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Research article

A practical model of low-volume high-intensity interval training induces performance and metabolic adaptations that resemble 'all-out' sprint interval training

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Abstract
Recently, a novel type of high-intensity interval training known as sprint interval training has demonstrated increases in aerobic and anaerobic performance with very low time commitment. However, this type of training program is unpractical for general populations. The present study compared the impact of a low-volume high-intensity interval training to a "all-out" sprint interval training. Twenty-four active young males were recruited and randomized into three groups: (G_1: 3-5 cycling bouts × 30-s all-out with 4 min recovery; G_2: 6-10 cycling bouts × 125% P_{max} with 2 min recovery) and a non-trained control group. They all performed a VO_{2max} test, a time to exhaustion at P_{max} (T_{max}) and a Wingate test before and after the intervention. Capillary blood lactate was taken at rest, 3, and 20 min after the Wingate trial. Training was performed 3 sessions per week for 4 weeks. In G_1, significant improvements ($p < 0.05$) following training were found in VO_{2max} (9.6%), power at VO_{2max} (12.8%), T_{max} (48.4%), peak power output (10.3%) and mean power output (17.1%). In G_2, significant improvements following training were found in VO_{2max} (9.7%), power at VO_{2max} (16.1%), T_{max} (54.2%), peak power output (7.4%; $p < 0.05$), but mean power output did not change significantly. Blood lactate recovery (20^{th} min) significantly decreased in G_1 and G_2 when compared with pre-testing and the CON group ($p < 0.05$). In conclusion, the results of the current study agree with earlier work demonstrating the effectiveness of 30-s all-out training program to aerobic and anaerobic adaptations. Of substantial interest is that the low volume high intensity training provides similar results but involves only half the intensity with double the repetitions.

Key words: Wingate test, repeated sprints, blood lactate, training adaptations.

Introduction

It is well established that factors such as poor cardiorespiratory fitness, adiposity, impaired glucose tolerance, hypertension, and arteriosclerosis are independent threats to health and that physical inactivity increases the risk for premature death and elevates the incidence of the above-mentioned unhealthy conditions. Epidemiologic cross-sectional investigations and longitudinal intervention studies have provided experimental evidence for the effectiveness of prolonged aerobic exercise training such as continuous running, brisk walking, or bicycling as interventions that may lower the relative risk for developing several metabolic diseases (Nybo et al., 2010). However, lack of time is a common reason why many people fail to accomplish the training programs (Godin et al., 1994). Recently, a novel type of high-intensity interval training (HIT) known as sprint interval training (SIT; repeated 30-s ''all-out'' efforts) has demonstrated increases in aerobic performance in a short time frame (Burgomaster et al., 2005; Gibala et al., 2006). It is interesting that SIT may induce similar improvements in cardiorespiratory fitness and skeletal muscle oxidative capacity as prolonged training, (Burgomaster et al., 2008; Nybo et al., 2010) and a recent study reported that SIT substantially improves insulin action (Babraj et al., 2009). Apparently, the repeated SIT bouts stress many of the physiological/biochemical systems used in aerobic efforts (Hazell et al., 2010) and poses a considerable metabolic challenge to skeletal muscle as large reductions in muscle glycogen and pH and increases in blood lactate as well as whole body carbohydrate and fat oxidation (Stepto et al., 2001). A wide range of adaptations have been shown after this type of training, including increased resting glycogen content (Barnett et al., 2004; Rodas et al., 2000), increased activity of various glycolytic and oxidative enzymes (Burgomaster et al., 2005; Jacobs et al., 1987), H^+ buffering capacity (Gibala et al., 2006), and time to exhaustion (Burgomaster et al., 2005; Gibala et al., 2006). Increased (Rodas et al., 2000; Tabata et al., 1996) or unchanged (Burgomaster et al., 2005) values of VO_{2max} after SIT have also been reported. As a result, it has been speculated that SIT could be a time-efficient strategy for health promotion. Nonetheless, use of 30-s ''all-out'' efforts as a training program is often dismissed outright as unsafe, unpractical, or intolerable for general populations. Therefore, an evaluation of the physiological adaptations induced by different interval-training strategies in a wide range of populations will permit evidence-based recommendations that may provide an alternative to current exercise prescriptions for time-pressed individuals. Accordingly, the purpose of this study was to compare the established SIT protocol (30-s ''all-out'' efforts with 4 min recovery) versus a modified type of HIT (30-s efforts with 125% P_{max} with 2 min recovery) on both aerobic and anaerobic performance. The intensity of the modified training is the half of the intensity of SIT but with doubled repetitions. We hypothesized that the training induced changes are the same.

Methods

Participants

Twenty-four healthy young male graduate students volunteered to participate in the investigation (age = 25 ± 0.8 years; height = 1.72 ± 0.08 m; mass = 70 ± 11 kg; Percent

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body fat = 18 ± 6%). All were habitually active but not engaged in any sort of structured training program nor had they been for at least 3 months prior to the study. Prior to any participation, the experimental procedures and potential risks were explained fully to the participants and all provided written informed consent. Dietary and physical activity patterns were maintained throughout the study. Subjects were matched into three groups based on the time to exhaustion test and VO_{2max}. The study was approved by the Ethical Committee of the Faculty of Medical Sciences of Tarbiat Modares University and was in accordance with the Declaration of Helsinki.

Baseline testing

Prior to any baseline testing, all participants attended a laboratory familiarization visit to introduce the testing/training procedures and to ensure that any learning effect was minimal for the baseline measures. Each participant first completed tests for body composition and graded exercise test (GXT) to determine their VO_{2max} and power at VO_{2max} (P_{max}). The second test was a T_{max} test used to determine the time to exhaustion at P_{max}. On the third day, the participants performed 30-second Wingate test to determine peak power output (PPO), mean power output (MPO) and total work (W_{tot}). All tests were separated by at least 24 h, post-testing occurred within 48 h of the final training session in the same order (Hazell et al., 2010).

Graded exercise test

VO_{2max} was determined using a progressive incremental test on a cycle ergometer until volitional fatigue. After 5 min warm-up and performing stretching exercises, the test commenced at an initial power output of 50 W and was increased by 30 W every minute until the participant was fatigued (Laursen et al., 2002a). Expired gas analysis was acquired with an automated metabolic system (ZAN600 CPET: ZAN Messgerate GmbH, Oberthulba, Germany), and VO_{2max} was calculated as the highest oxygen consumed over a 1 min period. VO_{2max} was confirmed when three or more of the following criteria were met: (1) a plateau in VO_2 despite an increase in work load; (2) a respiratory exchange ratio (RER) higher than 1.2 (Esfarjani and Laursen, 2007); (3) peak heart rate at least equal to 90% of the age-predicted maximum (Tanaka et al., 2001); and/or (4) visible exhaustion. P_{max} was defined as the final completed work rate (e.g., maintained for 10 s) (Laursen et al., 2002a).

Time to exhaustion at P_{max}

The second test performed was a T_{max} test used to determine the time to exhaustion at P_{max} that was defined as the minimal power output that elicited a VO_2 reading that was within 2 $ml \cdot kg^{-1} \cdot min^{-1}$ of the previous reading, despite an increase in workload (Laursen et al., 2005). After a 10-minute warm-up at 50 W, participants cycled to fatigue at P_{max} at a self-selected cadence; the test was stopped when cadence fell below 60 $rev \cdot min^{-1}$. The total amount of work completed during the T_{max} test (W_{Tmax}) was calculated as a product of P_{max} and T_{max} ($W_{Tmax} = (P_{max} \times T_{max})/1000$) and expressed in kJ.

Wingate test

Peak power output (PPO), mean power output (MPO) and total work (W_{tot}) were assessed over Wingate test on a mechanically braked cycle ergometer (model 894E, Monark, Sweden). Participants performed a Wingate test against a resistance equivalent to 0.075 kg/kg body mass. Participants were instructed to begin pedaling as fast as possible against the ergometer's inertial resistance, and then the appropriate load was manually applied. Participants were verbally encouraged to continue pedaling as fast as possible throughout the 30-s test. PPO was defined as the highest work output in a 5-second period, and MPO as the average work output for the 30-second test period (Nottle and Nosaka, 2007). W_{tot} was used to describe muscle endurance and was calculated by multiplying MP by 30 seconds and expressed in kJ. Previously determined intraclass correlation coefficient (ICC) for Wingate variables was 0.94 (Bar-Or, 1987).

Training intervention

Participants were assigned to one of the three groups: (G_1) 30-s all-out efforts: 4 min recovery; (G_2) 30-s with 125% P_{max}: 2 min recovery and control group (no training) (Table 1). Participants trained three times per week on alternate days for a total of 4 wk. The G_1 cycling program began with 3 intervals in week 1, progressing to 5 in week 3 followed by a reduction to 4 intervals in week four. The G_2 cycling program began with 6 intervals in week 1, progressing to 10 in week 3 followed by a reduction to 8 intervals in week four.

Blood lactate

Capillary blood samples were taken (by finger prick) from the forefinger at rest, 3, and 20 min after the Wingate trial. Blood lactate was analyzed on site using a Lactate Analyzer (Lactate Scout, Senslab GmbH Leipzig, Germany).

Data analysis

All results are reported as a mean ± SD. The Kolmogorov-Smirnov test was used to test the normality of the distribution. A 3 × 2 (Group × time) repeated measures analysis of variance (ANOVA) was performed. When a significant difference was revealed, Tukey's post hoc test was used to specify where the difference occurred. The alpha level for statistical significance was set at $p \leq 0.05$. Effect size was calculated using Cohen's *d* (d).

Results

Changes in physiological variables associated with the GXT are presented in Table 2. Following the 4-week

Table 1. Four-week training protocol for the two sprint interval training groups (G_1, G_2).

Group	Sessions/week	Bouts/session	Intensity	Work duration	Rest duration
G_1 (n = 8)	3	3-5	All out	30s	4min
G_2 (n = 8)	3	6-10	125% P_{max}	30s	2min

Table 2. Pre-test vs. Post-test values for maximal oxygen consumption (VO_{2max}), power at VO_{2max} (P_{max}) and time to exhaustion at P_{max} (T_{max}). Data are means (±SD).

		Pre-test	Post-test	P-value	95%CI
VO_{2max} ($ml \cdot kg^{-1} \cdot min^{-1}$)	G_1	44.6 (4.3)	48.9 (3.5)	.046	.08-8.5
	G_2	44.3 (3.9)	48.6 (3.3)	.048	.04-8.5
	CON	45.1 (3.9)	44.7 (4.2)	.876	3.9-4.5
P_{max} (W)	G_1	242.4 (26.2)	273.5 (22.8) †	.027	3.6-58.6
	G_2	238.5 (27.2)	277.1 (16.3) †	.007	11.1-66.0
	CON	241.8 (24.8)	237.1 (31.8)	.730	22.7-32.1
T_{max} (s)	G_1	131.5 (26.7)	195.2 (14.0) †	.000	36.4-90.9
	G_2	130.8 (26.8)	201.7 (13.4) †	.000	43.6-98.0
	CON	134.5 (35.5)	131.0 (26.7)	.792	23.6-30.8
W_{Tmax} (kJ)	G_1	32.4 (9.7)	53.6 (7.3) †	.000	12.6-29.7
	G_2	31.7 (9.9)	56.0 (6.2) †	.000	15.6-32.8
	CON	32.0 (7.2)	30.8 (6.1)	.771	7.3-9.8

† Significantly different compared with CON group ($p < 0.05$).

training program, VO_{2max} (G_1: d = 1.10; G_2: d = 1.19), P_{max} (G_1: d = 1.27; G_2: d = 1.77), T_{max} (G_1: d = 3.13; G_2: d= 3.53) and W_{Tmax} (G_1: d = 2.49; G_2: d = 3.02) were significantly increased in the G_1 and G_2 groups compared with pre-testing. As well, the increases in P_{max} (G_1: p = 0.011, d = 1.31; G_2: p = 0.006, d = 1.58), T_{max} (G_1: p = 0.00, d = 3.01; G_2: p = 0.00, d = 3.34) and W_{Tmax} (G_1: p = 0.00, d = 3.38; G_2: p = 0.00, d = 4.09) were significantly greater in G_1 and G_2 compared with CON group. No variables were significantly changed in the CON group.

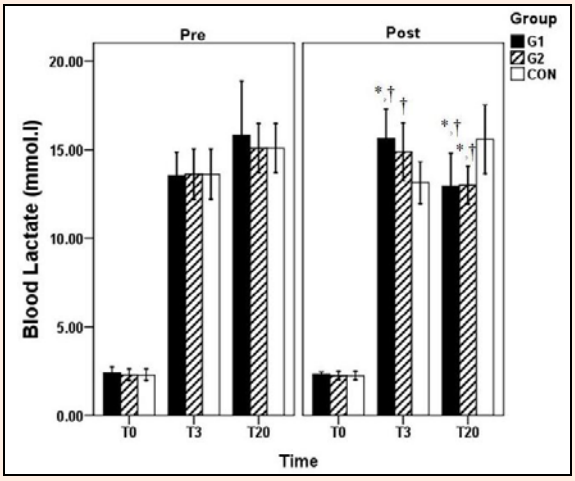

Figure 1. Blood lactate concentrations at rest, 3 and 20 min after the Wingate trial. Values are means ± SD. * Significantly different from pre-testing values ($p < 0.05$). † Significantly different compared with control group ($p < 0.05$).

PPO increased significantly in G_1 (p = 0.006, d = 1.48; Table 3) and G_2 (p = 0.04, d = 1.86) after the training. As well, the increase in PPO was significantly greater in G_1 compared with CON group (p = 0.009, d = 1.66). In addition, MPO increased significantly in G_1 compared with pre-testing (p = 0.025, d = 1.32; Table 3) and the CON group (p = 0.017, d = 1.49). MPO in G_2 did not change significantly with the training (p = 0.064). There were significant increases in W_{tot} in G_1 (p = 0.025, d = 1.38; Table 3), which were statistically significant compared with CON group (p = 0.017, d = 1.52).

After the 4-week training period, maximal BLa^- (3^{th} min) was significantly higher in G_1 compared with pre-testing (Post: 15.6 ± 1.6 *vs.* Pre: 13.5 ± 1.3 $mmol \cdot l^{-1}$; p = 0.01, d = 1.45; Figure 1) and CON group (G_1: 15.6 ± 1.6 *vs.* CON: 13.1 ± 1.2 $mmol \cdot l^{-1}$; p = 0.003, d = 1.76). Maximal BLa^- in G_2 was only significantly different compared with CON group (G_2: 14.8 ± 1.6 *vs.* CON: 13.1 ± 1.2 $mmol \cdot l^{-1}$; p = 0.032, d = 1.2). Blood lactate recovery (20^{th} min) significantly decreased in G_1 (Post: 12.9 ± 1.8 *vs.* Pre: 15.8 ± 3.0 $mmol \cdot l^{-1}$; p = 0.007, d = 1.21) and G_2 (Post: 13.0 ± 1.0 *vs.* Pre: 15.1 ± 1.4 $mmol \cdot l^{-1}$; p = 0.04, d = 1.75) when compared with pre-testing and the CON group ($p < 0.05$).

Discussion

Recent evidence suggests that sprint interval training is a time-efficient strategy to stimulate a number of skeletal muscle adaptations that are comparable to traditional endurance training (Gibala and McGee, 2008). It has been demonstrated that SIT up-regulates peroxisome proliferator-activated receptor-co-activator-1α (PGC-1α), a potent regulator of mitochondrial biogenesis (Gibala et al., 2009), which could be the underlying mechanism responsible for the observed aerobic adaptation. This

Table 3. Pre-test vs. Post-test values for peak power output, mean power output and total work. Data are means (±SD).

		Pre-test	Post-test	P-value	95%CI
Peak power (W)	G_1	776 (72)	856 (36) †	.006	24 - 136
	G_2	775 (41)	833 (20)	.045	1 - 133
	CON	783 (67)	780 (54)	.902	53 - 59
Mean power (W)	G_1	378 (61)	443 (37) †	.025	8 - 121
	G_2	375 (55)	417 (44)	.141	15 - 98
	CON	372 (54)	373 (54)	.955	55 - 58
Total work (kJ)	G_1	11.3 (1.8)	13.2 (1.1) †	.002	.2 - 3.6
	G_2	11.2 (1.6)	12.5 (1.3)	.100	.4 – 2.9
	CON	11.2 (1.6)	11.2 (1.6)	.900	1.6 – 1.7

† Significantly different compared with CON group ($p < 0.05$).

information is exciting because it means that such adaptations can be obtained with a substantial reduction in exercise training time. However, the feasibility of this type of training is questionable for general populations. Therefore, the purpose of the current study was to compare the established SIT protocol versus a modified type of HIT on both aerobic and anaerobic performance.

The first finding of the present study was that both training programs significantly and similarly improved VO_{2max} in untrained subjects (G_1: 9.6% *vs.* G_2: 9.7%; Table 2). Several studies have shown an increase in VO_{2max} following established SIT program (Creer et al., 2004; Barnett et al., 2004). In contrast to these findings, Burgomaster et al. (2005) recently showed in untrained subjects that six sessions of sprint interval training (30-s all-out Wingate sprints) increased citrate synthase activity by 38% and doubled cycle time to exhaustion. Interestingly, no change occurred in VO_{2max}. Improvements in VO_{2max} may occur through increases in both oxygen delivery (i.e., increases in stroke volume) and/or oxygen utilization by active muscles (i.e., increases in capillarization/mitochondrial density). Given that maximal heart rate remains unchanged in response to training, improvements in oxygen delivery to exercising muscles during high-intensity exercise can be attributed to an increase in stroke volume. Stroke volume can increase through a higher left-ventricular contractile force and/or through an increase in cardiac filling pressure, which raises end-diastolic volume and resultant stroke volume (Laursen and Jenkins, 2002b). In addition, the increases in VO_{2max} and oxidative enzymes that has been reported by several authors (Barnett et al., 2004; Gibala et al., 2006) indicate the present sprint-training protocol evoked changes in the capacity to produce energy via oxidative metabolism. It is also known that the aerobic contribution to sprint exercise increases, depending on the recovery between bouts, as a function of successive sprints (Bogdanis et al., 1996). The relatively short recovery periods used in the present study would have imposed a considerable demand on aerobic metabolism in meeting ATP resynthesis in the latter sprint bouts. All of these factors may explain the observed increase in VO_{2max}.

T_{max} significantly increased in both groups following training (G_1: 48% *vs.* G_2: 54%; Table 2). T_{max} improvements are greater than those reported by Esfarjani and Laursen (2007) who reported a 32% improvement in moderately trained runners using HIT prescribed at 130% vVO_{2max} with interval durations of 30-s. In addition, 30-s all-out SIT program (over 2 wk) has been shown to improve (100%) cycle time to exhaustion at 80% VO_{2max} in untrained subjects (Burgomaster et al., 2005). Franch et al. (1998) also reported a significant increase (65%) in time to exhaustion after 6 weeks of short-interval training. From a metabolic point view, the increased mitochondrial enzyme content, which is associated with an increase in the rate of lipid utilization and consequently a decrease in rate of glycogen depletion contributes to an improved exercise tolerance, the glycogen depletion being strongly linked with fatigue during prolonged exercise (Demarle et al., 2003). Interestingly, a comprehensive work by Harmer et al. (2000) indicated a reduced anaerobic ATP synthesis during intense exercise following seven weeks of sprint training, and also suggested an enhanced aerobic metabolism. Another possible reason for the improved T_{max} found in the training groups may be a decreased lactate accumulation throughout the test and/or a greater muscle buffering capacity, which has been demonstrated in repeated 30-s sprint training (Gibala et al., 2006). It is possible that the greater improvement in T_{max} in G_2 versus G_1 was aided through the higher training volume in G_2 (6-10 30-s bouts) compared with G_1 (3-5 30-s bouts).

PPO elicited during the Wingate test increased by 10.3% and 7.3% in G_1 and G_2, respectively, with no difference between groups (Table 3); whereas, MPO was increased only in G_1 by 17.1% ($p < 0.05$). This result is consistent with a greater reliance on glycolysis during training in G_1 and could explain at least partially why the less intense 30-s repeats seemed less difficult. Such results are in accordance with previous studies reporting an improvement in PPO with SIT. Burgomaster et al. (2005) reported increase in PPO after 2 weeks of SIT but MPO did not change in their investigation. Increased muscle phosphocreatine concentration (Rodas et al., 2000), anaerobic enzyme activities (MacDougall et al., 1998; Parra et al., 2000), and a significant increase in FTa fibers, along with a decrease in ST fibers (Jansson et al., 1990; Dawson et al., 1998), are possible explanations of our findings.

As expected with SIT, there was an increase in maximal blood lactate from pre to post training in G_1 (15.5%; Figure 1). An increase in maximal BLa^- in post-testing coinciding with increased peak power, mean power, and total work in G_1. Sharp et al. (1986) reported an increase in blood lactate concentrations and total work performed during a 45-s maximal cycle sprint after eight weeks of intense sprint training in untrained subjects. These data were reported in conjunction with an increase in the glycolytic enzyme phosphofructokinase (PFK), suggesting that increased lactate and total work values were due to improved glycolytic output. Increases in glycolytic enzymes such as hexokinase and PFK due to SIT have been shown as well (MacDougall et al., 1998; Rodas et al., 2000), indicating that increases in blood lactate and total work values in G_1 may be due to improved glycolytic function. Interestingly, Sharp and colleague (1986) saw no change in muscle pH in spite of increased lactate concentrations suggesting an increase in buffering capacity, which may have also contributed to increases in total work in the current study in spite of increased blood lactate concentrations. However, the changes in G_2 did not reach to significant level ($p > 0.05$). In addition, blood lactate recovery changed significantly and similarly with either training regimen (Figure 1).

As a result, 30-s all-out SIT should be easily incorporated into the training program of any athlete desiring to increase both aerobic and anaerobic power quickly but not into the training program of general population. The Wingate-based training model requires a specialized ergometer and an extremely high level of subject motivation. Given the extreme nature of the exercise, it is doubtful that the general population could safely or practically adopt the model. Like the our study, future studies should

examine modified interval-based approaches to identify the optimal combination of training intensity and volume necessary to induce adaptations in a practical time-efficient manner.

Conclusion

The results of the current study agree with earlier work demonstrating the effectiveness of 30-s all-out SIT to aerobic and anaerobic adaptations. However, of substantial interest is our observation that many of the same performance gains occurred in modified HIT protocol (30-s with 125% P_{max}).

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Key points

- Given the markedly lower training volume in the training groups, our results suggest that intense interval training is indeed a time-efficient strategy to induce rapid metabolic and performance adaptations.
- The results demonstrate that a practical low-volume HIT program is effective for improving metabolic and performance adaptations that resemble many of the same performance gains occurred in all-out SIT protocol.

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