ARTICLE

Impact of 4 weeks of interval training on resting metabolic rate, fitness, and health-related outcomes

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Abstract: Resting metabolic rate (RMR) and substrate oxidation (respiratory exchange ratio; RER) are important indicators of health. The effects of interval training on RMR have not been thoroughly investigated, which was the purpose of the present study. Thirty men and women (mean ± SD age and maximal oxygen uptake: 28.8 ± 7.6 years and 33.0 ± 8.3 mL·kg^{-1}·min^{-1}) completed 4 weeks of Wingate-based sprint interval training (SIT), repeated 1-min high-intensity intervals (HIIT), or served as controls. Before and after training, RMR, resting RER, maximal oxygen uptake, body composition, physical activity, and energy intake were recorded. Data were analyzed using a repeated-measures ANOVA. RMR increased in response to 4 weeks of SIT training (1789 ± 293 to 1855 ± 320 kcal·day^{-1}; $p = 0.003$) but did not increase after HIIT (1670 ± 324 to 1704 ± 329 kcal·day^{-1}; $p = 0.06$). While SIT increased RMR by ~2× the magnitude of HIIT, the difference was not significant ($p = 0.5$). Fasting substrate oxidation and RER did not change ($p > 0.05$). Maximal oxygen uptake increased, and small changes were also observed in percent body fat and fat mass ($p < 0.05$ for all). In conclusion, SIT provided a time-efficient stimulus to increase RMR after 4 weeks in healthy adults. However, the clinical relevance of the changes observed in this study remains to be determined. Further studies should be conducted in obese individuals and those with diabetes or insulin resistance to examine if interval training (≥4 weeks) influences resting metabolic rate in magnitudes similar to that reported here.

Key words: resting metabolic rate, respiratory quotient, interval training, substrate oxidation.

Résumé : Le métabolisme de repos (quotient respiratoire métabolique; « RMR ») et l'oxydation des substrats (« RER ») sont d'importants indicateurs de la santé. Cet article a pour objectif d'approfondir les connaissances au sujet des effets de l'entraînement par intervalle sur le RMR. Treize femmes et hommes (moyenne ± ÉT, âge : 28,8 ± 7,6 ans, consommation maximale d'oxygène : 33,0 ± 8,3 mL·kg^{-1}·min^{-1}) participent à quatre semaines d'entraînement de type Wingate au sprint par intervalle (SIT), à des intervalles d'intensité élevée d'une durée d'une minute (« HIIT ») ou à une condition de contrôle. Avant et après l'entraînement, on évalue le RMR, le RER de repos, la consommation maximale d'oxygène, la composition corporelle, l'activité physique et l'apport énergétique. Les données sont soumises à une ANOVA avec mesures répétées. Le RMR augmente en réponse aux 4 semaines de SIT (de 1789 ± 293 à 1855 ± 320 kcal·jour^{-1}; $p = 0,003$), mais n'augmente pas en réponse à HIIT (de 1670 ± 324 à 1704 ± 329 kcal·jour^{-1}; $p = 0,06$). Dans la condition SIT, RMR augmente de ~2× l'ampleur de HIIT, mais la différence n'est pas significative ($p = 0,5$). L'oxydation des substrats à jeun et le RER ne varient pas ($p > 0,05$). On observe une augmentation du VO$_2$ max et une faible modification du pourcentage de gras et de la masse adipeuse ($p < 0,05$ pour toutes les variables). En conclusion, SIT s'avère un stimulus rentable pour l'augmentation du RMR en 4 semaines chez des adultes en santé. Toutefois, il reste à établir la pertinence clinique des modifications enregistrées dans cette étude. Il faut réaliser d'autres études auprès de personnes obèses et d'individus aux prises avec le diabète ou l'insulinorésistance pour vérifier si l'entraînement par intervalle (≥4 semaines) modifie l'ampleur du métabolisme de repos de façon similaire aux observations dans la présente étude. [Traduit par la Rédaction]

Mots-clés : métabolisme de repos, quotient respiratoire, entraînement par intervalle, oxydation des substrats.

Introduction

It is well known that obesity rates have increased worldwide, and that obesity is a risk factor for several diseases (Cornier et al. 2011; Ogden et al. 2014). Much attention has been given to the relationships between the development of cardiovascular disease (CVD) and modifiable risk factors such as obesity, diabetes, and physical inactivity (Lamarche et al. 1996; van Lenthe et al. 1998; Kokkinos and Myers 2010). Furthermore, compelling evidence now exists that increases in cardiorespiratory fitness can reduce or even eliminate mortality risk of other CVD risk factors (Kodama et al. 2009; Lee et al. 2009; Fogelholm 2010). Unfortunately, despite clear evidence of the protective effects of physical activity, it has been estimated that physical inactivity and insufficient activity were associated with 11% of health care expenditures from 2004–2011 (Carlson et al. 2015). It was recently reported that physical inactivity has major health burdens, accounting for 6% of the burden for CVD, 7% of type 2 diabetes (T2D), 27% of obesity, 16% of metabolic syndrome, 10% of cancers, and 9% of premature deaths (Lee et al. 2012). Risks of developing hypertension, CVD, stroke, and T2D are all elevated when comparing inactive individuals with active individuals of various ethnicities (Liu et al. 2014). It has been reported that obese men only obtain 23.4 min of moderate activity and 36 s of vigorous activity per day, while obese women obtain 13.8 min and 11 s, respectively (Tudor-Locke et al. 2010; Archer et al. 2013). It has also been reported that only

1.7% of overweight and obese adults met guidelines for weight management (Young et al. 2009). In a comprehensive review, Booth et al. (2012) stated that

> conclusive evidence exists that physical inactivity is one important cause of most chronic diseases. In addition, physical activity primarily prevents, or delays, chronic diseases, implying that chronic disease need not be an inevitable outcome during life.

In addition to common clinical risk factors (i.e., blood pressure, cholesterol, triglycerides), there may be other variables associated with disease risk that can be assessed. For example, in sedentary individuals, resting energy expenditure (REE) accounts for the most significant (60%–70%) portion of total daily energy expenditure (TDEE). REE is also known as resting metabolic rate (RMR, used subsequently in this paper). A low RMR has been linked with weight gain over time in some studies (Luke et al. 2006) but not others (Shook et al. 2016; Anthanont and Jensen 2016). Besides RMR, fasting substrate oxidation has also been implicated as a measure of health and disease risk. Shook et al. reported that individuals with higher fasting respiratory quotients (respiratory exchange ratio (RER), a proxy of fasting substrate oxidation), indicative of a greater portion of carbohydrates being oxidized, gained larger amounts of weight than individuals with lower RERs (Shook et al. 2016). This has been mirrored in 24-h metabolic chamber studies where higher fed RERs were positively associated with weight gain, 24-h fat oxidation was inversely associated with weight change (in men), and a higher 24-h RER was predictive of greater ad libitum food intake (Piaggi et al. 2013, 2015). In sum, these prospective studies suggest that assessment of RMR and RER may be useful as clinical tools for risk assessment and stratification.

In light of current health issues, physical activity is heavily promoted to ameliorate disease risk. However, existing guidelines suggest anywhere from 150–300 min per week of moderate–vigorous physical activity (MVPA), which may be a significant barrier to adherence because of the time commitment as less than 5% of a nationally representative sample achieved 30 min of activity per day as assessed via accelerometry (Trost et al. 2002; Troiano et al. 2008). Low volume, high-intensity interval training (HIIT) has been promoted as a more time-efficient and similar or more potent stimulus than continuous moderate-intensity training (Burgomaster et al. 2008; Gibala et al. 2012; Gillen et al. 2016). HIIT and one of its relatives, sprint-interval training (SIT, defined as "supramaximal" or Wingate-based training (Weston et al. 2014)), have been shown to positively impact a number of health outcomes, including increases in cardiorespiratory fitness and maximal fat oxidation (Astorino et al. 2013a, 2013b; Gist et al. 2014), upregulation of skeletal muscle proteins and markers of mitochondrial function related to oxidative phosphorylation capacity (Gurd et al. 2010; Little et al. 2010; Gillen et al. 2013), reduced insulin resistance (Little et al. 2011; Earnest et al. 2013), and improved body composition (Gillen et al. 2013).

Despite these positive benefits, relatively little attention has been given to the influence of interval training on RMR and fasting substrate oxidation (Whyte et al. 2010; Sevits et al. 2013; Fisher et al. 2015; Martins et al. 2016). For example, Fisher and colleagues had 28 sedentary and overweight men perform 6 weeks of HIIT with 30-s bouts at 85% peak power (derived from a Wingate test) with 4-min recoveries at 15% peak power for 3 days/week or 45–60 min of moderate-endurance exercise between 55%–65% maximal oxygen uptake ($\dot{V}O_{2max}$) (Fisher et al. 2015). The authors did not report results for fasting substrate metabolism, but reported no significant changes in RMR after HIIT (–13 kcal/day) (Fisher et al. 2015). Further to this, Martins and colleagues report that after 12 weeks of HIIT, moderate-endurance exercise, or half-HIIT, had no significant effects on RMR and fasting substrate oxidation in sedentary and obese individuals (Martins et al. 2016). In contrast, acute studies have reported increases in RMR or TDEE at

~24 h postexercise (Kelly et al. 2013; Sevits et al. 2013; Skelly et al. 2014; Jabbour et al. 2017), but this could be due to the time interval between the final training session and the outcome assessment, as Whyte et al. showed an elevation in RMR 24, but not 72, hours post-intervention (Whyte et al. 2010).

Thus, it was the objective of this study to examine how 4 weeks of either HIIT or SIT influenced RMR and substrate oxidation. Secondary objectives were to examine changes body composition, physical activity, energy intake, cardiovascular fitness, and maximal fat oxidation in response to SIT and HIIT. We hypothesized that HIIT and SIT would increase RMR, resting and maximal fat oxidation, and $\dot{V}O_{2max}$ without alterations in body composition, physical activity, or energy intake. SIT and HIIT protocols were selected based on their use in the literature and to examine whether intensity affected the variables of interest.

Materials and methods

Participants

Healthy men ($n = 13$) and women ($n = 17$) were recruited from the university and surrounding community. Criteria for inclusion were moderately active (defined as >120 min of moderate–vigorous activity per week by self-report during the previous 6 months); between the ages of 18–50; nonsmokers; not currently taking any medication or supplements known to effect metabolism or blood pressure; and (for women) were eumenorrheic or on birth control, and not planning on becoming pregnant in the following 3 months. Participants typically completed aerobic-type exercise (walking, jogging, cycling) as well as resistance training for 3–5 days·week⁻¹. This study was approved by the Auburn University at Montgomery Institutional Review Board for Human Subjects Research and adhered to the guidelines laid out in the Declaration of Helsinki. All investigators completed Collaborative Institutional Training Initiative research compliance training.

Experimental design

This study was a quasi-randomized control trial. Interested participants were randomized to either an SIT ($n = 12$) or an HIIT ($n = 12$) group. Individuals who wished to undergo the testing procedures but were unwilling to modify their existing training regimes self-selected into the control group (CON, $n = 6$). All training and testing sessions were conducted on a Velotron DynaFit Pro cycle ergometer (RacerMate Inc., Seattle, Wash., USA). All groups were instructed to maintain their habitual activity and diet and not to begin any new exercise programs during the 4 weeks of the study. Women were tested in the same phase of the menstrual cycle before and after completion of the study; this was determined through self-report. A 4-week study duration was elected for several reasons. First, this would allow for examination of changes in habitual behaviors such as free-living physical activity that acute studies typically do not measure. Second, we wished to limit the study to a pre-/postassessment, and felt any duration greater than 4 weeks would require another $\dot{V}O_{2max}$ test to update the training prescription in the HIIT group, necessitating additional assessments.

The SIT group completed training as previously described by Gillen et al. (2016), with modifications. Training the first week of the study consisted of a 2-min warm-up at 10% peak power output (PPO), three 20-s "all-out" sprints at a resistance equivalent to 5% of baseline body weight with 2-min recoveries at 10% PPO, and a 3-min cool-down a 10% PPO. During the last 10 s of each recovery period, participants had 10 s of unloaded cycling and were instructed to pedal as quickly as possible. At the end of the 10 s, the resistance was applied automatically and participants were given strong verbal encouragement to maintain their cadence until the end of the 20 s sprint. Week 2 included 4 repeats and weeks 3 and 4 incorporated 5 repeats. Total training time per session ranged from 10 min during week 1 to ~15 min during weeks 3–4.

The HIIT group completed a training program that has been widely used in the literature, i.e., 1-min repeats at 90% PPO with 1-min recoveries at 10% PPO (Astorino et al. 2013*a*, 2013*b*; Kelly et al. 2013). As with SIT, all sessions began and ended with a 2-min warm-up and 3-min cool-down. During the first 2 weeks, participants completed 6 repeats and during the final 2 weeks they performed 8 repeats. Total training time per session ranged from 16 min to 20 min.

Sessions were completed at the same time of day within-participants (±1 h). Dietary standardization was not strictly enforced; however, participants were instructed to refrain from eating at ~2 h before to avoid any gastrointestinal discomfort. We aimed to keep all training sessions similar in length; thus work was not matched between conditions. Previously published equations using average heart rate during exercise, age, weight, sex, and baseline $\dot{V}O_{2max}$ (Dugas et al. 2005) were utilized to estimate energy expenditure (EE) per session. The equation was as follows:

$$(1) \quad \{-59.4 + [(\text{sex } 1 = M, \ 0 = F) \times 0.27 \times \text{age}]\} + (0.394 \times \text{weight})$$
$$+ (0.4 \times \text{baseline } \dot{V}O_{2max}) + (0.634 \times \text{exercise HR}) + (1 - \text{sex})$$
$$\times [(0.274 \times \text{age}) + (0.103 \times \text{weight}) + (0.38 \times \dot{V}O_2)$$
$$+ (0.45 \times \text{exercise HR})] = EE$$

where HR is heart rate and EE is in kJ·min^{-1}.

Assessments

All assessments were conducted during 1 visit before and after the study. Each assessment visit was conducted at the same time of day within-participants (±1 h). The order of assessments always began with height and weight, followed by RMR, circumference measurements, body composition, and $\dot{V}O_{2max}$.

RMR and substrate oxidation

RMR and substrate oxidation were determined using a ventilated hood and metabolic cart (ParvoMedics TrueOne 2400, Salt Lake City, Utah, USA). Participants were instructed to refrain from alcohol, caffeine, and moderate–vigorous exercise for a minimum of 14 h before each visit and to abstain from food for 12 h before testing and limit activity on their way to laboratory. Post-testing was done at 48–72 h after the final exercise session to decrease the chance of any transient effects (Whyte et al. 2010).

Upon arrival (0600–0900 h), participants rested in a supine position in dimly lit, temperature-controlled (21–22 °C) room and were asked to relax but avoid falling asleep. Following 10–15 min of rest, participants were placed under the hood, covered with the drape, and remained in the hood for at least 20 min. If participants did not achieve a steady state by 20 min, additional 5-min increments were allotted up to 40 min total duration (this was not necessary for any participants).

Criteria for a valid RMR assessment was predefined as a minimum of 10 min of steady state with less than 10% fluctuation in oxygen consumption ($\dot{V}O_2$) (Compher et al. 2006). RMR (kcal·day^{-1}) was calculated using the Weir equation (Weir 1949) and resting substrate oxidation was calculated using the equations of Frayn (Frayn 1983), as follows:

$$(2) \quad RMR = [(3.941 \times \dot{V}O_2) + (1.1106 \times \dot{V}CO_2)] \times 1440$$

(Weir 1949). Where $\dot{V}CO_2$ is carbon dioxide production.

$$(3) \quad \text{carbohydrate oxidation} = [(4.55 \times \dot{V}CO_2) - (3.21 \times \dot{V}O_2)]$$

(Frayn 1983).

$$(4) \quad \text{fat oxidation} = [(1.67 \times \dot{V}O_2) - (1.67 \times \dot{V}O_2)]$$

(Frayn 1983).

Coefficients of variation and typical error of the measurement from test–retest reliability for RMR and RER in our lab, determined from duplicate trials in 13 men and women of varying ethnicities and body composition, are 1.3% (3.8 mL·min^{-1}) for $\dot{V}O_2$, 2.75% (5.3 mL·min^{-1}) for $\dot{V}CO_2$, 1.5% (28 kcal·day^{-1}) for RMR, and 2.4% (0.015) for RER.

Maximal fat oxidation and $\dot{V}O_{2max}$

Maximal rates of fat oxidation and their corresponding workloads and intensities were determined during an incremental cycle test on a Velotron DynaFit Pro ergometer (RacerMate Inc.). Standardization procedures were identical to those described above for RMR. Briefly, men (40 W) and women (30 W) cycled for a 2-min warm-up followed by increases in workload of 20 W every 3 min until the RER was >1.0 for an entire stage. HR (Polar Electro Oy, Kempele, Finland) and gas exchange data were recorded continuously using the aforementioned metabolic cart with a face mask (V2 Mask; Hans-Rudolph Inc., USA). Leg pain (Cook et al. 1997) and rating of perceived exertion (RPE) (1–10 Borg) (Borg 1982) were recorded at the end of each stage.

After RER exceeded 1.0 for an entire stage, participants continued to cycle to exhaustion while the workload was increased 20 W every minute. HR and gas exchange were recorded continuously while leg pain and RPE were recorded every minute until exhaustion. After exhaustion, participants pedaled easily for 1 min and then were permitted to dismount the ergometer and rest for 10 min. At this time, they re-mounted the ergometer and completed a ride to exhaustion at 110% of their peak power output while gas exchange data and HR were recorded to verify $\dot{V}O_{2max}$ (typical duration ~90–180 s).

$\dot{V}O_{2max}$ was determined as the highest value recorded over a 30-s period from either test. PPO was the workload completed for an entire 60-s stage during the maximal test.

This assessment and verification of $\dot{V}O_{2max}$ has been shown to be valid, repeatable, and reliable in active (Bishop et al. 1998; Astorino and White 2010; Merry et al. 2016) men and women.

Body composition and anthropometrics

Immediately preceding RMR assessments, participants were weighed (BWB-800A; Tanita Corp. USA, Arlington Heights, Ill., USA) and had their height recorded without shoes in light clothing in duplicate using a wall-mounted stadiometer (ProDoc Detecto; Detecto Products, Webb City, Mo., USA). Waist and hip circumference were obtained in duplicate using a tape measure (Gulick, Creative Health Products, Ann Arbor, Mich., USA) and following standard procedures (Heyward and Gibson 2014).

Percent body fat (%BF), fat-free mass (FFM), and fat mass (FM) were determined utilizing air displacement plethysmography (BodPod; Cosmed USA, Concord, Calif., USA) immediately after RMR measures. From a sample of 32 young men and women, coefficients of variation and typical error of the measurement from test–retest reliability in our lab for %BF, FFM, and FM are 4.7% (1.3%), 1.4% (1.3 kg), and 4.8% (1.16 kg), respectively.

Physical activity and energy intake

Time spent in varying stages of activity (sedentary, light, and MVPA) and steps per day were determined using wrist-worn accelerometers placed on the nondominant wrist (GTX3+; Actigraph Corp.) over 7 days. Participants received the accelerometer after completion of their maximal test and returned the accelerometer 1 week later when they reported for their first training session, and were given the accelerometer after their last training session at the end of the study. All accelerometer data were analyzed and processed using the ActiLife software (version 6.13.2; Actigraph Corp., Pensacola, Fla., USA). Wear time was assessed using the criteria of Choi et al.(2011). A minimum of 10 h of wear time was considered a valid day and 3 valid days were required to be included in the analysis (Willis et al. 2014). The cut-points of Troiano

Table 1. Changes in body composition, diet, and activity.

	Control		HIIT		SIT	
	Pre-training	Post-training	Pre-training	Post-training	Pre-training	Post-training
Body mass (kg)	82.8±19.1	83.1±18.9	76.4±16.2	76.2±16.8	81.2±16.5	83.2±15.2
Body fat (%)	20.8±7.8	21.0±8.0	25.7±8.4	23.8±8.1*	29.4±10.7	28.1±10.8*
Fat mass (kg)	17.6±9.6	17.9±9.8	19.7±7.7	18.3±7.8*	24.7±12.4	24.3±12.4*
Fat-free mass (kg)	65.2±14.2	65.2±14.0	56.8±13.5	57.9±13.5	56.5±9.8	58.9±8.2
Body mass index (kg·m^{-2})	26.6±5.1	26.75±5.0	26.9±3.6	26.6±3.7	28.4±4.7	29.0±4.4
Waist circumference (cm)	79.0±7.4	79.5±7.0	80.1±9.9	80.0±9.4	84.4±11.1	85.1±10.6
Energy intake (kcal·d^{-1})	2584±638	2544±658	2371±511	2427±483	2515±427	2547±487
Sedentary time (%)	61.9±6.6	63.3±5.9	59.1±9.9	58.5±9.0	60.2±11.7	59.6±11.7
Light PA (%)	21.6±2.5	20.9±1.5	25.4±6.3	26.3±4.9	25.7±7.9	25.6±6.8
MVPA (%)	16.5±4.6	15.8±4.6	15.5±4.3	15.6±4.4	14.1±5.6	14.8±6.2
Steps·d^{-1}	12 378±3572	12 167±3773	11 057±2653	11 576±3886	9799±2985	9718±2842

Note: Data are means ± SD. HIIT, high-intensity interval training; MVPA, moderate–vigorous physical activity; PA, physical activity; SIT, sprint interval training.

*Significant within-group difference ($p < 0.05$).

et al. (2008) were utilized to determine time spent in sedentary, light, and MVPA.

Energy and macronutrient intake were obtained via 3-day food records. They received their food logs the same time they received their accelerometers. Participants were asked to record all food and drink over 3 consecutive days, including 1 weekend day. Energy and nutrient data were determined utilizing nutritional analysis software (Food Processor; ESHA Research, Salem, Ore., USA). While we acknowledge that self-reported energy intake is susceptible to underreporting, Goldberg cutoff values (energy intake (EI)/RMR) indicated that no participants fell below the threshold of 1.35 that is deemed physiologically implausible (Black et al. 1991; Goldberg et al. 1991).

Statistics

Data were analyzed using IBM SPSS (version 23; IBM Corp., Armonk, N.Y., USA). Our sample size was estimated based on prior research on interval training; additionally, based on an estimated $\beta = 0.8$, moderate effect size $f = 0.25$, and a correlation among repeated measures of 0.8 for resting metabolic rate, a total sample size of 21 participants was calculated using G*Power 3.1.9.2 (Faul et al. 2007). A 1-way ANOVA was used to assess group differences at baseline. A 2-way repeated-measures (group × time) ANOVA was used to examine differences between and within groups before and after training. Post hoc analysis with the Bonferroni correction was performed when significant main effects or interactions were detected. Sex-based differences were not examined in the present analysis because of an expected lack of power. Statistical significance was accepted at $p < 0.05$. Data are reported in text as means ± SD and in figures display individual values.

Results

Thirty-three participants began the study, with 3 withdrawing because of time ($n = 2$, one each from SIT and HIIT) or illness ($n = 1$, HIIT). Thus, 30 participants completed the study (CON = 6, SIT = 12, HIIT = 12). The training groups each consisted of 5 men and 7 women, with 3 men and 3 women in the CON group. The majority of the participants (67%) were Caucasian, 27% African-American, 3% Asian/Pacific Islander, and 3% Hispanic. Mean age was 28.8 ± 7.6 years across the groups. Body composition characteristics are reported in Table 1. No differences between groups for any variables were observed at the beginning of the study ($p > 0.30$ for all).

Descriptive characteristics of training

All participants completed the required 12 sessions over the 4 weeks of training. Mean percentage of HR maximum, averaged over all sessions, was 76.1% ± 3.8% in SIT and 73.4% ± 4.8% in HIIT. Peak percentage HR maximum (recorded at the end of each interval), averaged over all sessions, was 86.6% ± 3.4% in SIT and 78.0% ±

5.0% in HIIT. Averaged over all sessions, peak power during SIT was 256% ± 65% of baseline PPO. EE per session, as estimated from average HR, was 147 ± 28 and 157 ± 35 kcals for SIT and HIIT, respectively.

RMR, RER, and substrate oxidation

Individual data are displayed in Fig. 1 for RMR and Fig. 2 for RER. Means ± SD for RMR, RER, and substrate oxidation are displayed in Table 2. Repeated-measures ANOVA revealed a main effect of time ($p = 0.025$), no main effect of group ($p = 0.565$), and a significant group × time interaction ($p = 0.001$) for RMR. RMR significantly increased (+65 kcals; $p = 0.003$) in response to SIT and trended towards a significant increase (+34 kcal; $p = 0.062$) in response to HIIT. RMR in CON did not change (–36 kcal; $p = 0.085$).

With regards to fasting RER, no main effect of time ($p = 0.964$), a main effect of group ($p = 0.045$), but no group × time interaction ($p = 0.872$) were observed. Mean RER in the HIIT group was significantly lower than SIT ($p = 0.02$) and CON ($p = 0.02$), but no groups revealed an effect of training. No significant effects or interactions were detected for fasting carbohydrate or fat oxidation ($p > 0.06$ for all).

$\dot{V}O_{2max}$ and exercise metabolism

Data for $\dot{V}O_{2max}$ and exercise metabolism are displayed in Table 3. Baseline $\dot{V}O_{2max}$ was similar between groups ($p = 0.32$). $\dot{V}O_{2max}$ significantly increased over time ($p = 0.002$) and displayed a group × time interaction ($p = 0.012$), but no between-group ($p = 0.584$) effect. $\dot{V}O_{2max}$ significantly increased in the training groups ($p = 0.014$ in SIT; and $p = 0.006$ in HIIT) but not in the CON group ($p = 0.319$). Similar results were observed for PPO, including a significant effect of time ($p = 0.012$) and group × time interaction ($p = 0.026$), but no main effect of group ($p = 0.571$).

Maximal fat oxidation (absolute and relative to FFM) did not change in response to training ($p = 0.582$), nor was there an effect of group ($p = 0.61$) or significant group × time interaction ($p = 0.10$). Expressed as a percentage of $\dot{V}O_{2max}$, a trend for a main effect of time ($p = 0.061$) and a significant group effect ($p = 0.006$), but no group × time interaction ($p = 0.47$), were revealed. Participants in the HIIT group achieved their maximal fat oxidation (MFO) at a greater percentage of $\dot{V}O_{2max}$ compared with those in the SIT group during post-testing ($p = 0.045$).

Body composition and anthropometry

Data for body composition and anthropometry are displayed in Table 2. Body weight, body mass index, and waist circumference did not significantly change in response to training or differ between groups ($p > 0.22$ for all). For %BF, there was a main effect of time ($p = 0.01$), but no between-group differences ($p = 0.25$) or group × time interaction ($p = 0.067$). Post hoc paired t test revealed

Fig. 1. Resting metabolic rate data by group and time. Data are displayed as individual values with means displayed as thick lines and symbols. Top panel, control = circles; middle panel, high-intensity interval training (HIIT) = squares; bottom panel, sprint interval training (SIT) = triangles. Pre, before training; Post, after training.

Fig. 2. Resting respiratory exchange ratio (RER) data by group and time. Data are displayed as individual values with means displayed as thick lines and symbols. Top panel, control = circles; middle panel, high-intensity interval training (HIIT) = squares; bottom panel, sprint interval training (SIT) = triangles. Pre, before training; Post, after training.
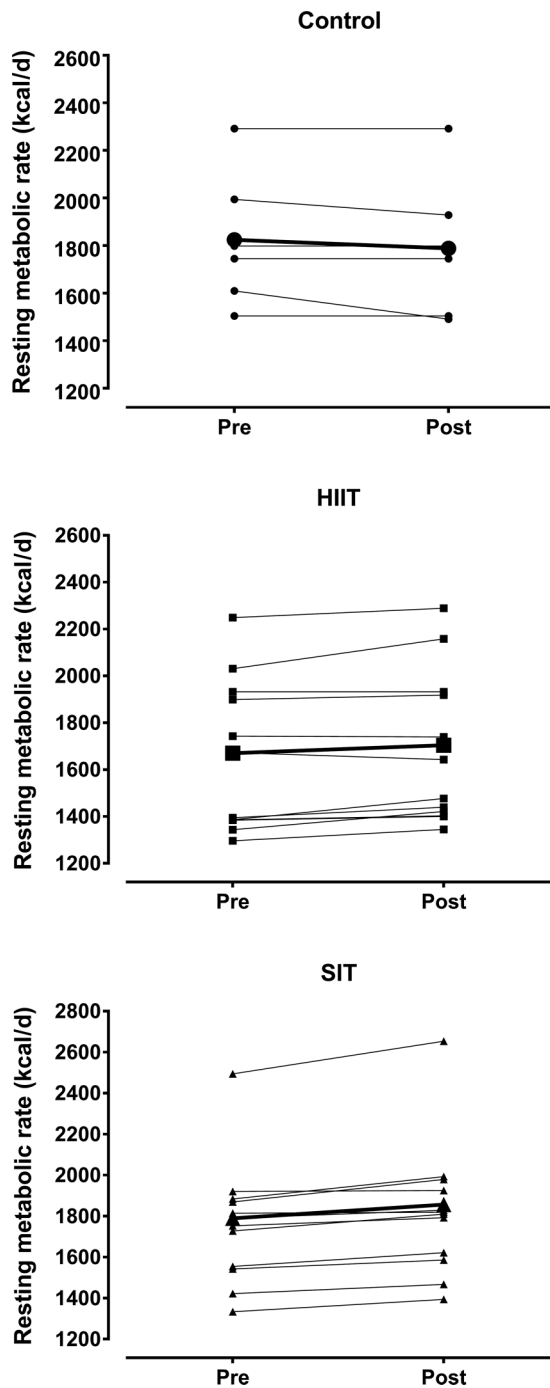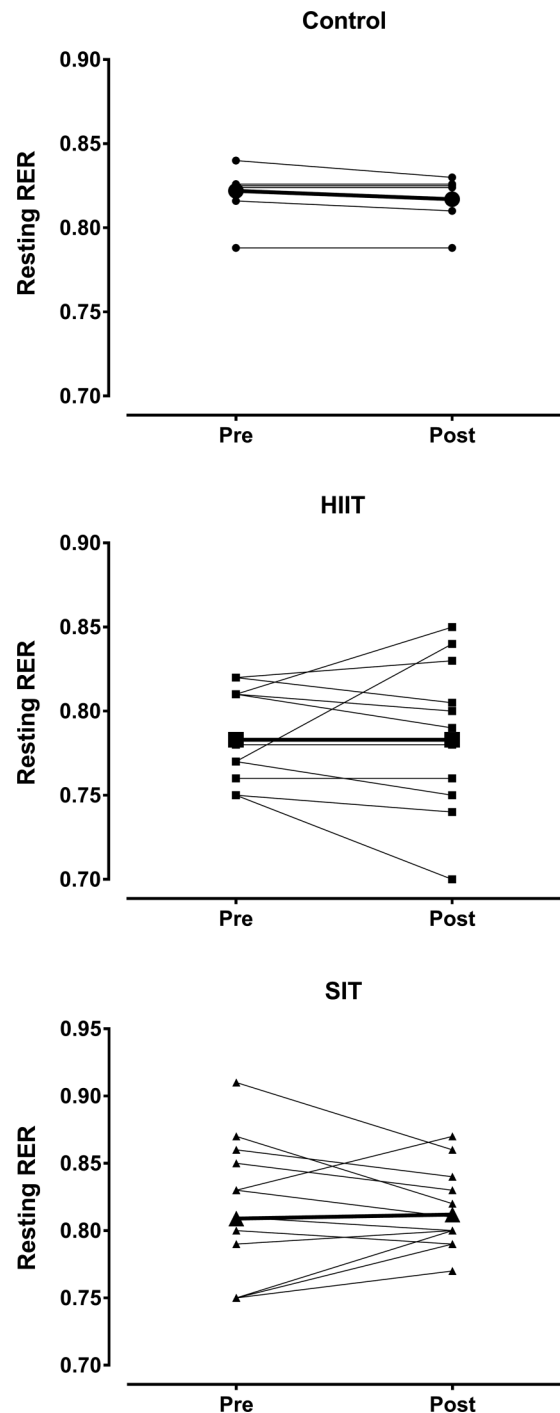

that %BF significantly decreased in SIT ($p = 0.014$) and HIIT ($p = 0.032$). Similar results were observed for FM, whereby a main effect of time ($p = 0.016$) but no between-group differences ($p = 0.279$) or group × time interaction ($p = 0.069$) were observed. Post hoc testing revealed significant reductions in FM in response to SIT ($p = 0.015$) and HIIT ($p = 0.043$). Regarding FFM, no main effect of time ($p = 0.06$), group ($p = 0.433$), or group × time interaction ($p = 0.345$) were observed.

Physical activity and energy intake

Physical activity levels, as assessed by percentage of time spent in sedentary activity, light physical activity, MVPA, and steps per day, were not different at baseline and were maintained over the intervention in all groups (all $p > 0.28$). Energy and macronutrient intake were not different between baseline and post-testing (all $p > 0.21$). Data for physical activity and energy intake are displayed in Table 1.

Table 2. Changes in RMR and resting substrate metabolism.

	Control		HIIT		SIT	
	Pre-training	Post-training	Pre-training	Post-training	Pre-training	Post-training
RMR (kcal·d^{-1})	1824±284	1788±291	1670±324	1704±329	1789±293	1855±320*
RER	0.82±0.02	0.82±0.02	0.78±0.03	0.78±0.05	0.81±0.04	0.81±0.03
Fat oxidation (g·min^{-1})	0.08±0.02	0.08±0.02	0.08±0.02	0.09±0.02	0.09±0.03	0.08±0.02
Carbohydrate oxidation (g·min^{-1})	0.13±0.02	0.13±0.02	0.09±0.04	0.09±0.05	0.12±0.05	0.13±0.04

Note: Data are means ± SD. HIIT, high-intensity interval training; RER, respiratory exchange ratio; RMR, resting metabolic rate; SIT, sprint interval training.

*Significant within-group difference ($p < 0.05$).

Table 3. Changes in fitness and exercise metabolism.

	Control		HIIT		SIT	
	Pre-training	Post-training	Pre-training	Post-training	Pre-training	Post-training
$\dot{V}O_{2max}$ (mL·kg^{-1}·min^{-1})	37.5±8.3	37.0±8.8	31.35±9.2	33.77±9.6*	32.3±7.1	35.0±7.4*
$\dot{V}O_{2max}$ (L·min^{-1})	3.06±0.89	3.04±0.92	2.38±0.79	2.56±0.82*	2.58±0.59	2.86±0.54*
Peak power output (W)	235±93	238±90	195±62	217±63*	211±46	242±35*
Maximal heart rate (beats·min^{-1})	187±8	188±6	179±8	183±7	184±9	186±7
Maximal $\dot{V}_E$ (L·min^{-1})	103±26	107±23	87±33	104±31	106±28	110±19
Maximal RER	1.18±0.05	1.24±0.06	1.19±0.07	1.21±0.08	1.18±0.06	1.22±0.07
Maximal rate of fat oxidation (g·min^{-1})	0.28±0.13	0.25±0.14	0.24±0.11	0.26±0.11	0.19±0.08	0.24±0.10
Maximal rate of fat oxidation (mg·kg FFM^{-1}·min^{-1})	3.0±1.4	3.5±1.5	4.0±1.8	4.0±1.7	4.7±2.0	4.6±2.0
Maximal rate of fat oxidation (%$\dot{V}O_{2max}$)	42.5±7.1	41.0±7.6	47.5±9.7	43.9±6.2	39.9±9.7	36.0±6.5

Note: Data are means ± SD. FFM, fat-free mass; HIIT, high-intensity interval training; RER, respiratory exchange ratio; SIT, sprint interval training; $\dot{V}_E$, minute ventilation; $\dot{V}O_{2max}$, maximal oxygen uptake.

*Significant within-group difference ($p < 0.05$).

Discussion

This study examined the influence of 4 weeks of HIIT or SIT on RMR, RER, and fasting substrate oxidation in healthy adults. We determined that interval training causes significant increases in RMR, with SIT causing about twice the increase as HIIT. $\dot{V}O_{2max}$ was improved and we also observed reductions in body fat levels. Importantly, the timing of assessments was scheduled so that transient effects of the training itself would be minimized. These data indicate that HIIT and SIT increase RMR by ~35–65 kcal, and this increase is within a level (~50–150 kcal·day^{-1}) estimated to prevent weight regain in most adults (Hill et al. 2003).

Prior studies that have evaluated changes in RMR and RER have treated these variables as secondary outcomes, or have looked at changes over an acute (≤48 h) window and have reported contrasting results (Whyte et al. 2010; Kelly et al. 2013; Sevits et al. 2013; Skelly et al. 2014; Fisher et al. 2015; Martins et al. 2016). For example, training studies of 6 and 12 weeks in duration reported no significant changes in RMR and RER comparing HIIT, SIT, and moderate-intensity continuous training (Fisher et al. 2015; Martins et al. 2016). This was despite the authors of these studies reporting decreases in FM and %BF in their participants, and increases in FFM – which may have been the main driver of the changes in RMR observed in the present study (discussed below). Furthermore, the divergent results between prior studies and the present study could be due to our healthier population being more sensitive to leptin, as changes in leptin levels in overweight/obese individuals have been associated with metabolic adaptations to weight loss, including a fall in RMR and increased food reward (Hopkins et al. 2014a, 2014b).

On the other hand, when authors have measured 24-h EE or RMR within a relatively short window after a single-interval training session, increases in RMR, 24-h EE, or both have been reported (Whyte et al. 2010; Kelly et al. 2013; Sevits et al. 2013; Skelly et al. 2014). In an elegant study, Whyte et al. conducted 2 post-tests after 2 weeks of SIT in overweight/obese men, one at 24-h post-intervention and the other at 72-h post-intervention (Whyte et al. 2010). RER was significantly lower at 24 h post-intervention com-pared with baseline, whilst at 72 h post-intervention it was not significantly different versus baseline. These data suggest that conducting assessments too closely to the final training session may lead to false positives, as the transient effects of a single SIT/HIIT bout may extend to 24 h or possibly longer (Whyte et al. 2010; Sevits et al. 2013; Skelly et al. 2014). Thus, in the present study, we scheduled all post-testing assessments at least 48 h after our participants' last training session to minimize transient effects.

The precise mechanism driving the change in RMR cannot be known. However, a number of authors have speculated that FFM is a significant determinant of RMR (Caudwell et al. 2013; Blundell et al. 2015). Though the change in FFM was not significant in the present study, both exercise groups had increases of 1–2 kg FFM. A simple correlation analysis between change scores revealed that only the change in FFM and change in RMR was significant ($r = 0.42$, $p = 0.028$) in the present study, but this is less than values reported in Blundell and colleagues' recent review of their work in overweight/obese individuals ($r = 0.51$–0.85) (Blundell et al. 2015). Furthermore, in a simple stepwise linear regression model, the change in FFM only explained 14.3% of the change in RMR ($p = 0.027$). As FFM is a highly metabolically active tissue, it is intuitive that increases in FFM would drive increases in RMR, though the precise signaling molecules can only be speculated. It also must be noted that the changes in FFM were close to, or within, our typical error. Thus, instead of seeing a true change in FFM, we could simply be observing biological variation.

In agreement with prior research, we report significant increases in $\dot{V}O_{2max}$ and PPO in response to interval training. It is now well established that interval training potently impacts $\dot{V}O_{2max}$ in multiple populations (Gist et al. 2014; Weston et al. 2014), and that $\dot{V}O_{2max}$ is an important predictor of mortality (Kodama et al. 2009). Both interval training groups increased $\dot{V}O_{2max}$ by ~7%–8%, which is broadly similar to that reported in studies using similar designs (Whyte et al. 2010; Fisher et al. 2015). Given that 1-MET increases in $\dot{V}O_{2max}$ levels confers anywhere from an 8%–19% reduction in CVD risk and all-cause mortality (Lee et al. 2012), the 0.7–0.8 MET increase our participants obtained could suggest risk reductions of 5.6%–7%.

We observed no significant changes in fuel metabolism during exercise, as assessed by MFO. This is in contrast to studies in active men and women (Astorino et al. 2011) and sedentary women (Astorino et al. 2013*a*) that have reported reductions in RER during submaximal exercise as well as increased MFO. However, the participants in our study were considerably more heterogenous than those in prior studies. The variation in our participants for $\dot{V}O_{2max}$ and body composition, therefore, likely prevented our ability to observe changes in MFO in response to SIT and HIIT.

The influence of interval training on body composition, such as RMR, has mixed results. In agreement with some studies, we observed small decreases in body fat. For example, Fisher et al. reported a decrease in body fat of a similar magnitude to the present study in their population of overweight/obese men, while Heydari and colleagues reported significant reductions in total weight, body fat, and visceral fat in overweight young men (Heydari et al. 2012; Fisher et al. 2015). Trapp also reported significant reductions in body weight, fat, and visceral fat in young women (Trapp et al. 2008). In contrast, other studies have not reported significant changes in body composition after 12 weeks of HIIT (Astorino et al. 2013*a*). Discrepancies among studies because of the design and body composition assessment likely account for this variation. While we did observe a significant decrease in body fat, there was no indication that this fat loss preferentially came from visceral fat (as assessed by waist circumference) as has been previously reported (Trapp et al. 2008; Heydari et al. 2012), and whether the magnitude of FM loss observed in the present study is clinically meaningful remains unclear.

There is some concern that given the intense nature of SIT/HIIT protocols, these types of exercise may lead to compensatory behaviors such as increased sedentary time or alterations in energy intake. We and others (Martins et al. 2016) have not found compelling evidence to support this notion; in fact, diet and physical activity remained relatively stable during our intervention. However, as we only utilized 3-day diet recalls as opposed to more objective measures of energy intake, it is possible we lacked the precision to observe meaningful changes.

Our study has a number of strengths and limitations that merit consideration. The quasi-randomized design, a control group, diverse sample, inclusion of men and women, and matching of caloric cost and time commitment between the intervention groups are all strengths. The tight control around all testing procedures provides further robustness to our results; however, further dietary standardization around the testing sessions could have yielded more robust results for resting substrate oxidation and MFO, which can be highly variable (Fletcher et al. 2017). The main limitation is likely the sample size – though we had sufficient power to detect some differences, for other variables where trends were observed, we did not. However, this sample size is in line with other work (Gillen et al. 2016). Even though the exercise groups were randomized, the "self-selecting" control group may have limited our ability to detect some differences given their comparatively high baseline $\dot{V}O_{2max}$, although the inclusion of a non-intervention control is a strength that is not always included in other studies (Fisher et al. 2015; Gillen et al. 2016). Another limitation is the laboratory-based nature of the intervention, which limits the generalizability of our results to the public. However, from a practical standpoint, incorporating a similar workout program in a fitness center would not be exceedingly difficult. It is also worth considering whether the observed changes in RMR are clinically meaningful. While it has been extensively argued that 50–150 kcal·day^{-1} is sufficient to offset weight gain, this is based on population level research and forecasting, and empirical evidence is somewhat limited (Hill 2006, 2009; Hill et al. 2009; Ross et al. 2016).

Conclusion

This study found that 4 weeks of SIT increased RMR from baseline, without influencing resting RER or substrate oxidation. Importantly, the observed increase in RMR were close to or within the "small changes" in energy expenditure that may help offset long-term weight gain (Hill et al. 2003). Additionally, $\dot{V}O_{2max}$ improved whilst FM decreased. Given the clinical significance of RMR and RER for predicting weight gain and metabolic health (Luke et al. 2006; Piaggi et al. 2013, 2015; Anthanont and Jensen 2016), further studies utilizing longer durations of training and individuals at increased risk of disease and mortality (such as those with obesity or T2D) should consider including assessments of RMR and fasting substrate oxidation.

Conflict of interest statement

The authors declare no conflicts of interest relevant to the present work.

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