

Glycemic and Insulinemic Responses to Different Preexercise Snacks in Participants With Impaired Fasting Glucose

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Purpose: To compare serum glucose and insulin responses to 3 preexercise snacks before, during, and after exercise in individuals with impaired fasting glucose (IFG) and healthy (H) men. In addition, in an IFG population, the authors sought to determine whether a natural fruit snack (i.e., raisins) yields more desirable glucose and insulin concentrations than an energy bar or a glucose solution. **Methods:** The IFG ($n = 11$, age = 54.5 ± 1.3 yr, fasting blood glucose [BG] = 6.3 ± 0.1 mmol/L) and H groups ($n = 9$, age = 48.0 ± 3.1 yr, fasting BG = 4.9 ± 0.1 mmol/L) cycled at 50% of VO_{2peak} for 45 min on 4 occasions after consuming water or 50 g of carbohydrate from raisins (R), an energy bar (EB), or a glucose beverage (GLU). Metabolic markers were measured before, during, and after exercise. **Results:** In all nutritional conditions, glucose concentrations of the IFG group were consistently higher than in the H group. Differences between IFG and H groups in insulin concentrations were sporadic and isolated. In the IFG group, preexercise glucose concentration was lower in the R condition than in GLU. Ten and 20 min into exercise, glucose concentrations in the R and EB conditions were lower than in GLU. Insulin concentrations were lower in the R condition than in EB and GLU immediately before exercise and at Minute 10 but at 20 min R remained lower than only GLU. **Conclusion:** Glucose concentrations were higher in the IFG group regardless of preexercise snack. Compared with the glucose solution, raisins lowered both the postprandial glycemic and insulinemic responses, whereas the energy bar reduced glycemia but not insulinemia.

Keywords: insulin resistance, prediabetic, preexercise meal, glycemic load

Research targeting individuals suffering from impaired fasting glucose is warranted because of the recent evidence suggesting that increased physical activity can markedly reduce the progression from impaired fasting glucose to Type 2 diabetes (Foster et al., 2002; Lakka & Laaksonen, 2007). Depending on the caloric density and timing of the meal eaten before exercise, an individual may choose to eat a snack before exercise. Therefore, identification of preexercise snacks that can be used to fuel physical activity without causing excessive increases in the postprandial blood glucose or insulin responses would be helpful to those with impaired fasting glucose.

We are unaware of any studies in an impaired fasting glucose population that have evaluated the influence of different preexercise foods with low to moderate glycemic index (GI) on exercise metabolism. Consequently, it is unclear whether those with impaired fasting glucose should consume different preexercise foods than those

having normal fasting glucose. A variety of studies, using subjects with normal fasting-glucose values, have investigated the influence of preexercise meals of various GI levels on blood glucose and insulin concentrations. The outcomes of these investigations have varied. A number of studies found that high-GI preexercise foods result in immediate hyperinsulinemia that is followed by hypoglycemia during the initial phase of exercise (DeMarco, Sucher, Cisar, & Butterfield, 1999; Earnest et al., 2004; Johannsen & Sharp, 2007; Li, Wu, Gleeson, & Williams, 2004; Sparks, Selig, & Febbraio, 1998; Thomas, Brotherhood, & Brand, 1991; Wee, Williams, Gray, & Horabin, 1999; Wu & Williams, 2006). However, an equal number of studies have found that high-GI foods result in immediate hyperinsulinemia that is not followed by hypoglycemia at the onset of exercise (Bennard & Doucet, 2006; Burke, Claassen, Hawley, & Noakes, 1998; Febbraio & Stewart, 1996; Horowitz & Coyle, 1993; Kirwan, O’Gorman, & Evans, 1998; Stevenson, Williams, Mash, Phillips, & Nute, 2006; Thomas, Brotherhood, & Miller, 1994; Wee, Williams, Tsintzas, & Boobis, 2005). The recent findings of Ferland et al. (2009) also suggest that ingesting a low-GI food or fasting before exercise may benefit those diagnosed with Type 2 diabetes. However, it is not clear if this is also the case in those with impaired fasting glucose.

Consequently, the first aim of this study was to evaluate the postprandial, exercise, and postexercise blood

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glucose and insulin responses to preexercise snacks in people with impaired fasting glucose compared with individuals with normal fasting blood glucose values. Because of the nature of the disease, we hypothesized that consuming food of any GI level before exercise would lead to higher postprandial insulin and glucose concentrations in individuals suffering from impaired fasting glucose than in those with normal fasting glucose. We hypothesized that during exercise, serum insulin concentrations would require additional time to return to baseline in those with impaired fasting glucose.

Postprandial insulin concentrations of those with impaired fasting glucose have been shown to be reduced as a result of incorporating low-GI foods into the diet (Ostman, Frid, Groop, & Bjorck, 2006). Therefore, the second purpose of this study was to determine whether different low- to moderate-GI foods produce lower serum glucose and insulin concentrations in those with impaired fasting glucose during exercise than a high-GI food. For this study, a low-GI snack of raisins (GI = 49; (Kim, Hertzler, Byrne, & Mattern, 2008) was compared with isocarbhydrate portions of an energy bar that is typically recommended for exercise (PowerBar, GI = 58; Brand-Miller, Wang, McNeil, & Swan, 1997) and a glucose solution. Specifically, we hypothesized that the raisins would result in lower glucose and insulin levels during exercise than either the energy bar or glucose solution in both healthy people and those with impaired fasting glucose.

Methods

Participants

Criteria to participate included the following: age 35–65 years, male, nonathletic (no participation in athletic competition during the past year), nonexercising (non-compliance to American College of Sports Medicine guidelines of 30 min of moderate-intensity exercise 5 days/week; Haskell et al., 2007), fasting plasma glucose concentration 5.5–6.9 mmol/L for people with impaired fasting glucose (IFG group), fasting plasma glucose value <5.5 mmol/L for those with normal fasting glucose (H group), no tobacco use, and no infection, surgery, or corticosteroid treatment within the past 3 months or antibiotic therapy within the past 3 weeks.

Eleven individuals in the IFG group and 9 men in the H group completed the study. The study protocol was reviewed and approved by the institutional review board human subjects committee. All subjects provided written informed consent and completed Health Insurance Portability and Accountability Act (HIPAA) forms. Subject characteristics are presented in Table 1.

Testing Procedures

Initially, all subjects performed a peak-oxygen-consumption ($\text{VO}_{2\text{peak}}$) test, which was used to determine the intensity of the aerobic exercise to perform during the experimental protocol. On their arrival at the labora-

Table 1 Subject Descriptive Characteristics, $M \pm SEM$

	H (n = 9)	IFG (n = 11)
Age (years)	48.0 \pm 3.1	54.5 \pm 1.3
Height (cm)	182.7 \pm 2.7	177.6 \pm 2.3
Weight (kg)	92.4 \pm 5.6	92.4 \pm 4.2
Body-mass index	27.7 \pm 1.5	29.3 \pm 1.2
% body fat	20.6 \pm 2.1	23.4 \pm 1.7
$\text{VO}_{2\text{peak}}$ ($\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$)	33.3 \pm 1.9	33.1 \pm 2.2
Average fasting glucose (mmol/L)	4.9 \pm 0.1	6.3 \pm 0.1*

Note. H = healthy normal fasting-glucose group; IFG = impaired fasting-glucose group.

*Significantly different from H group ($p < .001$).

tory, height was measured using a stadiometer (Seca, Hanover, MD) and weight was determined using a balance scale (Health O Meter, Rye, NY). Electrodes were then placed on the chest of each subject such that heart rate and rhythm could be monitored via electrocardiogram (Fukuda Denshi Cardiomax FX-3010, Redmond, WA) and to allow for blood-pressure determination (Colin STBP-780, San Antonio, TX) before, during, and after exercise. Exercise was performed on an electronically braked bicycle ergometer (Lode Examiner, Groningen, The Netherlands). The initial workload was set at 50 W and progressively increased by 25–50 W every 2 min until volitional exhaustion. Volume of expired air, oxygen consumption, and respiratory-exchange ratio (RER) were quantified every 20 s via a two-way breathing valve (Hans Rudolph, Inc., Kansas City, MO) attached to a metabolic cart (Parvomedics Truemax 2400, Salt Lake City, UT). The metabolic cart was calibrated according to the manufacturer's specifications with gases of known concentration and a 3-L syringe before each test. Blood pressure and rating of perceived exertion (RPE), using both the 1–10 (Borg, 1982) and 6–20 Borg (1970) scales, were determined every 3 min.

Research Design

The experimental protocol was designed to compare the exercise responses to preexercise meals of varied GI using a randomized and controlled crossover design. Hence subjects reported to the laboratory on four separate occasions, each corresponding to a different preexercise meal; the meals were assigned in random order. The meals consisted of 296 ml of water, 296 ml of a glucose-tolerance test beverage with a GI of 100 (Sun-Dex, Fisher HealthCare, Houston, TX), a 69-g portion of raisins with a GI (using a glucose standard) of 49 (Kim et al., 2008; Sun-Maid, Kingsburg, CA), and a 77.4-g portion of chocolate-flavored energy bar (Power Bar) with a GI of 58 (Powerfood, Inc., Berkeley, CA). The GI of the Power Bar was determined using a white-bread refer-

ence food ($GI_{WB} = 83$) and then converted to GI for a glucose reference food (GI_G) of 58 by dividing the GI_{WB} by 1.4 (Brand-Miller et al., 1997). Water was a control, and the amounts of glucose, raisins, and energy bar were calculated to provide 50 g of available carbohydrate (total carbohydrate minus dietary fiber) based on the nutrition-label information. Complete nutritional information for all four preexercise snacks is presented in Table 2.

Subjects were asked to fast overnight and refrain from exercise for 12 hr before each visit. For 3 days before each visit, they were asked to consume a minimum of 150 g of carbohydrate per day, verified by 3-day diet records, to ensure adequate glycogen stores for exercise.

On arrival at the laboratory, a finger-prick blood sample (1–2 ml) was obtained, using sterile technique, to determine baseline values. Subjects were then provided one of the four meals and instructed to consume the meal within 10 min. On the first bite or drink of the meal, a timer was started, and exercise began precisely 30 min later. During this 30-min period the subject sat quietly. The exercise bout consisted of riding on an electronically braked cycle ergometer at 50% of the subject's measured VO_{2peak} . The workload to elicit 50% of VO_{2peak} was initially calculated using the following equation (American College of Sports Medicine, 2006):

$$\text{Workload (kg/min)} = \{50\% \text{ } VO_{2max} \text{ [ml/min]} - [3.5 \times \text{weight (kg)}]\} / 2$$

If necessary, this workload was then adjusted during the first 10 min of the first exercise bout based on actual measured VO_2 values. Once the workload was finalized, it remained consistent for all four exercise trials. Although cadence was selected by the subjects, it was noted so that it could be held consistent among all four of the subject's trials.

Additional blood samples (1–2 ml) were obtained from a finger prick using sterile technique at the following time intervals: 30 min after the meal (start of exercise); 10, 20, 30, and 45 min into the exercise (the conclusion of the exercise); and 5 min and 30 min postexercise. Blood samples were collected into serum separator tubes, allowed to clot, and then centrifuged at 1,168 g for 15 min to obtain serum. The serum was then stored at -20°C until analysis. Serum was analyzed for glucose, insulin, and free fatty acids as described later in this article.

Measurements of RER were obtained via the metabolic cart during exercise at Minutes 14–16, 24–26, and

39–41. Before each measurement, the metabolic cart was calibrated according to the manufacturer's specifications.

On completion of each of the four trials, subjects were allowed to return to their normal diet and activity patterns. A minimum of 3 days separated visits. Body composition was estimated during the last visit to the laboratory with skinfold measures described by Jackson and Pollock (1985). All measurements were performed by the same experienced technician using Harpenden calipers. Percent body fat was calculated via the Siri equation (Siri, 1961).

Blood Analysis

Glucose concentration of serum samples (25 μl) was determined using the dual-channel YSI 2700 Select Plus Biochemistry analyzer (YSI Instruments, Yellow Springs, OH). This instrument employs the glucose oxidase method.

Insulin concentration of serum samples (25 μl) was determined via the enzyme-linked immunosorbent assay (ELISA) technique using the DSL-10-1600 insulin ELISA kit (Diagnostic Systems Laboratories, Webster, TX) and the ELx808 microtiter plate reader (Bio-Tek Instruments, Winooski, VT) set to a wavelength of 450 nm. This method is an enzymatically amplified, one-step, sandwich-type immunoassay.

Free-fatty-acid concentration of serum samples (20 μl) was determined spectrophotometrically using the NEFA-HR (2) kit (Wako Chemicals, Richmond, VA), modified for a microtiter plate reader. This kit employs the acyl CoA synthetase and acyl CoA oxidase colorimetric method. As with the serum insulin assay, the ELx808 microtiter plate reader, set to a wavelength of 550 nm, was used to determine color change.

Statistical Analysis

All statistical analyses were conducted using SPSS software (version 15.0.1, SPSS Inc., Chicago, IL). Descriptive statistics were calculated and variables were examined for meeting assumptions of normal distributions. All data are presented as $M \pm SEM$. A general linear-model repeated-measures analysis was used to test differences across the different nutritional trials within each subject group. Differences across the subject groups were examined using general linear-model multifactor analyses of variance (ANOVA). When significant differences were found ($p < .05$), a Tukey's post hoc test was used for pairwise comparisons.

Table 2 Nutritional Information for Preexercise Snacks

Preexercise food	Glycemic index	Available carbohydrate (g)	Protein (g)	Fat (g)	Energy (kcal)
296 ml Glucodex	100	50	0	0	200
77.4 g energy bar	58	50	11.9	2.4	274
69 g raisins	49	50	1.7	0	224
296 ml water	0	0	0	0	0

Results

Subject characteristics are presented in Table 1. Subjects in both experimental groups were similar in age, height, weight, cardiovascular fitness (VO_{2peak}), and percent body fat. As dictated by design, subjects in the IFG group had significantly higher fasting blood glucose concentrations than the H group.

The experimental design called for subjects to cycle at 50% of their VO_{2peak} . The actual percentage of VO_{2peak} of each exercise trial was $53.2\% \pm 1.2\%$ for glucose, $52.2\% \pm 0.9\%$ for energy bar, $52.0\% \pm 0.8\%$ for raisins, and $51.9\% \pm 0.9\%$ for water. There were no significant differences in exercise intensity among the four nutritional conditions.

Comparisons Between Those With Impaired Fasting Glucose and Healthy Populations

For both the raisin and energy-bar conditions, serum glucose concentrations of the IFG group were significantly higher than in the H group at all time points except 5 min after exercise. For the glucose condition, the IFG group was significantly higher than the H group at all time points except 5 and 30 min postexercise. For the water condition, the IFG group was significantly higher than the H group at all time points (Figure 1).

In both the raisin and water conditions, serum insulin concentration of the IFG group was significantly higher than in the H group at 20 min into the exercise; there were

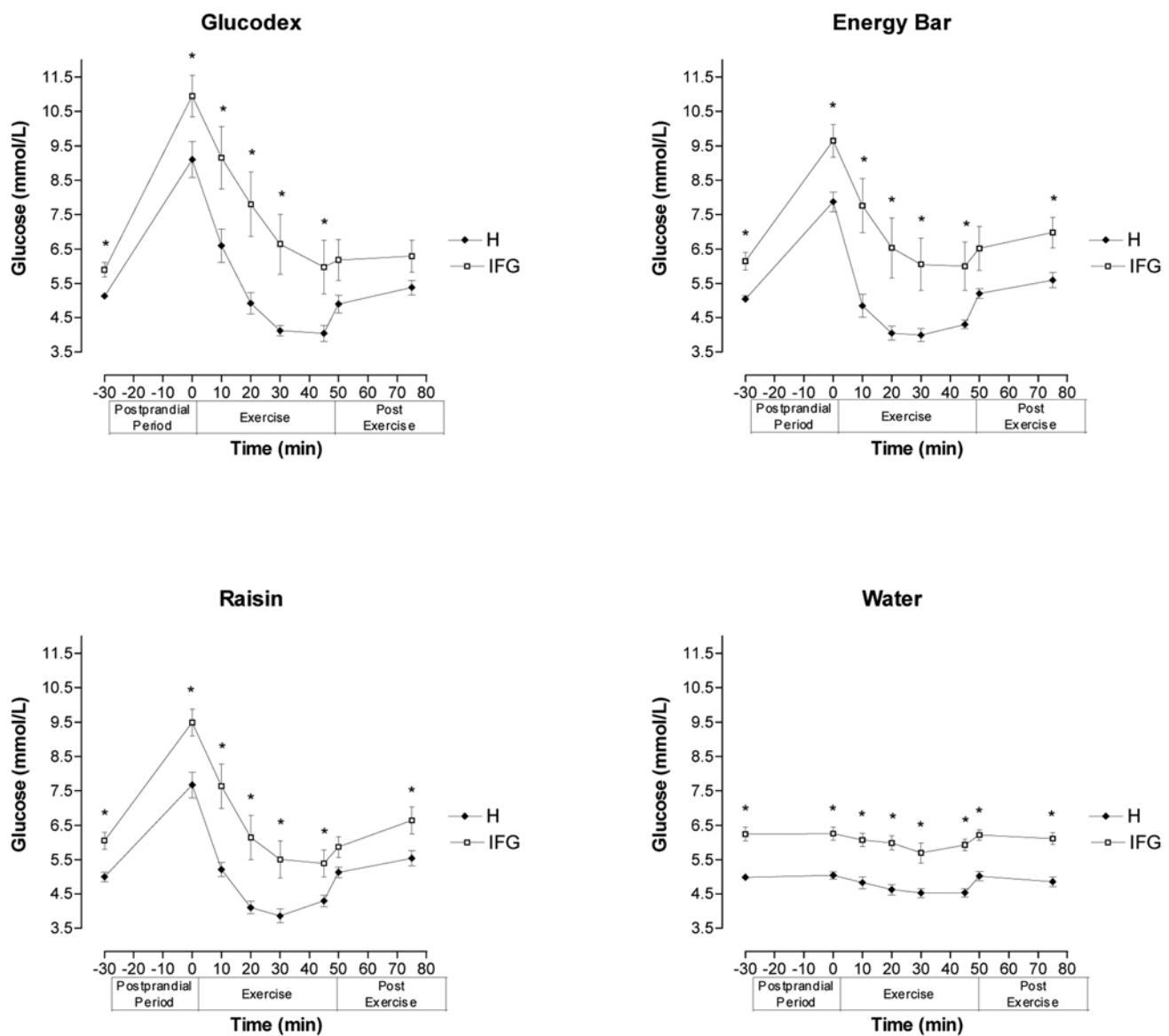


Figure 1 — Mean serum glucose responses in healthy (H) subjects and those with impaired fasting glucose (IFG) before, during, and after exercise in four different nutritional conditions, $M \pm SEM$. Foods were provided immediately after the blood collection at -30 min. *Significant differences at each time point between the two subject populations ($p < .05$).

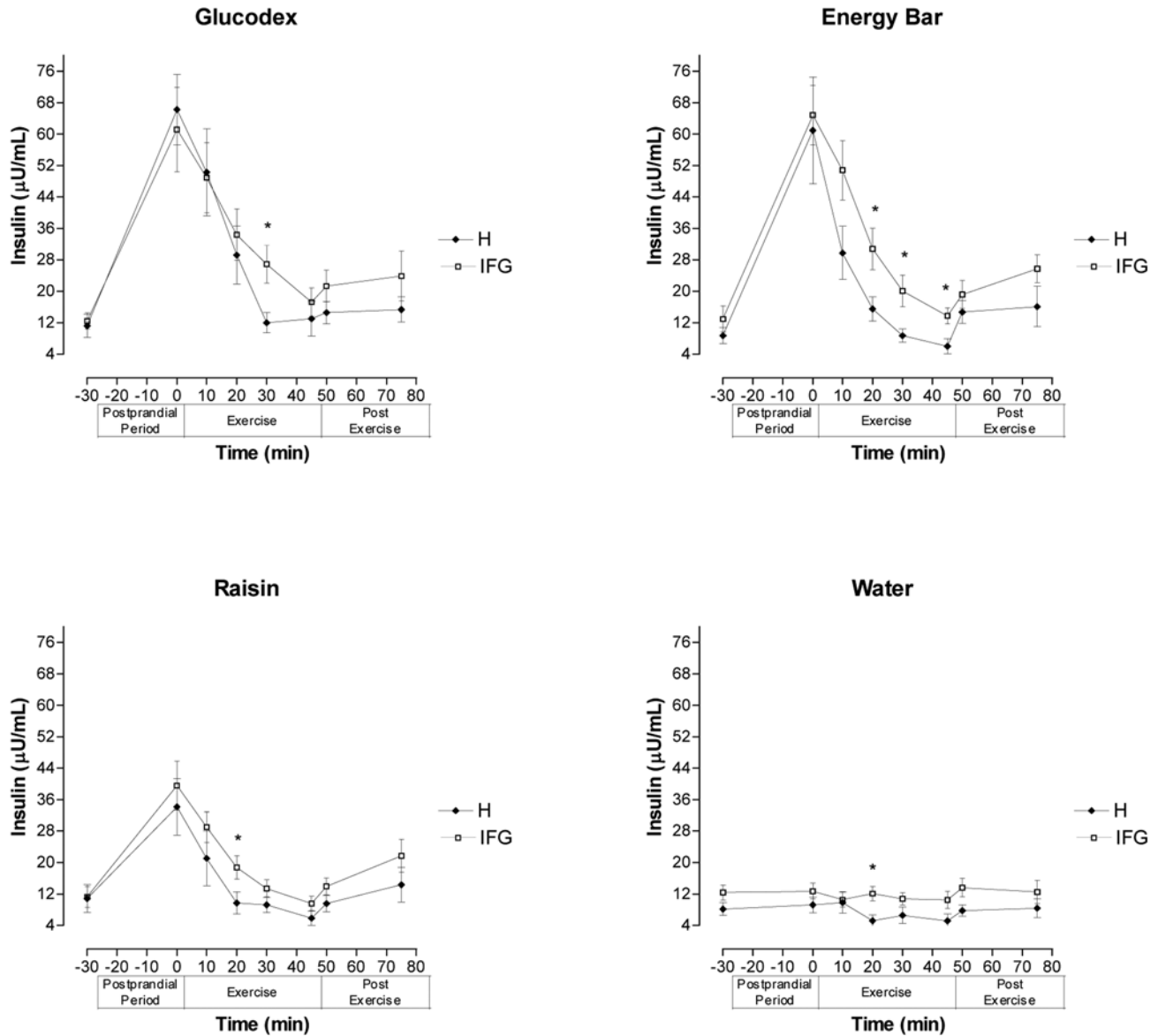


Figure 2 — Mean serum insulin responses in healthy (H) subjects and those with impaired fasting glucose (IFG) before, during, and after exercise in four different nutritional conditions, $M \pm SEM$. Foods were provided immediately after the blood collection at -30 min. *Significant differences at each time point between the two subject populations ($p < .05$).

no differences at any other time points. In the energy-bar condition, the IFG group was significantly higher than the H group at Minutes 20, 30, and 45 of exercise; there were no differences at any other time points. In the glucose condition, the IFG group was significantly higher than the H group at Minute 30 into the exercise; there were no differences at any other time points (Figure 2).

There were no significant differences in RER at any time points measured between the IFG and H groups for any nutritional trial.

There were no significant differences in serum free-fatty-acid concentration at any time points measured between the IFG and H groups for any nutritional trial.

Comparisons Among Preexercise Foods in Those With Impaired Fasting Glucose

There were no differences in blood glucose concentrations among the four nutritional conditions at 30 min before the start of exercise, 30 and 45 min into the exercise, and 5 and 30 min after the end of exercise. At 0 and 10 min into the exercise, blood glucose for the glucose, energy-bar, and raisin conditions was significantly higher than with water. At Minute 0, the glucose condition was significantly higher than raisins, and at Minutes 10 and 20, it was significantly higher than both energy bar and raisins (Figure 3).

There were no differences in insulin concentrations 30 min before the exercise, 45 min into the exercise, and 5 and 30 min after the end of exercise among the four nutritional conditions. However, during Minutes 0, 10 and 20, the glucose, energy-bar, and raisin conditions were significantly higher than water. At Minutes 0 and 10 into the exercise, the raisin condition was significantly lower than both the energy bar and glucose. However, at Minutes 20 and 30, it was only lower than glucose, not energy bar. In addition, at 30 min after the start of exercise, only

glucose and energy bar remained significantly higher than water (Figure 4).

At Minutes -30 and 0, there were no differences between nutritional trials. At Minute 10, energy bar, raisins, and glucose (energy bar = 0.38 ± 0.19 ; $R = .37 \pm 0.17$; glucose = 0.33 ± 0.20 mmol/L) were significantly lower than water (0.56 ± 0.27 mmol/L). These differences persisted through Minute 50. In addition, at Minute 45, the raisin condition (0.54 ± 0.22 mmol/L) was significantly higher than glucose (0.38 ± 0.17 mmol/L).

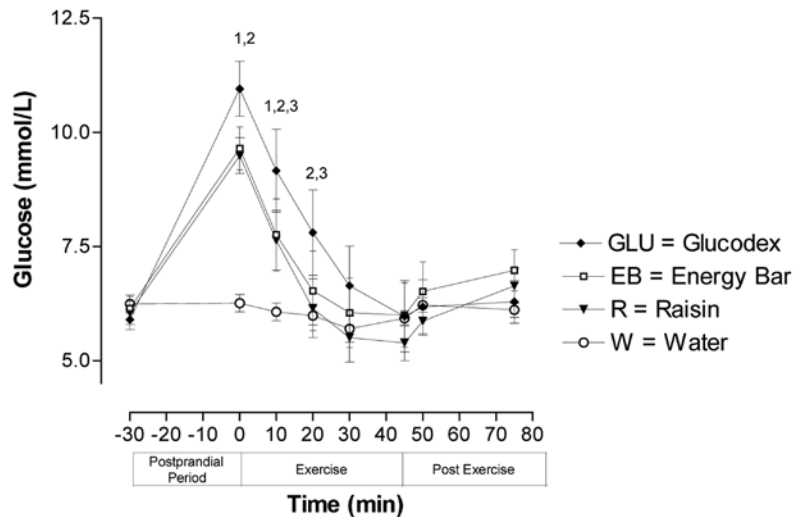


Figure 3 — Mean serum glucose responses in subjects with impaired fasting glucose before, during, and after exercise in four different nutritional conditions, $M \pm SEM$. Foods were provided immediately after the blood collection at -30 min. Significant differences ($p < .05$) between nutritional conditions are indicated with numbers 1–3: 1 = water is significantly ($p \leq .05$) different from glucose, energy bar, and raisins; 2 = glucose is significantly ($p \leq .05$) different from raisins; 3 = glucose is significantly ($p \leq .05$) different from energy bar.

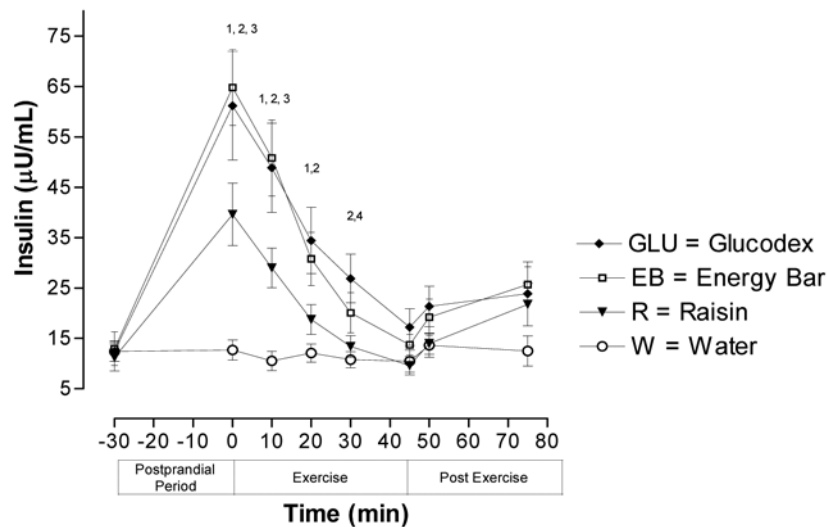


Figure 4 — Mean serum insulin responses in subjects with impaired fasting glucose before, during, and after exercise in four different nutritional conditions, $M \pm SEM$. Foods were provided immediately after the blood collection at -30 min. Significant differences ($p < .05$) between nutritional conditions are indicated with numbers 1–4: 1 = water is significantly ($p \leq .05$) different from glucose, energy bar, and raisins; 2 = glucose is significantly ($p \leq .05$) different from raisins; 3 = raisins is significantly ($p \leq .05$) different from energy bar; 4 = glucose and energy bar are significantly ($p \leq .05$) different from water.

At Minute 5 postexercise, raisins (0.77 ± 0.25 mmol/L) and energy bar (0.67 ± 0.21 mmol/L) were greater than glucose (0.47 ± 0.24 mmol/L). Finally, at Minute 30 postexercise, glucose (0.47 ± 0.22 mmol/L) and energy bar (0.50 ± 0.24 mmol/L) were lower than water (0.94 ± 0.55 mmol/L) and raisins (0.71 ± 0.33 mmol/L).

At Minute 15, water (0.92 ± 0.04) was significantly lower than energy bar (0.95 ± 0.05). At Minute 25 into exercise, the water condition was significantly lower (0.92 ± 0.05) than the glucose (0.94 ± 0.04) condition, and 40 min into exercise, both the water (0.91 ± 0.06) and raisin (0.90 ± 0.04) conditions were significantly lower than glucose (0.94 ± 0.04).

Discussion

The first purpose of this study was to evaluate the postprandial and exercise metabolic responses to preexercise snacks in people with impaired fasting glucose compared with individuals with normal fasting blood glucose concentrations. We hypothesized that consuming food of any GI level before exercise would lead to higher postprandial serum insulin and glucose concentrations in the IFG group than in the H group. Our first hypothesis was supported with postprandial serum glucose data—serum glucose concentrations were higher at all time points in the IFG group than in the H group for all three food-intake nutritional trials (glucose, energy bar, raisins). However, our hypothesis of elevated postprandial insulin concentrations in each nutritional trial in the IFG group was not supported across all time points (Figure 2). Our data demonstrate that although the postprandial insulin response was elevated at some time points, the mean fasting level was not. Although this finding was unexpected, it has been suggested that hyperinsulinemia might not occur early on in diabetic disease progression and that postprandial insulinemia appears to be affected before fasting insulinemia. Consequently, some people with impaired fasting glucose have normal insulin concentrations, and these concentrations increase as the disease state moves toward diabetes (Ivy, Zderic, & Fogt, 1999).

We hypothesized that serum glucose and insulin concentrations at exercise onset would be elevated in the IFG group and would therefore require additional time to return to baseline. In all trials and at most of the time points, serum glucose concentration was higher during exercise in the IFG than the H group. There were no differences in serum insulin concentrations between the two subject groups at the onset of exercise, yet in the glucose and raisin conditions, serum insulin took significantly longer to return to baseline in the IFG group. Therefore, most of our data suggest that serum insulin concentrations take longer to return to baseline in those with impaired fasting glucose than in “healthy” individuals, despite similar peak postprandial values. This is likely because of the insulin-resistant status of the IFG group—elevated serum glucose concentration requires the presence of insulin in the blood for a longer period of time to facilitate glucose disposal. Comparisons between our data and those of other investigators are difficult because, although

two studies (Ludwig, 2003; Sun, See, Huse, Tsai, & Lin, 2001) have investigated postprandial insulin responses of varying-GI diets in a diabetic population, we are unaware of other studies that have examined the insulin response during exercise in patients with diabetes or those with impaired fasting glucose.

The second purpose of this study was to determine whether a snack of raisins would alter the metabolic profile during exercise compared with an energy bar or glucose. Raisins have a high fructose content (roughly 50% of the total carbohydrate content; Matthews, Pehrsson, & Farhat-Sabet, 1987). Fructose has been shown to produce an attenuated effect on blood glucose and insulin responses compared with glucose and correspondingly has a low GI (Crapo, Kolterman, & Olefsky, 1980; Foster-Powell, Holt, & Brand-Miller, 2002; Lee & Wolever, 1998). For these reasons, we hypothesized that the preexercise raisins would induce lower glycemic and insulinemic responses than the energy bar or glucose.

We further hypothesized that the combination of the independent effects of fructose on substrate oxidation and the lower insulinemic response from fructose would result in higher rates of fat oxidation with the raisins relative to the other treatments, as indicated by a lower RER during exercise. Massicotte et al. (1986) showed lower exogenous carbohydrate oxidation and higher fat utilization after ingestion of [^{13}C] fructose than [^{13}C] glucose during 90 min of exercise on a cycle ergometer at 50% $\text{VO}_{2\text{max}}$. However, plasma free-fatty-acid concentrations were similar between [^{13}C] fructose and [^{13}C] glucose. Guezennec et al. (1987) also reported lower fructose oxidation and increased fat utilization after ingestion of [^{13}C] fructose than [^{13}C] glucose during 120 min of cycle-ergometer exercise at 60% $\text{VO}_{2\text{max}}$. The lower oxidation of fructose was compensated for by the increased fat utilization, which was caused by increased lipolysis because of low insulin concentrations (Guezennec et al., 1987). It is well known that insulin inhibits the release of free fatty acids from the adipose tissue. Thus, it might be expected that the preexercise raisins would result in lower glycemic and insulinemic responses and greater fat oxidation than the energy bar or glucose. This could thereby promote the goal of exercise-induced weight loss in an IFG population.

Our insulin data, before and in the early phase of exercise, fully support this hypothesis (see Figure 4), in so much as insulin concentrations in the raisin condition were significantly lower than with energy bar and glucose through Minute 10 of exercise. However, our glucose data at these same time points only partially support this hypothesis, because glucose concentrations in the raisin condition were only lower than with glucose, not energy bar. These findings showing the benefits of a low-GI food on insulin concentration are in agreement with those of others (Ludwig, 2003; Riccardi, Rivellese, & Giaco, 2008; Rizkalla, Taghrif, & Laramiguere, 2004). Despite these findings with regard to insulin, in our study we did not observe differences in either nonesterified fatty acids or changes in RER that would indicate increased fat oxidation with the raisins versus

either the energy bar or glucose. The reason for this is not clear but could be related to insufficient duration of exercise in our study.

Ludwig (2003) demonstrated that habitual low-GI-food intake reduces insulin concentrations in subjects with impaired fasting glucose, whereas Riccardi et al. (2008) documented that both habitual and acute consumption of low-GI foods have beneficial effects on blood glucose control in patients with diabetes. Rizkalla et al. (2004) demonstrated that 4 weeks of a low-GI diet induced lower postprandial plasma glucose and insulin profiles and areas under the curve than a high-GI diet in 12 subjects with Type 2 diabetes. Our acute data corroborate these findings in that the raisin condition yielded attenuated insulin concentrations.

This is in contrast, however, to findings (Liese et al., 2005) indicating no association between the GI of habitual dietary intake and fasting insulin concentrations in normal subjects and those with impaired glucose tolerance (total $N = 979$). That study, however, was cross-sectional in design, making comparisons with our data difficult.

During exercise, our data suggest that the raisin condition is minimally beneficial in terms of serum glucose concentration, in so much as exercising glucose concentrations in the raisin condition were significantly lower than with glucose but not energy bar (see Figure 3). It is interesting to note that the 9-unit difference in GI between the energy bar and raisins did not translate into actual differences in measured postprandial glycemic curves in either group of subjects. However, for insulin, the raisin condition produced attenuated insulin responses at two of four time points during exercise. One potentially confounding factor in our study is the protein content (11.9 g) of our energy-bar meal, because protein can be insulinotropic in nature. Protein has been shown to stimulate insulin release as a result of delayed gastric emptying and increases in glucose-dependent insulinotropic polypeptide and glucagon-like peptide 1 (Holst & Orskov, 2001). This possibly explains why the energy-bar condition demonstrated higher serum insulin concentrations than raisins, yet there was a similar blood glucose response between energy bar and raisins.

If the energy bar had not contained protein, it too may have shown a more attenuated insulin response. However, a bar with less protein might contain more carbohydrate, possibly causing higher glycemic and similar insulinemic responses as the bar with the original protein content.

The lower serum insulin concentrations during exercise in our study are not in agreement with the findings of Backhouse, Williams, Stevenson, and Nute (2007). Their study examined the metabolic responses during an hour of walking 3 hr after consuming a meal with a GI of 77 compared with a meal with a GI of 51. Insulin concentrations were not different at any time points during exercise. That study, however, used a small number of subjects with normal fasting blood glucose levels. In addition, they used a 3-hr postprandial period before exercise, whereas ours was only 30 min, which makes comparisons difficult.

It is conceivable that individuals would want to consume a preexercise snack, especially if they have not been able to eat a meal in the hours leading up to exercise. The major conclusion of this study indicates that information on both the glycemic and insulinemic responses to preexercise foods may be important, especially for those with impaired fasting glucose. In addition, the choice of low- or moderate-GI preexercise foods versus a high-GI food may decrease glycemia and/or insulinemia during exercise. In particular, foods containing fructose, as well as fiber, might be more beneficial in terms of decreased insulinemia. Chronically elevated insulin concentrations have been shown to promote fat storage, increase blood pressure, increase cholesterol, and ultimately increase the risk of cardiovascular disease. Therefore, from the perspective of insulin response before and during exercise, both individuals with impaired fasting glucose and those with normal fasting glucose values should consider consuming a low-GI preexercise snack.

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