Metabolic Effects of Dietary Sucrose in Type II Diabetic Subjects

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OBJECTIVE — To assess in diabetic subjects the effects of dietary sucrose on glycemia and lipemia.

RESEARCH DESIGN AND METHODS — Twelve type II diabetic subjects consumed, in random order, two isocaloric, 55% carbohydrate study diets for 28 days. In one diet, 19% of energy was derived from sucrose. In the other diet, <3% of energy was derived from sucrose, and carbohydrate energy came primarily from starch. Both study diets were composed of common foods. All meals were prepared in a metabolic kitchen where foods were weighed during meal preparation.

RESULTS — No significant differences were noted between the study diets at any time point in mean plasma glucose. At day 28, mean plasma glucose values for the sucrose diet were 9.6 ± 0.5 mM and for the starch diet were 9.4 ± 0.6 mM ($P = 0.63$). Also, no significant differences were observed between the study diets in urine glucose; fasting serum total, HDL, or LDL cholesterol; fasting serum TG; or peak postprandial serum TG.

CONCLUSIONS — A high sucrose diet did not adversely affect glycemia or lipemia in type II diabetic subjects.

For most of this century, the most widely held belief about the dietary treatment of diabetes mellitus was that sugars should be avoided. The origin of this belief is uncertain, but may be related to statements made by Allen (1) in the early part of the century describing sugar as a dangerous food for people with diabetes (2). This belief about the danger of sugar was based on the assumption that dietary sugars are more rapidly digested and absorbed than dietary starches and thereby aggravate hyperglycemia. However, little scientific evidence supports this assumption. When sucrose was fed to diabetic subjects as a single nutrient (3), as part of a meal (4–7), or as part of a snack (8,9), it did not cause a greater rise in blood glucose than isocaloric amounts of starch-containing foods. Although several studies have attempted to assess the more chronic effects of dietary sucrose in diabetic subjects, only a few have established adequate control of study nutrients by providing subjects with meals prepared in a metabolic kitchen. Of these studies, one reported that a diet containing 16% of energy as sucrose increased glycemia relative to a reference diet nearly devoid of sucrose in type II diabetic subjects (10). However, two other studies found that dietary sucrose did not increase glycemia relative to a high starch reference diet in either type I (11) or type II diabetic subjects (11,12). Our study was designed to further explore this issue and assess the effects of sucrose on glycemia and lipemia in diabetic subjects.

RESEARCH DESIGN AND METHODS

— Twelve type II diabetic subjects participated in the study. There were 8 women and 4 men whose mean age was 62 yr (range 40–72 yr) and whose mean duration of diabetes was 7 yr (range 3 mo-33 yr). One subject had retinopathy, 1 had mild nephropathy, and 3 had peripheral neuropathy. Diabetes treatments for the 12 subjects were as follows: diet alone for 5 subjects, an oral hypoglycemic agent for 2 subjects, and insulin for 5 subjects. The 5 type II subjects treated with insulin received NPH and regular insulins before breakfast and supper (2 subjects); NPH insulin before breakfast and supper (1 subject); NPH insulin before breakfast (1 subject); and Lente insulin before breakfast (1 subject). Mean relative weight of the type II subjects was 136% (range 99–170%). All type II subjects, including those

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Type I diabetes, insulin-dependent diabetes mellitus; type II diabetes, non-insulin-dependent diabetes mellitus; HDL, high-density lipoprotein; LDL, low-density lipoprotein; TG, triglyceride.

treated with insulin, demonstrated urinary C-peptide excretion that was >10.0 nmol/24 h.

A crossover design was used in which each subject consumed a high sucrose diet, a high starch diet, and a high fructose diet for 28 days. Diet sequences were assigned at random using a balanced scheme such that each of the 6 possible diet sequences was followed by 2 subjects. Results from the fructose diet have been reported previously (13) and are not included herein. Both the sucrose and the starch diets provided ~55% of total energy from carbohydrate, 15% of total energy from protein, and 30% of total energy from fat. In addition, both diets contained nearly identical amounts of dietary fiber, cholesterol, polyunsaturated fat, monounsaturated fat, and saturated fat (PS ratio ~1.0). In the sucrose diet, 19% of energy was derived from sucrose. In the starch diet, carbohydrate energy was derived primarily from starch with <3% of energy derived from sucrose. Of the starch, 52% came from breads and potatoes, which are foods of high glycemic index (3,14). Both study diets were composed of common foods. Meals were prepared in the General Clinical Research Center Metabolic Kitchen (Minneapolis, MN). All foods were weighed during meal preparation. Breakfast and dinner were eaten in the General Clinical Research Center; lunch and the evening snack were packaged and given to the subjects to eat at home or work. Subjects were required to eat all food provided and nothing other than the provided food. Ethanol was proscribed during study participation. A 48-h sample of each study diet was homogenized and submitted to an independent reference laboratory (Medallion, Golden Valley, MN) for nutrient analyses. The calculated and the analyzed nutrient compositions of the study diets were in close agreement.

On days 1, 14, 21, and 28 of each diet period, fasting blood samples were obtained 30 min before breakfast (at 0730) for measurement of plasma glucose, serum cholesterol, serum HDL cholesterol, and serum TG. Additional samples for measurement of plasma glucose were obtained at 0900, 1000, 1200, 1330, 1430, 1630, 1800, 1900, 2100, 2230, and 2330. Additional samples for measurement of serum TG were obtained at 0900, 1000, and 1200 h. A 24-h urine collection for glucose was also obtained. Plasma glucose, urine glucose, serum cholesterol, serum HDL cholesterol, and serum TG were determined as described previously (13). Serum LDL cholesterol was estimated from the formula LDL cholesterol = total cholesterol(HDL cholesterol + TG/2.2).

All subjects who received an oral hypoglycemic agent or insulin received constant daily doses throughout both diet periods. All subjects continued with their usual activities during the study. If necessary to accommodate an individual subject's needs, metabolic testing was performed 1 day before or 1 day after the target date. The research protocol was approved by the University of Minnesota Committee on the Use of Human Subjects in Research. Written consent was obtained from all subjects.

An interval of at least 2 days passed between study diets to allow subjects a break from the diets. The interval between diets was not considered a wash-out period; rather, the first 2 wk of each diet period were considered a wash-in or equilibrium period. Consequently, no attempt was made to make within-diet comparisons of end-point values.

Statistical analysis

Comparisons between the study diets at days 1, 14, 21, and 28 were two-sided and performed by paired Student's *t* test. For all comparisons in the text and table, nominal *P* values are provided. The Bonferrioni procedure was used to protect against the effects of multiple comparisons (15). The significance level required within a group of related end-point comparisons was $0.05/m$, where m was the number of comparisons of that specific end point. With 12 subjects, this study had 80% power of detecting differences between the study diets of 2.66 × SE for each of the end points. All values are presented as means ± SE.

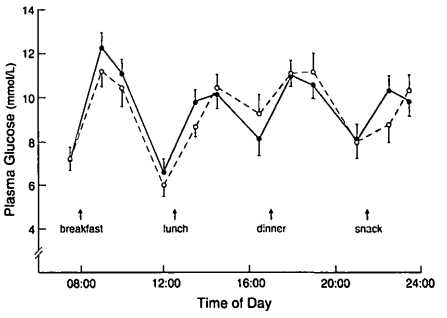

Figure 1—*Mean ± SE plasma glucose values at specific sampling times on the final day (day 28) of the sucrose (●) and starch (○) study diets. None of the time point differences were significant (P < 0.05) after correction for multiple comparisons.*

RESULTS — Both study diets were well tolerated by all 12 subjects. The mean body weights of subjects at the start of the study diets were 86.0 ± 6.5 and 86.9 ± 6.4 kg (P = 0.14) for the sucrose diet and starch diet, respectively. The mean body weights at day 28 of the study diets were 86.1 ± 6.5 and 85.9 ± 6.4 kg (P = 0.84) for the sucrose diet and starch diet, respectively. The effects of the two study diets on mean plasma and urine glucose are summarized in Table 1. No significant differences were noted between the study diets in these measures on any of the test days. Plasma glucose values at specific sampling times on day 28 of the study diets are shown in Fig. 1. None of the between-diet differences were significant. At day 28, the study had 80% power of detecting a difference between the diets in mean plasma glucose of 1.5 mM and in urine glucose of 1.3 g/24 h.

No significant differences were observed between the study diets in baseline or subsequent values for fasting serum cholesterol, HDL cholesterol, LDL

Table 1—*Effects of sucrose and starch study diets on end-point values*

	Day 1	Day 14	Day 21	Day 28
Mean plasma glucose (mM)				
Sucrose diet	10.9 ± 0.7	10.0 ± 0.5	9.8 ± 0.4	9.6 ± 0.5
Starch diet	10.8 ± 0.7	9.9 ± 0.7	9.7 ± 0.6	9.4 ± 0.6
P value	0.88	0.81	0.86	0.63
Urine glucose (g/24 h)				
Sucrose diet	5.2 ± 2.5	1.9 ± 1.0	1.2 ± 0.4	1.0 ± 0.3
Starch diet	1.9 ± 1.0	1.1 ± 0.4	0.6 ± 0.3	1.6 ± 0.7
P value	0.12	0.43	0.21	0.26
Fasting serum cholesterol (mM)				
Sucrose diet	5.32 ± 0.31	5.17 ± 0.26	5.19 ± 0.27	5.05 ± 0.27
Starch diet	5.20 ± 0.24	4.86 ± 0.24	4.93 ± 0.26	4.90 ± 0.27
P value	0.52	0.06	0.04	0.41
Fasting serum HDL cholesterol (mM)				
Sucrose diet	1.07 ± 0.07	1.02 ± 0.06	1.07 ± 0.07	1.05 ± 0.07
Starch diet	1.08 ± 0.07	1.03 ± 0.08	1.01 ± 0.07	1.02 ± 0.07
P value	0.97	0.85	0.03	0.17
Fasting serum LDL cholesterol (mM)				
Sucrose diet	3.32 ± 0.27	3.27 ± 0.25	3.29 ± 0.26	3.12 ± 0.26
Starch diet	3.21 ± 0.25	2.94 ± 0.21	3.07 ± 0.21	3.08 ± 0.21
P value	0.53	0.03	0.07	0.81
Fasting serum TG (mM)				
Sucrose diet	2.03 ± 0.22	1.92 ± 0.20	1.81 ± 0.19	1.91 ± 0.21
Starch diet	2.02 ± 0.29	1.87 ± 0.20	1.84 ± 0.22	1.77 ± 0.23
P value	0.97	0.60	0.76	0.26
Peak postprandial serum TG (mM)				
Sucrose diet	2.51 ± 0.28	2.58 ± 0.23	2.48 ± 0.27	2.64 ± 0.27
Starch diet	2.52 ± 0.31	2.47 ± 0.27	2.39 ± 0.23	2.34 ± 0.25
P value	0.99	0.42	0.59	0.08

Data are means ± SE.

cholesterol, or TG (Table 1). Also, no differences were noted in peak postprandial serum TG. Although the comparisons of serum cholesterol at day 21, serum HDL cholesterol at day 21, and serum LDL cholesterol at day 14 all yielded $P < 0.05$, these values were not significant after correction for multiple comparisons. Moreover, the differences in serum cholesterol, HDL cholesterol, and LDL cholesterol at day 28 were smaller and no longer approached significance. At day 28, the study had 80% power of detecting a difference between the diets in serum cholesterol of 0.72 mM, in serum HDL cholesterol of 0.19 mM, in serum LDL cholesterol of 0.63 mM, in fasting serum TG of 0.59 mM, and in peak postprandial serum TG of 0.69 mM.

CONCLUSIONS— These data demonstrated that a diet deriving 19% of energy from sucrose did not cause an increase in glycemia relative to a diet deriving carbohydrate energy primarily from starch. The data are thus consistent with previous studies comparing sucrose and starch-containing test meals in diabetic subjects (4–7) and with a short-term study (11) and an intermediate-term study (12) comparing sucrose and starch diets in diabetic subjects. However, the data are not consistent with a report by Coulston et al. (10). Coulston fed type II diabetic subjects a diet containing 16% of energy as sucrose for 15 days and found that day-long plasma glucose concentration was higher than when the subjects were fed a sucrose-free reference diet. However, 57% of the carbohydrate energy in the sucrose-free reference diet was derived from sugars other than sucrose. These other sugars were not identified by the authors. If a significant percentage of the other sugars was fructose, then Coulston may actually have compared a high sucrose diet with a high fructose diet. Because dietary fructose can reduce glycemia in diabetic subjects (11,13), the higher day-long plasma glucose concentration during the sucrose diet may have been attributable to fructose present in the reference diet.

The American Diabetes Association in its most recent dietary recommendations for people with diabetes stated that "In some individuals, modest amounts of sucrose and other refined sugars may be acceptable, contingent on metabolic control and body weight"

(16). This study suggests that this recommendation is unnecessarily cautious; that is, a diet providing 19% of total energy as sucrose did not aggravate glycemia in diabetic subjects. However, the subject sample size of the study was small and provided an 80% chance of detecting a difference between the study diets in mean plasma glucose of 1.5 mM. Thus, small differences in glycemia between the study diets might have been overlooked. Moreover, it is important to emphasize that the two study diets were isocaloric. If individuals with diabetes add sucrose or sucrose-containing foods to their diets without reducing other sources of carbohydrate energy, increased glycemia can be expected. Increased sucrose consumption would also increase the risk of dental caries (17).

Considerable controversy exists about the potential effects of dietary sucrose on lipemia in diabetic subjects (18). In this study, the sucrose diet did not result in any significant changes in serum cholesterol. The study had an 80% chance of detecting a difference between the study diets in fasting serum cholesterol of 0.72 mM at day 28. Thus, it is possible that a high sucrose diet does increase serum cholesterol, and this study simply did not have sufficient power to demonstrate such an effect. In this regard, Coulston et al. (10) reported that type II diabetic subjects fed a high sucrose diet and a sucrose-free reference diet for 15 days demonstrated increased fasting plasma cholesterol during the high sucrose diet. In contrast, Abraira et al. (12) reported that type II diabetic subjects fed high sucrose or high complex carbohydrate diets for 1 mo demonstrated no significant differences in fasting serum total or LDL cholesterol. The effects of dietary sucrose on serum TG values in diabetic subjects are also controversial. Abraira et al. (12) found that dietary sucrose had no effect on fasting or postprandial serum TG, whereas Coulston et al. (10) found that dietary sucrose increased fasting and day-long plasma TG values. In this study, dietary sucrose did not significantly increase fasting or peak postprandial serum TG.

Thus, it is not certain whether dietary sucrose has adverse effects on serum lipids in diabetic subjects, and this probably should be considered an open issue. However, the weight of scientific evidence indicates that dietary sucrose does not increase glycemia in diabetic subjects more than other common carbohydrate-containing foods. Therefore, the restriction of sucrose in the diabetic diet because of concern about adverse effects on glycemia cannot be adequately defended on scientific grounds. However, if individuals with diabetes are allowed to consume sucrose and sucrose-containing foods, such foods should be substituted for other foods in the diet in a rational way. To make such substitutions, information is needed about the nutrient content of sucrose-containing foods. This could be provided by addition of a dessert exchange to the American Diabetes Association's Exchange Lists for Meal Planning.

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