Physiological Adaptations to Sprint Interval Training with Matched Exercise Volume

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ABSTRACT

LEE, C.-L., W.-C. HSU, and C.-F. CHENG. Physiological Adaptations to Sprint Interval Training with Matched Exercise Volume. *Med. Sci. Sports Exerc.*, Vol. 49, No. 1, pp. 86–95, 2017. **Purpose**: This study aimed to determine how high-intensity interval training (HIIT) protocols featuring matched times but distinct sprint durations affect cardiorespiratory and metabolic responses and performance. **Methods**: Thirty-eight recreationally active men (age 21 ± 2 yr) were assigned to one of three interval training groups: long-duration high-intensity (HIIT60s; 8 × 60 s at 85%–90% $\dot{V}O_{2max}$; 120-s recovery at 30% $\dot{V}O_{2max}$), short-duration high-intensity (HIIT10s; 48 × 10 s at 85%–90% $\dot{V}O_{2max}$; 20-s recovery at 30% $\dot{V}O_{2max}$), and control (regular physical activity without HIIT). Before and after a 4-wk training period (three sessions per week), participants performed graded exercise tests and repeated sprint tests, based on which their aerobic and anaerobic capacities were assessed. Skinfold thickness, blood, and metabolic responses were also measured before and after intervention. **Results**: After the 4-wk training period, $\dot{V}O_{2max}$ was significantly increased ($P < 0.01$) in HIIT60s (52 ± 9 vs 61 ± 12 mL·kg^{-1}·min^{-1}) and HIIT10s (53 ± 10 vs 61 ± 10 mL·kg^{-1}·min^{-1}), but there were no changes in the control group (50 ± 7 vs 52 ± 7 mL·kg^{-1}·min^{-1}). Skinfold thickness in the abdomen and thigh did not differ significantly among the groups, but a significantly greater decrease in 14%–25% in HIIT60s and a decrease in 20% in HIIT10s after training ($P < 0.05$) were found. Blood lactate, total cholesterol, triglyceride, cortisol, and insulin concentrations were not significantly different among the three groups ($P > 0.05$), but testosterone concentration in the HIIT10s was higher after training than before ($P < 0.05$). **Conclusion**: The higher incremental aerobic performance and lower skinfold thickness in HIIT60s versus HIIT10s reflected similar adaptations, but the higher repeated sprint performance was observed only in responses to HIIT60s, which may elicit greater anaerobic adaptations. **Key Words:** AEROBIC INTERVAL TRAINING, HORMONE, LIPID, PERFORMANCE, TIME-EFFICIENT TRAINING

Low-volume high-intensity interval training (HIIT) is currently recognized as a time-efficient training strategy for improving performance in athletes (18) and overall health in adults (30). HIIT is typically defined as exercise performed at an intensity that is above the anaerobic threshold (AT), with the exercise duration ranging from repeated bouts of sprints (6–8 s per sprint) to a few minutes (20,30). HIIT not only supplies more energy to the muscle tissue under anaerobic metabolism but also depletes the energy from glycogenolysis (6) and has comparable effects with continuous moderate exercise training on cardiometabolic risks (30), including glucose metabolism, serum lipids, blood pressure, and anthropometric outcomes in terms of body fat and anabolic and catabolic hormone levels. Recently, interest has increased in determining whether HIIT improves health outcomes in nonathletes. In one study, untrained men ran 4 × 800 m at an intensity of 90% of the HR_{max}, 3 d·wk^{-1} for 8 wk, and the results revealed that this HIIT improved blood lipid profiles (i.e., HDL-C level was increased, and triglyceride [TG] and cholesterol [Chol] levels were decreased) (37). Intriguingly, metabolic adaptations were also reported to be induced by an extremely short-duration HIIT (HIIT10s) program: 2 wk of HIIT (6 × 30-s cycle sprints interspersed with 4-min recovery periods) correlated with improved insulin activity and glycemic control in young men (2). Thus, short-term sprint-type interval training might offer health and exercise-capacity benefits that are comparable with those provided by traditional endurance training.

Currently, several novel combinations of sprint interval training protocols are in use that have not been comprehensively investigated for the efficiency with which they affect metabolic responses and performance (23). Although the 12-wk sprint interval training might have similar adaptations with traditional endurance training program on the improvements in cardiometabolic health (22), the effects of HIIT10s (≤30 s) versus long-duration HIIT (HIIT60s; >30 s) on physiological adaptations and cardiorespiratory fitness are not thoroughly understood. The increase in glycolytic activity induced by 10 × 6-s all-out sprints performed with

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30-s recovery intervals on a cycle ergometer was reported to coincide with a 57% reduction in phosphocreatine degradation and a high level of muscle lactate; however, power output was not notably diminished during the final 6-s sprint, and this was because of an increase in aerobic metabolism (17). A few studies have demonstrated that HIIT involving 6–18 training sessions administered for 2–6 wk, with each session comprising 4–6 × 30-s cycling endeavors featuring a 4-min recovery period, is a time-saving exercise for rapidly inducing numerous metabolic adaptations, such as increases in muscle oxidative potential and endurance capacity in skeletal muscle (7,8,19). Another study showed that 10 × 6-s all-out sprints over an extremely short period resulted in a 10% improvement in 10-km time-trial performance (29). Regarding extremely short-duration sprinting, Trapp et al. (43) reported that active women who performed a maximum of 60 repetitions per session involving 8 s of intermittent sprints with a resistance of 0.5 kg and a 12-s recovery period for 20 min, 3 $d \cdot wk^{-1}$ for 15 wk, showed a 23.8% increase in maximal oxygen uptake ($\dot{V}O_{2max}$). Metcalfe et al. (36) also found that a 6 wk of very brief sprint interval training, consisted of 10 min of cycling at 60 W interspersed with 2 × 20-s all-out sprints, improved $\dot{V}O_{2max}$ for men (+15%) and women (+12%). Although certain studies showed that repeated ≤ 30-s all-out cycling interspersed with a 4-min recovery performed 3 $d \cdot wk^{-1}$ can enhance muscle activity and time-trial performance, the cardiorespiratory fitness (i.e., $\dot{V}O_{2max}$) and the anaerobic performance were not consistently improved after the HIIT10s (9,10).

Although the exercise intensity is substantially lower and the sprint duration is longer in HIIT60s than that in HIIT10s, a recent study (44) reported that HIIT60s is a potent stimulus for eliciting adaptations similar to those elicited by HIIT10s. Ziemann et al. (44) demonstrated that when 27 $min \cdot wk^{-1}$ of HIIT60s at an exercise intensity of 80% $\dot{V}O_{2max}$ was applied for 6 × 90-s bouts, followed by 180 s of rest, for a 6-wk period, $\dot{V}O_{2max}$ and power at AT were markedly increased in active male university students. Moreover, when 80%–90% of maximal workload was applied as an HIIT60s on a cycle ergometer for 3 $d \cdot wk^{-1}$ for 12 wk, with the HIIT60s including 6–10 × 60-s bouts interspersed with active recovery, fat oxidation was enhanced in sedentary women (1). A few previous studies have matched training protocols of distinct intensities for total work and frequency (15,28). However, most studies to date have presented comparisons of adaptations induced by interval training versus traditional continuous training of similar work volume; they have not compared adaptations induced by HIIT10s versus HIIT60s programs of interval training. Helgerud et al. (25) reported that using matched total work in 15/15 (15-s interval running at 90%–95% HR_{max}) and 4/4 (4 × 4-min interval running at 90%–95% HR_{max}) protocols resulted in a substantial increase in $\dot{V}O_{2max}$ and repeated sprint ability compared with the changes produced by slow, long-duration distance running and lactate threshold running intensity. The correlational data obtained thus far suggest that sprint training lasting from 8 s to 4 min might help enhance exercise capacity and blood metabolic profile. However, training studies are required to identify the matched-volume HIIT10s and HIIT60s programs and the training strategy involved that optimally improve both anaerobic and aerobic performance during a short-term training period. Specifically, no study to date has elucidated how using a matched volume of constant training intensities, but distinct sprint durations affects cardiometabolic factors and performance during high-intensity, repeated sprint performance, and graded endurance exercise. Therefore, the study was to examine the effects of a matched volume of HIIT10s and HIIT60s administered at an identical work-to-rest ratio (1:2) on physiological responses and metabolic variables during a graded exercise test (GXT) in active men. Here, the hypothesis tests revealed that HIIT10s (48 × 10-s sprint intervals with 20-s recovery) and HIIT60s (8 × 60-s intervals with 120-s recovery) programs yield similar benefits in cardiorespiratory and physiological adaptations compared with those of participants performing regular physical activity without HIIT.

METHODS

Participants. Forty-two young, healthy, recreationally active male university students were recruited to participate in this study; the students engaged in endurance training and leisure-time physical activity (e.g., badminton, basketball, and taekwondo) at least three times per week. A requirement of this study was that participants did not previously undertake a structured HIIT training program. To avoid any possible effects of sex differences on the physiological adaptations to the HIIT program, only males were selected as participants in this study. The participants were randomly allocated to one of three groups: HIIT60s, 14 students (mean ± SD: 21 ± 1 yr); HIIT10s, 14 students (21 ± 1 yr); and control (CON), 14 students (21 ± 3 yr). During the training period, four participants (one for HIIT60s, two for HIIT10s, and one for CON) dropped out of this study because of illness and injuries not related to this study. Each participant reviewed the study design and signed consent forms before participating in this study, which was approved by the institutional ethics committee and was conducted in accordance with the Helsinki Declaration. Regarding sample size considerations, a power of 0.80, a significance level of 0.05, and a 95% confidence interval (CI) necessitated a sample for each group of at least nine participants (16).

Experimental design. The experimental design comprised medical clearance and familiarization, baseline testing, and posttesting. Baseline aerobic and anaerobic performance and health parameters were determined before commencing the 4-wk training program. All groups completed testing at baseline and participated in a 4-wk training period following identical procedures. The participants engaged in training three times a week (on Mondays/Wednesdays/Fridays or on Tuesdays/Thursdays/Saturdays) of supervised HIIT for 4 wk at workloads equal to 85%–90% $\dot{V}O_{2max}$. After medical

clearance, the participants completed one to two familiarization sessions to become familiar with the GXT (represents aerobic capacity), 6 × 10-s repeated sprint test (RST; represents anaerobic capacity), and training regimes of HIIT60s or HIIT10s.

Baseline testing. On day 1, the participants reported to the laboratory and were asked to sit quietly in a chair for 20 min. The baseline resting arterial blood pressure was then measured and repeated on the upper arm of each participant by using an automated blood pressure device (YM1000 vital signs monitor; Mediana Technologies, San Antonio, TX). The average of these two values was recorded. For the GXT trial, the participants underwent a GXT on an electromagnetically braked cycle ergometer (Avantronic Cyclus II, h/p Cosmos, Germany). This test was used to determine $\dot{V}O_{2max}$, ventilatory parameters, and pulmonary gas exchange, which were measured using a Cortex Metamax 3B portable metabolic test system (Cortex Biophysik, Leipzig, Germany); the 15% O_2 and the 5% CO_2 analyzers were calibrated before each test by using standard gases of established concentrations in accordance with manufacturer guidelines. The ergometer seat height was individually adjusted to attain an appropriate angle of the bend in the knee at the lowest point in the pedal revolution. The seat of each participant was set at a consistent height for each training and testing session. Participants were allowed a 5-min warm-up period at an intensity of 70 W and a pedaling cadence of <60 rpm. Immediately after the warm-up, the participants began the $\dot{V}O_{2max}$ testing by cycling against progressively increasing workloads, with the resistance being increased by 30 $W \cdot min^{-1}$, and pedaling cadence was maintained at 60–70 rpm until the participants reached the point of volitional exhaustion. Maximal effort was considered when three of the following five criteria were achieved (39): a) a plateau in oxygen uptake defined as no expected increase higher than 150 $mL \cdot min^{-1}$, despite an increase in power output; b) a respiratory exchange ratio > 1.1; c) an HR ±10% of the age-predicted HR_{max} (210 − 0.65 × age); d) a blood lactate concentration >8 $mmol \cdot L^{-1}$; e) an RPE >19 on the Borg (6–20) Scale; and/or volitional exhaustion. The coefficient of variation (CV) for $\dot{V}O_{2max}$ for active populations in the laboratory was 2.6%. The $\dot{V}O_2$ data collected at each workload during the GXT were further analyzed using a simple linear regression to determine the exercise intensities (i.e., 30%, 85%, and 95% $\dot{V}O_{2max}$) of the training program. The oxygen pulse (O_2 pulse, mL per beat) is calculated as the ratio between the $\dot{V}O_2$ and the HR, which can represent the stroke volume (SV) of the heart and arteriovenous oxygen difference (5). As described previously, ventilatory threshold (VT, expressed as a percentage of $\dot{V}O_{2max}$) was determined as an increase in the ventilatory equivalent for oxygen ($\dot{V}_E/\dot{V}O_2$) with no associated increase in the ventilatory equivalent for carbon dioxide ($\dot{V}_E/\dot{V}CO_2$) (4). HR was monitored and recorded at 5-s intervals during the exercise (RX800 CX; Polar Electro, Finland). The Borg (6–20) Scale was used to assess the RPE at baseline and at the end of each stage until exhaustion.

After being allowed to recover for 3 d, the participants returned to the laboratory and were instructed to perform the 6 × 10-s maximal sprints with 60-s active recovery, the results of which yielded the anaerobic capacity presented here. Peak power output (PPO), mean power output (MPO), and percentage of sprint decrement (S_{dec}%) were measured during the operation of the electromagnetically braked cycle ergometer against a resistance equaling 0.075 kg per kilogram of body mass. Initially, the participants performed a standardized 10-min procedure, including a 5-min warm-up and three bouts of unloaded sprinting on a cycling ergometer, to prepare for the subsequent repeated sprints, followed by a 5-min routine stretching exercise. Participants were then instructed to begin pedaling as rapidly as possible against the inertial resistance of the ergometer, and the appropriate load was applied instantaneously. Verbal encouragement was provided throughout the 10-s sprint. During the 60-s recovery phases, the participants pedaled at 55–60 rpm against a load of 50 W for the active recovery and were instructed to prepare for the next sprint during the final 2 s. The S_{dec}% was calculated as follows (41): [1 − (sum of work done from Sprint $_1$ to Sprint $_6$) / (6 × work done in ideal Sprint)] × 100. The ideal sprint means the highest work performed among the six sprints. This RST was designed to induce the PCr degradation and anaerobic glycolysis system repeatedly (17). The CV for 6 × 10-s RST for active populations in the laboratory was 1.4%.

Body composition was determined by using the sum of three skinfold models and by following standardized procedures (26). Subcutaneous fat was measured twice at the chest, abdomen, and thigh in rotational order, typically on the right side of the body, with the participant standing in a relaxed posture. If the difference in measured values at each site is more than 1 mm, the third assessment would be performed.These measured values were used to calculate body density, which was then used to estimate the body fat percentage: [% body fat = (495/density) − 450]. Skinfold thickness was measured to the nearest millimeter by using Lange calipers (Cambridge Scientific Industries, Inc., Cambridge, MD), except in the case of low values, when it was measured to the nearest 0.5 mm. The entire procedure was performed in triplicate with an interval of at least 5 min between measurements. All skinfold measurements were handled by an experienced researcher certified as a health fitness instructor. The physical characteristics of the participants at pretraining (baseline), including age, height, body mass (BM), skinfold thickness, blood metabolite contents, and fitness level (i.e., $\dot{V}O_{2max}$), did not differ significantly among the three groups, as shown in Tables 1 and 2.

Training interventions. After the $\dot{V}O_{2max}$ test was administered, the participants were assigned to one of three groups: two training groups and one CON; the study was conducted for 4 wk, with three training sessions being held each week. Comprehensive calculations were performed to

equate the work volume for each of the training sessions. All sessions were performed at the same time of day.

1. HIIT10s: Each session consisted of 48 × 10-s cycling sprints at the power output corresponding to 85% $\dot{V}O_{2max}$ separated by 20 s of active recovery, which yielded a work-to-rest ratio of 1:2.
2. HIIT60s: Each session comprised 8 × 60-s cycling intervals at the power output corresponding to 85% $\dot{V}O_{2max}$ separated by 120 s of active recovery, which also yielded a work-to-rest ratio of 1:2.
3. CON: The participants maintained their daily physical activity and habitual diet but did not perform any HIIT.

In each training session, participants were asked to sprint at a given intensity as fast as possible during the exercise period (i.e., 10- or 60-s cycling) and to perform active recovery at an intensity corresponding to 30% $\dot{V}O_{2max}$ with a pedal cadence of 60–70 rpm. Training progression was accomplished by increasing the intensity of exercise from 85% $\dot{V}O_{2max}$ during the first two training weeks to 90% $\dot{V}O_{2max}$ for the final two training weeks. All participants completed the individual training protocols with at least 1 d of rest in between. Each training session included a 5-min warm-up period and a 3-min cooldown period at 30% $\dot{V}O_{2max}$. The total work completed per training session in the 4-wk HIIT program did not differ between the HIIT60s (213.7 ± 21.6 kJ) and the HIIT10s (226.6 ± 29.2 kJ) groups (P= 0.95). The training intensities in the HIIT60s were 232 ± 37 and 249 ± 39 W for 85% $\dot{V}O_{2max}$ and 90% $\dot{V}O_{2max}$, respectively; the training intensities in the HIIT10s were 237 ± 45 and 255 ± 47 W for the 85% $\dot{V}O_{2max}$ and 90% $\dot{V}O_{2max}$, respectively. No significant differences in training intensities or average peak HR (HIIT60s vs HIIT10s, 182 ± 8 vs 186 ± 7 bpm) were found between training conditions ($P > 0.05$). In addition, the average peak HR values under both training conditions were similar to HR_{max} during the GXT (HIIT60s, 182 ± 12 bpm; HIIT10s, 184 ± 9 bpm) with no significant differences ($P > 0.05$), suggesting the intensive nature of HIIT60s and HIIT10s.

Dietary notice and physical activity. The participants were asked to maintain their regular diet throughout the study period and to record daily food and drink intake before the GXT and the RST day. They were then asked to mimic this daily intake before all subsequent test days and to fast at least 3-h before engaging in the HIIT10s/HIIT60s training and exercise tests. One-day food and drink records were analyzed using nutritional analysis software (E-Kitchen Co., Taiwan). The dietary records comprised 62.5% carbohydrates, 14.5% protein, and 23% fat for each participant. The participants in the training and CON were asked to maintain their regular physical activity (apart from the training program) but to refrain from strenuous exercise during the 48 h immediately preceding the test days. The participants in the CON did not perform HIIT. On average, the participants engaged in recreational physical activity 2 to 3 $d \cdot wk^{-1}$ (approximately 6 $h \cdot wk^{-1}$) during the experimental period.

Blood collection. Blood samples for analyses were collected from the antecubital vein before and after training; samples were collected between 7:00 and 8:00 a.m. after overnight fasting (approximately 12 h) before the GXT trial. An 8-mL sample of blood was collected in a tube containing serum clot activator (Vacuette®, Switzerland) and centrifuged at 3000 rpm for 15 min; the samples were placed on ice during experiments and stored frozen at −80°C. The collected serum was subsequently analyzed for the levels of testosterone, cortisol, insulin, TG, total Chol, HDL-C, and LDL-C; the interassay CV values were 1%, 3.3%, 2.2%, 1.8%, 1.8%, 2.0%, and 2.1%, respectively. The remaining blood was placed in a tube (Vacuette®) containing k3-EDTA and later analyzed for glycosylated hemoglobin (HbAlc); the interassay CV was 1.1%. A whole-blood sample (approximately 1 μL) was collected from the earlobe during the GXT and 6 × 10-s RST. During the GXT, a whole-blood sample (approximately 1 μL) was collected from the earlobe before exercise and at 5 min after exercise for analyzing blood lactate concentrations and blood glucose concentrations. During the 6 × 10-s RST, the same blood sample was collected before, during every end-sprint, and at 5 min after exercise. Blood lactate concentrations were measured immediately using an automated analyzer (Lactate Pro™ LT-1730; Arkray KDK Corp., Japan), and blood glucose concentrations were measured using a portable device (Breeze®2, Bayer, Munich, Germany); the interassay CV values were 1.2% and 1%, respectively. Insulin resistance was assessed using the homeostatic model assessment for insulin resistance (HOMA-IR), which was computed as follows (40): ISI HOMA-IR = [fasting insulin ($\mu U \cdot mL^{-1}$) × fasting glucose ($mmol \cdot L^{-1}$)]/22.5. All assessments were performed at the same time (±1 h) of day within participants.

Statistical analysis. The Shapiro–Wilk normality test was performed to determine the homogeneity of all data. The aerobic exercise performance data (e.g., endurance time [time to exhaustion] during GXT), physiological parameters, and data on blood samples were analyzed using a two-way repeated-measures ANOVA (group [HIIT60s, HIIT10s, CON] × training [pre, post]) to investigate any significant differences between groups and between pretraining and post-training sessions, with Scheffe's *post hoc* test being used where necessary. A three-way repeated-measures ANOVA (group [HIIT60s, HIIT10s, CON] × training [pre, post] × repeated sprints [6 reps]) was used to detect differences for each anaerobic exercise performance (i.e., for PPO or MPO). Student's *t*-test was also used to test for significant differences in total work completed per training period between the two groups for the 4-wk program. Measures of reliability, presented as CV and intraclass correlation coefficients (ICC), were derived from the results of two-way repeated-measures ANOVA. The values of 95% CI for ICC were calculated using a previously reported method (35). Furthermore, a measure of effect size (Cohen's d) was calculated to compare the variables between the groups and the training effects. The thresholds for Cohen's d for small, moderate, and large effects

TABLE 1. Aerobic parameters among HIIT60s, HIIT10s, and CON groups at pretraining and posttraining (mean ± SD).

	HIIT60s (n = 13)		HIIT10s (n = 12)		CON (n = 13)	
	Pretraining	Posttraining	Pretraining	Posttraining	Pretraining	Posttraining
$\dot{V}O_{2max}$ ($L \cdot min^{-1}$)	3.5 ± 0.5	4.1 ± 0.8**	3.5 ± 0.8	4.1 ± 0.8**	3.6 ± 0.7	3.7 ± 0.7
$\dot{V}O_{2max}$ ($mL \cdot kg^{-1} \cdot min^{-1}$)	51.9 ± 9.2	61.4 ± 12.2*,**	52.6 ± 9.5	61.0 ± 9.6*,**	50.0 ± 6.8	52.4 ± 6.5
HR_{max} (bpm)	182 ± 11	180 ± 11	184 ± 9	183 ± 8	183 ± 11	188 ± 10
RPE (Borg's 6–20)	17 ± 1	18 ± 2	16 ± 1	17 ± 1	17 ± 1	17 ± 1
Blood lactate (mM)	9.6 ± 2.0	10.6 ± 2.0	9.0 ± 1.4	9.8 ± 1.9	9.4 ± 1.8	9.5 ± 1.8
O_2 pulse (mL per beat)	19.6 ± 2.9	23.6 ± 4.4*	19.8 ± 3.5	23.1 ± 4.7**	20.6 ± 4.1	19.8 ± 2.4
Endurance time (s)	573.2 ± 62.8	621.2 ± 46.7**	571.0 ± 79.5	624.0 ± 89.4**	579.7 ± 96.2	601.8 ± 76.2
$\dot{V}_E$ at $\dot{V}O_{2max}$ ($L \cdot min^{-1}$)	131.6 ± 21.4	138.7 ± 21.9	126.7 ± 27.2	137.9 ± 34.7	130.5 ± 27.7	131.6 ± 27.5
RER at $\dot{V}O_{2max}$	1.3 ± 0.1	1.3 ± 0.2	1.3 ± 0.1	1.2 ± 0.1	1.3 ± 0.2	1.3 ± 0.1
$\dot{V}O_2$ at AT ($L \cdot min^{-1}$)	2.2 ± 0.3	2.5 ± 0.4**	2.1 ± 0.3	2.5 ± 0.5**	2.3 ± 0.4	2.3 ± 0.3
$\dot{V}O_2$ at AT ($mL \cdot kg^{-1} \cdot min^{-1}$)	31.9 ± 5.6	36.6 ± 6.3**	31.5 ± 6.1	36.6 ± 6.4**	31.7 ± 5	32.5 ± 6.6
%$\dot{V}O_{2max}$ at AT (%)	57.7 ± 5.5	64.4 ± 7.9**	58.8 ± 9.3	61.6 ± 6.3	58.6 ± 6.5	61.9 ± 12.0

*Significantly different from CON.
**Significantly different from pretraining.

were defined as 0.20, 0.50, and 0.80, respectively (13). $P \leq 0.05$ was considered statistically significant. All data are presented as means ± SD.

RESULTS

Cardiorespiratory parameters and performance. A group–time interaction effect was identified in the case of $\dot{V}O_{2max}$ ($P = 0.03$). After the training interventions, $\dot{V}O_{2max}$ values significantly increased in the HIIT10s and HIIT60s groups ($P < 0.01$) but not in the CON group ($P = 0.87$). As compared with CON, the HIIT60s and HIIT10s interventions resulted in 18.4% ($d = 0.5$) and 17.9% ($d = 0.5$) increases in $\dot{V}O_{2max}$, respectively (Table 1). Although the endurance times measured in a GXT did not differ significantly among the groups ($P = 0.86$, $d = 0.2$), HIIT60s and HIIT10s produced an increase of 7.7% ($P = 0.01$, $d = 0.3$) and 8.5% ($P < 0.01$, $d = 0.3$) in endurance time at posttraining relative to the pretraining level, respectively. Moreover, O_2 pulse ($P < 0.01$), absolute $\dot{V}O_2$ at AT ($P < 0.01$), relative $\dot{V}O_2$ at AT ($P < 0.01$), and AT as a percentage of $\dot{V}O_{2max}$ ($P = 0.03$) in the HIIT60s or HIIT10s group or both groups were significantly higher at posttraining than at pretraining; however, no significant differences were detected in the other parameters among the HIIT60s, HIIT10s, or CON groups (Table 1). The corresponding ICC for $\dot{V}O_{2max}$ was 0.80 (95% CI = 0.61–0.90).

Anaerobic capacity. The PPO and the MPO values measured for the three group are presented in Figures 1 and 2. The three-way repeated-measures ANOVA revealed no significant interaction effects for group–training–repeated sprints in PPO ($P = 0.595$) or in MPO ($P = 0.187$), but significant interaction effects for training–repeated sprints were found in PPO ($P = 0.017$) and in MPO ($P = 0.05$). The PPO values in the HIIT60s significantly increased at sprint 4 ($P = 0.03$, $d = 1.1$) and sprint 6 ($P = 0.02$, $d = 1.1$) compared with the corresponding CON values; moreover, HIIT60s produced similar improvements in MPO at sprint 4 ($P = 0.05$, $d = 1.0$) and sprint 6 ($P = 0.05$, $d = 1.0$). By contrast, HIIT10s and CON induced no significant differences in PPO ($P > 0.05$, $d = 0.6$) or MPO ($P > 0.05$, $d = 0.5$). No significant interaction effect was found in S_{dec}% ($P = 0.31$). However, at posttraining, S_{dec}% was significantly different between HIIT60s and CON ($P = 0.04$, $d = 1.0$) but not between HIIT10s and CON ($P = 0.76$, $d = 0.4$) or HIIT60s and HIIT10s ($P = 0.56$, $d = 0.7$). The

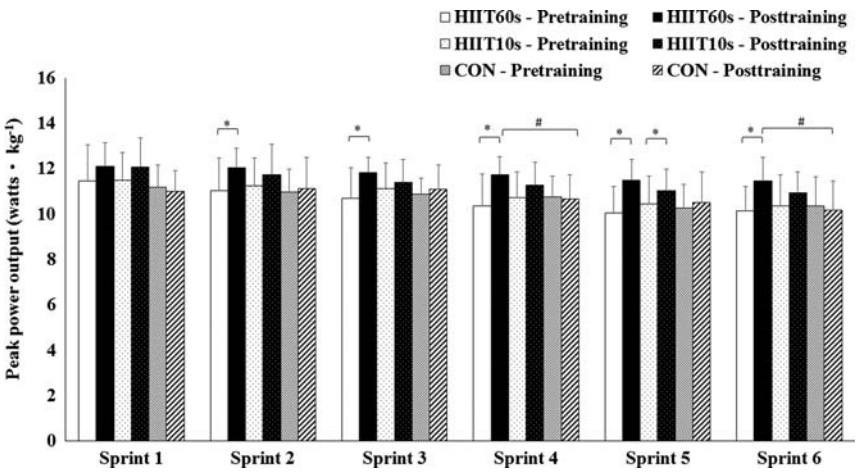

FIGURE 1—Changes in PPO (mean ± SD) between before and after training in HIIT60s, HIIT10s, and CON groups. #Significantly different from CON ($P < 0.05$). *Significantly different from pretraining ($P < 0.05$).

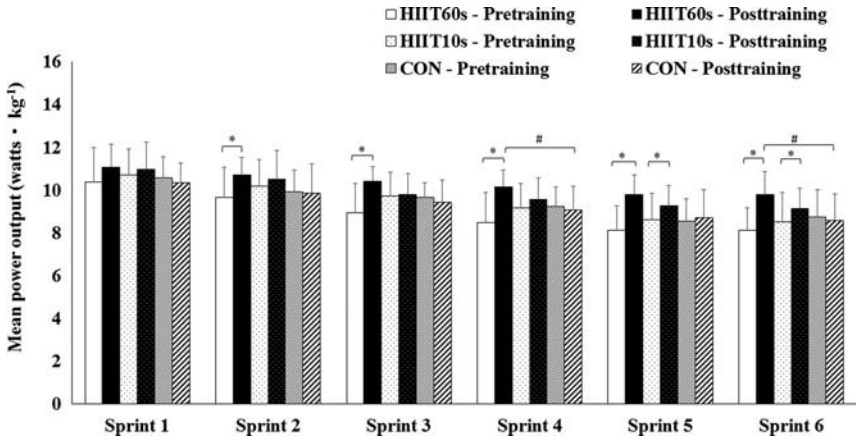

FIGURE 2—Changes in MPO (mean ± SD) between before and after training in HIIT60s, HIIT10s, and CON groups. #Significantly different from CON ($P < 0.05$). *Significantly different from pretraining ($P < 0.05$).

measured S_{dec}% values at pretraining versus posttraining were 13.7% ± 4.7% versus 7.1% ± 3.5% for HIIT60s, 11.1% ± 3.7% versus 10.0% ± 4.9% for HIIT10s, and 11.4% ± 7.9% versus 12.6% ± 7.0% for CON. Moreover, S_{dec}% was significantly lower at posttraining than at pretraining in the HIIT60s group, but not in the HIIT10s and CON groups. The ICC values were 0.93 (95% CI = 0.89–0.96) for PPO and 0.93 (95% CI = 0.89–0.96) for MPO.

Physiological characteristics. After 4 wk of HIIT intervention, no significant group–training interactions were detected in the case of systolic blood pressure (SBP) ($P = 0.33$), diastolic blood pressure (DBP) ($P = 0.56$), or BM ($P = 0.08$, $d = 0.5$), and no significant main effects were noted for time or group ($P > 0.05$). Although the results revealed no effects of group–training interaction in the case of skinfold thicknesses in the chest ($P = 0.86$) or thigh ($P = 0.31$), the effect measured for time was significant: skinfold thickness decreased at posttraining (relative to baseline) in both the chest ($P = 0.04$, d at HIIT60s and HIIT10s were 0.3 and 0.2) and the thigh ($P < 0.01$, d at HIIT60s and HIIT10s was 0.9). Skinfold thickness of the abdomen showed a significant interaction ($P = 0.04$) but again only decreased at posttraining (relative to baseline) in the HIIT60s ($d = 0.9$) and HIIT10s ($d = 0.6$) groups. The percent body fat was significantly lower at posttraining than at pretraining ($P < 0.01$) in the HIIT60s and ($d = 0.8$) HIIT10s ($d = 0.6$) groups but did not differ between the groups ($P = 0.63$, $d = 0.2$). In the case of the CON, none of the variables showed any significant change ($P > 0.05$, $d = 0.1$) at posttraining relative to pretraining. Detailed results are listed in Table 2.

Response of metabolic parameters to HIIT. The following blood metabolic parameters did not differ significantly among the HIIT60s, HIIT10s, and CON groups ($P > 0.05$): baseline insulin ($d = 0.3$), HbAlc ($d = 0.4$), HOMA-IR ($d = 0.2$), blood glucose ($d = 0.2$), TG ($d = 0.3$), total Chol ($d = 0.2$), HDL-C ($d = 0.2$), and LCL-C ($d = 0.2$). Moreover, these values at posttraining did not differ significantly from those at pretraining (Table 3). No training–group interaction was observed in the case of either testosterone ($P = 0.41$) or cortisol ($P = 0.87$) concentration, but significant main effects of training were observed for both testosterone ($P = 0.01$, $d = 0.5$) and cortisol ($P = 0.04$, $d = 0.6$), which showed that testosterone and cortisol concentrations were higher at posttraining than at pretraining (Table 3).

TABLE 2. Physical characteristics, blood pressure, and body composition among HIIT60s, HIIT10s, and CON groups at pretraining and posttraining (mean ± SD).

	HIIT60s ($n = 13$)		HIIT10s ($n = 12$)		CON ($n = 13$)	
	Pretraining	Posttraining	Pretraining	Posttraining	Pretraining	Posttraining
Height (m)	1.77 ± 0.06	1.76 ± 0.05	1.77 ± 0.09	1.77 ± 0.09	1.76 ± 0.05	1.76 ± 0.05
Body mass (kg)	68.4 ± 7.2	67.5 ± 6.3	67.9 ± 11.0	68.4 ± 9.9	71.9 ± 7.2	72.1 ± 7.3
BMI (kg·m^{-2})	21.8 ± 2.0	21.7 ± 1.8	21.5 ± 2.0	21.7 ± 1.8	23.1 ± 1.8	23.3 ± 1.6
SBP (mm Hg)	125 ± 7	126 ± 9	124 ± 9	123 ± 9	123 ± 14	127 ± 12
DBP (mm Hg)	67 ± 10	66 ± 8	67 ± 6	66 ± 8	71 ± 7	68 ± 8
Skinfold (mm)						
Chest*	9.4 ± 3.9	8.5 ± 3.0	9.4 ± 3.4	8.8 ± 3.3	9.6 ± 3.9	8.4 ± 3.7
Abdominal	15.2 ± 4.9	11.4 ± 3.5**	13.3 ± 3.3	11.5 ± 2.6**	14.6 ± 6.2	13.9 ± 6.5
Thigh	13.7 ± 3.4	10.9 ± 2.9**	11.6 ± 2.9	9.3 ± 2.0**	11.4 ± 3.7	10.5 ± 3.8
Body fat (%)	10.6 ± 3.4	8.3 ± 2.3**	9.3 ± 2.6	7.9 ± 2.1**	9.8 ± 4.3	9.3 ± 4.1

BMI, body mass index.
*Main effect identified for training ($P < 0.05$).
**Significantly different from pretraining.

TABLE 3. Blood metabolite contents among HIIT60s, HIIT10s, and CON groups at pretraining and posttraining (mean ± SD).

	HIIT60s (n = 13)		HIIT10s (n = 12)		CON (n = 13)	
Metabolite	**Pretraining**	**Posttraining**	**Pretraining**	**Posttraining**	**Pretraining**	**Posttraining**
Insulin ($\mu U \cdot mL^{-1}$)	7.9 ± 2.7	7.1 ± 2.3	7.2 ± 1.6	8.1 ± 3.	7.0 ± 1.5	6.8 ± 1.6
HbAlc (%)	5.5 ± 0.2	5.5 ± 0.1	5.3 ± 0.2	5.3 ± 0.2	5.4 ± 0.3	5.4 ± 0.3
HOMA-IR	1.9 ± 0.7	1.8 ± 0.6	1.7 ± 0.4	2.0 ± 0.8	1.7 ± 0.5	1.7 ± 0.5
Blood glucose ($mg \cdot dL^{-1}$)	95.8 ± 9.1	94.8 ± 4.9	99.9 ± 9.8	98.0 ± 8.1	96.6 ± 9.1	96.3 ± 7.7
TG ($mg \cdot dL^{-1}$)	58.5 ± 14.5	59.6 ± 13.8	50.7 ± 1.8	62.6 ± 19.9	54.6 ± 7.3	55.3 ± 19.8
Total Chol ($mg \cdot dL^{-1}$)	145.0 ± 28.5	151.2 ± 21.4	143.1 ± 29.7	150.3 ± 24.5	155.3 ± 29.4	148.6 ± 35.8
HDL-C ($mg \cdot dL^{-1}$)	54.9 ± 9.7	61.0 ± 11.2	56.2 ± 17.6	58.5 ± 11.1	56.2 ± 15.2	54.3 ± 14.2
LDL-C ($mg \cdot dL^{-1}$)	90.6 ± 19.8	89.0 ± 19.8	91.2 ± 23.6	90.0 ± 20.3	100.7 ± 22.1	90.6 ± 20.7
Testosterone ($ng \cdot dL^{-1}$)*	5.1 ± 1.5	5.2 ± 1.5	5.9 ± 1.7	6.7 ± 2.1	6.2 ± 1.4	6.8 ± 1.5
Cortisol ($ng \cdot dL^{-1}$)*	163.7 ± 51.3	179.4 ± 27.0	159.1 ± 34.7	184.8 ± 44.2	144.6 ± 25.8	153.9 ± 40.8

*Main effect identified for training ($P < 0.05$).

None of the blood lactate concentrations measured during the 6 × 10-s RST showed significant differences among the training groups at either pretraining or posttraining (P = 0.39, d = 0) (Fig. 3). Moreover, blood glucose concentrations were similar across all groups at baseline and after exercise (P = 0.19, d = 0.4). However, blood lactate and blood glucose concentrations after exercise were higher than at baseline.

DISCUSSION

This study produced a major novel result: when either one of two HIIT protocols matched in volume and frequency domain was administered for 4 wk (three times a week; including interval recovery, a total of 72 $min \cdot wk^{-1}$), 60 s of HIIT60s or 10 s of HIIT10s (both adhering to a work-to-recovery ratio of 1:2), $\dot{V}O_{2max}$, and $\dot{V}O$ at AT were enhanced to a larger extent than in the control group. Furthermore, the HIIT60s intervention yielded more favorable results in repeated sprint power output performance compared with the CON. Our results showed that 4 wk of HIIT60s and HIIT10s did not elicit changes in either physiological responses (in SBP, DBP, and BM) or blood responses (in baseline insulin, blood glucose, HbAlc, TG, total Chol, HDL-C, and LCL-C levels) in active young men. However, HIIT60s and HIIT10s resulted in respective reductions of 21% and 15% in percent body fat, 25% and 13% in skinfold thickness of the abdomen, and 21% and 19% in skinfold thickness of the thigh.

Both HIIT60s and HIIT10s exercise increased relative and absolute $\dot{V}O_{2max}$ by approximately 15%. This large increase after only 12 sessions of HIIT60s and HIIT10s at 85%–90% $\dot{V}O_{2max}$ training intensity for 4 wk is unexpected given that the volume of the aerobic component was considerably less than that of similar total work in the case of either moderate training at 70% HR_{max} for 8 wk (25) or steady-state exercise at 60% $\dot{V}O_{2max}$ for 15 wk (43). The HIIT60s and the HIIT10s protocols that amounted to 24 min per session, including recovery periods, have similar magnitudes of improvements in aerobic performance compared with the training length longer than 4 wk (25,43). Hazell et al. (24) demonstrated that various combinations of sprint interval training produced a 3.8%–9.3% increase in $\dot{V}O_{2max}$ after three sessions per week for 2 wk; by comparison, the protocol used in this study yielded a larger improvement in cardiorespiratory adaptation. Furthermore, the data obtained in this study revealed that both relative and absolute $\dot{V}O_2$ at AT were increased by 10.4% and 4.5% after HIIT60s and HIIT10s, respectively. Ziemann et al. (44) reported that $\dot{V}O_2$ at AT was meaningfully improved in active males after 6 × 90-s bouts at an exercise intensity of 80% $\dot{V}O_{2max}$, and

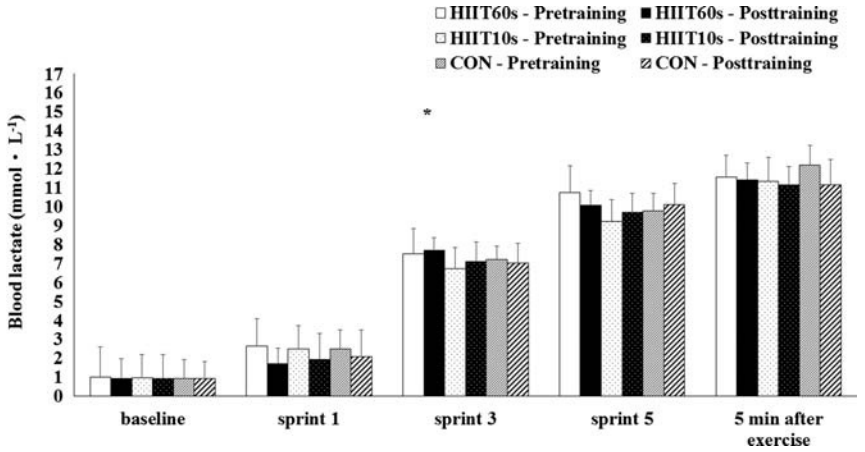

FIGURE 3—Blood lactate concentrations (mean ± SD) measured before and after training in HIIT60s, HIIT10s, and CON groups during the RST. *Significantly different across time ($P < 0.05$).

increases in the activity of glycolytic and muscle oxidative enzymes (38) and muscle buffering capacity (9) were implicated in these responses. Moreover, 4–7 × 30-s all-out sprint exercise (10) or 10 × 60-s cycling at 60% of peak power or both (27) upregulated peroxisome proliferator-activated receptor γ coactivator 1α, a potent regulator of mitochondrial biogenesis; in addition, they activated AMP-activated protein kinase and p38 mitogen-activated protein kinase signaling (21) in peripheral muscle tissue. Furthermore, the changes in $\dot{V}O_{2max}$ might be due to the increase in SV (5); the O_2 pulse was increased in this study, and the training-induced changes in O_2 pulse measured previously during exercise suggested that an elevation reflects SV alterations and an arteriovenous oxygen difference (25). Our study provides partial supports for a previous study (3), which found that the longer-duration (4-min) HIIT improved $\dot{V}O_{2max}$ to a greater extent compared with short-duration (1-min) HIIT and moderate-intensity continuous training program, most likely mediated through an increase in SV estimated by O_2 pulse.

PPO and MPO also increased after HIIT60s and HIIT10s programs but showed a marked improvement relative to the CON levels in the HIIT60s but not in the HIIT10s group. The anaerobic performance results obtained for distinct sprint durations in the low matched-volume exercise sessions used in this study might appear atypical; however, the results can be considered acceptable when they are compared closely with other findings related to responses to sprint durations in studies that involved comparatively longer sprints (≥30-s sprints). Previous studies have reported that the protein content of subunits (e.g., complex Π 70 kDa subunit and cytochrome *c* oxidase subunit IV in skeletal muscle), GLUT4, and neuromuscular-training adaptation outcomes were enhanced when the exercise regimen incorporated maximal-effort sprint bouts that lasted ≥30–60 s (11,33). By contrast, the effect of sprint training on the maximal activity of the enzyme phosphorylase increased and citrate synthase activity decreased after the administration of a repeated sprint training protocol involving short-duration sprints (<10-s sprints) for 6 wk (14). The energy for the first short-duration sprint lasting several seconds was provided by an equal contribution from creatine phosphate (CP) degradation and anaerobic glycolysis, and the energy for the subsequent repeated sprints was derived mainly from CP degradation and ATP resynthesis (17). The mechanisms responsible for the HIIT-induced increase in anaerobic sprint power could include increases in muscle CP concentration, anaerobic enzyme activity, and shifts from slow-twitch to fast-twitch muscle fibers (31). Thus, a sprint duration of 60 s coupled with 120 s of recovery is an optimal training protocol for active young men seeking to enhance repeated sprint ability during short-term HIIT. Collectively, these data suggest that long rather than short work intervals (e.g., 60- vs 10-s intervals) on a bicycle are preferable for active people attempting to improve sprint power performance, a view that is supported by a review of the literature (8).

In previous studies, similar reductions in fat mass and trunk fat were observed after a long-term (15 wk), high-intensity intermittent exercise training program (43) and a short-term (6 wk) sprint interval training program (34). Another study reported that the 2-wk HIIT program comprising 10 × 4-min intervals at approximately 90% of $\dot{V}O_{2max}$ increased activities of the muscle mitochondrial β-hydroxyacyl-CoA dehydrogenase, citrate synthase activity, and whole-body fat oxidation (42). However, a similar protocol in which the training period was extended to 6 wk further enhanced fat oxidation. This resulted from an increase in the rate of transfer across the muscle and mitochondrial membranes (e.g., fatty acid transport protein FAT/CD36) of the free fatty acid that is stored in peripheral adipose tissue and intramuscular TG (38). Moreover, the change observed in skeletal muscle capacity after seven sessions of HIIT for 2 wk was reported to be related to the increase in fat oxidation (42). In this study, there was no difference in carbohydrate or fat oxidation during the GXT in responses to training (data not reported). Furthermore, the concentrations of the products of lipid metabolism, such as TG and total Chol, did not differ significantly within or among the groups; however, the lipid profiles of each participant before training were already in the standard-value range. Thus, 4 wk of HIIT intervention might induce a posttraining loss in subcutaneous fat and whole-body fat, rather than induce a rapid change in blood lipid profiles in healthy people whose BM is in the standard range. The research behind the mechanisms is incomplete thus far, and more studies should be included to elucidate the skeletal muscle fat metabolism responsible for the decreases in body fat with HIIT. Previously, exercise-induced reductions in SBP and DBP were suggested to occur in response to long-term HIIT in healthy normotensive people (12). However, no changes were observed in SBP and DBP responses to 4 wk of HIIT, and this could be attributed to our study participants presenting a standard blood pressure value at pretraining and to the blood pressure being measured at rest rather than immediately after exercise. These findings support the view that in healthy active men, marked alterations in blood pressure might not be observed after short-term HIIT60s or HIIT10s.

Although HIIT60s and HIIT10s did not significantly affect resting concentrations of testosterone or cortisol, the post-training levels of these hormones in both groups were markedly higher than their pretraining levels. An elevated concentration of testosterone was shown to promote protein synthesis within the muscular system, but in contrast to testosterone, cortisol was reported to potentially lead to decreased muscle mass because of its catabolic effect (32). The observed lack of effect of either HIIT60s or HIIT10s on resting hormone levels in this study agrees with previous findings, which indicated that the potential beneficial effects produced by an HIIT-induced increase in testosterone concentrations resulted from stimulated red blood cell production and effects on neuroendocrine function, thus contributing substantially toward increasing muscular

power (45). Cortisol, which is commonly called a stress hormone, affects the metabolism of proteins and glucose. An elevated cortisol concentration might reflect exercise-induced stress and a disruption of homeostasis but can facilitate energy release and thus enhance performance. Here, the results did not observe any statistically significant differences in the testosterone and cortisol responses among the three groups. However, hormonal changes might arise because HIIT remains a potential influence that is not being fully understand. Moreover, given the widespread use of HIIT, additional research on this topic is warranted. Regarding insulin concentrations, HbAlc and HOMA-IR did not significantly change after 4 wk of training among the three groups. Fasting insulin concentrations and glucose remained unchanged in the young healthy males, which was a natural and expected outcome; however, the area under the plasma glucose and insulin responses to a 75-g oral glucose tolerance test might be improved after HIIT training (2). In addition, all the participants maintained a normal range in the TG and total Chol, whether pretraining or posttraining. A higher concentration of HDL-C may play a protective role against coronary atherosclerosis, and the HDL-C could be substantially ameliorated by longer periods of training, such as an 8-wk program of HIIT (37).

In conclusion, our results show that both HIIT60s and HIIT10s programs improve $\dot{V}O_{2max}$, oxygen uptake corresponding to the AT, and endurance time on a cycle ergometer during a GXT. Moreover, the HIIT60s protocol used here induced a notable improvement in both PPO and MPO and reduced sprint decrement in active men when administered for 4-wk. Although the 4-wk HIIT60s and HIIT10s programs did not markedly affect lipid outcomes or hormone levels in the active and health male university students, the training regimens has a time-efficient manipulation decreased skinfold thickness in the abdomen and thigh and the percent body fat. These data indicate that lipid metabolism did not respond to short-term HIIT60s or HIIT10s, which could be attributed to either an insufficient training period or the healthy condition at baseline of the adults enrolled in this study. Consequently, the magnitude of improvement in aerobic performance and partial skinfold thickness was clearly higher in the HIIT60s and HIIT10s groups than that in the CON group, but repeated sprint ability was improved only in the HIIT60s group relative to the CON group. These results suggest that HIIT60s might be desirable for inducing large, rapid changes in anaerobic repeated sprint performance in active people shortly after initiating exercise training.

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