Original Articles

Comparison of Predictive Capabilities of Diabetic Exchange Lists and Glycemic Index of Foods

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To determine whether the diabetic exchange lists or the glycemic index of foods better predicts postprandial responses to carbohydrate-containing foods eaten as part of a mixed meal, three test meals were developed and fed to 12 subjects with non-insulin-dependent diabetes mellitus (NIDDM) and 13 healthy subjects. Each test meal contained exactly the same exchanges (1 milk, 4 starch, 2 fruit, 2 meat, 3 fat, 1 vegetable). In one meal, foods of high glycemic index (GI) were used, in a second meal, foods of intermediate GI were used, and in a third meal foods of low GI were used. The total GIs of the meals were: high, 184; intermediate, 131; and low, 107, thus predicting responses to intermediate and low GI, which were 71 and 58%, respectively, of the responses to high GI. Although some of the observed differences in the glycemic responses to the test meals were statistically significant, primarily in healthy subjects, the differences were usually much less than predicted by the GIs of the meals. In NIDDM subjects, peak postprandial plasma glucose, plasma glucose area, plasma glucose area increment, and mean plasma glucose responses after intermediate and low GI were >90% of the corresponding responses to high GI. In healthy subjects, only the plasma glucose area increment after the low-GI meal was close to the predicted response. High GI produced significantly greater insulin responses than low GI in healthy subjects. We conclude that the diabetic exchange lists more accurately predict postprandial responses to carbohydrate-containing foods eaten as part of a mixed meal than does the GI of foods. Moreover, our data suggest that the responses of healthy subjects to test meals do not accurately predict the responses of NIDDM subjects. *Diabetes Care* 10:387–94, 1987

The Exchange Lists for Meal Planning were developed jointly by the American Dietetic Association, the American Diabetes Association, and the U.S. Public Health Service >35 yr ago to assist individuals with diabetes mellitus in meal planning. Six food groups (milk, fruit, vegetable, starch, meat, and fat) were established with the intent that one food in a group could be interchanged with an equivalent portion of another food in the same group. The assumption was made that foods with similar nutrient content would have similar effects on postprandial plasma glucose concentration. Crapo et al. (1) were among the first to question the validity of this assumption when they demonstrated that feeding healthy subjects glucose, sucrose, and various starches alone or in combination with other foods resulted in significantly different postprandial plasma glucose concentrations. Subsequently, differences in the responses to isocaloric amounts of various carbohydrate-containing foods have been demonstrated in healthy subjects (2), subjects with impaired glucose tolerance (3), and subjects with non-insulin-dependent diabetes mellitus (NIDDM) (4). Jenkins and colleagues (5–7) further refined this approach when they characterized foods by a glycemic index (GI), which compared blood glucose responses to 50-g carbohydrate portions of various test foods to 50 g of a reference food. Great variability in the GIs of common carbohydrate-containing foods, when eaten alone, was demonstrated.

Based on data such as these, the utility of the Exchange Lists for Meal Planning has been questioned (8). However, before the exchange lists are changed significantly or eliminated in favor of a new system based on the GI, we believe it is necessary to determine which system better predicts postprandial responses to typical servings of carbohydrate-containing foods eaten as part of a mixed meal. To accom-

plish this, we fed three test meals to healthy volunteers and individuals with NIDDM and compared their postprandial plasma glucose and serum insulin responses. All three meals contained exactly the same combination of diabetic exchanges. However, foods with high GI were used in one meal, foods with intermediate GI were used in the second meal, and foods of low GI were used in the third meal. An attempt was also made to assess carbohydrate malabsorption after each meal by means of breath H_2 determinations.

METHODS

Subjects. Thirteen healthy volunteers and 12 volunteers with NIDDM participated in the study. The 13 healthy subjects were 7 men and 6 women (mean age 27 yr, range 21–40). All were within 20% of ideal body weight as defined by the 1983 Metropolitan Life Insurance Company tables for individuals of medium frame (9). The 12 subjects with NIDDM were 6 men and 6 women (mean age 59 yr, range 38–70). The mean duration of diabetes was 6 yr with a range of 6 mo to 15 yr. Five subjects had evidence of peripheral neuropathy; however, symptoms suggestive of gastroparesis or diabetic enteropathy were absent in all subjects. Eleven of the 12 subjects were taking an oral hypoglycemic agent. All subjects met the National Diabetes Data Group criteria for the diagnosis of diabetes (10). The mean body weight of the NIDDM subjects was 31% above ideal weight (range 8–76%).

Study design. All subjects were served three different test meals composed of common foods on different mornings. The same combination of exchanges (1 milk, 4 starch, 2 fruit, 1 vegetable, 2 meat, and 3 fat) was used in each meal. Standard foods in all meals were skim milk, lean ground beef, tomatoes, and margarine. Generic carbohydrate-containing foods with nationwide availability were selected according to their GIs. Based on the data of Jenkins et al. (7), carbohydrate-containing foods of high GI were used in one meal, foods of intermediate GI were used in a second meal, and foods of low GI were used in a third meal. The GI of each food was calculated as published GI (7) × carbohydrate content (g)/50 g. The GIs of the foods contained in each meal were summed to determine the total GI of the meal. The specific nutrient content of each meal is summarized in Table 1. All meals were prepared in the metabolic kitchen of the General Clinical Research Center (GCRC). The order in which the meals were served to each subject was randomized so that each of the six possible sequences was used by at least two subjects in each group.

To avoid foods containing poorly or slowly absorbed carbohydrate, subjects were instructed to eat a wheat-, milk-, and bean-free evening meal the day before testing, to fast overnight, and to report to the GCRC at 0730 h on study mornings. Those diabetic subjects being treated with an oral hypoglycemic agent took their usual dose ~30 min before being served the test meals. An indwelling catheter was placed in a forearm vein, and 5 min before the test meals were served, blood samples were removed for measurement of plasma glucose and serum insulin. The study commenced with the first bite of food. Additional blood samples for measurement of plasma glucose and serum insulin were obtained 30, 60, 90, 120, 150, 180, 210, 240, and 270 min after each meal.

In five healthy subjects and 6 diabetic subjects, carbohydrate malabsorption was evaluated with breath H_2 determinations. Alveolar breath samples were obtained before the test meal and then every hour for 9 h after ingestion of the meal. Subjects were not allowed to smoke during these 9 h. Breath H_2 samples were also collected from these 11 subjects on a 4th test day when they received 10 g lactulose (containing 10 g nonabsorbable carbohydrate), which served as a standard.

The protocol was reviewed and approved by the University of Minnesota Committee on the Use of Human Subjects in Research. Informed written consent was obtained from all subjects.

Analytical techniques. Plasma glucose was determined by a glucose oxidase method. Serum insulin was measured by radioimmunoassay. The lower limit of detectability of the insulin assay was 4.0 μU/ml, and the interassay coefficient of variation was 9.3%. Breath samples were analyzed for percent CO_2 with a Beckman LB-2 medical gas analyzer and for H_2 with a Quintron MicroLyzer model 12 gas chromatograph. To correct for atmospheric contamination of breath samples, the observed breath H_2 concentration was adjusted for percent CO_2

$$(5.92/\%CO_2) \times H_2 \text{ (ppm)} = \text{corrected } H_2 \text{ (ppm)}$$

where ppm is parts per million.

Statistical analysis. The data for the healthy and NIDDM volunteers were analyzed separately. Plasma glucose and serum insulin areas were calculated as the total areas under the response curves from −5 to +270 min. Plasma glucose and serum insulin area increments were calculated by subtracting from the total area the area of a rectangle equal to fasting plasma glucose (serum insulin) times 270 min. Mean plasma glucose was calculated from the 10 values available for each subject during each meal test. Breath H_2 areas were calculated as the total areas under the response curves from −5 min to +9 h. Estimated carbohydrate malabsorption was computed for each subject by dividing the breath H_2 area after each test meal by the breath H_2 area after his/her 10-g lactulose standard.

For each of the response variables, mean values were determined, and an estimate of error was made with analysis of variance. When the analysis of variance indicated significant differences among test meals, the mean responses were then compared within subject groups using the honestly significant difference (HSD) between responses (12). Mean responses that differ by ≥1 HSD are significantly different from each other at the 5% level; mean responses that differ by <1 HSD are not significantly different. The size of the subject groups allowed an 80% chance (power = .80) of detecting differences at the 5% level of 1.3 HSD in healthy subjects and 1.35 HSD in NIDDM subjects. All statistical

analyses were done with the GCRC CLINFO computer system.

RESULTS

Measures of glycemic response after the three test meals are presented graphically in Fig. 1 and numerically in Table 2. In NIDDM subjects, fasting plasma glucose was similar before the three test meals. The intermediate-GI meal produced the highest and the low-GI meal the lowest peak postprandial plasma glucose. The difference in peak postprandial plasma glucose between the intermediate- and low-GI meals was significant, but the differences between the high-GI meal and the other two were not. There were no significant differences among the three test meals in plasma glucose area, plasma glucose area increment, and mean plasma glucose. The subject sample size of the NIDDM group (n = 12) allowed an 80% chance of detecting differences among the test meals of 32 mg/dl for peak postprandial plasma glucose, 4890 min · mg · dl^{-1} for plasma glucose area, 5370 min · mg · dl^{-1} for plasma glucose area increment, and 18 mg/dl for mean plasma glucose.

In the healthy subjects, fasting plasma glucose was also similar before the test meals. The high-GI meal produced significantly greater peak postprandial plasma glucose, plasma glucose area, and mean plasma glucose than did the low-GI meal. Values for peak postprandial plasma glucose, plasma glucose area, and mean plasma glucose for the intermediate-GI meal were between those of the high- and low-GI meals and not significantly different from either. The subject sample size of the healthy group (n = 13) allowed an 80% chance of detecting differences among the test meals of 16 mg/dl for peak postprandial plasma glucose, 1870 min · mg · dl^{-1} for plasma glucose area, 2040 min · mg · dl^{-1} for plasma glucose area increment, and 7 mg/dl for mean plasma glucose.

The calculated GIs of the test meal and the observed measures of glycemic response are expressed as percentages of the high-GI meal in Table 3. Although the calculated GIs of the intermediate- and low-GI meals predicted responses that were 71 and 58%, respectively, of those of the high-GI meal, all of the observed responses of the NIDDM subjects were >90% of those of the high-GI meal. Among the healthy subjects, most of the observed responses to the intermediate- and low-GI meals were also >90% of those of the high-GI

TABLE 1
Test meal composition

Food	Exchange group	Amount	Weight (g)	CHO* (g)	Protein* (g)	Fat* (g)	Fiber* (g)	kcal*	Glycemic index†
High-GI meal									
Milk, skim	Milk	1	240	12.2	8.6	0.2		86.4	11.2
Potato, instant mashed	Starch	2	40	31.1	4.4	0.4	2.8	143.2	72.2
Bread, white	Starch	2	50	25.3	4.4	1.6	1.6	135.0	50.6
Raisins	Fruit	2	35	27.1	0.9	0.1	2.7	101.2	50.4
Ground beef, lean	Meat	2	60	0.0	10.7	12.7		160.8	
Margarine	Fat	3	15	0.0	0.1	12.2		108.0	
Tomatoes	Vegetable	1	100	4.3	1.0	0.2	1.8	21.0	
Total				100.0	30.1	27.4	8.9	755.6	184.4
Intermediate-GI meal									
Milk, skim	Milk	1	240	12.2	8.6	0.2		86.4	11.2
Potato, sweet	Starch	2	100	24.9	2.0	0.2	2.4	108.0	34.9
Rice, white	Starch	2	40	32.2	2.7	0.2	1.4	145.2	53.4
Orange, peeled	Fruit	2	200	24.0	1.4	0.0	4.0	94.0	31.7
Ground beef, lean	Meat	2	60	0.0	10.7	12.7		160.8	
Margarine	Fat	3	15	0.0	0.1	12.2		108.0	
Tomatoes	Vegetable	1	100	4.3	1.0	0.2	1.8	21.0	
Total				97.6	26.5	25.7	9.6	723.4	131.2
Low-GI meal									
Milk, skim	Milk	1	240	12.2	8.6	0.2		86.4	11.2
Beans, butter	Starch	2	180	38.2	14.0	1.1	10.3	212.0	39.7
Beans, kidney	Starch	2	185	39.6	14.4	0.9	11.4	218.0	42.8
Cherries, water pack	Fruit	2	200	21.4	1.6	0.4	2.6	86.0	13.7
Ground beef, lean	Meat	2	60	0.0	10.7	12.7		160.8	
Margarine	Fat	3	15	0.0	0.1	12.2		108.0	
Tomatoes	Vegetable	1	100	4.3	1.0	0.2	1.8	21.0	
Total				115.7	50.4	27.7	26.1	892.2	107.4

GI, glycemic index.
*Calculated amounts based on ref. 11.
†GI of each food based on data of Jenkins et al. (7) and calculated as GI × CHO content (g)/50 g.

meal. Only the plasma glucose area increment for the low-GI meal was close to the predicted response.

Serum insulin responses are summarized in Fig. 2 and Table 4. In NIDDM subjects there were no significant differences among the test meals in fasting serum insulin, peak postprandial serum insulin, serum insulin area, and serum insulin area increment. In healthy subjects, fasting serum insulin values were similar, but the high-GI meal produced significantly greater peak postprandial serum insulin, serum insulin area, and serum insulin area increment than did the low-GI meal. Again, the intermediate-GI meal was between the other two meals and not significantly different from either.

Breath H_2 data for the three meals are presented in Table 5. In both subject groups, there were no significant differences among the test meals in breath H_2 area or estimated carbohydrate malabsorption.

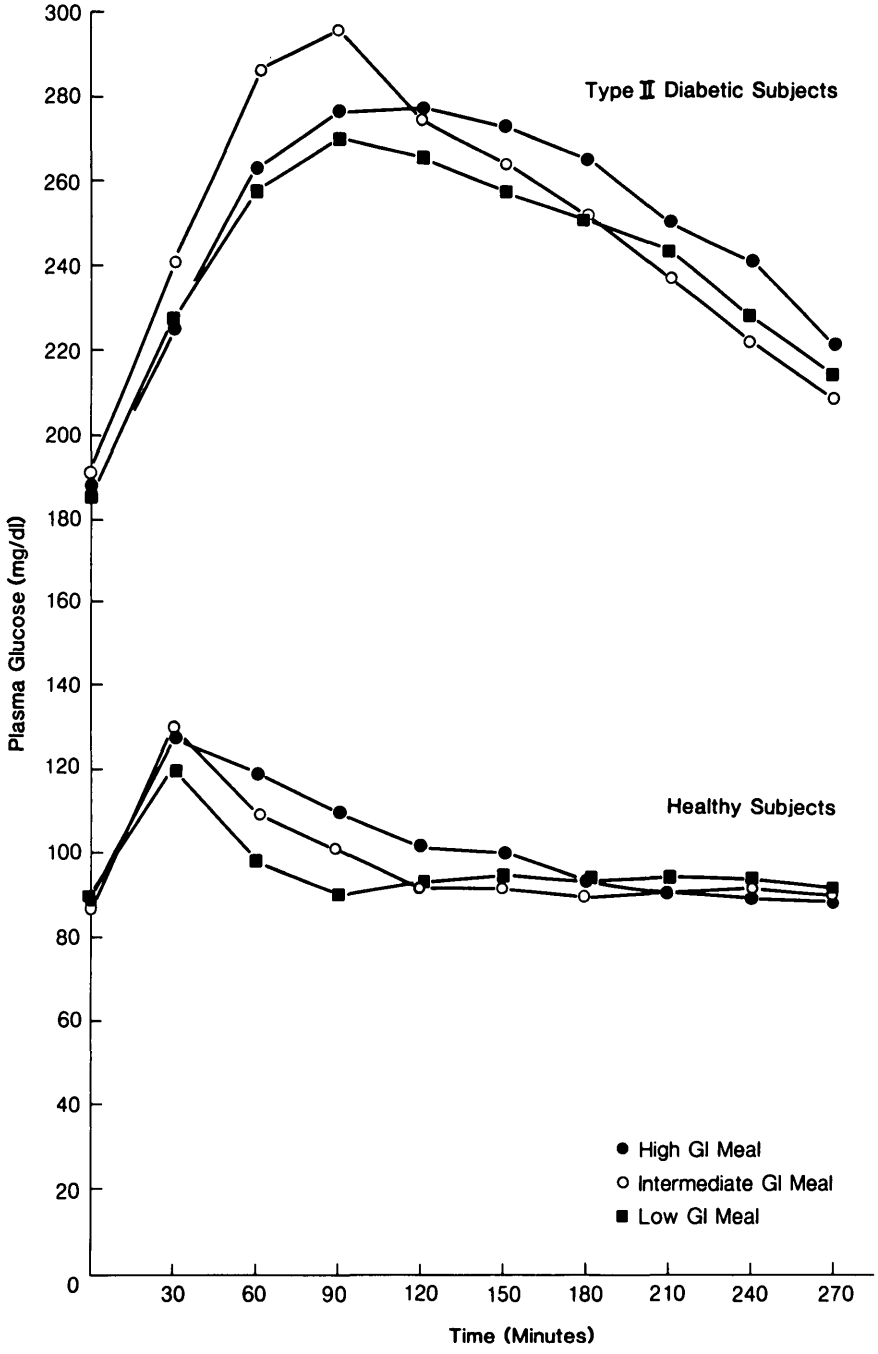

FIG. 1. Mean plasma glucose concentrations before and after ingestion of 3 test meals in type II diabetic and healthy subjects.

TABLE 2
Mean measures of glycemic response to three different test meals in NIDDM and healthy subjects

	High-GI meal	Intermediate-GI meal	Low-GI meal	HSD
NIDDM subjects (n = 12)				
Fasting plasma glucose (mg/dl)	188[a]	191[a]	185[a]	16
Peak postprandial plasma glucose (mg/dl)	296[bc]	305[b]	274[c]	24
Plasma glucose area (min · mg · dl^{-1})	68,780[d]	68,650[d]	66,510[d]	3620
Plasma glucose area increment (min · mg · dl^{-1})	17,100[e]	16,240[e]	15,520[e]	3980
Mean plasma glucose (mg/dl)	248[f]	247[f]	240[f]	13
Healthy subjects (n = 13)				
Fasting plasma glucose (mg/dl)	89[g]	87[g]	89[g]	6.2
Peak postprandial plasma glucose (mg/dl)	136[h]	134[hi]	124[i]	12
Plasma glucose area (min · mg · dl^{-1})	27,950[j]	26,790[jk]	26,320[k]	1440
Plasma glucose area increment (min · mg · dl^{-1})	3370[l]	3000[l]	1880[l]	1570
Mean plasma glucose (mg/dl)	101[m]	97[mn]	96[n]	5.0

GI, glycemic index; HSD, honestly significant difference. Values sharing same superscript letter are statistically indistinguishable.

DISCUSSION

Our data demonstrate that when the Exchange Lists for Meal Planning were used to develop three test meals, each containing the same number and types of exchanges but different foods, remarkably similar postprandial responses were observed in NIDDM subjects. When peak postprandial plasma glucose, plasma glucose area, plasma glucose area increment, and mean plasma glucose responses were expressed as percentages of the corresponding responses to the high-GI meal, the intermediate- and low-GI meals varied from only 91 to 103% (Table 3). Our data are thus consistent with those of Nuttall et al. (13), who also demonstrated similar postprandial responses of NIDDM subjects to mixed-meal breakfasts designed with the exchange lists. In our NIDDM subjects the only significant difference in the measures of glycemic control was the difference in peak postprandial plasma glucose between the intermediate- and low-GI meals. This difference was much less than predicted by the GIs of the meals and was of doubtful clinical importance. The results were thus similar to those of Coulston et al. (14), who found few significant differences in the postprandial responses of subjects with NIDDM to mixed meals containing foods of varying GI.

TABLE 3
Measures of observed glycemic response expressed as percentage of high-GI meal

	High-GI meal	Intermediate-GI meal	Low-GI meal
Calculated GI of meal	100	71	58
NIDDM subjects			
Peak postprandial plasma glucose	100	103	93
Plasma glucose area	100	100	97
Plasma glucose area increment	100	95	91
Mean plasma glucose	100	100	97
Healthy subjects			
Peak postprandial plasma glucose	100	99	91
Plasma glucose area	100	96	94
Plasma glucose area increment	100	89	56
Mean plasma glucose	100	96	95

Values are given as percentages. GI, glycemic index.

In healthy subjects we were able to demonstrate that the high-GI meal produced significantly greater peak postprandial plasma glucose, plasma glucose area, and mean plasma glucose than did the low-GI meal. Although these differences were statistically significant, only the plasma glucose area increment after the low-GI meal was close to what was predicted by the calculated GIs of the meals. We were surprised that the postprandial responses to our test meals were not more accurately predicted by the GI of foods. This may largely have been because the carbohydrate-containing foods in our meals were eaten as part of a mixed meal also containing fat and protein. Because fat inhibits gastric emptying (15,16), digestion and absorption of the nutrients in the test meals may have been delayed sufficiently to reduce the differences in response among the carbohydrate-containing foods in the meals. Similarly, it has been demonstrated that protein reduces the postprandial plasma glucose response to dietary carbohydrates (17). Further support for this possibility is provided by the observation that the addition of fat and protein to test carbohydrates fed to healthy volunteers reduced peak postprandial glucose concentrations compared with the test carbohydrates fed alone (2). Thus, dietary fat and protein, by delaying gastric emptying or by other mechanisms, appear to diminish the differences in response to dietary carbohydrates.

It is also possible that the size of our test meals diminished our ability to detect differences among them. The test meals

varied from 723 to 892 kcal, with 98–116 g carbohydrate, whereas the GI of foods was based primarily on 50-g carbohydrate (200 kcal) test portions and several 25-g carbohydrate (100 kcal) test portions. Thus, our test meals may have provided greater stimulus to insulin secretion and other homeostatic mechanisms than the test portions used in developing the GI of foods. If the stimulus provided by our meals was maximal or nearly so in NIDDM subjects, differences in response to the carbohydrate-containing foods included in them may have been obscured. Nevertheless, the amount of food served in the test meals was not greater than the amount of food consumed by most adults during the main meal each day.

Our results are in partial agreement with those of Wolever et al. (18), who analyzed increments in plasma glucose area and found an excellent correlation between the calculated GIs and the observed responses to carbohydrate meals fed to six healthy subjects. When we compared increments in plasma

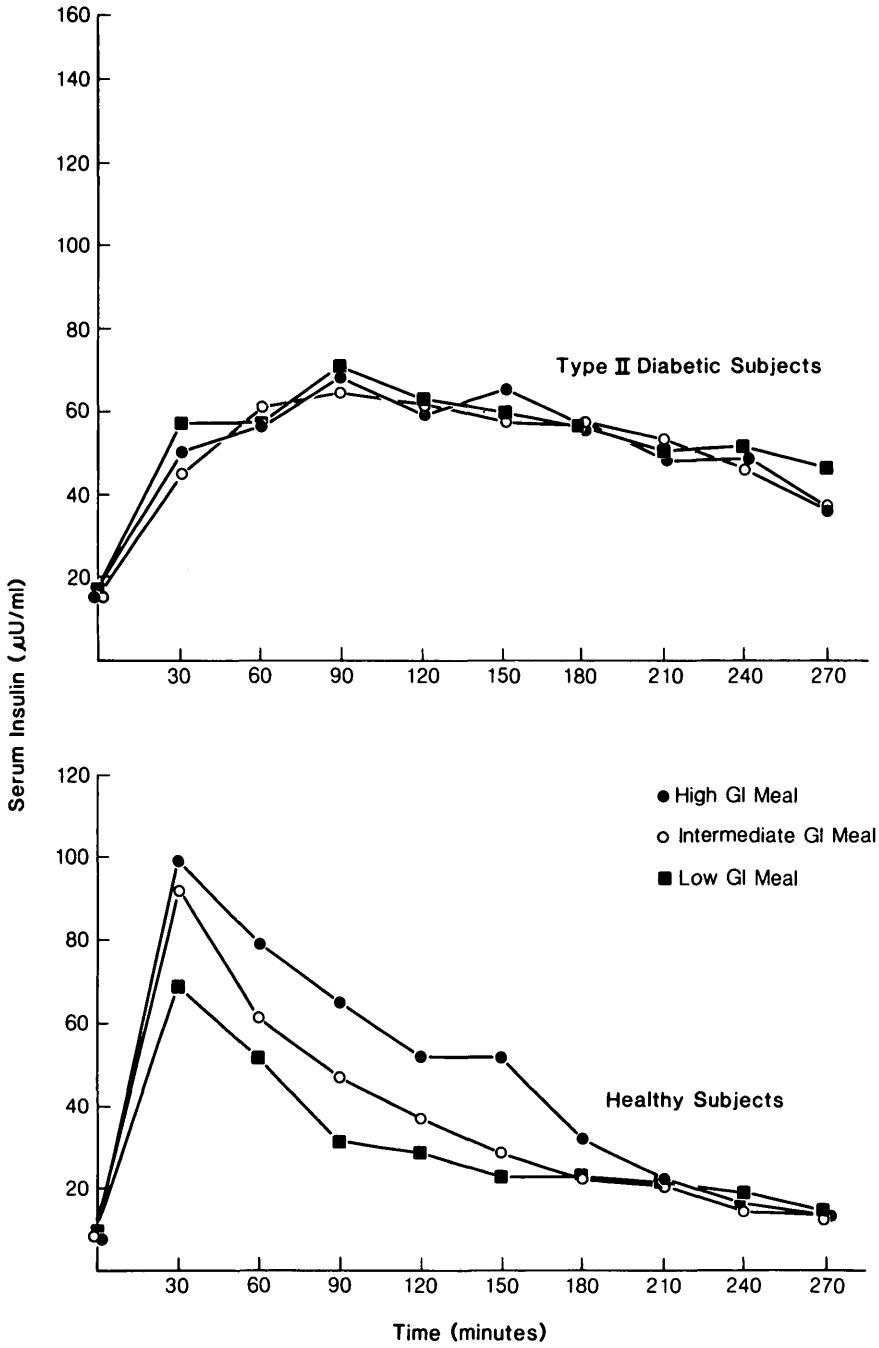

FIG. 2. Mean serum insulin concentrations before and after ingestion of 3 test meals in type II diabetic and healthy subjects.

TABLE 4
Mean serum insulin responses to three different test meals in NIDDM and healthy subjects

	High-GI meal	Intermediate-GI meal	Low-GI meal	HSD
NIDDM subjects (n = 12)				
Fasting serum insulin (μU/ml)	16[a]	17[a]	15[a]	3.6
Peak postprandial serum insulin (μU/ml)	82[b]	79[b]	78[b]	17
Serum insulin area (min · μU · ml^{-1})	14,470[c]	14,230[c]	15,040[c]	1940
Serum insulin area increment (min · μU · ml^{-1})	10,190[d]	9460[d]	10,990[d]	2420
Healthy subjects (n = 13)				
Fasting serum insulin (μU/ml)	9.3[e]	7.6[e]	7.7[e]	3.5
Peak postprandial serum insulin (μU/ml)	110[f]	98[fg]	79[g]	27
Serum insulin area (min · μU · ml^{-1})	12,830[h]	10,110[hi]	8370[i]	3100
Serum insulin area increment (min · μU · ml^{-1})	10,270[j]	8020[jk]	6260[k]	2710

GI, glycemic index; HSD, honestly significant difference. Values sharing same superscript letter are statistically indistinguishable.

glucose area in healthy subjects, we found a good correlation between the responses predicted by the GIs of the meals and the observed responses. However, this did not hold true in NIDDM subjects. Moreover, we believe that it is not optimal to analyze postprandial increments in plasma glucose, because it is the absolute plasma glucose level rather than the increment in plasma glucose that is biologically important. Analyzing postprandial increments in plasma glucose often tends to magnify small differences that are of doubtful importance.

In our NIDDM subjects, peak postprandial serum insulin, serum insulin area, and serum insulin area increments were similar and not significantly different after all three test meals. This suggests that all three test meals provided equal stimuli to insulin secretion in the diabetic subjects. In the healthy subjects, in contrast, there were significant differences in peak postprandial serum insulin, serum insulin area, and serum area increment among the test meals that paralleled those demonstrated for the measures of glycemic control. Thus, insulin secretion in the healthy subjects was not equally stimulated by all three test meals.

When we were designing our study, we were concerned about potential differences in glycemic response that might be caused by partial malabsorption of the carbohydrate foods contained in the test meals. Legumes in particular are known to contain digestive enzyme inhibitors and other antinutrients that may interfere with the digestive process (7,19). Consequently, we attempted to assess carbohydrate malabsorption by measuring breath H_2. H_2 is one of the gases produced in the colon when unabsorbed carbohydrate is fermented by the bacteria present there. Pulmonary H_2 excretion increases linearly with the amount of carbohydrate that reaches the colon (20), and the measurement of breath H_2 is thus a useful means of assessing carbohydrate malabsorption (21). However, in both of our subject groups there was little difference among the test meals in postprandial breath H_2 area or estimated carbohydrate malabsorption. The amount of estimated carbohydrate malabsorbed after each test meal could easily be accounted for by the dietary fiber contained in that meal, suggesting that little available carbohydrate was malabsorbed.

In summary, our data suggest that the Exchange Lists for Meal Planning more accurately predicts the postprandial response to a mixed meal than does the GI of foods. This may be because the addition of fat- and protein-containing foods to carbohydrate-containing foods reduces the differences in their postprandial response, even when the GIs of the carbohydrate-containing foods are quite different. Additional studies are necessary to determine whether clinically important differences in the physiologic responses to carbohydrate-containing foods really exist. Future studies should employ

TABLE 5
Mean breath H_2 responses to three different test meals in NIDDM and healthy subjects

	High-GI meal	Intermediate-GI meal	Low-GI meal	HSD
NIDDM subjects (n = 6)				
Breath H_2 area (ppm/h)	101.8[a]	106.9[a]	132.6[a]	72.6
Estimated CHO malabsorption (g)*	4.6[b]	5.1[b]	5.6[b]	4.3
Healthy subjects (n = 5)				
Breath H_2 area (ppm/h)	104.3[c]	136.5[c]	173.4[c]	140.1
Estimated CHO malabsorption (g)*	4.7[d]	7.6[d]	9.4[d]	9.6

GI, glycemic index; HSD, honestly significant difference. Values sharing same superscript letter are statistically indistinguishable.
*Estimated CHO malabsorption computed by comparing each subject's meal responses with response to 10-g lactulose standard. See text for details.

diabetic subjects because the responses of healthy subjects do not accurately predict those of individuals with diabetes mellitus.

ACKNOWLEDGMENTS: We gratefully acknowledge the contribution of General Clinical Research Center dietary, nursing, and laboratory personnel, the technical assistance of Reynold Francis and Julie Kneip, and the expert assistance of Kate Steinmeyer and Cynthia Huntington in preparation of the manuscript.

This study was supported by a grant from the American Diabetes Association, Minnesota Affiliate, and by General Clinical Research Center Grant RR-400 from the Division of Research Resources, National Institutes of Health.

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