyzed within 1 analysis.

Serum total cholesterol, high-density lipoprotein (HDL) cholesterol, and triglycerides (millimolars) were determined by enzymatic methods (Boehringer, Mannheim, Germany) with an intraserial variation of less than 2%. The serum concentration of low-density lipoprotein (LDL) cholesterol (millimolars) was calculated as total cholesterol − (HDL cholesterol + triglycerides/2.2).[14] Plasma FVIIc (%) was determined on a Schnittger-Gross hook-coagulometer (Amelung, Germany) in a 1-stage clotting assay using human brain tissue factor and human FVII-deficient plasma (Biopool, Umeå, Sweden) as previously described.[15] Plasma fibrinogen concentrations were also assessed in a functional assay on the coagulometer.[16] The intraserial variation was less than 5% for FVIIc and fibrinogen.

Dietary Assessments

The energy and nutrient content of the experimental diets were planned from the Danish National Food Agency's food database for the personal computer (Dankost 2.0). The final assessment of the dietary intake of each individual during each experimental period was calculated as the difference between food supplied and food (leftovers) returned by the participants.

Statistics

Dietary effects were examined with ANOVA including observations from all 3 experimental periods and all individuals and with period, diet, and individual as independent variables. Only if a significant diet effect was demonstrated (ANOVA, $P < .05$) was a *t* test performed between each pair of diets. We observed no statistically significant effect relating to the period or the sequence in which the diets were consumed.

RESULTS

Dietary Intake

The actual ad libitum intake was very close to the planned intake of macronutrients and fatty acids (Table 1). However, an unintended difference in the cholesterol content of the STARCH and SUCROSE diets was revealed during dietary data analysis. The difference was found to originate from the inclusion of certain egg-based foods in the SUCROSE diet. We took this unfortunate mistake into consideration when we interpreted our blood lipid results. STARCH was associated with an approximately 10% lower energy intake than FAT and SUCROSE. As a consequence, STARCH was associated with a significant decline in the mean body weight of 0.7 ± .2 kg (mean ± SEM) and in fat mass of 0.4 ± 0.1 kg over the 14-day period, whereas body weight did not change significantly on FAT and SUCROSE.[11] The weight loss on STARCH was close to the value predicted from the accumulated energy deficit (approximately 15 MJ over 14 days).

Table 1. Dietary Intake (mean ± SEM) for 20 Women Consuming FAT, STARCH, and SUCROSE Diets Ad Libitum for a Period of 14 Days

Component	STARCH	SUCROSE	FAT
Energy (MJ)	9.1 ± 0.4[b]	10.3 ± 0.4[a]	10.2 ± 0.4[a]
E%			
Protein	13 ± 0.1	13 ± 0.1	13 ± 0.1
Fat	28 ± 0.2[b]	29 ± 0.1[b]	46 ± 0.5[a]
Carbohydrate	59 ± 0.2[b]	59 ± 0.2[b]	41 ± 0.5[a]
Sucrose	2.5 ± 0.1[a]	23.2 ± 0.1[b]	2.2 ± 0.1[a]
Fiber (g/d)	31 ± 1[b]	20 ± 1[a]	22 ± 1[a]
Fatty acid composition (%)			
Saturated	35 ± 0.3[b]	38 ± 0.2[c]	45 ± 0.2[a]
Monounsaturated	40 ± 0.4[b]	37 ± 0.4[a]	37 ± 0.3[a]
Polyunsaturated	25 ± 0.2[b]	26 ± 0.2[c]	18 ± 0.2[a]
Cholesterol (mg/d)	114 ± 23[b]	209 ± 37[c]	259 ± 10[a]

NOTE. Calculations were based on recordings of supplied and returned foods. Values in the same row with different superscripts are significantly different ($P < .05$).

Blood Lipids and Coagulation Factors

Total and LDL cholesterol were 8% to 9% lower after STARCH than after SUCROSE, whereas HDL cholesterol did not differ significantly between the 2 diets (Table 2). As a result, the HDL/total cholesterol ratio was higher on STARCH (0.33) than on SUCROSE (0.30). STARCH and SUCROSE also differed with respect to triglycerides: STARCH was associated with 15% and 30% lower levels in the fasting and nonfasting state, respectively. Finally, STARCH was associated with 10% lower nonfasting FVIIc than SUCROSE.

The STARCH diet caused lower total, LDL, and HDL cholesterol concentrations and a lower HDL/total cholesterol ratio than the FAT diet (Table 2). Fasting triglycerides were 15% higher after STARCH than after FAT, but in nonfasting samples, FAT was associated with higher triglyceride concentrations (+25%). STARCH was associated with lower FVIIc values. The FVIIc difference was only statistically significant in nonfasting samples.

SUCROSE and FAT diets were similar with respect to total and LDL cholesterol, but SUCROSE caused lower HDL cholesterol and a lower HDL/total cholesterol ratio and higher fasting and nonfasting triglyceride concentrations than FAT. SUCROSE and FAT had similar effects on the hemostatic variables. Plasma fibrinogen was not significantly affected by any of the diets.

Differences Between Normal and Post-Obese Individuals

Serum triglycerides were significantly elevated in normals compared with post-obese individuals on all 3 diets in both fasting samples (average over all 3 diets, normal *v* post-obese, 0.91 *v* 0.71 mmol/L, $P = .002$) and nonfasting samples (1.19 *v* 0.86 mmol/L, $P = .004$). We found no other differences be-

Table 2. Fasting Serum Lipids (mmol/L), Plasma Fibrinogen (μmol/L), and FVIIc (%) in 20 Women After Consumption of FAT, STARCH, and SUCROSE Diets Ad Libitum for a Period of 14 Days (mean ± SEM)

Parameter	STARCH	SUCROSE	FAT	Differences		
				STARCH-SUCROSE	FAT-STARCH	FAT-SUCROSE
Total cholesterol (mmol/L)	4.13 ± 0.13	4.49 ± 0.14*	4.59 ± 0.17	−0.34 ± 0.08*	0.46 ± 0.09	0.12 ± 0.12
				$P = .001$	$P < .001$	$P = .35$
HDL cholesterol (mmol/L)	1.34 ± 0.09	1.38 ± 0.09	1.56 ± 0.09	−0.04 ± 0.04	0.22 ± 0.03	0.18 ± 0.05
				$P = .30$	$P < .001$	$P = .001$
HDL: total cholesterol ratio	0.33 ± 0.02	0.30 ± 0.02*	0.35 ± 0.02	0.02 ± 0.01	0.02 ± 0.01	0.04 ± 0.01
				$P = .015$	$P = .015$	$P < .001$
LDL cholesterol (mmol/L)	2.43 ± 0.15	2.72 ± 0.15*	2.71 ± 0.18	−0.25 ± 0.06	0.28 ± 0.07	0.04 ± 0.08
				$P = .001$	$P = .001$	$P = .59$
Triglycerides (mmol/L)	0.81 ± 0.05	0.96 ± 0.30	0.70 ± 0.04	−0.15 ± 0.04	−0.11 ± 0.03	−0.26 ± 0.05
				$P = .002$	$P = .002$	$P < .001$
Triglycerides, nonfasting (mmol/L)	0.84 ± 0.06†	1.29 ± 0.11†	1.03 ± 0.12*	−0.44 ± 0.07	0.19 ± 0.08	−0.23 ± 0.07
				$P < .001$	$P = .027$	$P = .005$
FVIIc (%)	97 ± 3.7*	103 ± 3.6	101 ± 3.6	−6.7 ± 3.1	3.4 ± 3.4	−2.9 ± 2.9
				—	—	—
FVIIc, nonfasting (%)	99 ± 4.4†	107 ± 4.6†	109 ± 4.9†	−9.8 ± 3.0	9.6 ± 2.7	2.1 ± 4.2
				$P = .004$	$P = .003$	$P = .63$
Fibrinogen (μmol/L)	7.7 ± 0.34†	7.8 ± 0.36	7.5 ± 0.34	−0.02 ± 0.39	−0.22 ± 0.28	−0.25 ± 0.26
				—	—	—
Fibrinogen, nonfasting (μmol/L)	7.5 ± 0.40†	7.5 ± 0.37†	7.2 ± 0.33*	0.02 ± 0.23	−0.29 ± 0.18	−0.22 ± 0.23
				—	—	—

NOTE. Nonfasting (6 PM) triglycerides, FVIIc, and fibrinogen after ad libitum consumption of breakfast (10 AM) and lunch (2 PM) on day 15 (FAT, STARCH, and SUCROSE, respectively) are also presented. *P* values refer to paired *t* test and are only presented if ANOVA including all 3 diets showed statistically significant differences ($P < .05$).

*n = 19.

†n = 18.

tween the normals and post-obese, and no differences in the way the 2 groups responded to the diets.

DISCUSSION

The present study mimics the real-life situation in which diets are eaten ad libitum, ie, until satiety is reached. This principle was rarely used in prior studies, in which the experimental diets were most often served in isoenergetic portions. The advantage of isoenergeticity is that the dietary change made in the experiment can be limited to very few foods or nutrients, giving an opportunity to identify specific effects of individual dietary factors. This possibility may be lost in trials with ad libitum consumption of experimental diets, because any difference in energy intake between diets will be accompanied by multiple differences in other nutritional variables, eg, protein intake. On the other hand, the ad libitum design gives an indication of how changes in the composition of the diet will affect the overall dietary intake, and how this will affect body weight, body composition, and biochemical markers of health and disease. These aspects are particularly relevant for the assessment of public health effects of dietary implementation.

In our study, participants received prepackaged foods in excess amounts and thus had the possibility to decide on the amount of food eaten. However, they had no choice regarding the type of food to consume. The palatability of each of the 3 experimental diets therefore may have influenced the results. We cannot exclude that a different composition of the experimental menus may have produced a different outcome, and one must be aware of this limitation of our study. Another weakness of the present study arises from the fact that the actual dietary intake during the experimental periods was not checked by the use of a biomarker. We relied on the participants and trusted that they would report any deviations from the protocol—our participants were highly motivated. In addition, we believe that the twice-weekly individual and personal contact with the participants secured a high degree of compliance.

The main question asked in the present study is whether diets low in saturated and total fat fed ad libitum affect cardiovascular risk markers differently depending on whether they are rich in sucrose or starchy foods. Our study shows this to be the case. The diet enriched with sucrose (23.2 E%) was associated with the more unfavorable blood lipid profile, and nonfasting FVIIc was elevated on this diet as compared with the starchy diet. These findings suggest that diets high in sucrose are more atherogenic and thrombogenic than diets high in starchy foods.

For blood lipids, our ad libitum observations are in line with the findings of most previous trials in which diets were compared under isoenergetic conditions. In an early report, Keys et al[1] reported a 10% elevation of total cholesterol when carbohydrates from fruit, vegetables, and legumes corresponding to 17% of the total dietary energy were replaced by sucrose and small amounts of lactose for a period of 6 weeks. The effect was found at both a dietary fat content of 16 E% and 31 E%. Similarly, Reiser et al[5] observed 10% higher serum total cholesterol after sucrose versus cooked wheat starch among 19 healthy subjects consuming diets composed of otherwise identical natural foods for a 6-week period. In contrast to the study by Keys et al,[1] serum triglycerides were also increased by sucrose in their study[5] (+33%). This difference between studies may relate to the unusual meal pattern with 90% of all food energy

consumed at dinner in the latter study. With a comparable design, Reiser et al[6] found that LDL cholesterol, HDL cholesterol, and triglycerides were elevated by sucrose as compared with starch also in hyperinsulinemic subjects. Some smaller studies support the finding that serum cholesterol and triglycerides, including nonfasting triglycerides, increase with sucrose, but others report no differences between sucrose and starch, maybe due to a lack of statistical power.[2-4,17] Our findings suggest great similarity between the ad libitum and energy-fixed consumption of diets rich in sucrose with respect to the impact on serum cholesterol.

There are several explanations for the adverse effects of our SUCROSE diet. As highlighted in the excellent review by Frayn and Kingman,[18] the fructose component of sucrose influences lipid metabolism at several levels. First, the metabolism of fructose to triose phosphates is more rapid and therefore may facilitate triacylglycerol synthesis in the liver as compared with glucose—the only hexose contained in starch. Second, fructose seems to promote the hepatic production and secretion of very–low-density lipoprotein (VLDL) particles. Finally, fructose may impair the lipolysis and clearance of VLDL particles from the circulation. The resulting higher plasma concentrations of VLDL explain why the SUCROSE diet was associated with higher fasting triglycerides. The higher energy intake (+1.2 MJ/d) with SUCROSE and the associated minor body weight differences (+0.7 kg over 14 days) may have contributed, but only very slightly. Weight changes must be larger to modify blood lipids importantly.[19] The accentuated triglyceride elevation in the nonfasting state is a consequence of the general VLDL increment. This general increment namely leads to augmented competition for lipoprotein lipase between chylomicrons and VLDL particles, causing delayed postprandial degradation and clearance of chylomicrons. Again, the 10% higher ad libitum energy intake with SUCROSE played some role, because it means that the absolute fat intake in grams also was approximately 10% higher versus the STARCH diet.[11]

The different influence of SUCROSE and STARCH on VLDL metabolism can explain why LDL cholesterol concentrations tend to be higher after diets enriched with sucrose. In our study, the starch diet was rich in a variety of starchy food and consequently had a higher dietary fiber content than the SUCROSE diet. This difference in fiber content most likely contributed to the observed differences in LDL cholesterol.[20] In our view, the fiber content should not be seen as a confounding factor in the context of our study, because starchy diets composed of a variety of natural food items and based on current nutrition recommendations will always be richer in dietary fiber than sucrose-rich diets. The unintended higher cholesterol content of the SUCROSE diet (an extra 95 mg/d from egg-based foods as compared with starch) may have contributed approximately 0.1 mmol/L to the difference in total cholesterol according to the Hopkins meta-analysis,[21] and thus also contributed to the LDL cholesterol differences (estimated contribution, 0.05 to 0.08 mmol/L). The slight differences in the fatty acid composition of the SUCROSE and STARCH diets were too small to have an important impact on blood cholesterol levels (less than 0.05 mmol/L can be explained that way). We are not aware of any studies that have compared the effect of diets rich in sucrose and starch on FVIIc. Like us, Szanto and Yudkin[22] found that fibrinogen was unaffected in a study where ad libitum diets rich in either sucrose or starch were compared. They also assessed platelet adhesiveness and clot lysis time and found no differences between the 2 diet types. Body weight and triglycerides were both elevated by sucrose in their study, but they did not observe any effects on serum total cholesterol.

In previous studies, we compared high-fat diets similar to the FAT diet of the present study with low-fat high-fiber diets similar to the STARCH diet.[23-26] The results of the present study are in close agreement with our prior observations, and confirm that replacing diets similar to the FAT diet with diets resembling the STARCH diet is associated with a sustained 10% reduction in LDL cholesterol in normolipidemic individuals. Plasma HDL cholesterol also declines after low-fat/high-fiber diets, but in our experience, the HDL/total cholesterol ratio remains close to or above the ratio found in populations at very low risk of coronary heart disease, ie, about 0.26 to 0.30.[27,28] A 10% to 20% increase in fasting triglycerides is a phenomenon frequently observed during the first few weeks after switching from a high-fat to low-fat diet, but in the case of diets similar to our STARCH diet, the triglyceride elevation seems to be intermittent and to disappear over time as first highlighted in the classic experiment by Antonis and Bersohn.[23,29-31] In addition, nonfasting triglycerides are decreased by this type of diet according to the present and another recent study.[32] The reduced postprandial triglyceride accumulation after STARCH was expected, because of its low fat content leading to little chylomicron formation. With the disappearance after a few weeks of the intermittent VLDL-related elevation of fasting triglycerides, postprandial triglycerides may decrease further as discussed before. For these reasons, we do not believe that the finding of elevated fasting triglycerides shortly after the introduction of low-fat, high-fiber diets is a matter of general concern. Besides the blood lipid differences, fasting FVIIc was insignificantly decreased and nonfasting FVIIc significantly decreased by STARCH as compared with FAT (and SUCROSE). These results confirm previous findings,[15,24,26] and suggest that low-fat, high-fiber diets similar to STARCH may possess an antithrombotic potential. The magnitude of the postprandial FVIIc decline (10% reduction) may be of considerable clinical relevance as judged from epidemiological findings.[8-10]

The duration of each dietary period of the present study was 2 weeks. Our results therefore cannot be extrapolated to indefinite periods, and longer-term studies are highly warranted. Also, our subjects were in slight negative energy balance while on the STARCH diet, which may have confounded the results slightly. Finally, our results were obtained in healthy females and may not apply to, eg, males or patients with hypertriglyceridemia or diabetes. Nevertheless, we find that the evidence available today suggests that (1) a low-fat diet rich in starchy foods and dietary fiber is associated with more favorable blood lipid and FVIIc levels than a diet rich in either sucrose or saturated fat, and (2) a low-fat diet rich in sucrose may lead to less desirable blood lipid and FVIIc levels compared with high–saturated-fat diets. Accordingly, low-fat diets rich in starchy foods and dietary fiber can be recommended for the prevention of coronary heart disease, whereas the replacement of saturated fat with sucrose in high-fat diets appears inadvisable.

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