A Low-Glycemic Diet Lifestyle Intervention Improves Fat Utilization during Exercise in Older Obese Humans

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Objective: To determine the influence of dietary glycemic index on exercise training-induced adaptations in substrate oxidation in obesity.

Design and Methods: Twenty older, obese individuals undertook 3 months of fully supervised aerobic exercise and were randomized to low- (LoGIX) or high-glycemic (HiGIX) diets. Changes in indirect calorimetry (VO2; VCO2) were assessed at rest, during a hyperinsulinemic-euglycemic clamp, and during submaximal exercise (walking: 65% VO2max, 200 kcal energy expenditure). Intramyocellular lipid (IMCL) was measured by 1H-magnetic resonance spectroscopy.

Results: Weight loss (−8.6 ± 1.1%) and improvements (P < 0.05) in VO2max, glycemic control, fasting lipemia, and metabolic flexibility were similar for both LoGIX and HiGIX groups. During submaximal exercise, energy expenditure was higher following the intervention (P < 0.01) in both groups. Respiratory exchange ratio during exercise was unchanged in the LoGIX group but increased in the HiGIX group (P < 0.05). However, fat oxidation during exercise expressed in relation to changes in body weight was increased in the LoGIX group (+10.6 ± 3.6%; P < 0.05). Fasting IMCL was unchanged, however, extramyocellular lipid was reduced (P < 0.05) after LoGIX.

Conclusions: A LoGIX/exercise weight-loss intervention increased fat utilization during exercise independent of changes in energy expenditure. This highlights the potential therapeutic value of low-glycemic foods for reversing metabolic defects in obesity.

Introduction

In view of the growing obesity epidemic, lifestyle interventions must be developed to aid not only weight loss but also to reverse the underlying metabolic impairments present in overweight individuals. Several published exercise and diet interventions have yielded promising results with regards to improving body composition, insulin sensitivity, and glucose tolerance in such populations (1,2), and while the mode of exercise can influence these outcome variables (3,4) it is also clear that dietary composition plays an important role (5,6).

We recently reported that a 3-month exercise training intervention when combined with a low-glycemic diet (LoGIX) induces a more favorable metabolic state in older obese humans when compared to a high-glycemic intervention (7-10). Further, our short-term (7-day) lifestyle intervention that combined a low-glycemic index (GI) diet with daily aerobic exercise caused an increase in fasting intramyocellular lipid (IMCL) and a decrease in extramyocellular lipid (EMCL), suggestive of preferential substrate oxidation (10). Prior experiments conducted on lean healthy individuals suggest that low-GI meals may alter substrate oxidation during acute exercise bouts, towards increased fat utilization (11-16). In addition, ingesting meals with a low-glycemic response has been shown to reduce fasting respiratory exchange ratios (RERs) in some (17,18) but not all (19,20) studies. Such findings have clinical implications in that the prescription of low-glycemic meals in an obese population may increase weight loss via increases in fat oxidation rates. However, no prior study has examined the effect of dietary GI on substrate metabolism...
during exercise in obese individuals following diet and exercise-induced weight loss.

This investigation examined the effects of a combined exercise training and LoGIX intervention on energy expenditure and substrate utilization at rest, during a hyperinsulinemic euglycemic clamp, and during submaximal exercise. In addition, we measured fasting skeletal muscle lipid using proton magnetic resonance spectroscopy (1H-MRS) prior to and following the intervention period. Given the evidence that low-GI meals can increase exercise-induced fat utilization, we hypothesized that following 3 months of exercise training a low-GI diet would increase fat oxidation rates during submaximal exercise.

Methods

Participants

Twenty older, obese men and women (age 65 ± 1 years; BMI 35.0 ± 1.0 kg/m²; mean ± S.E.M.) volunteered to participate in a 3-month lifestyle intervention. All participants were weight stable and sedentary. The study was conducted using a randomized, parallel-group, repeated measures design. We previously described the design in detail in (8). The variables in this paper have not been published previously. Medical screening excluded individuals with heart, kidney, liver, thyroid, intestinal, and pulmonary diseases, or those taking medications known to affect our outcome variables. Resting ECG and submaximal exercise stress tests excluded individuals contraindicated to increments in physical activity. At the screening visit, volunteers also underwent a 30-min assessment of resting energy expenditure using ventilated-hood indirect calorimetry. Resting energy expenditure was multiplied by a physical activity factor of 1.2 to calculate the caloric load of the subjects’ diets during inpatient testing and study interventions. The study was approved by the Cleveland Clinic Institutional Review Board, and all subjects provided informed written consent in accordance with our guidelines for the protection of human subjects.

Intervention

All participants performed fully supervised aerobic exercise (treadmill walking and cycle ergometry) 60 mins/day, 5 days/week for 3 months at ~65% of their VO2max [full details of the exercise intervention are available in (21)]. In addition, participants were randomized to receive either a LoGIX (40 GI units, LoGIX; n = 10) or a high-GI diet (80 GI units, HiGIX; n = 10). The two diets were macronutrient matched. For the duration of the 3-month intervention, all food and drink items were provided to participants on a daily basis by a registered dietician. We have previously published full details of the dietary interventions and sample menus in (7). Dietary analysis was performed using Nutritionist Pro software (Axxya Systems, Stafford, TX).

Inpatient control period

Prestudy and poststudy assessments of body composition (dual-energy X-ray absorptiometry; iDXA; Lunar, Madison, WI), oral glucose tolerance, insulin sensitivity [40 mU m⁻² min⁻¹ hyperinsulinemic euglycemic clamp, as previously described (7)], aerobic fitness [VO2max; as previously described (21)], and substrate metabolism (as described below) were performed during a 3-day inpatient stay in the Clinical Research Unit at the Cleveland Clinic. During the prestudy inpatient stay, participants received a diet with a moderate GI (55-60 GI units). During the poststudy inpatient stay, subjects continued their corresponding LoGIX or HiGIX interventions. Post-study metabolic measures (clamp and calorimetry) were performed ~16 h after the last exercise bout. Although energy balance was not measured directly, we ensured that all subjects received a daily caloric amount that was relative to their own caloric requirements as assessed by indirect calorimetry. Therefore, any deviations from energy balance would be equal between groups and not influence the metabolic outcomes of the study.

Substrate Metabolism

Resting and insulin-stimulated (clamp-derived) energy expenditure (22) and substrate oxidation rates (23) were determined via indirect calorimetry (Vmax Encore, Viasys, Yorba Linda, CA). Expired air was collected over a 30-min period immediately upon waking after an overnight fast and during the final 30 min of the clamp. Metabolic flexibility was calculated as the insulin-stimulated change in RER from resting conditions. In addition, following an overnight fast, each subject completed a steady-state submaximal treadmill walking exercise test at 65% of their VO2max until 200 kcal of energy had been expended. VO2 was continuously monitored via indirect calorimetry (Jaeger Oxycon Pro, Viasys, Yorba Linda, CA) so that energy expenditure (EE) could be assessed in real-time. The data reported are calculated as the mean of the final 5 min of exercise. Following the 3-month intervention, subjects’ repeated the submaximal exercise test. No blood samples were collected during these exercise tests. To control for the acute effects of exercise, pre-intervention exercise tests were conducted >48 h prior to metabolic measures.

IMCL and EMCL assessments

1H-MRS was used to quantify fasting lipid levels in the soleus (slow-twitch) muscle before and after the lifestyle intervention. Briefly, the right calf of each subject was positioned in a knee coil and positioned near isocenter within a 4T Bruker Medspec MRI scanner (Siemens, Malvern, PA). Following manual shimming to improve the spectral resolution of the acquisition [water linewidth (FWHM) ≤ 50 Hz (~0.3 ppm)], a single-voxel Point-Resolved Spectroscopy (PRESS) acquisition (TR/TE = 1500/135 ms, 128 averages, voxel size = 1 cm³, water suppression, ADC BW = 10.5 ppm) was used to acquire the MRS data. Repeatability of the voxel placement following the intervention period was facilitated with anatomic landmarks. Following the acquisition, all spectra were reconstructed and exported to Matlab (The Mathworks, Natick, MA) for quantitative analysis of the lipid peaks. Fiber orientation modeling, previously described by Khuu et al. (24), was then applied to the MRS spectra. The IMCL and EMCL methylene and methyl residues were calculated and the relative concentrations of IMCL and EMCL levels were derived using the creatine signal as a concentration standard, assuming the skeletal muscle creatine concentration is approximately equal to 30 mmol/kg muscle wet weight. We have reported the use of this method previously (10). A total of 34 subjects were originally randomized to our HiGIX/LoGIX intervention. Of these 34, substrate oxidation data during exercise was collected in 20 subjects (10 HiGIX, 10 LoGIX); this manuscript presents that data. 1H-MRS data were collected in the original cohort, but because of a computer failure a large amount of spectroscopy data were lost.
leaving us with MRS data in 15 subjects (11 HiGIX, 4 LoGIX). Of these 15, a total of 10 subjects (8 HiGIX, 2 LoGIX) also had substrate oxidation measurements during exercise.

### Biochemical analyses
Plasma glucose was determined on a YSI 2300 STAT Plus analyzer (Yellow Springs, OH); plasma insulin via radioimmunoassay (Millipore, Billerica, MA). Plasma triglycerides and cholesterol were analyzed by enzymatic methods on an automated platform (Roche Modular Diagnostics, Indianapolis, IN). Glycated hemoglobin (HbA1c) was measured via nonporous ion exchange HPLC (G7 HPLC Analyzer, Tosoh Bioscience, San Francisco, CA).

### Statistics
Analyses were carried out using Prism v4 (GraphPad, San Diego, CA) and Statview v5.0.1 (SAS Institute, Cary, NC). Unpaired t-tests were used to compare group differences at baseline. Two-way (group x time) repeated measures ANOVA was used to identify between-group (LoGIX vs. HiGIX) differences. Bonferroni post hoc tests were used when significant group x time interactions arose. Additional ANCOVA analyses revealed that gender did not affect any variable in this intervention, nor were changes in RER influenced by the baseline differences between HiGIX and LoGIX groups. Between group changes in non-normally distributed variables were examined via Mann-Whitney tests. Bivariate correlation analyses were used to identify relationships between baseline variables and changes (Δ) in variables following the intervention period using Spearman’s rank correlation. Statistical significance was accepted when \( P < 0.05 \) and data are expressed as mean ± S.E.M.

### Results

#### Intervention
During presubject inpatient studies, subjects consumed 1733 ± 137 kcal/day with a macronutrient composition of 53 ± 1, 30 ± 1, 17 ± 0% of kcal from carbohydrate, fat, and protein. During the intervention subjects consumed 1880 ± 105 kcal/day with a macronutrient composition of 54 ± 0, 29 ± 1, 17 ± 0% of kcal from carbohydrate, fat, and protein. The total energy intake and macronutrient composition was not different between LoGIX and HiGIX groups, as previously reported in full in (25). Dietary adherence was 97 ± 1% (percent calories consumed relative to calories provided), and the mean study GI for the LoGIX group was 39.8 ± 6.2 and 80.0 ± 6.6 for the HiGIX group. Mean adherence was 97 ± 1% and 97 ± 1%, respectively. Maximal glycemic index, exercise, and substrate oxidation

### Subject characteristics
Both LoGIX and HiGIX groups showed equal weight loss (−8.6 ± 1.1%) and equal decreases in whole body adiposity (Table 1; both \( P < 0.05 \)), plus equal improvements in glycemic control (Table 1: reduced fasting plasma glucose [FPG] and hemoglobin A1c [HbA1c]); increased glucose disposal rates [GDR] during euglycemic hyperinsulinemia, all \( P < 0.05 \)) and fasting lipemia (Table 1: decreased triglycerides and cholesterol, both \( P < 0.05 \)). Maximal oxygen uptake (VO2max) during exhaustive aerobic exercise was also equally increased in both LoGIX and HiGIX groups (+18.9 ± 3.1%; Table 1: \( P < 0.05 \)).
Resting and insulin-stimulated energy metabolism

EE (kcal kg\(^{-1}\) min\(^{-1}\)) during resting and insulin-stimulated conditions increased following the 3-month intervention in both groups (Table 2: \(P < 0.05\)). Absolute changes in EE (kcal/min) showed the same trends but the effect of time was not significant (\(P = 0.13\)). RERs were equally decreased in both LoGIX and HiGIX groups during resting conditions, and was equally increased in both groups under insulin-stimulated conditions (Table 2: \(P < 0.05\)). Finally, a statistically significant increase in insulin-stimulated metabolic flexibility was found in both groups (Table 2: \(P < 0.05\)).

Submaximal exercise energy metabolism

Time to completion of submaximal exercise (200 kcal EE) was 31 ± 2 and 27 ± 2 min, pre-intervention vs. postintervention in all subjects (\(P < 0.05\)). This was not different between groups. No differences in exercise intensity between groups or trials were found: submaximal exercise was performed at 65.1 ± 0.7% of the subjects’ VO\(_{2}\)max (Table 3). Compared to prestudy measures, the rates of oxygen uptake (VO\(_{2}\), l/min and ml kg\(^{-1}\) min\(^{-1}\)) and EE (kcal/min) during submaximal exercise were higher following the 3-month intervention (Table 3 and Fig. 1 Panel [A]; PRE vs. POST, \(P < 0.05\)). The increases in EE and VO\(_{2}\) were identical in both the LoGIX and HiGIX groups. RERs during exercise were unchanged in the LoGIX (\(P > 0.05\)) following the study but increased in the HiGIX group (Figure 1 [B], \(P < 0.05\)). This change in RER in the HiGIX was significantly different from the LoGIX group (\(P < 0.05\)). Rates of carbohydrate oxidation during exercise expressed in relation to changes in body mass (mg kg\(^{-1}\) min\(^{-1}\)) were increased in both groups following the intervention (Figure 1 [C], \(P < 0.05\)), whereas fat oxidation rates during exercise were increased in the LoGIX group only (mg kg\(^{-1}\) min\(^{-1}\); Figure 1 [D], \(P < 0.05\)). To further examine these changes in carbohydrate and fat oxidation, we calculated the percentage of total caloric EE that is derived from the two substrates (Figure 2). Following the intervention, calories derived from either carbohydrate or fat sources during exercise were unchanged in the LoGIX group (\(P > 0.05\)), whereas in the HiGIX group a significant increase in carbohydrate utilization (Fig.2 [A], \(P < 0.05\) and decrease in fat utilization was found (Figure 2 [B], \(P < 0.05\)).

Skeletal muscle lipids

We had complete MRS measurements in \(n = 15\) subjects (4 LoGIX; 11 HiGIX). The 12 week lifestyle intervention did not significantly (\(P > 0.05\)) affect resting skeletal muscle [IMCL] (change from pre-study, LoGIX vs. HiGIX: –2.3 ± 1.2 vs. –1.0 ± 1.3 mmol/kg.

### TABLE 2 Resting and insulin-stimulated energy metabolism

| Energy metabolism | LogIX | HiGIX |
|-------------------|-------|-------|
|                   | PRE   | POST  | PRE   | POST  |
| Rest RER, a.u.    | 0.861 ± 0.008 | 0.838 ± 0.011* | 0.866 ± 0.018 | 0.831 ± 0.014* |
| EE, ×10\(^3\) kcal kg\(^{-1}\) min\(^{-1}\) | 10.6 ± 0.2 | 11.1 ± 0.3§ | 10.8 ± 0.3 | 11.5 ± 0.2§ |
| Cox, mg kg\(^{-1}\) min\(^{-1}\) | 1.29 ± 0.08 | 1.08 ± 0.19 | 1.26 ± 0.18 | 1.22 ± 0.16 |
| Fox, mg kg\(^{-1}\) min\(^{-1}\) | 0.334 ± 0.047 | 0.420 ± 0.089 | 0.360 ± 0.072 | 0.378 ± 0.106 |
| Insulin RER, a.u. | 0.885 ± 0.009 | 0.905 ± 0.014 | 0.860 ± 0.011 | 0.908 ± 0.012 |
| EE, ×10\(^3\) kcal kg\(^{-1}\) min\(^{-1}\) | 10.7 ± 0.2 | 11.5 ± 0.6 | 11.4 ± 0.4 | 11.7 ± 0.6 |
| Cox, mg kg\(^{-1}\) min\(^{-1}\) | 1.58 ± 0.08 | 1.95 ± 0.13 | 1.49 ± 0.12 | 2.04 ± 0.16 |
| Fox, mg kg\(^{-1}\) min\(^{-1}\) | 0.307 ± 0.042 | 0.289 ± 0.060 | 0.439 ± 0.044 | 0.271 ± 0.055 |
| Metabolic flexibility, a.u. | 0.023 ± 0.012 | 0.067 ± 0.012* | 0.006 ± 0.017 | 0.077 ± 0.017* |

Data represent mean ± SEM RER; respiratory exchange ratio; a.u., arbitrary units; EE, energy expenditure; Cox, carbohydrate oxidation; Fox, fat oxidation; % of EE, percentage of energy expenditure derived from either Cox or Fox; Insulin, measurements made during the final 30 min of 40 mU m\(^{-2}\) min\(^{-1}\) hyperinsulinemic euglycemic clamp; Metabolic flexibility; the difference between the RER during insulin-stimulated conditions and at rest. Statistical analysis using two-way ANOVA indicated significant pre- vs. post-study differences (\(P < 0.05\)), but no significant effects of trial or time × trial interactions. A \(P = 0.05\) increase in EE is represented by §.

### TABLE 3 Oxygen consumption (VO\(_{2}\)) during submaximal exercise performed at 65% of VO\(_{2}\)max

| Exercise intensity | LogIX | HiGIX |
|--------------------|-------|-------|
|                   | PRE   | POST  | PRE   | POST  |
| \(\text{VO}_{2}\) l/min | 1.32 ± 0.11 | 1.50 ± 0.15* | 1.52 ± 0.15 | 1.77 ± 0.18* |
| \(\text{ml kg}^{-1}\text{min}^{-1}\) | 13.5 ± 0.6 | 17.7 ± 1.3* | 15.5 ± 0.9 | 20.2 ± 1.7* |
| % \(\text{VO}_{2}\)max | 66.8 ± 1.0 | 65.6 ± 0.7 | 66.9 ± 0.7 | 66.5 ± 0.9 |

Data represent mean ± SEM. Submaximal exercise was performed before (PRE) and after (POST) the intervention at 65% of prestudy \(\text{VO}_{2}\)max until 200 kcal of energy had been expended. \(\text{VO}_{2}\), steady state rate of oxygen consumption measured by indirect calorimetry; %\(\text{VO}_{2}\)max, steady state oxygen consumption during exercise expressed as a percentage of maximal oxygen uptake. Statistical analysis using two-way ANOVA indicated significant effects of time: pre- vs. post-study differences are indicated by * \(P < 0.05\).
muscle wet weight) or [IMCL/EMCL] (change from prestudy, LoGIX vs. HiGIX: 0.21 ± 0.23 vs. −0.18 ± 0.16) in either group. However, the change in resting [EMCL] following the intervention period was significantly different between groups (P = 0.01) (change from prestudy, LoGIX vs. HiGIX: −14.9 ± 6.4 vs. 11.4 ± 6.7 mmol/kg muscle wet weight). No main effects were seen for muscle creatine values. In addition, relationships between skeletal muscle lipids and substrate oxidation rates at rest, insulin-stimulation and submaximal exercise were explored via correlation analyses in 10 subjects (2 LoGIX; 8 HiGIX) who had complete data-sets from both MRS and indirect calorimetry measures. At baseline, [EMCL] was inversely correlated (Rho = −0.704, P = 0.02) with the rate of resting carbohydrate oxidation (mg kg⁻¹ min⁻¹) and resting RER (Rho = −0.642, P = 0.04). Following the intervention, the change in [EMCL] was positively correlated (Rho = 0.726, P = 0.02) with the change in carbohydrate oxidation (mg kg⁻¹ min⁻¹) measured during submaximal exercise, and inversely correlated with the change in fat oxidation during submaximal exercise (Rho = −0.808, P = 0.003). The changes in [IMCL]/[EMCL] were also correlated with the changes in carbohydrate (Rho = −0.862, P = 0.001) and fat oxidation (Rho = 0.748, P = 0.01) during submaximal exercise. As a result of low subject numbers in these comparisons, we acknowledge these data as preliminary observations and they must be interpreted with caution until they can be verified in a larger cohort.

**Discussion**

Our data indicate that exercise-induced alterations in body composition, insulin sensitivity, and EE are not influenced by dietary GI in obese subjects. However, substrate metabolism during submaximal exercise was differentially altered between the dietary GI groups. A high GI diet increased the RER during exercise and increased the reliance on carbohydrate oxidation to sustain exercise performed at a moderate intensity. Conversely, consumption of a low GI diet during the exercise training intervention led to increased partitioning of fat towards EE during exercise in our older, obese subjects.

Clapp et al. previously showed that prolonged habituation (20 days) to a low GI diet reduces fasting RERs and increases fat oxidation in healthy women (17). Similar findings have been obtained in animal models (25,26). In addition, Bouche et al. and Pittas et al. have
demonstrated improved weight loss following a low GI diet (27,28). In this study, weight loss and increases in fasting RER induced by the combination of diet and exercise were equal among older obese individuals consuming either a low or high GI diet. Thus, when a negative energy balance is induced by diet and exercise, the overall changes in body composition are not influenced by the glycemic responses of the diet. While weight loss is a surrogate marker of improved metabolic health, it may not be indicative of all metabolic variables. As we have previously demonstrated from this same intervention, variables such as insulin secretion and inflammation show divergent trends between the LoGIX and HiGIX interventions in the presence of equal improvements in body composition (8-10). Further to this dietary group disparity, we now demonstrate that substrate metabolism during submaximal exercise is also influenced by dietary GI. However, as a result of our study design we cannot determine whether this disparity is because of the last meal or a metabolic adaptation to 3-months of meal feeding. That said, submaximal exercise tests were performed following an overnight fast, limiting the possible acute effects of meal GI. The larger reliance on lipid as a fuel during exercise following the intervention in the LoGIX group indicates that carbohydrate is being spared. Previous work has shown that low-glycemic feeding prior to aerobic exercise elevates fat oxidation during exercise (11-16), and that this may occur because of the lower postprandial insulin responses compared to high-glycemic meals, thus increasing the availability of free fatty acids for oxidation in the mitochondria (29,30). These findings highlight dietary glycemic responses as an important consideration with regard to substrate oxidation during physical activity.

Several prior weight loss interventions have examined changes in energy metabolism (31-33). Our data indicate an increase in resting EE in the presence of ~9% weight loss, as well as increased EE during submaximal exercise. In this study, subjects’ aerobic fitness (VO₂max) was measured every 2-weeks throughout the intervention such that the work load of each exercise training session could be adjusted to maintain the same relative exercise intensity throughout the study. As a result the submaximal exercise tests were therefore performed at the same relative intensity as the exercise training sessions, allowing us to directly extrapolate data collected in these tests to reflect the physiological adaptations occurring during the training sessions.

Previous studies indicate that low GI diets may enhance weight loss (27,28). This study combines exercise training with either a low GI or high GI diet. We hereby show that when energy balance is equal between groups undergoing LoGIX vs. HiGIX intervention, body composition is not differentially influenced between groups. While no group divergence in body composition was found, we did find group differences in skeletal muscle ectopic lipid accumulation. It appears that EMCL stores may be sensitive to the GI of the diet as indicated by our correlations between EMCL and substrate oxidation rates. However, we must acknowledge that these correlation data are underpowered and, given the unequal group distribution, should only be interpreted as a preliminary observation that must be verified in a larger cohort and by more detailed mechanistic studies. With reference to changes in resting and insulin-stimulated metabolism, it was found that following the intervention both groups demonstrated equal decreases in resting RERs and equal increases in metabolic flexibility. Decreased RER at rest and increased metabolic flexibility has been reported following exercise and diet-induced weight loss in obese subjects, by our group and others (31,33). We confirm this finding and additionally show that dietary GI does not influence these trends. However, during submaximal exercise, substrate metabolism was differentially altered between groups, but this could not be expected to extrapolate to a between-group difference in weight loss because we matched between-group energy balance during the study.

Following this exercise training intervention, we found a marked increase in caloric EE during exercise in both groups. This is primarily driven by the higher absolute workload in the postintervention test (performed at the same percentage of VO₂max). However, independent of the increase in EE, there was an interesting divergence between the dietary groups with respect to substrate utilization. The RER was unchanged in subjects who had consumed a low GI diet throughout the intervention, whereas RER was increased during submaximal exercise in subjects consuming a high GI diet. Further to this, carbohydrate oxidation rates during exercise...
corrected for weight loss (mg kg\(^{-1}\) min\(^{-1}\)) were increased in both groups, whereas fat oxidation rates (mg kg\(^{-1}\) min\(^{-1}\)) were increased only in subjects consuming a low GI diet. When expressed as a percentage of total EE during exercise, no change in the percentage of calories derived from carbohydrate or fat oxidation was seen in the LoGIX group, whereas in the HiGIX group energy derived from carbohydrate was increased and energy from fat was decreased. In summary, this indicates that in older obese individuals undergoing diet and exercise-induced weight loss, a high GI diet actually impairs fat oxidation during exercise training sessions such that reduced fat oxidation occurs. These divergent group effects are perhaps a result of the intervention effects upon the compensatory hyperinsulinemia present in our obese cohort: we previously reported that following this intervention only the LoGIX group showed significant reduction in hyperinsulinemia [reported in (8)]. Therefore, the underlying circulatory insulin levels may have been higher during submaximal exercise tests in the HiGIX group, thus suppressing lipolysis to a greater degree than the LoGIX group, limiting free fatty acid availability for oxidation. This hypothesis cannot be tested in this study as no blood/muscle samples were collected during the exercise bout, but a previous study by Wee et al. strongly supports this interpretation (29).

In summary, these novel data further highlight the importance of dietary intake during exercise and diet-induced weight loss interventions in obese populations. We have shown that while dietary GI does not influence body composition following exercise training in obese individuals, a low GI diet is more favorable with respect to increasing fat oxidation during physical activity. Furthermore, it appears that a high GI diet actually prevents these beneficial exercise training responses. To conclude, while weight loss induced by a combination of diet and exercise may be a good marker of reduced disease progression in obese individuals, the dietary carbohydrate quality employed in such interventions must be carefully considered to address metabolic dysfunction in its entirety.

Acknowledgments

The authors wish to thank the research volunteers for their outstanding dedication and effort, and the nursing staff of the Clinical Research Unit and the staff and students who helped with the implementation of the study and assisted with data collection.

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