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

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Running head: Sprint duration and training adaptations

Abstract

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-s training group (15TG) consisting of 4 to 6 x 15-s sprints interspersed with 2-min recovery, whereas eight participants (M: 5; F: 3) were assigned to 30-s training group (30TG) consisting of 4 to 6 30-s sprints interspersed with 4-min 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 8 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%, $P < 0.05$) and TTE (15TG: 16.2%; 30TG: 12.8%, $P < 0.01$) 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%, $P < 0.001$), those in TTE were greatest at 9 weeks. CON did not change any of performance variables following 9 weeks. This study demonstrated that whilst 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: sprint-to-rest ratio, peak oxygen uptake, endurance capacity, time course of training adaptations

Introduction

It has been demonstrated that Wingate-based sprint interval training (SIT) consisting of 4 to 6 30-s sprints interspersed with 4 min of recovery promotes comparable metabolic and physiological adaptations (e.g. an improved mitochondrial function) to those obtained from traditional aerobic exercise training (e.g. 60 to 90 min of continuous cycling at 65% $\dot{V}O_{2max}$) despite its low training volume (2 to 3 min of all-out efforts) (Gibala et al., 2006; Burgomaster et al., 2008). Nevertheless, the conventional 30-s Wingate-based SIT may not be necessarily time-efficient if warm-up and recovery periods are included, amounting to ~30 minutes (Gibala et al., 2006; Burgomaster et al., 2008). It has been shown that the majority of anaerobic metabolism (i.e. the degradation of phosphocreatine and glycogen) occurs within the first 15 s during a 30-s maximal sprint (Bogdanis et al., 1996a, 1998; Parolin et al., 1999), and that aerobic and anaerobic metabolism increases and decreases, respectively, with successive bouts irrespective of sprint duration (6 to 30 s) when sprints are separated by 30-s to 4-min recovery (Gaitanos et al., 1993; Bogdanis et al., 1996a, 1998; Parolin et al., 1999). Therefore, shorter sprint protocol ($\leq$ 15 s) may 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. (2010) demonstrated that a reduced-volume of sprint interval training consisting of 4 to 6 10-s cycling sprints separated by 2- or 4-min recovery was as effective as typical 30-s Wingate-based SIT in improving aerobic and anaerobic performance. Similarly, Zelt et al. (2014) recently demonstrated that 4 to 6 15-s sprints interspersed with 4.75 min of recovery improved aerobic capacities such as $\dot{V}O_{2peak}$ and critical power to a similar extent compared to the conventional Wingate-based SIT. Hazell et al. (2010) found that training intensity (the reproducibility of power during training) was increased in the 10-s training groups compared to the 30-s 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 (10:120s, 10:240s, 30:240s) 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-s Wingate test) (Kavaliauskas et al., 2015). 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 utilisation of Wingate-based SIT to promote physiological and performance adaptations (Burgomaster et al., 2005; Gibala et al., 2006; Bailey et al., 2009; Macpherson et al., 2011; Trilk et al., 2011; Astorino et al., 2012), little is known regarding the time course of those adaptations. Whilst Burgomaster et al. (2008) 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. (2016) 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 x 20-s all-out cycle sprints (with 2-min 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 (i.e. 30s vs. 20s) might have had a different impact on the time course of training adaptations.

In short, the purposes of the current study were twofold, that is, to determine 1) effects of sprint duration (15 s vs. 30 s) on physiological and performance adaptations while matching sprint-to-rest ratio between training protocols (sprint: rest ratio of 1:8) and 2) time course of those adaptations over 9 weeks (baseline, weeks 3, 6 and 9). It was hypothesised 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.

Materials and methods

Participants

27 healthy active adults (male: 20, female: 7) who took part in a minimum of 3-h exercise per week initially participated in this study (Table 1). However, 1 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. 2 out of 7 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.

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 h prior to each session. Participants performed three baseline measurements on three different occasions, separated by 48h, 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-s training group consisting of 4 to 6 15-s cycle sprints interspersed with 2-min active recovery at 40% of $\dot{V}O_{2peak}$ (15TG) (N = 9; male: 7, female: 2) or 30-s training group consisting of 4 to 6 30-s cycle sprints interspersed with 4-min 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 (i.e. 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 (± 2h) in a controlled environment throughout the study period and each session was separated by at least a period of 48h.

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-h 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, 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, Varberg, Sweden). Prior to starting the test, participants were connected to a breath by breath gas analyser (Metalyzer®3B gas analyser, Cortex, Leipzig, Germany) and had a heart rate monitor attached (Polar Electro, Kempele, Finland). The oxygen (O_2) and carbon dioxide (CO_2) analyser 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 analyser was calibrated using a 3-L calibration syringe (Hans Rudolph, inc., Kansas city, USA). The test commenced at an initial power output of 70W, with an additional 35W 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 30s with $\dot{V}O_{2peak}$ calculated as the highest oxygen consumed over a 30-s period. Similarly, maximal heart rate (HR max) was defined as the highest heart rate recorded over a 30-s 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, since it has been shown that a better estimation of stroke volume is achieved when oxygen pulse is corrected for bodyweight (Lavie et al., 2004; Oliveira et al., 2011), 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. (2011).

3-min all-out cycling test

On the second visit, they performed a 3-min all-out cycling test to determine their critical power. They first completed a 3-min warm-up against 60W on a cycle ergometer (Monark Ergomedic 894E, Varberg, Sweden). The test then began when the participants reached 110 rpm where resistance was applied (4.5% of bodyweight). They pedalled 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 Exercise AB) and average power output over the final 30 sec 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-min all-out cycling test on an electronically braked cycle ergometer (Bergstrom et al., 2012).

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: 2kg; female: 1.5kg). They first completed a 3-min warm-up against 60W on a cycle ergometer (Monark Ergomedic 894E, 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.

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 (i.e. 4 sets for the initial 3 weeks, 5 sets for the second 3 weeks and 6 sets for the last 3 weeks). All participants

completed a 3-min warm-up against 60W on a cycle ergometer before performing all-out sprints. The recovery intensity (i.e. 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 4 sprints was calculated to determine the difference in training volume between the groups (i.e. 15TG vs. 30TG) in addition to peak power over the 4 sprints. Furthermore, to assess the reproducibility of power during the training, power drop rate across the 4 sprints was also calculated using the following formula;

Reproducibility of power: ((PO 1 + PO 2 + PO 3 + PO 4) / 4) / best PO x 100, where PO is power output (either peak or average) (Hazell et al., 2010).

Peak and average power were calculated automatically using the Monark software (Monark Anaerobic Test Software version 2.24.2, Monark Exercise AB), and total work was determined by integrating power output recorded every second. Total work, peak power and the reproducibility of power were assessed every 6 sessions over 9 weeks (4 times in total).

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 (Bogdanis et al., 1996a, Bogdanis et al., 1996b; Parolin et al., 1999). Blood lactate concentration was measured at 6 different time points, that is, pre-sprint, 0, 3, 5, 8 and 10 min 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^{TM} 2,

Zephyr Technology, MD, USA) in the first, 7th and 13th training sessions and average values over 4, 5 and 6 sprints were determined, respectively. Representative example of HR response during the first session in each training protocol is shown in Figure 1 (Fig.1a and 1b). In addition, HR was normalised to percentage of total time using a cubic spline method to directly compare heart rate responses between the two training protocols (Fig. 1c).

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.

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 analysed 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 analysed 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 (2002); $d = M_{diff} / SD_{pooled}\sqrt{2}(1 - r)$, where M_{diff} is mean difference between conditions, SD_{pooled} is pooled standard deviation, and r is correlation between means. Cohen's 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 (Cohen, 1992). All statistics were run on IBM® SPSS® Version 22.0 for Windows and the level of significance was set at $P < 0.05$.

Results

Blood pressure and anthropometric measures

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

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%, $d = 1.77$, $P < 0.001$; 30TG: 12.8%, $d = 1.27$, $P < 0.05$, Table 2), O_2 pulse (15TG: 10.5%, $d = 1.64$, $P < 0.01$; 30TG: 10.8%, $d = 1.05$, $P < 0.05$, Table 2), time to exhaustion (15TG: 16.2%, $d = 2.17$, $P < 0.001$; 30TG: 12.8%, $d = 1.70$, $P < 0.01$, Table 2) and10-km cycling time-trial performance (15TG: 8.6%, $d = 3.47$, $P < 0.01$; 30TG: 7.2%, $d = 0.86$, $P < 0.05$, Table 2), while only 15TG significantly increased critical power (7.8%, $d = 0.87$, $P < 0.05$, Table 2). Although 30TG also increased critical power to a similar extent (7.4%, $d = 0.67$), it did not reach a statistical significance ($P = 0.11$, 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).

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 (6 vs. 5 sprints, $P < 0.05$, $d = 0.98$, Table 3). Both training groups similarly increased blood lactate accumulation following four 15-s or 30-s sprints with the peak values observed immediately after sprint 4 (pre-sprint vs. peak post-sprint value; 15TG: $P < 0.001$, $d = 8.28$, 30TG: $P < 0.001$, $d = 14.7$, Table 3). Blood lactate concentration gradually decreased with time during the 10-min recovery phase in both groups (Table 3).

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 in both groups (15TG: 49.2 ± 5.4 $ml \cdot min^{-1} \cdot kg^{-1}$, 30TG: 47.5 ± 10.3 $ml \cdot min^{-1} \cdot kg^{-1}$, Fig.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 $ml \cdot beat^{-1} \cdot kg^{-1}$, Fig.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 sec, 30TG: 1076 ± 283 sec, Fig.2c).

Time course of changes in repeated sprint performance over 9 weeks

30TG did not improve any assessment of repeated sprint performance over 9 weeks (Fig.3b), whereas 15TG significantly increased peak power and total work over the first 4 sprints during the sessions 6, 12 and 18 compared with the first session (Fig.3a). Percent of changes from the session 1 in 15TG were 7.0% ($d = 2.12$, $P < 0.001$), 7.4% ($d = 1.60$, $P < 0.01$) and 7.9% ($d = 1.08$, $P < 0.05$) in peak power, and 4.6% ($d = 0.78$, $P < 0.05$), 5.4% ($d = 0.86$, $P < 0.05$) and 5.4% ($d = 0.77$, $P < 0.05$) in total work for the sessions 6, 12 and 18, respectively (Fig.3a). Total work was greater in 30TG compared to 15TG (15TG vs. 30TG: 38.6 ± 11.2

vs. 57.8 ± 17.6 kJ, $P < 0.05$, $d = 1.33$, Fig.3a & 3b), whereas there was no difference between the groups in peak power or the reproducibility of peak and average power (Fig.3a-d).

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-s and 30-s 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 (Fig. 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.

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, whilst Burgomaster et al. (2008) saw a 7.3% improvement in $\dot{V}O_{2peak}$ following 3 weeks of Wingate-based SIT, it 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. (2012) 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. (2012), Matsuo et al. (2014) 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 x 30-s cycling at 120%$\dot{V}O_{2max}$ separated by 15-s rests. Moreover, Matsuo et al. (2014) 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. (2016) have recently demonstrated that SIT consisting of 3 x 20-s all-out cycle sprints separated by 2 min 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. (2016) 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. (2008) (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. (2014) 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. (2014) saw greater session-averaged HR in their SIT group (161 beats·min^{-1}) compared to 15TG (141 to 144 bpm) or 30TG (142 to 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 (Kavaliauskas et al., 2015).

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 (Fig. 2c). Previously, increases in time to exhaustion were associated with improvements in mitochondrial function following 2 weeks of Wingate-based SIT (Burgomaster et al., 2005) or 8 weeks of aerobic high-intensity interval training (HIT) at

~ 90% $\dot{V}O_{2max}$ (Daussin et al., 2008). In addition, faster $\dot{V}O_2$ kinetics was also associated with the improvements in TTE after 8 weeks of HIT (Daussin et al., 2008) or 2 weeks of Wingate-based SIT (Bailey et al., 2009). Accelerated $\dot{V}O_2$ kinetics may delay the onset of depletion of muscle high-energy phosphates (e.g. PCr) and accumulation of fatigue related metabolites (e.g. H^+, P_i) (Jones et al., 2008; Vanhatalo et al., 2010), which likely results in an enhanced exercise tolerance (Bailey et al., 2009).Moreover, Daussin et al. (2008) also demonstrated an increased TTE in their continuous training group (20 to 35 min 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 to 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 (Daussin et al., 2008). 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 fibres (Jensen et al., 2004). 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, since 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.

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 sec (Hazell et al., 2010) or 15 sec (Zelt et al.,

2014). 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-s Wingate-based SIT group in the study by Zelt et al. (2014). Whilst eight of nine participants increased CP following 9 weeks in 15TG, two of eight participants decreased it in 30TG. Therefore, inter-individual variability may have precluded a statistical significance. A high level of power production during all-out sprinting would require an increased level of muscle fibre recruitment (Esbjornsson-Liljedahl et al., 2002) and rapid training grains derived from sprint interval training has been linked to the high degree of stress to working muscles, in particular to type II muscle fibres (Hazell et al., 2010; Buchheit et al., 2012; Sloth et al., 2013). In this study, sprint: rest ratio was matched between the training groups (1:8) as opposed to the study by Hazell et al. (2010) or Zelt et al. (2014) and as a result, there was no difference in the reproducibility of power production during the training between the groups (Fig. 3c & 3d). Moreover, although sprint duration was reduced by 50% in 15TG, training volume (total work) was only reduced to ~ 67% of that obtained in 30TG (Fig. 3a & 3b). This suggests that the majority of work achieved in a 30-s sprint is produced during the initial phases as previously reported (Bogdanis et al., 1996a, 1996b), which may negate the need for performing a prolonged sprint. The choice of sprint duration (15 s) was made based on the previous studies showing that the majority of anaerobic metabolism (i.e. the degradation of phosphocreatine and glycogen) occurs within the first 15 s when performing a maximal sprint (Bogdanis et al., 1996a, 1998; Parolin et al., 1999). 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, whilst heart rate during sprints tended to be increased in 30TG compared to 15TG, HR during recovery showed an opposite trend

(Fig. 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 (Fig. 4a). 30TG did not improve any of the sprint performance measures over the 4 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-s Wingate tests (Burgomaster et al., 2005; Hazell et al., 2010; Astorino et al., 2012). However, Whyte et al. (2010) 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 (Trilk et al., 2011) or 2 weeks (Burgomaster et al., 2005) of Wingate-based SIT. Prolonged sprint duration (≥ 30 sec) may have less impact on improvements in anaerobic metabolism and thus sprint performance compared to a shorter sprint protocol (≤ 15 sec) (Linossier et al., 1997; Dawson et al., 1998; Parra et al., 2000). 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 (Ross & Leveritt, 2001). 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 (St Clair Gibson et al., 2001; Ansley et al., 2004; Billaut et al., 2011). Sprint performance was assessed during the training intervention and the number of sprint was increased every 3 weeks (every 6 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 (i.e. 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.

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 min (Gibala et al., 2006; Burgomaster et al., 2008). The present study further demonstrated that only 50% of total time commitment is required to gain similar or even greater training benefits compared to 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 to 11.5 minutes in total (including recovery periods). Nevertheless, while both training groups increased time to exhaustion towards 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 (Kavaliauskas et al., 2015).

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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 (years)	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

Values are means ± SD.

SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; PP, pulse pressure.

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 (sec)*** ###	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 (sec)***	977 ± 160	893 ± 112‡‡	969 ± 82	899 ± 89‡	912 ± 103	902 ± 112

Values are means ± SD. $\dot{V}O_{2peak}$, peak oxygen uptake; HR max, maximal heart rate.

***Indicates $P < 0.001$ for main effect of time. **Indicates $P < 0.01$ for main effect of time. *Indicates $P < 0.05$ for main effect of time. ###Indicates $P < 0.001$ for time by group interaction effect. ##Indicates $P < 0.01$ for time by group interaction effect. ‡‡‡Indicates $P < 0.001$ vs. pre within the same group. ‡‡Indicates $P < 0.01$ vs. pre within the same group. ‡Indicates $P < 0.05$ vs. pre within the same group.

Note: A 2-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.

Table 3. Heart rate and blood lactate responses during the training

Physiological parameters	Training group	
Session-averaged HR (beats·min^{-1})*	15TG	30TG
4 sprints and 3 rest periods	141 ± 8	142 ± 12
5 sprints and 4 rest periods	144 ± 8	145 ± 12
6 sprints and 5 rest periods	144 ±8	148 ± 13‡
Blood lactate (mmol·l^{-1})***	15TG	30TG
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.

*Indicates $P < 0.05$ for main effect of sprint number. ***Indicates $P < 0.001$ for main effect of time. ‡Indicates $P < 0.05$ vs. 4 sprints and 3 rest periods within the same group.[aaa] Indicates $P < 0.001$ vs. pre-sprint within the same group. [bb] Indicates $P < 0.01$ vs. 10-min post within the same group. [b] Indicates $P < 0.05$ vs. 10-min post within the same group. [c] Indicates $P < 0.05$ vs. 5-min post within the same group

Note: A 2-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 1-way ANOVA with LSD post-hoc test.

Supplementary Table 1. Status of physical activity in each group

	15TG (n = 9)	30TG (n = 8)	CON (n = 8)
Jogging	2	1	2
Cycling	1	2	1
Resistance exercise	3	1	2
Football	1	0	3
Rugby	0	1	0
Boxing	2	1	0
Hockey	0	1	0
Netball	0	1	0

Numbers in the table indicate number of participants engaging in particular sporting or physical activity during the study period.

Figures

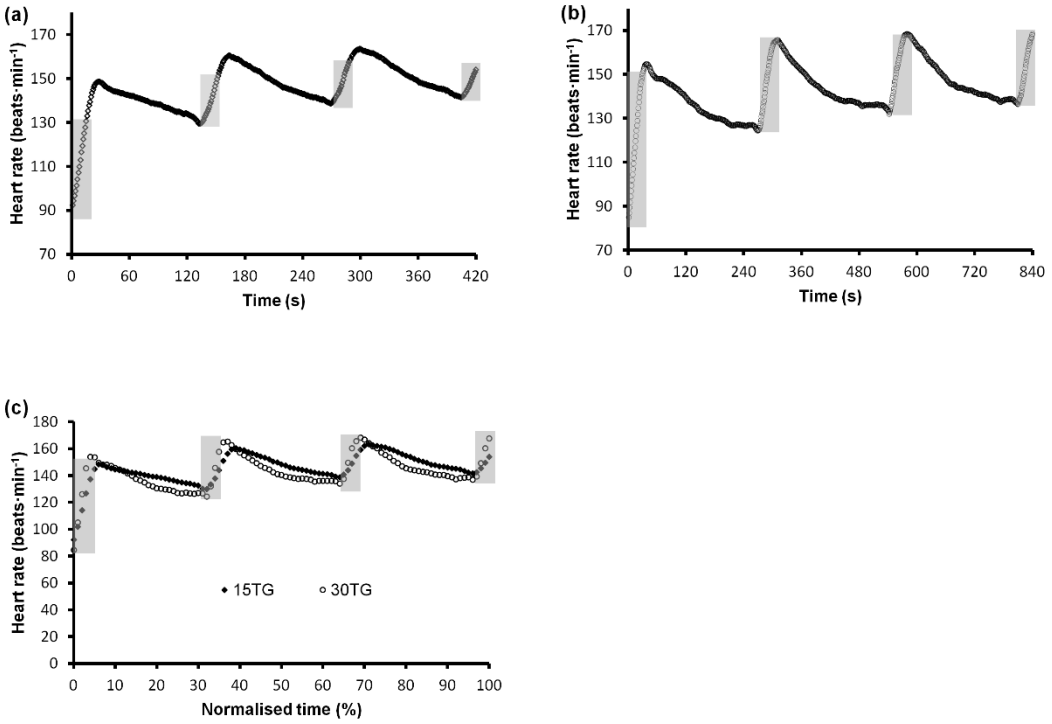

Fig. 1. Representative example of heart rate over 4 sprints and 3 rest periods (group mean) in 15TG (a), 30TG (b) and both groups (C). In Fig. 1a and 1b, HR with actual running time is shown whereas HR is normalised to percentage of total time in Fig.1c. Shaded areas indicate HR during sprints. Error bars are not shown for clarity.

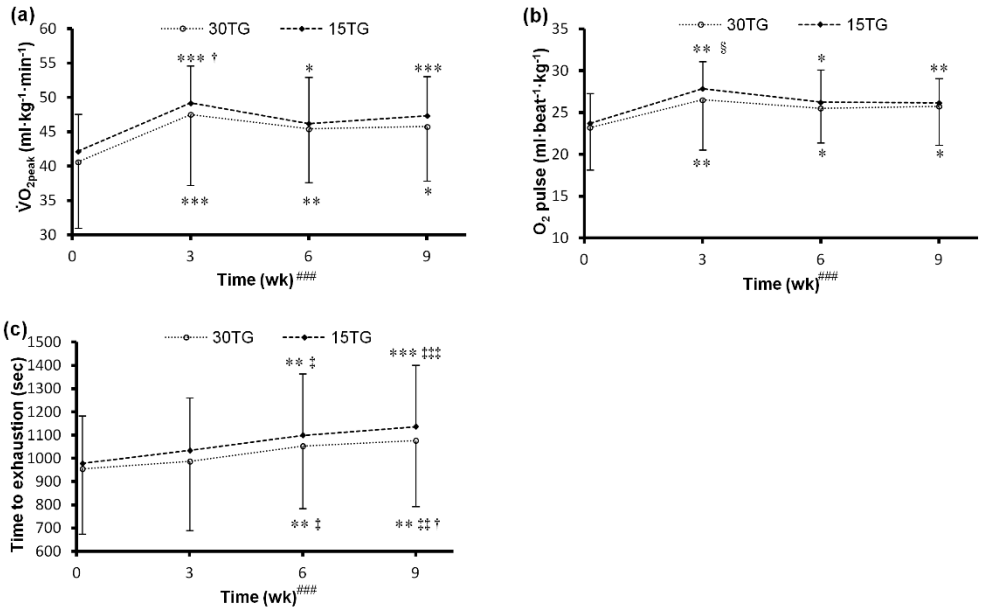

Fig. 2. Time course of changes in peak oxygen uptake (a), O_2 pulse (b) and time to exhaustion (c). ###Indicates $P < 0.001$ for main effect of time. ***Indicates $P < 0.001$ vs. baseline within the same group. **Indicates $P < 0.01$ vs. baseline within the same group. *Indicates $P < 0.05$ vs. pre within the same group. ‡‡‡Indicates $P < 0.001$ vs. week 3 within the same group. ‡‡Indicates $P < 0.01$ vs. week 3 within the same group. ‡Indicates $P < 0.05$ vs. week 3 within the same group. †Indicates $P < 0.05$ vs. week 6 within the same group. §Indicates $P < 0.05$ vs. week 9 within the same group.

Note: A 2-way mixed ANOVA showed no interaction effect or main difference between groups in all variables. A 1-way ANOVA with LSD post-hoc test was performed to determine changes in the variables over time for each group.

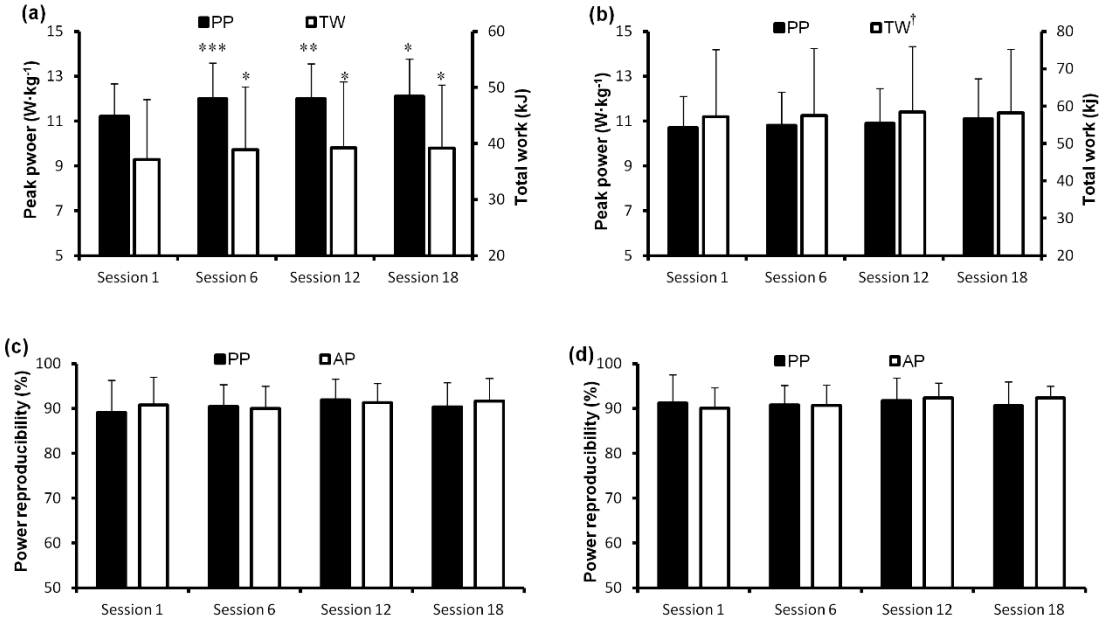

Fig. 3. Peak power and total work in 15TG (a) and 30TG (b) and the reproducibility of power in 15TG (c) and 30TG (d) during the first 4 sprints in the 1st, 6th, 12th and 18th training sessions. †Indicates that total work is greater than 15TG ($P < 0.05$). ***Indicates $P < 0.01$ vs. session 1 within the same group. **Indicates $P < 0.01$ vs. session 1 within the same group. *Indicates $P < 0.05$ vs. session 1 within the same group.

Note: A 2-way mixed ANOVA showed a main difference in total work between the groups, whereas a 1-way ANOVA with LSD post-hoc test demonstrated the improvements in sprint performance in 15TG.