Active Recovery Induces Greater Endurance Adaptations When Performing Sprint Interval Training

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Abstract

Yamagishi, T and Babraj, J. Active recovery induces greater endurance adaptations when performing sprint interval training. *J Strength Cond Res* 33(4): 922–930, 2019–This study sought to determine effects of recovery intensity on endurance adaptations during sprint interval training (SIT). Fourteen healthy young adults (male: 9 and female: 5) were allocated to 1 of 2 training groups: active recovery group (ARG, male: 4 and female: 3) or passive recovery group (PRG, male: 5 and female: 2). After having completed a 2-week control period, both groups performed 6 sessions of 4- to 6 30-second sprints interspersed with 4-minute recovery over 2 weeks. However, only ARG cycled at 40% $\dot{V}O_2$peak during the 4-minute recovery periods, while PRG rested on the bike or cycled unloaded. After the 2-week training intervention, both groups improved 10-km time-trial performance to a similar extent (ARG: 8.6%, $d = 1.60$, $p = 0.006$; PRG: 6.7%, $d = 0.96$, $p = 0.048$) without gains in $\dot{V}O_2$peak. However, critical power was increased by ARG only (7.9%, $d = 1.75$, $p = 0.015$) with a tendency of increased maximal incremental power output (5.3%, $d = 0.88$, $p = 0.063$). During the training, active recovery maintained $\dot{V}O_2$ and heart rate at a higher level compared with passive recovery ($\dot{V}O_2$: $p = 0.005$, HR: $p = 0.018$), suggesting greater cardiorespiratory demands with the active recovery. This study demonstrated that greater endurance performance adaptations are induced with active recovery when performing SIT over a short time frame. The findings of the current study indicate that, with active recovery, individuals can gain greater training benefits without increasing total training commitment time. Further studies are required to determine whether differences are seen with recovery intensity over a longer period.

Introduction

It has been established that a 2-week Wingate-based sprint interval training (SIT) program consisting of 4–6 × 30-second maximal efforts with 4-minute recovery can induce various training adaptations such as improvements in mitochondrial function, muscle buffering capacity, and exercise performance (12,13,20). In addition, the magnitude of physiological and performance adaptations seen after 2 weeks of Wingate-based SIT has been shown to be comparable with those obtained by traditional endurance training (e.g., 60–90 minutes of continuous cycling at 65% $\dot{V}O_2$max) over the same time course, despite its low-training volume (i.e., 2–3 minutes of all-out efforts per session) (20). This suggests that this training modality could be a time-efficient strategy to bring about training benefits rapidly.

However, although an improvement in endurance performance has been consistently reported after Wingate-based SIT over 2–6 weeks (3,13,20,27,40), it remains unknown whether an improvement in a single endurance parameter coincides with those in different parameters because most of the previous Wingate-based studies assessed endurance performance through a single performance test (e.g., time trial). There are several studies that have demonstrated a close association between different endurance parameters. For example, critical power (CP) derived from a 3-minute all-out test has been shown to significantly correlate with 16.1-km road time-trial time (8). Likewise, Balmer et al. (5) found a high correlation between maximal incremental power output and mean power output during a 16.1-km road time trial (5). Nevertheless, a high correlation does not necessarily imply causation, and indeed, the main physiological determinants have been shown to be different according to endurance parameters with oxygen transport capacity being the most important for maximal incremental power output, whereas skeletal muscle oxidative capacity being the strongest predictor for time-trial performance (24). Moreover, although both incremental and all-out

exercises can elicit maximal physiological responses (e.g., $\dot{V}O_2$peak), the pattern (time course) of aerobic and anaerobic energy utilization would be different between the exercise modalities (28,37), which may change the importance of muscle O_2 supply or utilization. Taken together, the magnitude of performance changes may be dependent on the form of its assessment and the modality of exercise training.

Despite the reported training benefits, most of the studies have not considered workload during the recovery period, and indeed, passive recovery or very light cycling ($<$30 W) are commonly used in the Wingate-based studies (4,12,13,20). However, in previous studies examining acute physiological and performance responses to different recovery modes during repeated 30-second Wingate tests, active recovery (cycling at 28–40% of $\dot{V}O_2$max) has been shown to facilitate maintenance of power production with an elevated cardiorespiratory demand (e.g., increased heart rate [HR] and $\dot{V}O_2$) compared with passive recovery (9,34). Although it has yet to be determined whether increased physiological responses induced by active recovery brings about gains in physiological and performance adaptations, when rest intervals were kept short during 2 weeks of repeated 10-second sprint training, it produced a greater HR demand, which resulted in greater endurance adaptations (26). Therefore, active recovery at low to moderate intensity ($\sim$40% $\dot{V}O_2$max) may induce greater endurance adaptations when performing Wingate-based SIT protocols.

Accordingly, this study aimed to determine effects of recovery intensity on endurance adaptations during Wingate-based SIT over 2 weeks by examining several endurance parameters. It was hypothesized that active recovery would induce greater endurance adaptations when compared with passive recovery because of a higher aerobic demand during the training.

METHODS

Experimental Approach to the Problem

This study aimed to investigate effects of recovery intensity on endurance performance adaptations during SIT over 2 weeks. All subjects were asked to maintain their normal diet and activity throughout the study period and to refrain from alcohol intake and any form of intense physical activity for 24 hours before each session. First, a 2-week (male subjects) or 4-week (female subjects) nonexercise period was set as a control period after 3 baseline measurements of peak oxygen uptake ($\dot{V}O_2$peak), critical power (CP) and 10-km time-trial performance had been completed (Table 1). Subsequently, the subjects performed the 3 preintervention measurements (i.e., $\dot{V}O_2$peak, CP, and 10-km time trial). They were then assigned to either active recovery group (ARG) ($N = 7$; M: 4; F: 3) or passive recovery group (PRG) ($N = 7$; M: 5; F: 2) according to their preintervention $\dot{V}O_2$peak, CP, and time-trial performance to ensure that both groups possessed similar pretraining values before the 2-week training (Tables 2 and 3). A 4-week control period was set for female subjects to ensure that they completed the measurements at the same stage of their menstrual cycles. Because of the same reason, the female subjects commenced their 2-week training interventions 2 weeks after they had completed the preintervention measurements. Three of 5 female subjects were taking oral contraceptive pills during the study period, but dose and type remained constant throughout. All subjects performed each session at a similar time of day ($\pm$2 hours) in a controlled environment throughout the study period. Each performance measurement was completed on a different occasion and separated by 48 hours.

Subjects

Fourteen healthy active male and female subjects (values measured as $\pm$ *SD*: M: 9; F: 5, ages 21–33 years) who took part in a minimum of 3-hour exercise per week participated in this study (Tables 1 and 2). All were physically active, but none of them were participating in regular sporting competitions during the study period. All subjects were fully informed

TABLE 1. Physical characteristics and endurance performance parameters of the subjects during the control period.*†

	ARG ($n = 6$)		PRG ($n = 6$)	
	Baseline	Preintervention	Baseline	Preintervention
Age (y)	23 ± 3	—	25 ± 4	—
Height (cm)	175.4 ± 10.1	—	172.6 ± 11.3	—
Body mass (kg)	75.7 ± 16.6	74.8 ± 16.5	68.2 ± 12.3	68.3 ± 11.6
Fat (%)	19.4 ± 9.4	19.5 ± 8.8	12.8 ± 7.0	13.0 ± 7.4
$\dot{V}O_2$peak (ml·min^{-1}·kg^{-1})	34.9 ± 4.7	38.2 ± 5.7	38.6 ± 4.7	38.9 ± 4.7
$\dot{V}O_2$peak (L·min^{-1})	2.66 ± 0.72	2.85 ± 0.64	2.65 ± 0.64	2.69 ± 0.68
HRmax (b·min^{-1})	185 ± 5	181 ± 6	178 ± 4	178 ± 10
Pmax (W)‡	209 ± 37	219 ± 42	207 ± 51	219 ± 56§
10-km time trial (s)	1,054 ± 81	1,007 ± 107	998 ± 165	994 ± 151
Critical power (W)	201 ± 44	212 ± 47	199 ± 52	203 ± 51
3-min total work (kJ)	47.9 ± 10.7	48.8 ± 11.5	45.6 ± 10.8	46.4 ± 11.0

*ARG = active recovery group; PRG = passive recovery group; $\dot{V}O_2$peak = peak oxygen uptake; HRmax = maximal heart rate; Pmax = maximal incremental power output.
†Values are mean values ± *SD*.
‡Main effect for time ($p < 0.05$). $n = 5$ for 10-km time trial, critical power, and 3-minute total work in PRG.
§$p = 0.076$ vs. baseline within the same group.

both verbally and in writing about the study before giving their written informed consent. The study was approved by the Research Ethics Committee of Abertay University and was performed in line with the Declaration of Helsinki.

Procedures

Body Composition Test. On the initial visit, subjects reported to the Human Performance Laboratory after a 4-hour fast before an incremental test. Body composition was recorded on a calibrated bioimpedance meter (Tanita 330; Tanita Co., Ltd., Tokyo, Japan) where body fat and body mass were recorded.

Performance Measurement During the Incremental Test. After having completed the body composition measurement, they performed an incremental test to exhaustion to determine their $\dot{V}O_2$peak on a cycle ergometer (Monark Ergomedic 874E; Monark, Varberg, Sweden). The subjects were connected to a breath-by-breath gas analyzer (Metalyzer 3B gas analyzer; Cortex, Leipzig, Germany), and the test commenced at an initial power output of 70 W, with an additional 35 W increase every 3 minutes, until volitional exhaustion or the subjects could not maintain 70 rpm, despite strong verbal encouragement. Maximal incremental power output (Pmax) was calculated from the last completed work rate, plus the fraction of time spent in the final noncompleted work rate multiplied by the work rate increment (i.e., 35 W) (1). Respiratory gas exchange measures were averaged every 30 seconds with $\dot{V}O_2$peak calculated as the highest oxygen consumed over a 30-second period. Heart rate was recorded throughout using a HR monitor (Polar Electro, Kempele, Finland) with maximal HR (HRmax) defined as the highest HR recorded over a 30-second period. Because of schedule diffi-

culties, 6 of 7 subjects from both groups competed the incremental tests during the control period (Table 1).

Three-Minute All-Out Cycling Test. On the second visit, they performed a 3-minute all-out cycling test to determine their CP. They first cycled against 60 W for 5 minutes on a cycle ergometer (Monark Ergomedic 894E; Monark) to warm up. Before starting the test, subjects were connected to a breath-by-breath gas analyzer (Metalyzer 3B gas analyzer; Cortex) and had a HR monitor attached (Polar) to record $\dot{V}O_2$ and HR during the test. The test then began when the subjects reached 110 rpm where resistance was applied (4.5% of body mass). They pedaled with an all-out effort for 3 minutes. Although strong verbal encouragement was given, no feedback on the elapsed time was provided to avoid pacing. Power output was recorded using Monark software (Monark Anaerobic Test Software Version 2.24.2; Monark), and average power output over the final 30 seconds was defined as CP. This method has been shown to provide a valid estimation of CP with no difference from the conventionally estimated CP or one derived from a 3-minute all-out cycling test on an electronically braked cycle ergometer (7). Power produced over each 30-second block and 3-minute total work was also calculated as the integral of power output recorded every second to find changes in performance throughout 3 minutes. Cardiorespiratory measures were averaged every 30 seconds, and the highest and average $\dot{V}O_2$ and HR over 3 minutes were determined. Cardiorespiratory data for ARG only include 6 subjects because of a mechanical error with the gas analyzer occurred during the post-test in 1 subject. Moreover, because of schedule difficulties, 6 of 7 subjects from ARG and 5 of 7

TABLE 2. Physical and physiological characteristics of the subjects before and after the training intervention.*†

	ARG ($n = 7$)		PRG ($n = 7$)	
	Pre	Post	Pre	Post
Age (y)	23 ± 3	–	25 ± 4	–
Height (cm)	173.0 ± 11.7	–	172.2 ± 10.7	–
Body mass (kg)	72.7 ± 16.1	72.9 ± 16.3	71.1 ± 12.9	72.3 ± 13.4
Fat (%)	20.6 ± 8.6	19.8 ± 8.6	16.7 ± 11.8	16.4 ± 12.2
$\dot{V}O_2$peak (ml·min^{-1}·kg^{-1})	37.1 ± 6.0	37.3 ± 7.4	36.8 ± 7.2	36.4 ± 6.9
$\dot{V}O_2$peak (L·min^{-1})	2.70 ± 0.69	2.75 ± 0.87	2.61 ± 0.67	2.60 ± 0.55
HRmax (b·min^{-1})	182 ± 5	180 ± 9	180 ± 10	177 ± 10
Pmax (W)‡§	210 ± 45	$221 \pm 47\|$	210 ± 56	212 ± 55
10-km time trial (s)¶	$1,012 \pm 99$	925 ± 106#	$1,040 \pm 155$	970 ± 138**

*ARG = active recovery group; PRG = passive recovery group; $\dot{V}O_2$peak = peak oxygen uptake; HRmax = maximal heart rate; Pmax = maximal incremental power output.
†Values are mean values $\pm$ *SD*.
‡Main effect for time ($p < 0.05$).
§A tendency for time by group interaction effect ($p = 0.088$).
‖$p = 0.063$ vs. pre within the same group.
¶Main effect for time ($p < 0.01$).
#$p < 0.01$ vs. pre within the same group.
**$p < 0.05$ vs. pre within the same group.

TABLE 3. Performance parameters and cardiorespiratory responses during the 3-minute all-out cycling tests.*†‡

	ARG		PRG	
	Pre	Post	Pre	Post
Critical power (W)§‖	202 ± 50	218 ± 59¶	192 ± 46	193 ± 49
Total work (kJ)#**	46.5 ± 12.1	48.2 ± 13.3¶	44.5 ± 9.5	44.9 ± 10.2
Peak $\dot{V}O_2$ (L·min⁻¹)	2.66 ± 0.77	2.62 ± 0.85	2.67 ± 0.61	2.69 ± 0.63
Average $\dot{V}O_2$ (L·min⁻¹)	2.34 ± 0.76	2.36 ± 0.75	2.42 ± 0.54	2.40 ± 0.56
Peak HR (b·min⁻¹)§	175 ± 7	171 ± 7††	176 ± 10	173 ± 10
Average HR (b·min⁻¹)#	167 ± 8	161 ± 8††	167 ± 10	166 ± 11

*ARG = active recovery group; PRG = passive recovery group.
†Values are mean values ± SD.
‡Cardiorespiratory data for ARG only include 6 subjects.
§Main effect for time ($p < 0.01$).
‖Time by group interaction effect ($p < 0.05$).
¶$p < 0.05$ vs. pre within the same group.
#Main effect for time ($p < 0.05$).
**A tendency for time by group interaction effect ($p = 0.082$).
††$p < 0.01$ vs. pre within the same group.

subjects from PRG completed the 3-minute all-out cycling tests during the control period, respectively (Table 1).

Ten-Kilometer Cycling Time Trial. On the third visit, the subjects performed a self-paced 10-km cycling time trial on a cycle ergometer (Monark Ergomedic 894E; Monark) against a fixed resistance (2.0 kg for male subjects; 1.5 kg for female subjects) where they were asked to complete the set distance as fast as possible. No information on time, power output, and pedal frequency was provided, whereas the amount of distance covered was visible on the screen. Because of schedule difficulties, 6 of 7

subjects from ARG and 5 of 7 subjects from PRG competed the 10-km time trial during the control period, respectively (Table 1).

Training Sessions. The training protocol was identical for both groups consisting of 4 to six 30-second sprints against 7.5% of body mass interspersed with 4-minute recovery (20). However, while ARG cycled at 40% $\dot{V}O_2$peak during the recovery, PRG either remained stationary on the bike or cycled unloaded at a low speed (<50 rpm) as previously reported (12,13). The recovery intensity for ARG was derived from the linear relationship between each individual's $\dot{V}O_2$ and work rate during the incremental test. Both groups performed their respective training protocol 3 times per week for 2 weeks (6 sessions in total) and sprint load increased with time (4 sprints for the first 2 sessions, 5 sprints for the mid 2 sessions, and 6 sprints for the last 2 sessions) as previously described (20). Respiratory gas measures (Metalyzer 3B gas analyzer, Cortex) and HR (Polar) were recorded during the first 4 sprints and recovery periods in the first and last training sessions to investigate differences in cardiorespiratory responses between and within groups. $\dot{V}O_2$ and HR were averaged every 5 seconds during the sprint and recovery intervals. Cardiorespiratory data for PRG only include 6 subjects because of increased feelings of discomfort and nausea in 1 subject resulting from wearing the measuring equipment.
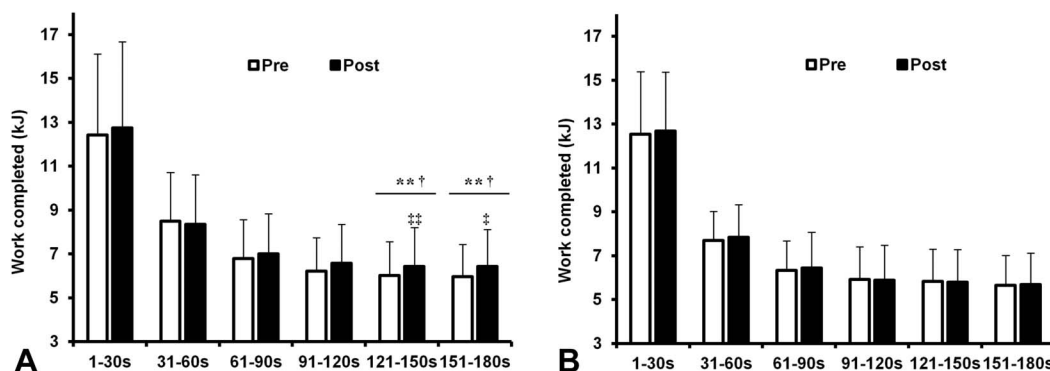

Figure 1. Power produced over each 30-second section during the 3-minute all-out cycling tests in active recovery group (A) and passive recovery group (B). **Main effect for time ($p < 0.01$). †Time by group interaction effect ($p < 0.05$). ‡‡$p < 0.01$ vs. pre within the same group. ‡$p < 0.05$ vs. pre within the same group.
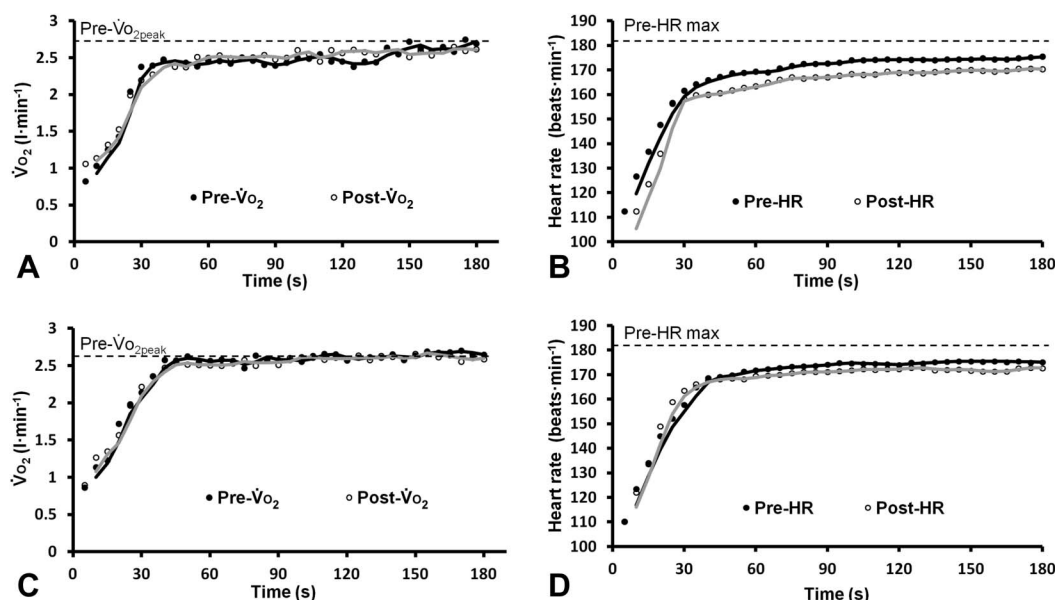
Figure 2. V̇o₂ and HR responses during the 3-minute all-out cycling tests in active recovery group (A and B) and passive recovery group (C and D). Dashed lines indicate V̇o₂peak and HRmax determined in the preincremental test. Error bars are not shown for clarity. Cardiorespiratory data for ARG only include 6 subjects. HR = heart rate.

To assess sprint work, the best peak power and total work during each session were determined. Furthermore, in an attempt to evaluate the reproducibility of power during the training, power drop rate across the sprints in each session was also calculated using the following formula;

Reproducibility of power :
$$([\text{sum of PO from all sprints}]/ \text{total number of sprints})/\text{best PO} \times 100$$

where PO is power output (either peak or average) (23).

Peak and average power were automatically determined through Monark software, whereas total work was determined by integrating power output recorded every second.

Postintervention Tests. Forty-eight hours after the last training sessions, the subjects performed the postintervention tests. The order of the measurements was identical to the preintervention tests, and each measurement was separated by 48 hours.

Statistical Analyses

All data are presented as mean values ± *SD*. Before conducting parametric tests, a Shapiro-Wilk test was performed to ensure that all values were normally distributed. Effects of training on each variable were analyzed using a 2-way analysis of variance with between (group) and repeated (time) factors. Where the analyses revealed a significant main effect for time or time × group interaction effect, individual paired-samples *t*-tests were performed to determine the origin of such effects. When the post hoc paired *t*-tests showed a significant training effect, Cohen's *d* was calculated to quantify the magnitude of such effect (i.e., pre-to-post difference). Because of the within-subject factor, it was corrected for dependence between mean values using the equation suggested by Morris and DeShon (30); $d = M_{\text{diff}}/SD_{\text{pooled}} \sqrt{2(1-r)}$, where M_{diff} is mean difference between conditions, SD_{pooled} is pooled *SD*, and *r* is correlation between mean values (30). Cohen's effect size was defined as follows: $d < 0.2$ trivial, 0.2–0.5 small, 0.6–1.1 moderate, and 1.2–1.9 as a large effect (15). All statistics were run on IBM SPSS Version 22.0 for Windows, and the level of significance was set at $p \leq 0.05$.

RESULTS

Anthropometric Measures and Performance Parameters During the Control Period

During the control period, body composition or endurance performance parameters were not significantly changed. However, PRG tended to increase maximal incremental power output during the 2-week control period ($p = 0.076$, Table 1).

Anthropometric Measures and Performance Variables in the Incremental Tests

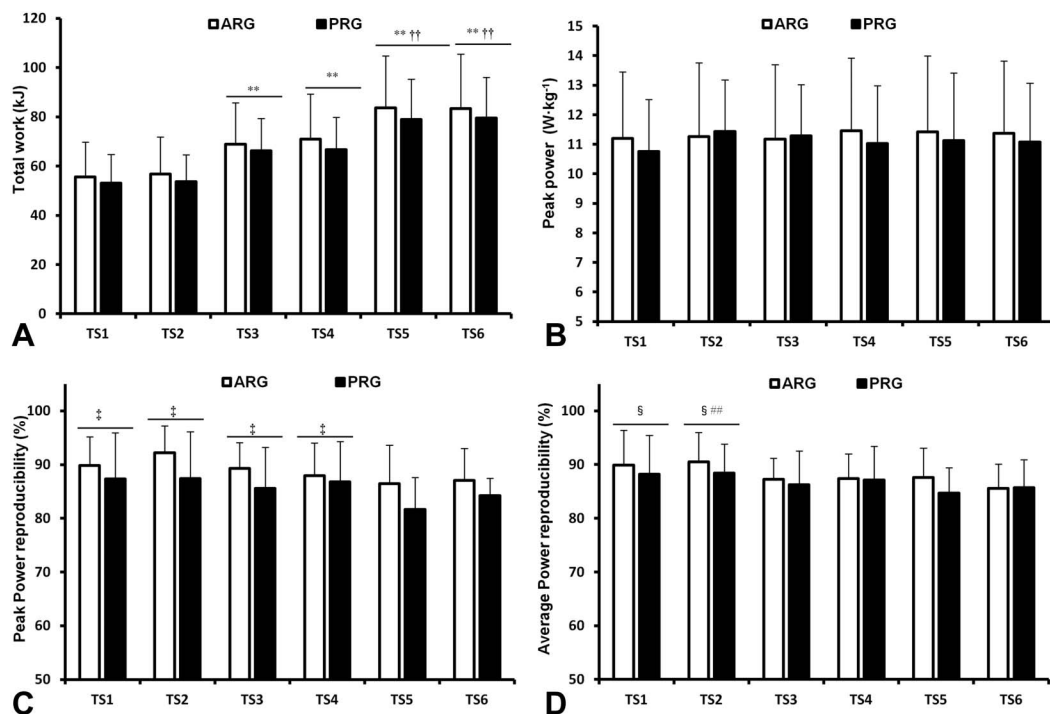There was no change in body composition after 2 weeks of SIT (Table 2).

Figure 3. Total work (A), peak power (B), and the reproducibility of peak power (C) and average power (D) during the training sessions. ** $p < 0.01$ vs. training sessions 1 and 2. †† $p < 0.01$ vs. training sessions 3 and 4. ‡ $p < 0.05$ vs. training session 5. § $p < 0.05$ vs. training sessions 5 and 6. ## $p < 0.01$ vs. training sessions 3. ARG = active recovery group; PRG = passive recovery group.

$\dot{V}O_2$peak, Pmax, and HRmax were similar between the groups at preintervention (Table 2). $\dot{V}O_2$peak and HRmax were unchanged after 2 weeks of SIT with either active or passive recovery (Table 2). However, there was a trend for Pmax to be increased from preintervention to postintervention in the ARG only (5.3%, $d = 0.88$, $p = 0.063$, Table 2).

Ten-Kilometer Cycling Time-Trial Performance

Ten-kilometer time-trial performance was similar between the groups at preintervention (Table 2). Ten-kilometer time-trial performance was significantly improved preintervention to postintervention in both ARG and PRG (ARG: 8.6%, $d = 1.60$, $p = 0.006$; PRG: 6.7%, $d = 0.96$, $p = 0.048$, Table 2).

TABLE 4. Cardiorespiratory responses during the first and last training sessions.*†‡

	ARG		PRG	
	Session 1	Session 6	Session 1	Session 6
$\dot{V}O_2$average over 4 sprints (L·min^{-1})	1.94 ± 0.61	2.02 ± 0.61	1.80 ± 0.36	1.76 ± 0.25
$\dot{V}O_2$average over 4 rest periods (L·min^{-1})§‖	1.77 ± 0.40	1.85 ± 0.42¶	1.16 ± 0.16	1.21 ± 0.15
HRaverage over 4 sprints (b·min^{-1})	154 ± 10	152 ± 10	147 ± 11	143 ± 11
HRaverage over 4 rest periods (b·min^{-1})#	152 ± 11	153 ± 11	136 ± 15	132 ± 12

*ARG = active recovery group; PRG = passive recovery group.
†Values are mean values ± SD.
‡Cardiorespiratory data for PRG only include 6 subjects.
§Main effect for time ($p < 0.05$).
‖Main difference between groups ($p < 0.01$).
¶$p < 0.05$ vs. session 1 within the same group.
#Main difference between groups ($p < 0.05$).

Three-Minute All-Out Cycling Test

Critical power was similar between the groups at preintervention (Table 3) and remained unchanged in PRG after 2 weeks (Table 3). After 2 weeks of SIT with active recovery, CP was significantly increased by 7.9% ($d = 1.75$, $p = 0.015$, Table 3). Passive recovery group was also unchanged after 2 weeks for 3-minute total work (Table 3) and power production over each 30-second block throughout 3 minutes (Figure 1B). In ARG, the total work was significantly increased by 3.7% after 2 weeks ($d = 1.84$, $p = 0.022$, Table 3), and there was a trend for 30-second power production to be increased with the elapsed time pre to post, reaching a significance during the fifth 30-second (6.0 ± 1.5 to 6.4 ± 1.8 kJ, $d = 2.64$, $p = 0.008$) and sixth 30-second blocks (6.0 ± 1.5 to 6.4 ± 1.7 kJ, $d = 1.72$, $p = 0.012$) (Figure 1A).

During the CP test, peak and average $\dot{V}O_2$ remained unchanged in both groups, while peak and average heart rates were significantly decreased in ARG after 2 weeks (Table 3 and Figure 2).

Performance and Cardiorespiratory Responses During the Training

Peak power remained unchanged throughout 2 weeks in both groups (Figure 3B). Although total work increased with sprint number ($p < 0.01$), no difference was observed between the groups (Figure 3A). The decline in peak power reproducibility was seen during session 5 compared with sessions 1, 2, 3, and 4 ($p < 0.01$, Figure 3C) with no significant difference between the groups (Figure 3C). Likewise, although the reproducibility of average power was reduced during sessions 5 and 6 compared with sessions 1 and 2 ($p < 0.05$, Figure 3D) and session 3 compared with session 2 ($p < 0.01$, Figure 3D), similar values were observed between the groups. There was no difference between the groups in $\dot{V}O_2$ and HR during the sprints, whereas those variables were significantly elevated in ARG compared with PRG during the recovery intervals ($p < 0.05$, Table 4). While recovery $\dot{V}O_2$ was significantly increased in ARG from session 1 to 6 ($p < 0.05$, Table 4), other cardiorespiratory measures were not significantly altered from session 1–6 in either group (Table 4).

DISCUSSION

This study demonstrated that endurance performance adaptations can be augmented by increasing recovery intensity during typical Wingate-based SIT over 2 weeks. Although both training groups improved time-trial performance, only the ARG significantly increased CP and 3-minute total work with a trend of increased maximal incremental power output. This suggests that the arrangement of recovery mode would play a role in bringing about training benefits when performing SIT.

There was no improvement in $\dot{V}O_2$peak in either training group in this study (Table 2). This is similar to what has been shown previously when using the same training protocol (12,13). When longer recovery, greater resistive force, or

more sprints are used, then 2 weeks of Wingate protocols has been shown to improve $\dot{V}O_2$max or $\dot{V}O_2$peak (2,4,23). Although gains in $\dot{V}O_2$max are not totally attributed to increased muscle oxidative capacity (12,13,33), peripheral adaptations have been associated with increased $\dot{V}O_2$max after SIT (22,27). Indeed, exercise intensity (i.e., the level of power production) has been suggested to be a key factor in inducing oxidative adaptations in skeletal muscles, type II fibers in particular (4,10,33), and therefore, the selection of recovery duration or resistive load may impact overall aerobic adaptations (2,23,38). Although Bailey et al. (4) used the traditional resistive load (7.5% of body mass) and recovery duration (4 minutes), greater training volume (35 sprints in total) performed in their study compared with the aforementioned 2-week Wingate studies or the current study (30 sprints in total) might have facilitated the increased $\dot{V}O_2$peak (4,21). In this study, active recovery at 40% of $\dot{V}O_2$peak did not improve power production compared with passive recovery during the training (Figure 3), which was somewhat contrary to previous findings (9,34). Considering the relatively low fitness level of our subjects, a lower recovery intensity (e.g., 20% of $\dot{V}O_2$peak) might have been more suitable for improving power production (17,35), and thus inducing greater peripheral adaptations (4,10,33). On the other hand, overall oxygen demand in ARG during the training was approximately 69% of pre-$\dot{V}O_2$peak in this study (Table 4). Given that a training program eliciting a high percentage of $\dot{V}O_2$max (i.e., $\geq 90\%$ $\dot{V}O_2$max) has been suggested to maximally stimulate the oxygen transport and utilization systems (11), adoption of a higher recovery intensity would be an option for ensuring a greater cardiorespiratory load especially when the main purpose of a training program is to obtain a central adaptation (e.g., increased stroke volume).

After 2 weeks of SIT, there was a 5.3% increase in maximal incremental power output (albeit not statistically significant) in ARG (Table 2). Previously, maximal incremental power output has been primarily attributed to oxygen transport capacity (24). However, considering the lack of improvement in $\dot{V}O_2$peak, there seems to have been limited changes in cardiac function (e.g., maximal cardiac output) after the training in the current study (6). Hence, the trend of increased Pmax observed in ARG would be accounted for peripheral adaptations such as improved capillarization. Daussin et al. (16) demonstrated that 8 weeks of moderate-intensity continuous training (CT, 20–35 minutes of cycling at ~61% $\dot{V}O_2$max) brought about greater improvements in capillary density and vascular conductance compared with the same duration of high-intensity aerobic interval training (IT) at ~90% $\dot{V}O_2$max (16). Because they matched total work between the training protocols, greater capillarization of skeletal muscle seen in the CT may be explained by constant cardiovascular load caused by the CT. Although the workload itself was rapidly reduced in ARG after each 30-second sprint (i.e., 40% of $\dot{V}O_2$peak) in

the current study, oxygen demand of recovery phases remained elevated (e.g., recovery $\dot{V}O_2$ in ARG was greater than 65% of pre-$\dot{V}O_2$peak, Table 4). Therefore, it is possible that there were greater improvements in capillary density after 2 weeks of SIT with active recovery, resulting in improved O_2 supply and thus maximal endurance capacity. Nevertheless, because muscle biopsies were not obtained in this study, this remains to be elucidated.

Both groups improved the 10-km time-trial performance after the training (ARG: 8.6%; PRG: 6.7%, Table 2). The magnitude of improvement is comparable with the previous 2-week Wingate-based studies that report improvements of between 5 and 10% in 5-km (23), 750-kJ (20), and 250-kJ (3,12) cycling time trials. The improvement in time-trial performance occurs without any improvement in $\dot{V}O_2$peak but with increased activity of mitochondrial enzymes such as citrate synthase and pyruvate dehydrogenase (12). Therefore, the observed time-trial improvements in this study may reflect an improved muscle oxidative potential previously reported after 2 weeks of Wingate-based SIT (12,13,20). Although improved time-trial performance cannot be totally attributed to mitochondrial adaptations (14) and other factors such as improved substrate utilization (12) or muscle buffering capacity (20) would also account for the improvements in time-trial performance, muscle oxidative capacity has been shown to be the primary predictor of cycling time-trial performance (24). Given that both training groups improved the performance to a similar extent in the current study, the intermittent nature of SIT seems to be a driving factor in increasing muscle oxidative capacity irrespective of recovery mode (14,16).

Zelt et al. (40) demonstrated that 4 weeks of Wingate-based SIT increased CP by approximately 5–7% (40), indicating that sprint-type (all-out) IT can also improve this parameter in addition to submaximal endurance training (18,25) and high-intensity but constant-load IT (18,32,36). Vanhatalo et al. (36) observed a 7% increase in $\dot{V}O_2$peak during a 3-minute all-out cycling test in addition to a 10% increase in CP after 12 sessions of aerobic-type high-intensity IT over 4 weeks (36). In the current study, only ARG improved CP and 3-minute total work, without any gains in peak or average $\dot{V}O_2$ but with a reduction in HR response during the 3-minute CP test after the intervention (Table 3 and Figure 2A, B). The decreased HR demand with unchanged $\dot{V}O_2$ could indicate an improved O_2 transport capacity. Similar to the maximal incremental power output, this may have been achieved through increased local muscle (microvascular) O_2 delivery (29). Indeed, locomotor skeletal muscle O_2 perfusion has been shown to be the limiting factor for maximal cycling exercise (31). The greater adaptations in CP than in Pmax seen after 2 weeks of SIT with active recovery may be attributable to the difference in the testing modality (i.e., incremental vs. all-out exercise). Anaerobic metabolism has been shown to occur from the initial phases of all-out exercise, whereas anaerobic energy production pro-gressively increases during incremental exercise (28,37). Therefore, there would be greater depletion of anaerobic energy sources in the final phases of 3-minute all-out exercise than those of incremental exercise, suggesting increased importance of muscle O_2 supply with time during the 3-minute all-out exercise (19). Taken together, the increased power production over the second 90 seconds of the test in ARG (Figure 1A) would indicate improved muscle O_2 perfusion after the training in this group (29).

Although Zelt et al. (40) used unloaded cycling as a recovery modality during rest periods, greater training volume in their study (12 sessions over 4 weeks) compared with the current study (6 sessions over 2 weeks) may have allowed for the increased CP in their study (40). Therefore, it could be assumed that, with active recovery at low to moderate intensity (~40% $\dot{V}O_2$peak), it induces rapid endurance adaptations and a greater training volume would be required to induce similar improvements with passive or very light active recovery.

In the current study, there was a slight difference in the number of subjects completing the 3-minute all-out and 10-km time-trail tests between the groups during the control period (ARG vs. PRG: 6 vs. 5), which might have affected the observed results. Nevertheless, there were no significant changes in these performance parameters in a control group consisting of recreationally active male and female adults in a recent study (39). Moreover, we made every effort to avoid any pacing during the 3-minute all-out tests by carefully looking at power production every 30 seconds throughout the tests (Figure 1). Indeed, if there was any sign of pacing (e.g., an increase in power production during the later phases of the tests), we asked our subjects to redo the test, until no sign of pacing was confirmed, suggesting that the difference in the performance adaptations between the groups are most likely explained by the difference in recovery intensity during the training.

PRACTICAL APPLICATIONS

This study demonstrates for the first time that active recovery induces greater endurance adaptations, especially in maximal whole-body exercise (i.e., CP and maximal incremental power output) when performing sprint-type IT. The findings from the current study indicate that active recovery at 40% $\dot{V}O_2$peak provides an elevated cardiorespiratory demand during the training, resulting in greater endurance adaptations. Therefore, when the goal of a training program is to enhance endurance performance, active recovery (~40% of $\dot{V}O_2$peak) should be used during SIT to gain greater training benefits without increasing total training time commitment.

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