

Acute Metabolic Response to High-Carbohydrate, High-Starch Meals Compared With Moderate-Carbohydrate, Low-Starch Meals in Subjects With Type 2 Diabetes

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OBJECTIVE — The monosaccharides resulting from the digestion of ingested carbohydrates are glucose, fructose, and galactose. Of these three monosaccharides, only ingested glucose resulted in a large increase in the plasma glucose concentration. Fructose (*Metabolism* 41:510–517, 1992) and galactose (*Metabolism* 42:1560–1567, 1993) had only a minor effect. Therefore, we were interested in determining whether we could design a mixed meal, using foods of known monosaccharide, disaccharide, and starch composition, the ingestion of which would result in only a small rise in plasma glucose concentration.

RESEARCH DESIGN AND METHODS — The experimental meal was composed of very little readily digestible starch but rather large amounts of fruits and vegetables. It contained 43% carbohydrate, 22% protein, and 34% fat. The results were compared with a second type of meal that contained 55% carbohydrate, 15% protein, and 30% fat, with an emphasis on complex carbohydrates (starch). It also was compared with a third meal that contained 40% carbohydrate, 20% protein, and 40% fat, typical of that consumed by the average American. The test meals were ingested in random order by people with type 2 diabetes who were not treated with oral hypoglycemic agents or insulin. Each subject ingested each type of meal. The same identical meal was ingested at 0800, 1200, and 1700.

RESULTS — The integrated 24-h plasma glucose area response was statistically significantly smaller ($P < 0.05$) after ingestion of the low-starch meals compared with the high-starch, high-carbohydrate meals or the typical American meals. The 24-h integrated serum insulin area response also was statistically significantly less ($P < 0.05$) after ingestion of the low-starch meals compared with the high-starch meals or the typical American meals. The serum triglyceride area response was similar after ingestion of all three test diets.

CONCLUSIONS — A diet in which fruits, nonstarch vegetables, and dairy products are emphasized may be useful for people with type 2 diabetes.

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Abbreviations: AAN, alpha amino nitrogen; ADA, American Diabetes Association; NEFA, nonesterified fatty acid; RIA, radioimmunoassay; SDTU, special diagnostic and treatment unit.

A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

The monosaccharides resulting from the digestion of carbohydrate-containing foods are glucose, fructose, and galactose. Therefore, in previous studies in subjects with untreated type 2 diabetes, we determined the effect on plasma glucose and insulin concentrations of single meals of these monosaccharides, both individually (1–3) and in various combinations (2,4). These studies indicated that it is primarily the glucose derived from digestion of foods that raises the blood glucose concentration. The effects of fructose and galactose were very modest. We also compared the results obtained after the ingestion of the commonly consumed disaccharides, sucrose and lactose, as well as various starches (2,4,5). Starch is a polymer of glucose, whereas fruits and most vegetables contain the monosaccharides glucose and fructose and the disaccharide sucrose. Milk contains the disaccharide lactose, which consists of equimolar amounts of glucose and galactose. Thus, non-starch-containing fruits and vegetables, as well as milk, should raise the blood glucose less than a readily digestible starch-containing food. This indeed was the case. In general, the plasma glucose rise correlated with the potentially available glucose content of the ingested food after digestion.

Based on these data, we were interested in determining the circulating glucose response integrated over 24 h to mixed meals that contained a typical amount of total carbohydrate (43%), but very little readily digestible starch (i.e., potential glucose) and rather large amounts of fruits and nonstarch vegetables (i.e., an increase in potential fructose). The results were compared with those obtained in the same individuals when they ingested meals containing a similar amount of carbohydrate (40%) but composed of foods typically present in American meals. The results also were compared with those obtained when the same subjects ingested high-carbohydrate (55%), high-starch (complex carbohydrate) meals,

Table 1—Composition of test diets

	High-carbohydrate, high-starch diet	Typical American diet (usual carbohydrate, usual starch)	Experimental diet (usual carbohydrate, low-starch)
Carbohydrate (%)	55	40	43
Protein (%)	15	20	22
Fat (%)	30	40	34
Total (kcal/day)	2,052	2,098	2,052
Meal			
Ground beef	42 g raw ~30 g cooked	92 g raw ~65 g cooked	100 g raw ~70 g cooked
Bun	40 g	40 g	—
Baked potato with skin	180 g	100 g	—
Milk	120 g (skim)	180 g (2%)	80 g (2%)
Canned pineapple (juice packed)	100 g	—	—
Lettuce leaves	15 g	15 g	15 g
Sliced tomato	25 g	25 g	or 25 g
Regular ice cream	—	55 g	—
Orange section (peeled)	—	—	100 g
Apple (include peel)	—	—	100 g
Stewed prunes with minimal juice	—	—	100 g
Mayonnaise	5 g	—	—
Saltines (two per package) (~5.7 g)	—	—	1 package
American cheese	—	—	30 g
Corn oil margarine	12 g	—	—
Butter	—	5 g	2.5 g
Decaffeinated or regular coffee	ad libitum	ad libitum	ad libitum
Salt, pepper, sugar substitute	ad libitum	ad libitum	ad libitum
Bedtime snack			
Graham cracker	21 g	21 g	—
Saltines two per package (~5.7 g)	—	—	1 package
Milk	180 g (skim)	180 g (2%)	—
Apple	—	—	120–130 g
American cheese	—	—	30 g
Corn oil margarine	5 g	—	—
Butter	—	—	2.5 g

For each group, identical meals were served for breakfast, lunch, and dinner at 0800, 1200, and 1700, respectively.

which are often recommended for people with diabetes and for the public at large. At the time this study was begun, the American Diabetes Association (ADA) had recommended that the percentage of carbohydrate in the diet be increased to 55–60%. Complex carbohydrates (presumably starch) were to be emphasized (6).

RESEARCH DESIGN AND

METHODS — Three types of test meals were designed. One meal was designed according to the recommendations of the ADA at the time the study was instituted (6). It contained 15% protein, 30% fat, and 55% carbohydrate, with an emphasis on starch-containing foods (2,052 kcal/day) (Table 1).

This is referred to in the text as the high-carbohydrate, high-starch meal. A second meal was designed to approximate the normal American diet. It consisted of 20% protein, 40% fat, and 40% carbohydrate (usual carbohydrate, usual starch) (2,098 kcal/day). It is referred to in the text as the American meal. The third type of test meal was designed to contain an amount of carbohydrate similar to that ingested in the normal American diet (43%). However, sucrose or its equivalent, equimolar amounts of glucose and fructose, were emphasized, rather than starch-containing foods (Table 1). It also contained 22% protein and 34% fat (2,052 kcal/day). This is referred to in the text as the experimental meal. The calorie intake was

distributed as three identical meals and a snack for each type of test meal. Dairy products were included because we had shown that milk protein strongly stimulates insulin secretion (4).

Six male subjects with mild type 2 diabetes who had not been treated with oral hypoglycemic agents or insulin were studied in a special diagnostic and treatment unit (SDTU). All subjects met the National Diabetes Data Group criteria for the diagnosis of type 2 diabetes (9). The patient characteristics are given in Table 2. Written informed consent was obtained from all subjects, and the study was approved by the Department of Veterans Affairs Medical Center and the University of Minnesota Committee on Human Subjects. All subjects had ingested a diet containing at least 200 g carbohydrate/day with adequate food energy for 3 days before testing.

Each subject was admitted to the SDTU on the evening before the study. After an overnight fast of 10–12 h, an indwelling catheter was inserted into an antecubital vein and kept patent with intravenous saline.

Each subject ingested each type of test meal, in random order, on days 1, 4, and 7 of the study. On the study day, identical meals were ingested for breakfast, lunch, and dinner, at 0800, 1200, and 1700. A snack was given at 2100. The caloric distribution was 29.5% each for breakfast, lunch, and dinner, and 11.5% for the snack. Blood was obtained at 0730, 0745, and 0800, 30 min after each meal, and hourly throughout the remainder of the 24-h period of the study. After the 24-h test period, the patients consumed a regular hospital diet ad libitum until the next study day. To determine a fasting baseline response, subjects also consumed water only for a 24-h period.

Plasma glucose concentration was determined by a glucose oxidase method using a Beckman glucose analyzer with an O₂ electrode (Beckman Instruments, Fullerton, CA). Serum immunoreactive insulin was measured using a standard double-antibody radioimmunoassay (RIA) method using kits produced by Incstar (Stillwater, MN); the antibody has 30% cross-reactivity with proinsulin and 0.01% with C-peptide. Glucagon was determined by RIA using 30K antiserum purchased from Health Science Center (Dallas, TX). C-peptide was measured using a double antibody RIA method with kits produced by Incstar; the antibody to C-peptide has only a 4% reactivity with proinsulin. Alpha amino nitrogen (AAN)

Table 2—Patient characteristics

Patient	Age (years)	Fasting glucose		BMI (kg/m ²)	GHb (%)	Time since diagnosis of diabetes	Concomitant diseases
		(mmol/l)	(mg/dl)				
1	61	7.1	128	31.9	8.1	6 weeks	Coronary artery disease, polyarthrititis of unknown etiology
2	56	7.5	135	34.7	8.6	2 months	Peripheral vascular disease with history of claudication, coronary artery disease, cerebrovascular disease
3	70	6.9	125	32.0	6.7	2 years	Hypercholesterolemia, hypertension
4	67	8.3	149	25.9	10.9	1 week	History of Barrett's esophagus (stable), chronic obstructive lung disease (mild), history of gout
5	57	10.3	185	33.4	11.3	3 years	Arteriosclerotic cardiovascular disease (coronary artery bypass graft times four), hypertension
6	62	11.4	206	29.9	11.4	4 years	Hypertension, angina
Mean ± SEM	62 ± 2.2	8.6 ± 0.8	155 ± 13.9	31.3 ± 1.3	9.5 ± 0.8		

was determined by the method of Goodwin (10). Serum nonesterified fatty acids (NEFAs) were determined enzymatically, using a kit purchased from Wako Chemicals (Dallas, TX). Triglycerides and urea nitrogen were determined using an EktaChem Analyzer (Eastman Kodak, Rochester, NY).

The net 24-h area responses were calculated using a computer program based on the trapezoid rule (11). Statistics were determined using either Student's *t* test for paired variates, analysis of variance, or the correlation coefficient, as appropriate, with the Statview 4.1 program (Abacus Concepts, Berkeley, CA) for the Macintosh computer (Apple Computer, Cupertino, CA). A *P* value of <0.05 was the criterion for significance. Data are presented as means ± SEM.

RESULTS — The mean fasting glucose concentration was 8.8 ± 0.34 mmol/l (158 ± 6.1 mg/dl). After ingestion of water only, the fasting glucose concentration decreased continuously, reaching a nadir of 92 ± 9 mg/dl (5.1 ± 0.5 mmol/l) at 1900. After ingestion of all meals, the plasma glucose concentration increased rapidly (Fig. 1A). The glucose response was slightly less for the typical American meals compared with the high-carbohydrate, high-starch meals. The incremental rise was much less after the experimental (low-starch) meal.

The mean 24-h integrated glucose area response using the glucose concentration at 0800 as baseline was 34% greater for the high-carbohydrate, high-starch meals compared with the American meals (Fig. 1B). It

was considerably lower after ingestion of the experimental (low-starch) meals. Indeed, this area was only 3% of that after the high-carbohydrate meals and 4% of that after the typical American meals ($P < 0.05$). When

the glucose concentration after ingestion of water only was used as baseline, the areas were larger, and the difference between groups was less, as expected (Fig. 1C). Nevertheless, the 24-h integrated glucose con-

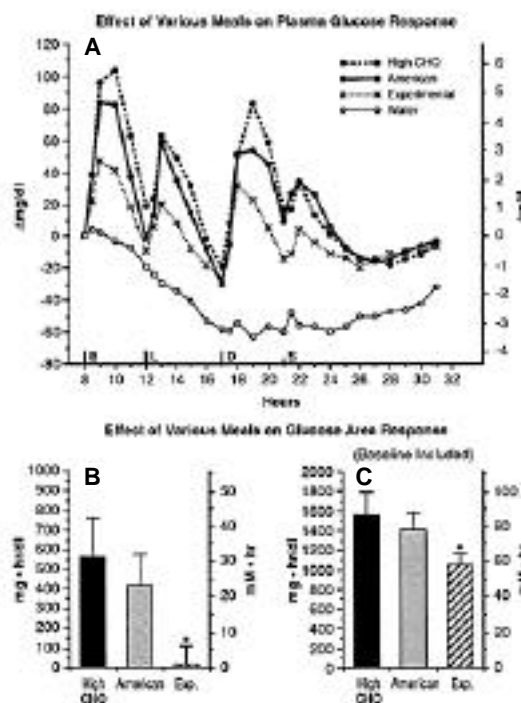


Figure 1—A: Plasma glucose response to various meals in six men with untreated type 2 diabetes. Identical meals were given at 0800, 1200, and 1700. The mean fasting plasma glucose concentration was 8.8 ± 0.34 mmol/l (158 ± 6.1 mg/dl) and was stable throughout the study. B: Glucose area response using the overnight fasting glucose concentration as baseline. C: Glucose area response using the concentration after ingestion of water only as baseline. *Statistically different from the high-carbohydrate (CHO) diet and the American diet, $P < 0.05$.

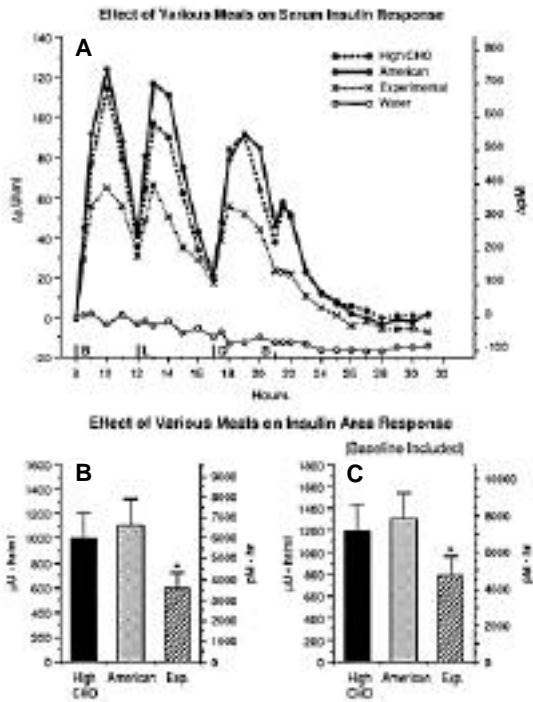


Figure 2—A: Serum insulin response to various meals in six men with untreated type 2 diabetes. Identical meals were given at 0800, 1200, and 1700. The mean fasting serum insulin concentration was 33 ± 14.8 pmol/l (33 ± 2.5 µU/ml) and was stable throughout the study. B: Insulin area response using the overnight fasting insulin concentration as baseline. C: Insulin area response using the concentration after ingestion of water only as baseline. *Statistically different from the high-carbohydrate (CHO) diet and the American diet $P < 0.05$.

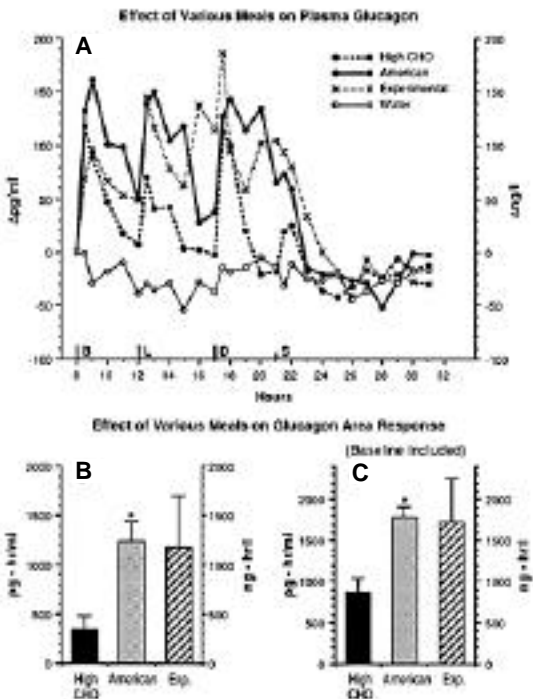


Figure 3—A: Plasma glucagon response to various meals in six men with untreated type 2 diabetes. Identical meals were given at 0800, 1200, and 1700. The mean fasting glucagon concentration was 280 ± 332.2 pg/ml (280 ± 32.2 ng/l) and was stable throughout the study. B: Glucagon area response using the overnight fasting glucagon concentration as baseline. C: Glucagon area response using the concentration after ingestion of water only as baseline. *Statistically different from the high-carbohydrate (CHO) meal $P < 0.05$.

centration was still significantly lower after ingestion of the experimental meals. The glucose area response was positively correlated with the starch ($P = 0.02$) and carbohydrate ($P < 0.05$) content of the diets and was negatively correlated with the fat content ($P < 0.05$). Other comparisons (e.g., protein, phosphorus, arginine, alanine, isoleucine, kilocalories, fiber, monounsaturated fatty acids, polyunsaturated fatty acids, saturated fat, potassium, lactose, and sucrose) were not statistically correlated with the glucose area response ($P > 0.05$ for all). Correlations in addition to starch and carbohydrate were requested during the review process. However, because the study was designed to detect differences in metabolic response based on starch and carbohydrate content of the diet only and the number of observations is relatively small, there may not have been adequate statistical power to detect other correlations.

The mean fasting serum insulin concentration was 33 ± 2.5 µU/ml (198 ± 14.8 pmol/l). After ingestion of water only, the fasting insulin concentration decreased continuously, reaching a nadir of 16 ± 3 µU/ml at 24 h. The serum insulin concentration increased rapidly after ingestion of each of the meals (Fig. 2A). The response was slightly higher after the American meal compared with the high-carbohydrate, high-starch meal. As with the glucose results, ingestion of the experimental (low-starch) meals resulted in a marked attenuation of the increase in postprandial serum insulin concentration.

The mean 24-h integrated insulin area response using the fasting value as a baseline was 10% lower after the high-carbohydrate, high-starch meals compared with the American meals (Fig. 2B). After ingestion of the experimental (low-starch) meals, the insulin area response was only 60% of the area measured after the high-carbohydrate, high-starch meals and 54% of that after the American meals (Fig. 2B and C) ($P < 0.05$).

The mean fasting C-peptide concentration was 1.80 ± 0.53 pmol/ml. After the ingestion of water only, the C-peptide concentration decreased modestly (data not shown). The relative relationship between the meals was similar to the insulin responses. The mean 24-h integrated C-peptide area response relationships also were similar to the insulin area responses (data not shown). However, these differences were not statistically significant.

The mean fasting glucagon concentration was 280 ± 32.2 pg/ml. After ingestion

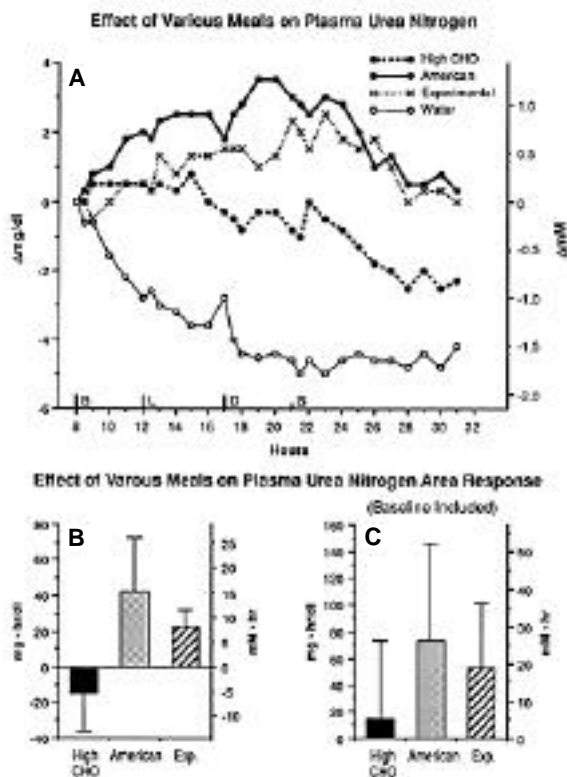


Figure 4—A: Plasma urea nitrogen response to various meals in six men with untreated type 2 diabetes. Identical meals were given at 0800, 1200, and 1700. The mean fasting plasma urea nitrogen concentration was 19 ± 0.6 mg/dl (3.14 ± 0.20 mmol/l) and was stable throughout the study. B: Plasma urea nitrogen area response using the overnight fasting urea nitrogen concentration as baseline. C: Plasma urea nitrogen area response using the concentration after ingestion of water only as baseline. CHO, carbohydrate.

of water only, the glucagon concentration decreased modestly. It increased rapidly after each type of meal (Fig. 3A). However, the incremental increase was much less after ingestion of the high-carbohydrate, high-starch meals.

The mean 24-h integrated glucagon area response also was significantly less after the high-carbohydrate, high-starch meal (Fig. 3B and C) ($P < 0.05$). After the typical American meals and the experimental (low-starch) meals, the area response was $\sim 350\%$ greater than that after the high-carbohydrate, high-starch meals when the overnight fasting value was used as a baseline (Fig. 3B).

The mean fasting AAN concentration was 4.2 ± 0.7 mg/dl. After ingestion of water only, the AAN concentration decreased modestly, but returned to baseline by 0800 the following morning. The AAN concentration increased rapidly after each meal (data not shown). The largest incremental rise occurred after ingestion of the typical American meal. The incremental rise was least after ingestion of the high-

carbohydrate meals. At 0700 the following morning, the AAN concentration after the American and the high-carbohydrate, high-starch meals was similar to that of the water control. It was modestly lower after the experimental meals.

As expected, the mean 24-h integrated AAN area response was least after ingestion of the high-carbohydrate, high-starch meals and greatest after ingestion of the typical American meals. However, the differences were not statistically significant (data not shown).

The mean fasting plasma urea nitrogen concentration was 19 ± 0.6 mg/dl (3.14 ± 0.02 mmol/l). After ingestion of water only, it decreased until 1800, at which time it then stabilized (Fig. 4A). After the ingestion of the high-carbohydrate, high-starch meals, the plasma urea nitrogen concentration remained relatively stable until ~ 1600 , after which it decreased modestly for the remainder of the study. However, it continued to be above that when only water was ingested. After the American and the experimental meals, the urea nitrogen

increased continuously until ~ 2000 – 2200 . It then decreased slightly.

The mean 24-h integrated plasma urea nitrogen area response was least after ingestion of the high-carbohydrate, high-starch meals and greatest after ingestion of the typical American meals (Fig. 4B and C). Because of the large variance, none of the differences were statistically significant.

The mean fasting NEFA concentration was 941 ± 63 μ Eq/l (263 ± 18 g/l). After ingestion of water only, the NEFA concentration increased modestly until 0300 and then returned to the original 0800 concentration (Fig. 5). After ingestion of the test meals, the NEFA concentration decreased, as expected, and returned toward the fasting baseline after the bedtime snack. However, the concentrations had not returned to the fasting baseline by 0800 the following morning. The mean 24-h integrated NEFA area responses to the different types of meal were similar (Fig. 5).

The mean fasting triglyceride concentration was 238 ± 21 mg/dl (2.7 ± 0.3 mmol/l). After ingestion of water only, the triglyceride concentration increased slightly until ~ 1600 and then was relatively stable at the higher concentration (Fig. 6). After ingestion of the test meals, the triglyceride concentration increased to a maximum between midday and early evening. After ingestion of the high-carbohydrate, high-starch meal and the typical American meal, the triglyceride concentration returned to the overnight fasting concentration by 0800 the following morning. With the experimental (low-starch) meals, it did not. However, it did return to the concentration measured after ingestion of water only.

The mean 24-h integrated triglyceride area response was similar for the high-carbohydrate, high-starch meal and the typical American meal. It was slightly, but not significantly, higher after ingestion of the experimental meals. This represented 152% of the area measured after the high-carbohydrate, high-starch meals.

Urinary glucose excretion was greatest after ingestion of the high-carbohydrate, high-starch meals, and least after ingestion of the experimental meals. Urine urea and creatinine excretion were not different with the different meals (Fig. 7).

CONCLUSIONS — As observed previously (12), after an overnight fast and an additional 8 h without food ingestion, there is normalization of the plasma glucose concentration in people with mild to moderately

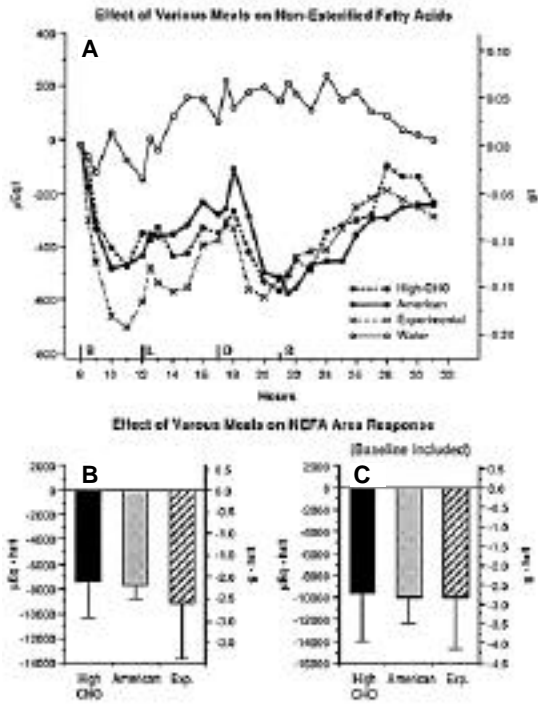


Figure 5—A: NEFA response to various meals in six men with untreated type 2 diabetes. Identical meals were given at 0800, 1200, and 1700. The mean fasting NEFA concentration was $941 \pm 63 \mu$ ($263 \pm 18 \text{ g/l}$) and was stable throughout the study. NEFA area response using the overnight fasting NEFA concentration as baseline. B: NEFA response using the concentration after ingestion of water or as baseline. CHO, carbohydrate.

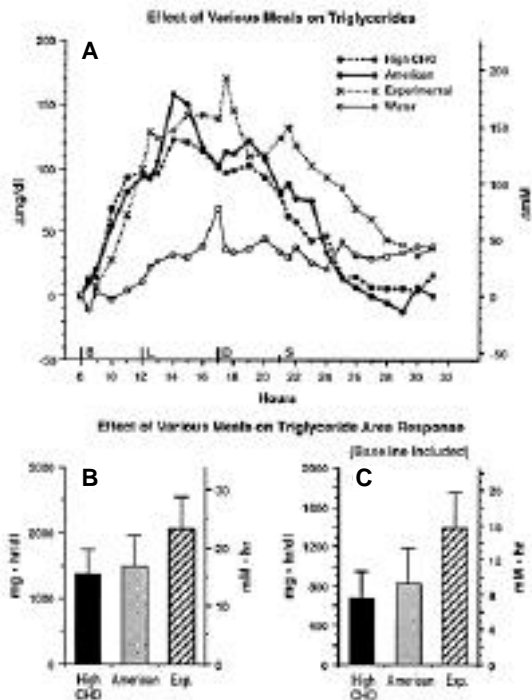


Figure 6—A: Triglyceride response to various meals in six men with untreated type 2 diabetes. Identical meals were given at 0800, 1200, and 1700. The mean fasting triglyceride concentration was $85 \pm 21 \text{ mg/dl}$ ($2.7 \pm 0.3 \text{ mmol/l}$) and was stable throughout the study. B: Triglyceride area response using the overnight fasting triglyceride concentration as baseline. C: Triglyceride response using the concentration after ingestion of water only as baseline. CHO, carbohydrate.

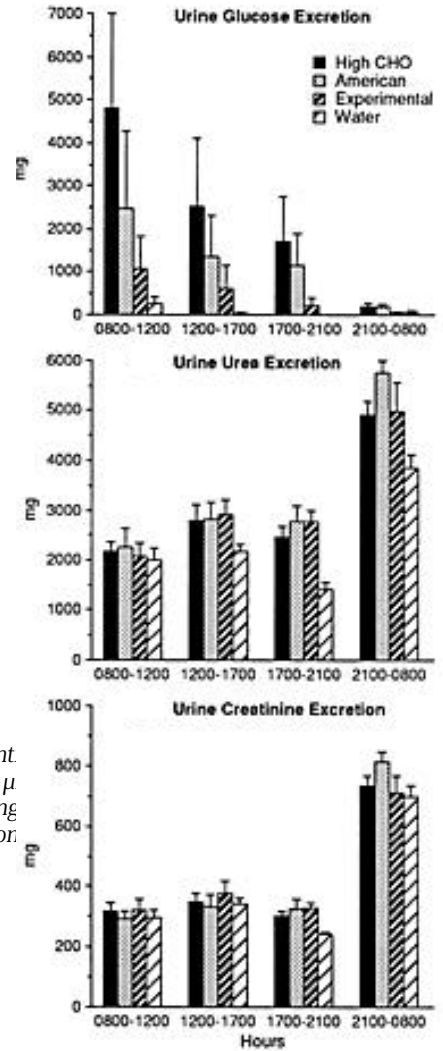


Figure 7—Mean urine glucose, urea, and creatinine excretion after various meals in six men with untreated type 2 diabetes. CHO, carbohydrate.

severe type 2 diabetes. This is associated with a significant decrease in serum insulin concentration as well. The glucagon concentration also decreased slightly (Figs. 1–3).

After ingestion of meals, regardless of the composition, there also is a trend toward lower premeal glucose concentrations throughout the day, as pointed out by Turner et al. (13) many years ago. By the following morning, the glucose concentration tends to rise modestly, just as it did when the subjects did not ingest food (Fig. 1).

Of considerable interest to us was the observation that the low-starch meals resulted in essentially no net increase in glucose concentration when compared with the morning fasting value (Fig. 1B). As indicated above, this may be explained in part by the usual circadian rhythm in glucose

concentrations that is present even when food is not being ingested. The basis for this circadian rhythm remains to be determined but clearly should be considered when interpreting the plasma glucose response to meals. The small increase in plasma glucose also can be explained by the lower potential glucose content of these meals.

It can be calculated that the high-carbohydrate, high-starch meals contained 247 g potential glucose, 32 g fructose, and 12 g galactose. The typical American meals contained 189 g potential glucose, 19 g fructose, and 22 g galactose. The experimental (low-starch) meals contained 134 g potential glucose, 75 g fructose, and 7 g galactose. The potentially available monosaccharides as a percentage of the total carbohydrate in the high-carbohydrate, high-starch meals is glucose 85%, fructose 11%, and galactose 4%; in the American meals, it is glucose 82%, fructose 8%, and galactose 10%; and in the experimental (low-starch) meals it is glucose 62%, fructose 35%, and galactose 3%. These calculations assume that the starch ingested is hydrolyzed equally in the three types of meals. If we consider that the plasma glucose area response to the amount of fructose and galactose present in these meals is only ~12% (1) and 19% (2), respectively, of that of a similar amount of glucose (3) and that protein contributed ~16% to the glucose response (14), we can account for 104% of the glucose area response to the high-carbohydrate, high-starch meal, 92% of the response to the American meals, and 98% of the response to the experimental (low-starch) meals. These calculations were based on the glucose response to 25 g of each monosaccharide and indicate that the predictability for glucose is excellent, i.e., is 98% for the average of the three types of meals.

The three monosaccharides, glucose, fructose, and galactose, as well as protein (14–16) and fat (17), all stimulate an increase in circulating insulin concentration in people with type 2 diabetes. In previous studies, the insulin area response to 25 g fructose was 44% of that to 25 g glucose (1); for galactose, it was 36% (2); and for protein, it was 73% when ingested alone (14). It was ~200% when 25 g protein was ingested with 50 g glucose (15), and for fat ingested with carbohydrate, the insulin area response to 25 g fructose was 13%. Using these calculations and assuming linearity of response with amount, which may not be correct (1,3), we can account for 83% of the insulin area response to the

high-carbohydrate, high-starch meals, 72% of the response to the American meals, and 109% of the response to the experimental (low-starch) meals. The overall predictability was 88% for insulin area response. We accounted for the synergism in the insulin response to glucose plus protein. As indicated above, the least accurate prediction occurred with the American meals, which contained the most protein and the most fat. We have not determined the insulin response to various ratios of protein:carbohydrate:fat, and butter is the only fat studied to date. Knowledge of such interactions should allow more accurate prediction of responses. Presumably when these data are available, the predictions will be more accurate.

Of interest is the fact that glucose tolerance improves between breakfast and lunch, and again between lunch and dinner, as evidenced by the lower premeal glucose concentration and attenuated increase after the meal. The glucose tolerance then deteriorates after dinner. In addition, there is a dissociation between the fasting concentration (water only) and the premeal and overnight glucose concentration. This dissociation increased throughout the 24-h period of the study. Thus, there is likely to be a circadian variation in glucose disposal rate or insulin resistance. Neither this variation, nor possible second and third meal effects (18–21) have been accounted for in our calculations to predict glucose and insulin area responses.

Protein is much more potent in stimulating a rise in glucagon concentration than is glucose in suppressing it, as we have pointed out previously. On a weight basis, the ratio is ~10:1, i.e., protein is 10 times more potent than glucose in affecting the glucagon area response in normal individuals (22). Similar data are not available for subjects with type 2 diabetes. To our knowledge, glucose is the only nutrient known to reduce the glucagon concentration. Ingested fructose modestly increases the glucagon concentration (1) and also may have contributed to the rise in glucagon after meals in the present study. Galactose, ingested alone, does not affect the glucagon concentration. However, when ingested with glucose it resulted in an inhibition of the expected glucose-mediated glucagon decrease (2). Based on the protein and carbohydrate content of the diets, the glucagon area response is approximately that expected. The high-carbohydrate meals had the greatest amount of glucose equivalents

and least amount of protein, and it resulted in the smallest glucagon area response. The American and experimental meals had similar amounts of carbohydrate and protein and similar glucagon area responses.

The relative serum AAN (total amino acids) and urea nitrogen concentration changes were largely those that would be expected from the protein content of the meals. The ordering of the area responses was similar to the ordering of the protein content of the meals, i.e., 15, 20, and 22% of total food energy, for the high-carbohydrate, high-starch, American, and experimental (low-starch) meals, respectively.

The triglyceride area response was greater with the experimental (low-starch) meals than with the high-carbohydrate, high-starch and American meals, even though the experimental meals were only 34% fat. The triglyceride concentration also remained elevated longer (Fig. 6). Dietary fructose has been reported to impair triglyceride clearance (23–25). The experimental diet contained the largest amount of fructose. Thus, the greater increase and more prolonged elevation after these meals is likely to have been due to the fructose content (26).

The decreases in NEFA concentration resulting from the three types of meals were similar, even though the meal composition and insulin response to the meals were different. Adipocyte lipolysis is known to be inhibited by insulin and to be sensitive to very small changes in concentration (27,28). The decrease in NEFAs was persistent. There were large increases in insulin after each meal. However, the insulin never returned to a fasting value between the meals. Thus, the between-meal insulin concentration was sufficient for a maximal effect on lipolysis to be observed. Ingested triglycerides result in a rise in circulating NEFAs (29). Thus, the results are likely due to an integration of these two opposing factors.

There is a reciprocal relationship between free fatty acid concentration and glucose oxidation, i.e., as the free fatty acid concentration decreases, glucose oxidation increases. Because the decrease in free fatty acid concentration was similar in all three diets, an effect of glucose oxidation on glucose disposition cannot be implicated mechanistically.

These data clearly indicate that it is possible to design a diet that is not high in fat and the ingestion of which results in a very small increase in the 24-h integrated plasma glucose response in subjects with untreated type 2 diabetes. This low-starch meal was

acceptable to the subjects for a single day. However, a similar diet is not likely to be acceptable to most people with type 2 diabetes on a long-term basis. Substituting some of the fruit content with poorly and/or slowly digestible starch sources, such as legumes and al dente pasta (30), etc., and perhaps increasing the protein content may result in a diet that is more acceptable. Long-term studies currently underway will be necessary to determine the acceptability of a diet low in readily digestible starch over a longer period of time, as well as the metabolic effects of such a diet.

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