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ABSTRACT

Purpose: To compare the metabolic, cardiorespiratory and perceptual responses to three isoenergetic high-intensity interval exercise (HIIE) protocols of different bout duration and an isoenergetic continuous exercise protocol. **Methods**: Eleven healthy males (age, 28±6 y) performed four 20-min cycling trials of equal mean power output one week apart. Participants cycled either continuously (CON) or intermittently with 10s (HIIE10), 30s (HIIE30), or 60s (HIIE60) bouts at intensities corresponding to 49% (CON) or 100% of power at peak oxygen uptake (VO_2peak). Recovery intervals during the HIIE trials were 15, 45, and 90s, respectively. **Results**: Average VO_2 was similar in the HIIE trials (2.29±0.42, 2.20±0.43, and 2.12±0.45 $L \cdot min^{-1}$, for HIIE10, HIIE30 and HIIE60, respectively); whereas, in CON (2.02±0.38 $L \cdot min^{-1}$), it was lower than HIIE10 (p=0.002) and HIIE30 (p=0.043). Average pulmonary ventilation (V_E) was higher in HIIE60 compared to HIIE10, HIIE30, and CON (75.8±21.8 vs. 64.1±14.5, 64.1±16.2, and 54.0±12.5 $L \cdot min^{-1}$, respectively, $p<0.001$). The peak values and oscillations of VO_2 and V_E in HIIE60 were higher compared to all other trials ($p<0.001$). Blood lactate concentration was higher in HIIE60 compared to HIIE10, HIIE30, and CON from the 5^{th} min onward, reaching 12.5±3.5, 7.2±2.1, 7.9±2.9, and 4.9±1.6 $mmol \cdot L^{-1}$, respectively, at the end of exercise ($p<0.001$). Rating of perceived exertion (RPE) was higher and affective responses were lower in HIIE60 compared to all other trials toward the end of exercise ($p<0.001$). **Conclusions**: These findings highlight the importance of bout duration in HIIE, since shorter bouts resulted in attenuated metabolic and cardiorespiratory responses, lower RPE and feelings of displeasure compared to a longer bout, despite equal total work, duration, and work-to-recovery ratio. These results may have implications for the prescription of HIIE in various populations. **Key Words:** HEART RATE, LACTATE, OXYGEN UPTAKE, PLEASURE-DISPLEASURE, RESPIRATORY FREQUENCY, VENTILATION

INTRODUCTION

High-intensity interval exercise (HIIE) is very popular among highly trained and physically active individuals due to its effectiveness in improving cardiorespiratory and metabolic fitness despite a low exercise volume (1–4). The most common HIIE protocols require either short bouts (e.g., 1 min x 10 repetitions) with recovery of equal duration or longer bouts (e.g., 4 min x 4 repetitions) with 3 min of recovery performed at intensities between 80 and 100% of maximal oxygen uptake (VO_2max) (3–6). Both types of protocols cause similar metabolic and physiological responses, characterized by near-maximal activation of aerobic metabolism and high anaerobic contribution from glycolysis (5), and they are effective in inducing cardiovascular and mitochondrial adaptations (7–9). However, due to the severity of metabolic perturbations, HIIE is deemed to be unpleasant and less tolerable compared to moderate-intensity continuous exercise (10).

Inherent in HIIE is the separation of intense exercise into shorter bouts, interspersed with active or passive recovery intervals. Early work (11) showed that when a certain power output is generated intermittently rather than continuously, the physiological strain is lower and the tolerable work is increased. A study using bilateral knee extension performed in a 3T magnetic resonance scanner showed that when a given volume of intermittent supramaximal exercise (≈ 110% of peak power) was divided into shorter bouts (16 instead of 64 s) while maintaining a work-to-recovery ratio of 1:2, the contribution of glycolysis was diminished despite a similar average external power and total ATP turnover (12). The authors suggested that, by manipulating the work and recovery durations, the magnitude of intramuscular metabolic perturbations can be dissociated from the

external power (12). They also reported a significantly lower absolute oxygen uptake (VO_2) during the shorter compared to the longer bouts of exercise (12).

In contrast, during 60 min of intermittent cycling at 70% of peak power output (PPO), with bouts lasting 30, 60 or 120 s (work-to-recovery ratio of 1:1), average VO_2 was similar, while the fluctuation of VO_2 increased with increasing bout duration (13). However, the contribution of anaerobic metabolism was minimal, as indicated by the blood lactate concentration (BLa) which ranged between 2 and 3.5 $mmol \cdot L^{-1}$ (13). Data showing that the metabolic and cardiorespiratory responses to HIIE may be manipulated by changing bout duration are sparse (11, 12), and only few studies have examined protocols of very short bout duration (14, 15). In addition, our understanding of ventilatory control during exercise and, especially during HIIE is limited, with only one study (to our knowledge) examining the regulation of pulmonary ventilation (V_E) and its components, i.e., respiratory frequency (f_R) and tidal volume (V_T) during intense exercise (16). Importantly, fluctuations of VO_2, V_E and f_R have been associated with perceptual and affective responses during HIIE (10, 17) and, thus, may be significant regulators of exercise tolerance, as well as of the degree of physiological and metabolic adaptations. While most studies examining the physiological responses to HIIE use an equal duration of work and recovery (13), a ratio of 1:1.5 has been used in some studies (15, 18), which may allow greater degree of recovery and in turn, greater fluctuation of physiological parameters.

The aim of this study was to compare the metabolic, cardiorespiratory, and perceptual responses to three isoenergetic HIIE protocols of different bout duration and equal work-to-recovery ratio, as well as to an isoenergetic continuous bout. We hypothesized that differences in

bout duration would modify the cardiorespiratory, metabolic, perceptual and affective responses to the four protocols despite identical total duration and average exercise intensity.

METHODS

Participants. Eleven healthy males (mean ± SD age, 28 ± 6 y; height, 1.77 ± 0.07 m; body mass, 70.4 ± 10.6 kg; body fat, 9.4 ± 2.3 %) volunteered to participate in the study. Participants were recreationally active (performing light to moderate-intensity activities, such as jogging, 2-3 times per week) and had not participated in any organized sports training program for at least 6 months before testing. They were also characterized as "moderately active" according to the results of the long form of the International Physical Activity Questionnaire (19) (total score 2105 ± 1120 MET-min·wk^{-1}). Prior to data collection, a medical history questionnaire was completed, and all participants were screened by a physician for cardiorespiratory conditions that would preclude participation. Participants were free of musculoskeletal injuries for at least one year prior to the study, were non-smokers, and were not taking any drugs or nutritional supplements. Written informed consent was obtained after a thorough verbal and written description of possible discomforts and risks. The study was approved by the Aretaieion Hospital Ethics Committee (B-153/04-02-2016), and all procedures were in accordance with the Code of Ethics of the World Medical Association (Helsinki declaration of 1964, as revised in 2013).

An a priori power analysis was performed using G∗Power (version 3.1.9.2; Kiel University, Kiel, Germany). A sample size of 10 was calculated as needed to detect a medium effect size (partial eta squared, or η^2, of 0.137), based on a power of 0.80, alpha of 0.05, and correlation coefficient of 0.5 between repeated measures.

Experimental design. A repeated-measures design was used to examine the effects of exercise protocol on metabolic, cardiorespiratory, affective, and perceptual responses to interval and continuous exercise. Following familiarization and preliminary exercise testing, participants performed four trials, spaced one week apart, in random and counterbalanced order. In each trial, participants cycled for 20 min at a mean power output equal to 49% of PPO attained during the VO_2peak test. In one trial, participants cycled at a constant intensity (CON), while, in the other three trials, intensity alternated between 100% and 15% of PPO for either 10 and 15 s (HIIE10), 30 and 45 s (HIIE30), or 60 and 90 s (HIIE60), respectively, resulting in an exercise-to-recovery ratio of 1 to 1.5 (Fig. 1). The primary dependent variables included the following cardiorespiratory and metabolic responses: VO_2, carbon dioxide output (VCO_2), pulmonary ventilation (V_E), tidal volume (V_T), respiratory frequency (f_R), respiratory exchange ratio (RER), heart rate (HR) and BLa concentration. The secondary dependent variables were rating of perceived exertion (RPE) and affective valence.

Preliminary tests and familiarization. Participants completed four preliminary sessions, three to four days apart. In the first session, participants were familiarized with continuous and alternating-intensity cycling by performing 10 min of incremental cycling until HR reached 70-80% of age-predicted maximum (220 – age). Then, after 3 min of rest, they cycled for 8 min, alternating between the highest power achieved during the preceding incremental trial and 30 W every 30 s. In the second session, anthropometric measurements were obtained, and VO_2peak was measured using an incremental cycling protocol to exhaustion. Also, participants completed the long form of IPAQ for the assessment of habitual physical activity. In the third session, a submaximal graded cycling test was performed, while, in the last session, participants were

familiarized with HIIE by completing a 9-min HIIE bout, which included 3 min of HIIE10, HIIE30, and HIIE60 in that order.

Anthropometric measurements. Body height was measured to the nearest 0.5 cm using a stadiometer (Charder HM200P Portstad, Charder Electronic, Taichung City, Taiwan), and body mass was measured to the nearest 0.1 kg (TBF-300A Body Composition Analyzer, Tanita, Tokyo, Japan). Seven skinfold thickness measurements were obtained using Harpenden skinfold calipers (British Indicators, West Sussex, UK) for the estimation of body fat (20).

VO_2peak test. VO_2peak was measured during an incremental test to exhaustion on an electronically braked cycle ergometer (Ergo bike premium 8i, Daum Electronic, Fürth, Germany), where power was independent of cycling frequency, which was set at 70 rpm. Starting power ranged from 30 to 40 W and was increased by 20 to 30 W every minute until volitional exhaustion (attained within 8 to 13 min). Gas exchange data were monitored breath-by-breath using a metabolic system interfaced with a computer (MedGraphics ULTIMA, MedGraphics, St. Paul, MN). The flow sensor was calibrated using a 3-L syringe, and the gas analyzers were calibrated with two precision gas mixtures (16% O_2, 4% CO_2, 80% N_2; and 21% O_2, 79% N_2) before each test. Respiratory data were averaged every 10 s, and VO_2peak represented the mean of the highest two VO_2 measures. PPO was defined as the average power output corresponding to the time interval at which VO_2peak was attained. HR was continuously measured by telemetry (Polar S410, Kempele, Finland) and averaged every 5 s (also during all experimental trials). VO_2peak attainment was declared if at least three out of the following five criteria were satisfied: (a) inability to maintain the required pedaling frequency of 70 rpm (drop below 50 rpm), (b) RER > 1.15, (c)

HR within 10 $b.min^{-1}$ of the age-predicted maximum, (d) a plateau in VO_2, i.e., change of less than 150 $mL \cdot min^{-1}$ despite an increase in power, and (e) RPE > 17.

Submaximal graded test. The submaximal graded cycling test consisted of five 4-min stages at 40%, 50%, 60%, 75%, and 90% of PPO. Respiratory data were monitored continuously as described above and were averaged during the last minute of each stage. Also, BLa was measured in capillary blood at the end of each stage using the Lactate Scout+ portable analyzer (EKF Diagnostics, SensLab, Leipzig, Germany). Lactate threshold was determined as the exercise intensity (as percentages of HRpeak and VO_2peak) corresponding to a BLa equal to 4 mmol/L, based on the curvilinear relationship between blood lactate and exercise intensity (21).

Experimental trials. Participants were asked to abstain from exercise and to repeat their habitual diet for 24 h before each trial. They reported to the laboratory between 8 and 10 am in a hydrated state and after an overnight fast. Each trial commenced with 3-min of cycling at 15% of PPO (warm-up), followed by 3 min of passive rest on the cycle ergometer. The main part of each trial included 20 min of cycling at a mean power output equal to 49% of PPO, at a cadence of 70 rpm, which was maintained by the participants using both visual (on a display) and audio feedback (through a metronome). For the HIIE trials, power was alternated automatically by increasing or decreasing the resistance applied by the electronically braked cycle ergometer. Cardiorespiratory responses were continuously monitored during each trial, including 3 min before and 3 min after exercise, using the same equipment as during the VO_2peak test. Respiratory data were averaged every 10 s, and HR data were averaged every 5 s.

The average, highest and lowest values, as well as the oscillations (highest minus lowest value) of all respiratory parameters (VO_2, VCO_2, V_E, V_T, R_f, RER) and HR were calculated every 2.5 min, to include the same duration of exercise (60 s) and the same duration of recovery (90 s) in all trials. Also, the times spent above the intensity corresponding to the lactate threshold, above 70%, 80%, 85%, 90%, and 95% of VO_2peak, as well as above 80%, 85%, 90% and 95% of HRpeak were determined (22, 23).

BLa was measured in capillary blood before and every 5 min during each trial, using the same analyzer as in the submaximal test. RPE on a 6-20 rating scale was obtained before and every 5 minutes during each trial. Also, affective valence (11-point Feeling scale evaluating pleasure/displeasure) was obtained at the same time points (24). Participants were familiarized with these scales in the preliminary sessions.

Statistical analysis. All statistical analyses were performed using SPSS (IBM SPSS Statistics, version 23). The distribution of all variables was found not to differ significantly from normal according to the Shapiro-Wilk test. Results were analyzed using 2-way (trial by time) analysis of variance (ANOVA) for repeated measures. Cardiorespiratory, BLa, and perceptual data were analyzed using 2-way ANOVA (4 trials x 8 time points for cardiorespiratory data and 4 trials x 5 time for BLa, RPE and affective valance). For average VO_2, time spent above the lactate threshold and above certain percentages of VO_2peak and HRpeak, a one-way ANOVA for repeated measures was used. When a significant interaction or main effect was observed, Tukey's post-hoc test was used to locate significant differences between means. Effect sizes for main effects and interactions were determined by η^2 values, which were classified as small (0.01 to 0.058), medium

(0.059 to 0.137), or large (>0.137) according to Cohen (1988). For pairwise comparisons, effect size was determined by Hedges' *g* (small, 0.01 to 0.49; medium, 0.50 to 0.79, large, ≥ 0.8). Correlation was examined through Pearson's correlation analysis. Data are presented as mean ± standard deviation. Statistical significance was set a priori at $\alpha = 0.05$.

RESULTS

Respiratory gases. VO_2peak of the participants was equal to 47.1 ± 6.6 $ml \cdot kg^{-1} \cdot min^{-1}$ (3.28 ± 0.70 $L \cdot min^{-1}$) and was attained at 242 ± 50 W with a HR equal to 192 ± 7 $b \cdot min^{-1}$. Peak values of V_E, V_T, and f_R during the VO_2peak test were equal to 134 ± 31 $L \cdot min^{-1}$, 2.61 ± 0.31 $L \cdot min^{-1}$, and 51.8 ± 8.3 $breaths \cdot min^{-1}$.

The time course of VO_2 during exercise is presented in Fig. 2A. One-way ANOVA for average VO_2 showed a significant and large trial effect ($p = 0.003$, $\eta^2 = 0.38$). Post-hoc tests revealed that VO_2 did not differ significantly between all three HIIE trials (2.29 ± 0.42, 2.20 ± 0.43, and 2.12 ± 0.45 $L \cdot min^{-1}$ for HIIE10, HIIE30 and HIIE60, respectively, corresponding to 70.3 ± 4.1%, 67.7 ± 4.6%, and 65.3 ± 3.7% of VO_2peak); whereas, that in the CON trial (2.02 ± 0.38 $L \cdot min^{-1}$, or 62.4 ± 6.9% VO_2peak) was lower than those in HIIE10 and HIIE30 ($p = 0.002$ and 0.043, $g = 0.64$ and 0.44, respectively).

Two-way ANOVA on peak VO_2 within each 2.5-min interval showed significant trial and time effects ($p < 0.001$, $\eta^2 = 0.66$ and 0.91, respectively), but no interaction ($p = 0.08$). As shown in Table 1, peak VO_2 increased gradually until the middle of all trials (10.0-12.5 min) and remained relatively stable thereafter. The overall peak VO_2 value (average of all peaks) for HIIE60 was

significantly greater compared with all other trials ($p < 0.001$, g = 1.48 to 1.81). The overall peak VO_2 values for HIIE10 and HIIE30 did not differ, while they were both greater than CON (p = 0.034 and 0.026, g = 0.57 and 1.04, respectively) (Table 1).

VO_2 oscillations within each 2.5-min interval exhibited significant trial and time effects ($p < 0.001$, η^2 = 0.93 and 0.55, respectively), as well as a trial x time interaction ($p < 0.001$, $\eta^2 = 0.28$). Post-hoc tests showed that, in CON and HIIE10, oscillations were small and remained similar across time (2.1 ± 1.0 and 2.0 ± 0.7 $ml \cdot kg^{-1} \cdot min^{-1}$, respectively). Oscillations in HIIE30 (4.3 ± 2.5 $ml \cdot kg^{-1} \cdot min^{-1}$) were greater than in CON and HIIE10 (p = 0.036 and 0.048, g = 1.11 and 1.16, respectively), but remained unchanged over time. Oscillations in HIIE60 increased over time (from 16.1 ± 4.3 to 20.5 ± 5.9 $ml \cdot kg^{-1} \cdot min^{-1}$, $p < 0.001$, g = 1.19) and were greater than in all other trials ($p < 0.001$ and g = 3.52 to 4.37).

Changes in VCO_2 during exercise and recovery are presented in Fig. 2B. Two-way ANOVA on peak VCO_2 within each 2.5-min interval showed significant trial and time effects ($p < 0.001$, η^2 = 0.77 and 0.76, respectively), but no interaction (p = 0.31). Overall peak VCO_2 was greater in HIIE60 compared with HIIE10 and HIIE30 (3.03 ± 0.72 vs. 2.45 ± 0.46 and 2.42 ± 0.44 $L \cdot min^{-1}$, respectively, p = 0.001, g = 0.91 and 1.47, respectively). In addition, overall peak VCO_2 in CON (2.15 ± 0.38 $L \cdot min^{-1}$) was lower than the values in all three HIIE trials ($p < 0.001$, g = 0.65 to 0.70).

VCO_2 oscillations within each 2.5-min interval showed significant trial and time effects ($p < 0.001$, η^2 = 0.87 and 0.63, respectively), as well as a trial x time interaction ($p < 0.001$, $\eta^2 = 0.28$).

Post-hoc tests showed that oscillations were small in CON and HIIE10 (< 0.13 $L \cdot min^{-1}$). However, in HIIE30, oscillations (0.27 ± 0.15 $L \cdot min^{-1}$) were greater than in CON and HIIE10 ($p < 0.001$, $g = 1.18$ and 1.22, respectively), while oscillations in HIIE60 (1.20 ± 0.46 $L \cdot min^{-1}$) were greater than in all other trials ($p < 0.001$, $g = 3.13$ to 3.28).

Peak RER within each 2.5-min interval showed significant trial and time effects ($p < 0.001$, $\eta^2 = 0.53$ and 0.29, respectively), as well as a trial x time interaction ($p = 0.005$, $\eta^2 = 0.17$). Peak RER remained stable over time within each trial ($p > 0.05$, Fig. 2C). However, overall peak RER was greater in HIIE60 compared with CON and HIIE10 (1.18 ± 0.16 vs. 1.00 ± 0.05 and 1.03 ± 0.06, respectively, $p < 0.001$, $g = 1.48$ and 1.22). Peak RER was similar in CON and HIIE10 ($p = 0.83$), but was lower than HIIE30 (1.11 ± 0.10, $p = 0.001$, $g = 1.24$).

Pulmonary ventilation. The time course of V_E is presented in Fig. 3A. Average V_E within each 2.5-min interval showed significant trial and time effects ($p < 0.001$, $\eta^2 = 0.77$ and 0.84, respectively), as well as a trial x time interaction ($p < 0.001$, $\eta^2 = 0.36$). Although VO_2 was similar between HIIE trials, V_E was greater in HIIE60 compared with HIIE10 and HIIE30 (75.8 ± 21.8 vs. 64.1 ± 14.5 and 64.1 ± 16.2 $L \cdot min^{-1}$, respectively, $p < 0.001$, $g = 0.59$ and 0.61). V_E during CON (54.0 ± 12.5 $L \cdot min^{-1}$) was lower than in response to all HIIE trials ($p < 0.001$, $g = 0.67$ to 1.18).

Peak V_E within each 2.5-min interval showed significant trial and time effects ($p < 0.001$, $\eta^2 = 0.77$ and 0.85, respectively) and a trial x time interaction ($p < 0.001$, $\eta^2 = 0.47$). As shown in Table 1, values increased over time until the middle of each trial (10.0-12.5 min) and remained

unchanged thereafter. Overall peak V_E was greater in HIIE60 compared with all other trials (p < 0.001, g = 0.88 to 1.40) and overall peak V_E in HIIE10 and HIIE30 was greater than CON (p < 0.01, g = 0.71 and 0.75, respectively).

V_E oscillations within each 2.5-min interval (Fig. 3A) showed significant trial and time effects (p < 0.001, η^2 = 0.86 and 0.75, respectively) and a trial x time interaction (p < 0.001, η^2 = 0.57). Oscillations were small in CON and HIIE10 (< 4.0 $L \cdot min^{-1}$). Oscillations in HIIE30 (10.2 ± 5.3 $L \cdot min^{-1}$) were greater than in CON and HIIE10 (p < 0.001, g = 1.68 and 1.78, respectively), while oscillations in HIIE60 (34.5 ± 17.0 $L \cdot min^{-1}$) were several-fold greater than in all other trials (p < 0.001, g = 1.86 to 2.51).

Respiratory frequency. The time course of f_R is presented in Fig. 3B. Average f_R within each 2.5-min interval showed significant trial and time effects (p < 0.001, η^2 = 0.71 and 0.91, respectively) and a trial x time interaction (p < 0.001, η^2 = 0.27). Post-hoc tests showed that average f_R was greater in HIIE60 compared with HIIE10 and HIIE30 (31.6 ± 7.1 vs. 29.1 ± 4.7 and 27.9 ± 5.4 $breaths \cdot min^{-1}$, respectively, p = 0.034 and 0.001, g = 0.40 and 0.57, Fig. 3B). Average f_R during CON (24.3 ± 4.3 $breaths \cdot min^{-1}$) was lower than all HIIE trials (p < 0.001, g = 0.71 and 1.04).

Peak f_R within each 2.5-min interval showed significant trial and time effects (p < 0.001, η^2 = 0.81 and 0.90, respectively) and a trial x time interaction (p < 0.001, η^2 = 0.46). As shown in Table 1, values increased over time until the middle of each trial and remained unchanged thereafter. Overall peak f_R was greater in HIIE60 compared with all other trials (p < 0.001, g =

0.93 to 1.54, Table 1). Overall peak f_R in HIIE10 and HIIE30 was greater than CON ($p < 0.01$, $g = 0.71$ and 0.92, Table 1).

Oscillations in f_R within each 2.5-min interval showed significant trial and time effects ($p < 0.001$, $\eta^2 = 0.77$ and 0.47, respectively), as well as a trial x time interaction ($p < 0.001$, $\eta^2 = 0.22$). Oscillations were small in CON and HIIE10 (< 2.0 breaths·min^{-1}). Oscillations in HIIE30 (4.3 ± 2.5 breaths·min^{-1}) were greater than in CON and HIIE10 ($p < 0.001$, $g = 1.31$ and 1.35, respectively), while oscillations in HIIE60 were greater than in all other trials (20.0 ± 5.5 breaths·min^{-1}; $p < 0.001$, $g = 3.54$ to 4.48, Fig. 3B).

Tidal volume. The time course of V_T is presented in Fig. 3C. Average V_T within each 2.5-min interval showed only a trial effect ($p = 0.049$, $\eta^2 = 0.23$) with no significant time effect or interaction. However, post-hoc tests revealed that average V_T was similar in all trials (1.97 ± 0.37, 1.96 ± 0.32, 2.04 ± 0.32, and 2.10 ± 0.29 L·min^{-1}, for CON, HIIE10, HIIE30 and HIIE60, respectively, $p > 0.05$). Likewise, peak V_T within each 2.5-min interval exhibited only a trial effect ($p < 0.001$, $\eta^2 = 0.39$), with post-hoc tests showing that the overall peak V_T was higher in HIIE60 than in all other trials ($p = 0.007$ to 0.046, $g = 0.42$ to 0.65).

V_T oscillations within each 2.5-min interval showed only an effect of trial ($p < 0.001$, $\eta^2 = 0.87$). Oscillations were small in CON and HIIE10 (< 0.14 L·min^{-1}). However, oscillations in HIIE30 (0.27 ± 0.18 L·min^{-1}) were greater than in CON and HIIE10 ($p < 0.001$, $g = 0.94$ and 1.04, respectively), while oscillations in HIIE60 (0.65 ± 0.26 L·min^{-1}) were greater than all other trials ($p < 0.001$, $g = 1.51$ to 2.45).

Heart rate. The time course of HR during exercise is presented in Fig. 4. Average values every 2.5 min showed significant trial and time effects ($p < 0.006$, $\eta^2 = 0.37$ and 0.91, respectively) and a trial x time interaction ($p = 0.003$, $\eta^2 = 0.23$). Average HR was similar in all three HIIE trials (153 ± 18, 152 ± 16, and 155 ± 17 bpm for HIIE10, HIIE30, and HIIE60, respectively), corresponding to 80 ± 7%HRpeak. Average HR during CON (148 ± 17 bpm, or 77 ± 7%HRpeak) was lower than that during HIIE60 ($p = 0.003$, $g = 0.39$, Fig. 4).

Peak HR within each 2.5-min interval exhibited significant trial and time effects ($p < 0.001$, $\eta^2 = 0.71$ and 0.90, respectively) and a significant trial x time interaction ($p < 0.001$, $\eta^2 = 0.30$). As shown in Table 1, values increased over time until the middle of each trial (10.0-12.5 min) and remained unchanged thereafter. Overall peak HR was significantly higher in HIIE60 compared with all other trials ($p < 0.001$, $g = 0.72$ to 0.99, Table 1).

Oscillations of HR within each 2.5-min interval showed significant trial and time effects ($p < 0.001$, $\eta^2 = 0.93$ and 0.66, respectively) and a significant trial x time interaction ($p < 0.001$, $\eta^2 = 0.53$). Values were small in CON and HIIE10 (< 3 bpm). However, oscillations in HIIE30 (7 ± 5 bpm) were greater than in CON and HIIE10 ($p < 0.001$, $g = 1.40$ and 1.51), while oscillations in HIIE60 were several-fold greater than in all other trials (24 ± 7 bpm; $p < 0.001$, $g = 2.59$ to 4.30).

Time spent at different intensity zones. Time spent above the lactate threshold (which corresponded to 61.7 ± 10.9% VO_2peak and 77.1 ± 7.7% HRpeak) exhibited no trial effect ($p = 0.39$). Specifically, the time spent above the relative exercise intensity corresponding to the lactate threshold, when expressed as %VO_2peak, was 10.7 ± 8.0, 14.2 ± 6.4, 12.4 ± 6.3, and 10.9 ± 4.6

min ($p > 0.05$), while, when expressed as %HRpeak, it was equal to 10.6 ± 6.8, 11.3 ± 7.6, 10.7 ± 7.0, and 12.1 ± 6.5 min ($p > 0.05$), for CON, HIIE10, HIIE30 and HIIE60, respectively.

Time spent above 70% (t70), 80% (t80), 85% (t85), 90% (t90) and 95% VO_2peak (t95) showed significant trial effects ($p = 0.01$ to 0.04, Fig. 5A). t70 was longer in HIIE10 compared with CON ($p < 0.001$, $g = 1.53$; Fig. 5), and t80, t85, t90, and t95 were longer in HIIE60 compared with all other trials ($p < 0.001$, $g = 0.94$ to 2.46).

Similarly, time spent above 80% (t80), 85% (t85), 90% (t90), and 95% HRpeak (t95) showed significant trial effects ($p = 0.002$ to 0.04, Fig. 5B). t80 and t85 were longer in HIIE60 compared with CON ($p < 0.05$, $g = 0.61$ and 0.86, respectively), while t90 and t95 were longer in HIIE60 compared with all other trials ($p < 0.05$, $g = 0.82$ to 1.78).

Blood lactate concentration: BLa showed significant effects of trial and time ($p < 0.001$; $\eta^2 = 0.85$ and 0.91, respectively) and a trial x time interaction ($p < 0.001$; $\eta^2 = 0.76$). BLa increased over time in all trials ($p < 0.01$), except in CON, where it remained unchanged after the 5th minute of exercise (Fig. 6A). Also, BLa was higher in HIIE60 compared to HIIE10, HIIE30 and CON from the 5th min onward, reaching 12.5 ± 3.5, 7.2 ± 2.1, 7.9 ± 2.9, and 4.9 ± 1.6 mmol·L^{-1}, respectively, at the end of exercise ($p < 0.001$, $g = 0.66$ to 2.97). There was no significant difference in BLa between HIIE10 and HIIE30.

Ratings of perceived exertion and affective responses: RPE exhibited main effects of trial and time ($p < 0.001$; $\eta^2 = 0.33$ and 0.92, respectively), as well as a trial x time interaction (p

< 0.001; $\eta^2 = 0.35$). The post-hoc tests showed that RPE increased similarly in CON, HIIE10, and HIIE30 until the 10th minute and did not change significantly thereafter (Fig. 6B). However, RPE was higher in HIIE60 than in all other trials at 10, 15 and 20 min of exercise ($p < 0.001$, $g = 0.73$ to 1.58). RPE during CON, HIIE10, and HIIE30 was similar. RPE was highly correlated with f_R in each HIIE trial ($r = 0.86$. 0.82, and 0.77 for HIIE10, HIIE30, and HIIE60, respectively; $p < 0.01$ for all) as well as when all HIIE trials were pooled together ($r = 0.78$, $p < 0.001$).

Ratings of affective valence (pleasure-displeasure) showed significant main effects of trial and time ($p = 0.007$, $\eta^2 = 0.32$, and $p < 0.001$, $\eta^2 = 0.54$, respectively), as well as a significant trial x time interaction ($p < 0.001$; $\eta^2 = 0.32$; Fig. 6C). Affective valence decreased 5 min after the start of exercise and remained stable thereafter in CON, HIIE10, and HIIE30. However, in HIIE60, affective valence decreased over time until the 15th minute of exercise and was lower compared with all other trials at the 15th and 20th min of exercise ($p < 0.02$ to 0.01, $g = 0.57$ to 1.06).

DISCUSSION

We found that during a 20-min HIIE session, a decrease in exercise bout duration from 60 s to 30 s to 10 s resulted in attenuated fluctuations and lower peaks of cardiorespiratory parameters, accompanied by lower BLa, RPE, and aversive affective responses. These effects were observed despite equal total work and similar average VO_2, V_T, HR, and time spent above the lactate threshold in all three HIIE trials (Figs 2 to 4). Thus, it appears that physiological, metabolic, perceptual, and affective responses to HIIE may be manipulated by changing bout duration while maintaining all other parameters identical (i.e., exercise intensity, total exercise and recovery duration, exercise-to-recovery ratio, total mechanical work). Specifically, reducing bout duration

from the commonly used 60 s to 10 s attenuates the fluctuations and peak values of all cardiorespiratory parameters (Figs 2 to 4), thus reducing the severity of metabolic (e.g., BLa), perceptual, and affective responses by more than 40% (Fig. 6). Thus, by altering bout duration while keeping neuromuscular load and average VO_2 identical, we can adjust the acute metabolic and cardiorespiratory strain according to one's physical fitness and training goals.

Previous studies examining fluctuations in VO_2 and BLa responses to short interval exercise used submaximal exercise intensities which elicited low BLa (<6 $mmol \cdot L^{-1}$) (13–15). In contrast, the high intensity (100% PPO) used in the present study induced BLa greater than 6 $mmol \cdot L^{-1}$ when bout duration was 10 s and up to 12 $mmol \cdot L^{-1}$ when bout duration increased to 60 s (Fig. 6). Similar large differences in BLa have been reported between HIIE bout durations of 30 and 180 s (26). BLa may serve as an index of the contribution of glycolysis and the associated metabolic disturbances during HIIE (7). Indeed, glycolytic contribution to ATP supply is very low and phosphocreatine contribution predominates during HIIE bouts of 16 s or 32 s, compared with bouts of 64 s (work-to-recovery ratio of 1:2) during work-matched bilateral knee extension exercise performed at 110% of peak aerobic power (12). This result may be due to greater usage of oxygen stored in myoglobin during short bouts of HIIE (11, 27) and, thus, lower reliance on glycolysis, as indicated by the lower BLa in the shorter HIIE bouts (Fig. 6).

The magnitude of metabolic disturbances is considered a regulator of chronic responses and adaptations to HIIE (7, 8). It has been suggested that exercise-induced accumulation of ions, such as Ca^{2+}, H^+, lactate, P_i and AMP, within skeletal muscle induce signaling cascades regulating mitochondrial biogenesis (7, 28, 29). In contrast, a previous study reported that metabolic acidosis

may reduce the amount of mRNAs encoding proteins related to mitochondrial biogenesis (30), which has also been demonstrated in vitro (31). Although the effect of metabolic acidosis on mitochondrial adaptations is not clear, the widely different muscle metabolic milieu between the longer (HIIE60) and shorter protocols (HIIE10 and HIIE30), as indicated by differences in BLa, may elicit dissimilar peripheral adaptations to HIIE (32, 33). Furthermore, previous studies have shown that, during HIIE, large fluctuations in oxygen uptake, rather than global energy expenditure, are key factors for improving muscle oxidative capacity and VO_2max (33, 34). Based on these results, practitioners may prescribe HIIE protocols that induce different adaptations by manipulating the contribution of glycolysis and the fluctuations of cardiorespiratory parameters through bout duration while maintaining the same high intensity and total work.

In this study, large differences were observed in the fluctuations of most cardiorespiratory parameters between HIIE trials. It is noteworthy that in HIIE10, VO_2 did not fluctuate throughout exercise and was sustained at a value 10-15% above the lactate threshold, while in HIIE30, fluctuations were evident, but the peaks did not exceed the VO_2 of HIIE10 (Fig. 2A). Thus, protocols involving shorter duration bouts may serve as a means of training close to the lactate threshold, although with a much higher neuromuscular load (i.e., 100% PPO). HR continued to increase over time in all trials and followed fluctuation patterns similar to VO_2. The smaller fluctuations of VO_2 and HR when bout duration was decreased confirm previous results with lower-intensity exercise (70% PPO) (13) and demonstrate that large reductions in peak values and oscillation amplitudes of VO_2 and HR may be attained depending on the structure of a HIIE session while keeping total work, intensity, and work-to-recovery ratio the same.

In addition to VO_2 and HR, other respiratory parameters were affected by bout duration. Specifically, V_E reached peak values during the HIIE60 trial that were approximately 40-50% higher than in the other two HIIE protocols and almost 70% higher than in CON (Table 1). Interestingly, peak V_T was similar in response to CON, HIIE10, and HIIE30, yet was 8-11% higher in HIIE60. Changes in f_R were most pronounced during HIIE60, especially after 10 min of exercise (30-35% higher peak f_R than in the other HIIE trials), contributing largely to the increases in V_E. These findings may have implications for modifying the structure HIIE protocols when the target is to stimulate (or to progressively load) the respiratory system in healthy populations and in individuals with various respiratory diseases (35). The mechanism behind the rapid increase in f_R in the second half of HIIE60 may be the greater degree of metabolic acidosis, as reflected by the increased BLa (Fig. 6), which enhances stimulation of the carotid bodies resulting in a respiratory compensation for the increased H^+ production (36). Thus, the greater metabolic acidosis in HIIE60 was possibly related with the higher f_R and V_E compared with the other trials.

Besides the physiological differences between trials, it appears that perceptual responses are also impacted by bout duration. Consistent with the results of the present study, there is evidence that RPE is higher with longer bout duration, and this may influence enjoyment and adherence to this type of exercise, especially in previously sedentary and unfit individuals (37, 38). A higher RPE may reflect greater metabolic and cardiorespiratory disturbances and increased discomfort. In agreement with previous studies (38–40), feelings of displeasure were greater in HIIE60 compared to the other trials. These findings may have considerable implications for the use of HIIE protocols in different populations, as they show that RPE and feelings of displeasure during exercise may be manipulated by changing bout duration even while exercising at a maximal

intensity (100% PPO) and maintaining the same total work. Previous work shows that affective responses closely match the severity of homeostatic perturbation (41). A recent study from the same research group showed that affective valence mirrored changes in VO_2 during repeated 3 and 5 min HIIE trials (10). The design of the present study, where shorter bouts of exercise were performed, does not allow us to examine this concept. However, it has been suggested that during interval exercise, f_R reflects physiological strain better than other parameters traditionally used (e.g., VO_2 or HR) (17), and in addition, f_R is strongly associated with RPE during HIIE and CON (16, 17). These results support our data in which strong correlations were found between f_R and RPE, and suggest that changes in f_R may be a promising monitoring tool of the magnitude of physiological and perceptual strain during HIIE. Overall, our data indicate that fluctuations in f_R and changes in metabolic stress, as reflected by BLa, may be more related to affective responses to HIIE than changes in VO_2.

Our findings also reveal that RPE and affective valence during HIIE may be manipulated by changing bout duration. By modifying bout duration, practitioners may adjust not only metabolic and physiological stress, but also the perceptual and affective responses to HIIT, thus fine-tuning load progression and introducing healthy and diseased individuals to the benefits of HIIE more efficiently. It is possible that HIIT in the form of very short bouts may be used as a "gateway" to intense exercise, while "harder" HIIE protocols may be introduced later in a progressive manner, enabling periodization of HIIT and promoting adherence and future engagement to exercise (42, 43). One advantage of the work-to-recovery ratio used in this study (1:1.5) is that it enables a greater degree of restoration of energy sources and metabolic

disturbances between bouts, thus delaying large increases in RPE and declines in affective responses, in contrast with studies using a 1:1 work-to-recovery ratio (38, 44–46).

This study had some limitations. First, data can only be generalized to young, healthy men performing HIIE on a cycle ergometer. Changing the size of exercising muscle mass leads to different metabolic and perceptual responses during HIIE (47); thus, it remains to be shown whether results will be similar in response to HIIE performed on the treadmill. In addition, our "long" exercise bout was only 1 min despite the 4 x 4 protocol being widely used in various populations (48, 49). Further studies are needed to compare responses to repeated 4-min bouts versus shorter ones.

CONCLUSIONS

In conclusion, the findings of the present study highlight the importance of bout duration in HIIE, since shorter bouts attenuated cardiorespiratory and metabolic responses, as well as RPE and feelings of displeasure, compared to a longer bout, despite equal total work, duration, and work-to-recovery ratio. Very short HIIE bouts induce lower cardiorespiratory and metabolic strain along with more positive perceptual responses. These differences may enable manipulation of the metabolic and cardiorespiratory load during HIIE, which may be useful for introducing individuals to HIIE or as means of load progression or periodization. Additionally, shorter bouts of HIIE could be preferred when prescribing interval exercise to unfit or overweight adults, or to individuals with cardiorespiratory or metabolic diseases who may be intolerant to severe cardiorespiratory or perceptual responses.

Disclosure of funding

No funding to declare.

Conflict of interest

The authors declare no conflict of interest. The results of the present study do not constitute endorsement by ACSM and are presented clearly, honestly, and without fabrication, falsification, or inappropriate data manipulation.

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FIGURE LEGENDS

Figure 1. Schematic representation of the four experimental trials. PPO, peak power output during the VO_2peak test.

Figure 2. Time course of oxygen uptake (expressed as %VO_2peak, A), VCO_2 (B), and respiratory exchange ratio (C) during the four protocols. Data are mean ± SD of time-aligned 10-s averages. Dotted line in A represents the lactate threshold. Inserts depict averages of 2.5-min intervals.

Figure 3. Time course of pulmonary ventilation (A), respiratory frequency (B), and tidal volume during exercise (C). Data are mean ± SD of time-aligned 10-s averages. Inserts depict averages of 2.5-min intervals.

Figure 4. Time course of heart rate (expressed as % HRpeak) during the four protocols. Data are mean ± SD of time-aligned 5-s averages. Dotted line represents the lactate threshold.

Figure 5. Time spent above certain percentages of VO_2peak (A) and HRpeak (B). †$p < 0.05$ from CON, #$p < 0.001$ from all other trials at the same zone.

Figure 6. Blood lactate concentration (A), ratings of perceived exertion (RPE, B) and affective responses (feeling scale, C) before and during exercise. †$p < 0.01$ from HIIE10, #$p < 0.01$ from HIIE30, *$p < 0.01$ from CON.

Figure 1

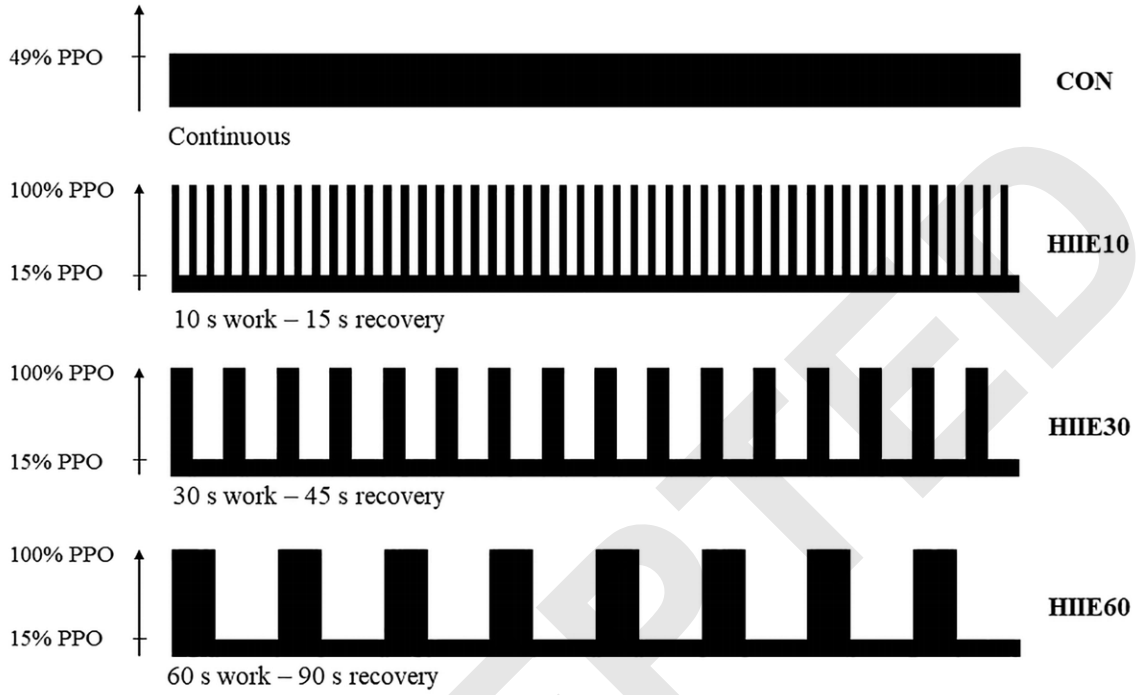

Figure 2

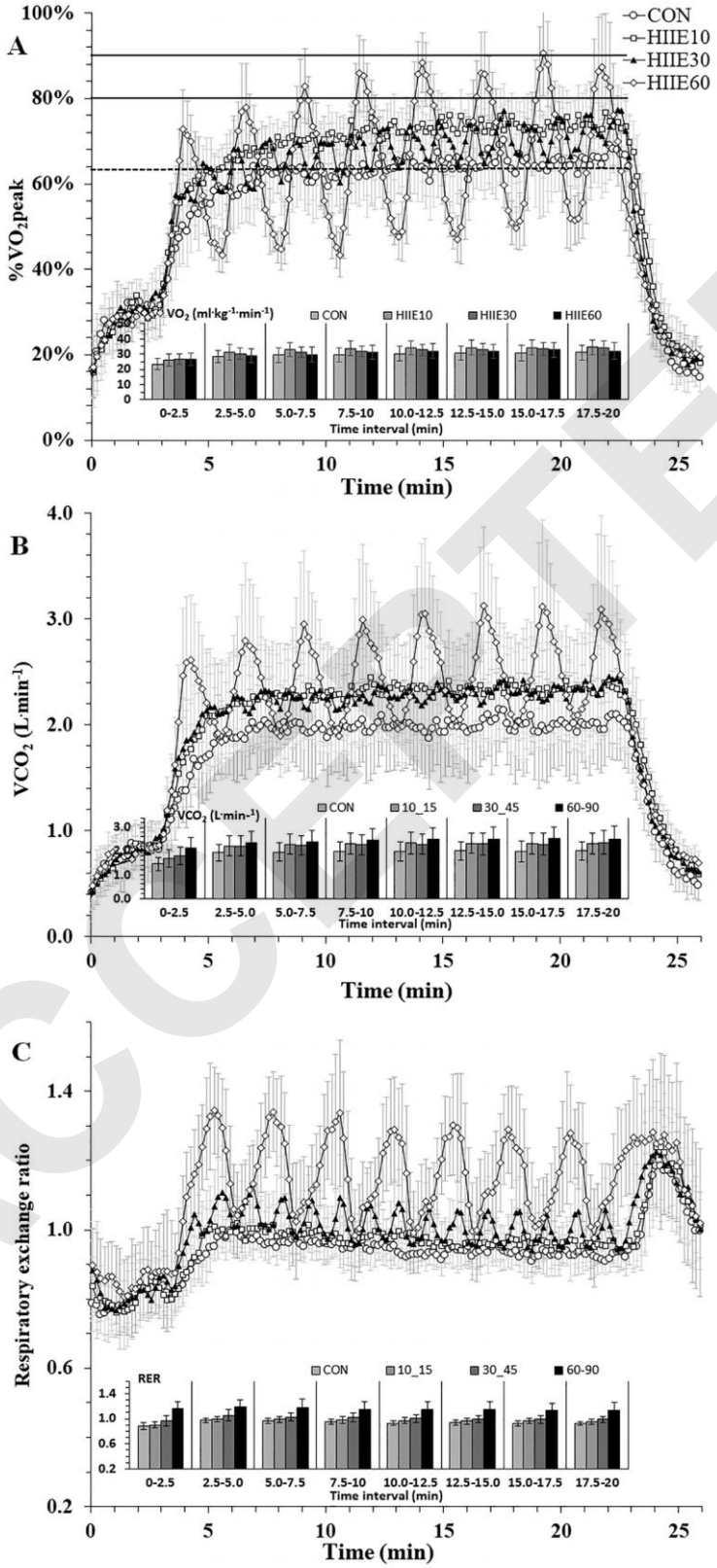

Figure 3

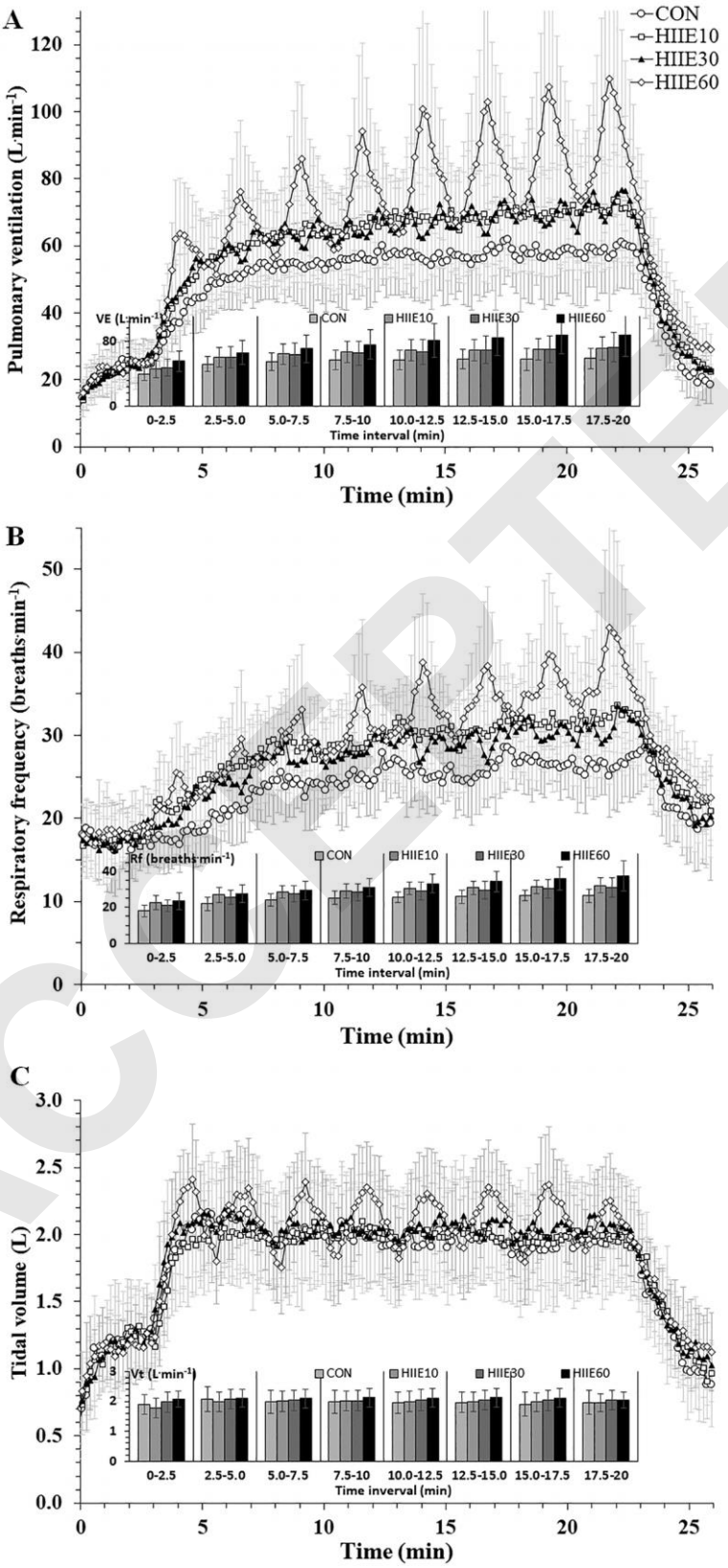

Figure 4

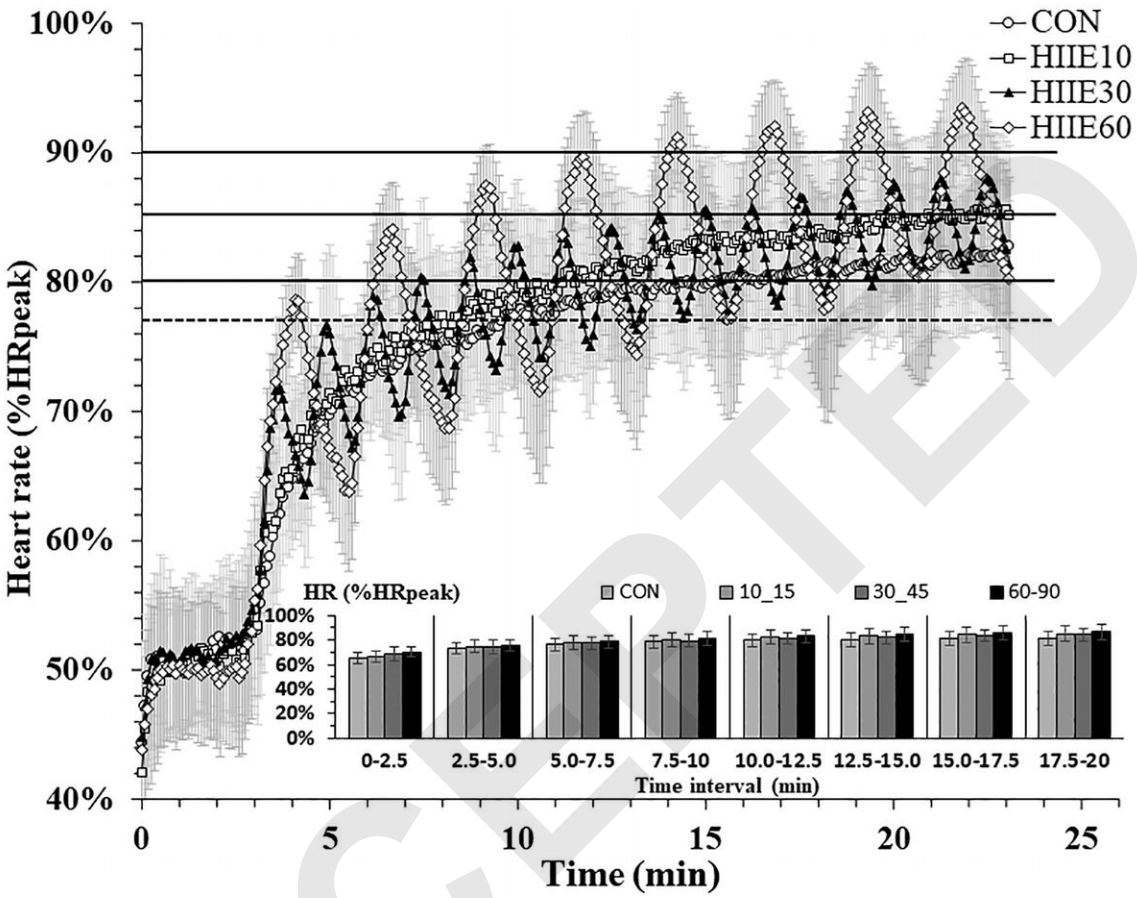

Figure 5

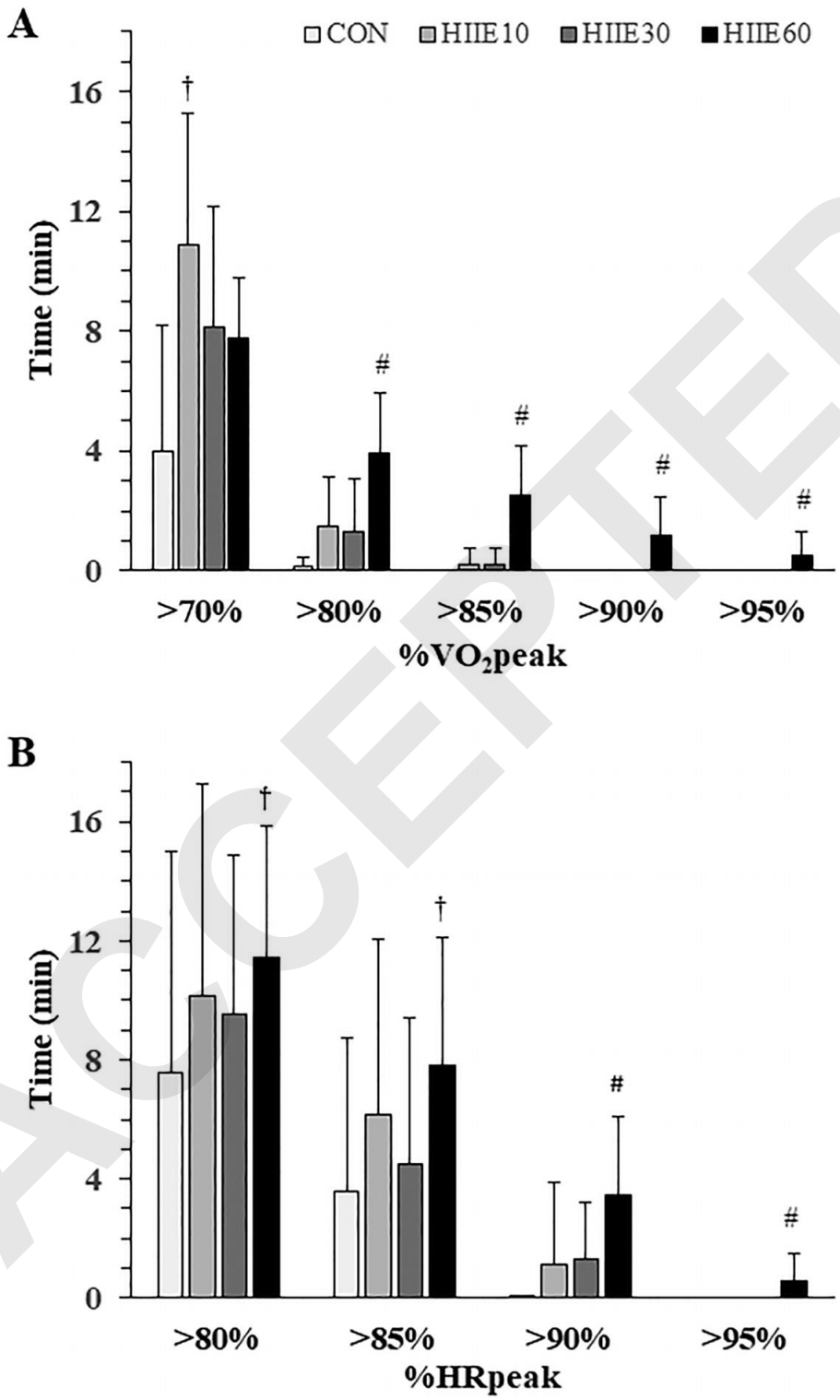

Figure 6

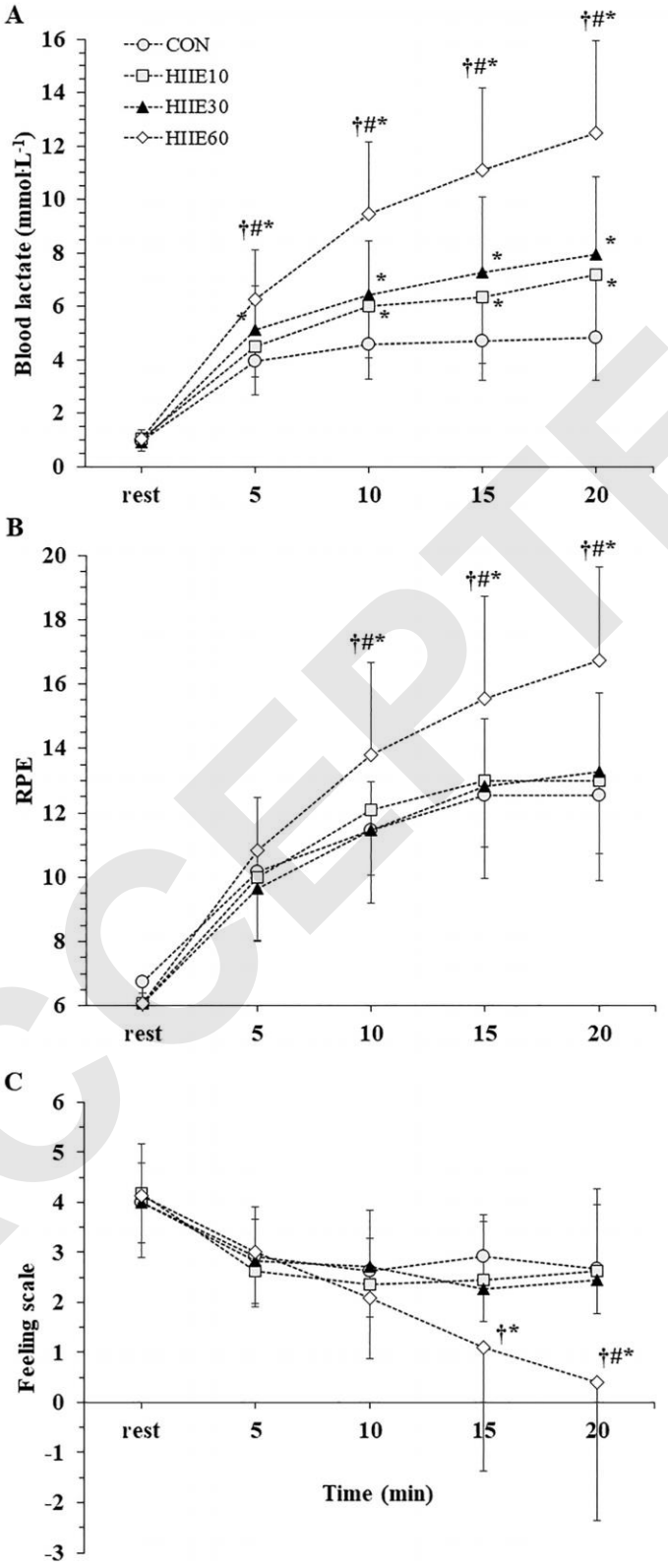

Table 1. Peak values of oxygen uptake (VO_2), pulmonary ventilation (V_E), tidal volume (V_T), respiratory frequency (f_R), and heart rate (HR), calculated within each 2.5-min interval of the 20-min bouts and overall in the four experimental trials

		Interval (min)								
		0-2.5	**2.5-5.0**	**5.0-7.5**	**7.5-10.0**	**10.0-12.5**	**12.5-15.0**	**15.0-17.5**	**17.5-20.0**	**Overall**
VO_2 ($\%VO_2peak$)	**CON**	60.0 ± 6.9	67.6 ± 8.5*	69.2 ± 8.4*	68.4 ± 6.5*	71.7 ± 8.6*	72.9 ± 8.3	71.4 ± 7.1	72.3 ± 5.9	**69.2 ± 8.3**
	HIIE10	66.1 ± 3.5	72.6 ± 3.8*	75.9 ± 3.3*	78.1 ± 4.4*	78.9 ± 4.7*	78.6 ± 4.4	79.3 ± 4.	81.4 ± 3.7	**76.4 ± 6.0†**
	HIIE30	65.4 ± 4.1	70.9 ± 6.3*	71.5 ± 6.8*	73.6 ± 6.1*	75.8 ± 7.3*	74.3 ± 5.9	76.4 ± 5.7	78.3 ± 4.6	**73.3 ± 6.8†**
	HIIE60	75.3 ± 8.2	80.9 ± 9.5*	84.9 ± 7.8*	88.2 ± 9.1*	91. ± 7.2*	88.9 ± 8.?	91.7 ± 9.1	90.5 ± 10.2	**86.4 ± 9.9#**
HR ($\%HRpeak$)	**CON**	71.5 ± 4.4	75.8 ± 4.8*	78.2 ± 5.3*	79.9 ± 5.1*	80.8 ± 5.0*	81.6 ± 5.3	82.5 ± 5.2	82.7 ± 5.5	**79.1 ± 6.1**
	HIIE10	71.2 ± 5.0	76.5 ± 5.5*	79.6 ± 6.1*	81.5 ± 5.8*	83.6 ± 5.9*	84.2 ± 6.2	85.3 ± 5.6	86.0 ± 6.0	**81. ± 7.3**
	HIIE30	74.6 ± 4.9	77.6 ± 4.9*	79.7 ± 5.4*	81.4 ± 4.8*	82.7 ± 5.3*	83.5 ± 5.7	84.5 ± 5.3	85.6 ± 5.3	**81.2 ± 6.1**
	HIIE60	77.8 ± 3.3	83.2 ± 3.9*	86.3 ± 3.4*	88.5 ± 3.9*	89.5 ± 4.1*	90.1 ± 4.4	90.4 ± 4.6	90.9 ± 4.6	**87.1 ± 5.8#**
V_E ($L{\cdot}min^{-1}$)	**CON**	48.1 ± 8.7	57.0 ± 9.4*	59.5 ± 10.8*	60.8 ± 12.6*	62.1 ± 13.2*	63.8 ± 11.5	62.6 ± 13.5	65.2 ± 13.8	**59.9 ± 12.4**
	HIIE10	55.9 ± 13.3	64.3 ± 12.2*	69.1 ± 12.6*	71.4 ± 13.8*	72.6 ± 14.0*	72.6 ± 12.9	75.3 ± 13.2	77.4 ± 14.0	**69.8 ± 14.3†**
	HIIE30	57.3 ± 13.0	67.3 ± 15.7*	69.9 ± 17.0*	72.4 ± 16.6*	73.8 ± 15.4*	75.1 ± 18.0	77.2 ± 18.7	80.3 ± 20.0	**71.7 ± 17.6†**
	HIIE60	65.3 ± 16.9	77.9 ± 20.8*	88.6 ± 23.0*	95.4 ± 27.1*	104.0 ± 29.9*	106.0 ± 30.8	109.4 ± 33.7	114.0 ± 41.1	**95.1 ± 31.9#**
V_T ($L{\cdot}min^{-1}$)	**CON**	2.26 ± 0.35	2.31 ± 0.42	2.21 ± 0.40	2.16 ± 0.40	2.20 ± 0.35	2.17 ± 0.37	2.08 ± 0.41	2.20 ± 0.43	**2.20 ± 0.38**
	HIIE10	2.11 ± 0.37	2.20 ± 0.33	2.18 ± 0.36	2.24 ± 0.38	2.16 ± 0.41	2.17 ± 0.37	2.15 ± 0.32	2.14 ± 0.34	**2.17 ± 0.35**
	HIIE30	2.27 ± 0.40	2.33 ± 0.44	2.19 ± 0.39	2.23 ± 0.38	2.20 ± 0.37	2.30 ± 0.42	2.21 ± 0.45	2.23 ± 0.36	**2.24 ± 0.39**
	HIIE60	2.36 ± 0.30	2.40 ± 0.37	2.46 ± 0.35	2.42 ± 0.37	2.37 ± 0.30	2.40 ± 0.37	2.45 ± 0.42	2.39 ± 0.41	**2.41 ± 0.35#**
f_R ($br{\cdot}min^{-1}$)	**CON**	20.6 ± 3.7	25.4 ± 4.6*	26.8 ± 4.1*	27.9 ± 4.0*	28.7 ± 3.6*	30.5 ± 4.1	29.7 ± 4.5	29.2 ± 4.5	**27.3 ± 5.0**
	HIIE10	26.4 ± 4.7	30.0 ± 5.2*	31.8 ± 3.6*	32.1 ± 3.6*	33.1 ± 4.3*	33.5 ± 4.0	34.5 ± 3.7	35.5 ± 5.2	**32.1 ± 5.0†**
	HIIE30	25.4 ± 4.3	28.6 ± 5.1*	31.6 ± 4.9*	31.0 ± 5.0*	32.0 ± 5.7*	32.4 ± 5.0	34.6 ± 5.7	35.3 ± 6.2	**31.4 ± 5.9†**
	HIIE60	27.7 ± 5.2	32.5 ± 5.9*	35.8 ± 6.5*	39.4 ± 5.9*	43.9 ± 7.5*	43.6 ± 7.5	44.5 ± 6.6	47.7 ± 10.2	**39.4 ± 9.4#**

*p < 0.01 compared to all previous intervals; #p < 0.001 compared to all other trials, †p < 0.05 compared to CON