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ORIGINAL ARTICLE

Effects of breakfast meal composition on second meal metabolic responses in adults with type 2 diabetes mellitus

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Objective: We tested the relative importance of a low-glycemic response versus a high glycemic response breakfast meal on postprandial serum glucose, insulin and free fatty acid (FFA) responses after consumption of a standardized mid-day meal in adult individuals with Type 2 diabetes mellitus (DM).
Design: Following an overnight fast of 8–10 h, a randomized crossover intervention using control and test meals was conducted over a 3-week-period. A fasting baseline measurement and postprandial measurements at various time intervals after the breakfast and mid-day meal were taken.
Subjects: Forty-five Type 2 DM subjects completed the requirements and were included in the study results.
Interventions: Two different breakfast meals were administered during the intervention: (A) a high glycemic load breakfast meal consisting of farina (kJ 1833; carbohydrate (CHO) 78 g and psylium soluble fiber 0 g), (B) a low-glycemic load breakfast meal consisting of a fiber-loop cereal (kJ 1515; CHO 62 g and psyllium soluble fiber 6.6 g). A standardized lunch was provided approximately 4 h after breakfast. Blood plasma concentrations and area under the curve (AUC) values for glucose, insulin and FFA were measured in response to the breakfast and mid-day lunch. Statistical analyses were performed using SAS software (8.02). Comparisons between diets were based on adjusted Bonferroni *t*-tests.
Results: In post-breakfast analyses, Breakfast B had significantly lower area under the curve (AUC) values for plasma glucose and insulin compared to Breakfast A ($P<0.05$) (95% confidence level). The AUC values for FFA were higher for Breakfast B than for Breakfast A ($P<0.05$) (95% confidence level). Post-lunch analyses indicated similar glucose responses for the two breakfast types. Insulin AUC values for Breakfasts B were significantly lower than Breakfast A ($P<0.05$) (95% confidence level). The AUC values for FFA were unaffected by breakfast type.
Conclusions: These data indicate that ingesting a low-glycemic load meal containing psyllium soluble fiber at breakfast significantly improves the breakfast postprandial glycemic, insulinemic and FFA responses in adults with Type 2 DM. These data revealed no residual postprandial effect of the psyllium soluble fiber breakfast meal beyond the second meal consumed. Thus, there was no evidence of an improvement postprandially in the glycemic, insulinemic and FFA responses after the consumption of the lunch meal.
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Introduction

Adults with Type 2 diabetes mellitus (DM) are at increased risk for cardiovascular disease (CVD) and other comorbidities. This risk can be lowered through lifestyle modifications such as changes in dietary intake, weight loss and exercise to reduce postprandial glycemia, insulin resistance and lipid abnormalities (Gavin, 1999; Gerich, 2003; Abraham, 2004).

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The major mechanism by which diet decreases risk is by improvement in insulin sensitivity (Mayer-Davis *et al*., 1998). Reducing visceral adiposity, which is associated with a reduction in free fatty acid (FFA) levels, can help ameliorate insulin resistance and hence lower diabetes and CVD risk (Short *et al*., 2003).

Consensus among medical organizations emphasizes dietary modification that is high in complex carbohydrate (CHO) and low in fat to improve glycemic control, lower low-density lipoprotein (LDL)-cholesterol concentrations, and reduce insulin requirements (Lui *et al*., 2000; Hu *et al*., 2001; Roberts and Barnard, 2005).

Studies indicate that plant soluble fibers such as guar, psyllium and pectin were more effective than insoluble fibers in moderating postprandial glucose and insulin concentrations in patients with noninsulin-dependent diabetes. There have been discrepant findings in the efficaciousness of psyllium, which may be due to the timing and/or the method of administration. Fiber has been shown to be efficacious when administered as a component of a mixed solid food meal or as a supplemental drink (Jarjis *et al*., 1984; Wolever *et al*., 1997; Sierra *et al*., 2002).

Psyllium in a supplemental drink form, has been specifically shown to lower postprandial glycemia in individuals with Type 2 DM when consumed just before a meal (Anderson *et al*., 1999; Sierra *et al*., 2002) and to have a residual effect after a second meal (Pastors *et al*., 1991; Anderson *et al*., 1999). Few studies have examined second meal responses to soluble fiber (Pastors *et al*., 1991; Sierra *et al*., 2002), but there is limited data on glycemic load and soluble fiber in influencing Type 2 DM (Salmeron *et al*., 1997; Liu *et al*., 2000).

The aim of this study was first to determine the effects of a breakfast meal with a low-glycemic load containing psyllium soluble fiber as compared to a breakfast meal with a high glycemic load without psyllium soluble fiber on postprandial plasma glycemic, insulinemic and FFA responses in adult subjects diagnosed with Type 2 DM, and second to determine whether or not there are any residual postprandial effects of these breakfast meals on glycemic, insulinemic and FFA responses after a standardized, mid-day lunch has been consumed.

Subjects

A total of 75 adult male and female subjects between the ages of 20 and 75 with Type 2 DM were recruited by newspaper advertising and from patients attending hospital clinics at Sparrow Hospital, Lansing, Michigan. Individuals were eligible for the study if they were over 18 years old; were previously medically diagnosed with Type 2 DM (fasting plasma glucose 126 mg/dl or higher) for a minimum of 6 months; controlled their diabetes with diet only or diet plus oral hypoglycemic agents; had no other chronic disease diagnosis, were regular breakfast eaters (4 out of 7 days), and had no known allergy to psyllium seed husk since individuals were informed that psyllium was included in the test meal. Individuals were excluded from the study if they had a history of myocardial infarction, other chronic medical conditions, or major surgical procedures within the previous 6 months, as were individuals who were unable to participate for a 3-week consecutive time period. Forty-five subjects provided informed consent and completed the study. The supplementary questionnaire also indicated that these subjects did not smoke, drink nor exercise on a regular basis. As such, it was not deemed necessary to stratify our analyses by exercise level nor medication status. Subjects using medication were asked to bring the medication with them at each visit and were allowed to take the medication as usual following the fasting glucometer reading. The Michigan State University Committee on Research Involving Human Subjects and Sparrow Hospital reviewed and approved the study protocol.

Materials and methods

The study utilized a randomized, cross-over design where subjects consumed three breakfast and lunch meal combinations over the course of a 3-week period. Each subject ingested each meal type. Breakfast A was a high-glycemic breakfast meal with farina; Breakfast B was a low-glycemic breakfast meal with a psyllium ready-to-eat cereal, and a third breakfast (C) consisting of farina was administered to address objectives not relevant to this particular study.

The food items contained in the breakfast control and test meals are provided in Table 1. Breakfast B had a 26 % lower-carbohydrate content than Breakfast A. The psyllium fiber ready-to-eat cereal was developed and manufactured by Kellogg Company, Battle Creek, MI, USA, and the farina was purchased commercially from Malt-O-Meal Company, Northfield, MN, USA. The glycemic index (GI) values of the psyllium loop and farina cereals were analyzed using white bread as reference and the results were 56 and 64, respectively. The GI of the mixed meal was not measured as the methodology has yielded mixed results (Brouns *et al*., 2005). The calculated difference between the low-glycemic load response breakfast meal containing psyllium soluble

Table 1 Food composition of breakfast meals[a,b]

Food Items	*Breakfast A*	*Breakfast B*
Breakfast cereal (2 servings)	Farina	Psyllium ready-to-eat (RTE)
Milk (1 cup)	Skim	Skim
Bread (1 slice)	Wheat toast	Wheat toast
Spread (1 Tsp)	Margarine	Margarine
Beverage	Coffee or tea	Coffee or tea

[a]Goals for meals were 30% of total daily energy intake based on an 1800 kilocalorie meal plan.
[b]A serving of farina is 1 cup (3 tbsp/ 1 cup cooked); a serving of psyllium loop cereal is 2/3 cup.

fiber was 38% lower than the high-glycemic load response breakfast meal.

Lunch meals consisted of split-top, cracked wheat bread (Taystee), American cheese, fat-free turkey, lettuce, a slice of tomato, fat-free mayonnaise, a mustard package and non-caloric beverages. The nutrient composition of the breakfast and lunch meals is outlined in Table 2. The goal of both the breakfast and lunch meals was for each meal to provide approximately 30% of a total daily energy intake. The meals were adjusted to meet the caloric requirement of each subject, with the majority of the sample population prescribed to 7531 kilojoules (KJ) (1800-kilocalorie) diet plans. Unrestricted amounts of coffee, tea, water, artificial sweetener and nonfat creamer were also offered to subjects at breakfast and lunch meals.

Before each breakfast meal, fasting glucometer readings were taken to ensure subjects were not hypoglycemic. Only subjects who had glucose readings at $\geqslant$126 mg/dl were allowed to proceed. Subjects were then served breakfast and were permitted to take 15–20 min to complete the meal. Postprandial blood samples were taken at several intervals both after breakfast and lunch. All testing and clinical measurements were conducted at the G. Malcolm Trout Food Science and Human Nutrition Building at Michigan State University.

Blood samples were taken at various time intervals: 30, 45, 60, 90, 120, 180 and 210 min after the breakfast, and postlunch at 285, 300, 330, 360, 390, 420 and 450 min. Postprandial concentrations of plasma glucose, insulin and FFA were then determined. The statistical analyses for these concentrations were computed as area under the curve (AUC) values, and are represented in Table 5. AUC calculations were expressed in hours, 0–210 min for the AM period, and 285–450 min for the PM period. The time period for the PM period AUC calculations began at 285 min due to the absence of a baseline blood draw at lunch. Of the total PM measurements, approximately 10% is missing for the treatment analyses. The glucose is expressed as mg/dl h, insulin AUC is expressed in μIU/ml h, and FFA concentration is expressed in mg/dl h.

Table 2 Nutrient composition of breakfast and lunch[a,b,c]

	Breakfast A	*Breakfast B*	*Lunch*
Energy (kJ)	1833	1515	2378
Protein (g)	19	15	24
Carbohydrate (g)	78	62	75
Tat (g)	5.5	6.0	19
Dietary fiber (g)	3.4	12.4	2
Soluble fiber (g)	1.0	6.6	

[a]The nutrient content of meals was calculated using Nutritionist V Data Analysis software (First Databank, Inc., 1999–2000) and Kellogg Chemistry Laboratory.
[b]The differences in fiber content between Breakfasts A and B were due to the amount of fiber contained in the psyllium loop cereal. The loop cereal provided 6.6 g of soluble fiber and 4.4 grams insoluble fiber. The remaining fiber present was contained in the farina (1 g each of soluble and insoluble fiber) and the cracked wheat bread (1.4 g insoluble fiber).
[c]Values shown are for serving sizes based on an 1800 kilocalorie meal plan.

The blood samples were processed for commercial analyses according to the instructions provided by the Sparrow Hospital Laboratory, Lansing, MI, USA. Fasting blood glucose, HbA_{1C}, insulin and lipoprotein analyses, and the treatment glucose and insulin analyses were performed by Sparrow Hospital. The Mayo Clinic (Rochester, MN, USA) provided the FFA analyses.

Serum glucose concentrations were determined using the hexokinase and ultraviolet methods with an Olympus AU640 Spectrophotometer (Olympus America Inc., Melville, NY, USA). Serum insulin concentrations were determined using immunoenzymatic and chemiluminescence methods with the Beckman Access Detector (Beckman Instruments, Brea, CA, USA). The HbA_{1C} was determined using HPLC with the Tosoh A1C 2.2 Detector (Tosoh Medics Inc., South San Francisco, CA, USA). The FFA concentrations were determined with enzymatic and colorimetric methods using the Hitachi 912 Spectrophotometer (Roche Diagnostic Corp., Indianapolis, IN, USA). Other lipid measurements were done locally. Serum HDL-C was determined using homogenous, liquid selective detergent) and TG concentrations were determined using the enzymatic method with the Olympus AU640 Detector (Olympus America Inc., Melville, NY, USA). LDL-C was calculated with the Friedewald formula (Friedewald *et al*., 1972).

Area under the curve was computed by the trapezoidal rule, with the baseline being zero. The analysis of each AUC measure was performed separately, and all analyses are expressed as the log-transformed AUC as a dependent variable in a mixed model with each patient as random effect, and diet and week as fixed effects. The log transformation helps mitigates the effect of skewness in AUC values. Graphical checks of normality were made by quantile-quantile (Q-Q) plots for each treatment, and for the entire test period. The hypothesis of log AUC values for each of the diets was formally verified by the Shapiro–Wilk test, *P*-values exceeded 0.77 (Shapiro *et al*, 1991).

Statistical analyses

Analyses of the effects of diet on AUC values were based on a mixed model in which the log-transformed AUC was the dependent variable, with subject as random effect, and diet and period as fixed effects. Analyses of AUC values in the postlunch period were controlled by including the post-breakfast AUC as a covariate in the model. Comparisons between diets were based on *t*-tests. Confidence intervals at 95% were calculated for differences used the Bonferroni adjustment for multiple comparisons. A *P*-value of $\leqslant$0.05 was used as the level of significance. All statistical analyses were performed using SAS Software version 8.02 (SAS Institute, Cary, NC, USA).

Results

Subjects

Fifty adult subjects, 32 males and 13 females, previously diagnosed with Type 2 DM participated in the study. Five subjects failed to fulfill the requirements for blood draws and their records were not evaluated. On the basis of subject interviews and observations, compliance to pre-experimental guidelines was determined to be excellent for both control (Breakfast A) and treatment (Breakfast B). Previous research comparing subjects with diet alone and diet in combination with oral hypoglycemic agents revealed no substantive differences in postprandial glycemic responses and thus, a separate analysis was not conducted in this study (Pastors *et al*., 1991; Anderson *et al*., 1999).

Baseline characteristics of the study population are summarized in Table 3. Fasting baseline serum characteristics of the subjects were similar prior to the consumption of the control and the test breakfast meals in this randomized, controlled, crossover design study (Table 4).

Table 3 Characteristics of study participants[a,b]

Characteristic	*Males (n = 32)*	*Females (n = 13)*
Age (years)	64 ± 2	59 ± 3
Height (m)	1.77 ± 0.01	1.65 ± 0.02
Weight (kg)	96.4 ± 2.8	92.1 ± 4.2
Body mass index (kg/m^2)	34.1 ± 1.8	30.9 ± 0.9
Diabetes management[b]		
Diet alone	13	8
Oral hypoglycemic agents		
Sulfonylurea	10	2
Metformin	4	2
Sulfonylurea and metformin	4	0
Antihypertensive medication	2	0
Lip id-lowering medication	0	1

[a]Mean ± s.e.m. of baseline values at week 0 before breakfast treatments.
[b]Totals are not additive as some subjects were using multiple prescribed medications.

Table 4 Baseline fasting serum glycemic, insulin and lipid parameters of subjects by breakfast treatment[a,b,c]

Variable	*Breakfast A*	*Breakfast B*
Fasting serum glucose (mg/dl)	130.9S ± 4.93	133 16 ± 4.37
Serum insulin (μIU/ml)	11.45 ± 1.27	10.86 ± 1.14
HbA_{1C} (%)	6.91 ± 0.18	691 ± 0 16
Free fatty acids (mg/dl)	532.77 ± 43.22	550.27 ± 33.68
Triglycerides (mg/dl)	188.33 ± 29.44	185.56 ± 19.32
HDL cholesterol (mg/dl)	41 89 ± 1.43	41.53 ± 1.28
LDL cholesterol (mg/dl)	111.09 ± 4.93	113.67 ± 6.02
Total cholesterol (mg/dl)	186.02 ± 4.66	189.91 ± 5.40
Total cholesterol: HDL Chol[c]	4.68 ± 0.23	4.75 ± 0.19

[a]Mean ± s.e.m. Baseline values of key outcomes prior to each breakfast treatment. There were no significant differences between groups ($P > 0.05$).
[b]Meal types consist of (1) Breakfast A including farina with no psyllium soluble fiber and, (2) Breakfast B including ready-to-eat psyllium loop cereal.
[c]Ratio of total cholesterol to HDL cholesterol.
Abbreviations: HDL, high-density lipoprotein; LDL, low-density lipoprotein.

First meal responses

Glucose, insulin and free fatty acid concentrations. Fasting and postprandial morning (AM) and afternoon (PM) glucose, insulin and FFA concentrations are shown in Figures 1–3. Glucose concentrations peaked at 60–90 min for both the control and treatment: Breakfast A (230 ± 8 mg/dl) and Breakfast B (200 ± 8 mg/dl). Concentrations of glucose steadily began to decline, and returned to near baseline levels by 210 min (Figure 1). Insulin concentrations for Breakfast A peaked at 60 min (74 ± 5 μIU/ml), while Breakfast B serum insulin peaked at 90 min (45 ± 5 μIU/ml) and remained significantly lower than Breakfast A throughout the AM period (Figure 2). The pattern of change for FFA concentrations was markedly different from the glucose and insulin observations. Initially, morning FFA concentrations declined, and then increased at the 90-min time period for Breakfast A (615 ± 57 mg/dl), and at 60 min for Breakfast B (721 ± 69 mg/dl). The FFA concentrations then dropped to ~50% of these peak concentrations, and overall remained below baseline concentrations for both breakfast types for the duration of the AM morning period (Figure 3).

Glucose, insulin and free fatty acids area under the curve

Summary comparisons for AUC values for the subject responses were analyzed by diet and by week for the AM morning period and the PM postlunch period. There was no effect of week for breakfast or lunch, and thus any carryover effect is presumed to be the same for both breakfast treatments.

Table 5 collapses the AM morning and PM afternoon data for all subjects ($n = 45$) by breakfast treatment with compar-

Table 5 Area under curve values for glucose, insulin, and free fatty acids by breakfast treatment in the AM and PM ($n = 45$)[a,b,c,d]

Time of day	*Breakfast A*	*Breakfast B*
AM		
Glucose (mg/dl/h)	6.45 ± 0.04[a]	6.36 ± 0.04[b]
Insulin (μIU/ml/h)	4.86 ± 0.11[a]	4.47 ± 0.10[b]
Free fatty acid (mg/dl/h)	7.00 ± 0.07[a]	7.16 ± 0.07[b]
PM		
Glucose (mg/dl/h)	5.84 ± 0.03[a]	5.92 ± 0.03[a]
Insulin (μIU/ml/h)	4.23 ± 0.06[a]	4.55 ± 0.06[b]
Free fatty acid (mg/dl/h)	7.13 ± 0.05[a]	7.07 ± 0.05[a]

[a]Values are expressed as log AUC values.
[b]Values in the same row with different superscript letters are significantly different ($P < 0.05$).
[c]Mean values and standard error of the mean.
[d]Meal types consist of (1) Breakfast A including farina with no psyllium soluble fiber and, (2) Breakfast B including ready-to-eat psyllium loop cereal.
Abbreviations: AUC, area under the curve.

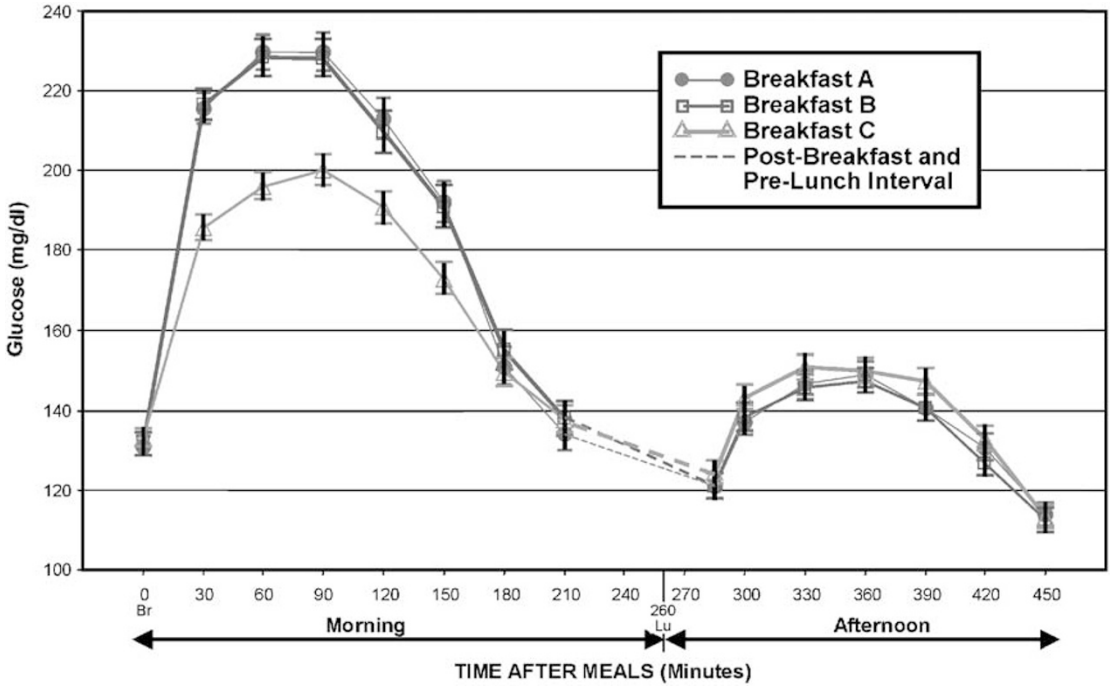

Figure 1 Mean±s.e.m. fasting and postprandial serum glucose concentrations during the AM and PM in subjects with Type 2 diabetes mellitus.

isons for glucose, insulin and FFA responses using area under the concentrations-versus-time curves (using a log scale and expressed as log AUC). The AM period includes values at 0 min–210 min. As expected, a high glycemic response meal (Breakfast A) resulted in significantly greater glucose AUC (6.45±0.04 mg/dl h; $P \leqslant 0.05$) versus Breakfast B (6.36± 0.04 mg/dl h) and insulin AUC values (4.86±0.11 μIU/ml h; $P \leqslant 0.05$) than for subjects fed Breakfast B (4.47±0.10 μIU/ml h). The FFA AUC values were significantly lower for Breakfast A (7.00±0.07 mg/dl h) versus Breakfast B (7.16± 0.07 mg/dl h) ($P < 0.05$).

Second meal responses

Glucose, insulin and free fatty acid concentrations. After lunch, at 285 min, plasma glucose averaged 121±6 mg/dl and 124±6 mg/dl for Breakfasts A and B, respectively. These concentrations were below AM fasting baseline values (Table 4). Glucose values during the PM period were fairly flat for both breakfast types between 330 and 360 min, with Breakfasts A and B at 149±7, and 151±6 mg/dl, respectively. At the end of the postlunch period, values were below fasting baseline levels in the AM period (Figure 1).

Insulin concentrations leveled by the end of the AM period, but remained above the prebreakfast concentrations. Plasma insulin concentrations of subjects fed Breakfast A peaked at 52±14 μIU/ml by 300 min (40 min after consumption of the lunch). In contrast, insulin concentrations for subjects fed Breakfasts A peaked at time 360 min (45±4 μmIU/ml), while insulin concentrations for subjects fed Breakfast B peaked at 390 min after lunch (47±9 μmIU/ml). At the end of the postlunch period, insulin concentrations were lowest for Breakfast A (24±3 μIU/ml.) and highest for Breakfast B (31±7 μIU/ml), with insulin concentrations 22% higher in Breakfast B at the end of the PM period (Figure 2).

The FFA concentrations continued to level off postlunch for both control and test breakfasts. Serum FFA concentrations for subjects fed both breakfast types peaked at 360 min, ranging from 705±58 to 762±64 mg/dl. At the end of the PM period, FFA concentrations declined below baseline fasting values both in the AM and PM periods. Breakfast B resulted in the highest FFA concentrations for both the AM and PM periods. However, by the end of the PM period (450 min) FFA concentrations were similar for both breakfast types: 299±21 mg/dl and 293±23 mg/dl for Breakfast A and B, respectively (Figure 3).

Glucose, insulin and free fatty acids area under the curve (area under the curve)

The postlunch analyses for AUC calculations began at 285 min. The glucose AUC values were slightly lower with control Breakfast A (5.84±0.03 mg/dl h) versus Breakfast B (5.92±0.03 mg/dl h), but neither of the treatments differed statistically. Insulin AUC values for Breakfast A (4.23±0.06 μIU/ml h) differed statistically from Breakfast B (4.55±0.06 μIU/ml h; $P \leqslant 0.05$). In the postlunch (PM) analyses, the FFA AUC values were unaffected by breakfast type.

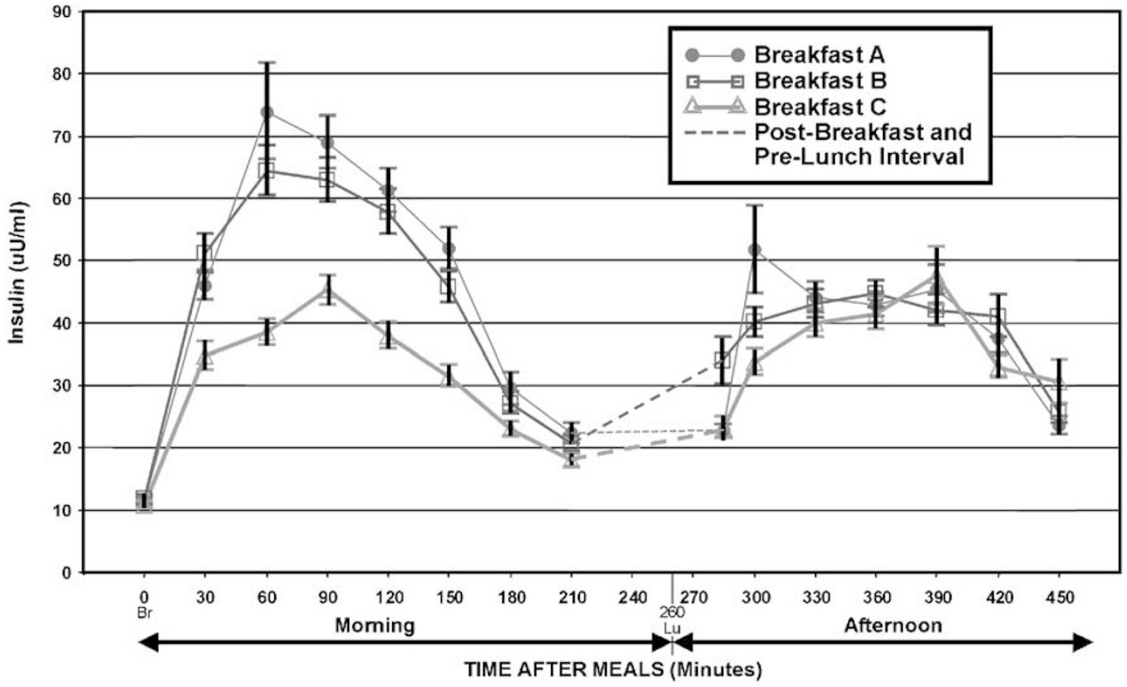

Figure 2 Mean±s.e.m. fasting and postprandial serum insulin concentrations during the AM and PM in subjects with Type 2 diabetes mellitus.

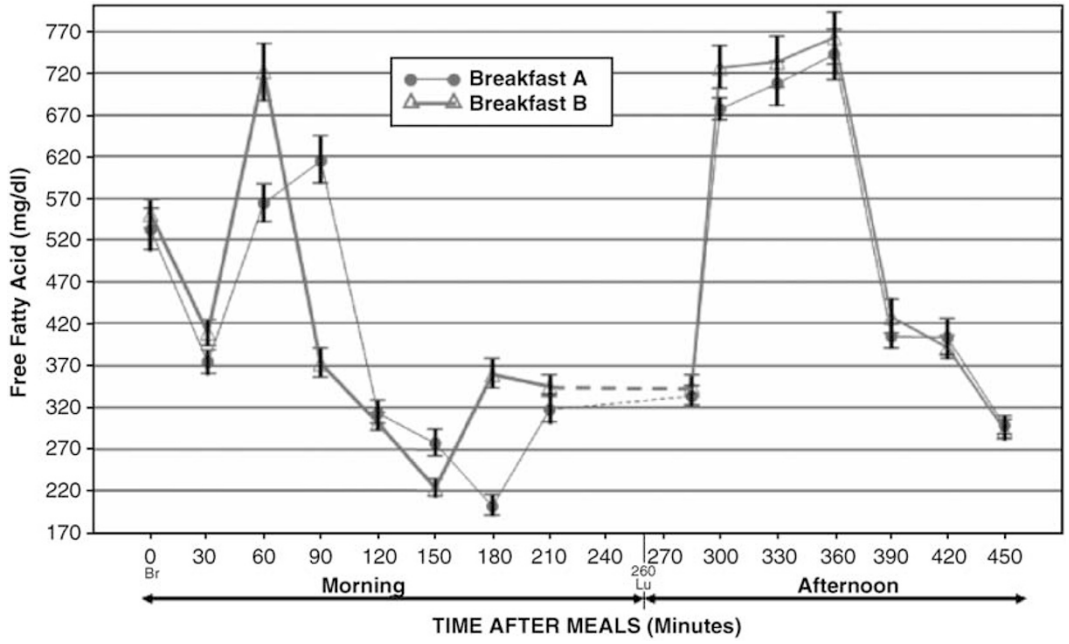

Figure 3 Mean±s.e.m. fasting and postprandial serum free fatty acids concentrations during the AM and PM in subjects with Type 2 diabetes mellitus.

Discussion

The effects of psyllium dietary fiber as a meal supplement on glycemic response have been studied previously in individuals with Type 2 DM (Pastors *et al*., 1991; Anderson *et al*., 1999; Sierra *et al*., 2001, 2002). There has been limited work with psyllium incorporated into the meal (Wolever *et al*., 1991). Intimate mixing of food and fiber seems to be an

important factor in effecting an improvement in glycemic response (Rendell, 2000). The goal of this study was to first test the efficacy of a low-glycemic load breakfast meal containing psyllium soluble fiber, and, then to assess its residual effects after a second meal.

As expected, postprandial glucose concentrations and AUC values were lower for the psyllium Breakfast B as compared to the control Breakfast A containing no psyllium. Previous studies have similarly reported that psyllium reduced fasting serum glucose concentrations in individuals diagnosed with Type 2 DM (Anderson *et al*., 1999; Sierra *et al*., 2001, 2002). Postprandial insulin responses were similar to the glycemic responses. The elevated insulin secretion associated with Breakfast A is consistent with other acute and chronic studies administering high glycemic response meals (Garg *et al*., 1994; Holt *et al*., 1996). Conversely Breakfast B had lower postprandial insulin concentrations and AUC values, consistent with other short-term studies that suggest an improvement in insulin action and pancreatic function associated with low glycemic index and low-glycemic response meals (Liljeberg *et al*., 1999; Bjorck *et al*., 2000).

Postprandial FFA concentrations dropped at the beginning of the AM period and then increased sharply before declining below baseline levels. The morning AUC values for FFA were significantly lower for the control Breakfast A versus the psyllium test Breakfast B meal. This may reflect the lowered insulin response to the low-glycemic response meal, consistent with previous research suggesting that a low glycemic index meal yielded a lower-insulin response with an increase in FFA concentrations (Wolever and Mehling, 2003).

Doses of guar, psyllium, and pectin ranging from 5.1–14.5 g consumed in the first meal shown to exhibit postprandial effects immediately following the first meal have also resulted in residual effects that blunt postprandial glycemia after meals eaten several hours after the fiber ingestion (Pastors *et al*., 1991; Anderson *et al*., 1999; Sierra *et al*., 2001). The afternoon (PM) postprandial serum glucose AUC values were actually slightly higher (6% on an unlogged scale) when subjects consumed Breakfast B than when Breakfast A was consumed. Similarly, the insulin demand was statistically higher (AUC value of 23% on an unlogged scale) for Breakfast B as compared with Breakfast A. Thus, the PM glycemic and insulinemic responses did not parallel the AM responses.

As the postprandial glucose profile does not exhibit serum glucose concentrations at or below the initial fasting concentration, it cannot be concluded that the psyllium test meal had a residual second meal effect. Sierra *et al*., 2002 observed an individualized response to psyllium by subjects diagnosed with diabetes, and we noted similar individualized responses. Additional research examining the mechanisms underlying the second-meal effect should be further explored. This would potentially entail the measurement of various hormonal concentrations such as glucagon, catecholamine and other counter-regulatory hormones to elucidate the mechanism.

It has been suggested that altering the amount and type of carbohydrate many influence FFA rebound in healthy subjects (Wolever *et al*., 1995) and in individuals with Type 2 DM (Gannon *et al*., 1998). Consumption of high-, moderate- and low-carbohydrate meals in subjects with Type 2 DM on glycemic control showed a significantly smaller glucose plasma area under the curve after a low-starch meal compared to a high and moderate starch meal with a similar decrease in NEFA response after all meals. It was suggested that the NEFA response was due to elevated insulin concentrations, presenting a maximal effect on lipolysis. We observed a similar reciprocal response after the breakfast meal with an elevated FFA response associated with the low-glycemic load breakfast meal. This was likely due to the difference in the CHO and energy content of the meal, which is the consequence of the addition of dietary fiber to the breakfast meal. However, the dramatic increase immediately postlunch in FFA concentrations is not readily explainable based on the biomarkers measured. As the fat content of the standardized lunch was at 30% of the total energy, we would not have expected this level of fat to exert an exorbitant influence on plasma FFA response. It would have been useful to determine if triacylglycerol and lipid levels were affected in a similar manner to the FFA, given that chronic ingestion of low-glycemic response meals are associated with a rise in triacylglycerols (Wolever and Mehling, 2003).

Dietary management can lead to a reduction in risk associated with cardiovascular disease and other co-morbidities among individuals with Type 2 DM. Our results indicate that the morning glycemic and insulinemic responses were favorably influenced by the low-glycemic load response meal. It would appear that carbohydrate foods with high-fiber content may alter glucose absorption, and thereby result in lower-glycemic and insulinemic responses. The American Diabetes Association recommends that priority is given to the amount of carbohydrate present in a food. Our findings suggest that both the type of and amount of carbohydrate may be important determinants of glycemic control. However, while low-glycemic load response foods may have an impact on glycemic control, it is unclear whether or not this benefit is achievable long-term. The acute nature of our study and the absence of isoenergetic meals renders this finding inconclusive.

This study showed that the acute ingestion of psyllium soluble fiber combined with a lower total carbohydrate intake at the morning meal did not result in a residual second meal effect after consuming a standardized lunch. The sharply elevated plasma FFA concentrations postlunch underscore the importance of understanding the influence of the increased availability of FFA in inhibiting insulin-stimulated glucose uptake (Boden, 1997; McGarry 2002). Hence, medical nutrition therapy among individuals with Type 2 DM must be individualized to reflect abnormalities in

both fat and carbohydrate metabolism. The approach of taking into consideration the metabolic effects of the glycemic response to a low-glycemic load breakfast meal containing psyllium soluble dietary fiber offers an additional dietary tool that may help improve the management and treatment of Type 2 DM.

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