The acute physiological and perceptual effects of individualising the recovery interval duration based upon the resolution of muscle oxygen consumption during cycling exercise

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<th>Journal:</th>
<th>International Journal of Sports Physiology and Performance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Manuscript ID</td>
<td>IJSPP.2020-0295.R2</td>
</tr>
<tr>
<td>Manuscript Type:</td>
<td>Original Investigation</td>
</tr>
<tr>
<td>Date Submitted by the Author:</td>
<td>n/a</td>
</tr>
<tr>
<td>Complete List of Authors:</td>
<td>Fennell, Christopher; University of Kent, School of Sport and Exercise Sciences Hopker, James; University of Kent, Centre for Sport Studies</td>
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<tr>
<td>Keywords:</td>
<td>recovery interval duration; high intensity interval training; near-infrared spectroscopy; muscle oxygen consumption.</td>
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ABSTRACT

Purpose. There has been paucity in research investigating the individualisation of recovery interval duration during cycling based high intensity interval training (HIIT). The main aim of the study was to investigate whether individualising the duration of the recovery interval based upon the resolution of muscle oxygen consumption (mVO$_2$), would improve the performance during work intervals and the acute physiological response of the HIIT session, when compared to a standardised (STD; 2:1 work:recovery ratio) approach. Methods. Sixteen well-trained cyclists (VO$_{2\text{max}}$: 60 ± 7 ml.kg$^{-1}$.min$^{-1}$) completed six laboratory visits: V1) Incremental exercise test, V2) Determination of the individuals mVO$_2$ recovery duration to baseline (IND) from a 4-min and 8-min work interval, V3 - V6) Participants completed a 6 x 4-min and a 3 x 8-min HIIT session twice, using the IND and STD recovery intervals. Results. Recovery duration had no effect on the percentage of the work intervals spent at >90% and >95% of VO$_{2\text{max}}$, maximal minute power output (MMP) and HR$_{\text{max}}$, during the 6 x 4-min and 3 x 8-min HIIT sessions. Recovery duration had no effect on mean work interval PO, HR, VO$_2$, B[La] and RPE. There were no differences in reported sRPE between recovery durations for the 6 x 4-min and 3 x 8-min HIIT sessions. Conclusion. Individualising HIIT recovery duration based upon the resolution of mVO$_2$ to baseline levels, does not improve the performance of the work intervals or the acute physiological response of the HIIT session, when compared to a STD recovery duration.

Keywords. recovery interval duration; high intensity interval training; near-infrared spectroscopy; muscle oxygen consumption.
INTRODUCTION

High intensity interval training (HIIT) programming comprises of five main components: work interval intensity, work interval duration, number of work intervals, recovery interval intensity, recovery interval duration. The work interval components have received the greatest amount of research attention as they ultimately facilitate the majority of the training stimulus produced by the HIIT session. However, optimal HIIT session performance (i.e. achieving the greatest training stimulus for the specific HIIT session) can only be achieved if adequate recovery separates the work intervals. If there is an imbalance between the demands of the work interval and the recovery provided, this can lead to HIIT sessions that are too hard to complete, or HIIT sessions that are too easy.

Surprisingly, despite the importance of the recovery interval duration to HIIT session programming, there has been paucity of research investigating the effect of recovery interval duration on subsequent work interval performance. Previous researchers investigating the acute effects of recovery interval duration have predominantly used fixed recovery durations and/or work recovery ratios (i.e. 1:1 or 2:1) to prescribe recovery interval duration. While fixed durations and work recovery ratios might be the most common and practical approach to prescribing recovery interval duration, it is based upon the assumption that every individual requires the same recovery duration during HIIT sessions. On the contrary, the optimal duration is most likely highly individual, dependent on training status and desired session outcome.

Researchers have attempted to use self-selected recovery durations as a method of individualisation demonstrating the method to be effective when participants are well familiarized with the procedures and physical demands of the HIIT protocol. While self-selected recovery durations take into consideration the day-to-day variation in the individuals' environmental and/or psychological state, it does not take into account the individuals' recovery status in order to recommence exercise. If the individual’s physiological status during recovery is not considered it could lead to inadequate or excessive recovery between work intervals, potentially compromising the training session.

The use of heart rate (HR) is a physiologically based method to individualise the duration of the recovery interval. However, the method has received limited research attention, this is most likely due to the inherent limitations of using HR to prescribe recovery duration. More recently, the $W'_\text{BAL}$ model has been proposed as a method to individualise interval training. For example, when working at intensities above the Critical Power (CP) a cyclist would deplete the finite energy capacity defined by $W'$, and in recovery below CP, $W'$ would replenish over time. During intermittent exercise the balance of $W'$ remaining has been suggested to predict an athlete’s interval training capacity, accounting for both the work and recovery elements of a given training prescription. However, the robustness of $W'_\text{BAL}$ has been questioned.

Near infrared spectroscopy (NIRS), is a well-known non-invasive method used to measure muscle oxygenation, which reflects the ratio of oxygen ($O_2$) delivery to the working muscle and muscle oxygen uptake in the capillary beds. The recovery of muscle oxygen consumption (mVO$_2$) considers the condition of the exercising muscle, as measurements are derived directly from the muscle body. It has been suggested that the recovery duration of mVO$_2$ after high intensity exercise is likely related to a greater depletion of adenosine triphosphate (ATP), phosphocreatine (PCr) and/or myoglobin $O_2$ stores, which logically take longer to be restored. In addition, it is possible that mVO$_2$ remains elevated above baseline values after high intensity exercise to compensate for the detrimental effect of a decreased muscle pH on PCr recovery. Therefore, it is possible that mVO$_2$ recovery coincides with
the return of the exercising muscle to a state of metabolic homeostasis. The recovery rate of mVO\(_2\) also takes into account the intensity of the prior exercise \(20\), the individuals training status \(21,22\) and age \(23\). Based on the aforementioned evidence, current authors propose that the recovery duration of mVO\(_2\) may provide a method to individualise HIIT recovery interval duration.

The current study therefore sought to investigate whether individualising the duration of the recovery interval based on the participants’ mVO\(_2\) recovery duration to baseline (IND) would improve the performance of self-paced work intervals and the acute physiological response, when compared to a standardised recovery duration (STD; 2:1 work recovery ratio). It was hypothesised that the IND recovery duration would increase work interval power output (PO) resulting in a greater acute physiological response during the work intervals, when compared to the STD recovery duration.

**METHODS**

**Participants.** Sixteen trained cyclists with a minimum of 2 years competitive racing experience participated in the study. The study was completed with full ethical approval, according to the Declaration of Helsinki standards. All participants provided signed informed consent prior to testing.

**Study design.** Each participant completed six visits to the laboratory. Visit 1 being an incremental exercise test to identify VO\(_{2\text{max}}\) and familiarise the participants with the laboratory environment. Visit 2 was the determination of the participants IND recovery duration. In visits 3 to 6, participants performed the four HIIT sessions in a randomised order within two weeks.

Visits were conducted on non-concurrent days and participants were instructed to refrain from any exercise in the day prior to testing and intense exercise in the two days prior. Participants were instructed not consume caffeine within 4 hours and alcohol within 24 hours of testing, and to arrive euhydrated, having eaten at least 4 hours prior to testing. Participants completed all their visits at the same time of day to avoid any circadian variance.

Participants used their own bike at all visits, affixed to a Cyclus2 ergometer (Leipzig, Germany). At all visits respiratory gas exchange data were assessed using breath by breath gas analysis (Metalyzer 3B; CORTEX Biophysik GmbH, Leipzig, Germany). Prior to all testing the analyser was calibrated according to the manufacturer recommendations.

**Incremental exercise test.** The VO\(_{2\text{max}}\) test protocol started with a 10 min warm-up at 100 W, after which the required cycling PO was increased by 20 W every 1 min until volitional exhaustion. PO and HR were measured continuously throughout the test, with rating of perceived exertion (RPE) taken in the last 10 s of each 1 min stage of the test, using the Borg 6 - 20 scale \(24\). The participant’s VO\(_{2\text{max}}\) was assessed as the highest pulmonary O\(_2\) uptake that was attained during a 1 min period. Maximal minute power (MMP) and maximal minute heart rate (HR\(_{\text{max}}\)) were assessed as the highest 1 min PO and HR achieved during the test.

**Methods for the determination of mVO\(_2\) recovery duration.** NIRS data were acquisitioned at 10 Hz from the right vastus lateralis muscle (VL; approximately 8 cm from the knee joint on the vertical axis) using a continuous-wave NIRS device (Portamon, Artinis Medical Systems, The Netherlands). Skinfold thickness at the site of application of the NIRS optode was determined before the session using Harpenden skinfold callipers (British indicators Ltd,
Burgess Hill, UK). A rapid inflating blood pressure cuff (Hokanson E20 cuff inflator, SC12 cuff; Bellevue, WA) was placed around the thigh proximal to the NIRS device.

Prior to the commencement of exercise, participants adopted a standardised resting position, seated with the knee flexed at 90° for a 2 min period, during which baseline NIRS parameters were established. A 5 min ischemic calibration procedure was then performed to scale the NIRS oxyhaemoglobin (O$_2$Hb) and deoxyhaemoglobin (HHb) signals to the maximal physiological range \(^{25}\). After warming up at 100 W for 10 min the participants completed a single self-paced 4 min interval. Immediately (5 s) following the interval a series of 20 brief (i.e. 10 s) arterial occlusions were applied to measure mV$\text{O}_2$ recovery \(^{25}\). Participants were instructed to keep the leg under occlusion at the bottom of the pedal stroke, remaining completely still and to hold the same posture throughout the occlusion procedure. After cooling down at 100 W for 10 min, participants then completed a seated rest for 20 min before repeating the above protocol, this time completing a single self-paced 8 min interval.

A blood volume correction was applied to the NIRS data prior to the calculation of mV$\text{O}_2$ \(^{25}\). mV$\text{O}_2$ was calculated as the initial slope of change in corrected HHb during the arterial occlusion using simple linear regression. The linear slope of increase in corrected HHb expressed in micromolar units was converted to millilitres O$_2$ per minute per 100 g tissue (ml.O$_2$.min$^{-1}$.100 g$^{-1}$) using the following equation \(^{26}\).

\[
[1] \quad \text{mV}O_2 = \frac{((\text{HHb} \times 60) / (10 \times 1.04) \times 4) \times 22.4}{1000}
\]

Data derived from the repeated arterial occlusions were then plotted versus recovery time to show the time course of mV$\text{O}_2$ recovery after the 4 min and 8 min intervals (Figure 2A). Participant’s IND recovery duration was calculated as the time at which the mV$\text{O}_2$ recovery curve intercepts the 95% mV$\text{O}_2$ value output from equation 2 (Figure 2A). A 95% mV$\text{O}_2$ value was used to ensure a plateau in mV$\text{O}_2$ was reached, take into account differences in the rate of mV$\text{O}_2$ recovery and allow for easy replication across participants. The 95% mV$\text{O}_2$ value was calculated as 95% of the difference between the peak mV$\text{O}_2$ value and the end mV$\text{O}_2$ value (equation 2).

\[
[2] \quad \text{mV}O_2 \text{value} = (\text{mV}O_2\text{peak} - \text{mV}O_2\text{end}) - \left(\frac{(\text{mV}O_2\text{peak} - \text{mV}O_2\text{end})}{100} \times 95\right) + \text{mV}O_2\text{end}
\]

Where:

- mV$\text{O}_2$peak = first mV$\text{O}_2$ value following the first cuff inflation.
- mV$\text{O}_2$end = last mV$\text{O}_2$ value at the end of the measurement period.

HIIT sessions. Participants completed both the 6 x 4-min and 3 x 8-min HIIT sessions twice (4 HIIT sessions in total), once with the STD recovery duration and once with the IND recovery duration (Figure 3). The STD recovery durations used were 120 s and 240 s for the 6 x 4-min and 3 x 8-min HIIT sessions respectively (2:1 work:recovery ratio). The participants IND recovery durations were 205 ± 79 s and 200 ± 81 s for the 6 x 4-min and 3 x 8-min HIIT sessions respectively, as measured in visit 2.
Work intervals were prescribed as self-paced on a ‘maximal session effort’ basis, with participants instructed to achieve the highest PO possible during each interval. HIIT sessions commenced with a 10 min warm-up at 100W and finished with a 10 min cool down at 100W. All recovery intervals were passive with participants instructed to remain seated with their right leg at the bottom of the pedal stroke.

PO, HR and respiratory gases were measured continuously throughout the HIIT sessions. NIRS derived HHb and NIRS data were acquisitioned at 10 Hz measured at the VL using a continuous-wave NIRS device during the recovery intervals throughout all of all HIIT sessions. HHb data were reported as percentages of a 5 min ischemic calibration performed prior to each HIIT session. Blood lactate (B[La]) samples were taken pre warm-up and during the last 30 s of each work interval via the fingertip ( Biosen C-Line, EKF Diagnostic, London, UK).

RPE measurements were taken during the last 15 s of each work interval using the Borg 6 - 20 scale. Session RPE (sRPE) measurements using the 0 - to 10 scale were taken at the end of the 10 min cool down.

Statistical analyses. Data were presented as individual values or mean ± SD (unless specified otherwise). Statistical analyses were conducted using IBM SPSS Statistics 26 (IBM, Armonk, New York, USA). Visual inspection of Q-Q plots and Shapiro-Wilk statistics were used to check whether data were normally distributed. Three separate two-way repeated measure analysis of variance (ANOVA), 1) two HIIT protocols (6 x 4-min vs 3 x 8-min) X two recovery durations (STD vs IND); 2) two recovery durations (STD vs IND) X number of work intervals; 3) two recovery durations (STD vs IND) X number of recovery intervals; were used to determine between and within condition effects for all dependent variables. Bonferroni post hoc comparisons were used when a main effect or interaction was significant. Partial eta squared (ηp²) were computed as effect size estimates and were defined as small (ηp² = .01), medium (ηp² = .06), and large (ηp² = .14). The significance level was set at P < .05 in all cases.

RESULTS

Participants characteristics are presented in Table 1.

Recovery duration had no effect on the time spent at >80%MMP (P = .14; ηp² = .14), >90%MMP (P = .17; ηp² = .12) and >95%MMP (P = .48; ηp² = .03) during the work intervals of the 6 x 4-min and 3 x 8-min HIIT sessions. Recovery duration had no effect on the time spent at >90%VO2max (P = .18; ηp² = .12) and >95%VO2max (P = .26; ηp² = .08) during the work intervals of the 6 x 4-min and 3 x 8-min HIIT sessions. Recovery duration had no effect on the time spent >90%HRmax (P = .17; ηp² = .15) and >95%HRmax (P = .17; ηp² = .15) during the work intervals of the 6 x 4-min and 3 x 8-min HIIT sessions (Table 2).

Statistics and effect-size estimations from the second ANOVA for each work interval variable are shown in Table 3. There were interactions found between recovery duration and work interval for PO (6 x 4) and B[La] (6 x 4). No interactions between recovery duration and work intervals were found for PO (3 x 8), HR, VO2, B[La] (3 x 8) and RPE and TSI%. There were
no main effects of recovery duration for HR, VO₂, B[La] and RPE and TSI%. There was a main effect of work interval number found for PO (6 x 4), HR, VO₂, B[La] and RPE and TSI%, but not for PO (3 x 8). A main effect of session type was found for PO and HR.

There was no effect of recovery duration on perceptual responses, with similar sRPE values reported for the 6 x 4-min (STD, 8.4 ± 0.6 vs IND, 8.3 ± 0.8) and the 3 x 8-min HIIT session (STD, 8.5 ± 0.7 vs IND, 8.3 ± 0.6; P = .26; η² = .08).

Mean recovery interval HR (144 ± 5 vs 134 ± 6 bpm; P = .005; η² = .47) and VO₂ (1.88 ± 0.29 vs 1.52 ± 0.32 L.min⁻¹; P = .002; η² = .49) were significantly lower during the IND 6 x 4-min compared to the STD 6 x 4-min HIIT sessions. There was no significant difference in mean recovery interval HR (130 ± 4 vs 133 ± 3 bpm; P = .29; η² = .08) and VO₂ (1.36 ± 0.21 vs 1.46 ± 0.22 L.min⁻¹; P = .17; η² = .12) during the STD and IND 3 x 8-min HIIT sessions.

Recovery duration had no effect on %O₂HbHb at the end of the recovery intervals (last 30 s average) for the 6 x 4-min (STD, 11.7 ± 3.2% vs IND, 17.3 ± 5.2%) and 3 x 8-min HIIT sessions (STD, 15.8 ± 3.8% vs IND, 15.1 ± 4.9%; P = .07; η² = .22).

DISCUSSION
The main finding of this study was that the IND recovery duration, did not improve the performance or acute physiological response of the work intervals, when compared to the STD recovery duration in well-trained cyclists. Specifically, mean POs were not significantly different between the IND and STD recovery conditions, for both the 6 x 4-min and 3 x 8-min HIIT sessions (Figure 4A & 4B). As recovery duration had no effect on the mean work interval intensity (Figures 4A & 4B), it is not surprising that there was no significant effect on the physiological and metabolic response during the work intervals for both the 6 x 4-min and 3 x 8-min HIIT sessions (Figure 4 & Table 3).

Based on the mVO₂ recovery response of the current study, it can be assumed the 120 s STD recovery duration would have not provided the same recovery at the exercising muscle, in comparison to the longer IND recovery durations (205 ± 79 s) intended to provide a more complete metabolic recovery during the 6 x 4-min HIIT session. However, despite the shorter recovery provided during the STD 6 x 4-min HIIT session, the performance of the work intervals was not affected. Within session NIRS data demonstrates a similar %HbO₂Hb at the end of the 120 s recovery intervals when compared all other recovery interval durations. These data demonstrate that 120 s recovery may be long enough for adequate O₂ delivery to the exercising muscle, allowing key recovery process to occur to such an extent that work interval performances could be maintained (i.e. resynthesis of ATP, PCr, restoration of myoglobin O₂ stores and muscle lactate utilisation). This may provide further insight for previous research which similarly found increases in recovery interval duration beyond 120 s during 6 x 4-min HIIT sessions do not induce any additional benefits for subsequent work bouts 3-5. In the case of the 3 x 8-min HIIT sessions the STD recovery duration (240 s) was longer than the IND recovery duration (200 ± 81 s). Therefore, it would be assumed that a similar metabolic recovery was attained during both recovery prescriptions, hence the similar work interval
performances. This suggests that a full recovery of $m\text{VO}_2$ may not be required to maximise work interval performance during HIIT.

In agreement with Schoenmakers & Reed\(^4\) and Smilios et al.\(^3\) the current study found recovery interval duration to have no effect on the time participants spent exercising $>90$ and $>95\%$ of $\text{VO}_2\text{max}$ and $\text{HR\text{max}}$ during the work intervals (Table 2), despite subsequent work intervals starting from a lower $\text{VO}_2$ after the longer recovery intervals. Schoenmakers & Reed\(^5\) reported that shorter recovery intervals (1 min) resulted in an increased metabolic rate at the start of the next work interval, which lengthened the time needed to reach a $\text{VO}_2$ plateau. Furthermore, the mean response time of $\text{VO}_2$ and HR was found to be faster after longer recovery intervals ($\geq 3$ min) and was accompanied by higher $\text{VO}_2$ and HR amplitude.\(^3,4\) This explains why similar times spent at $>90$ and $>95\%$ of $\text{VO}_2\text{max}$ and $\text{HR\text{max}}$ were found between recovery durations, despite the work intervals starting from a lower $\text{VO}_2$ and HR after the longer recovery intervals.

The HR and $\text{VO}_2$ results of the current study do not support the implementation of the IND recovery duration, over the STD 2:1 work recovery ratio. Nevertheless, the current study results and those of Schoenmakers & Reed\(^4\) and Smilios et al.\(^3\) show that shorter recovery intervals ($\leq 2$ min) allow for a higher percentage of the overall session to be completed at $>90\%\text{VO}_2\text{max}$, resulting in greater accumulation of physiological stress relative to the total time spent training, making for a more time efficient HIIT session.

Recovery interval duration had no effect on reported RPE or sRPE values, during both the 6 x 4-min and 3 x 8-min HIIT sessions (Figures 4I & 4J). Throughout all four HIIT sessions there was a linear increase in work interval RPE, with reported values reaching between 18 and 19 at the last work interval. This linear increase in RPE occurred despite mean PO being relatively consistent across the work intervals (Figures 4A & 4B). Similar increases in RPE have been observed in previous HIIT studies involving well trained runners, despite the participants maintaining a relative constant running velocity across the work intervals.\(^4,5,13\) The upward drift in RPE can be attributed to the increasing physiological, biomechanical, and psychological stress the participants experienced as the HIIT sessions progressed.\(^28,29\)

To establish recovery duration, the current study measured $m\text{VO}_2$ response after a single 4 or 8 min high intensity work interval, and then applied this to a HIIT session of multiple high intensity work intervals. It is important to note that restoration of PCr, which is closely linked to the time constant for $m\text{VO}_2$ recovery, takes longer as HIIT sessions progress,\(^30\) and so it is likely that the optimal recovery duration required between work intervals changes across a HIIT session. Therefore, the measurement of $m\text{VO}_2$ response, and thus recovery duration, after a single high intensity work interval may not be reflective of that performed following a series of HIIT intervals.

**PRACTICAL APPLICATIONS**

By increasing or decreasing the recovery interval duration within the range of the 2:1 work recovery ratio, this study has found there to be no significant effect on the performance of subsequent work intervals and the acute physiological and perceptual response to the HIIT session (when using passive recoveries). Coaches and athletes should consider utilising the 2:1 work recovery ratio when programming 4 or 8 min work interval duration HIIT sessions. In doing so, they can be reasonably confident they are achieving adequate recovery between work intervals, while maximising the time spent training. Importantly, this study used a cohort of trained cyclists, and so caution is advised when extrapolating findings beyond the scope of the current study.
CONCLUSION

Individualising HIIT recovery duration based upon the resolution of mVO₂ to baseline levels, does not improve the performance of the work intervals or the acute physiological response of the HIIT session, when compared to a STD 2:1 recovery duration.

ACKNOWLEDGEMENTS

N/A

REFERENCES


FIGURE CAPTIONS

Fig. 1. Schematic of repeated occlusion protocol for the determination of mVO$_2$ recovery duration.

Fig. 2. (A) Example of mVO$_2$ recovery curve. In this example the 95% mVO$_2$ value output from equation [1] was 0.78 (ml.O$_2$.min$^{-1}$.100g$^{-1}$). The time point at which the mVO$_2$ curve intercepted 0.78 (ml.O$_2$.min$^{-1}$.100g$^{-1}$) provides the IND recovery duration (i.e. 260-s). (B) Complete HHb trace from determination of mVO$_2$ recovery duration protocol.

Fig. 3. Schematic for the 6 x 4-min HIIT protocol (top), Schematic for the 3 x 8-min HIIT protocol (bottom).

Fig. 4. (A/B) mean PO, (C/D) mean HR, (E/F) mean VO$_2$, (G/H) mean B[La], (I/J) mean RPE, (K/L) mean TSI %. Data are displayed per work interval as mean ± SD for the 6 x 4-min and 3 x 8-min HIIT sessions with STD recovery duration (closed triangles) and IND recovery duration (open circles). $\phi$ Significant difference from interval 1 (all $P < 0.05$). $\phi$ Significant difference from previous interval (all $P < 0.05$). $\phi$ Main effect of work interval number (all $P < 0.001$). $\phi$ Interaction between recovery duration and work interval (all $P < 0.05$). *Significant difference between recovery durations (all $P < 0.05$).
Table 1 Participant characteristics and preliminary test results (mean ± SD)

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<tr>
<td>Height (cm)</td>
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<tr>
<td>Mass (kg)</td>
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<td>4-min IND mVO2 duration (s)</td>
<td>205 ± 79</td>
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<td>8-min IND mVO2 duration (s)</td>
<td>200 ± 81</td>
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<td>VL Skin Fold (mm)</td>
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<td>Thigh Circumference (cm)</td>
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<tr>
<td>VO2max (L.min⁻¹)</td>
<td>4.3 ± 0.6</td>
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<td>Relative VO2max (ml.kg.min⁻¹)</td>
<td>60 ± 7</td>
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<td>MMP (W)</td>
<td>373 ± 57</td>
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<tr>
<td>Relative MMP (W.kg⁻¹)</td>
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<td>HRmax (bpm)</td>
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<tr>
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<td>Years competing</td>
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<td>Mean weekly training hours</td>
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mVO2, muscle oxygen consumption; VL, vastus lateralis muscle; VO2max, maximal oxygen consumption; MMP, maximal minute power; HRmax, maximal minute heart rate.
### Table 2  
**Time in seconds** spent above percentages of VO$_{2\text{max}}$, $HR_{\text{max}}$ and MMP during work intervals

<table>
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<tr>
<th>Prescription</th>
<th>Time at %VO$_{2\text{max}}$</th>
<th>Time at %$HR_{\text{max}}$</th>
<th>Time at %MMP</th>
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<tr>
<td></td>
<td>80</td>
<td>90</td>
<td>95</td>
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<tr>
<td>STD 6 x 4</td>
<td>1178 ± 139</td>
<td>821 ± 311</td>
<td>502 ± 332</td>
</tr>
<tr>
<td>IND 6 x 4</td>
<td>1156 ± 153</td>
<td>749 ± 364</td>
<td>451 ± 390</td>
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<tr>
<td>STD 3 x 8</td>
<td>1202 ± 126</td>
<td>753 ± 396</td>
<td>398 ± 330</td>
</tr>
<tr>
<td>IND 3 x 8</td>
<td>1176 ± 176</td>
<td>649 ± 345</td>
<td>278 ± 258</td>
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VO$_{2\text{max}}$, maximal oxygen consumption; $HR_{\text{max}}$, maximal minute heart rate; MMP, maximal minute power.
<table>
<thead>
<tr>
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<th>Prescription</th>
<th>Interaction (Duration X Interval)</th>
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<th>Main effect of work interval number</th>
<th>Main effect of session type (6x4 vs 3x8)</th>
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<td>.001*</td>
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<td></td>
<td>3 x 8</td>
<td>1.26</td>
<td>.30</td>
<td>.08</td>
<td>1.61</td>
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Abbreviations: PO, power output; HR, heart rate; VO₂, oxygen consumption; B[La], blood lactate concentration; RPE, rating of perceived exertion; TSI%, tissue saturation index. *Statistical significance.
Fig. 1. Schematic of repeated occlusion protocol for the determination of mVO₂ recovery duration.
Fig. 2. (A) Example of mVO$_2$ recovery curve. In this example the 95% mVO$_2$ value output from equation [1] was 0.78 (ml.O$_2$.min$^{-1}$.100g$^{-1}$). The time point at which the mVO2 curve intercepted 0.78 (ml.O$_2$.min$^{-1}$.100g$^{-1}$) provides the IND recovery duration (i.e. 260-s), (B) Complete HHb trace from determination of mVO$_2$ recovery duration protocol.
Fig. 3. Schematic for the 6 x 4-min HIIT protocol (top), Schematic for the 3 x 8-min HIIT protocol (bottom).
Fig. 4. (A/B) mean PO, (C/D) mean HR, (E/F) mean VO$_2$, (G/H) mean B[La], (I/J) mean RPE. Data are displayed per work interval as mean ± SD for the 6 x 4-min and 3 x 8-min HIIT sessions with STD recovery duration (closed triangles) and IND recovery duration (open circles). φ Significant difference from interval 1 (all P < 0.05). T Significant difference from previous interval (all P < 0.05). $ Main effect of work interval number (all P < 0.001). # Interaction between recovery duration and work interval (all P < 0.05). * Significant difference between recovery durations (all P < 0.05).