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ARTICLE

Effects of high-intensity interval exercise versus continuous moderate-intensity exercise on postprandial glycemic control assessed by continuous glucose monitoring in obese adults

Jonathan P. Little, Mary E. Jung, Amy E. Wright, Wendi Wright, and Ralph J.F. Manders

Abstract: The purpose of this study was to examine the impact of acute high-intensity interval training (HIIT) compared with continuous moderate-intensity (CMI) exercise on postprandial hyperglycemia in overweight or obese adults. Ten inactive, overweight or obese adults (41 ± 11 yrs, BMI = 36 ± 7 kg/m^2) performed an acute bout of HIIT (10 × 1 min at approximately 90% peak heart rate (HR_{peak}) with 1-min recovery periods) or matched work CMI (30 min at approximately 65% HR_{peak}) in a randomized, counterbalanced fashion. Exercise was performed 2 h after breakfast, and glucose control was assessed by continuous glucose monitoring under standardized dietary conditions over 24 h. Postprandial glucose (PPG) responses to lunch, dinner, and the following day's breakfast were analyzed and compared with a no-exercise control day. Exercise did not affect the PPG responses to lunch, but performing both HIIT and CMI in the morning significantly reduced the PPG incremental area under the curve (AUC) following dinner when compared with control (HIIT = 110 ± 35, CMI = 125 ± 34, control = 162 ± 46 mmol/L × 2 h, $p < 0.05$). The PPG AUC (HIIT = 125 ± 53, CMI = 186 ± 55, control = 194 ± 96 mmol/L × 2 h) and the PPG spike (HIIT = Δ2.1 ± 0.9, CMI = Δ3.0 ± 0.9, control = Δ3.0 ± 1.5 mmol/l) following breakfast on the following day were significantly lower following HIIT compared with both CMI and control ($p < 0.05$). Absolute AUC and absolute glucose spikes were not different between HIIT, CMI, or control for any meal ($p > 0.05$ for all). We conclude that a single session of HIIT has greater and more lasting effects on reducing incremental PPG when compared with CMI.

Key words: exercise intensity, exercise metabolism, insulin resistance, obesity, carbohydrate metabolism.

Résumé : Cette étude se propose d'analyser l'effet d'une séance d'entraînement par intervalle de haute intensité (HIIT) comparativement à un exercice continu d'intensité modérée (CMI) sur l'hyperglycémie postprandiale chez des adultes en surpoids et obèses. Dix adultes inactifs en surpoids ou obèses (41 ± 11 ans, IMC = 36 ± 7 kg/m^2) participent à une séance HIIT (10 × 1-min à environ 90 % fréquence cardiaque de pointe (HR_{pointe})) suivie de 1 minute de récupération ou à une séance CMI appariée (30 min à environ 65 % HR_{pointe}) selon un ordre aléatoire contrebalancé. Les sujets participent à l'exercice 2 h après le petit déjeuner et on effectue un contrôle de la glycémie en continu dans des conditions strictes d'alimentation sur une période de 24 h. On détermine la concentration postprandiale de glucose (PPG) au dîner, au souper et au petit déjeuner du lendemain et on compare les résultats à la condition de contrôle, soit une journée sans exercice. L'exercice ne modifie pas la PPG au dîner, mais les séances HIIT et CMI réalisées le matin suscitent une diminution significative de l'accroissement de la surface sous la courbe (AUC) de PPG après le souper comparativement à la condition de contrôle (HIIT = 110 ± 35, CMI = 125 ± 34, contrôle = 162 ± 46 mmol/L × 2 h, $p < 0,05$). L'AUC de la PPG (HIIT = 125 ± 53, CMI = 186 ± 55, contrôle = 194 ± 96 mmol/L × 2 h) et la pointe de PPG (HIIT = Δ 2,1 ± 0,9, CMI = Δ 3,0 ± 0,9, contrôle = Δ 3,0 ± 1,5 mmol/L) après le déjeuner le jour suivant sont significativement plus faibles à la suite de HIIT comparativement à CMI et à la condition de contrôle ($p < 0,05$). On n'observe pas de différences des AUC absolues et des pointes absolues de glucose entre les trois conditions (HIIT, CMI, contrôle) à tous les repas ($p > 0,05$). En conclusion, une seule séance HIIT produit un effet meilleur et plus durable sur la diminution de l'accroissement du PPG comparativement à une séance CMI. [Traduit par la Rédaction]

Mots-clés : intensité d'exercice, métabolisme à l'effort, insulinorésistance, obésité, métabolisme des sucres.

Introduction

Postprandial hyperglycemia is an independent risk factor for cardiovascular disease and mortality in individuals with (Cavalot et al. 2006, 2011) and without type 2 diabetes (T2D) (Lin et al. 2009). Exercise improves glycemic regulation (Roberts et al. 2013), an effect that can be attributed, at least in part, to the acute increase in skeletal muscle insulin sensitivity that persists for approximately 24–48 h following each bout (Newsom et al. 2013). Although certainly important, assessments of insulin sensitivity in response to exercise do not necessarily provide information about glucose control, particularly on the occurrence of postprandial hyperglycemia or the real-life glycemic response to meals consumed throughout the day (Roberts et al. 2013). The emergence of continuous glucose monitoring (CGM) technology has allowed researchers to characterize the influence of acute exercise on glucose regulation under free-living conditions (i.e., postprandial hyperglycemic excursions in response to actual meals) (Manders et al. 2010; Mikus et al. 2012*a*; van Dijk et al. 2012). CGM provides novel information on the direction and magnitude of glycemic excur-

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sions in response to mixed meals and therefore can provide unique insight into postprandial glycemic responses over the course of the day under free-living conditions (Monnier and Colette 2008).

Assessing glucose control following meals in free-living conditions is particularly important, because recent studies demonstrate that common laboratory methods such as oral glucose tolerance testing may not accurately reflect the impact of physical activity on glycemic regulation (Mikus et al. 2011, 2012*a*, 2012*b*). Glucose control assessed by CGM is improved over 24 h following acute resistance and continuous moderate-intensity (CMI) exercise in individuals with T2D (van Dijk et al. 2012). Studies examining the impact of exercise on free-living glucose control using CGM in individuals without T2D are sparse, but van Dijk et al. (2012) recently reported that a single session of both CMI exercise and resistance exercise improved glycemic regulation in participants with impaired glucose tolerance (i.e., prediabetes). Thus, CGM appears to be a valuable tool to assess the impact of exercise on glucose control in individuals with, or at risk for, T2D.

High-intensity interval training (HIIT) is a form of exercise that involves short repeated bursts of vigorous exercise interspersed with periods of rest or recovery. A growing body of evidence demonstrates that HIIT may be superior to CMI exercise training for improving a variety of cardiovascular risk factors including cardiorespiratory fitness, endothelial function, and muscle metabolic capacity (Wisløff et al. 2009; Kessler et al. 2012), at least when compared on a matched-work basis. It was recently shown that an acute bout of HIIT reduces postprandial hyperglycemia assessed by CGM in patients with T2D (Gillen et al. 2012). In this study, HIIT was not compared with traditional CMI and therefore it is unclear if HIIT is superior to CMI for acutely improving free-living glucose control. As many of the acute effects of exercise on glycemic regulation are related to enhanced muscle insulin sensitivity and (or) replenishment of muscle glycogen stores (Devlin et al. 1987; Holloszy 2005), HIIT may be particularly effective for improving glucose control because of higher muscle fiber recruitment (Edgett et al. 2013) and (or) greater muscle glycogen depletion (Vollestad and Blom 1985). To our knowledge, the impact of acute HIIT versus CMI on glucose control assessed in free-living conditions by CGM has never been studied.

The primary purpose of this study was to examine postprandial glucose (PPG) control assessed by CGM following an acute bout of HIIT and CMI in overweight or obese, inactive adults. We hypothesized that an acute bout of both types of exercise would improve postprandial hyperglycemia over the subsequent 24 h but that HIIT would lead to greater improvements. Long-term patient compliance and adherence is required to improve glycemic control and to reduce the prevalence of diabetes-related comorbidities (Snowling and Hopkins 2006). Thus, in addition to metabolic benefits, it is also important to assess whether an exercise strategy aimed to improve health elicits psychological responses that are favourable and might encourage adoption. Our secondary aim was to assess psychological factors associated with exercise adherence, including affect, enjoyment, and preference for HIIT and CMI. To our knowledge, these psychological responses to HIIT versus CMI have not been previously reported in overweight or obese, inactive adults.

Materials and methods

Participants

Ten overweight or obese individuals (body mass index (BMI) >25 kg/m^2, 8 females and 2 males) who were inactive (<2 bouts of exercise lasting a minimum of 30 min per bout per week) were recruited through poster advertisements and online community message boards. Participant characteristics are provided in Table 1. Six participants had impaired fasting glucose (≥5.6 mmol/L) based on American Diabetes Association (ADA) criteria (ADA 2010). All females were premenopausal. The study protocol was approved by the University of British Columbia Clinical Research Ethics Board, and all participants provided written informed consent.

Table 1. Descriptive characteristics of participants.

Characteristic	Data
Sex (M/F)	2/8
Age (yr)	40.6±10.7
Height (m)	165.7±6.2
Body mass (kg)	99.6±20.0
BMI (kg/m^2)	36.1±7.1
Fasting glucose (mmol/L)	5.6±0.8
Peak power output (W_{peak}, Watts)	162.6±9.6
Estimated maximal oxygen uptake ($\dot{V}O_{2peak}$, ml/kg/min)	22.3±2.3

Note: $\dot{V}O_{2peak}$ was estimated based on Storer et al. (1990). Values are means ± SD (n = 10).

Baseline testing and familiarization

Approximately 1 week prior to experimental trials, participants reported to the laboratory for fitness testing and familiarization. Height and weight were measured using a stadiometer and physician's scale (Seca 700, Hamburg, Germany). A ramp increase (15 W/min) maximal exercise test to volitional exhaustion was completed on a cycle ergometer (Monark 839E, Sweden) with heart rate (HR) recorded continuously through a chest strap integrated with the ergometer software (Polar T31, Polar Electro, Kempele, Finland). Participants were provided verbal encouragement throughout the test and peak HR (HR_{peak}) and peak power output (W_{peak}) were recorded. Seat height was recorded and kept constant for all subsequent tests. Following a 15–20 min rest period, participants performed a 3-min familiarization with both CMI and HIIT exercise. CMI was performed at 35% W_{peak} (Manders et al. 2010), which was determined during pilot testing to elicit approximately 65% HR_{peak} and thus consistent with moderate-intensity exercise described in American College of Sports Medicine physical activity guidelines (Garber et al. 2011). HIIT was modeled after previously published work (Gillen et al. 2012; Little et al. 2011) and consisted of 1-min intervals of vigorous intensity at approximately 90% W_{peak} separated by a 1-min rest at 15% W_{peak}. Participants were allowed to adjust the workload of the vigorous intervals up or down by 2.5%–10% W_{peak} during this familiarization, and the CMI workload was adjusted accordingly to maintain matching of external work for the subsequent experimental trials. Based on our pilot work, these workloads for CMI and HIIT elicit approximately 65% HR_{peak} and approximately 90% HR_{peak}, respectively. During this visit, participants also selected foods to be included for experimental trials, with 3 predetermined energy and macronutrient matched options for breakfast, lunch, dinner, and snacks provided, therefore ensuring standardized dietary intake during the experimental period.

Experimental trials

All participants completed two 3-day experimental testing periods in a randomized, counterbalanced fashion separated by 7 days. Participants reported to the laboratory after an overnight fast and a continuous glucose monitor (CGM; Dexcom Seven Plus, San Diego, CA, USA) was inserted approximately 5 cm to the left or right of the umbilicus. The Dexcom Seven Plus measures interstitial glucose concentration via a glucose oxidase sensor implanted into the subcutaneous adipose tissue. Data are transmitted to a receiver that displays real-time blood glucose values, which are calculated using an algorithm calibrated to finger stick blood glucose values. The receiver records and stores blood glucose values as 5-min averages. The receiver must remain within 1.5 m of the monitor for communication so participants wore the pager-size receiving device on their waistband and were instructed to leave it by their bedside while sleeping. According to the manufacturer instructions, the sensor was allowed 2 h to initialize before the

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first calibration involving the input of 2 finger stick blood glucose samples (OneTouch UltraMini, LifeScan, Zug, Switzerland) into the receiver. During the initialization period, a standardized breakfast providing 25 kJ/kg (instant oatmeal, blueberry muffin, and orange juice; 70% energy from carbohydrate, 20% energy from fat, 10% energy from protein) was provided. Participants were then provided detailed instructions and training on how to obtain their own finger stick samples, which were collected twice per day (upon awakening and prior to going to bed) and input into the CGM receiver for calibration, as per manufacturer instructions. Individualized control diets, with energy and macronutrient intake based on sex, height, weight, and activity level (Health Canada 2010) were provided to participants based on their selections from the previous visit. All meals, snacks, and beverages were provided for a 48-h period to ensure fully standardized dietary intake during the experimental periods, as described previously (Manders et al. 2010). Diets provided 121 kJ/kg body mass and consisted of prepackaged foods purchased from a grocery store and preweighed foods prepared by researchers providing 66 ± 4% energy from carbohydrate, 23 ± 2% energy from fat, and 11 ± 2% energy from protein. A food and event logbook was provided with detailed instructions for meal timing, and participants were asked to record the food they ate and whether they deviated from the provided meals and snacks or prescribed times. Participants were also instructed to not engage in any purposeful exercise outside of activities of daily living, which was verified using the logbook.

Upon CGM initialization, participants left the laboratory with their individualized diets. CGM data were collected for the next 48 h with the first 24 h (lunch and dinner, following day breakfast) being a no-exercise control period. On Day 2, participants reported to the laboratory 2 h following breakfast and completed a single session of HIIT or CMI in a randomized, counterbalanced order. HIIT involved 10 × 1 min at 90% W_{peak} separated by 1-min rest periods at 15% W_{peak}. CMI involved 30 min at 35% W_{peak}. The HIIT protocol matched previously published work shown to improve glucose control in patients with T2D (Gillen et al. 2012). We designed the CMI protocol to be exactly matched for external work within each participant and be consistent with typical physical activity guidelines (i.e., 30 min of moderate-intensity exercise on most days of the week) (Garber et al. 2011). Participants then left the laboratory and consumed meals provided to them, which were identical to the previous 24 h. The energy content of the diet was the same on Control and exercise days (i.e., diet was not adjusted for the energy expended during exercise.) The next day (Day 3), participants reported back to the laboratory 2 h following breakfast for removal of the CGM device and data download. In this manner, Day 2 lunch and dinner and Day 3 breakfast provided for a 24 h comparison between HIIT, CMI, and the no-exercise control days.

Exercise measures

HR was measured continuously during exercise and ratings of perceived exertion (RPE) were assessed at set time points throughout exercise (20%, 50%, and 90% session completion) using the Borg 0–10 category/ratio scale (Borg 1982). Affective responses to exercise were assessed in-task during each bout of exercise using the one-item feeling scale, which is a validated measure of generalized affective valence (–5 very bad to +5 very good) (Hardy and Rejeski 1989). Enjoyment immediately after exercise was assessed using a one-item scale from 1 (not at all) to 7 (extremely) that asked: "Use the following scale to indicate how much you enjoyed the exercise session". Post-exercise enjoyment was assessed using the physical activity enjoyment scale, which is an 18-item scale providing an overall score out of 100 related to the overall enjoyment of exercise just experienced (Kendzierski and DeCarlo 1991). Following completion of both types of exercise (i.e., at the end of the study), participants were asked to choose which exercise they preferred.

CGM measures

CGM data were downloaded to a PC with Dexcom Seven Plus software according to the manufacturer's instructions. Data were exported to Excel for initial inspection and analyses and subsequently transferred to GraphPad Prism (Version 6.0a, San Diego, CA, USA) for detailed analyses. Initial data inspection involved verification and identification of meal times using the food diaries completed by participants. CGM data from the Dexcom Seven Plus provides average blood glucose every 5 min throughout the wear period (i.e., 288 values per 24-h period). Missing data were identified and treated according to the following a priori rules. (*i*) If there were <3 5-min periods of missing data (i.e., 15 min) the mean of the blood glucose value before and after was inserted. (*ii*) If there were >3 5-min periods of missing data during waking hours, this day was inspected further. In such instances, if >3 consecutive missing data points occurred during a 2-h postprandial period, this particular post-meal glucose response was omitted from analyses for all conditions for that participant. (*iii*) If >3 5-min periods were missing during sleep, 24-h data were adjusted for this day to omit these timepoints. Mean blood glucose concentration was calculated over 24 h for Control, HIIT, and CMI days using Excel. Mean amplitude of glycemic excursions (MAGE; Service et al. 1970), SD, time spent >8 mmol/L, and time spent <4 mmol/L (Mikus et al. 2012*b*) over 24 h were also calculated using a customized Excel macro as indices of glycemic variability.

Postprandial hyperglycemic responses

Once exported to Prism, 2 h post-meal glucose responses were plotted for each subject for each day. Incremental area under the glucose curve (AUC) and postprandial spike (PPS) in glucose from pre-meal (average of 15 min prior to each meal) to the highest post-meal value were calculated. Pre-meal, 2 h post-meal, and absolute glucose spikes and AUCs were also calculated.

Data analyses and statistics

To initially compare CGM data on the no-exercise control days under identical dietary intake, paired sample *t* tests were performed. The coefficient of variation was calculated using the reproducibility method error technique (Zehr and Sale 1997) to compare values for all variables between the 2 control days. After establishing that there were no significant differences between no-exercise control days for any measure and demonstrating the coefficient of variation <10%, 24 h glycemic responses were averaged for these days and compared with the 24 h following HIIT and CMI. For one participant (female), the CGM device appeared to malfunction during the first 3 h of data collection on the first no-exercise control day and at intermittent periods throughout the first experimental trial, resulting in incomplete data for this participant for the HIIT condition. With this participant removed, the CGM captured 92 ± 7% of all 5-min average glucose values (265 ± 19 of all potential data points per 24 h per participant). The most common time for missing data was at night, which is likely due to the receiver being out-of-range with the implanted monitor while a participant was sleeping. For one participant (female) there were >3 missing data points in a row during a 2-h post-lunch period and this participant's data was removed from the postprandial analyses for this meal. Thus, data are presented for 8 subjects for postprandial glycemic response to lunch and 9 subjects for all other CGM measures. All data were analyzed using IBM SPSS Statistics for Windows, Version 20.0 (IBM Corp. 2011. Armonk, NY, USA). One-way repeated measures ANOVA was performed using general linear models for repeated measures to compare HIIT, CMI, and control day data for CGM variables. For data with a time component (HR, RPE, in-task feeling scale) 2 repeated measures factors (condition × time) were included in the general linear

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model to compare HIIT versus CMI over time. Significant F values were further probed with least significant difference post-hoc tests. Overall exercise enjoyment for HIIT and CMI was analyzed by paired-sample t test. A p value <0.05 was considered statistically significant.

Results

Characteristics of exercise sessions

All participants were able to complete both exercise sessions as prescribed. As expected, HIIT resulted in higher average HR compared with CMI (Table 2).

Effect of exercise on glucose control

All participants consumed the provided diets as prescribed and reported no purposeful physical activity outside of the supervised laboratory sessions. There were no significant differences between glucose responses for any of the calculated parameters between the no-exercise control days, which were completed 7 d apart (all $p > 0.40$, data not shown). Therefore, the average response to both no-exercise control days was used for comparison with HIIT and CMI.

There were no differences between HIIT, CMI, or control for 24 h average blood glucose ($p = 0.11$, Table 3), MAGE ($p = 0.13$, Table 3), SD ($p = 0.22$, Table 3), or the postprandial glycemic response to lunch (i.e., the first meal eaten after the morning exercise sessions; $p = 0.55$ for AUC_{Lunch} and $p = 0.52$ PPS_{Lunch}, Fig. 1 and Table 3, respectively). There were no significant differences in the absolute peak glycemic responses to any of the meals between conditions nor were any differences noted between pre-meal values or 2 h post-meal values (all p values >0.22, Table 3), except the pre-meal glucose concentration before lunch, which was significantly higher in HIIT compared with both CMI and control ($p = 0.02$, Table 3). However, when calculated as the difference from pre-meal values significant differences in the AUC and PPS for dinner and breakfast existed, indicating that exercise affected postprandial glycemic excursions to these meals. Dinner AUC was significantly different between conditions ($p = 0.04$), with post-hoc tests revealing approximately 32% and approximately 22% lower AUC_{Dinner} following both HIIT and CMI compared with control, respectively ($p < 0.05$ for both, Fig. 2). The PPS_{Dinner} was significantly different between conditions ($p = 0.004$). Post-hoc tests revealed that HIIT reduced PPS_{Dinner} by approximately 41% when compared with control and by approximately 39% when compared with CMI ($p < 0.05$ for both), with no difference between CMI and control (Table 3). For the postprandial glycemic response to breakfast on the day following exercise, both $AUC_{Breakfast}$ ($p = 0.001$, Fig. 3) and $PPS_{Breakfast}$ ($p = 0.013$, Table 3) were significantly different between conditions. HIIT resulted in reduced $AUC_{Breakfast}$ compared with control (approximately 36% lower) and CMI (approximately 33% lower, both $p < 0.05$), whereas CMI was not different from the no-exercise control day (Fig. 3). The significant effects of HIIT over control and CMI were also found for $PPS_{Breakfast}$. $PPS_{Breakfast}$ was approximately 30% lower in HIIT compared with both control and CMI (both $p < 0.05$, Table 3). Minutes per day spent above 8 mmol/L glucose were not significantly different ($p = 0.49$) between HIIT (259 ± 338), CMI (208 ± 272), or control (269 ± 344). Minutes per day spent below 4 mmol/L were also not significantly different ($p = 0.11$) between HIIT (22 ± 65), CMI (52 ± 52), or control (83 ± 71).

Psychological responses to exercise

As expected, RPE was higher during HIIT compared with CMI (main effect of condition, $p < 0.01$, Table 2). RPE also increased over time during both HIIT and CMI (main effect of time, $p < 0.01$, data across time not shown). There were no significant differences between HIIT and CMI for exercise affect (i.e., pleasure or displeasure) assessed using the feeling scale (Table 2). Measures of exercise enjoyment were similar for HIIT and CMI (Table 2). In total, 6 participants preferred HIIT, whereas 4 preferred CMI.

Table 2. Heart rate, ratings of perceived exertion, and psychological responses to high-intensity interval training and continuous moderate-intensity exercise.

	HIIT	CMI
Average HR (% maximum)	87.3±6.8*	66.0±6.4
RPE (0–10)	6.1±1.9*	2.6±1.1
External work (kJ)	102±6	102±6
Average feeling scale (–5 to +5)	1.3±2.6	2.8±1.7
Immediate enjoyment (1–7)	5.1±1.4	5.0±1.2
Overall enjoyment (0–100)	87.4±13.8	91.2±13.1
Preference (no. of participants)	6/10	4/10

Note: Values are means ± SD ($N = 10$). *$p < 0.05$ (main effect). (HIIT, high-intensity interval training; CMI, continuous moderate-intensity; HR, heart rate; RPE, rating of perceived exertion.)

Table 3. Mean glucose and postprandial glucose assessments following meals measured by continuous glucose monitoring over 24 h following high-intensity interval training, continuous moderate-intensity exercise, or no-exercise control.

	Control	HIIT	CMI
24 hr average blood glucose	6.5±1.4	7.0±0.9	6.4±1.4
PPS_{Lunch}	2.2±1.3	1.9±1.3	2.7±1.7
PPS_{Dinner}	2.9±0.9	1.7±0.8*,**	2.8±1.4
$PPS_{Breakfast}$	3.0±1.5	2.1±0.9*,**	3.0±0.9
$Pre\text{-}meal_{Lunch}$	5.5±1.3	7.1±0.8*,**	5.7±0.9
$Pre\text{-}meal_{Dinner}$	6.8±2.2	7.2±1.1	6.2±0.6
$Pre\text{-}meal_{Breakfast}$	6.6±1.4	6.8±1.0	6.2±2.0
2 hr PPG_{Lunch}	6.4±2.9	7.2±1.0	6.5±1.6
2 hr PPG_{Dinner}	7.5±2.2	6.9±1.6	7.0±1.2
2 hr $PPG_{Breakfast}$	7.1±0.9	7.1±1.6	6.9±1.7
Abs $spike_{Lunch}$	7.7±2.9	9.0±0.6	8.4±1.6
Abs $spike_{Dinner}$	9.7±1.7	8.9±1.3	9.0±1.6
Abs $spike_{Breakfast}$	9.6±1.6	8.9±1.3	9.2±1.8
Abs AUC_{Lunch} (mmol/L × 2 h)	830±290	1020±100	890±145
Abs AUC_{Dinner}	980±325	925±200	865±140
Abs $AUC_{Breakfast}$	1015±190	1000±175	950±275
MAGE	2.5±0.8	2.6±0.9	1.8±0.4
SD	1.3±0.5	1.0±0.3	1.3±0.4

Note: All values are in mmol/L glucose unless otherwise indicated. *$p < 0.05$ vs. control, **$p < 0.05$ vs. CMI. Values are means ± SD ($n = 9$, except for Lunch variables where $n = 8$). (HIIT, high-intensity interval training; CMI, continuous moderate-intensity; PPS, postprandial spike; PPG, postprandial glucose; Abs, absolute; AUC, area under the curve; MAGE, mean amplitude of glycemic excursions.)

Discussion

The major novel finding in this study was that a single session of HIIT improved postprandial glycemia in overweight or obese adults during the 24 h following a single training session. Furthermore, HIIT was superior to more traditionally prescribed CMI, as the overall effects on postprandial glycemia were greater and lasted into the next day despite the fact that the 2 bouts were matched for total work. These results extend current knowledge on the potential health benefits of HIIT as they suggest this type of exercise is effective for improving glucose control in individuals who may be at elevated risk for T2D.

Although an acute bout of exercise is known to improve insulin sensitivity for approximately 24–48 h into recovery (Newsom et al. 2013), the influence of relative work intensity on free-living glucose control (i.e., PPG responses to actual meals) has received little attention. This is an important distinction because several recent studies have demonstrated that altering physical activity levels leads to significant effects on CGM-derived measures of postprandial glycemia without influencing glycemic responses to a traditional laboratory oral glucose tolerance test (Mikus et al. 2011, 2012*a*, 2012*b*). Manders et al. (2010) reported that 60 min of moderate-intensity exercise at 35% W_{peak} reduced hyperglycemia assessed by CGM in

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Fig. 1. (A) Postprandial glucose responses and (B) area under the glucose curve (AUC) following a standardized lunch meal on a no-exercise control day (control) and on a day when a single bout of high-intensity interval training (HIIT) or continuous moderate-intensity (CMI) exercise was performed. Exercise sessions took place 2 h following a standardized breakfast, approximately 1.5 h prior to lunch. Error bars are omitted in (A) for clarity. Means ± SD are shown in (B).

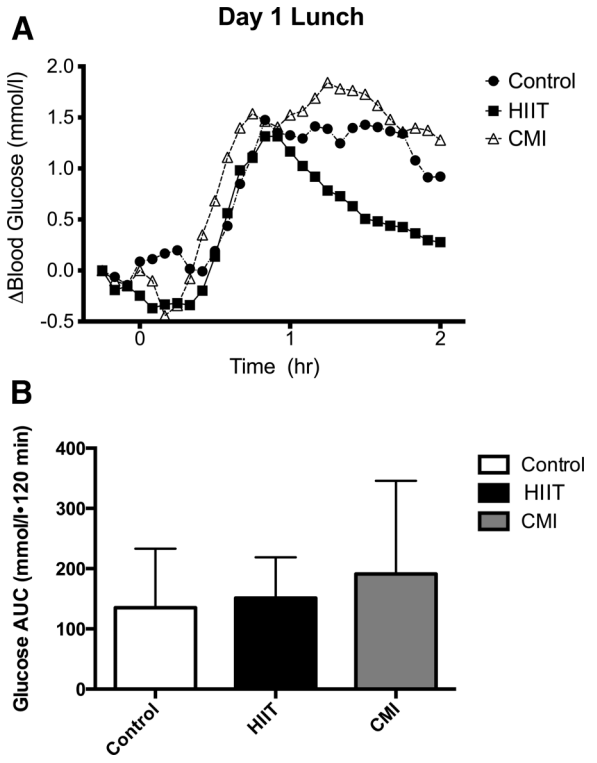
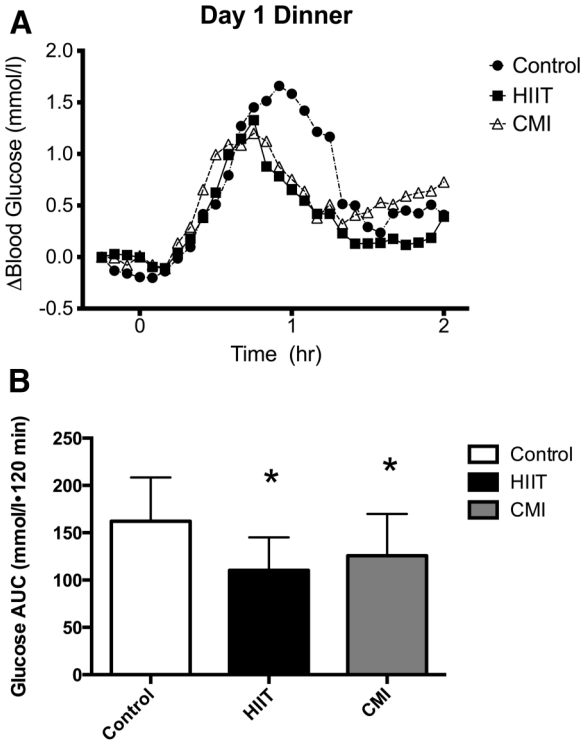


Fig. 2. (A) Postprandial glucose responses and (B) area under the glucose curve (AUC) following a standardized dinner meal on a no-exercise control day (control) and on a day when a single bout of high-intensity interval training (HIIT) or continuous moderate-intensity (CMI) exercise was performed. Error bars are omitted in (A) for clarity. Means ± SD are shown in (B). *$p < 0.05$ vs. control.



T2D patients, whereas 30 min of exercise at 70% W_{peak} had no apparent effect, suggesting that lower exercise intensity (or greater time spent exercising) may be more important for improving glucose control. Our findings are in contrast because HIIT was superior to continuous low-intensity exercise at improving measures of glucose control, despite requiring less total exercise time (20 min for HIIT vs. 30 min for CMI). However, differences in the length of continuous low-intensity exercise, nature of high-intensity exercise, or subject population make it difficult to directly compare our study with Manders et al. (2010).

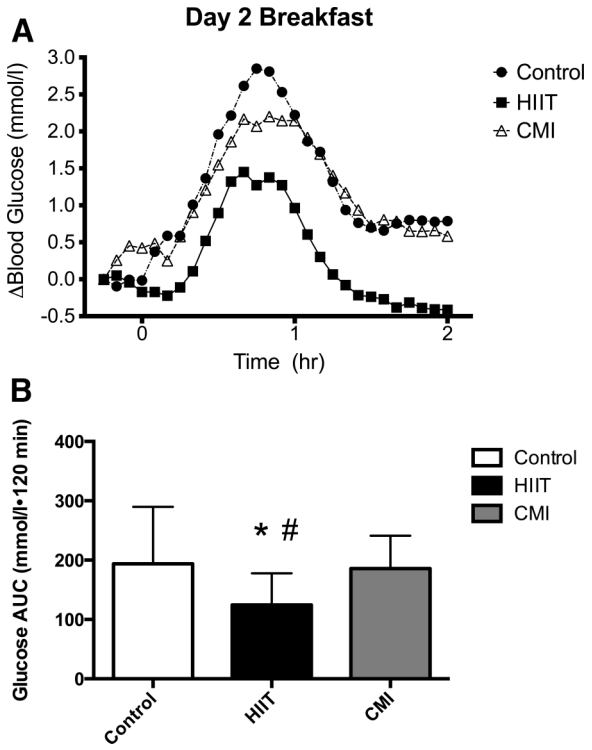
Studies examining the impact of acute exercise on free-living glucose control assessed by CGM in nondiabetic subjects are limited. Both acute endurance and resistance exercise can improve glucose control assessed by CGM in obese subjects at risk for T2D (van Dijk et al. 2012). Our results build upon these findings and show that acute interval exercise can benefit glycemic regulation in overweight or obese subjects. In particular, HIIT reduced post-meal glycemic excursions for up to 24 h following a single session. The current study extends previous research showing that HIIT improves postprandial glycemia in T2D patients (Gillen et al. 2012), by indicating that HIIT may be superior to more traditional CMI exercise. Recent work has also shown that a single session of HIIT is more effective than matched-work continuous exercise for improving postprandial triglyceridemia assessed on the morning following exercise (Trombold et al. 2013). Taken together, these findings indicate that HIIT may have important benefits on PPG and lipid metabolism that extend into the day following exercise. It remains to be determined if repeated sessions of HIIT over time would reduce clinical markers of glucose control and (or) lower overall risk of T2D and cardiovascular disease.

In addition to measuring the impact of HIIT and CMI on glycemic responses, we also aimed to assess several psychological parameters that influence exercise adherence. These measures are important because regardless of the potency of exercise to improve health markers, it is unlikely individuals will adhere to an exercise modality if it is unpleasant or not enjoyable (Ekkekakis et al. 2011). As expected, HR and RPE were significantly higher during HIIT compared with CMI. Using the feeling scale, a validated measure of the pleasure–displeasure experienced during exercise (Hardy and Rejeski 1989), we assessed the in-task affective response to both HIIT and CMI. Previous studies have shown that higher in-task affect is predictive of greater exercise adherence over 6–12 months (Williams et al. 2008). Although there were no statistically significant differences between HIIT and CMI, the mean values during HIIT were on average lower than CMI (Table 2). It is possible that we were underpowered to detect significant differences between conditions. Nonetheless, the mean values in response to HIIT were above zero at all time points, suggesting that HIIT did not elicit feelings of displeasure as is consistently seen during continuous vigorous-intensity exercise (Ekkekakis et al. 2011). Participants reported equal enjoyment for both HIIT and CMI, and 6/10 participants indicated that they preferred HIIT to CMI following completion of both types of exercise. These preliminary findings are intriguing as they suggest that HIIT is effective in improving aspects of glucose control while eliciting high enjoyment and preference in overweight or obese inactive adults despite eliciting higher RPE.

As expected, individual differences exist in the overall psychological responses and preference for HIIT, and further research is needed to determine whether certain characteristics are predictive of individuals who might prefer or enjoy HIIT more. Our sample size is too small to assess these aspects, but interestingly

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Fig. 3. (A) Postprandial glucose responses and (B) area under the glucose curve (AUC) following a standardized breakfast meal on a no-exercise control day (control) and on the day after a single bout of high-intensity interval training (HIIT) or continuous moderate-intensity (CMI) exercise was performed. Error bars are omitted in (A) for clarity. Means ± SD are shown in (B). *$p < 0.05$ vs. control. #$p < 0.05$ vs. CMI.



those who preferred HIIT had higher affective response to the bout of HIIT (2.8 ± 2) compared with those who preferred CMI (0.8 ± 0.6). Therefore, individual feelings of pleasure–displeasure during HIIT may influence exercise preference, a hypothesis that we are currently exploring.

Our study was not designed to examine the exact mechanisms contributing to exercise-induced improvements in postprandial hyperglycemia. We speculate that increased muscle fibre recruitment during high-intensity exercise (Edgett et al. 2013) results in an overall greater increase in muscle insulin sensitivity in the 24 h post-exercise recovery period. Also, high-intensity intermittent exercise has previously been shown to result in a rapid depletion of muscle glycogen (MacDougall et al. 1977). Because replenishment of muscle glycogen plays a key role in the post-exercise increase in peripheral insulin sensitivity (Devlin et al. 1987), HIIT may result in a more prolonged increase in muscle glucose uptake when compared with CMI to restore glycogen. It has been shown previously shown that short-term HIIT increases resting muscle glycogen (Little et al. 2010) and GLUT4 (Hood et al. 2011; Little et al. 2011), which supports this hypothesis.

Hazell et al. (2012) recently reported that a single session of low-volume HIIT elevated oxygen consumption for up to 24 h following exercise. In their study, the increase in post-exercise oxygen consumption following HIIT followed a different pattern when compared with continuous endurance training, with HIIT appearing to be elevated above continuous exercise from 8–24 h into recovery. Thus, it is possible that the improvement in next day PPG control following HIIT may be related to an elevated basal metabolic rate. In addition, HIIT may influence hepatic glucose output and (or) beta-cell function. The ability of insulin to inhibit hepatic glucose production following meals is a key factor controlling the PPG response (Rizza 2010) and may have been influenced by HIIT in the present study. Whyte et al (2013) have recently reported fasting homeostasis model assessment of insulin resistance and glucose–insulin responses to an oral glucose tolerance test on the morning after a session of HIIT (i.e., at a time point corresponding to the breakfast meal in the present study) in overweight or obese adults. These authors found no significant effects of HIIT, providing evidence that HIIT may not have lasting effects on hepatic insulin sensitivity or postprandial insulin secretion. The acute effects of exercise intensity and modality on hepatic glucose output and beta-cell function remain largely unexplored and are areas that are ripe for investigation. Interestingly, the increase in pre-meal glucose concentration prior to lunch (i.e., the first meal after acute exercise in our study) was slightly higher after HIIT, which is possibly due to a greater counter-regulatory hormone response. This did not affect the postprandial glycemic response to lunch or glycemic responses to any of the subsequent meals.

Despite reducing PPG responses, neither exercise modality led to significant reductions in 24 h mean blood glucose concentration (Table 3). Previous research in patients with T2D is mixed with some studies reporting reductions in 24 h mean blood glucose (Oberlin et al. 2013; Manders et al. 2010), whereas others only detect significant reductions in postprandial hyperglycemia (Gillen et al. 2012; Mikus et al. 2012*a*). Because elevated postprandial hyperglycemia (Cavalot et al. 2006, 2011) and within-day glucose fluctuations (Monnier and Colette 2008) are independent risk factors for cardiovascular disease, improvements in post-meal glucose control following exercise are likely of importance. In our view, this highlights the unique ability of CGM to assess how exercise impacts glucose control. It is important to note, however, that we did not see any statistically significant effects of HIIT (or CMI) on absolute post-meal glucose responses or overall glycemic variability (MAGE, SD). Therefore, the potential benefits of HIIT on glucose control for overweight or obese individuals will require further research.

Our results are based on a relatively small sample of overweight or obese individuals, some of whom had impaired fasting glucose, but we did not screen for impaired glucose tolerance prior to the study. Thus, generalizability of the findings may be limited. Nonetheless, we were still able to detect statistically significant differences between both exercise modalities and no-exercise control for PPG responses to dinner, and significantly greater effects of HIIT compared with both CMI and control for breakfast. Our sample included 8 females (7 included in full analyses) and only 2 males, which prevented analyses of potential sex differences. When data for only females were analyzed, the results were the same; with the exception that ANOVA results for dinner AUC and dinner PPS no longer reached statistical significance ($p = 0.15$ and $p = 0.10$, respectively). As such, in the context of our small sample, males and females appeared to respond similarly. Although we instructed participants not to engage in any purposeful activity outside of the exercise sessions and verified this via self-report logs, a limitation of this study is that we did not objectively monitor activity using a pedometer or accelerometer while we collected CGM data.

Conclusions

In conclusion, this study demonstrates that a single session of HIIT involving 10 × 1-min intervals eliciting approximately 90% HR_{peak} improved PPG control in overweight or obese adults for up to approximately 24 h following exercise. The lasting effects of an acute bout of HIIT appeared superior to 30 min of CMI, as the postprandial glucose spike and AUC following breakfast on the following day were lower with HIIT compared with CMI. HIIT was well tolerated, equally enjoyed, and preferred by 6/10 participants when compared with CMI. These findings offer preliminary evidence that HIIT may be an effective exercise strategy for improving glycemic control and reducing the risk of micro- and macro-vascular

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disease associated with postprandial hyperglycemia. Future research is required to understand the underlying mechanisms that contribute to improved postprandial glycemic regulation following HIIT and to adequately determine how this type of exercise compares with a prescription of traditional CMI exercise for adherence and improving health outcomes over time.

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