Accepted: 5 December 2016

DOI: 10.1111/sms.12831

ORIGINAL ARTICLE

WILEY

# Effects of reduced-volume of sprint interval training and the time course of physiological and performance adaptations

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This study sought to determine the time course of training adaptations to two different sprint interval training programmes with the same sprint: rest ratio (1:8) but different sprint duration. Nine participants (M: 7; F: 2) were assigned to 15-second training group (15TG) consisting of 4-6 × 15-second sprints interspersed with 2-minute recovery, whereas eight participants (M: 5; F: 3) were assigned to 30-second training group (30TG) consisting of 4-6 × 30 second sprints interspersed with 4-minute recovery. Both groups performed their respective training twice per week over 9 weeks and changes in peak oxygen uptake ($\dot{V}O_{2peak}$) and time to exhaustion (TTE) were assessed every 3 weeks. Additional eight healthy active adults (M: 6; F: 2) completed the performance assessments 9 weeks apart without performing training (control group, CON). Following 9 weeks of training, both groups improved $\dot{V}O_{2peak}$ (15TG: 12.1%; 30TG: 12.8%, $P<.05$) and TTE (15TG: 16.2%; 30TG: 12.8%, $P<.01$) to a similar extent. However, while both groups showed the greatest gains in $\dot{V}O_{2peak}$ at 3 weeks (15TG: 16.6%; 30TG: 17.0%, $P<.001$), those in TTE were greatest at 9 weeks. CON did not change any of performance variables following 9 weeks. This study demonstrated that while the changes in cardiorespiratory function plateau within several weeks with sprint interval training, endurance capacity (TTE) is more sensitive to such training over a longer time frame in moderately-trained individuals. Furthermore, a 50% reduction in sprint duration does not diminish overall training adaptations over 9 weeks.

**KEYWORDS**
endurance capacity, peak oxygen uptake, sprint-to-rest ratio, time course of training adaptations

## 1 | INTRODUCTION

It has been demonstrated that Wingate-based sprint interval training (SIT) consisting of 4-6 30-second sprints interspersed with 4 minutes of recovery promotes comparable metabolic and physiological adaptations (eg, an improved mitochondrial function) to those obtained from traditional aerobic exercise training (eg, 60-90 minutes of continuous cycling at 65% $\dot{V}O_{2max}$) despite its low training volume (2-3 minutes of all-out efforts).[1,2] Nevertheless, the conventional 30-second Wingate-based SIT may not be necessarily time-efficient if warm-up and recovery periods are included, amounting to ~30 minutes.[1,2] It has been shown that the majority of anaerobic metabolism (ie, the degradation of phosphocreatine and glycogen) occurs within the first 15 seconds during a 30-second maximal sprint[3–5] and that aerobic and anaerobic metabolism increases and decreases, respectively, with successive bouts irrespective of sprint duration (6-30 seconds) when sprints are separated by 30-second to 4-minute recovery.[3–6] Therefore, shorter sprint protocol (≤15 seconds) may

**TABLE 1** Resting measures before and after the experimental period

| | 15TG (n=9) | | 30TG (n=8) | | CON (n=8) | |
|---|---|---|---|---|---|---|
| | **Pre** | **Post** | **Pre** | **Post** | **Pre** | **Post** |
| Age (y) | 27.0±2.7 | – | 27.5±4.5 | – | 25.9±5.1 | – |
| Height (cm) | 177.4±10.4 | – | 174.6±9.5 | – | 174.8±8.6 | – |
| Body mass (kg) | 77.1±18.6 | 76.7±17.5 | 71.8±13.6 | 70.8±11.8 | 76.1±14.2 | 76.5±13.8 |
| Fat mass (kg) | 13.3±7.9 | 13.2±8.2 | 11.0±5.9 | 10.8±6.4 | 13.1±7.4 | 13.2±7.2 |
| Lean body mass (kg) | 63.8±15.1 | 63.5±14.6 | 60.8±16.3 | 60.0±15.4 | 63.0±13.2 | 63.3±13.0 |
| SBP (mm Hg) | 123±11 | 120±9 | 121±15 | 123±11 | 127±8 | 129±7 |
| DBP (mm Hg) | 73±8 | 75±7 | 73±11 | 76±7 | 76±8 | 77±7 |
| MAP (mm Hg) | 87±8 | 88±9 | 86±15 | 87±6 | 91±8 | 89±8 |
| PP (mm Hg) | 49±12 | 45±6 | 48±10 | 47±9 | 51±5 | 52±5 |
| HR rest (beats·min$^{-1}$) | 63±13 | 63±8 | 64±11 | 59±8 | 61±12 | 57±12 |

SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; PP, pulse pressure.
Values are means±SD.

induce a similar metabolic demand to that seen in Wingate-based SIT and reducing sprint duration may not result in a diminished training adaptation. Indeed, Hazell et al.[7] demonstrated that a reduced-volume of sprint interval training consisting of 4-6 10-second cycling sprints separated by 2- or 4-minute recovery was as effective as typical 30-second Wingate-based SIT in improving aerobic and anaerobic performance. Similarly, Zelt et al.[8] recently demonstrated that 4-6 15-second sprints interspersed with 4.75 minutes of recovery improved aerobic capacities such as $\dot{V}O_{2peak}$ and critical power to a similar extent compared with the conventional Wingate-based SIT. Hazell et al.[7] found that training intensity (the reproducibility of power during training) was increased in the 10-second training groups compared with the 30-second group and suggested that the level of power production rather than sprint duration would be important for inducing training benefits. Nevertheless, neither sprint duration nor sprint-to-rest ratio was matched among the groups (10:120, 10:240, 30:240 seconds) in their study, and thus, the improved reproducibility of power might have been attributed to greater sprint:rest ratio rather than shorter sprint duration itself. Indeed, it has recently been demonstrated that work-to-rest ratio alters the training adaptations to SIT, with 1:3 sprint-to-rest ratio producing more aerobic adaptations (improvements in time to exhaustion and 3-km running time-trial performance) and 1:12 sprint-to-rest ratio producing more anaerobic adaptations (increased power production during a 30-second Wingate test).[9] Therefore, further research is required to confirm whether shorter sprint protocol with the same sprint:rest ratio as Wingate-based SIT (1:8) also brings about an improved power restoration during training and consequently comparable training gains.

Despite the increased utilization of Wingate-based SIT to promote physiological and performance adaptations,[2,10–14] little is known regarding the time course of those adaptations. While Burgomaster et al.[1] found an increase in $\dot{V}O_{2peak}$ following 3 weeks of Wingate-based SIT, no further improvements were confirmed with another 3 weeks of the training in healthy young adults (baseline $\dot{V}O_{2peak}$: 41±2 ml·kg$^{-1}$·min$^{-1}$). In contrast, Gillen et al.[15] saw a continuous improvement in $\dot{V}O_{2peak}$ in their sedentary male participants (baseline $\dot{V}O_{2peak}$: 33±7 ml·kg$^{-1}$·min$^{-1}$) using 3 × 20-second all-out cycle sprints (with 2-minute recovery in between) over a 12-week intervention period. Although the discrepancies between the two studies may be attributed to the difference in baseline fitness level, the duration of sprint (ie, 30 vs 20 seconds) might have had a different impact on the time course of training adaptations.

In short, the purposes of the current study were two-fold, that is, to determine (i) effects of sprint duration (15 vs 30 seconds) on physiological and performance adaptations while matching sprint-to-rest ratio between training protocols (sprint: rest ratio of 1:8) and (ii) time course of those adaptations over 9 weeks (baseline, weeks 3, 6, and 9). It was hypothesized that the difference in sprint duration would not affect overall training adaptations and that the majority of cardiorespiratory and performance adaptations would occur during the initial phases.

## 2 | MATERIALS AND METHODS

### 2.1 | Participants

Twenty-seven healthy active adults (male: 20, female: 7) who took part in a minimum of 3-hour exercise per week initially participated in this study (Table 1). However, one male participant of each training group withdrew from the study due to injuries unrelated to the study. Consequently, 25 (18 males and

7 females) participants completed the current study. All were physically active performing various physical activities such as jogging, cycling, and resistance exercise on a regular basis (Table S1), but none of them were participating in regular sport competitions during the period of the study. All participants completed a Physical Activity Readiness Questionnaire (PARQ) to ensure there were no underlying health issues, and they were fully informed both verbally and in writing about the study before giving their informed consent. Two of seven female participants were taking oral conceptive pills during the study period, but dose and type remained constant throughout. The study was approved by the Institutional Ethics Committee and was carried out in line with the Declaration of Helsinki.

## 2.2 | Experimental design

All participants 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 prior to each session. Participants performed three baseline measurements on three different occasions, separated by 48 hours, to determine their peak oxygen uptake ($\dot{V}O_{2peak}$), critical power (CP), and 10-km cycling time-trial performance, respectively. They were then assigned to either 15-second training group consisting of 4-6 15-second cycle sprints interspersed with 2-minute active recovery at 40% of $\dot{V}O_{2peak}$ (15TG) (N=9; male: 7, female: 2) or 30-second training group consisting of 4-6 30-second cycle sprints interspersed with 4-minute active recovery at 40% of $\dot{V}O_{2peak}$ (30TG) (N=8; male: 5; female: 3) according to their baseline $\dot{V}O_{2peak}$, CP, and time-trial performance to ensure that both groups possessed similar baseline values before the training intervention. Additional eight recreationally active adults (male: 6, female: 2) performed the three baseline measurements 9 weeks apart without performing any training to act as a control group (CON). The participants from the training groups performed an incremental test to exhaustion to determine $\dot{V}O_{2peak}$ at 3 and 6 weeks in addition to the pre- and post-intervention tests (ie, the determination of $\dot{V}O_{2peak}$, CP, and 10-km time-trial performance). All participants performed each session at a similar time of day (±2 hours) in a controlled environment throughout the study period, and each session was separated by at least a period of 48 hours.

## 2.3 | Anthropometric measurements and performance assessments during incremental test

On the initial visit, participants reported to the laboratory at a time suitable for them after a 4-hour fast. Firstly, body composition was recorded. They removed their shoes and socks and had their height measured prior to stepping onto a calibrated bioelectrical impedance meter (SC-330ST Tanita Body Composition Analyser, Tanita Europe BV, Amsterdam, the Netherlands) where body fat mass and lean body mass were recorded (Table 1). Resting blood pressure and heart rate were then recorded using an automatic blood pressure monitor (Watch BP® office, Microlife Health Management Ltd., Cambridge, UK) after the participants had been seated for 5 minutes. They then performed an incremental test to exhaustion to determine their $\dot{V}O_{2peak}$ on a cycle ergometer (Monark Ergomedic 894E, Monark, Varberg, Sweden). Prior to starting the test, participants were connected to a breath-by-breath gas analyzer (Metalyzer®3B gas analyzer, Cortex, Leipzig, Germany) and had a heart rate monitor attached (Polar Electro, Kempele, Finland). The oxygen ($O_2$) and carbon dioxide ($CO_2$) analyzer systems were calibrated using ambient air with a gas mixture of known $O_2$ and $CO_2$ immediately before each test. Partial $O_2$ and $CO_2$ in ambient air were assumed to be 20.93% and 0.03%, respectively. The reference gas concentrations in $O_2$ and $CO_2$ used for the calibration were 17.10% and 5.00%, respectively. The turbine flowmeter of the Metalyzer®3B gas analyzer was calibrated using a 3-L calibration syringe (Hans Rudolph, inc., Kansas City, USA).

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 participants could not maintain 70 rpm despite strong verbal encouragement. Exercise duration at exhaustion was recorded to the nearest second and defined as time to exhaustion (TTE). Respiratory gas exchange measures were averaged every 30 seconds with $\dot{V}O_{2peak}$ calculated as the highest oxygen consumed over a 30-second period. Similarly, maximal heart rate (HR max) was defined as the highest heart rate recorded over a 30-second period. Oxygen pulse at $\dot{V}O_{2peak}$ was also calculated using the following equation: $\dot{V}O_2$ ($ml \cdot min^{-1}$)/HR ($beats \cdot min^{-1}$). Moreover, as it has been shown that a better estimation of stroke volume is achieved when oxygen pulse is corrected for bodyweight,[16,17] $O_2$ pulse was divided by weight in kilograms ($ml \cdot beat^{-1} \cdot kg^{-1}$) and multiplied by 100 as suggested by Oliveira et al.[17]

## 2.4 | 3-minute all-out cycling test

On the second visit, they performed a 3-minute all-out cycling test to determine their critical power. They first completed a 3-minute warm-up against 60 W on a cycle ergometer (Monark Ergomedic 894E, Monark, Varberg, Sweden). The test then began when the participants reached 110 rpm where resistance was applied (4.5% of bodyweight). They pedaled with an all-out effort for 3 minutes. While strong verbal encouragement was given, no feedback on the elapsed time was provided in an attempt to avoid pacing. Power output was recorded using Monark software (Monark Anaerobic Test Software version 2.24.2, Monark, Varberg, Sweden) 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.[18]

## 2.5 | 10-km cycling time trial

On the third visit, the participants performed a self-paced 10-km cycling time trial against a fixed resistance (male: 2 kg; female: 1.5 kg). They first completed a 3-minute warm-up against 60 W on a cycle ergometer (Monark Ergomedic 894E, Monark, Varberg, Sweden). They were then 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.

## 2.6 | Training protocol and assessment of repeated sprint performance

The training groups performed their respective training protocol against a predetermined resistance (male: 7.5% of bodyweight; female: 6.5% of bodyweight) twice per week over 9 weeks (18 sessions in total) and sprint load increased with time (ie, four sets for the initial 3 weeks, five sets for the second 3 weeks, and six sets for the last 3 weeks). All participants completed a 3-minute warm-up against 60 W on a cycle ergometer before performing all-out sprints. The recovery intensity (ie, 40% $\dot{V}O_{2peak}$) was derived from the linear relationship between each individual's $\dot{V}O_2$ and work rate during the incremental test, and it was recalculated for each participant every 3 weeks according to $\dot{V}O_{2peak}$ measurements.

Total work across the first four sprints was calculated to determine the difference in training volume between the groups (ie, 15TG vs 30TG) in addition to peak power over the four sprints. Furthermore, to assess the reproducibility of power during the training, power drop rate across the four sprints was also calculated using the following formula:

$$\text{Reproducibility of power: } ((PO1+PO2+PO3+PO4)/4)/\text{best PO}\times 100,$$

where PO is power output (either peak or average).[7]

Peak and average power were calculated automatically using the Monark software (Monark Anaerobic Test Software version 2.24.2, Monark, Varberg, Sweden), and total work was determined by integrating power output recorded every second. Total work, peak power, and the reproducibility of power were assessed every six sessions over 9 weeks (four times in total).

## 2.7 | Assessment of anaerobic and aerobic demands during training

The level of blood lactate was determined via fingertips' blood samples (Lactate pro, Arkray Inc., Kyoto, Japan) during the first training session, and it was used as a marker for anaerobic energy turnover.[3,5,19] Blood lactate concentration was measured at six different time points, that is, pre-sprint, 0, 3, 5, 8, and 10 minutes after the last sprint (sprint 4). The skin was punctured using an Accu-check single use lancet (Roche Diagnostics, UK) and pressure applied to the finger to draw the blood. The initial drop was discarded and the second drop was taken for analysis. Moreover, to assess aerobic demand during each training protocol, heart rate was recorded (Bioharness™ 2, Zephyr Technology, MD, USA) in the first, 7th and 13th training sessions and average values over four, five, and six sprints were determined, respectively. Representative example of HR response during the first session in each training protocol is shown in Figure 1a, b. In addition, HR was normalized to percentage of total time using a cubic spline method to directly compare heart rate responses between the two training protocols (Figure 1c).

## 2.8 | Post-intervention tests

A minimum of 48 hours and maximum of 72 hours after the last training sessions, participants from the training groups performed the post-intervention tests. The order of the testing was identical to the pre-intervention tests.

## 2.9 | Statistical analyses

All data are presented as means±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 two-way analysis of variance (ANOVA) with between (group) and repeated (pre to post) factors for all groups. A two-way ANOVA was also run with repeated measures (0, 3, 6, and 9 weeks) to determine the time course of physiological and performance adaptations to training, with training group used as a between-subjects factor (15TG vs 30TG). Likewise, heart rate and blood lactate accumulation during training were analyzed using a two-way mixed ANOVA. Where the analyses revealed a significant main effect for time or time x group interaction effect for all groups, individual paired samples t tests were performed to determine the origin of such effects. In the case of a significant main effect for time in the two-way mixed ANOVA for the training groups only, a one-way ANOVA with least significant difference (LSD) post-hoc test was performed to examine changes in variables over time for each training group. Where appropriate, Cohen's *d* was calculated to quantify the magnitude of difference within or between subjects. In the case of a within-subjects factor, it was corrected for dependence between means using the equation suggested by Morris and DeShon[20]; $d = M_{diff}/SD_{pooled}\sqrt{2(1-r)}$, where $M_{diff}$ is mean difference between conditions, $SD_{pooled}$ is pooled standard deviation, and $r$ is correlation between means. Cohen's

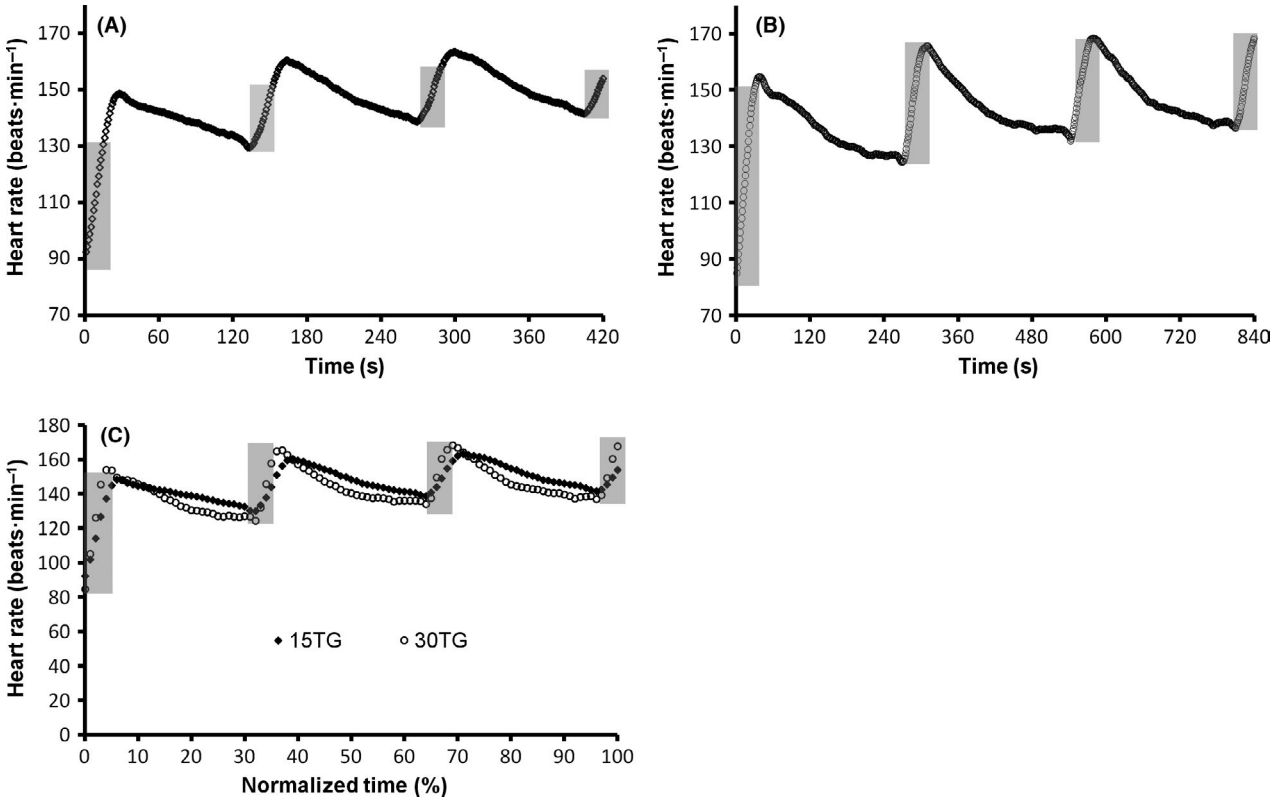


**FIGURE 1** Representative example of heart rate over four sprints and three rest periods (group mean) in 15TG (A), 30TG (B), and both groups (C). In A, and B, HR with actual running time is shown whereas HR is normalized to percentage of total time in C. Shaded areas indicate HR during sprints. Error bars are not shown for clarity

effect size was defined as follows: $d$<0.2 trivial effect, 0.2-0.5 small effect, 0.6-1.1 moderate effect, and 1.2-1.9 as a large effect.[21] All statistics were run on IBM® SPSS® version 22.0 for Windows, and the level of significance was set at $P$<.05.

## 3 | RESULTS

### 3.1 | Blood pressure and anthropometric measures

There was no change in blood pressure or body composition following 9 weeks of SIT or in the control group (Table 1).

### 3.2 | Performance variables

All performance measures were similar in all groups at baseline (Table 2). Both training groups significantly improved $\dot{V}O_{2peak}$ (15TG: 12.1%, $d$=1.77, $P$<.001; 30TG: 12.8%, $d$=1.27, $P$<.05, Table 2), $O_2$ pulse (15TG: 10.5%, $d$=1.64, $P$<.01; 30TG: 10.8%, $d$=1.05, $P$<.05, Table 2), time to exhaustion (15TG: 16.2%, $d$=2.17, $P$<.001; 30TG: 12.8%, $d$=1.70, $P$<.01, Table 2), and 10-km cycling time-trial performance (15TG: 8.6%, $d$=3.47, $P$<.01; 30TG: 7.2%, $d$=0.86, $P$<.05, Table 2), while only 15TG significantly increased critical power (7.8%, $d$=0.87, $P$<.05, Table 2). Although 30TG also increased critical power to a similar extent (7.4%, $d$=0.67), it did not reach a statistical significance ($P$=.11, Table 2). HR max was not significantly changed with 9 weeks of SIT (Table 2). All performance measures were not altered in CON following 9 weeks (Table 2).

### 3.3 | Heart rate and blood lactate responses during training sessions

There was no significant difference between the groups in session-averaged heart rate, whereas only 30TG increased the session-averaged HR with successive bouts (six vs four sprints, $P$<.05, $d$=0.98, Table 3). Both training groups similarly increased blood lactate accumulation following four 15- or 30-second sprints with the peak values observed immediately after sprint 4 (pre-sprint vs peak post-sprint value; 15TG: $P$<.001, $d$=8.28, 30TG: $P$<.001, $d$=14.7, Table 3). Blood lactate concentration gradually decreased with time during the 10-minute recovery phase in both groups (Table 3).

### 3.4 | Time course of changes in $\dot{V}O_{2peak}$, $O_2$ pulse, and time to exhaustion over 9 weeks

$\dot{V}O_{2peak}$ rapidly increased with both training protocols, and the highest values were observed at week 3

**TABLE 2** Performance measures before and after the experimental period

| | 15TG (n=9) | | 30TG (n=8) | | CON (n=8) | |
|---|---|---|---|---|---|---|
| | **Pre** | **Post** | **Pre** | **Post** | **Pre** | **Post** |
| $\dot{V}O_{2peak}$ ($ml^{-1}\cdot min^{-1}\cdot kg^{-1}$)$^{**,###}$ | 42.2±5.4 | 47.3±5.7$^{‡‡‡}$ | 40.6±9.6 | 45.8±7.9$^{‡}$ | 47.4±7.9 | 44.0±5.8 |
| $\dot{V}O_{2peak}$ ($L^{-1}\cdot min^{-1}$)$^{*,###}$ | 3.25±0.84 | 3.61±0.79$^{‡‡‡}$ | 2.92±0.83 | 3.23±0.65$^{‡}$ | 3.59±0.73 | 3.33±0.57 |
| HR max ($beats\cdot min^{-1}$) | 180±7 | 182±6 | 178±8 | 179±6 | 186±7 | 179±11 |
| $O_2$ pulse ($ml\cdot beat^{-1}\cdot kg^{-1}$)$^{**,##}$ | 23.7±3.6 | 26.2±2.9$^{‡‡}$ | 23.2±5.1 | 25.7±4.7$^{‡}$ | 25.7±4.2 | 24.6±2.9 |
| Time to exhaustion (s)$^{***,###}$ | 978±205 | 1136±264$^{‡‡‡}$ | 954±280 | 1076±283$^{‡‡}$ | 1037±221 | 1014±216 |
| Critical power (watts)$^{**}$ | 218±47 | 235±49$^{‡}$ | 204±47 | 219±52 | 219±54 | 222±55 |
| 10-km time trial (s)$^{***}$ | 977±160 | 893±112$^{‡‡}$ | 969±82 | 899±89$^{‡}$ | 912±103 | 902±112 |

$\dot{V}O_{2peak}$, peak oxygen uptake; HR max, maximal heart rate.

Values are means±SD. A two-way analysis of variance with repeated (time) and between (group) factors was performed to determine main effect of time, time-by-group interaction effect and main difference between groups, whereas paired *t* tests were employed to determine pre- to post-differences within the same group.

***Indicates $P<.001$ for main effect of time.

**Indicates $P<.01$ for main effect of time.

*Indicates $P<.05$ for main effect of time.

###Indicates $P<.001$ for time-by-group interaction effect.

##Indicates $P<.01$ for time-by-group interaction effect.

‡‡‡Indicates $P<.001$ vs pre within the same group.

‡‡Indicates $P<.01$ vs pre within the same group.

‡Indicates $P<.05$ vs pre within the same group.

**TABLE 3** Heart rate and blood lactate responses during the training

| | Training group | |
|---|---|---|
| **Physiological parameters** | **15TG** | **30TG** |
| Session-averaged HR ($beats\cdot min^{-1}$)$^{*}$ | | |
| Four sprints and three rest periods | 141±8 | 142±12 |
| Five sprints and four rest periods | 144±8 | 145±12 |
| Six sprints and five rest periods | 144±8 | 148±13$^{‡}$ |
| Blood lactate ($mmol\cdot L^{-1}$)$^{***}$ | | |
| Pre-sprint | 1.5±0.4 | 1.9±0.2 |
| Immediate post-sprint 4 | 13.4±2.1$^{aaa,bb}$ | 14.0±1.0$^{aaa,b,c}$ |
| 3-min post | 13.2±1.4$^{aaa,bb}$ | 13.8±1.2$^{aaa,b,c}$ |
| 5-min post | 12.6±1.8$^{aaa,bb}$ | 13.1±1.1$^{aaa}$ |
| 8-min post | 12.4±1.8$^{aaa,b}$ | 13.0±1.2$^{aaa}$ |
| 10-min post | 11.4±2.4$^{aaa}$ | 12.6±1.8$^{aaa}$ |

Values are means±SD. A two-way analysis of variance with repeated (time) and between (group) factors showed no interaction effect or main difference between groups in either heart rate or blood lactate values. Changes in the variables over time for each group were determined via a one-way ANOVA with LSD post-hoc test.

*Indicates $P<.05$ for main effect of sprint number.

***Indicates $P<.001$ for main effect of time.

‡Indicates $P<.05$ vs four sprints and three rest periods within the same group.

aaaIndicates $P<.001$ vs pre-sprint within the same group.

bbIndicates $P<.01$ vs 10-min post within the same group.

bIndicates $P<.05$ vs 10-min post within the same group.

cIndicates $P<.05$ vs 5-min post within the same group.

in both groups (15TG: 49.2±5.4 $ml\cdot min^{-1}\cdot kg^{-1}$, 30TG: 47.5±10.3 $ml\cdot min^{-1}\cdot kg^{-1}$, Figure 2a), indicating that the gain of $\dot{V}O_{2peak}$ plateaued after 3 weeks. Likewise, the highest $O_2$ pulse was observed following 3 weeks of the training in both groups (15TG: 27.9±3.2 $ml\cdot beat^{-1}\cdot kg^{-1}$, 30TG: 26.5±6.0 $ml\cdot beat^{-1}\cdot kg^{-1}$, Figure 2b). On the other hand, time to exhaustion was not significantly increased with training until week 6 and the greatest values were obtained at week 9 in both groups (15TG: 1136±264 seconds, 30TG: 1076±283 seconds, Figure 2c).

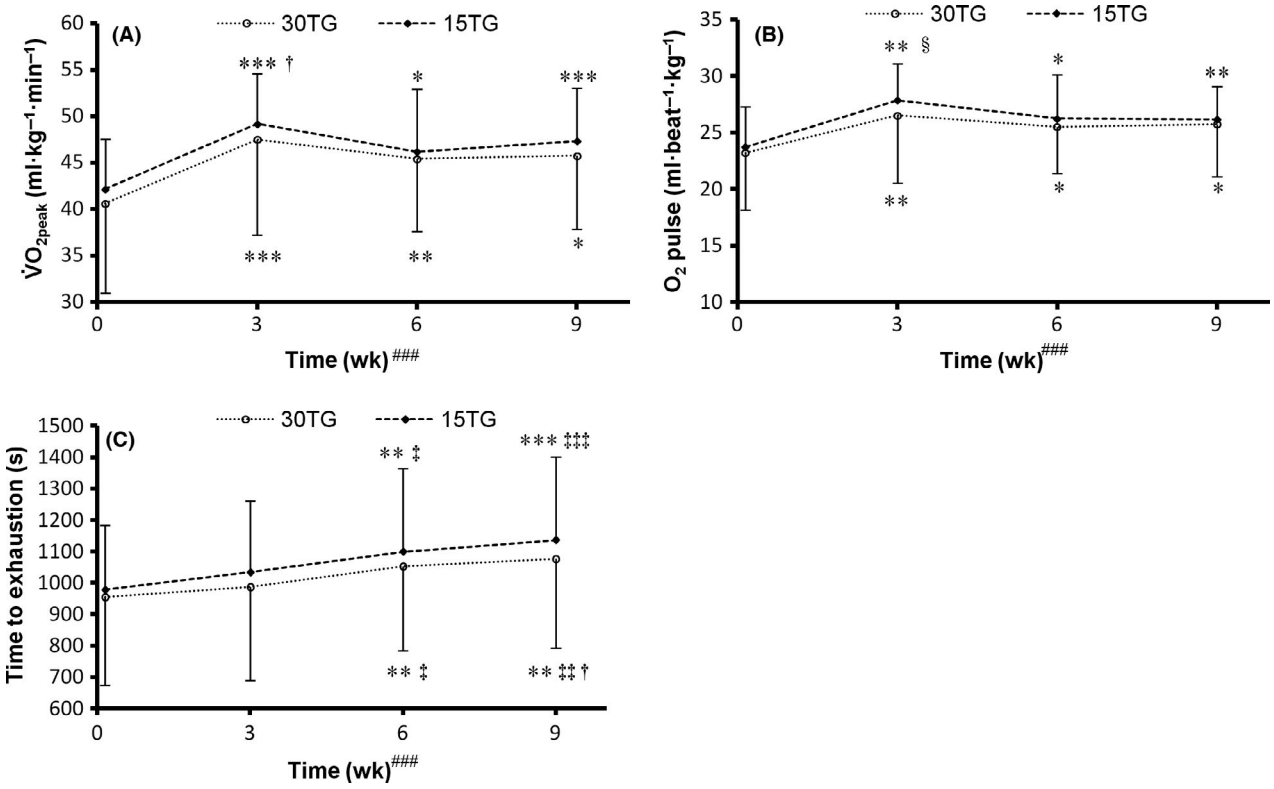


**FIGURE 2** Time course of changes in peak oxygen uptake (A), $O_2$ pulse (B), and time to exhaustion (C). ###Indicates $P<.001$ for main effect of time. ***Indicates $P<.001$ vs pre within the same group. **Indicates $P<.01$ vs pre within the same group. *Indicates $P<.05$ vs pre within the same group. ‡‡‡Indicates $P<.001$ vs week 3 within the same group. ‡‡Indicates $P<.01$ vs week 3 within the same group. ‡Indicates $P<.05$ vs week 3 within the same group. †Indicates $P<.05$ vs week 6 within the same group. §Indicates $P<.05$ vs week 9 within the same group. A two-way mixed ANOVA showed no interaction effect or main difference between groups in all variables. A one-way ANOVA with LSD post-hoc test was performed to determine changes in the variables over time for each group

### 3.5 | Time course of changes in repeated sprint performance over 9 weeks

30TG did not improve any assessment of repeated sprint performance over 9 weeks (Figure 3b), whereas 15TG significantly increased peak power and total work over the first four sprints during the sessions 6, 12, and 18 compared with the first session (Figure 3a). Percentages of changes from the session 1 in 15TG were 7.0% ($d$=2.12, $P<.001$), 7.4% ($d$=1.60, $P<.01$), and 7.9% ($d$=1.08, $P<.05$) in peak power, and 4.6% ($d$=0.78, $P<.05$), 5.4% ($d$=0.86, $P<.05$), and 5.4% ($d$=0.77, $P<.05$) in total work for the sessions 6, 12, and 18, respectively (Figure 3a). Total work was greater in 30TG compared with 15TG (15TG vs 30TG: 38.6±11.2 vs 57.8±17.6 kJ, $P<.05$, $d$=1.33, Figure 3a, b), whereas there was no difference between the groups in peak power or the reproducibility of peak and average power (Figure 3a-d).

## 4 | DISCUSSION

The present study demonstrated divergent effects of sprint interval training on physiological and performance adaptations in moderately-trained individuals. While the gain in $\dot{V}O_{2peak}$ reached a plateau following 3 weeks, time to exhaustion kept increasing until the end of the study in both 15- and 30-second training groups. In addition, reducing sprint duration did not diminish overall training adaptations, and indeed, only 15TG significantly increased critical power and repeated sprint performance. During the training, both sprint protocols resulted in similar session-averaged HR and blood lactate accumulation (Table 3) as well as power reproducibility (Figure 3), suggesting that when sprint-to-rest ratio is fixed (1:8), sprint duration can be reduced by 50% to provide a similar training stimulus.

### 4.1 | Performance measures in the incremental test

Both training groups increased $\dot{V}O_{2peak}$ to a similar extent following 9 weeks of training (12.1% and 12.8% for 15TG and 30TG, respectively); however, the greatest gains were observed at week 3 in both groups (16.6% and 17.0% for 15TG and 30TG, respectively). Similar to the current study, while Burgomaster et al.[1] saw a 7.3% improvement in $\dot{V}O_{2peak}$ following 3 weeks of Wingate-based SIT, it

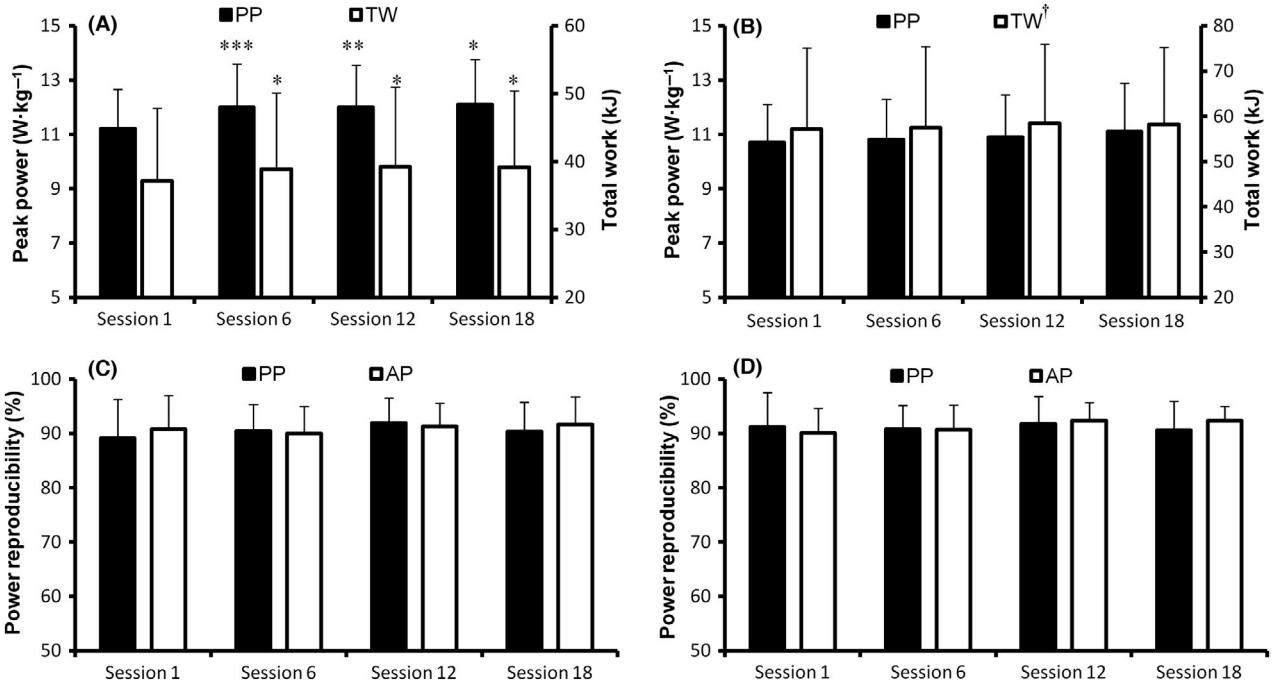


**FIGURE 3** Peak power and total work in 15TG (A) and 30TG (B) and the reproducibility of power in 15TG (C) and 30TG (D) during the first four sprints in the 1st, 6th, 12th, and 18th training sessions. †Indicates that total work is greater than 15TG ($P<.05$). ***Indicates $P<.001$ vs session 1 within the same group. **Indicates $P<.01$ vs session 1 within the same group. *Indicates $P<.05$ vs session 1 within the same group. A two-way mixed ANOVA showed a main difference in total work between the groups, whereas a one-way ANOVA with LSD post-hoc test demonstrated the improvements in sprint performance in 15TG

remained unchanged with additional 3 weeks of the training. In addition, none of the resting cardiovascular measures (resting HR and blood pressure variables) were altered in either training group (Table 1) which is in line with the study by Astorino et al.[10] who also observed an improvement in peak $O_2$ pulse but not in resting HR or blood pressure after 2 weeks of Wingate-based SIT. This indicates that sprint-type interval training may improve cardiac function during exercise without changes in resting cardiovascular mechanisms in healthy active adults. Nevertheless, contrary to the current study or the study by Astorino et al.,[10] Matsuo et al.[22] observed improvements in resting stroke volume and heart rate as well as gains in left ventricular mass following 8 weeks of SIT consisting of 7 × 30-second cycling at 120% $\dot{V}O_{2max}$ separated by 15-second rests. Moreover, Matsuo et al.[22] demonstrated a continuous increase in $\dot{V}O_{2max}$ throughout 8 weeks in healthy young men (baseline $\dot{V}O_{2max}$: 43.9±6.7 ml·kg$^{-1}$·min$^{-1}$). Likewise, Gillen et al.[15] have recently demonstrated that SIT consisting of 3 × 20-second all-out cycle sprints separated by 2 minutes of active recovery at 50W provides a continuous improvement in $\dot{V}O_{2peak}$ over 12 weeks in sedentary men ($\dot{V}O_{2peak}$: 33±7 ml·kg$^{-1}$·min$^{-1}$). The findings from Gillen et al.[15] may be explained by the low aerobic capacity of their participants given that the improved $\dot{V}O_{2peak}$ (~38 ml·kg$^{-1}$·min$^{-1}$) following 12 weeks of SIT was still lower than the baseline values observed in the study by Burgomaster et al.[1] (41±2 ml·kg$^{-1}$·min$^{-1}$) or the current study (15TG: 42.2±5.4 ml·kg$^{-1}$·min$^{-1}$; 30TG: 40.6±9.6 ml·kg$^{-1}$·min$^{-1}$). Although Matsuo et al.[22] recruited participants with similar level of baseline cardiorespiratory fitness to the current study, the short recovery periods (work: rest ratio: 2:1) employed by them may have provided a greater stimulus for adaptations of cardiorespiratory function. Indeed, Matsuo et al.[22] saw greater session-averaged HR in their SIT group (161 beats·min$^{-1}$) compared with 15TG (141-144 bpm) or 30TG (142-148 bpm) in the current study, indicating that when designing a SIT programme, the length of recovery would be a key factor in determining cardiorespiratory load and thus aerobic adaptations.[9]

In contrast to $\dot{V}O_{2peak}$, time to exhaustion showed a trend to be increased until the end of the study in both training groups (Figure 2c). Previously, increases in time to exhaustion were associated with improvements in mitochondrial function following 2 weeks of Wingate-based SIT[12] or 8 weeks of aerobic high-intensity interval training (HIT) at ~90% $\dot{V}O_{2max}$.[23] In addition, faster $\dot{V}O_2$ kinetics was also associated with the improvements in TTE after 8 weeks of HIT[23] or 2 weeks of Wingate-based SIT.[11] Accelerated $\dot{V}O_2$ kinetics may delay the onset of depletion of muscle high-energy phosphates (eg, PCr) and accumulation of fatigue-related metabolites (eg, $H^+$, $P_i$),[24,25] which likely results in an enhanced exercise tolerance.[11] Moreover, Daussin et al.[23] also demonstrated an increased TTE in their continuous training group (20-35 minutes cycling at ~61% $\dot{V}O_{2max}$) in

the absence of mitochondrial adaptation but due to greater improvements in capillary density and vascular conductance compared with the HIT group. This indicates that improved muscle perfusion and thus $O_2$ supply in addition to enhanced muscle $O_2$ uptake may increase tolerable exercise duration.[23] Following 4 weeks of high-intensity training consisting of repeated one-legged knee extensor exercise at 150% of leg $\dot{V}O_{2max}$, there was an increased capillary density in both type I and II muscle fibers.[26] Therefore, it is possible that improvements in capillary density following SIT as well as mitochondrial adaptations are regulating the improvement in time to exhaustion; however, as muscle biopsies were not obtained in this study, this remains to be elucidated. Taken together, it seems that peripheral adaptations mainly account for improvements in exercise tolerance with SIT and increasing sprint number may play a role in continuous improvements in tolerable exercise duration.

## 4.2 | Effects of sprint duration on endurance and repeated sprint performance adaptations

This study sought to determine whether the length of sprint would affect overall training adaptations, and the findings of the current study reinforce previous work that also showed no difference in performance adaptations such as 5-km cycling time trial or critical power when sprint duration was reduced from 30 to 10 seconds[7] or 15 seconds.[8] In the current study, similar magnitude of improvement was seen in the two training groups in 10-km time trial (15TG vs 30TG: 8.6% vs 7.2%) and critical power (15TG vs 30TG: 7.8% vs 7.4%) (Table 2). Although changes in critical power did not reach a statistical significance in 30TG, the magnitude of gain was higher than that (5.2%) seen in 30-second Wingate-based SIT group in the study by Zelt et al.[8] While eight of nine participants increased CP following 9 weeks in 15TG, two of eight participants decreased it in 30TG. Therefore, interindividual variability may have precluded a statistical significance. A high level of power production during all-out sprinting would require an increased level of muscle fiber recruitment[27] and rapid training grains derived from sprint interval training have been linked to the high degree of stress to working muscles, in particular to type II muscle fibers.[7,28,29] In this study, sprint:rest ratio was matched between the training groups (1:8) as opposed to the study by Hazell et al.[7] or Zelt et al.,[8] and as a result, there was no difference in the reproducibility of power production during the training between the groups (Figure 3c, d). Moreover, although sprint duration was reduced by 50% in 15TG, training volume (total work) was only reduced to ~67% of that obtained in 30TG (Figure 3a, b). This suggests that the majority of work achieved in a 30-second sprint is produced during the initial phases as previously reported,[3,19] which may negate the need for performing a prolonged sprint. The choice of sprint duration (15 seconds) was made based on the previous studies showing that the majority of anaerobic metabolism (ie, the degradation of phosphocreatine and glycogen) occurs within the first 15 seconds when performing a maximal sprint.[3–5] Similar level of blood lactate accumulation was recorded between the groups (Table 3), which would support a similar anaerobic demand of both sprint protocols. Moreover, while heart rate during sprints tended to be increased in 30TG compared with 15TG, HR during recovery showed an opposite trend (Figure 1), indicating that when sprint:rest ratio is matched, overall aerobic demand is not affected by sprint duration.

Improvements in repeated sprint performance during the training intervention were only seen in 15TG (Figure 3a). 30TG did not improve any of the sprint performance measures over the four sprints throughout the study period, which is not in line with previous Wingate-based studies that report improvements in performance during a single or repeated 30-second Wingate tests.[7,10,12] However, Whyte et al.[30] did not observe a gain in peak power during a single Wingate test following 2 weeks of Wingate-based SIT, and total work or mean power during repeated Wingate tests was not increased with 4 weeks[14] or 2 weeks[12] of Wingate-based SIT. Prolonged sprint duration (≥30 seconds) may have less impact on improvements in anaerobic metabolism and thus sprint performance compared with a shorter sprint protocol (≤15 seconds).[31–33] Nevertheless, in the current study, it seems that similar aerobic and anaerobic metabolic demands were achieved between the training groups as reflected by session-averaged HR and blood lactate accumulation during the training. Therefore, it is unlikely that there was a significant difference in metabolic or morphological adaptations between the groups.[34] Prior knowledge of sprint number or prolonged sprint duration has been shown to induce anticipatory pacing strategy, resulting in reduced neuromuscular activity and power generation.[35–37] Sprint performance was assessed during the training intervention and the number of sprint was increased every 3 weeks (every six sessions) in the current study and the lack of improvement seen in 30TG might be explained by the adoption of pacing strategy due to longer sprint duration as well as increased sprint repetitions.

In short, this study demonstrated for the first time that when sprint-to-rest ratio is fixed (1:8), the length of sprint does not affect training intensity (ie, reproducibility of power) or aerobic and anaerobic demands, resulting in comparable training adaptations irrespective of sprint duration in our moderately-trained participants. Furthermore, there are divergent effects of sprint interval training on the time course of physiological and performance adaptations over 9 weeks with improvements in $\dot{V}O_{2peak}$ being completed within 3 weeks, whereas exercise capacity (time to exhaustion) being increased throughout 9 weeks.

## 5 | PERSPECTIVES

Physiological and performance improvements induced by Wingate-based SIT have been shown to be comparable to those derived from traditional endurance training lasting more than 60 minutes.[1,2] The present study further demonstrated that only 50% of total time commitment is required to gain similar or even greater training benefits compared with typical Wingate-based SIT. In other words, the findings from the current study indicate that individuals (albeit not well-trained) can improve their cardiorespiratory function and endurance performance by performing SIT requiring only 7-11.5 minutes in total (including recovery periods). Nevertheless, while both training groups increased time to exhaustion toward the end of the study, the improvements in $\dot{V}O_{2peak}$ plateaued following 3 weeks despite the increase in sprint number, suggesting that training stimulus needs to be altered through a different strategy to see continuous cardiorespiratory improvements. In this study, recovery intensity (40% $\dot{V}O_{2peak}$) was kept constant throughout the study period; however, gradual increase in recovery intensity or reduction in recovery duration may be required to ensure a progressive cardiorespiratory overload.[9]

## REFERENCES


1. Burgomaster KA, Howarth KR, Phillips SM, et al. Similar metabolic adaptations during exercise after low volume sprint interval and traditional endurance training in humans. *J Physiol*. 2008;586:151–160.
2. Gibala MJ, Little JP, van Essen M, et al. Short-term sprint interval versus traditional endurance training: similar initial adaptations in human skeletal muscle and exercise performance. *J Physiol*. 2006;575:901–911.
3. Bogdanis GC, Nevill ME, Boobis LH, Lakomy HK. Contribution of phosphocreatine and aerobic metabolism to energy supply during repeated sprint exercise. *J Appl Physiol (1985)*. 1996a;80:876–884.
4. Bogdanis GC, Nevill ME, Lakomy HK, Boobis LH. Power output and muscle metabolism during and following recovery from 10 and 20 s of maximal sprint exercise in humans. *Acta Physiol Scand*. 1998;163:261–272.
5. Parolin ML, Chesley A, Matsos MP, Spriet LL, Jones NL, Heigenhauser GJ. Regulation of skeletal muscle glycogen phosphorylase and PDH during maximal intermittent exercise. *Am J Physiol*. 1999;277:E890–E900.
6. Gaitanos GC, Williams C, Boobis LH, Brooks S. Human muscle metabolism during intermittent maximal exercise. *J Appl Physiol (1985)*. 1993;75:712–719.
7. Hazell TJ, Macpherson RE, Gravelle BM, Lemon PW. 10 or 30-s sprint interval training bouts enhance both aerobic and anaerobic performance. *Eur J Appl Physiol*. 2010;110:153–160.
8. Zelt JG, Hankinson PB, Foster WS, et al. Reducing the volume of sprint interval training does not diminish maximal and submaximal performance gains in healthy men. *Eur J Appl Physiol*. 2014;114:2427–2436.
9. Kavaliauskas M, Aspe RR, Babraj J. High-intensity cycling training: the effect of work-to-rest intervals on running performance measures. *J Strength Cond Res*. 2015;29:2229–2236.
10. Astorino TA, Allen RP, Roberson DW, Jurancich M. Effect of high-intensity interval training on cardiovascular function, VO2max, and muscular force. *J Strength Cond Res*. 2012;26:138–145.
11. Bailey SJ, Wilkerson DP, Dimenna FJ, Jones AM. Influence of repeated sprint training on pulmonary O2 uptake and muscle deoxygenation kinetics in humans. *J Appl Physiol (1985)*. 2009;106:1875–1887.
12. Burgomaster KA, Hughes SC, Heigenhauser GJ, Bradwell SN, Gibala MJ. Six sessions of sprint interval training increases muscle oxidative potential and cycle endurance capacity in humans. *J Appl Physiol*. 2005;98:1985–1990.
13. Macpherson RE, Hazell TJ, Olver TD, Paterson DH, Lemon PW. Run sprint interval training improves aerobic performance but not maximal cardiac output. *Med Sci Sports Exerc*. 2011;43:115–122.
14. Trilk JL, Singhal A, Bigelman KA, Cureton KJ. Effect of sprint interval training on circulatory function during exercise in sedentary, overweight/obese women. *Eur J Appl Physiol*. 2011;111:1591–1597.
15. Gillen JB, Martin BJ, MacInnis MJ, Skelly LE, Tarnopolsky MA, Gibala MJ. Twelve weeks of sprint interval training improves indices of cardiometabolic health similar to traditional endurance training despite a five-fold lower exercise volume and time commitment. *PLoS ONE*. 2016;11:e0154075.
16. Lavie CJ, Milani RV, Mehra MR. Peak exercise oxygen pulse and prognosis in chronic heart failure. *Am J Cardiol*. 2004;93:588–593.
17. Oliveira RB, Myers J, Araújo CG. Long-term stability of the oxygen pulse curve during maximal exercise. *Clinics (Sao Paulo)*. 2011;66:203–209.
18. Bergstrom HC, Housh TJ, Zuniga JM, et al. A new single work bout test to estimate critical power and anaerobic work capacity. *J Strength Cond Res*. 2012;26:656–663.
19. Bogdanis GC, Nevill ME, Lakomy HK, Graham CM, Louis G. Effects of active recovery on power output during repeated maximal sprint cycling. *Eur J Appl Physiol Occup Physiol*. 1996b;74:461–469.
20. Morris SB, DeShon RP. Combining effect size estimates in meta-analysis with repeated measures and independent-groups designs. *Psychol Methods*. 2002;7:105–125.
21. Cohen J. A power primer. *Psychol Bull*. 1992;112:155–159.
22. Matsuo T, Saotome K, Seino S, et al. Effects of a low-volume aerobic-type interval exercise on VO2max and cardiac mass. *Med Sci Sports Exerc*. 2014;46:42–50.
23. Daussin FN, Zoll J, Dufour SP, et al. Effect of interval versus continuous training on cardiorespiratory and mitochondrial functions: relationship to aerobic performance improvements in sedentary subjects. *Am J Physiol Regul Integr Comp Physiol*. 2008;295:R264–R272.
24. Jones AM, Wilkerson DP, DiMenna F, Fulford J, Poole DC. Muscle metabolic responses to exercise above and below the "critical power" assessed using 31P-MRS. *Am J Physiol Regul Integr Comp Physiol*. 2008;294:R585–R593.
25. Vanhatalo A, Fulford J, DiMenna FJ, Jones AM. Influence of hyperoxia on muscle metabolic responses and the power-duration relationship during severe-intensity exercise in humans: a 31P magnetic resonance spectroscopy study. *Exp Physiol*. 2010;95:528–540.

26. Jensen L, Bangsbo J, Hellsten Y. Effect of high intensity training on capillarization and presence of angiogenic factors in human skeletal muscle. *J Physiol*. 2004;557:571–582.
27. Esbjörnsson-Liljedahl M, Bodin K, Jansson E. Smaller muscle ATP reduction in women than in men by repeated bouts of sprint exercise. *J Appl Physiol (1985)*. 2002;93:1075–1083.
28. Buchheit M, Abbiss CR, Peiffer JJ, Laursen PB. Performance and physiological responses during a sprint interval training session: relationships with muscle oxygenation and pulmonary oxygen uptake kinetics. *Eur J Appl Physiol*. 2012;112:767–779.
29. Sloth M, Sloth D, Overgaard K, Dalgas U. Effects of sprint interval training on VO2max and aerobic exercise performance: a systematic review and meta-analysis. *Scand J Med Sci Sports*. 2013;23:e341–e352.
30. Whyte LJ, Gill JM, Cathcart AJ. Effect of 2 weeks of sprint interval training on health-related outcomes in sedentary overweight/obese men. *Metabolism*. 2010;59:1421–1428.
31. Dawson B, Fitzsimons M, Green S, Goodman C, Carey M, Cole K. Changes in performance, muscle metabolites, enzymes and fibre types after short sprint training. *Eur J Appl Physiol Occup Physiol*. 1998;78:163–169.
32. Linossier MT, Dormois D, Perier C, Frey J, Geyssant A, Denis C. Enzyme adaptations of human skeletal muscle during bicycle short-sprint training and detraining. *Acta Physiol Scand*. 1997;161:439–445.
33. Parra J, Cadefau JA, Rodas G, Amigó N, Cussó R. The distribution of rest periods affects performance and adaptations of energy metabolism induced by high-intensity training in human muscle. *Acta Physiol Scand*. 2000;169:157–165.
34. Ross A, Leveritt M. Long-term metabolic and skeletal muscle adaptations to short-sprint training: implications for sprint training and tapering. *Sports Med*. 2001;31:1063–1082.
35. Ansley L, Robson PJ, St Clair Gibson A, Noakes TD. Anticipatory pacing strategies during supramaximal exercise lasting longer than 30 s. *Med Sci Sports Exerc*. 2004;36:309–314.
36. Billaut F, Bishop DJ, Schaerz S, Noakes TD. Influence of knowledge of sprint number on pacing during repeated-sprint exercise. *Med Sci Sports Exerc*. 2011;43:665–672.
37. St Clair Gibson A, Schabort EJ, Noakes TD. Reduced neuromuscular activity and force generation during prolonged cycling. *Am J Physiol Regul Integr Comp Physiol*. 2001;281:R187–R196.


## SUPPORTING INFORMATION

Additional Supporting Information may be found online in the supporting information tab for this article.

**How to cite this article:** Yamagishi T, and Babraj J. Effects of reduced-volume of sprint interval training and the time course of physiological and performance adaptations. *Scand J Med Sci Sports*. 2017;27:1662–1672. https://doi.org/10.1111/sms.12831