One Bout of Exercise Alters Free-Living Postprandial Glycemia in Type 2 Diabetes

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ABSTRACT

OBERLIN, D. J., C. R. MIKUS, M. L. KEARNEY, P. S. HINTON, C. MANRIQUE, H. J. LEIDY, J. A. KANALEY, R. S. RECTOR, and J. P. THYFAULT. One Bout of Exercise Alters Free-Living Postprandial Glycemia in Type 2 Diabetes. *Med. Sci. Sports Exerc.*, Vol. 46, No. 2, pp. 232–238, 2014. **Purpose**: Elevated postprandial glycemic (PPG) excursions are significant risk factors for cardiovascular disease in type 2 diabetes patients. In this study, we tested if and for how many meals a single bout of exercise would reduce PPG responses to subsequent meals in type 2 diabetes (T2D) patients using a continuous glucose monitor system (CGMS). **Methods**: We recruited nine sedentary (<30 min·wk^{-1} of exercise) individuals with T2D (mean ± SD; body mass index = 36.0 ± 1.1 kg·m^{-2}, age = 60.3 ± 1.0 yr, HbA1c = 6.3% ± 0.2%). The subjects consumed a eucaloric diet (51% carbohydrate, 31% fat, and 18% protein) consisting of three meals, identical in composition, for a 2-d period while wearing a continuous glucose monitor system in two different conditions (exercise [EX], one 60-min bout at 60%–75% of heart rate reserve performed before breakfast), vs a sedentary [SED] condition). We quantified 24-h average glucose, PPG area under the curve (AUC; 4-h glucose AUC after meals), and PPG-2 h (2 h postprandial glucose). **Results**: EX significantly reduced average [glucose] during the first 24-h period ($P = 0.03$). EX caused a reduction in PPG-AUC ($P = 0.02$) for all of the meals during the 2 d (main effect between conditions). A comparison between the EX and the SED conditions at each meal revealed that EX reduced PPG-AUC after the second meal of day 1 (lunch) ($P = 0.04$). PPG-2 h was not significantly different between EX and SED. **Conclusions**: Although a single EX bout does lower 24-h average [glucose], it only significantly lowered PPG-AUC at the second meal after the bout, suggesting that daily exercise may be needed to most effectively improve PPG at the advent of exercise training in T2D patients. **Key Words:** POSTPRANDIAL GLUCOSE, GLYCEMIC CONTROL, EXERCISE, TYPE 2 DIABETES, CONTINUOUS GLUCOSE MONITORING

Hyperglycemia is linked to increased risk for cardiovascular disease (CVD) as well as all-cause mortality; thus, improved glycemic control is a critical target for diabetes management (6,33). Individuals with type 2 diabetes (T2D) are therefore given the goal of maintaining glycated hemoglobin (HbA1c), a measure of long-term glycemic control, less than 7% (2,31,39). However, although HbA1c is a good indicator of average blood glucose levels for several months, it does not necessarily reflect the magnitude of changes in glucose levels for shorter periods of time, such as during the course of a day when meals of varying macronutrient content are consumed (19,26,27). During the course of a day, blood glucose levels can rise and fall several times, depending on frequency of feeding and the type of food that is consumed as well as the level of physical activity (19,26–28,41). Fasting glucose levels, another common indicator of glycemic control, also fails to assess changes in glucose in response to feeding. Therefore, both HbA1c and fasting glucose may not adequately capture changes in blood glucose concentrations experienced over a typical day (19,28,41). Recently, it has been reported that postprandial (or postglucose challenge) glucose excursions (PPG) may be more tightly linked to risk for CVD than HbA1c or fasting glucose levels (6,33). Therefore, therapies that limit the magnitude of PPG should also lower risk for CVD (4,6,7,21,33).

Exercise is a powerful method to improve long-term glycemic control (3,11,14,15,18,30,32,34,35). It has been demonstrated that exercise can 1) act in an acute manner to increase glucose uptake in the absence of insulin and 2) acutely improve insulin-stimulated glucose uptake in skeletal muscle (30). Moreover, exercise transiently improves insulin sensitivity for a prolonged period, suggesting that PPG may also be improved for several meals after one bout of exercise depending on the health status of the population (15,16,22). We have recently shown that 7 d of exercise training improves postprandial glucose levels measured by a continuous glucose monitoring system (CGMS) in free-living

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humans with T2D (24); however, the design did not allow us to determine the residual effects of each exercise bout on PPG. Because of the known effects of exercise to transiently improve skeletal muscle insulin sensitivity, we next questioned if, and for how many subsequent meals (identical in composition and caloric load), one bout of exercise would improve PPG measured by CGMS in free-living individuals with T2D. Importantly, PPG is driven by not just muscle insulin sensitivity but also by hepatic insulin sensitivity, pancreatic β-cell function, and other physiological responses. Although other laboratories have shown reduced concentrations in PPG and postprandial insulin at a specific time points postprandially (2.5 h) (40), the total area under the glucose curve has not been assessed, nor did subjects consume the same foods at each meal after the exercise bout. We hypothesized that a single, morning exercise session would only improve PPG area under the curve (AUC) for meals consumed during the same day (meals 1–3), and PPG-AUC would subsequently return to preexercise levels on day 2 (meals 4–6). Our hypothesis for a 1-d effect was based on 1) previous data showing one bout of exercise may not improve insulin sensitivity for as long as duration in T2D as in health cohorts 2) and that subsequent meals would replete muscle glycogen thus reducing the positive effects of exercise on PPG (9,15,22). If proven correct, this hypothesis would indicate that exercise on a daily basis may be needed to most effectively improve PPG in individuals with T2D, at least in the initial stages of training.

MATERIALS AND METHODS

Subjects. Sedentary and low-activity individuals with T2D were recruited in and around Columbia, MO, who, on average, took fewer than 6000 steps per day (pedometer) and did not participate in any formal exercise program (>30 min of planned exercise two times a week). Pedometers were worn for a 3-d period to measure daily step count. All subjects were nonsmokers with a body mass index between 30 and 42 $kg \cdot m^{-2}$ who did not have orthopedic limitations to exercise safely on a treadmill and stationary bike. They were weight stable (±5%) and had no changes in medication for at least 3 months before entering the study. In addition, the subjects had controlled diabetes with HbA1c < 7.5% with no insulin use and no reported advanced retinopathy or neuropathy. Other exclusion criteria included breakfast skippers, pregnancy, sleep perturbations, night shift workers, people who have recently traveled across more than two time zones, or individuals with irregular daily schedules to avoid odd sleep or eating patterns that may have conflicted with the study design. All women enrolled in the study were postmenopausal. All subjects signed an informed consent, which was approved by the institutional review board of the University of Missouri.

After the consent meeting, the subjects came to the exercise physiology laboratory during the morning after a 10-h fast for measurement of height, weight, blood pressure, fasting blood glucose, glycated hemoglobin (HbA1c), and blood lipids (total cholesterol, LDL, HDL, and triglycerides). HbA1c was analyzed on a Siemens DCA Vantage analyzer using blood drawn in a heparin tube. Blood lipids (cholesterol, triglycerides, HDL, LDL, and total cholesterol) and fasting glucose were measured by a commercial laboratory as done previously (24). Subjects were then given a diet log and a pedometer to measure dietary consumption and daily steps, respectively, for a 3-d period. On another visit, the subjects had their body composition measured via dual-energy x-ray absorptiometry (Hologic QDR 4500A; Hologic, Bedford, MA). The subjects then performed an exercise stress test to determine maximal oxygen consumption ($\dot{V}O_{2peak}$), maximal heart rate, and to screen for any potential cardiac abnormalities via EKG. The exercise stress test was performed on a treadmill using a standardized Bruce protocol as performed previously (24). During the test, respiratory gases were measured by a metabolic cart (ParvoMedics True One 2400 Metabolic Measurement System; ParvoMedics, Sandy, UT), and cardiovascular function was monitored using a 12-lead EKG (Quinton Q-Stress v3.5 Exercise Test Monitor; Quinton Cardiology, Inc./Burdick, Deerfield, WI). The criteria for a maximal test were three of the following: volitional exhaustion, perceived exertion of 17 or greater, respiratory exchange ratio of greater than 1.0, or a leveling off or slight decrease in oxygen consumption. The EKG data from each exercise stress test were reviewed by a cardiologist to ensure that the participants could safely participate in an exercise session. There was a 5- to 15-d washout after the $\dot{V}O_{2peak}$ test before the subjects began the study protocol.

Study design. The study incorporated a randomized crossover design and consisted of measuring minute-to-minute interstitial glucose concentrations by CGMS during a 2-d sedentary condition (subjects maintained normal daily activities) and another 2-d condition in which the subjects exercised during the morning of the first day (followed by maintaining normal daily activities). Therefore, subjects served as their own controls. We have previously used CGMS to measure glycemic control in free-living subjects who are healthy as well as those with T2D (24,25). During each condition, the subjects consumed the study diet, which consisted of three meals a day representing breakfast (8:00 a.m.), lunch (1:00 p.m.), and dinner (6:00 p.m.) for 4 d. The first 2 d of the diet were to acclimate the subject to the new diet. The following 2 d of the standard diet coincided with CGMS measurements to ensure that any changes to glycemic control were due to exercise and not due to dietary changes. Figure 1 shows the 4-d study period, which the subjects repeated twice, once performing an exercise (EX) bout in the morning before breakfast and once while remaining sedentary (SED). During the SED condition, the subjects continued their typical (sedentary) physical activity, which was verified using a Walk4Life Duo pedometer (Walk4Life Inc., Plainfield, IL) and an accelerometer (BodyMedia SenseWear armband body monitoring system; BodyMedia Inc., Pittsburgh, PA). Both pedometer and accelerometer were used primarily to

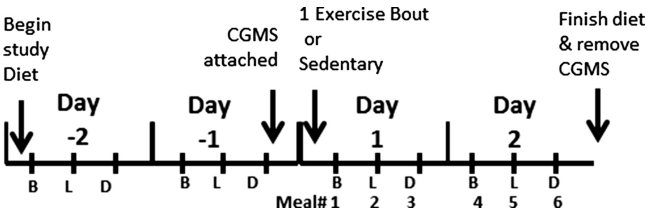

FIGURE 1—Study design for the exercise (EX) versus sedentary (SED) phases of the study. Subjects repeated two separate phases in which they wore CGMS and consumed provided meals three times per day. In both phases, CGMS was placed on the evening of the second wash-in day. On the morning of day 1, subjects either performed EX or remained SED. Glucose levels were measured by CGMS, and three prepared meals were consumed per day (six meals total) during the next 2 d.

assess physical activity within subjects, between conditions. Validation studies on the accuracy of SenseWear to measure energy expenditure have provided mixed results (12,17); however, the device allowed a basis of comparison between the two conditions and showed that exercise increased energy expenditure. There was a 5- to 15-d washout period between the two conditions of the study.

Exercise testing and exercise session. Graded exercise tests to measure $\dot{V}O_{2peak}$ were performed on a treadmill using a Bruce Protocol as done previously (23). The EX bout consisted of 60 min of aerobic exercise broken into three 20-min sections starting at approximately 6:30 a.m. This included 20 min on a treadmill, 20 min on a stationary cycle, and another 20 min on a treadmill. The exercise session was broken into these segments because the subjects could not do 60 min on the treadmill at this intensity without needing to sit down. The exercise intensity was within 5 beats per minute of 60% of HRR for the duration of the exercise bout (as determined from a previous graded exercise stress test). Intensity was controlled during the exercise session by adjusting speed or grade on the treadmill or adjusting resistance on the stationary cycle, to maintain the target heart rate throughout the exercise bout. The exercise prescription falls in line with the dual recommendation of 150 $min \cdot wk^{-1}$ at an intensity of 40%–60% $\dot{V}O_{2peak}$ for patients with T2D by the American Diabetes Association and the American College of Sports Medicine (1). In addition, the same exercise prescription was used in a previous study from our laboratory, which showed a decrease in PPG during 5–7 d of exercise training in previously SED subjects with T2D (24). Subjects were instructed to take their medications as prescribed throughout both conditions and were monitored postexercise to avoid hypoglycemia. Thus, in this study, we wanted to determine whether and for how many meals one bout of exercise prescribed at the same intensity and duration would improve postprandial glycemic (PPG) responses to subsequent meals.

Study diet. As stated previously, the subjects consumed the same study diet in both phases (Table 1). The diet was prepared and packed out by study staff. The subjects were instructed to eat the meals at the same times each day and to allow 5 h between meals. Every meal had the same nutrient composition and caloric content and contained the exact same food items prepared as either breakfast, lunch, or dinner. Breakfast was a potato hash with seasoned ground beef topped with salsa and cheese served with buttered toast, applesauce, and a juice drink. Lunch was mini cheeseburgers with salsa mixed into the patties and baked french fries served with a side of applesauce and a juice drink. Dinner was a mini-meatloaf with salsa and cheese baked in and mashed potatoes served with garlic toast, applesauce, and a juice drink. Subject were instructed to leave uneaten foods in containers when returning them to the laboratory, allowing us to asses food compliance to a certain degree. True compliance to the diet was not directly assessed as we did not monitor them eating the meals directly.

The macronutrient distribution based on energy content was 51.4% carbohydrate, 30.9% fat, and 17.8% protein for the total energy content for each meal (Table 1). The glycemic load of each meal was approximately 55. The daily energy requirement for each subject was estimated using the Harris–Benedict equation and also quantified using a 3-d dietary record completed by the subject. The 3-d mean was then averaged with the Harris–Benedict estimate to determine individual energy requirements and to avoid deficits due to underreporting in the dietary records. From this information, the subjects were provided a diet containing 1600, 1800, 2000, 2200, or 2400 $kcal \cdot d^{-1}$, whichever kilocalorie level was within 100 kcal of their predicted requirements. For example, if a person was estimated at 2063 kcal, they would receive the 2000-kcal diet while an individual estimated at 2115 would receive a 2200-kcal diet. The diet was designed to simulate a typical American diet and provide consistent diet composition between meals and across subjects; it was not meant to serve as an intervention, although it did differ from their normal routine.

The subjects were given a log sheet to track when they ate their meals. They were instructed to eat all of the portions within a 15- to 20-min time frame. Subjects also noted when they went to bed at night and when they got up in the morning. No additional calories were given to replace those lost in the exercise session, which created a deficit. Although this is a known confounder, replacing the calories would have lead to a mismatch of macronutrients between conditions, which would have also been a confounding variable. Therefore, the meals were kept constant through both conditions.

PPG control. A Medtronic iPro CGMS (Minneapolis, MN) monitor was attached to the subject's abdomen with a probe inserted beneath the skin, and the monitor was attached and taped down with Smith & Nephew IV3000 adhesive pads the night before the first measurement day. The CGMS was then worn for two consecutive days. While the CGMS was worn, the subjects recorded four daily blood glucose levels with an Accu-Chek Compact Plus glucometer, which were then used to calibrate the CGMS. We used the CGMS glucose concentration data to quantify the 24-h average glucose for each day in each condition. We also used the glucose concentrations along with subject-recorded meal times to calculate the PPG-AUC for each of the six meals (quantified as the 4-h glucose AUC response after each meal). Blood glucose values

TABLE 1. Foods and quantities in the study diet for a 2000-kcal·d^{-1} diet.

Food Item	Amount in Meal (g)	Kilocalories	CHO (g)	Fat (g)	Protein (g)
White sandwich bread	52.00	137.00	28.00	1.00	4.00
Potatoes	140.00	105.95	24.59	0.00	1.89
Salsa	33.00	8.00	2.00	0.00	0.00
Ground beef, 93/7	101.00	147.89	0.00	7.21	20.74
Salted butter, light	14.00	45.00	0.00	5.00	0.00
Olive oil, extra virgin	4.75	42.00	0.00	4.67	0.00
Applesauce	98.00	68.44	17.11	0.00	0.00
Cheese, medium cheddar	14.00	57.00	0.00	5.00	3.00
Fruit juice	125.00	56.00	14.00	0.00	0.00
Total	581.75	667.28	85.70	22.88	29.63

The table shows the amount of each food item in one meal for the study diet at the 2000-kcal level. The amount of food was adjusted for each calorie level to achieve 200-kcal differences.

recorded every 15 min during the 4-h period were used to calculate total AUC. We also quantified 2-h postprandial glucose concentration for each of the six meals (PPG-2 h) because it is a strong predictor of cardiovascular events (6).

Physical activity monitoring. Subjects wore pedometers and BodyMedia SenseWear Pro II arm band accelerometers during both phases of the study. Data from the accelerometers (estimated energy expenditure) and pedometers (daily steps) were collected.

Statistical analysis. Statistical analysis was analyzed using the Statistical Package for the Social Sciences (version 15; IBM, Armonk, NY) on PPG-AUC, PPG-2 h, average 24-h glucose concentration, estimated caloric expenditure from accelerometer, and daily steps using a two-way repeated-measures ANOVA. The two levels were condition (EX or SED) and meal (1–6) for all except the average 24-h glucose, steps, and energy expenditure, for which the two levels were condition and day (1 or 2). *Post hoc* analyses were conducted using Fisher least significant difference. The level of statistical significance was set at a P value of 0.05, and the data are reported as means ± SE.

RESULTS

Nine subjects completed the study. Baseline anthropometric characteristics, energy intake, blood chemistry, and aerobic fitness data are shown in Table 2. Medication usage for the subjects were as follows: 6/9 were taking metformin (biguanides), 9/9 were taking statins, 6/9 were taking ACE inhibitors, 1/9 were taking angiotensin receptor blockers, and 3/9 were taking diuretics.

Figure 1 depicts the order of events for the study. The exercise sessions were all supervised and were performed at an intensity of 60% HRR (similar to 60% $\dot{V}O_{2peak}$) for 60 min. All subjects performed 60 min of exercise at an average percent heart rate reserve of 58.1±5.5. Table 3 shows that the subjects had a statistically significant increase in both steps and energy expenditure on EX day.

PPG control. EX lowered average glucose concentration significantly during the first 24-h period compared with SED (EX: 5.98 ± 0.0.49 vs SED: 6.62 ± 0.73 mmol·L^{-1}; P = 0.038), but differences were not observed in day 2 (EX: 6.59 ± 0.78 vs SED: 6.62 ± 0.53 mmol·L^{-1}; P = 0.3). Figure 2 shows the average glucose responses measured every 5 min by CGMS during the entire first 24 h of EX or SED conditions, demonstrating lower circulating glucose levels in the EX versus SED condition.

Repeated-measures ANOVA revealed a main effect of EX to lower PPG-AUC across all six meals compared with SED (P = 0.015) (Fig. 3A); this lower PPG-AUC was primarily due to the effects seen during day 1. The *post hoc* analysis between EX and SED at each of the six meals revealed that only meal 2 was significantly lower in the EX compared with SED condition (14% lower; P = 0.04) (Fig. 3A). Although other meals showed trends to be lower in the EX versus SED condition (meal 1 EX is 15% lower than SED, P = 0.08; meal 3 EX is 12.8% lower than SED, P = 0.11; meal 6 EX is 19% lower than SED, P = 0.16), they were not statistically different. In addition, there was also a main effect for the 2-h PPG to be lower during the exercise compared with the SED condition (P = 0.005) (Fig. 3B). However, none of the meals were different between EX and SED after *post hoc* analysis.

DISCUSSION

The current study determined if and for how many subsequent meals one bout of exercise improved PPG in individuals with T2D. Importantly, this was the first study that fed six controlled meals (same type and energy content of food at each meal) during a 2-d period, allowing us to ensure that EX-induced differences in PPG were not affected by type or quantity of food. We found that a single bout of

TABLE 2. Anthropometric characteristics of the subjects.

Age (yr)	60.3 ± 1.0
Sex	5 females/4 males
Body mass index (kg·m^{-2})	36.0 ± 1.1
Body fat (%)	39.6 ± 1.9
Total cholesterol (mg·dL^{-1})	172.9 ± 15.8
LDL (mg·dL^{-1})	88.8 ± 12.6
HDL (mg·dL^{-1})	48.1 ± 2.7
Triglycerides (mg·dL^{-1})	179.7 ± 23.6
Fasting glucose (mmol·L^{-1})	6.5 ± 0.6
Hemoglobin A1c (%)	6.3 ± 0.2
Relative $\dot{V}O_{2peak}$ (mL·kg^{-1}·min^{-1})	20.4 ± 0.7
Average daily caloric intake (self-reported, kcal)	1937.8 ± 176.5

Baseline characteristics including lipid profile, glucose, and hemoglobin A1c results, metabolic cart readouts from an exercise stress test, and the average daily caloric intake of the subjects for the study participants are shown. Data are shown as means ± SE.

TABLE 3. Physical activity levels: average daily steps and energy expenditure during EX and SED conditions.

Activity Data		
Variable	**SED**	**Exercised**
Accelerometer energy expenditure (kcal·d^{-1})	2343.0 ± 148.2	2659.2 ± 50.7*
Pedometer steps	3621.1 ± 391.3	5489.5 ± 845.0

Data are shown as means ± SE.
*Statistically significant difference ($P < 0.05$).

exercise significantly lowered PPG-AUC responses to the second meal after the exercise bout by 15%, whereas a similar but nonsignificant effect was also shown at the first (15%) and third (12%) meal to also be lowered. These changes in PPG resulted in a significant 10% reduction in average blood glucose concentrations among the subjects during the first 24 h after the exercise session, similar to findings by other groups (13,20); however, the exercise-induced effect on the average 24-h glucose concentration was no longer present on day 2. Because the effects of exercise were undetectable by the second day, these data would suggest that exercise should be performed every day to have an optimal effect on postprandial glucose responses at least at the initiation of an exercise program for individuals with T2D. Of note, a previous study from van Dijk et al. (40) did find that that one 60-min bout of exercise improved 24-h glucose values on the second day, but key differences between the studies are noted in the following paragraphs.

A recent study by van Dijk et al. (40) showed that a single exercise bout (30 min) each day was sufficient to lower the 2.5-h glucose concentration after breakfast in individuals with T2D. The same study also showed that one 60-min exercise bout lowered post 2.5-h breakfast glucose on both the day of the bout and the following morning. Thus, they showed that one exercise bout that was the same volume of ours did affect postprandial glucose values on the second day. Key differences exist between the studies, which may have affected the outcomes. First, as already mentioned, our study used controlled meals consisting of the same foods and energy content for each meal limiting the effect that macronutrient content or caloric loads could have altered glycemic responses. Differences in the glycemic control of subjects could have also affected the results. Our T2D patients had good glycemic control (HbA1c = 6.3%) compared with the subjects in the van Dijk et al. (40) study (HbA1c = 7.2%), suggesting that our subjects may have not had as much room for improvement. In addition, our subjects were primarily only taking metformin as a glucose-lowering medication, whereas the study from van Dijk et al. (40) used some individuals who were on metformin and some who were also receiving insulin therapy, which may have positively interacted with exercise responses and provided even greater improvements in glycemia. Finally, the timing of the exercise bout could have also affected the results. Our subjects performed an early morning exercise bout and then consumed meal 1 approximately 2 h later, whereas the van Dijk et al. study performed exercise after breakfast, leaving only two meals to be consumed during the postexercise period of day 1.

Although the current study demonstrates that a single exercise bout transiently improves PPG, it is not known whether prolonged exercise training would result in more persistent effects in type 2 diabetics after each exercise bout. In an acute training study, the effects observed are most likely either due to a change in energy homeostasis or directly due to skeletal muscle contractions, which result in a period of enhanced insulin sensitivity that has been documented to last for up to 48 h (38). However, after prolonged exercise training, many other adaptations may contribute to witnessed improvements in glycemic control (8), such as increased GLUT-4 content (10), increased capillary density in skeletal muscle and improved vascular function (29), and altered body composition (8). Although these adaptations may allow a greater volume of glucose to be taken up by the working tissue, it is unknown whether they significantly change the duration of improvement in skeletal muscle insulin sensitivity and glucose uptake, resulting in prolonged reductions in PPG after an exercise bout. Furthermore, exercise training–induced adaptations that improve control of hepatic glucose output (18) and pancreatic β-cell function (36) could also play a role in potentiating the lasting effect of an exercise bout on glycemic control during subsequent postprandial periods. These concepts deserve further study.

In the current study, we attempted to control as many variables as possible without keeping subjects in a laboratory setting for the duration of the study. The number of steps per day and estimated caloric expenditure were both monitored

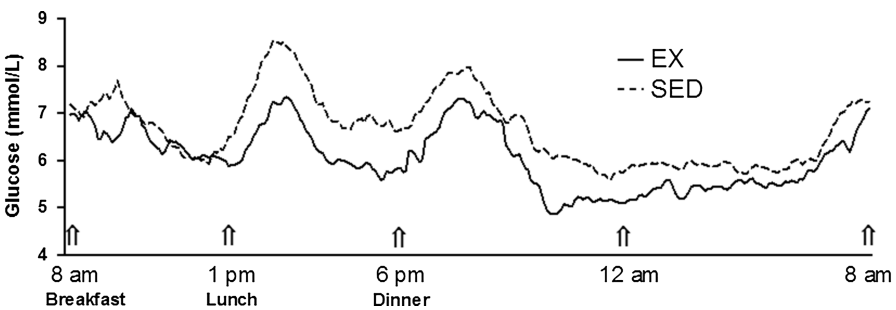

FIGURE 2—Glucose measured every 5 min by CGMS during the first 24-h period. Time points for breakfast (8 a.m.), lunch (1 p.m.), and dinner (6 p.m.) are approximate times when subjects consumed the three meals.

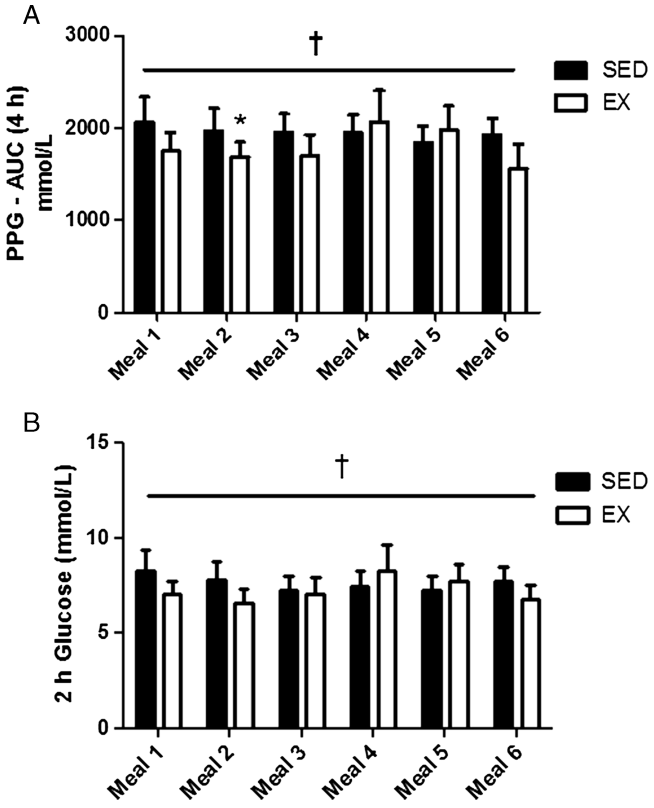

FIGURE 3—A. Postprandial glucose area under the curve (PPG-AUC) responses to six meals under EX and SED conditions: †main effect of EX to lower PPG-AUC ($P = 0.015$) across all meals compared with SED; *EX significantly lowered PPG-AUC response at meal 2 ($P = 0.04$) compared with SED. Data are shown as means ± SE. B. PPG-2 h responses to six meals after EX versus SED conditions: †main effect of EX to lower PPG-AUC ($P = 0.005$) across all meals. Data are shown as means ± SE.

throughout the exercise and SED conditions. The significant difference in steps per day and the estimated caloric expenditure between the first day of the exercised (including the exercise session) and the first day of the SED condition confirm that the subjects did not reduce steps or activity after the exercise bout to compensate for the exercise session in the morning. As previously stated, the diet provided in this study maintained the same food items at every meal, reducing the likelihood of differences in macronutrient content or glycemic load affecting the changes seen in glycemic control. This is a unique feature of this study, and we are not aware of a previous study utilizing this approach. Further, because the diet was provided and constant throughout the study, the observed changes that were seen in glycemic control were most likely due to the increased physical activity from the exercise session. However, a limitation of controlling the diet was that the subjects were not refed calories to compensate for the energy expended during the exercise bout. This confounds whether the effects seen in the current study were effects of muscle contraction, energy deficit, or a combination of both (5,37).

In conclusion, the current study demonstrated that a single exercise bout is sufficient to reduce 24-h average blood glucose concentrations during the first day (day of the exercise bout) as well as PPG-AUC for a single meal (meal 2) in individuals with T2D. This study confirms other recent findings that have shown a single bout of endurance exercise or short-duration, high-intensity exercise reduces average blood glucose concentrations in individuals with T2D for 24 h and also reduces postprandial glucose concentrations (13,40). Our findings suggest that for optimal blood glucose control, it is important for individuals with T2D to perform exercise on a daily basis, particularly at the onset of exercise training.

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The results of the current study do not constitute endorsement by the American College of Sports Medicine.

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The authors have no conflicts of interest to report.

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