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BRIEF COMMUNICATION

High-intensity interval exercise induces 24-h energy expenditure similar to traditional endurance exercise despite reduced time commitment

Lauren E. Skelly, Patricia C. Andrews, Jenna B. Gillen, Brian J. Martin, Michael E. Percival, and Martin J. Gibala

Abstract: Subjects performed high-intensity interval training (HIIT) and continuous moderate-intensity training (END) to evaluate 24-h oxygen consumption. Oxygen consumption during HIIT was lower versus END; however, total oxygen consumption over 24 h was similar. These data demonstrate that HIIT and END induce similar 24-h energy expenditure, which may explain the comparable changes in body composition reported despite lower total training volume and time commitment.

Key words: high intensity exercise, energy expenditure.

Résumé : Les sujets effectuent un entraînement par intervalle de haute intensité (« HIIT ») et en mode continu d'intensité modérée (« END ») et on évalue la consommation d'oxygène sur une période de 24 h. La consommation d'oxygène est plus faible en HIIT comparativement à END, mais la consommation d'oxygène total est similaire sur une période de 24 h. Ces résultats démontrent que HIIT et END suscitent la même dépense énergétique en 24 h, ce qui semble expliquer pourquoi les variations de composition corporelle sont semblables malgré le plus faible volume d'entraînement et le moins de temps consacré. [Traduit par la Rédaction]

Mots-clés : exercice de haute intensité, dépense énergétique.

Introduction

Short-term high-intensity interval training (HIIT) induces physiological adaptations that are similar to continuous moderate-intensity endurance training (END) despite a reduced total exercise volume and time commitment (Gibala et al. 2012). A common model employed in low-volume HIIT studies is repeated Wingate tests, which consists of 4–6 × 30-s "all-out" cycling efforts interspersed with 4 min of recovery. Although this type of training is effective, the effort required and need for specialized equipment has been criticized as impractical. Recent studies have considered the potential for other HIIT models — which involve relatively intense but submaximal efforts and thus might have wider application — to elicit adaptations in a time-efficient manner (Little et al. 2011, Gillen et al. 2013). For example, a model consisting of 10 × 60-s efforts at an intensity eliciting ~90% maximal heart rate (HR_{max}), interspersed with 60 s of recovery, has been shown to induce similar adaptations to Wingate-based and END protocols (Little et al. 2011; Gillen et al. 2013).

Limited work has considered the potential role for HIIT in promoting favourable body composition changes, but there are suggestions it may be a time-efficient alternative to END in this regard (Boutcher 2011). Trapp et al. (2008) found that 15 weeks of all-out HIIT was superior to END for reducing whole-body and abdominal fat mass. Other studies employing all-out protocols have reported reductions in waist circumference and fat mass following 2–6 weeks of HIIT that were comparable to END (Whyte et al. 2010; Macpherson et al. 2011). Consistent with results from Wingate-based HIIT studies, the 10 × 60-s model of constant-load HIIT has also been shown to reduce abdominal and whole-body fat mass (Gillen et al. 2013).

Energy expenditure (EE) associated with a single session of low-volume HIIT is typically lower than a bout of END, in large part because of differences in total time spent exercising (Hazell et al. 2012). However, it has been suggested that HIIT induces a larger excess postexercise oxygen consumption (EPOC) than END because of the intense nature of the protocol (Boutcher 2011). Recently, Hazell et al. (2012) reported no difference in total oxygen consumption ($\dot{V}O_2$) between both groups over 24 h, suggesting that HIIT induced a larger EPOC than END. Conversely, Williams et al. (2013) measured EPOC for 3 h following exercise and found no differences between all-out HIIT and END.

To date no study has examined whether a more practical model of constant-load HIIT acutely alters postexercise metabolism and (or) 24-h EE. Accordingly, our purpose was to compare 24-h EE following an acute bout of constant-load HIIT and END. We hypothesized that 24-h EE would be similar between treatments, and greater compared with a control period, despite the difference in total exercise volume and time commitment.

Materials and methods

Subjects

Nine men (age = 21 ± 1 years, peak oxygen consumption ($\dot{V}O_{2peak}$) = 46 ± 7 mL/(kg·min), body mass = 91 ± 15 kg, body mass index = 26 ± 1 kg/m²) took part in the experiment. The protocol was approved by the Hamilton Integrated Research Ethics Board.

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L.E. Skelly, P.C. Andrews, J.B. Gillen, B.J. Martin, M.E. Percival, and M.J. Gibala.* Department of Kinesiology, Ivor Wynne Centre, McMaster University, Hamilton, ON L8S 4K1, Canada.

Corresponding author: Martin J. Gibala (e-mail: gibalam@mcmaster.ca).

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Pre-experimental procedures

A canopy system attached to an on-line gas collection unit (Moxus modular oxygen uptake system, AEI Technologies, Pittsburgh, Pa., USA) was used to measure $\dot{V}O_2$ during resting conditions. To determine an appropriate canopy flow rate to be used during the experimental trials, subjects completed a 30-min resting EE session prior to testing. Subjects lay supine on a bed in a dark room and a tight seal was created using a plastic skirt on the canopy. During a separate visit a maximal exercise test was completed as described previously (Gillen et al. 2013).

Experimental trials

Subjects performed 3 trials in random order in a repeated-measures design. The HIIT treatment involved 10 × 60-s intervals at a workload that elicited ~90% HR_{max} with 60 s of active recovery at 50 W. The END protocol consisted of cycling at a workload that elicited ~70% of HR_{max} for 50 min. This protocol was selected because previous research has reported similar changes in body composition between HIIT and longer (40–60 min) bouts of END (Macpherson et al. 2011). Also, given that HIIT studies typically involve 3 training sessions per week, and given current physical activity guidelines are calling for 150 min of moderate-intensity exercise per week, a comparable END training program would consist of 3 × 50-min sessions per week. A 3-min warm-up at 50 W was performed prior to the main exercise bout in HIIT and END. No exercise was performed in the control trial (CON). All trials commenced following a 10-h overnight fast and subjects were advised to perform no physical activity other than the prescribed exercise and normal activities of daily living for 24 h prior to each trial and over the course of the 24-h collection period. All meals were standardized for a given subject across all trials. Heart rate (HR) and activity was measured continuously for 24 h using a chest-worn device (Actiheart; Camntech, Cambridge, UK) and $\dot{V}O_2$ was collected for 30–60 min at 7 different time points. The general design of the study and timing of $\dot{V}O_2$ measurements (Fig. 1) was modeled after previous work by Hazell et al. (2012).

Measurements

$\dot{V}O_2$

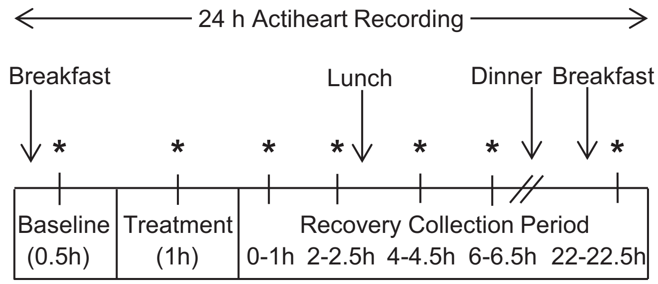
For all measurements except those performed during exercise, $\dot{V}O_2$ data was collected using the same methods described previously in the pre-experimental session. The flow rate was adjusted to match the previously determined rate. The first 10 min of data was excluded to avoid using data influenced by the initial application of the equipment. During the exercise periods in the HIIT and END trials, gas measurements were performed by the separate on-line gas collection unit (Moxus) used for the determination of $\dot{V}O_{2peak}$.

To determine the total $\dot{V}O_2$ during exercise and over 24 h, rate of $\dot{V}O_2$ (L/min) was plotted and combined area under the curve was calculated using GraphPad Prism 5 (GraphPad Software Inc., La Jolla, Calif., USA). To permit time-matched comparisons between treatments, baseline resting data was extrapolated to make the total "intervention" time standardized to 60 min in duration. For the CON trial, no specific $\dot{V}O_2$ measurements during treatment were performed, but this was determined based on the baseline resting data. Total EE was derived from $\dot{V}O_2$ measurements as described by Williams et al. (2013).

HR and activity

The Actiheart was placed on the subject's chest via electrodes (3M Health Care, St. Paul, Minn., USA) and determined activity EE based on HR and activity counts using an equation that has been previously validated (Crouter et al. 2008). Total 24-h EE was determined as described previously (Camntech 2010).

Fig. 1. Experimental protocol. *, Oxygen consumption measurement.



Statistical analyses

Data were analyzed using Statistical Package for Social Sciences (version 20.0; SPSS Inc., Chicago, Ill., USA). A 1-way repeated measures ANOVA was used to determine differences between treatments for total $\dot{V}O_2$, total EE, mean HR, and activity counts. For the 24-h period a 2-way repeated measures ANOVA (time × treatment) was used to determine differences in total $\dot{V}O_2$, respiratory exchange ratio (RER) and mean HR. Post hoc tests were performed using Tukey's HSD test.

Results

Exercise workload

Workload during the intervals in the HIIT session was 277 ± 45 W, which corresponded to 77% ± 3% of peak power output achieved at the end of the $\dot{V}O_{2peak}$ test. The corresponding values during the END session was 129 ± 20 W, which corresponded to 36% ± 3% of peak power output. Total exercise volume was 205 ± 27 and 410 ± 64 kJ in the HIIT and END trials, respectively.

$\dot{V}O_2$

Mean resting $\dot{V}O_2$ at baseline was 0.42 ± 0.06, 0.44 ± 0.07, and 0.42 ± 0.05 L/min for the HIIT, END, and CON trials, respectively. Total $\dot{V}O_2$ during exercise (including warm-up) was 53 ± 5.5 L (mean = 2.5 ± 0.3 L/min) during HIIT and 106 ± 13 (mean = 2.0 ± 0.2 L/min) during END. Total $\dot{V}O_2$ calculated over each 1-h intervention period was higher in END versus HIIT, and both were greater compared with CON (Fig. 2A). Total $\dot{V}O_2$ estimated over 24 h was similar between HIIT and END and both were greater compared with CON (Fig. 2B). There were no significant ($p < 0.05$) differences in RER at any measurement point over 24 h.

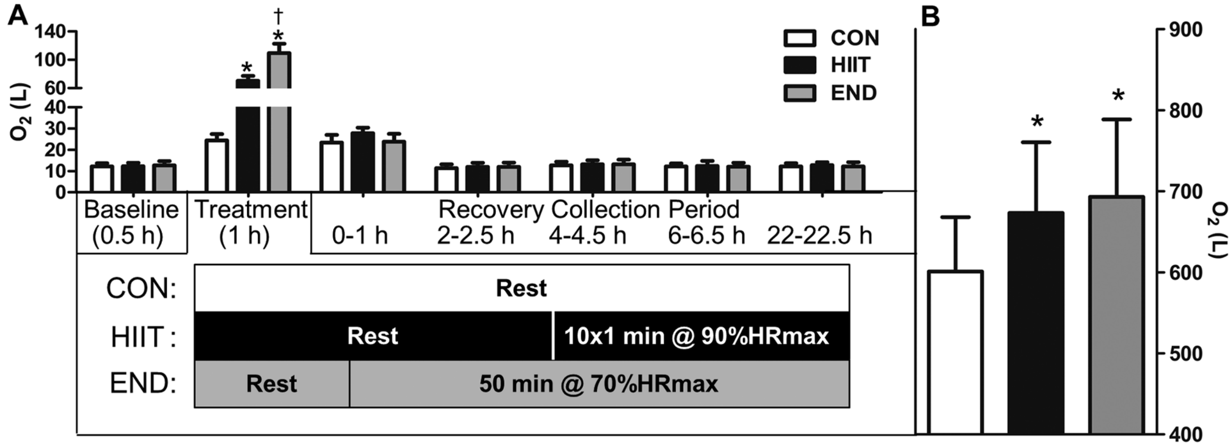
HR

Mean HR during the HIIT intervals corresponded to 90% ± 2% of HR_{max} (170 ± 9 bpm) and mean HR over the entire 22-min session was 80% ± 3% of HR_{max} (151 ± 9 bpm). Mean HR during END was 69% ± 1% of HR_{max} (131 ± 6 bpm). Mean HR during the 1-h intervention was higher in END than HIIT (HIIT: 93 ± 6, END: 120 ± 7 bpm, $p < 0.05$). In the first hour of recovery, mean HR was higher in HIIT versus END and CON (HIIT: 88 ± 8, END: 69 ± 7, CON: 56 ± 6 bpm, $p < 0.05$). In the second hour of recovery, mean HR following HIIT (76 ± 9 bpm) was higher ($p < 0.05$) compared with CON (60 ± 10) but not different versus END (68 ± 11).

EE

During the 1-h intervention period, $\dot{V}O_2$-derived estimates of EE were higher for END vs. HIIT, and both were greater than CON (HIIT: 352 ± 34, END: 547 ± 65, CON: 125 ± 15 kcal, $p < 0.01$). Total 24-h EE was similar between END and HIIT, and both were greater than CON (HIIT: 3368 ± 443, END: 3464 ± 469, CON: 3005 ± 335 kcal, $p < 0.01$). Accelerometry-derived estimates of EE over the 1-h intervention period were higher for END vs. HIIT, and both were greater than CON (HIIT: 380 ± 72, END: 564 ± 133, CON: 124 ± 30 kcal, $p < 0.01$). Total 24-h EE was similar between END and HIIT, and HIIT was greater than CON (HIIT: 3766 ± 523, END: 3526 ± 504, CON: 3107 ± 602 kcal, $p < 0.01$).

Fig. 2. Total oxygen consumption at each measurement point (A) and over 24 h (B). CON, control; HIIT, high-intensity interval training; END, continuous moderate-intensity training; HR_{max}, maximal heart rate. *, $p < 0.05$ vs. CON; †, $p < 0.05$ vs. HIIT.



Activity counts

Total activity counts in the postexercise period were not different between treatments (HIIT: 10763 ± 3240, END: 10077 ± 4400, CON: 10372 ± 1828, $p > 0.05$).

Discussion

The main finding from the present study was that constant-load HIIT elicited a measurable increase in 24-h EE, to an extent similar to that induced by moderate-intensity continuous exercise that lasted more than twice as long and involved twice as much mechanical work. Our results are similar to that of Hazell et al. (2012), who showed that total $\dot{V}O_2$ over 24 h was similar after a bout of Wingate-based HIIT and a 30-min END bout. The present study, however, employed a constant-load protocol that might be more feasible than more demanding all-out protocols, while still being time-efficient. In contrast to the present results and those of Hazell et al. (2012), Williams et al. (2013) found no difference in 3-h EPOC following HIIT and END, reporting that both had returned to baseline levels by 45 min postexercise. We also did not detect a measurable difference in $\dot{V}O_2$ in the hours immediately postexercise, and thus the divergent findings between studies may primarily be due to differences in the duration of data collection following exercise. Given the relatively small differences from baseline, a longer postexercise collection period may be necessary to capture small changes in EE induced by HIIT. It is also possible that the relatively small sample size may have limited our ability to detect potential differences that may have existed in 24-h $\dot{V}O_2$ between HIIT and END. Finally, it is possible that a small increase in $\dot{V}O_2$ during recovery, potentially in combination with other factors such as appetite suppression following HIIT, could contribute to reductions in body fat or alterations in body composition observed after HIIT and END training (Boutcher 2011).

The present study also included continuous HR recordings over 24 h. The rate of HR recovery is determined by the interaction of parasympathetic re-activation and sympathetic withdrawal (Pierpont et al. 2000) and maximal exercise stimulates the sympathetic nervous system to a greater extent than submaximal exercise (Borresen and Lambert 2008). In the recovery phase following high-intensity exercise, prolonged sympathetic activation can occur and override the parasympathetic response causing a delay in the rate of recovery (Borresen and Lambert 2008). In the present study, HIIT induced a greater mean HR than both END and CON for the first 1 h postexercise and the difference between HIIT and CON persisted for an additional 1 h. These data are similar to the findings from a study that examined HR following Wingate-based HIIT and reported that HR was elevated over END and CON for the first hour postexercise and that HIIT remained above CON for 15 min longer (Williams et al. 2013). Interestingly, in that study no change in oxygen consumption was seen between HIIT and END, suggesting that the influence of HIIT on HR recovery may largely be determined by the level of sympathetic activation during exercise, rather than oxygen demand during recovery. Elevated HR following HIIT may also occur to compensate for alterations in cardiac function immediately following exercise. Scott et al. (2010) observed lower biventricular stroke volumes and elevated HR from baseline values in normally active individuals for ~38 min following HIIT. Further investigations of the influence of HIIT on the nervous system and cardiac function during and after exercise will aid in better understanding the mechanisms facilitating an extended HR recovery following acute HIIT.

In summary, using both $\dot{V}O_2$ and accelerometry-derived measures of EE, the present study found that while EE during HIIT was lower versus END, 24-h EE was similar between treatments. Our data confirm that similar to Wingate-based HIIT, an acute bout of constant-load HIIT is a time-efficient method to elicit an increase in 24-h EE, similar to a bout of END that involved a longer time commitment and greater amount of mechanical work. HIIT may represent a practical exercise option to improve body composition in healthy and clinical populations in a time-efficient manner.

Conflict of interest statement

The authors have no conflicts of interest to declare.

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