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Optimizing the food guide pyramid to increase fat oxidation in young adult men

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TABLE OF CONTENTS

ABSTRACT

I tested two hypotheses: The first hypothesis was that a mixed diet containing lower GI foods would lower blood glucose and insulin levels and increase fat oxidation (FOX) compared with a diet consisting of higher GI foods. Secondly, I tested the hypothesis that variability in resting FOX would predict FOX during moderate exercise after consuming the lower and higher GI diets. Lower and higher GI diets with similar macronutrient contents were constructed using low and high GI versions of cereal, bread, fruits, snacks and vegetables. Diets were fed to 12 normal, untrained, young men for 4 d using a crossover design with a 3-day washout. The lower GI diet decreased postprandial blood glucose and insulin concentrations ($P<0.05$), increased plasma fatty acids ($P<0.05$), but did not affect plasma triglycerides or fasting plasma glucose compared with the higher GI diet. Macronutrient oxidation at rest and during the first 40 min of moderate exercise was not affected by diet. The lower GI diet decreased FOX by 20 % at 60 min of exercise compared with either the habitual or the higher GI diets. Body weight decreased slightly (-0.7 kg, $P<0.05$) after the lower GI diet, but did not change after the higher GI diet. Subjects were separated according to their mean resting FOX into higher and lower fat oxidizers. Both groups responded similarly to the diets in all measures. Higher fat oxidizers consumed more fat habitually, had lower postprandial glucose and insulin levels on both diets, and had higher pre-meal fatty acid concentrations irregardless of the diet ($P < 0.05$). I concluded that inclusion of low GI foods in a balanced diet modestly elevated FOX after at least 60 min of moderate exercise in normal young men. Subject having higher resting FOX rates had a

muted glycemic response and insulin response to an evening meal and had the higher FOX rates the next day during exercise. These data may be helpful in developing better diets for weight control and assist in identifying individuals at rest of weight gain.

INTRODUCTION

The glycemic index is the ratio of the incremental area under blood glucose response curve for carbohydrate-containing foods to the corresponding area after a standard food (white bread or glucose) having an equivalent amount of carbohydrate (1). Carbohydrate foods having similar macronutrient composition may have different plasma glucose responses known as glycemic response (2). The concept "glycemic index" was proposed to better understand the digestion, absorption and the glycemic responses of different carbohydrate foods. Many experiments examined the metabolic responses produced by individual high or low glycemic index foods. Low glycemic index foods may lower the postprandial glycemic responses (3, 5), postponed the appearance of plasma glucose and insulin peaks (3), reduced the serum total cholesterol (4), LDL cholesterol, Apolipoprotein B, free fatty acids and triacylglycerol concentrations (6).

The glycemic index concept has been proved to be applicable in mixed meals (7, 8, 9, 10). Low glycemic index foods combined in a mixed meal may still produce different metabolic effects when compared with the combined high glycemic index foods. However, the ability of the low glycemic index foods in the mixed meals to produce beneficial effects is still controversial (48). Only a few experiments studied the application of glycemic index concept into meal planning for a complete diet and then linked the consumption of a mixed diet containing low and high glycemic index foods in a mixed diet to subsequent metabolic effects. It is still not clear whether eating a mixed diet consisting of low glycemic index foods everyday for a period of time will be beneficial for people's health other than better

glycemic control for diabetes patients. It is also not clear whether a low glycemic index diet can be designed to provide similar nutrients as a balanced ordinary diet. To answer these questions, I selected commonly available low glycemic foods based on their glycemic indices measured in original scientific reports and used them to replace higher glycemic index foods. I varied the servings of the lower and higher glycemic index foods to make both diets have similar macronutrient composition. All the foods selected are commonly eaten and can be purchased at the local grocery stores. Subjects consumed the high and low glycemic index diets respectively for four days and their metabolic changes after the high and the low glycemic index diets were observed. The baseline and post-pradial plasma glucose, post-prandial insulin, free fatty acids and triacylglycerol levels were measured to study the metabolic effects after these diets. Subjects' evaluation of the diets was also recorded.

To study the effects of low glycemic index foods on the fuel utilization during the exercise, some experiments observed the effects of pre-exercise foods or meals having the low or high glycemic index on the fat and carbohydrate oxidation in the following exercise. They also studied the metabolic responses after those meals and during the exercise. However, the findings of those experiments were not consistent. Some studies found that the ingestion of low glycemic index meal before exercise could increase carbohydrate oxidation during following exercise (11, 12), but others observed that a pre-exercise low glycemic index meal lowed the carbohydrate oxidation and accelerated fat oxidation during the exercise (13, 14). Most, if not all, experiments tested the effects of glycemix index on fuel utilization used pre-exercise foods or meals.. I am not aware of any experiment that examined the effects of eating high versus low glycemic index foods as part of a mixed diet on fuel utilization during the exercise. We proposed the hypothesis that the consumption of

low glycemic index foods as part of a nutritionally balanced diet would increase the fat oxidation at rest and during exercise. Obesity is the risk factor of atherosclerosis, hypertension, cardiovascular disease, type II diabetes, insulin resistance, hypercholesterolemia, hyperlipidemia and is related to renal disease. If including the low glycemic index foods improves fat oxidation and lowers plasma glucose and insulin levels, an optimized food guide pyramid to construct diets to prevent overweight and obesity could be developed. Low fat oxidation is a factor that could contribute to obesity and a diet including low glycemic index foods that optimize fat oxidation may be useful for the prevention of obesity.

This thesis is organized into four chapters. The first chapter reviews previous research on glycemic index, glycemic responses, and the physiological effects glycemic index foods during postprandial and exercise states. The second chapter is a manuscript prepared for submission to the American Journal of Clinical Nutrition. This manuscript describes the study design, the methods used in this study and presents the study results. Chapter four summarizes the findings and discusses the potential future work of this area. Cited literature is included at the end of each section.

LITERATURE REVIEW

The Glycemic Index

The glycemic response of a food is defined as the elevation of blood glucose after eating that food. Different carbohydrate foods may have various glycemic responses. Even the carbohydrate foods with the same macro-nutrient composition may produce different glycemic responses (2). Scientists may compare the glycemic responses of different foods measured in same experiment when that experiment uses the same subjects and same measuring methods. However, the glycemic responses of foods measured from different studies are not always comparable because many factors affect the measurement of glycemic responses. To make measurements of glycemic responses more comparable, Jenkins and Wolever proposed the concept of the glycemic index (1). They proposed that if glycemic responses of different foods were compared to a common standard first, then the results from different subjects in different experiments could be compared. Glycemic index is defined as the ratio of the incremental area under the blood glucose response curve for a test food against the corresponding area after a standard food (white bread or glucose) containing the same carbohydrate amount (1). By comparing the glycemic indices of different foods, glycemic responses of foods measured in different groups of subjects and different experiments may be compared. Foods having high or low glycemic indices produce different plasma glucose responses. High glycemic index foods have higher post-prandial plasma glucose responses than low glycemic index foods. Theoretically, high and low glycemic index foods would produce blood glucose response curves as shown in Figure 1.1.

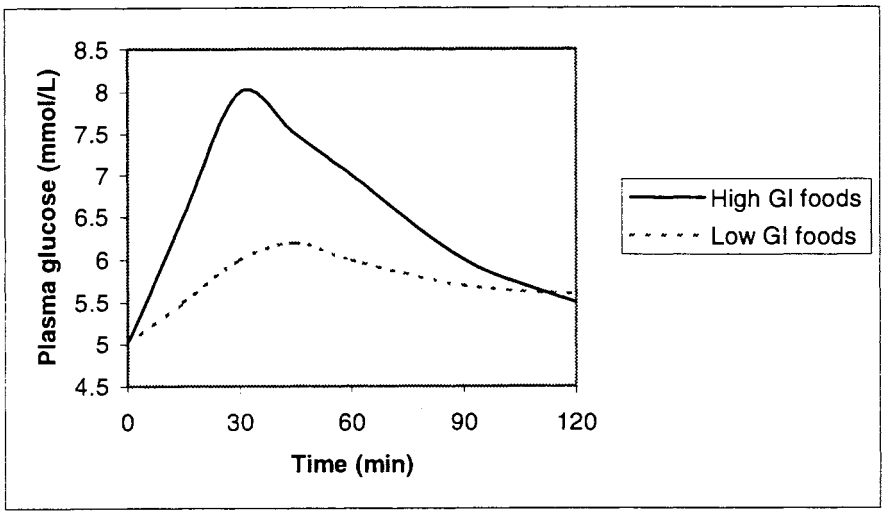

Figure 1.1. Theoretically plasma glucose response curves of high and low GI foods.

Factors Affecting Glycemic Responses

Glycemic responses are not soley determined by its chemical composition because carbohydrate foods with similar chemical composition induce various glycemic responses. Aside from the carbohydrte stucutre, i.e. amylose versus amylopectin, the soluble fiber content, , presence of antinutrients , particle size of the food and food processing methods can affect the glycemic responses to a carbohydrate containing food.

Fiber and glycemic responses

Total fiber content had been believed to affect glycemic responses, but many studies showed that the fiber effect on lowering the glycemic responses is due to soluble fiber not insoluble fiber. Jenkins et al. (15) found that adding viscous guar to a test meal had the greatest attenuating effect on post-prandial glucose responses in healthy adults compared with the other test meals containing equivalent amount of other fiber such as pectin, gum tragacanth, methylcellulose, wheat bran, or cholestyramine. When hydrolyzed non-viscous guar was added, the difference in glucose response between the viscous guar and other fiber

containing foods disappeared. From these experiments, Jenkins et al. concluded that the viscosity of a fiber was directly correlated with the reduction of the post-prandial peak in blood glucose concentrations and further suggested that viscous types of dietary fiber were important in lowering glycemic responses. Gel-forming fibers were able to lower the post-prandial glucose and insulin responses (16, 17). Hallfrisch et al. (18) compared the post-prandial glucose responses following oral challenges of glucose, glucose plus highly viscous oat extract containing 1% glucan, or glucose plus highly viscous oat extract containing 10% glucan. Both extracts lowered the glycemic responses relative to the glucose oral challenge. Furthermore, insoluble fiber had no similar effect as soluble fiber to reduce the glycemic responses. McMurry et al. (19) observed the effects of adding a large amount of wheat bran to a mixed meal in seven insulin-dependent diabetic patients. Despite the test meal containing the wheat bran having a higher total fiber amount than the test meal without the wheat bran, the glucose responses including the magnitude and the timing of the peak blood glucose concentration were similar. Similarly, Hall et al. (20) studied the effect of bran on glucose kinetics and plasma insulin levels, and found that bran did not affect the glucose tolerance and the metabolic clearance rate of glucose in non-insulin-dependent diabetes mellitus patients controlled by diet alone. Using another source of insoluble fiber, cellulose, Monnier et al. (21) observed that cellulose supplementation did not affect the peak plasma glucose in eight diabetic patients after they consumed an oral glucose load. Plasma insulin levels were also not significantly affected when glucose was consumed with or without pectin, cellulose or cellulose phosphate respectively. Together these results clearly demonstrate that insoluble fiber does not play an important role in changing the glycemic responses.

To summarize, adding viscous soluble fiber, not insoluble fiber, lowers the glycemic response. The lower glycemic responses observed with viscous fiber may due to delay of the glucose diffusion rate in small intestine caused by viscosity (22, 23).

Not all studies clearly demonstrate the effectiveness of viscous fiber. For example, Anette et al. (24) examined the effects of a low glycemic index diet with variable insoluble and soluble fiber content. The low glycemic index diet lowered the post-prandial insulin and glucose concentrations compared with a high glycemic index diet despite having higher content of insoluble fiber and similar amount of soluble fiber as the high glycemic index diets. These results suggest that either the glycemic index can be separated from fiber effects or maybe the function of soluble fiber can be diminished in the mixed meals. The relative contributions of soluble fiber and the glycemic index, independent of soluble fiber due to carbohydrate structure or other factors remains a subject of investigation..

Starch structure affects the glycemic index

Amylose is a straight chain starch in which glucose residues are linked by alpha-1, 4 linkages. Each chain of amylose can form an alpha- helix and the adjacent alpha-helic chains are bound together by hydrogen bonds. Amylopectin is a highly branched starch consisting of alpha-1, 4 linkages between the glucose molecules and alpha-1, 6 linkages at the branch points. The highly branched amylopectin has more sites accessible to amylase such that the hydrolysis of amylopectin results in a greater release rate of glucose.. Additionally, amylose can form stable interactions with lipid as the hydrocarbon chains of the monoglycerides can insert into the holes of the amylose helix (25) making amylose more difficult to be hydrolyzed by amylase. Goddard et al. (26) observed the glucose and insulin responses of three types of rice containing 0, 14 to 17, or 23 to 25% of carbohydrate in the form of

amylose in 33 normal subjects. In their study, the glucose and insulin responses were decreased with the high amylose content rice, though all the rice had the same fiber content. These structural differences between amylose and amylopectin are the reason why foods containing high amounts of amylose produce lower glycemic responses than foods with high amylopectin content.

Particle size influences the glycemic responses

Particle size of carbohydrate foods also affects glycemic responses. Significantly higher glucose responses are observed after eating fine wholemeal flour than after consuming coarse wholemeal flour (27), Bread with intact kernels results in a lower glycemic response than white bread (28). These results suggested that the food having carbohydrates with smaller particle size and a correspondingly greater surface area for access to digestive enzymes would be digested more rapidly than the food containing carbohydrate in larger particle sizes and subsequently the smaller particle size generates a higher glycemic response than the larger particle size.

Cooking methods and time affect the glycemic responses

The glycemic responses of starch are ultimately due to the rates of starch digestion and absorption. Cooking and duration of cooking increase the glycemic responses after eating the starch by increasing the gelatinization of starch which facilitates the digestion of starch by enzymes (29, 30). Different food processing methods can influence the glycemic response to foods. Lentils boiled for 20 minutes decreased blood glucose response compared with white bread (30), but boiling lentils for 60 minutes or drying lentils dried for 12 hours after boiling eliminated the low glycemic response of the lentils to such an extent that the responses to lentils and and white bread were similar (30). Lentils blended to a paste also did

not lower blood glucose response compared with the white bread (30). Other processing methods such as extruding (31), flaking and popping (32) increases glycemic responses.

Phytochemicals that impair bioavailability of foods affect the glycemic responses

Antinutrients such as lectins (33) or phytate (34) decrease the glycemic responses by decreasing the digestion rate of starch. Lectins may bind to the amylase or the starch and inhibit the access of starch to the amylase active site (35). Lectins also could impair glucose transit in the intestine by binding to the surface glycoprotein of intestine mucosa and preventing glucose molecules from reaching transport sites (35). Phytate can also prohibit the access of starch to the active site of amylase, because phytate could bind to starch molecules by forming hydrogen bonds or bind to the protein of a starch-protein complex and make starch molecule inaccessible to digestive enzymes (36). The chelating action of phytate could sequester metal ions such as calcium, which is essential for the amylase activity. Phytate can also bind to the calcium already bound to amylase causing the enzyme to lose activity (36).

Dietary fat influences the glycemic responses

Gatti et al. proposed that the type of fatty acids could affect the glycemic response (37). They fed subjects a standard carbohydrate meal with equal amounts of unsaturated oils or butter. Both unsaturated oils and butter lowered the glycemic response; however, butter only delayed the rise of plasma glucose without significantly changing the plasma glucose concentrations. Insulin concentrations were unaffected by the type of dietary fat. This study suggested that the degree of saturation degree of fatty acids might also affect glycemic responses, but not insulin levels.

Sugar influences the glycemic responses

Janette et al. (38) studied the glycemic index of foods naturally containing sugars and found that the absolute amount of sugars in food could not predict the glycemic index of food. However, they did find that adding sugars to food increased the glycemic index of that food (38). Hughes et al. found that consuming equivalent amounts of fructose and glucose produced a similar glycemic response as that produced by consuming glucose alone in insulin-dependent diabetes mellitus patients (39). In another experiment, they found that adding 25g glucose to either bread or apples produced similar glycemic responses as that produced by feeding 25g glucose alone in insulin-dependent diabetes mellitus patients (39). According to these results, they proposed that the glucose content of food was the major determinant of the glycemic responses in the patients with insulin-dependent diabetes mellitus (39). To address the factors which might influence glycemic responses I standardized cooking methods and used the same source of dietary fat when constructing the high and low GI diets. Because I selected foods with demonstrated GI that could be easily found in a grocery store there are no differences in fiber and antinutrients between the low and higher GI diet as a result of the specific foods used in the diets. The factor we used to select foods was an experimentally determined glycemic index, which may have been due to a variety of factors endogenous to the chosen food.

Other Sources of Variability of the Glycemic Index

The glycemic index can vary widely depending on the difference of the test meals, the variability within subjects, the variability between subjects, using different standard

foods, collecting blood samples from veins or arteries, different length of sampling time after meals, and different repeating times of measurement (43).

Some food, i.e. fruit, picked in different experiments could produce differential glycemic responses depending on ripeness degrees (40, 41) or different processing methods (42).

Perhaps the most significant source of variability in measuring glycemic index is found when the same subject eats the identiical carbohdyrte foods under idientical experimental conditions and still produces different glycemic responses (7). Obviously different subjects could exhibit different glycemic responses to the same food as well. It is therefore unreasonable to compare the glycemic responses of subjects without using a standard (44). If we compare the glycemic responses observed in different groups to the glycemic responses caused by a standard food tested by the same subjects, then the relative glycemic response in different groups will become comparable (43). However, if examiners compare the glycemic response of certain food with different standard foods, like the white bread or the glucose used in most studies, then we have to carry out some converting between different standard foods for comparing those relative glycemic responses (45).

Glycemic responses can vary depending on from where blood is sampled. Glucose concentrations are lower in venous blood than arterial blood by as much as 2mmol/L (46) because veins collect the blood from capillaries, whereas arteries are carrying glucose to tissues. Another experiment showed that glucose concentrations are similar in capillary and arterial blood (47). The glycemic index calculated as the ratio of the area under blood glucose curve for a test carbohydrate food and the area for an equivalent carbohydrate standard (43) can therefore be affected by using venous or arterial blood as a source for blood samples.

To calculate the glycemic index multiple blood samples need to be collected from subjects to measure the incremental area under blood glucose curve after meal. To obtain an accurate glycemic index sampling time must include the complete post-prandial blood glucose curve. In normal subjects, the glycemic response usually peaks between 30 to 45 minutes after ingesting a glucose load and the glycemic response will return to baseline values within 30 to 45 minutes after the appearance of the peak value (43). In diabetic subjects, the glycemic response continues to increase for at least 90 to 120 minutes and may take 3 to 5 hours before reaching a peak (43). Failure to sample over a sufficient time period may cause inaccurate estimate of the area under the plasma glucose curve and subsequently an inaccurate glycemic index. The sampling time used to calculate the glycemic indices needs to be standardized in order to be able to compare result from different experiments.

Multiple observations are needed to establish glycemic indices because the same subject may have different glycemic responses to the same carbohydrate food at different times. Most studies repeated the glycemic index measurements for several times to minimize within subject variability. The glycemic indices calculated by repeating measurements for at least three times are thought to be more reliable (43).

We selected the foods for the higher and lower glycemic index diet according to their glycemic indices. In recognizing the above limitations in the glycemic index measurement we used the following critiera to choose a particular food for the lower and higher GI diets: 1. White bread or glucose was used as a standard food; 2. The sampling time was no less than 120 min; 3. The measurement of the glycemic index was repeated for at least 3 times.

Glycemic Responses to Mixed Meals

Most studies have measured the glycemic index for individual foods, but only a few experiments measured the glycemic index of mixed meals that contain not only the carbohydrates but also protein and fat. Hollenbeck et al. found in 1986 (48) that single food with different glycemic responses would have similar glycemic responses if they were consumed in mixed meals. Other experiments demonstrated the opposite, as glycemic indices of individual foods were useful in predicting the glycemic responses of the mixed meals (7, 8, 9, and 10). The addition of fat and/or protein to food does change the glycemic responses. However, Collier et al. (49) found that the addition of the same amount of fat to carbohydrate foods did not change the relative relationship between the lower and higher glycemic index foods with correspondingly lower and higher glycemic responses (49). Wolever et al. confirmed that although the addition of fat and protein affected the glycemic responses, the addition of fat and protein did not mask the difference in glycemic responses produced by mixed meals containing foods with different glycemic indices (50). Adding protein and fat has no additional glycemic effect in mixed meals containing glucose (39). Based on the hypothesis that the glycemic response is contributed by each individual carbohydrate food in the mixed foods proportionally to their glycemic index (51), Wolever and Jenkins proposed a formula to calculate the glycemic index of mixed meals (9). The meal glycemic index can be calculated as the sum of the products of the individual glycemic index of food and the carbohydrate proportion contributed by that food (9). However, the feasibility of this formula still needs to be verified by additional experiments.

Physiologic Parameters Influenced by the Glycemic Index

The glycemic index of food has been verified to have profound physiological effects. Low glycemic foods because of the lower blood glucose response similarly have lower the post-prandial plasma insulin concentrations in addition to lower blood lipid concentrations. (53).

Jenkins et al. observed lower 12-h blood glucose profiles and total serum cholesterol concentrations in 6 healthy subjects after a low glycemic index mixed diet (52). Twenty-four hour urinary C-peptide levels were used as the index of insulin secretion. These measurements are consistent with lower insulin secretion in response to a low glycemic index diet because C-peptide results from protease activation of proinsulin and is released together with insulin. However, lower 24-h urinary C-peptide levels can not indicate whether a low glycemic index diet lowers insulin secretion during a short time, i.e., immediately after the meal, or decrease the insulin secretion over a longer time period (52). Ritz et al. (53) were able to narrow the specific time frame by which low glycemic foods change plasma insulin, glucose and free fatty acids concentrations. They found lower plasma insulin and glucose concentrations occurred from 150 to 210 minutes and lower free fatty acids concentrations occurred from 210 to 360 minutes after consuming low glycemic foods. However, because the metabolic effects were measured after a single low glycemic index food (manioc starch) and a single high glycemic index food (glucose), the results may not represent accurately the physiological effects following a mixed diet containing low and high glycemic index foods. Anette et al. (24) compared plasma glucose, insulin and C-peptide concentrations after two groups of high vs. low glycemic index diets. The high and low glycemic index diets in the first group were same except the low glycemic index meal included a whole apple and the

pasta made from durum wheat whereas the high glycemic index meal included bread made from durum wheat flour and a crushed apple. In the second group, the low glycemic index meal included parboiled rice, whole kidney beans, whole wheat grain bread and the high glycemic index meal included sticky rice, ground kidney beans and whole wheat grain bread. The remaining foods in the high and low glycemic index diets were the same. Comparing the first group of diets, they observed that the time intervals at which the low glycemic index diet significantly lowered plasma glucose concentrations compared with the high glycemic index diet were between 60 and 120 minutes after meal. And at 90, 120 and 180 minutes after meal, the plasma insulin levels had significantly lower values after low glycemic index diet than after high glycemic index diets. The effects of the low glycemic index diet in the second group of diets were similar to that of the low glycemic index diet in the first group of diets with the exception of the time intervals at which the low glycemic diet produced significantly lower glucose and insulin levels. The plasma glucose levels were lowered earlier and for a longer time after meal by the low glycemic index diet in second group (at 30, 60, 90 and 180 minutes). Plasma insulin concentrations were lowered earlier by the low glycemic index diet in second group at 30, 60, 90 and 120 minutes after meal. The difference between those two groups of diets was that the low glycemic index diet in the second group had more total energy percentage coming from the low glycemic food than the low glycemic index diet in the first group. These results suggested that mixed meals having lower glycemic indices might not only have relatively longer effects of lowering post-prandial plasma glucose concentrations, but also have relatively earlier appearance and longer maintaining of the effects of lowering post-prandial plasma insulin concentrations. But in Anette's study, they did not observe any difference between the serum lipid concentrations after the low and high

glycemic index diets (24). Bente et al. (54) studied the change of plasma insulin, glucose and fatty acids after each meal in 7 healthy young men after they consumed three high and low glycemic index meals a day having similar macronutrient composition. In their study, differences of plasma insulin, glucose and fatty acids due to the glycemic index occurred after lunch and before meals. The lower plasma insulin, glucose and fatty acids concentrations occurred within 3 day of consuming different glycemic index diets (54). However, the difference decreased after consuming different glycemic index diets for 30 days, suggesting that the glucose metabolism might adapt to different glycemic index diets (54).

Ingesting carbohydrate foods with different glycemic indices may also affect the amino acid metabolism. Lyons et al. (55) compared the ratio of the plasma tryptophan to the branched chain amino acids after two carbohydrate meals. The ratio of plasma tryptophan to the branched chain amino acids was higher after sucrose than after starch meal. Tryptophan is the direct precursor of serotonin and the alteration of the ratio of the plasma tryptophan to the large neutral amino acids will change the rate of serotonin synthesis in brain (56). Therefore, starch meal may stimulate lower serotonin synthesis in brain than sucrose meal, which indicates that the lower glycemic index food may stimulate less serotonin synthesis than higher glycemic index food (55). But this point is still controversial; Frank et al. have completely opposite findings (57).

Some studies also found that the consuming of high glycemic index diets was related to the response of plasma leptin (58, 59) in both type I and II diabetic patients, but the mechanism of this effect is still unclear.

Physiologic Effects during Exercise Related to Glycemic Index

Many experiments have investigated the effects of the glycemic index of pre-exercise meals on substrate utilization and performance during exercise. Kirwan et al. observed the suppression of plasma free fatty acids and glycerol and the increased carbohydrate utilization during the first 120 minutes of exercise after a pre-exercise moderate glycemic index meal in six young adult females, but they did not observe a significant change of effects of glycemic index on endurance (60). They also compared the performance and the metabolic responses of 6 young adult males during exercise after lower, higher glycemic index test meals eaten 45 minutes prior to the exercise. Both the lower and higher glycemic index pre-exercise meals suppressed plasma free fatty acids compared with the control (water alone). Lower glycemic index meal tended to increase the carbohydrate oxidation during exercise compared with the control. The latter effect was thought to be the reason that exercise performance time was longer. On the other hand, they did not find any difference of exercise performance time or fuel utilization between consuming higher glycemic index test meal and control (60). The effects of increasing exercise endurance and increasing the carbohydrate oxidation of low glycemic index meal were observed by several other experiments (12, 63, and 64). But those results were controversial. Febbraio et al. (61) observed increased carbohydrate utilization during exercise only after pre-exercise ingestion of high glycemic index meal and they observed no exercise performance increase after high (instant mashed potatoes), low glycemic index test meals (muesli) or control (diet jelly). The finding of lower carbohydrate utilization during exercise after the low glycemic index diet was consistent with the findings of Wee et al (13) that the carbohydrate oxidation was lower at 20, 40, 60 and 80 minutes of the exercise and the fat oxidation was higher during the first

80 minutes of exercise after low glycemic index test meal. No existence of different exercise performance and different fuel utilization was observed in some other experiments (14, 62). Febbraio et al. also found that plasma free fatty acids concentrations and insulin concentrations were continuously lower during exercise after high glycemic index test meal and blood glucose concentrations were lower after high glycemic index test meal at 15 and 30 minutes during exercise (61).

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INCREASE FAT OXIDATION IN ADULT MEN BY OPTIMIZING DIETS

A Paper to be submitted to the American Journal of Clinical Nutrition

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Abstract

Background: Eating a low glycemic index (GI) food or meal prior to exercise increases fat oxidation (FOX).

Objective: We tested the hypothesis that a mixed diet containing lower GI foods will increase FOX compared with a similar mixed diet containing higher GI foods.

Design: Low and high GI diets with similar macro-nutrient contents were constructed using low and high GI versions of cereal, bread, fruits, snacks and vegetables. Diets were fed to 12 normal, untrained, young men for 4 d using a crossover design with a 3 day washout. Daily fasting blood glucose and post-prandial glucose, insulin, fatty acids and triacylglyceride concentrations after the last meal of each diet were measured. After an overnight fast following the end of each diet FOX and carbohydrate oxidation (CHOX) were estimated by indirect calorimetry during rest and one-hour moderate exercise.

Results: The lower GI diet decreased post-prandial blood glucose and insulin concentrations by 2 to 3 fold ($P<0.05$), increased plasma fatty acids ($P<0.05$), but did not affect plasma triacylglycerides or fasting plasma glucose compared with the higher GI diet. Macro-nutrient

oxidation at rest and during the first 40 min of moderate exercise was not affected by diet. The low GI diet increased FOX by 20 % at 60 min of exercise compared with either baseline (habitual diet) or the higher GI diet (4.4 ± 0.3 vs. 3.6 ± 0.3 Kcal/min and 3.8 ± 0.3 kcal/min, mean ± SD). Despite similar energy intakes body weight decreased slightly (-0.7 kg, $P < 0.05$) after the lower GI diet, but did not change after the higher GI diet. Subjects were separated according to their mean resting FOX into higher fat oxidizers and lower fat oxidizers. Both groups responded similarly to the diets in all measures. Higher fat oxidizers habitually consumed more fat, had lower post-prandial glucose and insulin levels on both diets, and had higher pre-meal fatty acid concentrations than low fat oxidizers ($P < 0.05$).

Conclusion: Inclusion of low GI foods in a balanced diet can modestly elevate FOX after at least 60 min of moderate exercise in normal young men.

Key words: Young adult men, glycemic index, fat oxidation, exercise, balanced diet

Introduction

Glycemic index (GI) reflects the rate of digestion and absorption of dietary carbohydrate and is defined as the ratio of the incremental area under the blood glucose response curve for a test food against the corresponding area after a standard food (white bread or glucose) containing the same carbohydrate amount (Jenkins 1981). The Glycemic indices for most commonly consumed foods that contain carbohydrate have been measured under standardized experimental conditions (Wolever 1990). The physiological significance of eating a mixed diet consisting of high or low GI foods, other than affecting glucose control in diabetes (Brand, 1994), is not clear because most GI data are derived from experiments

involving intake of single foods or intake of highly manipulated meals (Bessesen 2001). Furthermore, other factors such as macro-nutrient and fiber composition, cooking methods, chewing and intrinsic differences in carbohydrate digestion and absorption may affect the rate of appearance of glucose in the blood following a meal. Recently, Ludwig (2000) suggested that the intake of high GI foods may contribute to the increased rate of obesity. This hypothesis is strengthened by the effects of elevated blood glucose and insulin levels on increased insulin resistance in animals (Bynes et al. 1995) and humans (Frost et al 1998, Bessesen 2001) and decreased appetite control (Ludwig 2000) and fat oxidation (Jeukendrup et al. 1998) in humans.

Most of the effects of GI on fat oxidation are from studies utilizing single foods fed prior to exercise (Kirwan et al. 2001, Febbrario et al. 2000). We tested the hypothesis that a lower GI diet could be constructed from published data and could be fed in a mixed diet to young men to lower fasting and post-prandial blood glucose, insulin and enhance fat oxidation rates at rest and during 1 h of moderate exercise.

Subjects and Methods

Subjects

Twelve young healthy male volunteers aged 22 ± 3 y (mean ± SD) with normal body mass indexes (23.9 ± 2.5 kg/m^2, mean ± SD) were recruited from the undergraduate and graduate student body at Iowa State University. Ten subjects were non-Hispanic Caucasians, one was Hispanic and one was Asian. All subjects signed a consent form prior to the study. The Iowa State University Human Subjects Review Committee approved the study protocol.

Methods

Subjects were randomly assigned to one of three cohorts (n = 4 per cohort). Prior to starting the experiment the maximal aerobic capacity ($VO_{2\,max}$) of each subject was measured at 0700 h under fasting conditions. Baseline carbohydrate oxidation (CHOX) and fat oxidation (FOX) were measured during rest and 1-h moderate exercise (60% $VO_{2\,max}$) after the subjects consumed their habitual diet for the first week (Monday through Thursday). For two successive weeks the lower and higher GI diets were fed (Monday through Thursday) to each cohort using a cross-over design with a three day habitual diet washout period (Friday through Sunday). The GI diets were fed in a random order to each cohort. Subjects recorded their habitual and experimental diet intakes using the 2D Food Portion Visual (Miller and Morgan, 1996). Actual intakes of the experimental diets were directly weighed by the investigators-unknown to the subjects. Fasting blood glucose was measured at 0700 h from Monday to Thursday during the lower and higher diet periods. On Thursday evenings blood samples were drawn from the antecubital vein prior to dinner (18:00 PM) and every 30 minutes after dinner was completed. The evening meal was consumed within about 30 minutes.

Maximal aerobic capacity ($VO_{2\,max}$) and indirect calorimetry measurements

Subjects were asked to do exercise on an electronically braked cycle ergometer (Lode Excalibur) under fasting conditions at 0600 h on Friday morning prior to starting the experiment. Subjects maintained pedal cadence at 70 rpm during the exercise. After a four-minute warm-up period, workload was increased by 40 watts every three min until subjects could not maintain the required pedal cadence. Expired air was directed through a breathing

valve into the Physiodyne Max I metabolic cart (Physio-Dyne Instrument Corporation, Quogue, NY) for measurement of VO_2 and VCO_2. Expired air was sampled on a breath by breath basis and averaged over 1 min.

On Friday mornings after each diet CHOX and FOX were measured during rest and 1-h moderate exercise (60% $VO_{2\ max}$) by indirect calorimetry. One-minute expired air samples were collected every 20 min during the exercise period. CHOX and FOX were calculated from VO_2 and VCO_2 values obtained at rest, adapt 20, 40 and 60 minutes of moderate exercise (ZuntzN 1901)

Diets

We identified foods with lower and higher GI from original publications in which the reported GI's were relative to white bread, measured for at least two hours, and repeated at least twice. We purchased similar foods in a grocery store. The one exception was the use of low GI Sustagrain™ Barley provided by ConAgra (Omaha, Nebraska). Lower and higher GI diets were constructed by exchanging high and low GI versions of breads, cereals, vegetables, fruit and snacks (Table 1). Both GI diets contained the same meats, condiments, fluids, some fruits and vegetables. The Nutritionist V program (N-Squared Computing, Salem, OR) was used to design the low and high GI diets to provide as much as possible similar energy and macro-nutrient composition (Table 2).

The low and higher GI diets included breakfast, morning snack, lunch, afternoon snack, dinner, and evening snack. Lunch and dinner meals were different each day and were randomly assigned to Monday, Tuesday, etc. Breakfast and snacks were fairly constant on each day. Carbohydrates in higher GI diet consisted of high GI carbohydrates mainly and

carbohydrates in low GI diet consisted of low GI carbohydrates mainly. The actual foods that comprised the lower and higher GI diets are summarized in Appendix 1.

Analytical methods

Fasting blood glucose concentrations were measured with a portable blood glucose monitor system (OneTouch® Basic®, LifeScan Company, Milpitas, CA). Blood samples were collected into EDTA tubes and immediately centrifuged at 4° C (1200×g, 15 min). Plasma was stored at -80°C until analyzed. Plasma was assayed for glucose by using the GL-2500 Glucose Lactate Analyzer (Yellow Springs Instrument Co, Yellow Springs, OH). Plasma insulin was determined by using ^{125}I radioimmunoassay kit (Diagnostic Products Company, Inc, Los Angeles, CA). Blood fatty acids were measured by the free fatty acid kit (Wako Diagnostics, Wako Chemicals USA, Inc., Richmond, VA). Plasma triglyceride concentrations were measured by using the triglyceride kit (Sigma Diagnostics, Sigma-Aldrich Corp., St. Louis, MO).

Statistical analyses

The effects of the habitual diet and the lower and higher GI diets on blood components, weight and macro-nutrient intakes and oxidation rates were analyzed by repeated- measures analysis of variance (ANOVA, Systat Inc., Evanston, IL). Some analyses were analyzed using data at 20m 40 and 60 min of exercise and the 0 time point was a covariate. Diet and time were within-subject factors. In some analyses, subjects were separated into higher fat and lower fat oxidizers and these groups were analyzed with group as between subject factor and diet and time as within-subject factors in repeated measures ANOVA analysis. Whenever there was a significant effect as determined by the repeated-measures ANOVA, we used the independent t test to examine between-group differences.

One subject hyperventilated during one exercise period and CHOX was extremely high and no fat oxidation was measured (Jequier et al. 1987). These data were omitted from the analyses. Significance was set at α =0.05 level, but differences with $P \leq 0.1$ are also reported to better balance Type I and Type II statistical errors. Data are presented as means and standard deviations.

Results

Energy, macro-nutrient and fiber intakes on the habitual and lower and higher GI mixed diets

The lower and higher GI mixed diets provided during the four day diet period similar energy and macro-nutrient intakes, but the lower GI diet provided significantly more fiber ($P < 0.05$) and to a lesser extend less sugar ($P < 0.05$) than the higher GI diet (Table 2). Estimations of the subjects habitual intakes from self-reported four-day food records were significantly lower compared with the GI mixed diets for energy and fiber ($P < 0.05$). The reported lower energy intake was entirely due to reported low intake of carbohydrate including sugar in self-reported four-day food record. Subjects reported eating very little fruit whereas the higher and lower GI diets contained 3-6 servings of fruit per day. The subjects self-reported approximately 10% less intake than the weighed intakes for both the lower and the higher GI diets ($P < 0.05$) (data not presented).

Fasting and post-prandial measurements

Within 24 h (Tuesday or day 2) of eating either GI mixed diet, fasting blood glucose declined by 7% ($P < 0.05$) from the baseline blood glucose concentrations measured at the

end of a habitual diet period (Monday or day 1), including after the 3 day weekend washout period between the two GI diets (4.5 vs. 4.2 and 4.2 mmol/L for habitual, low and higher GI diets respectively). On the last day (Thursday or day 4) of the diet period the GI mixed diets did not influence glucose and insulin concentrations measured at 1800 h (immediately prior to the evening meal and 3 h since the afternoon snack) (Figure 1a and 1b). Post-prandial glucose (P=0.006) and insulin concentrations (P=0.001) were significantly lowered after eating the lower GI meal than after the higher GI meal.

On the last day of the diet period, plasma fatty acids concentrations were higher immediately prior to the evening meal and within 1h after eating lower GI meal than when eating the higher GI meal (Figure 2). Plasma fatty acids declined after eating the lower GI meal but did not change after the higher GI meal (diet by time interaction, $P < 0.05$). Pre-meal and post-prandial triacylglycerol concentrations were not affected by the GI diets (data not presented).

Diet effects on fat and carbohydrate oxidation at rest and during moderate cycling exercise

There were no differences in subject's CHOX and FOX rates measured at rest after an overnight fast following 4 d of eating their habitual diets or the higher or the lower GI diets (Figure 3a and b). During the first 20 min of exercise CHOX increased ($P < 0.05$, main time effect) providing most of the energy expended. The CHOX rate peaked at 20 min then declined slightly. At 60 min of exercise CHOX was significantly lower ($P < 0.05$) following the lower GI diet compared with the habitual and higher GI diet (Figure 3a). FOX rate progressively increased throughout the 60 min of exercise ($P < 0.05$, main time effect). The increase of FOX rate after 40 min of exercise tended to be greater ($P < 0.09$, time by diet

interaction) after the lower GI diet than either the habitual or higher GI diet such that at 60 min of exercise FOX was 20% greater ($P < 0.05$) on the lower GI diet than the habitual and higher GI diets.

Weight

There was a small (-0.70 kg) but statistically significant decline ($P<0.05$) in body weight within 5 days after consuming the lower GI diet, whereas weight did not significantly change when subjects consumed the higher GI diet (+ 0.11 kg).

Higher and lower fat oxidizers

Resting FOX rate was positively associated ($P < 0.05$, covariant analysis $n = 12$) with the FOX rate during exercise, whereas, VO_{2max}, weight and BMI were not associated with either resting or exercise FOX. Subjects were arbitrarily separated into higher and lower fat oxidizer groups according to their average resting FOX rates at rest. The difference in FOX rates between the two groups was maintained and expanded ($P < 0.05$) throughout the exercise period (Figure 4). Subjects with the higher FOX rates not only had lower ($P < 0.05$) post-prandial plasma glucose and insulin concentrations (Figure 5) but also had higher ($P < 0.05$) post-prandial plasma fatty acid concentrations (Figure 6) than subjects with lower FOX rates. Resting fat oxidation status did not change after the higher and lower GI diets when compared with the habitual diet. There was not diet by fat oxidizer group effect on any measured parameter. Although the higher fat oxidizers reported consuming more fat on their habitual diet (92.25 ± 13 vs. 69.55 ± 15 g, $P < 0.05$), fat intake between the groups, by experimental design was the same.

Discussion

This study demonstrates that replacing commonly consumed high GI foods featured in the USDA food guide pyramid with lower GI alternatives as part of a nutritionally balanced diet over a four day period significantly increased FOX, albeit, during what must be construed as endurance exercise. Previous studies established that pre-exercise intake of single foods with low GI alone (Thomas et al. 1991) or as part of a non-balanced (i.e. 3% energy from fat) meal (Wee et al. 1999) increased FOX. The current experiment shows that FOX during exercise can be enhanced by eating low GI foods as part of a daily diet with up to 13 h separating the last low GI meal and the start of exercise.

Effect of GI diets on macronutrient oxidation during exercise

Adding lower GI foods to a mixed diet was hypothesized to increase FOX at rest and during exercise. FOX was not increased at rest, but was increased modestly after 40 to 60 minutes of exercise. The changes in both CHOX and FOX with exercise duration in this study are similar to those reported for untrained subjects at 40% VO_{2max} (Kiens et al. 1993). Subjects indicated, while lifting weights, their VO_2max measured was not consistent with endurance trained individuals. The increased FOX with duration of exercise after 4 days of eating a lower GI diet is similar to increased fat oxidation, inferred from respiratory exchange ratio, during 80 min of exercise at 70%VO_{2max} 3 h after a low GI meal with 30% of energy as protein and 3% as fat (Wee et al. 1999). However, in both experiments with moderate and intense exercise FOX was less than CHOX. The ability of lower GI foods to increase FOX during less intense physical activity has to our knowledge not been reported.

The current study did not address the mechanisms by which higher or the lower GI diets influence fat oxidation. Elevated plasma fatty acids during fasting state, which are

likely in excess of that needed to influence low oxidation rtes during rest (Rasmussen and Wolfe, 1999) could mask effects of dietary GI on FOX at rest. As FOX increases during continued exercise plasma fatty acid concentrations could affect FOX (Rasmussen and Wolfe, 1999). One possibility is that the lower GI diet could increase lipolysis resulting in increased plasma fatty acids which could affect FOX (Wolfe and Peters, 1987). Although small increases in blood glucose and insulin concentrations suppress lipolysis (Wolfe and Peters, 1987), the GI diets did not affect glucose concentrations but plasma fatty acids were increased prior to the evening dinner during the low GI diet period compared with the higher GI diet period. These data suggest that the lower GI diet increased fatty acid availability and, if present during exercise, elevated plasma fatty acids could have contributed to the enhanced FOX when subjects ate the lower GI diet.

Alternatively, FOX may be determined by glucose availability in exercising skeletal muscle (Wolfe, 1998) as glycogen content predicts FOX rates during exercise (Goedecke et al. 2000).. The high GI diet, eliciting a higher glycemic response than the lower GI diet (Figure 1), could promote more rapid glycogen synthesis (Coyle, 1995; Kiens and Richeter, 1996) which is evident within 24 h (Burke et al, 1993) than the lower GI diet. Lower glycogen stores following 4 days on the lower GI diet could have limited the supply of glucose during prolonged exercise contributing and this could contribute to the observed increase in FOX and decrease in CHOX.

Short term effects of the GI diets on blood glucose and insulin concentrations

Although replacing high GI foods with lower GI alternatives did not influence fasting plasma glucose levels, both high and low GI diets significantly lowered fasting glucose concentrations within 24 h after subjects switched from their habitual diet. It is likely that

higher amounts of fiber in the low and higher GI diets compared with the habitual diet (Table 2) contributed to this decrease in glucose levels during the transition from the habitual to experimental diets. High fiber foods lower postprandial glucose levels within a day of adding fiber to the diet (Liljeberg et al. 1999).

We did not directly measure the GI of any single food used in the lower and higher GI diets. However, the lower postprandial glucose and insulin concentrations following the last meal with low GI foods confirms that the foods chosen provide similar glycemic responses attributed to low GI diets (Morris and Zemel, 1999). The glycemic responses observed are not likely due to dietary fat (Bessesen, 2001) because dietary fat and saturated fat intakes were nearly identical between the two GI diets. These results help clarify the role of dietary carbohydrate and GI on glycemic responses in mixed diets. Differences in GI may, but not necessarily, disappear when carbohydrate foods are eaten as part of a normal meal (Wolever, 1990). The decreased postprandial blood glucose and insulin concentrations following the last meal of the 4 day diet period are consistent with the concept that the glycemic effect of mixed meals can be predicted from the constituent carbohydrate food's GI (Collier et al. 1986) and provides additional evidence supporting the use of GI in planning meals (Wolever et al. 1985).

Stable plasma glucose and mildly elevated insulin concentrations together with a drop in fatty acid levels within the 2h after the last low GI meal suggests that subjects exhibited greater insulin sensitivity when eating the lower GI diet than when eating the higher GI diet. Increased insulin sensitivity has been noted as a benefit of eating a low GI diet (Morris and Zemel, 1999). The difference in blood glucose and insulin between the low and high GI diets may reflect both the nutrient and GI differences of the last meal, but also the previous meal.

Liljeberg et al. (1999) showed that after a low GI breakfast the plasma glucose and insulin concentrations after the subsequent lunch were attenuated. Fiber slows the glucose digestion (Behall 1997) and the postprandial rise in glucose in diabetes (Bessesen, 2001) and our diets markedly differ in fiber content. Therefore it is important to note that the effects observed in the study cannot be solely attributed to GI but rather a high fiber diets with low GI foods.

Variability in FOX rates among subjects

We observed considerable variability in FOX rates similar to that reported by Goedecke et al. (2000). Others have shown that variability of FOX during exercise was associated with post-exercise FOX (Almeras et al. 1995). In our study resting FOX was strongly and positively associated with FOX during exercise. Resting FOX rates were a significant determinant of FOX at 50% peak power output in cyclists (Goedecke et al. 2000). The major determinates of FOX, indicated by the respiratory exchange ratio, were muscle glycogen content, training, proportion of type I muscle fibers, plasma fatty acids and lactate concentrations.

Low resting FOX rates could be a risk factor for weight gain (Ravussin and Gautier, 1999). In similar young adults, a higher fat intake was also associated with a lower resting respiratory quotient reflecting higher fat oxidation (Cooling and Blundell, 1998). Similarly, the higher fat oxidizer group reported considerably greater dietary fat intake than the lower fat oxidizer group. Such a relationship suggests a metabolic adaptation that would affect the weight promoting effects of a high fat diet (Cooling and Blundell, 1998). There was no difference in BMI between the high and low fat oxidizer groups. It is notable that the higher fat oxidizer group continued to have higher FOX rates than the low fat oxidizer group when fat intake was the same between each group during the feeding studies. The higher fat

oxidizer group accounted for the observed decrease in body weight when subjects ate the lower GI diet, although it is difficult to determine if the small weight loss was attributable to fat or lowered glycogen-water content.

Higher fat oxidizers also had much lower glycemic responses and insulin responses, irrespective of the type of diet, than the low fat oxidizers. This suggests inherent metabolic differences in carbohydrate metabolism may manifest in variable FOX rates. A study of 916 Caucasians and Pima Indians could only account for about 13% of the variability in the 24 average respiratory exchange quotient (Wee et al. 1999). Glycemic tolerance was found not to be associated with 24h respiratory quotient. Because of the known relationship between glucose metabolism, insulin metabolism, lipolysis and FOX, more research is needed to determine the genetic factors that link insulin and glucose metabolism with FOX.

Although the mechanism is not clear the current experiment demonstrates that incorporating low GI foods that are commonly available in a mixed diet can enhance the utilization of fat during exercise. Subjects in this study felt that the low GI diet was acceptable and healthy, and only reported mild flatulence. These results provide direct evidence that introducing low GI foods in a diet is one practical and reasonable approach to optimize fat oxidation in exercise programs for the prevention of weight gain and obesity (Ludwig, 2000).

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Table 1. Lower and higher GI food exchanges used to construct the experimental diets.

Higher GI	GI	Reference	Lower GI	GI	Reference
Bread and cereal					
White bread	100	Crapo et al. 1980	Pumpernickel bread	58	Otto and Niklas 1988
Rice/corn chex	127±5/118 ±11	Wolever et al. 1994	Barley flakes	39±6	Jenkins et al. 1988
Short grain white rice	83	Crapo et al. 1980	Long grain white rice	67±5	Wolever et al. 1986
			Spaghetti	38±4	Wolever et al. 1986
Vegetable					
Baked potato	134	Crapo et al. 1980	Yam	73	Jenkins et al. 1981
carrots	131	Jenkins et al. 1981	sweet corn	67±4	Wolever et al. 1994
Sweet peas	77	Kurup and Krishnamurthy 1992	Pinto beans	64±6	Wolever et al. 1987
Split peas soup	86±12	Wolever et al. 1994	Green beans	74±5	Wolever et al. 1987
Baked beans	80±8	Wolever et al. 1987	Lima beans	46±13	Wolever et al. 1994
Fruits					
Watermelon	103	Brand et al. 1995	Apple	56	Jenkins et al. 1981
Pineapple	94	Brand et al. 1995	Pear	58±7	Wolever et al. 1993
Orange juice	81±8	Gannon et al. 1986	Orange	61±11	Wolever et al. 1993
Raisins	91	Jenkins et al. 1981	Peach halves	43	Brand et al. 1995
Snacks					
Tortilla chips	106±8	Wolever et al. 1994	Yogurt	47	Jenkins et al. 1981
Rice cakes	117	Brand et al. 1992			
Vanilla wafers	106±9	Wolever et al. 1994			

Table 2. Energy, macro-nutrient, fiber and sugar intake[1] (means ± SD) of volunteers eating the lower and higher GI diets.

Diet	Total energy (kJ)	Carbohydrate (g)	Protein (g)	Fat (g)	Sugar (g)	Total fiber (g)
Habitual	41,027 ± 9,425[2]	1,271 ± 284[2]	479 ± 183	320 ± 101	410 ± 104[2]	76 ± 27[2]
High GI	51,221± 3,386	1858 ± 130	501 ± 45	333 ± 25	925 ± 32	106 ± 8.6
Low GI	51,807± 3,051	1864 ± 99	587 ± 43	332 ± 35	892 ± 41[3]	204 ± 10.7[3]

[1] The habitual diet data were calculated according to the subjects' four day food record. The high and low GI test diet data were recorded according to weighed food record. All the values were calculated using the Nutritionist V program.

[2] Different from lower and higher GI diets, $P < 0.05$.

[3] Different from the High GI diet, $P < 0.05$

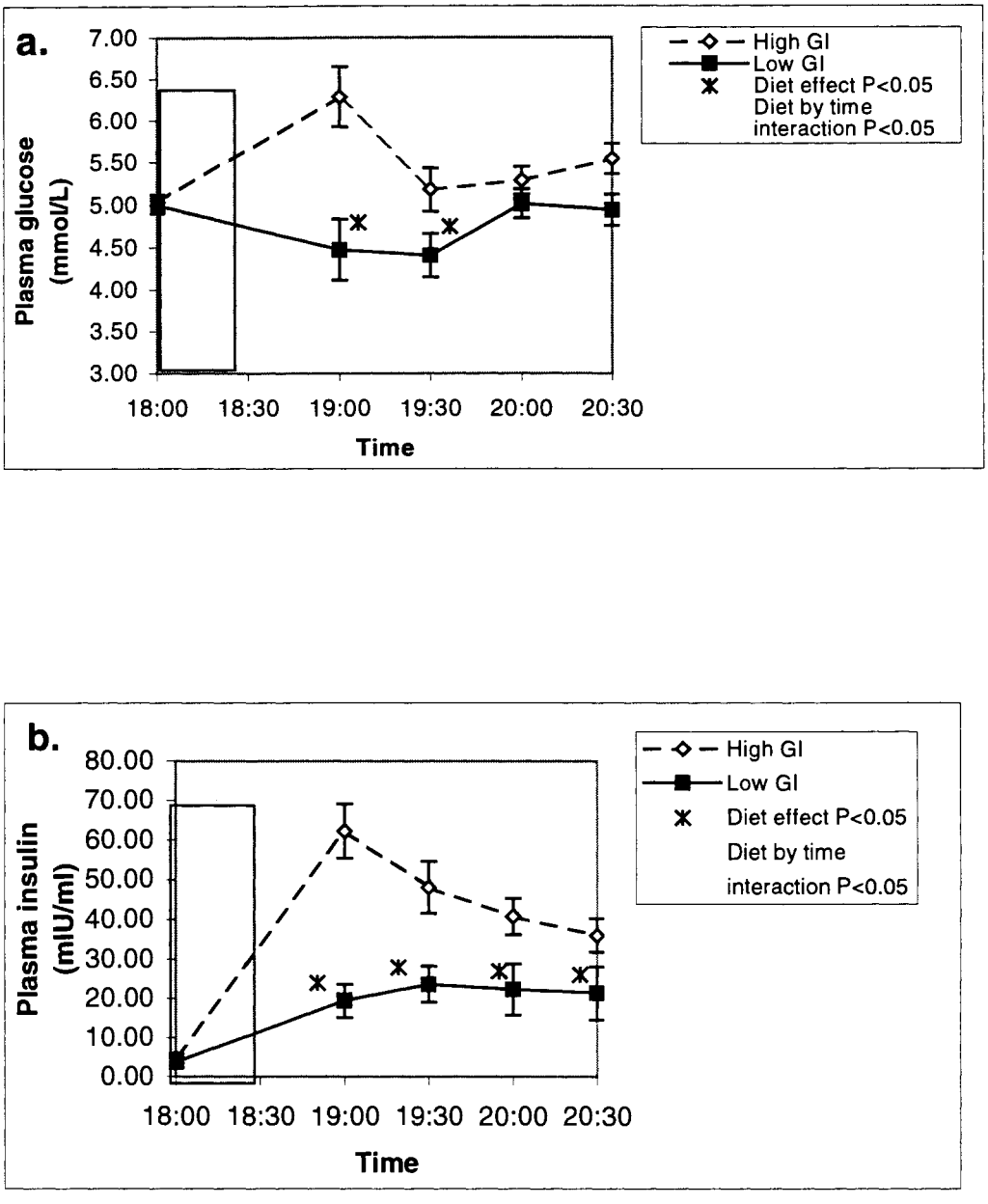


Figure 1. Post-prandial plasma glucose (a) (mmol/L) and insulin (b) (mIU/ml) responses after the higher and lower GI dinners (mean ± SD). Shaded area represents the time it took to consume the dinner. Lower GI diet decreased glucose levels (within subject's diet effect and diet by time interaction, $P < 0.05$). * Indicates individual values different ($P < 0.05$) from higher GI diet value ($P < 0.05$).

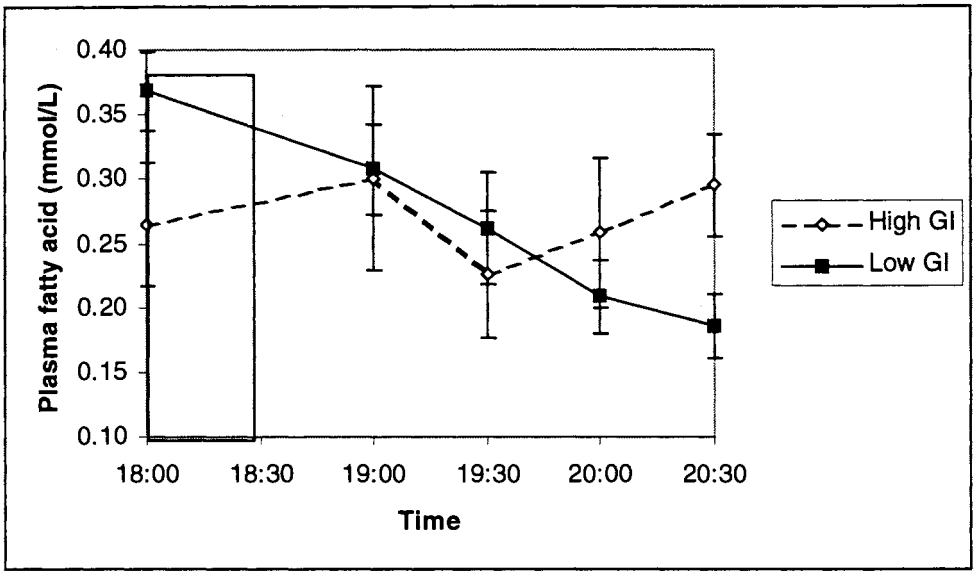


Figure 2. Post-prandial plasma fatty acid concentrations after the higher and lower GI diets (means ± SD). Shaded area represents the time it took to eat the dinner. * Indicates different from the higher GI diet ($P < 0.05$).

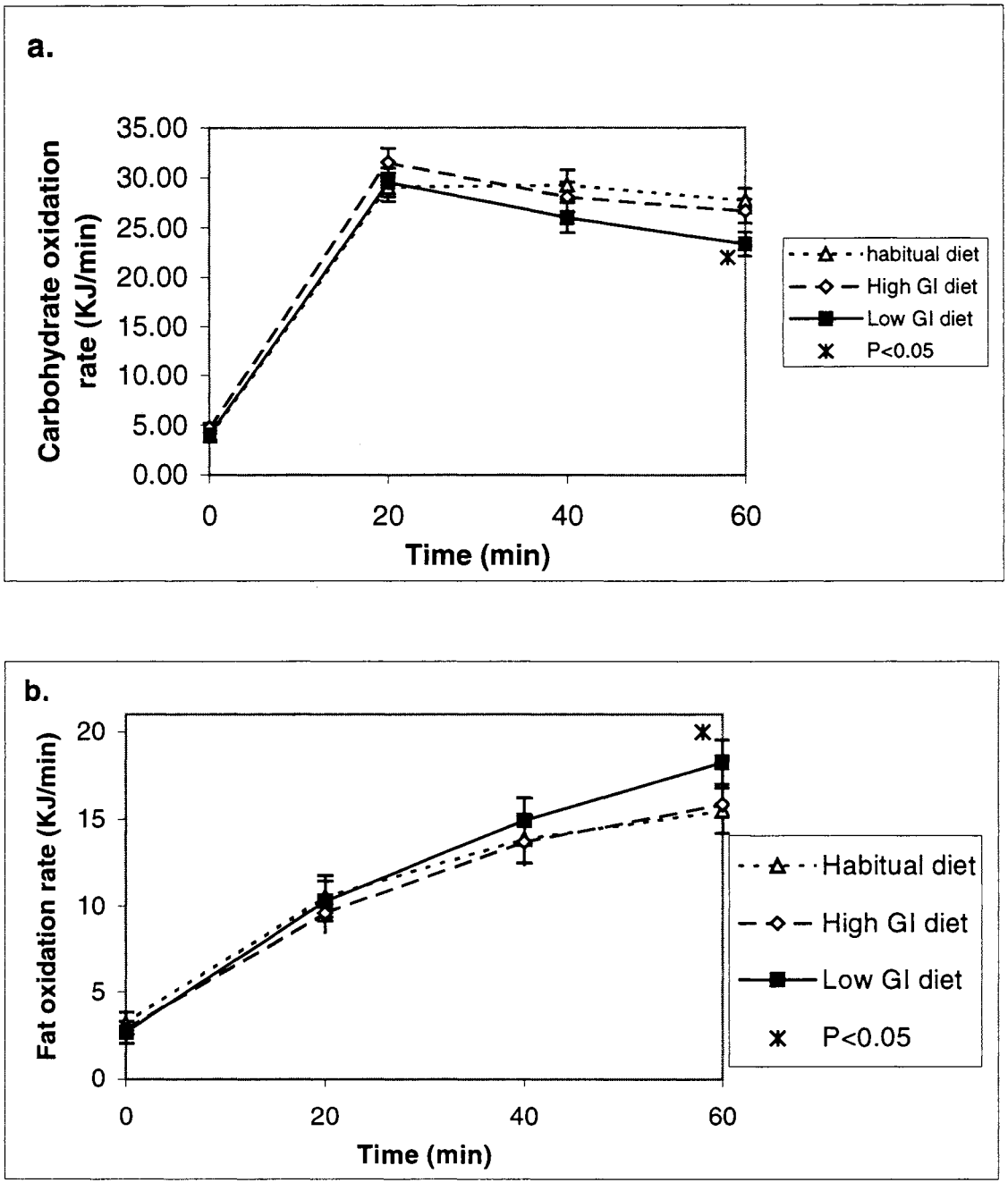


Figure 3. Mean carbohydrate (a) and fat (b) oxidation rates (kJ/min) at rest (time = 0) and during one-hour of exercise after eating habitual diet (dashed triangles) and the higher (dashed open circles) and lower GI diets (solid closed circles) (mean ± SD). * Indicates values significantly different from the higher and habitual diet values ($P < 0.05$).

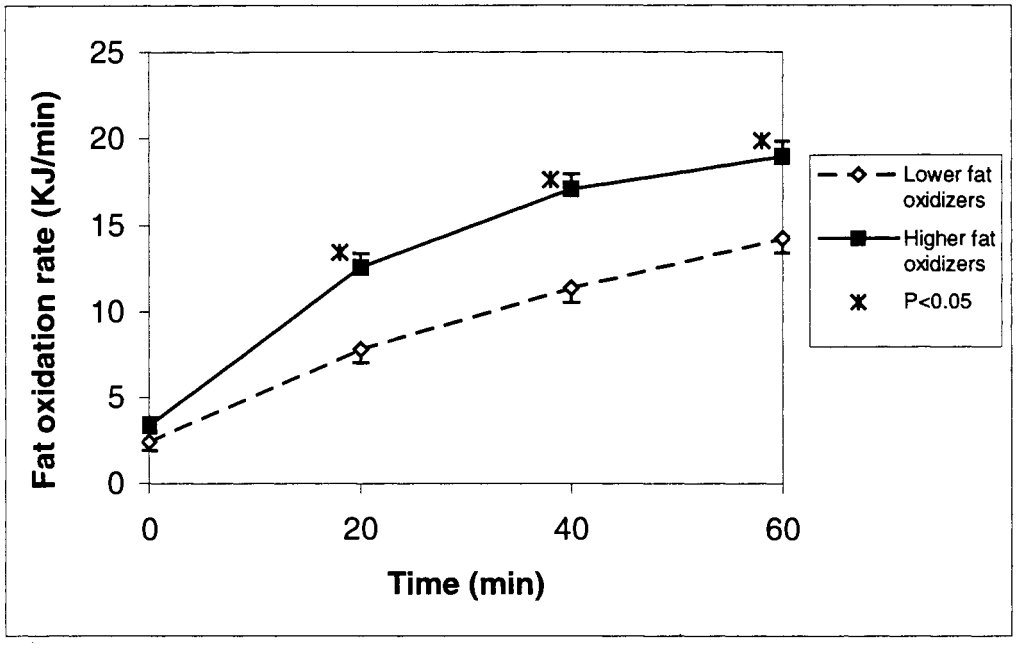
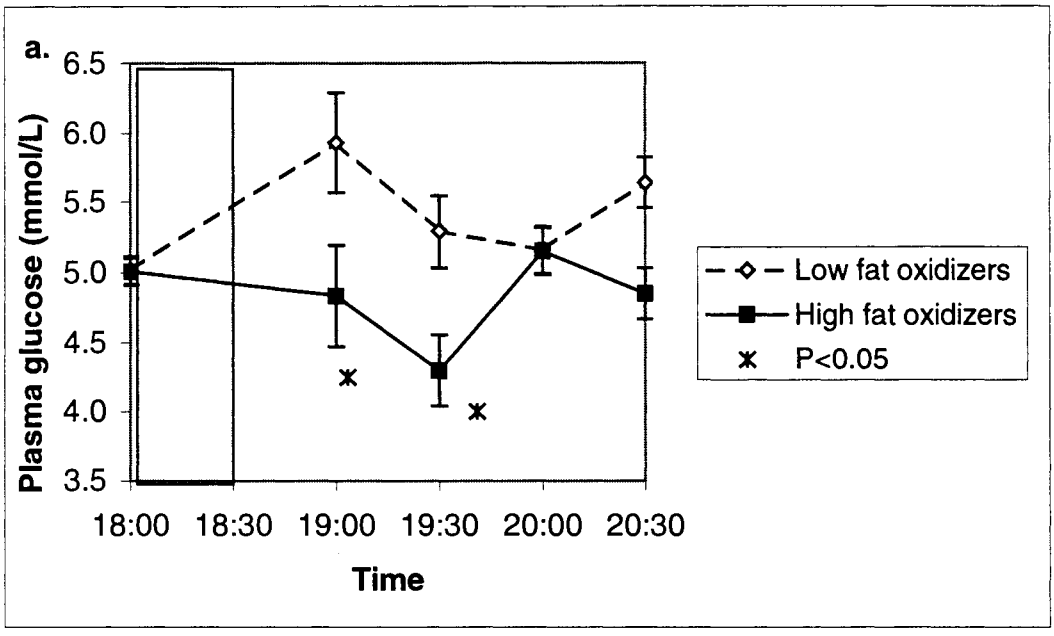


Figure 4. Mean fat oxidation rate of higher and lower fat oxidizers at rest and during exercise after higher GI and lower GI diets (fat oxidizer group, time and oxidizer by time interaction effects, $P < 0.05$).

* Indicates single data point for higher fat oxidizer group is different from the lower fat oxidizer group ($P < 0.05$).



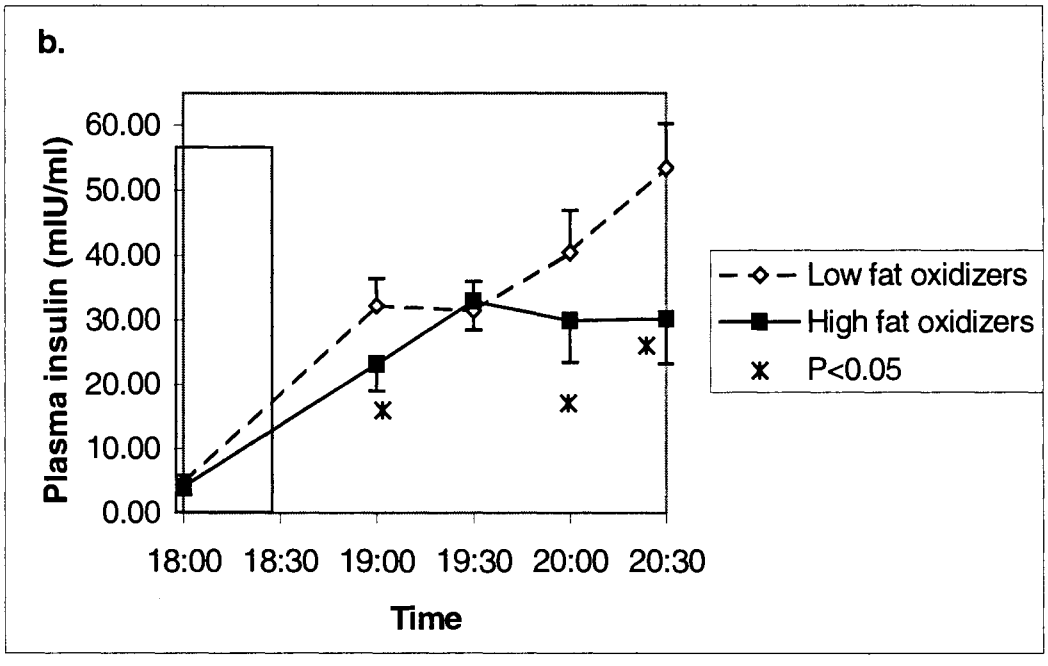


Figure 5. Post-prandial plasma glucose (a) and insulin (b) concentrations of higher and lower fat oxidizers after the higher and lower GI diets (combined). Shaded area represents the time it took to consume the evening meal. Higher fat oxidizer group was different from lower fat oxidizer group (oxidizer group effect, within subject time and time by oxidizer group effect, $P < 0.05$). * Indicates single data point for higher fat oxidizer group is different from the lower fat oxidizer group ($P < 0.05$).

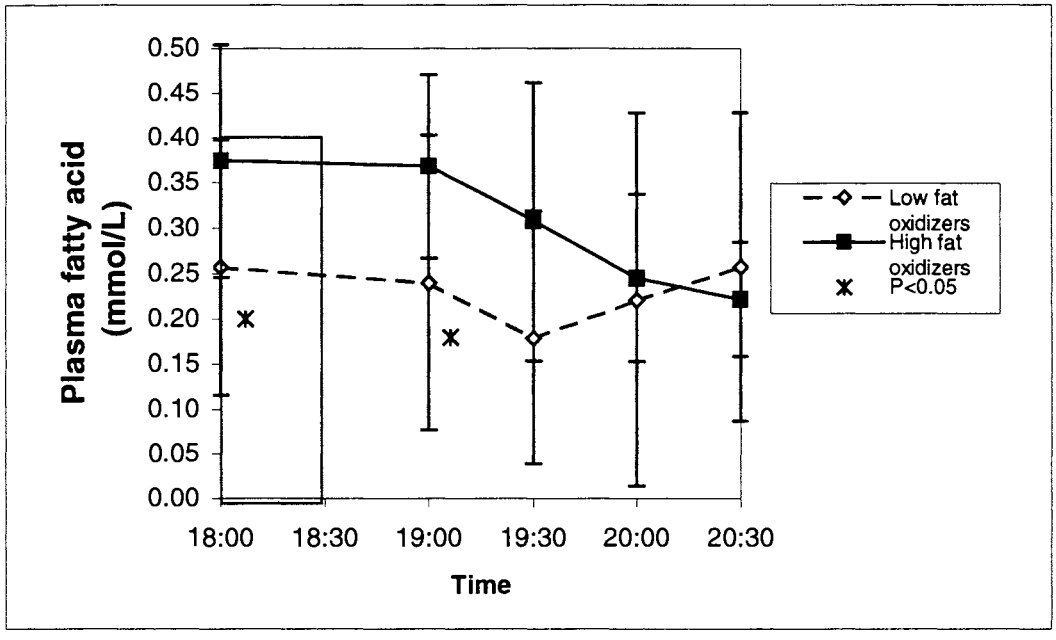


Figure 6. Plasma fatty acids in higher and lower fat oxidizers. Shaded area represents time to consume dinner. Higher fat oxidizer group (solid line and symbol) was different from lower fat oxidizer group (oxidizer group by time interaction, $P < 0.05$). * Indicates single data point for higher fat oxidizer group is different from the lower fat oxidizer group ($P < 0.05$).

SUMMARY AND FUTURE WORK

Two hypotheses were tested in this experiment: The first hypothesis was that a mixed diet containing lower GI foods would lower blood glucose and insulin levels and increase fat oxidation (FOX) compared with a diet consisting of higher GI foods. The second hypothesis was that variability in resting FOX would influence FOX during moderate exercise after consuming the lower GI and higher diets.

We designed two sets of diets including high and low GI foods, and these two diets were as similar as people's ordinary diets. When we designed the test meals for this study, we tried to make the change from high GI diet to low GI diet easy and convenient. A reasonable way is to build a glycemic index database having as many foods as possible, then select and separate common foods into high and low GI groups, since lots of studies have been done to measure the glycemic index of carbohydrate foods. The amount of carbohydrate foods can be modified so that high and low GI diets may have similar nutrients. The same amount of meat and fat can be added to both diets. A problem happened naturally is that all kinds of beans always supply large amount of protein for the low GI diet, which is hard to be balanced by the high GI carbohydrate foods in the high GI diet. Since the objective of this study is to compare the effects of high and low GI diets and to give useful suggestions for people to choose their food everyday, the possible problem caused by changing the carbohydrate foods in the normal life should be tested. So we left the diets as natural as possible, as if people change the carbohydrate food in their diets themselves and maintain their other dietary habits. Finally we made two sets of diets, one set of diet consisted of more

low GI carbohydrate foods and the other set consisted of more high GI carbohydrate foods. For the balance of nutrients, high GI diet may have some low GI foods and low GI diet may have some foods not having very low GI. When we analyzed these two diets by using the Nutritionist V program, we found that the designed low GI diet naturally had more total fiber content than high GI diet. The fiber in the low GI diet was two folds more than the fiber in the high GI diet. And the low GI diet tended to have less sugar than the high GI diet. Another difference is that the low GI diet had higher total energy than the high GI diet.

These two sets of diets, four days each, were fed to 12 young male college students. Beneficial metabolic responses were observed after the low GI diet. So it is feasible to design much healthier diet for healthy individuals by applying the GI concept to choose carbohydrate foods. We measured the baseline plasma glucose and the body weight every morning before the subjects had breakfast. The body weight of subjects decreased after they consumed the low GI diet for only four days, though they consumed more calories than the calories they consumed on the high GI diet. However, we can not verify the body weight reduction effect only by this experiment. The study time was not long enough to observe the body weight change to diets and the body weight change was not a large amount so that it might be caused by the body fluid volume change.

During exercise CHOX was significantly lower following the lower GI diet compared with the habitual and higher GI diet. FOX rate progressively increased during the 60 min of exercise. The increase of FOX rate after 40 min of exercise became larger after the lower GI diet than either the habitual or higher GI diet and at 60 min of exercise FOX was 20% greater on the lower GI diet than the habitual and higher GI diets.

Subjects were separated into higher and lower fat oxidizer groups according to their average resting FOX rates at rest. The difference in FOX rates between the two groups was maintained and expanded ($P < 0.05$) throughout the exercise period. Subjects with the higher FOX rates not only had lower ($P < 0.05$) post-prandial plasma glucose and insulin concentrations but also had higher ($P < 0.05$) post-prandial plasma fatty acid concentrations than subjects with lower FOX rates. There was no association between the diet type and resting FOX.

However, this study still had limitations. The study time was not long enough to observe long term effects of low GI diet on body metabolism. Body adaptation to low GI diet may develop after consuming low GI diet for a longer time. The subjects chosen in this study were healthy. It is still not known whether low GI diet would be effective to increase FOX in patients having metabolism related diseases. More studies should be done in those patients to observe the effect of low GI diet. We did not measure body composition of subjects in this study. Though increased FOX during exercise was observed after low GI diet, accurate body composition measurement could provide extra evidence to prove the FOX increasing effect of low GI diet. Another limitation of this study is that we did not observe metabolic change in plasma and muscle during the exercise. We did not know the effect of low GI diet on plasma glucose, insulin, free fatty acids, triacylglycerol during exercise. We also did not know the muscle composition change after low GI diet, which may supply some information for us to learn the mechanism of FOX increasing effect after low GI diet.

Though the Nutritionist V program supplied complete nutrient information for both test diets in present study, the lab analysis of some important nutrients that may influence the glycemic index of foods and the analysis of some nutrients having significantly different

amount in two diets should be helpful. The food amount was measured during the experiment and the nutrient amount was calculated by using the Nutritionist V program according to the food serving record made during the experiment, but we still need the lab analysis result to verify and compare the exact nutrient amount in both diets. The amylose content, soluble fiber content, sugar content and the existence of antinutrient were all proposed as the factors that may affect the food glycemic index (1, 2, 3). The Nutritionist V program does not supply the information about the amylose content of the diets, so it is valuable to measure and compare the amylose content in the two diets. The soluble fiber content and sugar content are still need to be verified by the lab analysis result. The lab analysis result may compare the nutrient difference between high and low GI diets and testify the hypothesis about the factors affecting the food glycemic index. The food analysis result may also contribute to the mechanism explanation of the responses to different GI diets.

In future studies, it will be very necessary to test low GI diet in overweight people. Obesity is the most common chronic disorder in the United States, affecting one third of the adult population. Health risks associated with overweight include hypertension, diabetes, cardiovascular disease, cancer, endocrine abnormalities, gall bladder disease, respiratory problems and arthritis. If low GI diet can increase FOX in overweight people, it will be very helpful for people to control body weight and lose weight.

It is also very helpful to determine the body composition of subjects in future studies. By measuring the body composition of subjects before eating low GI diet and after eating low GI diet, we may know the fat storage change, body fluid change of subjects during experiments. Body composition can explain why body weight change during experiments. If

body composition indicate that fat storage decreases after consuming low GI diet, it will be reasonable to propose that low GI diet may make people lose weight.

This experiment observed lower post-prandial plasma glucose and insulin levels on the low GI diet, higher plasma free fatty acid levels before meal on the low GI diet and higher fat oxidation and lower carbohydrate oxidation after 1 hour exercise after the low GI diet in healthy subjects. The lower post-prandial plasma glucose and insulin level may be explained as the result of slow digestion and absorption after consuming the low GI meal. But the mechanism of plasma FFA increase before meal is still unclear. Decreased insulin secretion during the day (4) may reduce the fat storage and increase fat utilization, which may increase the lipolysis and increase the plasma FFA concentration. Decreased plasma glucose level also indicated lower inhibitory effect of glucose on lipolysis via insulin activity (5). But we can not exclude other possibilities, for fat storage and utilization are regulated by many other factors. The intracellular hormone-sensitive lipase, which is responsible for the breakdown of stored triglycerides in adipose tissue, may be activated by protein kinase A. Increased intracellular CAMP concentration activates protein kinase A. Glucagon, the catecholamines, ACTH, TSH, LH, serotonin and vasopressin may activate the adenylyl cyclase and thus increase intracellular CAMP level, so they may also increase lipolysis (6). Growth hormone, glucocorticoids and thyroid hormones may also increase lipolysis via the synthesis of new protein (6). Studies need to be done to observe the effects of low GI diets on the secretion of those hormones that are also responsible for the increase of lipolysis. And low GI diet has also been related to the decreased insulin level and the increased serotonin level after meal (7). Prostaglandin E may also decrease the activity of the hormone-sensitive

lipase (6), so it is also necessary to study whether low GI diet has any relationship with the prostaglandin E level.

Consuming the low GI diet for four days increased fat oxidation rate after 1-hour exercise. In present study, we did not study the mechanism of how low GI diet increased FOX during exercise. We knew that low GI diet would not affect resting FOX and it increased FOX during exercise from present experiment. We observed increased subject plasma FFA concentrations on the low GI diet before meal, which may partly explain the increased FFA utilization during exercise. The reason for this is that high plasma FFA concentration indicates high intracellular FFA concentration, high concentration of fatty acyl-CoA in cytosol, and more fatty acyl-CoA transferred into the mitochondria, since the FFA was assumed to pass cell membrane and mitochondria membrane via passive diffusion. Though fatty acid binding protein, fatty acid translocase and fatty acid transport protein have been identified recently (8, 9, 10), which therefore highly suggested that FFA transport may via carrier-mediated process and may be saturable, the utilization of FFA may still increase with the increase of plasma FFA concentration at the lower concentrations before saturation. Romijn et al. (11) found that fat oxidation is normally impaired during exercise at 85% VO_2max, which is due to the low plasma FFA concentration during strenuous exercise. And they also found that impaired fat oxidation can be increased by intravenous infusion of lipid (Intralipid) and heparin to maintain the plasma FFA concentration between 1 and 2 mM. But the increased fat oxidation during strenuous exercise was still lower than the fat oxidation during moderate exercise (65% VO_{2max}). These results indicated that fat oxidation is regulated not only by the plasma FFA concentration, but also by other factors. Low GI diet would not affect resting FOX and it increased FOX during exercise, this phenomenon may be

explained partially by hormone change and plasma change, but we also speculate that low GI diet changed muscle composition. Muscle autopsy before and during exercise in future studies would be useful to verify our speculation.

In rats, skeletal muscle malonyl-CoA concentration has been found having negative relationship with fatty acid oxidation in the muscle in experiment (12), though this relationship has not been observed in human studies. The hypothesis is that the muscle malonyl-CoA higher than certain concentration may inhibit the carnitine palmitoyltransferase-1, and regulates the fat oxidation by regulating the uptake of long-chain fatty acid into the mitochondria (13). And muscle malonyl-CoA content is controlled by acetyl-CoA carboxylase, which is activated by citrate and inhibited by palmitoyl-CoA (14, 15, and 16). So malonyl-CoA content in the skeletal muscle is also related to glucose uptake. However, this hypothesis needs to be testified in human subjects.

The fact that some subjects naturally had significantly different macro-nutrient oxidation during exercise than other subjects can only be explained by individually difference. People who having higher fat oxidation rate during exercise had lower post-prandial glycemic response and higher pre-meal plasma fatty acid concentration may have higher lipolysis and lower plasma glucose and insulin levels during the day when low GI diet is consumed. And only subjects having higher fat oxidation rate during exercise had obvious response to the low GI diet. To explain those different metabolic changes between higher fat oxidizers and the lower fat oxidizers, we need to observe some metabolic change during the exercise. The hormone level change during the exercise, including the insulin and the epinephrine change may supply some important clues for clarifying the mechanism. The concentration change of some critical substrates, such as the plasma and muscle free fatty

acids, the muscle malonyl-CoA, and the muscle triglyceride, may also supply some information of fuel utilization. Also the separation of the higher and the lower fat oxidizers may be used to predict the possible metabolic changes of the subjects. In the future studies, subjects may be grouped according to their baseline fat oxidation rate, except for the difference on the plasma glucose, insulin, free fatty acid concentration between the two groups, the difference between those two groups on the insulin sensitivities, the incidence of the diabetes, insulin resistance, and some other metabolism related diseases may be expected. Also people having different baseline fat oxidation rate may have different exercise performance. So it will be very interesting to study whether the baseline fuel utilization rate of the athletes during the exercise may supply another evaluation rule to predict the exercise performance of the athletes after different diets having different glycemic index. But there is no exact value of fat oxidation rate alone or combined with the plasma fatty acid level and the glycemic response for predicting the response to the low GI diet on improving the fat oxidation in healthy subjects. Experiments may be designed to observe the response of subjects with different fat oxidation rates during exercise on low GI diet to determine the evaluation criteria.

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APPENDIX

Test Meals In the Experiment

Test meals on Monday

Meal	Foods (portion)	GI	High GI meal	Low GI meal	reference
Breakfast	White bread (slice)	100	2		1
	Pumpernickel bread (slice)	58		2	2
	RICE CHEX cereal (cup)	127±5	1		3
	Barley flakes (g)	39±6		27	4
	Banana, peeled (item)	83±7	1	1	5
	Skim milk (cup)	46	1.5	1.5	6
	Margarine (tsp)		1	1	
Morning	Rice cakes (g)	117	50		7
snack	1% fat yogurt (cup)	47		1	6
Lunch	White bread (slice)	100	2		1
	Pumpernickel bread (slice)	58		2	2
	Potatoes, flesh only, baked (g)	134	130		8
	Sweet corn (cup)	67±4		0.5	3
	Carrots (cup)	131	0.5		6
	Tomato (slice)			4	
	1% fat yogurt (cup)	47	1		9
	Skim milk (cup)	46	1	1	6
	Grilled Chicken Breast (g)		84	84	
	Margarine (tsp)		1		
	Swiss Cheese (g)			21	
Afternoon	Tortilla chips (g)	106±8	50		3
snack	Apple (item)	56		1	6
Dinner	White bread (slice)	100	1		1
	Pumpernickel bread (slice)	58		1	2
	Spaghetti (cup)	38±4		1	10
	Carrots (cup)	131	0.5		6
	Pinto Beans (cup)	64±6		0.5	11
	Sweet peas (cup)	77	1		12
	Green Beans (cup)	74±5		0.5	11
	Watermelon (cup)	103	2.5		9
	Orange (item)	69±11		1	13
	Skim milk (cup)	46	1	1	6
	Vanilla ice cream (cup)		1	1	
	Beef steak (g)		75	75	
	Margarine (tsp)		1	1	
	American cheese (g)			19	
Evening	Kiwi fruit (item)	83	1	1	9
snack	Orange (item)	69±11		1	13

Test meals on Tuesday

Meal	Foods (portion)	GI	High GI meal	Low GI meal	reference
Breakfast	White bread (slice)	100	2		1
	Pumpernickel bread (slice)	58		2	2
	Corn chex cereal (cup)	118±11	1		3
	Barley flakes (g)	39±6		27	4
	Banana, peeled (item)	83±7	1		5
	Orange juice (cup)	81±8	1		14
	Orange (item)	69±11		1	13
	Skim milk (cup)	46	1.5	1.5	6
	Margarine (tsp)		1	1	
Morning	Vanilla wafers (item)	106±9	10		3
Snack	1% fat yogurt (cup)	47		1	9
Lunch	White bread (slice)	100	2		1
	Pumpernickel bread (slice)	58		2	2
	Carrots (cup)	131	0.3		6
	Pinto Beans (cup)	64±6		0.5	11
	Pineapple (g)	94	115		9
	Apple (item)	56		1	6
	1% fat yogurt (cup)	47	1		9
	Skim milk (cup)	46		1	6
	Corned beef (g)	70	70		
	Miracle whip salad dressing (tsp)		1	1	
	Swiss Cheese (g)			21	
Afternoon	Fritos(g)	106±8	50		3
Snack	Pear (item)	58±7		1	13
Dinner	White bread (slice)	100	2		1
	Pumpernickel bread (slice)	58		2	2
	Potatoes, flesh only, baked (g)	134	130		8
	Yams (g)	73		140	6
	Baked beans (cup)	80±8	0.5		11
	Green Beans (cup)	74±5		0.5	11
	Watermelon (cup)	103	2.5		9
	Peach halves (cup)	43		1	9
	Skim milk (cup)	46	1	1	6
	Vanilla ice cream (cup)		1	1	
	Margarine (tsp)		2		
	Swiss Cheese (g)			21	
Evening	White bread (slice)	100	2		1
Snack	Pumpernickel bread (slice)	58		2	2
	Raisins (tbsp)	91	4		6
	Peanut butter (tsp)		3	3	

Test meals on Wednesday

Meal	Foods (portion)	GI	High GI meal	Low GI meal	reference
Breakfast	White bread (slice)	100	2		1
	Pumpernickel bread (slice)	58		2	2
	Corn chex cereal (cup)	118±11	1		3
	Barley flakes (g)	39±6		27	4
	Pineapple (g)	94	115		9
	Pear (item)	58±7		1	13
	Orange juice (cup)	81±8	1	1	14
	Skim milk (cup)	46	1.5	1.5	6
	Peanut butter (tsp)		2	2	
Morning	Rice cakes (g)	117	50		7
snack	1% fat yogurt (cup)	47		1	9
Lunch	White bread (slice)	100	1		1
	Pumpernickel bread (slice)	58		2	2
	French fries (item)	107±6	26		3
	Split peas soup	86±12	0.5		3
	Green Beans (cup)	74±5		0.5	11
	Lettuce leaves (piece)		2	2	
	Skim milk (cup)	46	1	1	6
	Turkey breast (g)		84	84	
	American cheese (g)		19	19	
Afternoon	Potato chips (g)	81	50		15
snack	Apple (item)	56		1	6
Dinner	White bread (slice)	100	2		1
	Pumpernickel bread (slice)	58		1	2
	Long grain white rice (cup)	67±5		1	16
	Carrots (cup)	131	0.5		6
	Pinto Beans (cup)	64±6		0.5	11
	Watermelon (cup)	103	2.5		9
	Apple (item)	56		1	6
	Skim milk (cup)	46	1	1	6
	1% fat yogurt (cup)	47	1		9
	Pumpkin pie (g)	107*	131		17
	Vanilla ice cream (cup)		1.5		
	Pork chop (g)	100	100		
	Margarine (tsp)		2	2	
Evening	Oatmeal cookies (item)	79	3		9
Snack	Orange (item)	69±11		1	13

*GI for pumpkin

Test meals on Thursday

Meal	Foods (portion)	GI	High GI meal	Low GI meal	reference
Breakfast	White bread (slice)	100	2		1
	Pumpernickel bread (slice)	58		2	2
	RICE CHEX cereal (cup)	127±5	1		3
	Barley flakes (g)	39±6		27	4
	Banana, peeled (item)	83±7	1	1	5
	Orange juice (cup)	81±8	1		14
	Skim milk (cup)	46	1.5	1.5	6
	Margarine (tsp)		1	2	
Morning	Banana, peeled (item)	83±7	1		5
Snack	1% fat yogurt (cup)	47		1	9
Lunch	White bread (slice)	100	2		1
	Pumpernickel bread (slice)	58		2	2
	Carrots (cup)	131	0.5		6
	Sweet corn (cup)	67±4		0.5	3
	Pineapple (g)	94	115		9
	Apple (item)	56		1	6
	Skim milk (cup)	46	1	1	6
	Ham (g)		84	84	
	American cheese (g)		19	19	
	Vanilla ice cream (cup)		1.3		
Afternoon	Potato crisps (g)	73	50		2
Snack	Orange (item)	61±11		1	13
Dinner	White bread (slice)	100	2		1
	Pumpernickel bread (slice)	58		2	2
	Medium grain white rice (cup)	83	0.8		1
	Long grain white rice (cup)	67±5		1	16
	Pumpkin pie (g)	107	131		17
	Green salad (cup)		2	2	
	Pinto Beans (cup)	64±6		0.5	11
	Lima beans (cup)	46±13		0.5	3
	Watermelon (cup)	103	2.5		9
	Pear (item)	58±7		1	13
	Skim milk (cup)	46	1	1	6
	Roast turkey breast (g)		100	100	
	Ranch salad dressing (tsp)		2	2	
	Cool whip whipped topping (tsp)		2		
Evening	White bread (slice)	100	2		1
Snack	Pumpernickel bread (slice)	58		2	2
	Peanut butter (tsp)		3	3	
	Fritos(g)	106±8	50		3
	Banana, peeled (item)	83±7	1		5

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