

TITLE

Influence of recovery intensity on oxygen demand and repeated sprint performance

T.Yamagishi,¹ J. Babraj ¹

¹Division of Sport and Exercise Sciences, Abertay University, Dundee, UK

Congress: This paper has not been presented elsewhere.

Funding: There was no external funder for this project.

Conflict of Interest: The authors report no conflict of interest.

Corresponding Author:

T. Yamagishi,

Division of Sport and Exercise Sciences,

Abertay University,

Bell street,

Dundee, DD1 1HG,

United Kingdom

E-mail: 1206876@abertay.ac.uk

ABSTRACT

Aim. This study aimed to determine effects of recovery intensity (passive, 20, 30 and 40% $\dot{V}O_{2peak}$) on oxygen uptake kinetics, performance and blood lactate accumulation during repeated sprints.

Methods. 7 moderately-trained male participants ($\dot{V}O_{2peak}$: 48.1 ± 5.1 ml·kg⁻¹·min⁻¹) performed 4 x 30-s repeated Wingate tests on 4 separate occasions.

Results. Recovery of $\dot{V}O_2$ between sprints was prolonged with recovery intensity (time required to reach 50% $\dot{V}O_{2peak}$: Passive: 50 ± 9 ; 20%: 81 ± 17 ; 30%: 130 ± 43 ; 40%: 188 ± 62 sec, $P < 0.001$), while $\dot{V}O_2$ -to-sprint work ratio was mainly increased by the higher intensities (Passive: 138 ± 17 ; 20%: 149 ± 14 ; 30%: 159 ± 15 ; 40%: 158 ± 17 ml·min⁻¹·kJ⁻¹, $P = 0.001$). The decline in peak power tended to be greater in the higher intensity conditions during sprint 2 (Passive: 7.4 ± 5.4 ; 20%: 5.8 ± 7.9 ; 30%: 12.7 ± 7.4 ; 40%: $12.7 \pm 5.5\%$, $P = 0.052$), whereas average power was less decreased with recovery intensity during sprint 4 (Passive: 22.4 ± 8.9 ; 20%: 19.9 ± 6.1 ; 30%: 18.4 ± 7.3 ; 40%: $16.6 \pm 6.2\%$, $P = 0.036$). Blood lactate was not different with recovery intensity ($P = 0.251$).

Conclusion. The present study demonstrated that while the higher recovery intensities induce prolonged oxygen recovery and impaired peak power restoration during the initial sprints, those intensities provide a greater aerobic contribution to sprint performance, resulting in better power maintenance during the latter sprints.

Key Words: Intermittent exercise - Active recovery - Passive recovery - Anaerobic metabolism - Aerobic metabolism.

TEXT

Introduction

Recovery mode (i.e. active vs. passive) has been shown to be important during high-intensity intermittent exercise.¹⁻⁴ When recovery duration is brief (15 to 21 sec) relative to sprint duration (e.g. sprint: rest ratio between 1:1 to 1:5), active recovery results in a greater performance decline in subsequent sprints.²⁻⁴ Furthermore, there was a shorter time to exhaustion during repeated sprints with active recovery regardless of intensity.^{5, 6} Conversely, active recovery improves sprint power production when repeated efforts are interspersed with longer recovery periods (180 to 300 sec; sprint: rest ratio between 1:8 to 1:12).^{1, 7, 8} This difference could be due to the oxygen cost of active recovery which may inhibit re-oxygenation of the haemoglobin and myoglobin^{2, 5, 6} or re-synthesis of PCr^{3, 4} in short recoveries. With longer recovery, the increased aerobic metabolism induced by active recovery may allow greater oxygen availability to facilitate PCr restoration during recovery^{1, 9, 10} and increase aerobic energy production during repeated sprints.¹¹

Over recent years the use of repeated Wingate-based exercise training (four to six all-out 30-s cycling efforts interspersed with 4-min recovery, sprint: rest ratio of 1:8) has become increasingly utilised to improve metabolic functions (e.g. an improved mitochondrial function) and endurance performance.¹²⁻¹⁵ However, most of the studies have not considered workload during the recovery period. Previously, active recovery (28 to 40% of $\dot{V}O_{2max}$) during repeated Wingate tests has shown a greater maintenance of power production with similar blood lactate accumulation compared to passive recovery.^{1, 8, 16} Despite this, only Bogdanis *et al.*¹ investigated effects of recovery mode on oxygen uptake during two 30-s Wingate tests interspersed with 4-min recovery. Furthermore, although they found that active recovery increased $\dot{V}O_2$ during the 4-min recovery compared to passive recovery, only

average $\dot{V}O_2$ was reported due to the method used (Douglas bag method).¹ Therefore, effects of recovery mode on the time course of oxygen uptake recovery is yet to be determined. Moreover, it is unknown whether the intensity of recovery affects overall sprint performance, cardiorespiratory or blood lactate response as these studies merely compared active or passive but not intensity of recovery.

Therefore, this study sought to determine the effects of four different recovery intensities (passive, 20, 30 and 40% $\dot{V}O_{2peak}$) on oxygen uptake kinetics, sprint performance, and blood lactate accumulation during repeated 30-s Wingate tests that have been utilised previously to promote training adaptations.¹⁵ It was hypothesised that oxygen demand would be increased with recovery intensity, whereas all active recovery intensities would result in improved sprint performance with a similar level of blood lactate when compared with passive recovery.

Materials and Methods

Subjects

Seven healthy active males who took part in a minimum of 3-h exercise per week participated in the present study (Table I). Subjects were fully informed both verbally and in writing about the study as well as any risks before giving their informed consent. The study was approved by the Institutional Ethics Committee and was carried out in line with the Declaration of Helsinki.

Experimental design

All subjects were asked to maintain their normal diet and activity throughout the study period and to refrain from alcohol intake and any form of intense physical activity for 24 h prior to each session. Subjects reported to the Human Performance Laboratory having only

consumed water 4h prior to arriving at the lab. All subjects performed 5 sessions at a similar time of day (± 2 h) in a controlled environment (temperature: 19.8 ± 0.7 °C; humidity: $34.0 \pm 3.8\%$) throughout the study period. Each session was separated by at least a period of 48h but by no more than 2 weeks. On the initial visit, body composition was recorded on a calibrated bio-impedance meter (Tanita TBF 300, Tanita Co., Ltd. Japan) where body fat and mass were recorded (Table I).

Determination of $\dot{V}O_{2peak}$

Subjects performed an exhaustive incremental cycling test to determine $\dot{V}O_{2peak}$ via breath by breath analysis (Metalyzer[®]3B gas analyser, Cortex, Leipzig, Germany). Subjects warmed up by cycling for 4 minutes at any speed above 60 rpm on an unloaded cycle ergometer (Monark Ergonomic 874E, Varberg, Sweden). The weight of the bike cradle was then increased by 0.5 kg every minute until the subjects could no longer maintain a speed of 60 rpm or until volitional exhaustion occurred. After the incremental test, an additional supra-maximal verification test was performed to ensure that true $\dot{V}O_{2peak}$ had been elicited. Subjects rested for 5 minutes either passively or actively (unloaded cycling). They then cycled again until they reached the limit of tolerance (~ 2 min) at a work rate equivalent to one stage higher (0.5kg heavier) than that of the last stage in the incremental test.¹⁷ A high correlation between peak $\dot{V}O_2$ achieved during the incremental and verification tests was obtained using a linear regression model ($r \geq 0.99$ in both relative and absolute values), and the difference in peak $\dot{V}O_2$ between the two tests (77.4 ± 36.4 ml \cdot min⁻¹ or 1.0 ± 0.5 ml \cdot kg⁻¹ \cdot min⁻¹) was less than the conventional concept of a plateau in $\dot{V}O_2$ (i.e. ≤ 150 ml \cdot min⁻¹ or ≤ 2.1 ml \cdot kg⁻¹ \cdot min⁻¹)¹⁸, suggesting the attainment of a true $\dot{V}O_{2peak}$. Respiratory gas exchange measures were averaged every 10s with $\dot{V}O_{2peak}$ calculated as the highest oxygen consumed over a 10-s period, while power output elicited at $\dot{V}O_{2peak}$ was defined as the maximal aerobic power

(MAP). Heart rate was recorded throughout using a heart rate monitor (Polar Electro, Kempele, Finland) and was averaged every 5s. Maximal heart rate (HR_{max}) was defined as the highest heart rate recorded over a 5-s period. Recovery intensities (i.e. 20, 30 and 40% of $\dot{V}O_{2\text{peak}}$) were determined according to the linear relationship between each individual's $\dot{V}O_2$ and work rate during the incremental test.

Procedures of repeated Wingate tests and determination of cardiorespiratory kinetics

Subjects performed 4 x 30 second cycle sprints with 4 minutes of recovery (Monark Ergonomic 894E, Varberg, Sweden) against 7.5% bodyweight on 4 different days separated by at least 48 h. Upon cessation the workload was adjusted to a given recovery intensity (20, 30, or 40% $\dot{V}O_{2\text{peak}}$), which had been randomly allocated, and the subjects cycled at this intensity for 230 sec. 10 sec before the next sprint, the workload was adjusted again to 7.5% of the subjects' bodyweight. In the case of passive recovery, the subjects remained still on the bike for 240 sec. Heart rate (Polar Electro, Kempele, Finland) and on-line gas analysis (Metalyzer[®]3B gas analyser, Cortex, Leipzig, Germany) were both recorded continuously throughout. Oxygen uptake and HR were averaged every 5 seconds during the sprint protocol, and average values were expressed as percentage of $\dot{V}O_{2\text{peak}}$ and HR_{max} determined in the incremental test respectively. During recovery, each $\dot{V}O_2$ or HR was divided by $\dot{V}O_{2\text{peak}}$ or HR_{max}, and the slope of $\dot{V}O_2$ or HR versus time was determined for each recovery condition using a second-order polynomial regression (Figure 3A & 3B).¹⁹ Although the decrease in $\dot{V}O_2$ was well described by the mathematical model chosen as a group (Figure 3A), a large intra- or inter-individual difference was observed ($r=0.55$ to 0.98) mainly due to the different recovery conditions. Therefore, recovery kinetics of $\dot{V}O_2$ was determined by measuring the time required to reach 50% $\dot{V}O_{2\text{peak}}$ (T to $50\dot{V}O_{2\text{peak}}$) in accordance with the previous study.¹⁹ If $\dot{V}O_2$ did not reach 50% $\dot{V}O_{2\text{peak}}$ during the 4-min

recovery (3 out of 84 cases), T to $50\dot{V}O_{2\text{peak}}$ was defined as 240 sec. However, $\dot{V}O_2$ reduced to 51% $\dot{V}O_{2\text{peak}}$ during the 4-min recovery in the above all 3 cases, suggesting that this method does not seem to underestimate T to $50\dot{V}O_{2\text{peak}}$ and is independent of the mathematical model chosen.

Determination of repeated sprint performance and blood lactate accumulation

Peak power (PP) and average power (AP) during each 30-s sprint was automatically calculated using software (Monark Anaerobic Test Software version 2.24.2, Monark Exercise AB). Power drop of PP or AP across sprints was also determined using the following formula; $(PP \text{ or } AP^{S2, S3 \text{ or } S4} - PP \text{ or } AP^{S1}) / PP \text{ or } AP^{S1} \times 100$, where PP or AP^{S1} is peak or average power of sprint 1 and PP or $AP^{S2, S3 \text{ or } S4}$ is peak or average power of sprint 2, 3, or 4. $\dot{V}O_2$ – to – sprint work ratio ($\dot{V}O_2/\text{kJ}$) was also calculated in an attempt to determine aerobic contribution relative to mechanical work produced across the sprints and recovery conditions.²⁰ Blood samples were taken from the subjects' fingertips before the first sprint and 180 sec after each sprint to determine blood lactate concentration (Lactate pro, Arkray Inc., Kyoto, Japan). Briefly, 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.

Statistical Analyses

All data are presented as means \pm standard deviation. Before conducting parametric tests, a one sample Kolmogorov-Smirnov test was performed to ensure that all values were normally distributed. A two-way analysis of variance (ANOVA) with repeated measures was used to determine overall differences between recovery conditions (passive, 20, 30 and 40% $\dot{V}O_{2\text{peak}}$) and sprint/recovery number for Wingate performance, cardiorespiratory and blood lactate

variables. Greenhouse-Geisser corrections were used where the violation of sphericity was detected. In the case of a significant main effect of condition, the test was followed by post-hoc Least significant difference (LSD) test. Where a significant sprint/recovery number by condition interaction effect was observed, one-way repeated ANOVA with post-hoc LSD test was performed to determine differences among the conditions for each sprint/recovery. Moreover, where the post-hoc test revealed a significant difference between conditions, effect size (Cohen's d) was calculated. Due to the study design (i.e. repeated measures), Cohen's d was corrected for dependence between means using the equation suggested by Morris and DeShon;²¹ $d = M_{\text{diff}} / SD_{\text{pooled}} \sqrt{2(1 - r)}$, 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.²² Changes over time ($P < 0.01$ in all cases) are only mentioned where appropriate for clarity. All statistics were run on IBM® SPSS® Version 21.0 for Windows and the significance level was set at $P < 0.05$.

Results

Wingate performance

There was no main effect of recovery condition in the overall peak power, average power or power drop rate (Table II). However, a significant sprint by condition interaction effect was observed in peak power, and the drop rates of both peak and average power (Table II).

Although the 30% recovery condition temporarily decreased peak power compared to the 20% recovery during sprint 2 ($P < 0.01$, $d = 1.44$), all active recovery conditions improved it during the last sprint compared with the passive recovery ($P < 0.05$ in all cases, $d = 0.94$, 1.01 and 0.95 for passive vs. 20, 30 and 40%, respectively) (Table II). A greater drop rate in peak power was observed following the higher recovery intensities during sprint 2, which nearly

reached significance ($P=0.052$), whereas that in average power was significantly improved by the 30 and 40% recovery conditions compared with the passive recovery ($P<0.05$ in both cases, $d=1.46$ and 1.18 for passive vs. 30 and 40%, respectively), or for the 40% recovery compared to the 20% recovery ($P<0.05$, $d=1.17$) during the last sprint (Table II).

Oxygen uptake during sprints

There was a main effect of recovery condition in overall $\dot{V}O_2$ during sprints (Figure 1A: Passive: 57 ± 5 ; 20%: 63 ± 6 ; 30%: 66 ± 5 ; 40%: 67 ± 5 % $\dot{V}O_{2peak}$, $P<0.001$). Overall sprint $\dot{V}O_2$ was significantly elevated for all active recovery conditions compared with the passive recovery ($P<0.05$ for passive vs. 20%, $d=1.16$; $P<0.01$ for passive vs. 30 and 40%, $d=2.12$ and 2.33 for passive vs. 30 and 40%, respectively), while there was no significant difference among active recovery conditions (Figure 1A). There was a main effect of condition in $\dot{V}O_2$ -to-sprint work ratio (Figure 1C: Passive: 138 ± 17 ; 20%: 149 ± 14 ; 30%: 159 ± 15 ; 40%: 158 ± 17 ml·min⁻¹·kJ⁻¹, $P=0.001$). $\dot{V}O_2$ -to-sprint work ratio was significantly increased in the 30 and 40% recovery groups compared to the passive recovery ($P<0.01$ in both cases, $d=1.64$ and 2.23 for passive vs. 30 and 40%, respectively). It was also significantly elevated for the 30% recovery condition compared with the 20% recovery condition ($P<0.05$, $d=1.16$) (Figure 1C).

Heart rate during sprints

There was a main effect of recovery condition in overall heart rate during sprints (Figure 1B: Passive: 78 ± 4 ; 20%: 80 ± 4 ; 30%: 83 ± 6 ; 40%: 84 ± 4 % HR_{max}, $P<0.001$). Overall sprint HR was significantly elevated for the 30 and 40% recovery groups compared with the passive recovery ($P<0.01$ for passive vs. 30%, $d=2.74$; $P<0.001$ for passive vs. 40%, $d=4.04$) or 20% recovery group ($P<0.05$ for 20% vs. 30%, $d=1.14$; $P<0.01$ for 20% vs. 40%, $d=2.74$), but

there was no significant difference between the 20% recovery and the passive recovery (Figure 1B).

Oxygen uptake during recovery

There was a main effect of recovery condition in overall $\dot{V}O_2$ during recovery (Figure 2A: Passive: 35 ± 4 ; 20%: 51 ± 5 ; 30%: 57 ± 4 ; 40%: 63 ± 7 % $\dot{V}O_{2peak}$, $P < 0.001$). Overall recovery $\dot{V}O_2$ was significantly increased for all active recovery conditions compared with the passive recovery ($P < 0.001$ in all cases, $d = 4.52, 6.49$ and 6.61 for passive vs. 20, 30 and 40%, respectively) (Figure 2A). It was also significantly elevated for the 30 and 40% recovery groups compared with the 20% recovery group ($P < 0.01$ for 20% vs. 30%, $d = 2.22$; $P < 0.001$ for 20% vs. 40%, $d = 5.66$), and for the 40% recovery compared to the 30% recovery condition ($P < 0.05$, $d = 1.96$) (Figure 2A). Likewise there was a main effect of condition in time required to reach 50% $\dot{V}O_{2peak}$ (Figure 2C: Passive: 50 ± 9 ; 20%: 81 ± 17 ; 30%: 130 ± 43 ; 40%: 188 ± 62 sec, $P < 0.001$). T to 50% $\dot{V}O_{2peak}$ was increased for all active recovery conditions compared with the passive recovery ($P < 0.01$ in all cases, $d = 2.22, 2.53$ and 2.99 for passive vs. 20, 30 and 40%, respectively) (Figure 2C). It was also significantly elevated for the 30 and 40% recovery groups compared with the 20% recovery group ($P < 0.05$ for 20% vs. 30%, $d = 1.69$; $P < 0.01$ for 20% vs. 40%, $d = 2.88$), and for the 40% recovery compared to the 30% recovery condition ($P < 0.05$, $d = 1.53$) (Figure 2C). Example of recovery kinetics of $\dot{V}O_2$ is shown in Figure 3A.

Heart rate during recovery

There was a main effect of recovery condition in overall HR during recovery (Figure 2B: Passive: 74 ± 4 ; 20%: 80 ± 4 ; 30%: 82 ± 3 ; 40%: 84 ± 4 %HRmax, $P < 0.001$). Overall recovery HR was significantly increased for all active recovery conditions compared with the

passive recovery ($P < 0.01$ in all cases, $d = 2.15$, 7.36 and 2.73 for passive vs. 20, 30 and 40%, respectively) (Figure 2B). It was also significantly elevated for the 30 and 40% recovery groups compared with the 20% recovery group ($P < 0.05$ for 20% vs. 30%, $d = 0.96$; $P < 0.01$ for 20% vs. 40%, $d = 1.62$) (Figure 2B). Example of recovery kinetics of HR is shown in Figure 3B.

Blood lactate

Although blood lactate level significantly rose with repeated sprints, there was no main or sprint by condition interaction effect in blood lactate concentration (Table III).

Discussion

This study sought to determine the effects of recovery intensity on the oxygen uptake kinetics, repeated 30-s Wingate performance and blood lactate accumulation. To the best of our knowledge, this is the first study to examine the effects of four different recovery intensities (passive, 20, 30 and 40% $\dot{V}O_{2peak}$) during the typical Wingate-based exercise training protocols.¹⁵ The novel findings of the study are that oxygen cost during recovery is increased with the intensity of recovery, aerobic contribution to repeated sprint performance is only elevated by the higher recovery intensities and any active recovery intensity does not cause an alteration of blood lactate accumulation when compared with the passive recovery.

Although average $\dot{V}O_2$ during the repeated sprints was increased in all active recovery conditions compared with the passive recovery, $\dot{V}O_2$ - to- sprint work ratio was only significantly increased by the 30 and 40% recovery groups (Figure 1A & 1C). $\dot{V}O_2$ at the end of the recovery periods was greater in the higher recovery conditions (30%: 46.5 ± 2.7 ; 40%: 47.6 ± 6.5 % $\dot{V}O_{2peak}$) compared to the 20% recovery group (38.1 ± 1.7 % $\dot{V}O_{2peak}$, $P < 0.01$) as

well as the passive recovery ($21.9 \pm 4.6 \% \dot{V}O_{2\text{peak}}$, $P < 0.001$; Figure 3A), suggesting that the subjects began sprints with an elevated oxidative metabolism following the higher recovery intensities compared to the passive or 20% recovery. Further, HR during the sprints was significantly increased by the higher recovery intensities compared to the passive or 20% recovery condition (Figure 1B), indicating an increased muscle blood flow and thus a greater O_2 delivery to the working muscles²³ during the sprints in these conditions. The elevated whole body $\dot{V}O_2$ along with the increased HR following the higher recovery intensities seems to have become increasingly important with the successive sprint repetitions where muscle O_2 extraction and thus aerobic contribution to mechanical work progressively increase (Figure 1C).^{20, 24} The attenuated drop in average power induced by the higher recovery intensities during the last sprint (Table II) would support this assumption.

Conversely, there was a tendency for the 30 and 40% recovery groups to cause greater peak power decline during sprint 2 compared with the passive or the lower recovery condition (Table II). In contrast to the current study, Bogdanis *et al.*¹ demonstrated that active recovery at 40% $\dot{V}O_{2\text{max}}$ increased power production in sprint 2 compared to passive recovery, which was totally attributed to a 3.1% higher power output produced during the initial 10s, when two 30-s cycle sprints were separated by 4 min. Since they also found a high correlation between re-synthesis of PCr and recovery of power output during the initial 10s of the second 30-s sprint ($r=0.84$, $P < 0.05$),²⁵ the improved power production might be attributed to a greater O_2 availability for PCr re-synthesis induced by the active recovery (as reflected by greater $\dot{V}O_2$ compared with passive recovery, $P < 0.01$) during the 4-min recovery.¹ In support of this, Haseler *et al.*⁹ demonstrated that hyperoxia caused by greater fractions of inspired O_2 enhanced PCr restoration during 5-min recovery following submaximal exercise. Nevertheless, considering that a close relationship has been shown between time course of

$\dot{V}O_2$ recovery and that of PCr restoration,^{10, 19} the greater decrease in peak power seen in the higher recovery conditions during sprint 2 could be explained by the prolonged $\dot{V}O_2$ recovery (Figure 2C). Indeed, it has been demonstrated that a prolonged T to $50\dot{V}O_{2peak}$ results in a slower rate of PCr recovery,¹⁹ and within our study T to $50\dot{V}O_{2peak}$ is greater with higher intensity recoveries (Figure 2C). Although Bogdanis *et al.*¹ only reported average $\dot{V}O_2$ during the 4-min recovery (55% $\dot{V}O_{2max}$) and thus the time course of $\dot{V}O_2$ recovery is unknown, a greater maximal aerobic capacity ($4.28 \pm 0.13 \text{ l}\cdot\text{min}^{-1}$; approximately $55 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) of their subjects compared to the current study ($3.6 \pm 0.6 \text{ l}\cdot\text{min}^{-1}$, or $48.1 \pm 5.1 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) might have allowed faster $\dot{V}O_2$ recovery,^{26, 27} possibly resulting in faster and/or greater PCr restoration. Similar to the current study, Lopez *et al.*¹⁶ employed six 30-s cycle sprints alternated by 4-min recovery, and saw a greater peak power drop during sprint 2 in active recovery condition compared with passive recovery, while the active recovery improved average power during sprint 5 and 6. Although they did not report $\dot{V}O_{2max}/\dot{V}O_{2peak}$ of their subjects and therefore their findings cannot be directly compared with those of the current study or the study by Bogdanis *et al.*,¹ it could be assumed that active recovery may not be beneficial when only two sprints are performed whereas it would facilitate maintaining power production with the sprint repetitions, as the sprints become more aerobically demanding.^{20, 25, 28, 29} Lopez *et al.*¹⁶ also found that the active recovery tended to improve peak power during sprint 5 and 6 compared to the passive recovery (improved by $0.3 \text{ W}\cdot\text{kg}^{-1}$ in both cases), which is in agreement with the current study (i.e. peak power during the last sprint; Table II). This may suggest that greater aerobic metabolism caused by active recovery during the rest periods may become increasingly beneficial to PCr restoration as sprints are repeated²⁷ where muscle re-oxygenation rate progressively increases.²⁰

The blood lactate level markedly rose after sprint 1, however the magnitude of increase in

blood lactate notably decreased with the successive bouts (Table III), indicating that anaerobic glycolysis progressively reduced with repeated sprints.^{25, 28, 29} The present study did not find any difference in blood lactate concentration across the recovery intensities. This is not in line with the previous studies employing longer recovery periods (> 450 sec) where active recovery promoted a greater clearance of lactate from the blood,^{8, 30} but similar to those employing shorter recovery periods (15 to 240 sec) where recovery mode did not affect blood lactate concentration despite the difference in repeated sprint performance between recovery conditions.^{1, 5-7} This indicates that the level of blood lactate may not be a decisive factor in repeated sprint exercise and longer recovery duration might be needed to see effects of an increased blood flow induced by active recovery on lactate transport and uptake by other tissues.³¹⁻³³

Practical implications

It is now well established that Wingate-based exercise training induces various physiological and metabolic adaptations comparable to those seen following traditional endurance training despite its markedly lower training volume.^{14, 15} It could be argued that active recovery, especially at higher intensities, ensures greater aerobic demand (e.g. higher $\dot{V}O_2$ and HR) during the training without diminishing overall exercise intensity (i.e. mechanical work) and/or anaerobic demand. Moreover, the smaller power decrement achieved by active recovery in the latter sprints might be related to higher levels of muscle fibre recruitment (chiefly Type II fibres)²⁰ which may bring about greater training benefits. Therefore when designing HIT programmes the demands of the rest period should be considered to ensure maximal adaptation to this type of training.

Conclusions

The present study demonstrates that $\dot{V}O_2$ recovery kinetics and aerobic contribution to power generation during repeated Wingate tests are dependent on the intensity of recovery while no difference was observed in blood lactate accumulation among the recovery conditions. Peak power tended to be decreased with the higher recovery intensities (30 and 40% $\dot{V}O_{2peak}$) during sprint 2 and this might reflect an impaired intramuscular recovery (e.g. PCr recovery) due to the prolonged $\dot{V}O_2$ recovery induced by those conditions. On the other hand, aerobic contribution to sprint performance was only increased by the higher intensities which likely resulted in the less decreased average power during the last sprint. It is currently unknown whether acute alterations in sprint performance and physiological responses caused by the manipulation of recovery modality (i.e. active vs. passive) have an impact on chronic physiological and performance adaptations. Therefore, further research is required to investigate effects of recovery mode on training adaptations (e.g. $\dot{V}O_{2max}$, endurance performance) to sprint interval training.

REFERENCES

1. 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* 1996; 74:461-9.
2. Dupont G, Moalla W, Matran R, Berthoin S. Effect of short recovery intensities on the

- performance during two Wingate tests. *Med Sci Sports Exerc* 2007; 39:1170-6.
3. Spencer M, Bishop D, Dawson B, Goodman C, Duffield R. Metabolism and performance in repeated cycle sprints: active versus passive recovery. *Med Sci Sports Exerc* 2006; 38:1492-9.
 4. Spencer M, Dawson B, Goodman C, Dascombe B, Bishop D. Performance and metabolism in repeated sprint exercise: effect of recovery intensity. *Eur J Appl Physiol* 2008; 103:545-52.
 5. Dupont G, Blondel N, Berthoin S. Performance for short intermittent runs: active recovery vs. passive recovery. *Eur J Appl Physiol* 2003; 89:548-54.
 6. Dupont G, Moalla W, Guinhouya C, Ahmaidi S, Berthoin S. Passive versus active recovery during high-intensity intermittent exercises. *Med Sci Sports Exerc* 2004; 36:302-8.
 7. Connolly DA, Brennan KM, Lauzon CD. Effects of active versus passive recovery on power output during repeated bouts of short term, high intensity exercise. *J Sports Sci Med* 2003; 2:47-51.
 8. Spierer DK, Goldsmith R, Baran DA, Hryniewicz K, Katz SD. Effects of active vs. passive recovery on work performed during serial supramaximal exercise tests. *Int J Sports Med* 2004; 25:109-14.
 9. Haseler LJ, Hogan MC, Richardson RS. Skeletal muscle phosphocreatine recovery in exercise-trained humans is dependent on O₂ availability. *J Appl Physiol* 1999; 86:2013-8.
 10. McMahon S, Jenkins D. Factors affecting the rate of phosphocreatine resynthesis following intense exercise. *Sports Med* 2002; 32:761-84.
 11. Dorado C, Sanchis-Moysi J, Calbet JA. Effects of recovery mode on performance, O₂ uptake, and O₂ deficit during high-intensity intermittent exercise. *Can J Appl Physiol* 2004; 29:227-44.
 12. Burgomaster KA, Cermak NM, Phillips SM, Benton CR, Bonen A, Gibala MJ. Divergent response of metabolite transport proteins in human skeletal muscle after sprint interval

- training and detraining. *Am J Physiol Regul Integr Comp Physiol* 2007; 292:R1970-6.
13. Burgomaster KA, Heigenhauser GJ, Gibala MJ. Effect of short-term sprint interval training on human skeletal muscle carbohydrate metabolism during exercise and time-trial performance. *J Appl Physiol* 2006; 100:2041-7.
 14. Burgomaster KA, Howarth KR, Phillips SM, Rakobowchuk M, Macdonald MJ, McGee SL, *et al.* Similar metabolic adaptations during exercise after low volume sprint interval and traditional endurance training in humans. *J Physiol*. 2008; 586:151-60.
 15. Gibala MJ, Little JP, van Essen M, Wilkin GP, Burgomaster KA, Safdar A, *et al.* Short-term sprint interval versus traditional endurance training: similar initial adaptations in human skeletal muscle and exercise performance. *J Physiol* 2006; 575:901-11.
 16. Lopez EI, Smoliga JM, Zavorsky GS. The effect of passive versus active recovery on power output over six repeated wingate sprints. *Res Q Exerc Sport* 2014; 85:519-26.
 17. Rossiter HB, Kowalchuk JM, Whipp BJ. A test to establish maximum O₂ uptake despite no plateau in the O₂ uptake response to ramp incremental exercise. *J Appl Physiol* 2006; 100:764-70.
 18. Duncan GE, Howley ET, Johnson BN. Applicability of VO₂max criteria: discontinuous versus continuous protocols. *Med Sci Sports Exerc* 1997; 29:273-78.
 19. Cohen-Solal A, Laperche T, Morvan D, Geneves M, Caviezel B, Gourgon R. Prolonged kinetics of recovery of oxygen consumption after maximal graded exercise in patients with chronic heart failure. Analysis with gas exchange measurements and NMR spectroscopy. *Circulation* 1995; 91:2924-32.
 20. 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; 112:767-79.
 21. Morris SB, DeShon RP. Combining effect size estimates in meta-analysis with repeated

- measures and independent-groups designs. *Psychol Methods* 2002; 7:105-25.
22. Cohen J. A power primer. *Psychol Bull* 1992; 112:155-59.
23. Tordi N, Perrey S, Harvey A, Hughson RL. Oxygen uptake kinetics during two bouts of heavy cycling separated by fatiguing sprint exercise in humans. *J Appl Physiol* 2003; 94:533-41.
24. Jones AM, Koppo K, Burnley M. Effects of prior exercise on metabolic and gas exchange responses to exercise. *Sports Med* 2003; 33:949-71.
25. Bogdanis G, Nevill M, Boobis L, Lakomy H. Contribution of phosphocreatine and aerobic metabolism to energy supply during repeated sprint exercise. *J Appl Physiol* 1996:876-84.
26. Dupont G, McCall A, Prieur F, Millet GP, Berthoin S. Faster oxygen uptake kinetics during recovery is related to better repeated sprinting ability. *Eur J Appl Physiol* 2010; 110:627-34.
27. Tomlin D, Wenger H. The relationship between aerobic fitness and recovery from high intensity intermittent exercise. *Sports Med* 2001:1-11.
28. Gaitanos GC, Williams C, Boobis LH, Brooks S. Human muscle metabolism during intermittent maximal exercise. *J Appl Physiol* 1993; 75:712-9.
29. 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:E890-900.
30. Wahl P, Mathes S, Köhler K, Achtzehn S, Bloch W, Mester J. Effects of active vs. passive recovery during Wingate-based training on the acute hormonal, metabolic and psychological response. *Growth Horm IGF Res* 2013; 23:201-8.
31. Belcastro AN, Bonen A. Lactic acid removal rates during controlled and uncontrolled recovery exercise. *J Appl Physiol* 1975; 39:932-6.

32. Brooks GA. Intra- and extra-cellular lactate shuttles. *Med Sci Sports Exerc* 2000; 32:790-9.

33. Gladden LB. Muscle as a consumer of lactate. *Med Sci Sports Exerc* 2000; 32:764-71.

Table 1 Physical and Physiological characteristics of the subjects.

	Subjects (n = 7)
$\dot{V}O_{2\text{peak}}$ ($\text{l}\cdot\text{min}^{-1}$)	3.6 ± 0.6
$\dot{V}O_{2\text{peak}}$ ($\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$)	48.1 ± 5.1
MAP (W)	321 ± 71
HRmax ($\text{beats}\cdot\text{min}^{-1}$)	181 ± 11
Age (year)	23 ± 5
Height (cm)	180 ± 6
Body mass (kg)	74 ± 8
Body fat (%)	14.4 ± 4.9

Abbreviations: $\dot{V}O_{2\text{peak}}$, peak oxygen uptake; MAP, maximal aerobic power; HRmax, maximal heart rate. Values are means \pm SD.

Table 2 Peak power, average power and respective power drop rate across the sprints and recovery conditions.

Peak power ($\text{W}\cdot\text{kg}^{-1}$) [#]	Passive	20%	30%	40%
Sprint 1	12.6 ± 2.2	12.8 ± 2.1	12.4 ± 1.9	13.1 ± 1.7
Sprint 2 [§]	11.7 ± 2.1	12.1 ± 2.1 ^{‡‡}	10.8 ± 2.0	11.4 ± 1.4
Sprint 3	10.4 ± 1.9	10.9 ± 2.0	10.0 ± 1.5	11.0 ± 1.6
Sprint 4 [§]	9.5 ± 1.7	10.1 ± 1.8*	10.3 ± 1.8*	10.6 ± 1.8*
PP drop relative to Sprint 1 (%) ^{##}	Passive	20%	30%	40%
Sprint 2	7.4 ± 5.4	5.8 ± 7.9	12.7 ± 7.4	12.7 ± 5.5
Sprint 3	17.0 ± 9.9	14.8 ± 7.2	19.5 ± 3.4	16.0 ± 5.8
Sprint 4	23.7 ± 11.8	20.9 ± 10.3	16.6 ± 8.5	19.3 ± 10.0
Average power ($\text{W}\cdot\text{kg}^{-1}$)	Passive	20%	30%	40%
Sprint 1	8.3 ± 0.5	8.4 ± 0.5	8.3 ± 0.5	8.3 ± 0.4
Sprint 2	7.3 ± 0.7	7.6 ± 0.7	7.3 ± 0.9	7.3 ± 0.5
Sprint 3	6.8 ± 0.9	7.0 ± 0.6	6.7 ± 0.6	7.0 ± 0.3
Sprint 4	6.4 ± 0.8	6.8 ± 0.7	6.8 ± 0.6	6.9 ± 0.5
AP drop relative to Sprint 1 (%) [#]	Passive	20%	30%	40%
Sprint 2	11.2 ± 5.0	10.4 ± 5.2	11.6 ± 7.9	12.0 ± 4.9
Sprint 3	17.5 ± 9.3	16.8 ± 4.5	18.5 ± 6.3	15.7 ± 5.6
Sprint 4 [§]	22.4 ± 8.9	19.9 ± 6.1	18.4 ± 7.3*	16.6 ± 6.2* [†]

Abbreviations: PP, peak power; AP, average power. Values are means ± SD. ^{##}Indicates sprint by condition interaction effect ($P < .01$). [#]Indicates sprint by condition interaction effect ($P < .05$). [§]Indicates main effect of condition during each sprint ($P < .05$). *Indicates $P < .05$ vs. passive recovery. [†]Indicates $P < .05$ vs. 20%. ^{‡‡}Indicates $P < .01$ vs. 30%.

Table 3 Blood lactate concentration across the sprints and recovery conditions.

Blood lactate (mmol·l ⁻¹)	Passive	20%	30%	40%
Pre-Sprint	1.6 ± 0.5	1.7 ± 0.5	1.5 ± 0.4	1.9 ± 0.3
Post-Sprint 1 at 180 sec***	10.7 ± 0.9	12.1 ± 2.1	11.3 ± 1.2	12.2 ± 1.9
Post-Sprint 2 at 180 sec***†††	14.0 ± 0.7	14.6 ± 2.5	14.6 ± 1.9	13.9 ± 1.1
Post-Sprint 3 at 180 sec***†††	14.8 ± 0.8	15.8 ± 2.0	14.7 ± 2.9	15.3 ± 1.7
Post-Sprint 4 at 180 sec***†††‡	15.0 ± 1.6	14.9 ± 0.7	15.8 ± 1.2	15.7 ± 1.6

Values are means ± SD. ***Indicates $P < .001$ vs. pre-sprint. †††Indicates $P < .001$ vs. post-sprint 1. ‡Indicates $P < .05$ vs. post-sprint 2.

Figure 1 – Percentage of $\dot{V}O_{2peak}$ (A), percentage of HRmax (B) and $\dot{V}O_2$ –to- sprint work ratio (C) across sprints. ***Indicates $P < .001$ vs. passive recovery. **Indicates $P < .01$ vs. passive recovery. *Indicates $P < .05$ vs. passive recovery. ††Indicates $P < .01$ vs. 20%. †Indicates $P < .05$ vs. 20%. Significant differences across the recovery conditions are only shown for clarity.

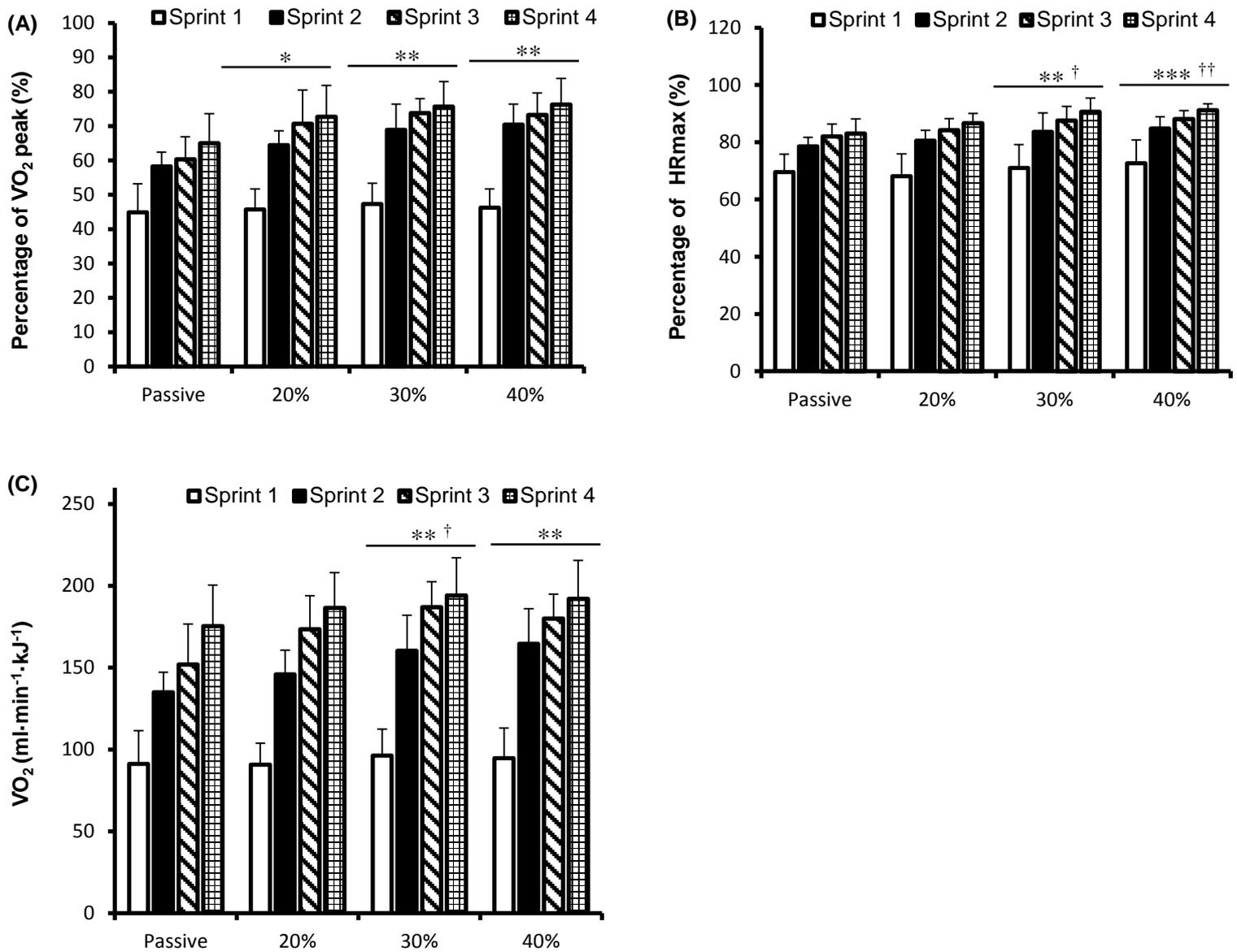


Figure 2 – Percentage of $\dot{V}O_{2peak}$ (A), percentage of HRmax (B) and time required to reach 50% $\dot{V}O_{2peak}$ (C) during recovery periods. ***Indicates $P < .001$ vs. passive recovery. **Indicates $P < .01$ vs. passive recovery. *Indicates $P < .05$ vs. passive recovery. †††Indicates $P < .001$ vs. 20%. ††Indicates $P < .01$ vs. 20%. †Indicates $P < .05$ vs. 20%. ‡Indicates $P < .05$ vs. 30%. Significant differences across the recovery conditions are only shown for clarity.

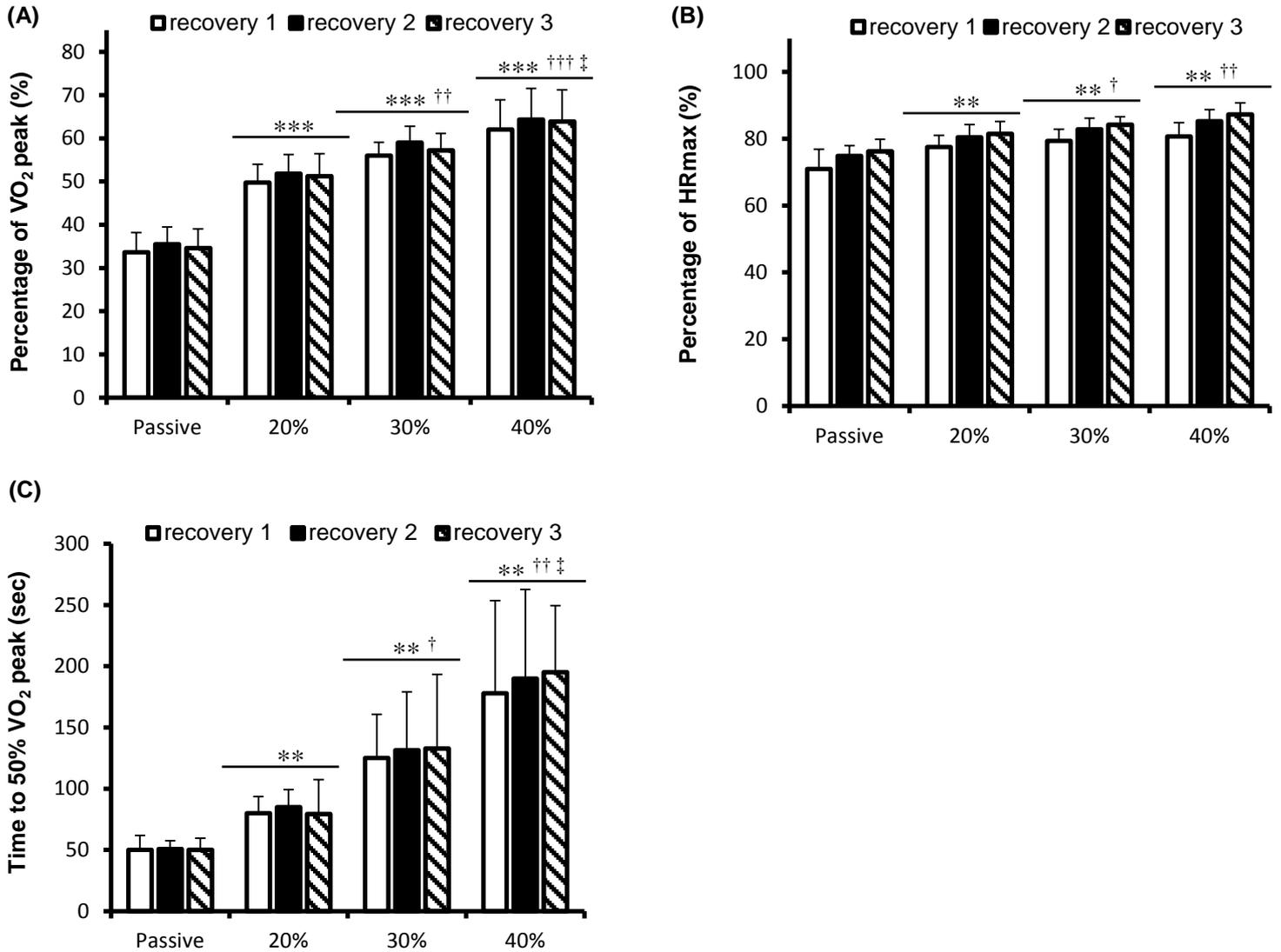


Figure 3 - Example of recovery kinetics of oxygen uptake (A) and HR (B) during recovery (group mean). Error bars are not shown for clarity.

