

Kent Academic Repository

Full text document (pdf)

Citation for published version

Nicolò, Andrea and Marcora, Samuele Maria and Bazzucchi, Ilenia and Sacchetti, Massimo (2017) Differential control of respiratory frequency and tidal volume during high-intensity interval training. *Experimental Physiology*, 102 (8). pp. 934-949. ISSN 0958-0670.

DOI

<https://doi.org/10.1113/EP086352>

Link to record in KAR

<https://kar.kent.ac.uk/62585/>

Document Version

Author's Accepted Manuscript

Copyright & reuse

Content in the Kent Academic Repository is made available for research purposes. Unless otherwise stated all content is protected by copyright and in the absence of an open licence (eg Creative Commons), permissions for further reuse of content should be sought from the publisher, author or other copyright holder.

Versions of research

The version in the Kent Academic Repository may differ from the final published version.

Users are advised to check <http://kar.kent.ac.uk> for the status of the paper. **Users should always cite the published version of record.**

Enquiries

For any further enquiries regarding the licence status of this document, please contact:

researchsupport@kent.ac.uk

If you believe this document infringes copyright then please contact the KAR admin team with the take-down information provided at <http://kar.kent.ac.uk/contact.html>

Differential control of respiratory frequency and tidal volume during high-intensity interval training

Andrea Nicolò¹, Samuele M. Marcora², Ilenia Bazzucchi¹ and Massimo Sacchetti¹

¹ Department of Movement, Human and Health Sciences, University of Rome “Foro Italico”,
Piazza Lauro De Bosis 6, Rome 00135, Italy

² Endurance Research Group, School of Sport and Exercise Sciences, University of Kent,
Chatham Maritime, Kent ME4 4AG, UK

Correspondence:

Massimo Sacchetti, PhD, Department of Movement, Human and Health Sciences, University
of Rome “Foro Italico”, Piazza Lauro De Bosis, 15 - 00135 Rome, Italy; Phone: +39
0636733281; Fax: +39 0636733214; E-mail: massimo.sacchetti@uniroma4.it

This is an Accepted Article that has been peer-reviewed and approved for publication in the
Experimental Physiology, but has yet to undergo copy-editing and proof correction. Please cite this
article as an Accepted Article; [doi: 10.1113/EP086352](https://doi.org/10.1113/EP086352).

This article is protected by copyright. All rights reserved.

Running Title: Control of respiratory frequency and tidal volume during HIIT

Keywords: Ventilatory pattern, effort, high-intensity interval training

Total number of words: 6375

Total number of references: 47

Subject area: Human/environmental and exercise physiology

New Findings

- **What is the central question of this study?**

By manipulating recovery intensity and exercise duration during high-intensity interval training (HIIT) we tested the hypothesis that fast inputs contribute more than metabolic stimuli to respiratory frequency (f_R) regulation.

- **What is the main finding and its importance?**

f_R , but not tidal volume, responds rapidly and in proportion to changes in workload during HIIT, and is dissociated from some markers of metabolic stimuli in both experimental manipulations, suggesting that fast inputs contribute more than metabolic stimuli to f_R regulation. Differentiating between f_R and tidal volume may help unravel the mechanisms underlying exercise hyperpnoea.

Accepted Article

This article is protected by copyright. All rights reserved.

Abstract

Since respiratory frequency (f_R) has been proposed as a good marker of physical effort, furthering the understanding of how f_R is regulated during exercise is of great importance. We manipulated recovery intensity and exercise duration during high-intensity interval training (HIIT) to test the hypothesis that fast inputs (including central command) contribute more than metabolic stimuli to f_R regulation.

Seven male cyclists performed an incremental test, a 10-min and a 20-min continuous time trial (TT) as preliminary tests. Subsequently, recovery intensity and exercise duration were manipulated during HIIT (30s work and 30s active recovery) by performing four 10-min and one 20-min trial (recovery intensities: 85%, 70%, 55% and 30% of the 10-min TT mean workload; 85% of the 20-min TT mean workload). The work intensity of the HIIT sessions was self-paced by participants to achieve the best performance possible.

When manipulating recovery intensity, f_R , but not tidal volume (V_T), showed a fast response to the alternation of the work and recovery phases, proportional to the extent of workload variations. No association between f_R and gas exchange responses was observed. When manipulating exercise duration, f_R and Rating of Perceived Exertion (RPE) were dissociated from V_T , $\dot{V}CO_2$ and $\dot{V}O_2$ responses. Overall, RPE was strongly correlated with f_R ($r=0.87$; $P<0.001$) but not with V_T .

These findings may reveal a differential control of f_R and V_T during HIIT, with fast inputs appearing to contribute more than metabolic stimuli to f_R regulation. Differentiating between f_R and V_T may help unravel the mechanisms underlying exercise hyperpnoea.

Abbreviations. fMRI, functional Magnetic Resonance Imaging; f_R , respiratory frequency; HIIT, high-intensity interval training; HR, heart rate; La^- , blood lactate; PET_{CO_2} , end tidal PCO_2 ; PPO, peak power output; RPE, rating of perceived exertion; TT, time trial; $\dot{V}CO_2$, carbon dioxide output; \dot{V}_E , minute ventilation; $\dot{V}O_2$, oxygen uptake; V_T , tidal volume; 10_85%, 10-min HIIT trial with 85% recovery intensity; 10_70%, 10-min HIIT trial with 70% recovery intensity; 10_55%, 10-min HIIT trial with 55% recovery intensity; 10_30%, 10-min HIIT trial with 30% recovery intensity; 20_85%, 20-min HIIT trial with 85% recovery intensity.

Introduction

Recent evidence suggests that respiratory frequency (f_R) is a valid marker of effort during cycling exercise (Nicolò *et al.*, 2014a, 2016). Unlike traditionally monitored physiological variables such as $\dot{V}O_2$, heart rate (HR) and blood lactate (La^-), f_R is strongly associated with Rating of Perceived Exertion (RPE) when manipulating both the work-to-rest ratio during high-intensity interval training (HIIT) (Nicolò *et al.*, 2014a) and exercise duration during continuous time trials (TT) (Nicolò *et al.*, 2016). Moreover, RPE and f_R respond in a similar way to several experimental interventions that affect performance such as muscle fatigue (Marcora *et al.*, 2008), muscle damage (Davies *et al.*, 2009), increases in body temperature (Hayashi *et al.*, 2006) and hypoxia (Koglin & Kayser, 2013).

The strong experimental link found between perceived exertion and f_R suggests that the two variables may share a common regulation mechanism. It was proposed that f_R and perceived exertion are at least partially regulated by central command (Nicolò *et al.*, 2016), i.e. the activity of motor and premotor areas of the brain relating to voluntary muscle contractions (de Morree *et al.*, 2012). Strong indirect evidence in favour of central command being the sensory signal for perceived exertion comes from exercise studies using partial blockade of sensory signals from skeletal muscle afferents, because they show that RPE is unchanged or even augmented with partial sensory blockade (Fernandes *et al.*, 1990; Kjaer *et al.*, 1999). Further evidence comes from recent electroencephalographic data showing a relationship between RPE and movement-related cortical potentials – a direct measure of central command – in a series of experimental manipulations (de Morree *et al.*, 2012, 2014; Berchicci *et al.*, 2013), and the strongest evidence comes from the observation that the

experimental disruption of the activity of cortical areas upstream of primary motor cortex affects perceived exertion (Zénon *et al.*, 2015).

Central command, together with signals from muscle afferent fibres and metabolic stimuli, is a major regulator of ventilation during exercise (see Forster *et al.* (2012) for a detailed review). These inputs on ventilation act with different timings when abrupt changes in exercise workload occur. While the rapid increase in ventilation at the beginning of exercise is accounted for by fast inputs like central command and afferent feedback, the contribution of metabolic stimuli to ventilation is delayed (Duffin, 2014). Despite the regulation of f_R and tidal volume (V_T) being less studied than the regulation of minute ventilation (\dot{V}_E), some findings suggest that the putative inputs driving ventilation may act separately on f_R and V_T . Isolating the effect of central command on cardiorespiratory responses by imagining exercise under hypnosis, Thornton *et al.* (2001) found an increase in f_R , with no change of V_T . Bell & Duffin (2006) reported an immediate response of f_R , but not of V_T , in the transition from rest to exercise (and vice versa) and in the transition from passive to active leg extension exercise, where the increase in ventilation is largely regulated by central command. Hence the hypothesis that central command could play a role in the regulation of f_R , but not of V_T (Nicolò *et al.*, 2015). However, as seen in the aforementioned studies, the challenge to experimentally isolate the contribution of central command to ventilation from that of other inputs has often resulted in experimental conditions imperfectly replicating the true physiological state of exercise, particularly the hyperpnoea of heavy exercise (Sheel & Romer, 2012). Consequently, little is known on the regulation of f_R and V_T during high-intensity exercise.

Instead of attempting to isolate the effect of central command on ventilation, in our study we aimed to test the hypothesis that during high-intensity exercise fast inputs (including central command) contribute more than metabolic stimuli to f_R regulation. We used HIIT as an appropriate exercise paradigm to test this hypothesis, in view of both the nature and practical relevance of this type of exercise. Indeed, abrupt changes in workload, as in HIIT, have the potential to partially dissociate the contribution of fast and slow inputs to ventilation. Besides, HIIT is a widely used and recommended training modality, but our understanding of the ventilatory responses to HIIT is limited. Specifically, we manipulated both recovery intensity and exercise duration during HIIT, while measuring self-selected work intensity, RPE and ventilatory and metabolic responses. We expected the manipulation of recovery intensity to determine systematic variations in workload across trials, probably accompanied by proportional variations in the magnitude of central command (Siemionow *et al.*, 2000; Dai *et al.*, 2001). Based on previous findings during continuous exercise (Nicolò *et al.*, 2016), we expected the manipulation of exercise duration to dissociate f_R and RPE from V_T and from some markers of metabolic stimuli driving ventilation. Together, the two experimental manipulations aimed to provide further insight into the regulation of f_R and V_T during HIIT, with potentially important implications for the monitoring of f_R as a marker of effort during exercise.

Methods

Ethical approval

This study was approved by the Ethics Committee of the University of Rome Sapienza in compliance with the *Declaration of Helsinki*. Written informed consent was obtained from all participants.

Subjects

Seven male participants (mean \pm SD: age 24 ± 3 years, stature 1.77 ± 0.04 m, body mass 68 ± 7 kg) volunteered to participate in this study. They were well-trained competitive cyclists (De Pauw *et al.*, 2013) with a minimum of 5 years' cycling experience and 250 km training per week. Participants were asked to refrain from strenuous exercise, consumption of alcohol and caffeine for at least 24 h before each test.

Experimental overview

All testing was completed in the laboratory with a room temperature of 19–21°C and at the same time of day (\pm 1h). Participants reported to the laboratory on 8 separate occasions over a five-week period, with visits separated by at least 48 hours. On the first visit, participants performed a preliminary ramp incremental exercise test, followed by a familiarization trial. During visits 2 and 3, they performed a 10-min and a 20-min continuous TT in random order. On the following visits participants performed 5 experimental HIIT trials in random order on

separate days. These consisted of four 10-min and one 20-min HIIT trial with different recovery intensities (i.e. 85%, 70%, 55% and 30% of the 10-min continuous TT mean power output, and 85% of the 20-min continuous TT mean power output). A standardized warm-up was performed before the two continuous TTs and the five HIIT trials. This consisted of 3 min at 100 W, 6 min at 50% of the peak power output (PPO) reached in the incremental test, 1 min at 60% of the PPO, and 1 min at 100 W. Trials were then preceded by 3 min of rest and 3 min pedalling at 20 W. All the protocols were performed on an electromagnetically-braked cycle ergometer (Lode Excalibur Sport, Groningen, the Netherlands). The position of the seat and handle bar on the ergometer was adjusted and recorded for each participant during the first visit to be reproduced in the following visits.

Ramp incremental test

Before the incremental test was performed, participants were presented with the Borg 6–20 RPE scale, and instructions were given according to established recommendations (Borg, 1998). During the ramp incremental test, participants were asked to point out their perceived exertion on the RPE scale every minute during exercise and immediately after exhaustion. Perceived exertion rating during this test served as a familiarization with the scale, and RPE values were not included in subsequent data analysis.

The ramp incremental test to exhaustion was preceded by a 5 min warm-up at 100 W, 3 min of rest and 3 min pedalling at 20 W. The test consisted of a continuous ramped increase in work rate of $30 \text{ W} \cdot \text{min}^{-1}$, starting from 20 W. Preferred pedalling cadence was selected by each participant and was kept constant throughout the test which terminated when cadence

fell by more than 10 rpm despite strong verbal encouragement. $\dot{V}O_2$ peak was defined as the highest value of a 30-s average, and PPO as the highest power output achieved at exhaustion, registered to the nearest 1 W. After recovering from the incremental test, participants were familiarized with the linear mode of the ergometer used in the continuous TTs and the HIIT protocols, as previously described (Nicolò *et al.*, 2014a).

Continuous time trials

A 10-min and a 20-min TT were performed in random order according to previously reported procedures (Nicolò *et al.*, 2014a, 2016). Briefly, participants were asked to self-pace the power output to achieve the maximal mean power output possible in each trial. With the exception of elapsed time and time to be completed, no feedback on performance or physiological measurements and no encouragement was given to participants, to minimize external factor influence (Currell & Jeukendrup, 2008). The mean power output of the two TTs was used to set the recovery power output for the HIIT trials as detailed below.

Experimental HIIT trials

Four 10-min and one 20-min HIIT trial were performed in random order. The same work-recovery cycle of 60 s (30 s work and 30 s active recovery) was selected for all the trials. The HIIT recovery intensity was fixed throughout each trial, but differed across the four 10-min trials, corresponding to 85% (10_85%), 70% (10_70%), 55% (10_55%) and 30% (10_30%) of the 10-min continuous TT mean power output. The recovery intensity of the 20-min HIIT

trial corresponded to 85% (20_85%) of the 20-min continuous TT mean power output.

During all HIIT trials, the work intensity was self-paced by participants in order to achieve the best performance possible (i.e. the highest average power output). To this end, participants were allowed to choose the most effective pacing strategy, and this choice was not influenced by the experimenter. During the recovery phases, the ergometer was set in the hyperbolic mode (cadence independent mode), which fixes the power output irrespective of the cadence selected by the participant. This is a convenient ergometer modality for manipulating recovery intensity. Instead, during the work phases, the ergometer was set in the linear mode (cadence dependent mode), where changes in power output are determined by changes in pedalling cadence. This is a convenient ergometer modality for allowing the participant to self-pace the work-intensity during HIIT, as previously reported (Nicolò *et al.*, 2014a).

The exercise prescription approach used in the present study, previously defined as “isoeffort” and “isotime” (Nicolò *et al.*, 2014a), is an important feature of our experimental design. This approach guarantees the same exercise duration and a similar effort across different exercise protocols. Therefore, it allowed for the investigation of the effect of recovery intensity manipulation on ventilatory responses, while excluding the effect of potentially confounding factors such as session effort and exercise duration. A similar between-trial effort was also guaranteed when investigating the effect of exercise duration on ventilatory responses, by asking participants to achieve the best performance possible in both the 10_85% and the 20_85% trial. Given that the work intensity was self-paced, we tested experienced cyclists to ensure correct execution of the trials. During all trials, with the exception of elapsed time and time to be completed, no feedback on performance or

physiological measurements and no encouragement was given to participants, to minimize external factor influence (Currell & Jeukendrup, 2008). On the other hand, participants were informed on both absolute and relative (% of the continuous TT) recovery power output prior to performing each trial, because during HIIT with active recovery the sustainable work intensity is influenced by recovery intensity. Specifically, self-selected work intensity was expected to increase with the decrease in recovery intensity, thus progressively enhancing the difference between work and recovery power output across trials. These expected systematic changes in workload across trials make the experimental design of the present study suitable to verify whether f_R changes in proportion to variations in workload during HIIT. All the HIIT trials started with the work phase. Breath-by-breath data, RPE and La^- were measured during HIIT as reported below.

Measurements

Pulmonary gas exchange, \dot{V}_E , f_R , V_T , end-tidal PCO_2 (PET_{CO_2}) and HR were measured breath-by-breath using a metabolic cart (Quark b2, Cosmed, Rome, Italy). Appropriate calibration procedures were performed following the manufacturer's instructions.

Capillary blood samples were drawn from the earlobe and lactate was measured by a portable lactate analyser (Lactate Plus, Nova Biomedical, USA). Blood lactate was collected every 2 min during the 10-min HIIT trials and every 4 min during the 20_85% trial, in order to express values for every 20% of relative exercise duration.

Data analysis

To account for varying kinetics of physiological variables due to work and recovery phase alternation characterizing HIIT, variables were expressed as a function of the work-recovery cycle (complete cycle including work and a recovery phase) as previously described (Nicolò *et al.*, 2014b). Briefly, breath-by-breath data were averaged over 10 seconds, and the 60-s work-recovery cycle (30 s work and 30 s active recovery) was subdivided into 6 parts of 10 seconds each. To reduce the influence of the kinetics of physiological variables at the start of exercise, data from the first minute of exercise were removed from this analysis.

To investigate the time course of physiological variables, values from work and recovery phases were averaged together. When the time course of physiological variables was normalized to relative exercise duration, values from the 20_85% protocol were averaged over 2 min. Values from work and recovery phases were also averaged together when correlating f_R , \dot{V}_E and V_T with RPE. Since RPE was measured at discrete time points (every minute during exercise, immediately after the end of each work phase), values collected every 2 min were considered when normalizing the RPE values of the 20_85% protocol to relative exercise duration. For all the trials, the first data point (first 10% of the trial) was not included when correlating ventilatory variables with RPE.

For all the physiological variables reported in table 1 and all the HIIT trials, the peak value was defined as the highest value of a 60-s average.

Statistical analysis

An a priori power analysis was performed using G*Power (version 3.1.9.2; Kiel University, Kiel, Germany). Expecting a large effect size for the effect of recovery intensity manipulation on the response of f_R within the work-recovery cycle, a sample size of 6 was required based on $1-\beta = 0.80$ and $\alpha = 0.05$. Seven participants were recruited to account for potential drop out.

Statistical analysis was conducted using IBM SPSS Statistics 20 (SPSS Inc, Chicago, Illinois, USA). Data were checked for normality prior to analysis. A Student's t-test was used to compare mean and peak values of mechanical and physiological variables of the 10_85% with those of the 20_85% trial, while a one-way repeated-measures ANOVA was used to compare mechanical and physiological variables of the four 10-min HIIT trials. In the case of a significant effect of trial, the Bonferroni test was used for follow-up analysis. A two-way repeated-measures ANOVA (trial x time) was used to analyse the effect of trial on mechanical, physiological and perceptual variables as a function of absolute (when comparing the four 10-min HIIT trials) and relative (when comparing the 10_85% with the 20_85% trial) exercise duration. A two-way repeated-measures ANOVA (trial x time) was also used to analyse the effect of recovery intensity manipulation on work-recovery-cycle physiological responses. When the sphericity assumption was violated, the Greenhouse-Geisser adjustment was performed. Partial eta squared (η_p^2) effect sizes were calculated; an effect of $\eta_p^2 \geq 0.01$ indicates a small, $\eta_p^2 \geq 0.059$ a medium, and $\eta_p^2 \geq 0.138$ a large effect (Cohen, 1988).

Within-subjects correlation coefficients (r) were computed for the correlations between RPE and f_R , RPE and \dot{V}_E and RPE and V_T , using the method described by Bland & Altman (1995). This method adjusts for repeated observations within participants, by using multiple regression with “participant” treated as a categorical factor using dummy variables. A correlation coefficient and a P value were obtained considering the five HIIT trials together, as well as for each trial considered separately. A P value < 0.05 was considered statistically significant in all analyses. The results are expressed as mean \pm SD in text and table and as mean \pm SE in figures.

Results

The $\dot{V}O_{2\text{peak}}$ and the PPO measured during the ramp incremental test were 4366 ± 412 $\text{mL}\cdot\text{min}^{-1}$ (65 ± 6 $\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) and 437 ± 38 W respectively. The mean power output of the 10-min and 20-min continuous TTs was 344 ± 35 W and 311 ± 38 W respectively.

Table 1 reports mean and peak values of mechanical and physiological variables for the five HIIT trials. When manipulating the recovery intensity (comparing the four 10-min HIIT trials), an effect of trial ($P < 0.038$, $\eta_p^2 > 0.368$) was found for power output_{mean}, power output_{work}, total work, cadence_{mean}, $\dot{V}O_2$, and HR_{peak}, but not for the other variables. When manipulating the exercise duration (comparing the 10_85% with the 20_85%) an effect of trial ($P < 0.033$, $\eta_p^2 > 0.509$) was found for all variables except for cadence, $\dot{V}O_2$, $\dot{V}_{E\text{mean}}$ and f_R . No significant difference was found when comparing the $V_{T\text{peak}}$ of the incremental test (3.2 ± 0.5) with the $V_{T\text{peak}}$ of any of the four 10-min HIIT trials.

Figure 1 reports the time course of power output for the five HIIT trials. For the 10-min HIIT trials, a decrease in self-paced work intensity was observed with the increase in recovery intensity. Figure 2 reports the time course of f_R and V_T for the four 10-min HIIT trials. f_R showed a fast response to the alternation of work and recovery phases, proportional to the extent of workload variations across trials, while modest changes, opposite to the alternation of work and recovery phases, were observed for V_T . Of note, a difference in response of f_R and V_T can be observed already in the very first work-recovery cycle, where an increase and decrease in f_R consistent with the alternation of the work and recovery phases was observed, while V_T increased throughout the first minute of exercise, and even during recovery. The responses of f_R and V_T were analysed considering either the responses within

the work-recovery cycle (figure 3) or the time course (figure 4) as described below. Changes in power output, ventilatory variables, gas exchange variables and HR within the work-recovery cycle are reported in figure 3. Unlike for f_R , a delayed response to the alternation of work and recovery phases was found for most of the variables. An interaction ($P < 0.001$, $\eta_p^2 > 0.387$) was observed for all the variables reported except for V_T ($P = 0.062$). An effect of time ($P < 0.002$, $\eta_p^2 > 0.566$) was observed for all variables, while an effect of trial ($P < 0.037$, $\eta_p^2 > 0.369$) was only observed for $\dot{V}O_2$ and HR.

Figure 4 depicts the time course of RPE and ventilatory variables for the four 10-min trials (A, C, E and G), and for 10_85% and 20_85% expressed as a % of relative exercise duration (B, D, F and H). When manipulating the recovery intensity, neither interaction nor main effect of trial was found for any variable, but they all showed a main effect of time ($P < 0.001$, $\eta_p^2 > 0.768$). For RPE, a statistical trend ($P = 0.067$) was observed for the main effect of trial. When manipulating the exercise duration, an interaction ($P < 0.001$, $\eta_p^2 > 0.476$) was found for \dot{V}_E and V_T , but not for RPE and f_R . A main effect of trial was only found for V_T ($P < 0.001$, $\eta_p^2 = 0.891$), while a main effect of time ($P < 0.001$, $\eta_p^2 > 0.719$) was found for all variables.

Figure 5 depicts the time course of $\dot{V}O_2$, $\dot{V}CO_2$, P_{ETCO_2} and HR for the four 10-min HIIT trials (A, C, E and G), and for 10_85% and 20_85% expressed as a % of relative exercise duration (B, D, F and H). When manipulating the recovery intensity, an interaction ($P < 0.002$, $\eta_p^2 > 0.276$) was observed for all variables, except for P_{ETCO_2} . A main effect of trial ($P = 0.004$, $\eta_p^2 = 0.516$) was found for $\dot{V}O_2$, but not for the other variables (HR, $P = 0.099$), while a main effect of time ($P < 0.001$, $\eta_p^2 > 0.883$) was found for all variables. When

manipulating the exercise duration, an interaction ($P < 0.001$, $\eta_p^2 > 0.687$) was found for $\dot{V}O_2$, $\dot{V}CO_2$ and HR, as well as for P_{ETCO_2} ($P = 0.014$, $\eta_p^2 = 0.302$). An effect of trial ($P < 0.034$, $\eta_p^2 > 0.559$) was found for $\dot{V}CO_2$ and HR, but not for $\dot{V}O_2$ and P_{ETCO_2} , while an effect of time ($P < 0.001$, $\eta_p^2 > 0.843$) was found for all variables. When manipulating the recovery intensity, neither interaction nor main effect of trial, but a significant effect of time ($P < 0.001$, $\eta_p^2 = 0.885$), was found for La^- (figure 6, panel A). When manipulating the exercise duration, a statistical trend towards an interaction was found for La^- (figure 6, panel B) after applying the Greenhouse-Geisser correction ($P = 0.074$), with a main effect of time ($P < 0.001$, $\eta_p^2 = 0.844$), but no main effect of trial.

Figure 7 depicts the correlations between RPE and f_R , RPE and \dot{V}_E and RPE and V_T , either when considering the four 10-min intermittent trials (panel A) or 10_85% and 20_85% (panel B). Correlation coefficients and P values of these correlations are reported in table 2. When the five intermittent trials were considered together, RPE was significantly correlated ($P < 0.001$) with f_R ($r = 0.87$) and \dot{V}_E ($r = 0.80$), but not with V_T ($r = -0.11$; $P = 0.053$). When the intermittent trials were considered separately, significant correlations were found between RPE and both f_R and \dot{V}_E , but generally not between RPE and V_T .

Discussion

The present study clearly shows different responses of f_R and V_T during HIIT. Specifically, the main findings of the study are as follows: 1) When manipulating recovery intensity, f_R ,

but not V_T , showed a fast response to the alternation of the work and recovery phases, which was proportional to the extent of workload variations; 2) When manipulating exercise duration, f_R and RPE responses were dissociated from those of V_T and some markers of metabolic stimuli driving ventilation; 3) A similar time course and a strong correlation were found between f_R and RPE in all the conditions tested, while generally no correlation was found between V_T and RPE. These findings are in line with our hypothesis that fast inputs (possibly including central command) contribute more than metabolic stimuli to f_R regulation.

The abrupt changes in workload characterizing HIIT determined a fast increase and decrease in f_R at the beginning of the work and recovery phases respectively, unlike that for any other physiological variable investigated. This was particularly evident in the trial with the larger difference between work and recovery intensity (10_30%), and in line with what was found in a previous study (Nicolò *et al.*, 2014b). Furthermore, variations in f_R to the alternation of the work and recovery phases were proportional to the extent of workload variations (figure 3), and thus presumably to changes in the magnitude of central command. Indeed, higher mechanical workload is accompanied by greater movement-related cortical potentials (Siemionow *et al.*, 2000; de Morree *et al.*, 2012) and functional Magnetic Resonance Imaging (fMRI) signals (Dai *et al.*, 2001). Nonetheless, it has to be acknowledged that muscle afferent feedback may also vary proportionally with workload, and is known to contribute to the fast response of f_R (Bell & Duffin, 2006). While both muscle afferent feedback and central command likely contributed to the fast response of f_R observed herein, the ecological experimental design used makes it difficult to understand their relative contributions, and consequently, this is beyond the scope of the present study. A delayed response of $\dot{V}O_2$ and $\dot{V}CO_2$ to the alternation of the work and recovery phases was observed,

thereby showing a dissociation between f_R and some indicators of metabolic stimuli driving ventilation. Opposite to f_R , V_T showed a modest decrease and increase at the beginning of the work and recovery phases, respectively. Furthermore, the time course of V_T was similar across different trials, resembling to some extent the time course of $\dot{V}CO_2$.

When considering the overall time course, the link between f_R and physical effort appears to be reinforced. Given the “isoeffort” and “isotime” prescription of the four 10-min HIIT sessions, a similar effort was expected across trials. In line with this, no significant between-trial differences were found for RPE and f_R . For both variables a similar and progressive increase over time was observed, and this is a feature of fatiguing exercise, as also found in other experimental conditions (Nicolò *et al.*, 2014a, 2016). Between-trial differences were, however, observed for HR and $\dot{V}O_2$. The manipulation of exercise duration further revealed an association between f_R and physical effort. Indeed, when values were normalized to relative exercise duration (an analysis that equates performance trials differing in exercise duration in terms of effort (Nicolò *et al.*, 2016)), no significant between-trial differences in f_R and RPE were found. Conversely, \dot{V}_E , V_T , HR, $\dot{V}CO_2$, $\dot{V}O_2$, and La^- (only a trend) were lower in the longest trial (20_85%). Therefore, the responses of f_R and RPE were dissociated from the responses of V_T and some markers of metabolic stimuli driving ventilation, in line with what has previously been found during continuous time trials of different duration (Nicolò *et al.*, 2016). Considering the two experimental manipulations together, a strong correlation was found between RPE and f_R in all the conditions tested, while generally no correlation was found between RPE and V_T . This confirms and extends previous findings showing a strong relationship between f_R and RPE in different exercise paradigms (Noble *et al.*, 1973; Nicolò *et al.*, 2014a, 2016).

Although the present design allows for a partial understanding of the mechanisms underlying the regulation of f_R and V_T during high-intensity exercise, our findings suggest the relevance of looking at the link between effort and f_R regulation. This supports the possible contribution of central command to f_R regulation, central command being defined in the cardiovascular field as an “effort-induced modulation of autonomic function” (Williamson *et al.*, 2006). This definition was proposed because of the strong link between perceived effort – rather than exercise intensity per se – and the central-command mediated cardiovascular response (Williamson *et al.*, 2006). From this perspective, not only the proportional variation of f_R to work and recovery intensity alternation, but also the f_R increase over time found in all the HIIT trials may have been partially driven by central command. That central command may have a role in the increase in f_R over time is supported by the increase in movement-related cortical potentials (Berchicci *et al.*, 2013; de Morree *et al.*, 2014) and fMRI signals (Liu *et al.*, 2003) during constant workload fatiguing trials. Alternatively, the increase in f_R over time may have been influenced by the well-documented increase in body temperature during fatiguing trials (González-Alonso *et al.*, 1999), given the strong association between body temperature and tachypnoea during exercise (Hayashi *et al.*, 2006). Conversely, the fast response of f_R to the work and recovery alternation can hardly be explained by changes in body temperature, which shows a substantially delayed response compared to variations in power output (Todd *et al.*, 2014). In fact, the influence of body temperature and central command on f_R is not mutually exclusive, since the increase in body temperature may lead to an increase in the activity of central command (Hayashi *et al.*, 2006).

The similar V_T responses observed across the four 10-min HIIT trials (figure 2) suggest the occurrence of the V_T plateau phenomenon, which is usually documented during

maximal incremental exercise (Sheel & Romer, 2012). This phenomenon is often attributed to mechanical constraints that can be observed at relatively high levels of \dot{V}_E , and the tachypnoeic breathing pattern that occurs is supposed to be a consequence of the attainment of maximal V_T values (Sheel & Romer, 2012). Accordingly, we cannot exclude that mechanical constraints could have partially prevented V_T from increasing further and thus from changing in proportion to variations in workload during HIIT. This may be supported by the fact that the V_{Tpeak} registered during the incremental test was not significantly different from any of the V_{Tpeak} values found in the four 10-min HIIT trials. However, the V_T plateau is a complex phenomenon and the underlying mechanisms are not well understood. Some findings (Martin & Weil, 1979; McParland *et al.*, 1991; Fan *et al.*, 2012) but not others (Gallagher *et al.*, 1987) suggest that V_T stabilizes before the attainment of truly maximal V_T levels. For instance, Martin & Weil (1979) suggested that V_T is lowered by exercise-induced hypocapnia, because the V_T plateau observed during a maximal incremental test was raised when CO_2 was added to the inspired gas for maintenance of isocapnia. Similar findings were reported more recently by Fan *et al.* (2012). Therefore, the decrease in P_{ETCO_2} observed during the four 10-min HIIT trials, which indicates progressive development of hypocapnia, may have contributed to stabilizing V_T in the present study. However, this explanation is not supported by the findings of Gallagher *et al.* (1987). The fact that V_T was similar in the four 10-min HIIT trials apparently contrasts with the relatively low values of $\dot{V}O_2$ and $\dot{V}CO_2$ found in the 10_30% trial. On the other hand, the 10_30% trial showed the highest work-intensity power output and the highest La^- (no significant differences across trials for La^-). A similar picture was found by Nicolò *et al.* (2014a) when using the “isoeffort” and “isotime” approach to compare continuous and different HIIT trials with a session duration of 30 min.

The experimental design used in that study resulted in substantial differences in $\dot{V}O_2$, $\dot{V}CO_2$ and La^- across trials. However, similar responses of f_R , V_T and \dot{V}_E were found, and the HIIT trial with the lowest values of $\dot{V}O_2$ and $\dot{V}CO_2$ had the highest values of work-intensity power output and La^- , as in the present study. Therefore, it is conceivable that V_T may be partially driven by metabolic stimuli associated with high values of La^- , such as relatively low values of blood pH, and this is supported by mechanistic studies on animals (Borison *et al.*, 1977, 1978). The tachypnoeic breathing pattern observed by Nicolò *et al.* (2014a) was not due to the attainment of maximal V_T values, because V_T has been shown to be considerably lower during a 30-min maximal effort trial compared to a 10-min trial (Nicolò *et al.*, 2016). Likewise, a tachypnoeic breathing pattern was found during a high-intensity constant workload trial despite the fact that V_T values were lower than those observed during a maximal incremental test (Syabbalo *et al.*, 1994). Other evidence suggests the existence of a differential control of V_T and f_R that is not dependent on the attainment of maximal levels of V_T during high-intensity exercise. Looking at the very first work-recovery cycle of the four 10-min HIIT trials (figure 2) where \dot{V}_E values were below $110 \text{ L}\cdot\text{min}^{-1}$, different responses of V_T and f_R can already be observed. f_R increased during the work phase and decreased during the recovery phase. Conversely V_T increased throughout the first minute of exercise even during recovery, possibly driven by metabolic stimuli. A similar ventilatory pattern was documented during 10-s impulse bouts of exercise, showing a fast increase in f_R and a delayed increase in V_T and some metabolic stimuli, with \dot{V}_E reaching relatively low values (Bakker *et al.*, 1980; Miyamoto *et al.*, 1983).

Our interpretation that central command may contribute to regulating f_R but not V_T is in line with findings from previous studies that have attempted to isolate the effect of central

command on ventilatory responses. When asking participants to imagine heavy exercise under hypnosis, an increase in f_R and HR, with no change in V_T , was observed (Thornton *et al.*, 2001). At the end of the imagined exercise, a fast decrease in f_R and a delayed decrease in HR occurred, while an increase in V_T and P_{ETCO_2} was observed (Thornton *et al.*, 2001). It is impressive how these responses resemble those found in the present study during actual exercise despite the huge difference between the experimental conditions. A fast response of f_R with no substantial change in V_T was observed both at the beginning and at the end of a moderate-intensity exercise bout (Bell & Duffin, 2006). Similar responses were also observed in the transition from passive to active exercise, where central command is a major drive to breathe (Bell & Duffin, 2006). During high-intensity continuous exercise, an increase in central command, secondary to exercise-induced muscle fatigue (Marcora *et al.*, 2008) and epidural anaesthesia (Amann *et al.*, 2008), determined an increase in f_R with a mild decrease in V_T compared to control conditions. Furthermore, the reported increase in f_R was independent of muscle afferent feedback, which was either reduced because of epidural anaesthesia (Amann *et al.*, 2008) or unchanged because of a fatiguing protocol specifically designed to induce muscle fatigue without affecting afferent neural feedback related to metabolic stress (Marcora *et al.*, 2008). That central command can affect f_R independently of muscle afferent feedback, does not imply that afferent feedback does not contribute to f_R regulation. Conversely, the increase in f_R during passive exercise (Bell & Duffin, 2006), and the decrease in f_R with partial blockade of muscle afferent feedback during active exercise (Amann *et al.*, 2010) clearly indicate that afferent feedback plays a role in regulating f_R . Nonetheless, the relative contribution of afferent feedback to f_R regulation was found to be lower at high exercise intensity compared to moderate intensity (Amann *et al.*, 2010).

Our interpretation that f_R is not largely regulated by metabolic stimuli, unlike V_T , appears even more convincing in the light of evidence of a differential control of f_R and V_T in various experimental conditions. When voluntarily controlling f_R , Haouzi & Bell (2009) elegantly showed that there is an inherent, fundamental mechanism that, according to the background level of CO_2 and metabolic rate, elaborates V_T based on f_R to keep alveolar ventilation constant. This was consistent irrespective of the experimental conditions tested, that is, increased dead space, hypercapnia and light exercise (Haouzi & Bell, 2009). Interestingly, Ohashi *et al.* (2013) extended these findings and found that at rest CO_2 homeostasis is impaired when voluntarily controlling V_T rather than f_R , suggesting that f_R is regulated by non-metabolic factors. Furthermore, metabolic stimuli known to increase ventilation such as hypercapnia and hypoxia have been reported to act primarily on V_T . With a progressive increase in hypercapnia, V_T increases first but f_R does not increase until hypercapnia reaches a significant level (Duffin *et al.*, 2000). A substantial increase in V_T , with negligible changes in f_R , was also found in response to an abrupt change of inspired CO_2 (Gelfand & Lambertsen, 1973). During intermittent hypoxia, an increase in V_T , with no change in f_R , was observed (Mateika *et al.*, 2004).

When attempting to understand the mechanisms underlying exercise hyperpnoea, ecological human studies are subject to important limitations that have to be acknowledged. The present design did not attempt to isolate the contribution of the putative inputs regulating ventilation. Besides, the use of self-paced HIIT performance trials limited the possibility of including measurements of some relevant physiological variables like pH, potassium, bicarbonate and the arterial partial pressures of O_2 and CO_2 . Therefore, the present findings cannot exclude that some of these metabolic stimuli may have played a role in regulating f_R .

Nevertheless, the limitations of ecological studies should not discourage researchers to elaborate experimental designs aiming to investigate ventilatory control during high-intensity exercise. Indeed, our findings highlight the relevance of conducting such studies, as well as the importance of manipulating exercise paradigms as experimental intervention. Given the physiological and practical value of monitoring f_R as a marker of physical effort, further investigations aiming to improve our understanding of the ventilatory control during high-intensity performance trials are needed.

Conclusions

We manipulated recovery intensity and exercise duration during HIIT to further our understanding of the regulation of f_R and V_T during high-intensity exercise, with a special interest in the link between f_R and physical effort. When manipulating recovery intensity, f_R , but not V_T , responded rapidly and in proportion to changes in workload. Furthermore, both experimental manipulations used managed to dissociate the responses of f_R and RPE from those of V_T and some markers of metabolic stimuli driving ventilation. These findings may reveal a differential control of f_R and V_T during HIIT, with fast inputs (possibly including central command) appearing to contribute more than metabolic stimuli to f_R regulation. The understanding of the proportional contribution of central command and muscle afferent feedback to f_R regulation was beyond the scope of the present study, and thus requires further investigation. Our findings underline the importance of differentiating between f_R and V_T , which may help unravel the mechanisms underlying exercise hyperpnoea. From a practical

perspective, the monitoring of f_R as a marker of effort is further encouraged by the present findings.

Additional information***Competing interests***

None declared.

Author contributions

Conception or design of the work: A.N. and M.S. Acquisition, analysis or interpretation of data for the work: A.N., S.M.M., I.B. and M.S. Drafting the work or revising it critically for important intellectual content: A.N., S.M.M., I.B. and M.S. All authors approved the final version of the manuscript and agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All persons designated as authors qualify for authorship, and all those who qualify for authorship are listed.

Funding

The present study received no external funding.

This is an Accepted Article that has been peer-reviewed and approved for publication in the Experimental Physiology, but has yet to undergo copy-editing and proof correction. Please cite this article as an Accepted Article; [doi: 10.1113/EP086352](https://doi.org/10.1113/EP086352).

This article is protected by copyright. All rights reserved.

References

- Amann M, Blain GM, Proctor LT, Sebranek JJ, Pegelow DF & Dempsey JA (2010). Group III and IV muscle afferents contribute to ventilatory and cardiovascular response to rhythmic exercise in humans. *J Appl Physiol* **109**, 966–976.
- Amann M, Proctor LT, Sebranek JJ, Eldridge MW, Pegelow DF & Dempsey JA (2008). Somatosensory feedback from the limbs exerts inhibitory influences on central neural drive during whole body endurance exercise. *J Appl Physiol* **105**, 1714–1724.
- Bakker HK, Struikenkamp RS & De Vries GA (1980). Dynamics of ventilation, heart rate, and gas exchange: sinusoidal and impulse work loads in man. *J Appl Physiol* **48**, 289–301.
- Bell HJ & Duffin J (2006). Rapid increases in ventilation accompany the transition from passive to active movement. *Respir Physiol Neurobiol* **152**, 128–142.
- Berchicci M, Menotti F, Macaluso A & Di Russo F (2013). The neurophysiology of central and peripheral fatigue during sub-maximal lower limb isometric contractions. *Front Hum Neurosci* **7**, 135.
- Bland JM & Altman DG (1995). Calculating correlation coefficients with repeated observations: Part 1--Correlation within subjects. *BMJ* **310**, 446.
- Borg G (1998). Borg's perceived exertion and pain scales. Human Kinetics, Champaign.
- Borison HL, Gonsalves SF, Montgomery SP & McCarthy LE (1978). Dynamics of respiratory VT response to isocapnic pHa forcing in chemodenervated cats. *J Appl*

Physiol **45**, 502–511.

Borison HL, Hurst JH, McCarthy LE & Rosenstein R (1977). Arterial hydrogen ion versus CO₂ on depth and rate of breathing in decerebrate cats. *Respir Physiol* **30**, 311–325.

Cohen J (1988). Statistical power analysis for the behavioural sciences. (2nd ed). Hillsdale, NJ: L. Erlbaum Associates.

Currell K & Jeukendrup AE (2008). Validity, reliability and sensitivity of measures of sporting performance. *Sports Med* **38**, 297–316.

Dai TH, Liu JZ, Sahgal V, Brown RW & Yue GH (2001). Relationship between muscle output and functional MRI-measured brain activation. *Exp Brain Res* **140**, 290–300.

Davies RC, Rowlands A V. & Eston RG (2009). Effect of exercise-induced muscle damage on ventilatory and perceived exertion responses to moderate and severe intensity cycle exercise. *Eur J Appl Physiol* **107**, 11–19.

de Morree HM, Klein C & Marcora SM (2014). Cortical substrates of the effects of caffeine and time-on-task on perception of effort. *J Appl Physiol* **117**, 1514–1523.

de Morree HM, Klein C & Marcora SM (2012). Perception of effort reflects central motor command during movement execution. *Psychophysiology* **49**, 1242–1253.

De Pauw K, Roelands B, Cheung SS, De Geus B, Rietjens G & Meeusen R (2013). Guidelines to classify subject groups in sport-science research. *Int J Sports Physiol Perform* **8**, 111–122.

Duffin J (2014). The fast exercise drive to breathe. *J Physiol* **592**, 445–451.

- Duffin J, Mohan RM, Vasiliou P, Stephenson R & Mahamed S (2000). A model of the chemoreflex control of breathing in humans: Model parameters measurement. *Respir Physiol* **120**, 13–26.
- Fan JL, Leiggener C, Rey F & Kayser B (2012). Effect of inspired CO₂ on the ventilatory response to high intensity exercise. *Respir Physiol Neurobiol* **180**, 283–288.
- Fernandes A, Galbo H, Kjaer M, Mitchell JH, Secher NH & Thomas SN (1990). Cardiovascular and ventilatory responses to dynamic exercise during epidural anaesthesia in man. *J Physiol* **420**, 281–293.
- Forster HV, Haouzi P & Dempsey JA (2012). Control of breathing during exercise. *Compr Physiol* **2**, 743–777.
- Gallagher CG, Brown E & Younes M (1987). Breathing pattern during maximal exercise and during submaximal exercise with hypercapnia. *J Appl Physiol* **63**, 238–244.
- Gelfand R & Lambertsen CJ (1973). Dynamic respiratory response to abrupt change of inspired CO₂ at normal and high PO₂. *J Appl Physiol* **35**, 903–913.
- González-Alonso J, Teller C, Andersen SL, Jensen FB, Hyldig T & Nielsen B (1999). Influence of body temperature on the development of fatigue during prolonged exercise in the heat. *J Appl Physiol* **86**, 1032–1039.
- Haouzi P & Bell HJ (2009). Control of breathing and volitional respiratory rhythm in humans. *J Appl Physiol* **106**, 904–910.
- Hayashi K, Honda Y, Ogawa T, Kondo N & Nishiyasu T (2006). Relationship between

- ventilatory response and body temperature during prolonged submaximal exercise. *J Appl Physiol* **100**, 414–420.
- Kjaer M, Hanel B, Worm L, Perko G, Lewis SF, Sahlin K, Galbo H & Secher NH (1999). Cardiovascular and neuroendocrine responses to exercise in hypoxia during impaired neural feedback from muscle. *Am J Physiol* **277**, R76-85.
- Koglin L & Kayser B (2013). Control and sensation of breathing during cycling exercise in hypoxia under naloxone: a randomised controlled crossover trial. *Extrem Physiol* **2**, 1.
- Liu JZ, Shan ZY, Zhang LD, Sahgal V, Brown RW & Yue GH (2003). Human brain activation during sustained and intermittent submaximal fatigue muscle contractions: an fMRI study. *J Neurophysiol* **90**, 300–312.
- Marcora SM, Bosio A & de Morree HM (2008). Locomotor muscle fatigue increases cardiorespiratory responses and reduces performance during intense cycling exercise independently from metabolic stress. *Am J Physiol Regul Integr Comp Physiol* **294**, R874–R883.
- Martin BJ & Weil JV (1979). CO₂ and exercise tidal volume. *J Appl Physiol* **46**, 322–325.
- Mateika JH, Mendello C, Obeid D & Badr MS (2004). Peripheral chemoreflex responsiveness is increased at elevated levels of carbon dioxide after episodic hypoxia in awake humans. *J Appl Physiol* **96**, 1197–1205.
- McParland C, Mink J & Gallagher CG (1991). Respiratory adaptations to dead space loading during maximal incremental exercise. *J Appl Physiol* **70**, 55–62.

- Miyamoto Y, Nakazono Y, Hiura T & Abe Y (1983). Cardiorespiratory dynamics during sinusoidal and impulse exercise in man. *Jpn J Physiol* **33**, 971–986.
- Nicolò A, Bazzucchi I, Haxhi J, Felici F & Sacchetti M (2014a). Comparing continuous and intermittent exercise: an “isoeffort” and “isotime” approach. *PLoS One* **9**, e94990.
- Nicolò A, Bazzucchi I, Lenti M, Haxhi J, Scotto di Palumbo A & Sacchetti M (2014b). Neuromuscular and metabolic responses to high-intensity intermittent cycling protocols with different work-to-rest ratios. *Int J Sports Physiol Perform* **9**, 151–160.
- Nicolò A, Marcora SM & Sacchetti M (2016). Respiratory frequency is strongly associated with perceived exertion during time trials of different duration. *J Sports Sci* **34**, 1199–1206.
- Nicolò A, Sacchetti M & Marcora SM (2015). Are respiratory frequency and tidal volume regulated by different inputs during exercise? *J Appl Physiol* **118**, 1559.
- Noble BJ, Metz KF, Pandolf KB & Cafarelli E (1973). Perceptual responses to exercise: A multiple regression study. *Med Sci Sports* **5**, 104–109.
- Ohashi S, Izumizaki M, Atsumi T & Homma I (2013). CO₂ homeostasis is maintained in conscious humans by regulation of tidal volume, but not of respiratory rhythm. *Respir Physiol Neurobiol* **186**, 155–163.
- Sheel AW & Romer LM (2012). Ventilation and respiratory mechanics. *Compr Physiol* **2**, 1093–1142.
- Siemionow V, Yue GH, Ranganathan VK, Liu JZ & Sahgal V (2000). Relationship between

motor activity-related cortical potential and voluntary muscle activation. *Exp brain Res* **133**, 303–311.

Syabbalo NC, Krishnan B, Zintel T & Gallagher CG (1994). Differential ventilatory control during constant work rate and incremental exercise. *Respir Physiol* **97**, 175–187.

Thornton JM, Guz A, Murphy K, Griffith AR, Pedersen DL, Kardos A, Leff A, Adams L, Casadei B & Paterson DJ (2001). Identification of higher brain centres that may encode the cardiorespiratory response to exercise in humans. *J Physiol* **533**, 823–836.

Todd G, Gordon CJ, Groeller H & Taylor NAS (2014). Does intramuscular thermal feedback modulate eccrine sweating in exercising humans? *Acta Physiol* **212**, 86–96.

Williamson JW, Fadel PJ & Mitchell JH (2006). New insights into central cardiovascular control during exercise in humans: a central command update. *Exp Physiol* **91**, 51–58.

Zénon A, Sidibé M & Olivier E (2015). Disrupting the supplementary motor area makes physical effort appear less effortful. *J Neurosci* **35**, 8737–8744.

Table 1. Mean and peak values of performance and physiological variables for the five HIIT trials.

	10_85%	10_70%	10_55%	10_30%	20_85%
Power output _{mean} (W)	341 ± 33 ^{cd}	336 ± 29 ^c	324 ± 30 ^c	297 ± 25	313 ± 33
Power output _{work} (W)	390 ± 40 ^{abcd}	429 ± 35 ^c	458 ± 42 ^c	488 ± 39	360 ± 36
Total work (kJ)	205 ± 20 ^{cd}	201 ± 18 ^c	195 ± 18 ^c	178 ± 15	375 ± 39
Cadence _{mean} (rpm)	96 ± 2 ^{abc}	100 ± 1 ^c	102 ± 2	105 ± 3	95 ± 3
$\dot{V}O_{2\text{mean}}$ (mL·min ⁻¹)	4054 ± 361	4030 ± 291 ^c	3993 ± 270	3785 ± 324	3933 ± 335
$\dot{V}O_{2\text{peak}}$ (mL·min ⁻¹)	4287 ± 388	4350 ± 239 ^c	4280 ± 280	4007 ± 321	4133 ± 308
$\dot{V}CO_{2\text{mean}}$ (mL·min ⁻¹)	3952 ± 351 ^d	4012 ± 231	3981 ± 340	3858 ± 325	3723 ± 377
HR _{mean} (beats·min ⁻¹)	176 ± 8 ^d	173 ± 7	173 ± 7	172 ± 11	172 ± 7
HR _{peak} (beats·min ⁻¹)	186 ± 8 ^d	183 ± 7	182 ± 8	182 ± 9	181 ± 8
$\dot{V}E_{\text{mean}}$ (L·min ⁻¹)	144 ± 20	142 ± 20	141 ± 22	142 ± 26	140 ± 24
$\dot{V}E_{\text{peak}}$ (L·min ⁻¹)	172 ± 24 ^d	175 ± 21	171 ± 25	170 ± 26	164 ± 24
$f_{R\text{mean}}$ (breaths·min ⁻¹)	50 ± 7	48 ± 7	48 ± 5	47 ± 7	50 ± 6
$f_{R\text{peak}}$ (breaths·min ⁻¹)	59 ± 6	59 ± 7	58 ± 5	57 ± 6	59 ± 6
V _{Tmean} (L)	2.9 ± 0.4 ^d	2.9 ± 0.3	2.9 ± 0.4	3 ± 0.4	2.8 ± 0.4
V _{Tpeak} (L)	3.1 ± 0.4	3.1 ± 0.4	3.1 ± 0.4	3.2 ± 0.5	2.9 ± 0.4

10_85% = 10-min HIIT trial with recovery intensity corresponding to 85% of the 10-min continuous TT mean workload. 10_70% = 10-min HIIT trial with recovery intensity corresponding to 70% of the 10-min continuous TT mean workload. 10_55% = 10-min HIIT trial with recovery intensity corresponding to 55% of the 10-min continuous TT mean workload. 10_30% = 10-min HIIT trial with recovery intensity corresponding to 30% of

the 10-min continuous TT mean workload. 20_85% = 20-min HIIT trial with recovery intensity corresponding to 85% of the 20-min continuous TT mean workload. TT = time trial.

Power output_{work} = work-phase mean power output, $\dot{V}O_2$ = oxygen uptake, $\dot{V}CO_2$ = carbon dioxide output, HR = heart rate, \dot{V}_E = minute ventilation, f_R = respiratory frequency, V_T = tidal volume. Values are mean \pm SD.

^a $P < 0.05$ vs. 10_70%, ^b $P < 0.05$ vs. 10_55%, ^c $P < 0.05$ vs. 10_30%, ^d $P < 0.05$ vs. 20_85%.

Table 2. Correlations between RPE and f_R , RPE and \dot{V}_E and RPE and V_T for the five HIIT trials.

		Overall	10_85%	10_70%	10_55%	10_30%	20_85%
RPE and f_R (breaths·min ⁻¹)	r	0.87	0.92	0.92	0.88	0.92	0.91
	P	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
RPE and \dot{V}_E (L·min ⁻¹)	r	0.80	0.87	0.89	0.86	0.86	0.86
	P	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
RPE and V_T (L)	r	-0.11	-0.12	0.05	0.11	-0.25	-0.54
	P	0.053	0.392	0.721	0.422	0.058	<0.001

Overall = five HIIT trials considered together. 10_85% = 10-min HIIT trial with recovery intensity corresponding to 85% of the 10-min continuous TT mean workload. 10_70% = 10-min HIIT trial with recovery intensity corresponding to 70% of the 10-min continuous TT mean workload. 10_55% = 10-min HIIT trial with recovery intensity corresponding to 55% of the 10-min continuous TT mean workload. 10_30% = 10-min HIIT trial with recovery intensity corresponding to 30% of the 10-min continuous TT mean workload. 20_85% = 20-min HIIT trial with recovery intensity corresponding to 85% of the 20-min continuous TT mean workload. TT = time trial.

f_R = respiratory frequency, \dot{V}_E = minute ventilation, V_T = tidal volume, r = correlation coefficient, P = alpha level.

Figure legends

Figure 1. Group mean power output for 10_30% (A), 10_55% (B), 10_70% (C), 10_85% (D) and 20_85% (E).

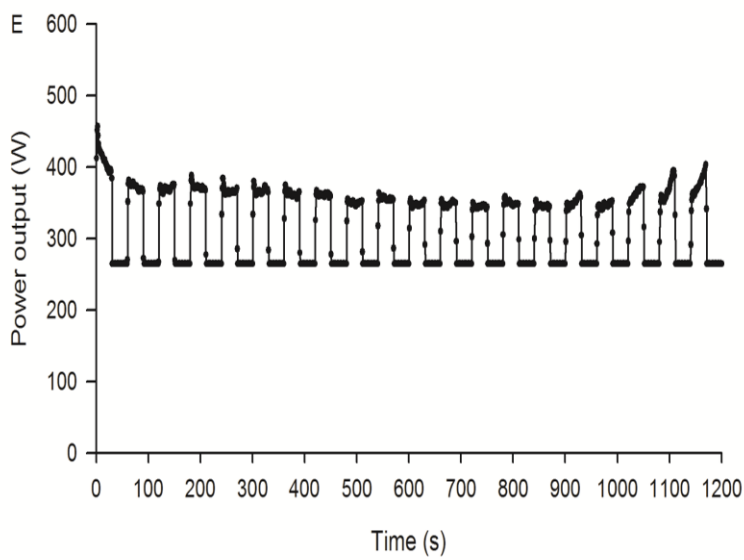
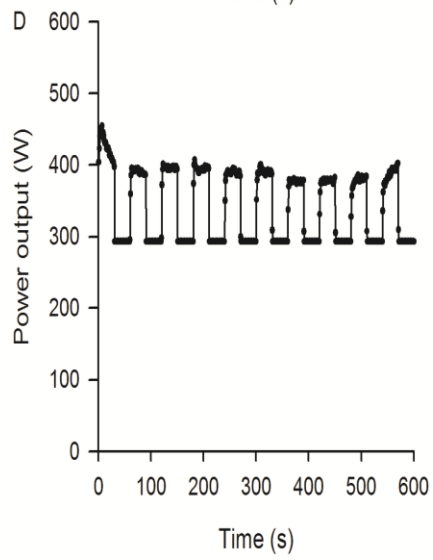
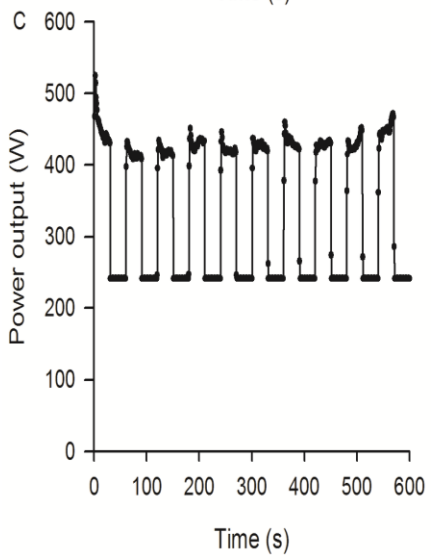
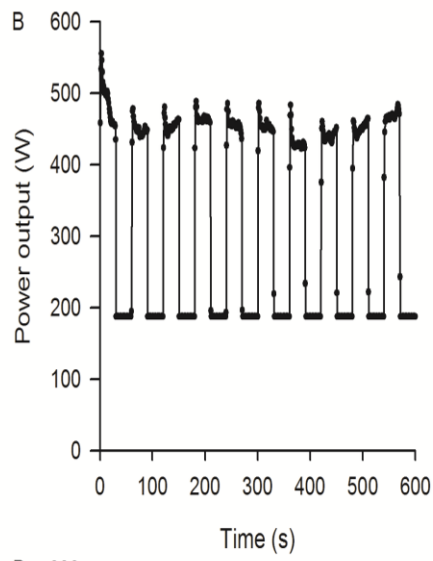
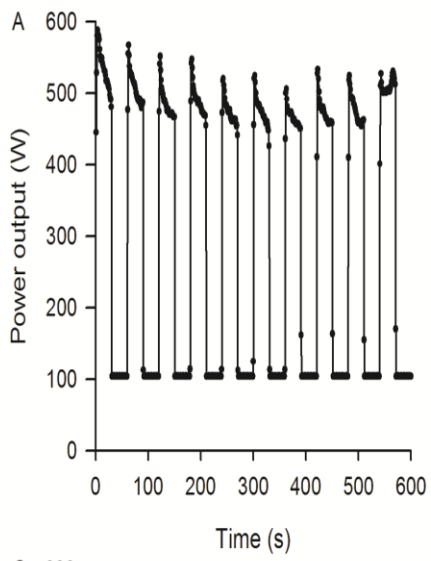


Figure 2. Group mean f_R (A) and V_T (B) for 10_85% (closed circles), 10_70% (open circles), 10_55% (closed triangles) and 10_30% (open triangles).

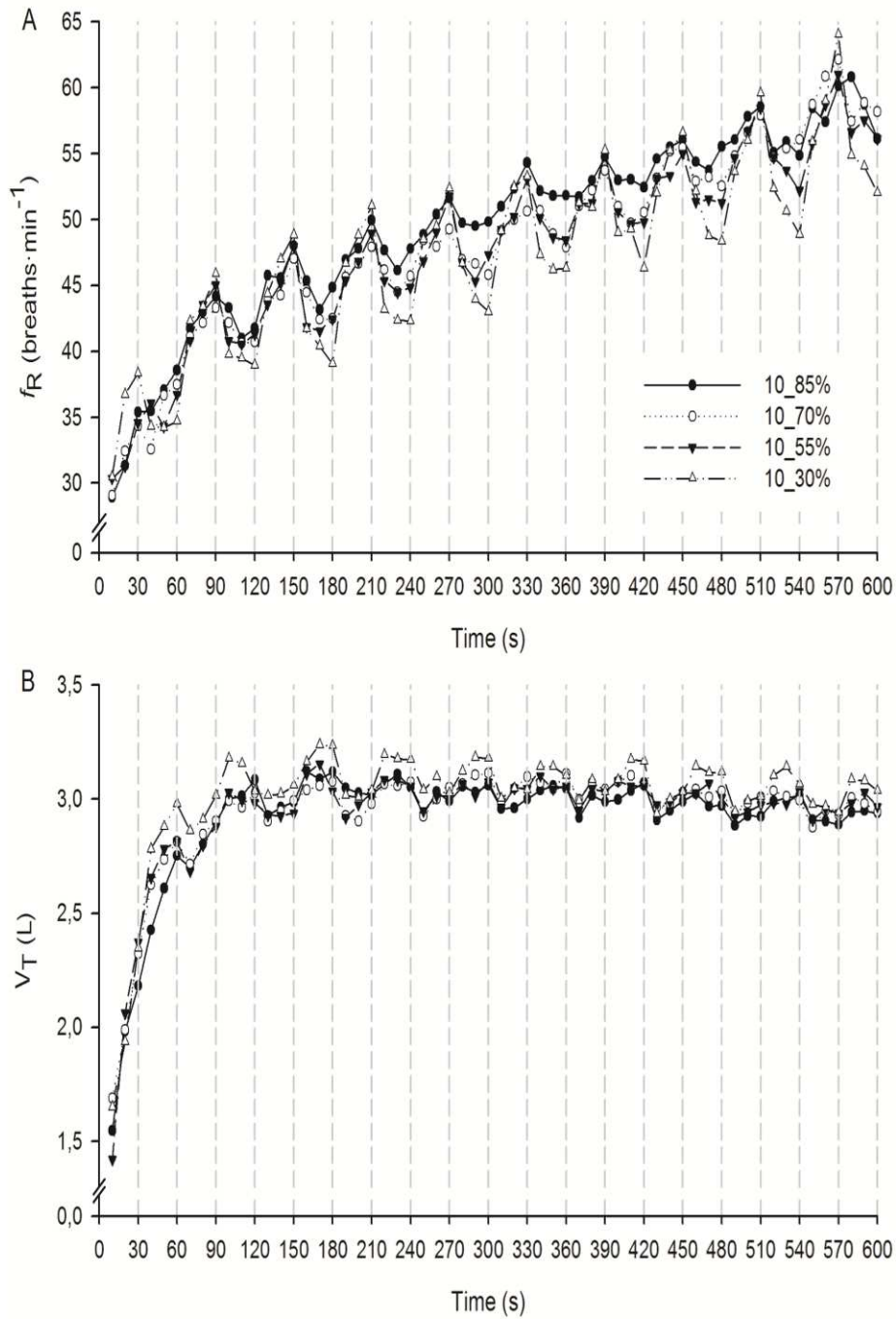


Figure 3. Work-recovery cycle responses of power output (A), f_R (B), P_{ETCO_2} (C), V_T (D), $\dot{V}CO_2$ (E), \dot{V}_E (F), $\dot{V}O_2$ (G) and HR (H) for 10_85% (closed circles), 10_70% (open circles), 10_55% (closed triangles) and 10_30% (open triangles). * Significant interaction ($P<0.001$). † Significant main effect of trial ($P<0.037$). ‡ Significant main effect of time ($P<0.002$).

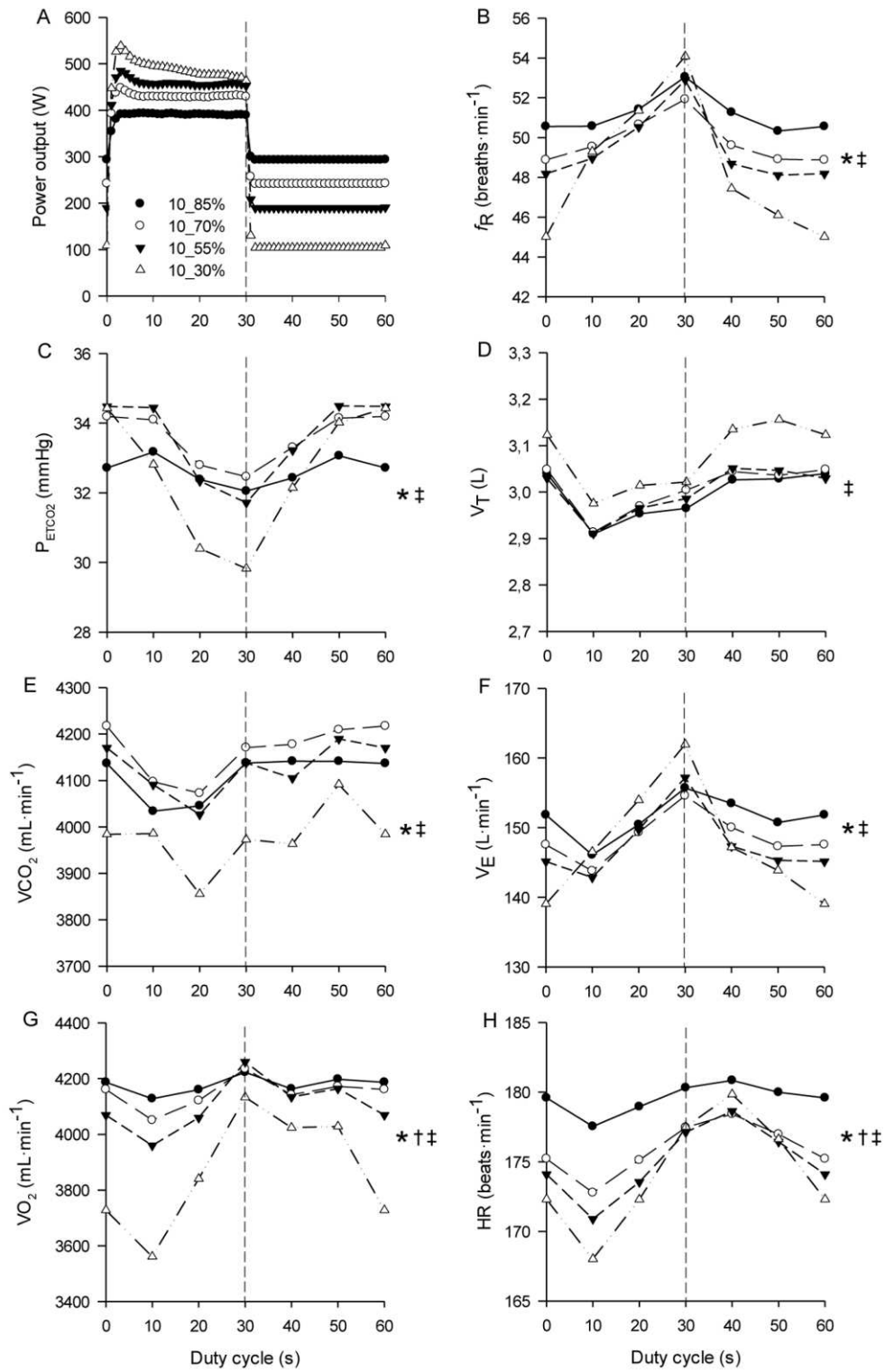


Figure 4. Effect of recovery intensity manipulation (left panels) and exercise duration manipulation (right panels) on RPE (A and B), f_R (C and D), \dot{V}_E (E and F) and V_T (G and H) for 10_85% (closed circles), 10_70% (open circles, left panels), 10_55% (closed triangles), 10_30% (open triangles) and 20_85% (open circles, right panels). * Significant interaction ($P<0.001$). † Significant main effect of trial ($P<0.001$). Data are mean \pm SE.

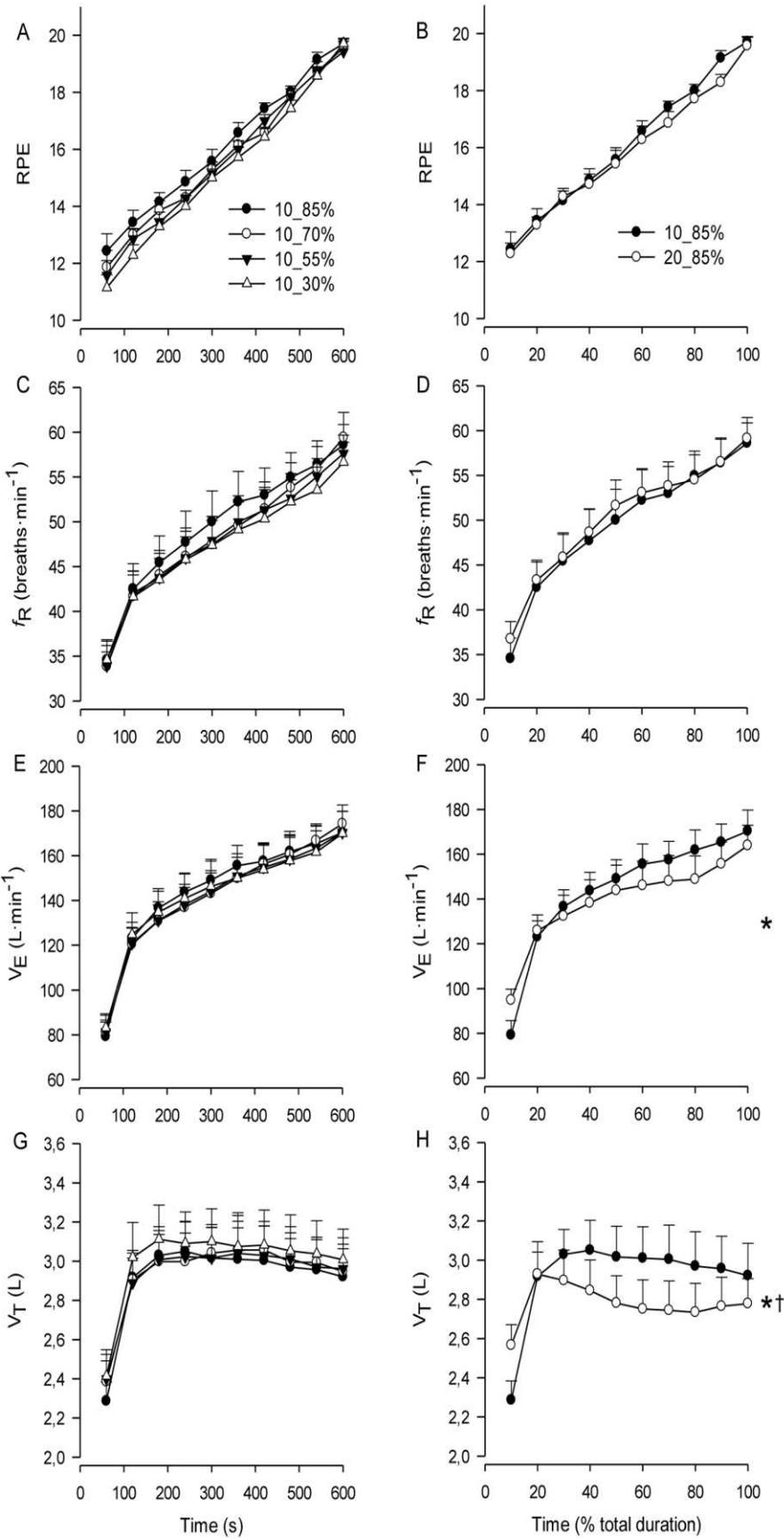


Figure 5. Effect of recovery intensity manipulation (left panels) and exercise duration manipulation (right panels) on $\dot{V}O_2$ (A and B), $\dot{V}CO_2$ (C and D), P_{ETCO_2} (E and F) and HR (G and H) for 10_85% (closed circles), 10_70% (open circles, left panels), 10_55% (closed triangles) and 10_30% (open triangles) and 20_85% (open circles, right panels). * Significant interaction ($P<0.014$). † Significant main effect of trial ($P<0.034$). Data are mean \pm SE.

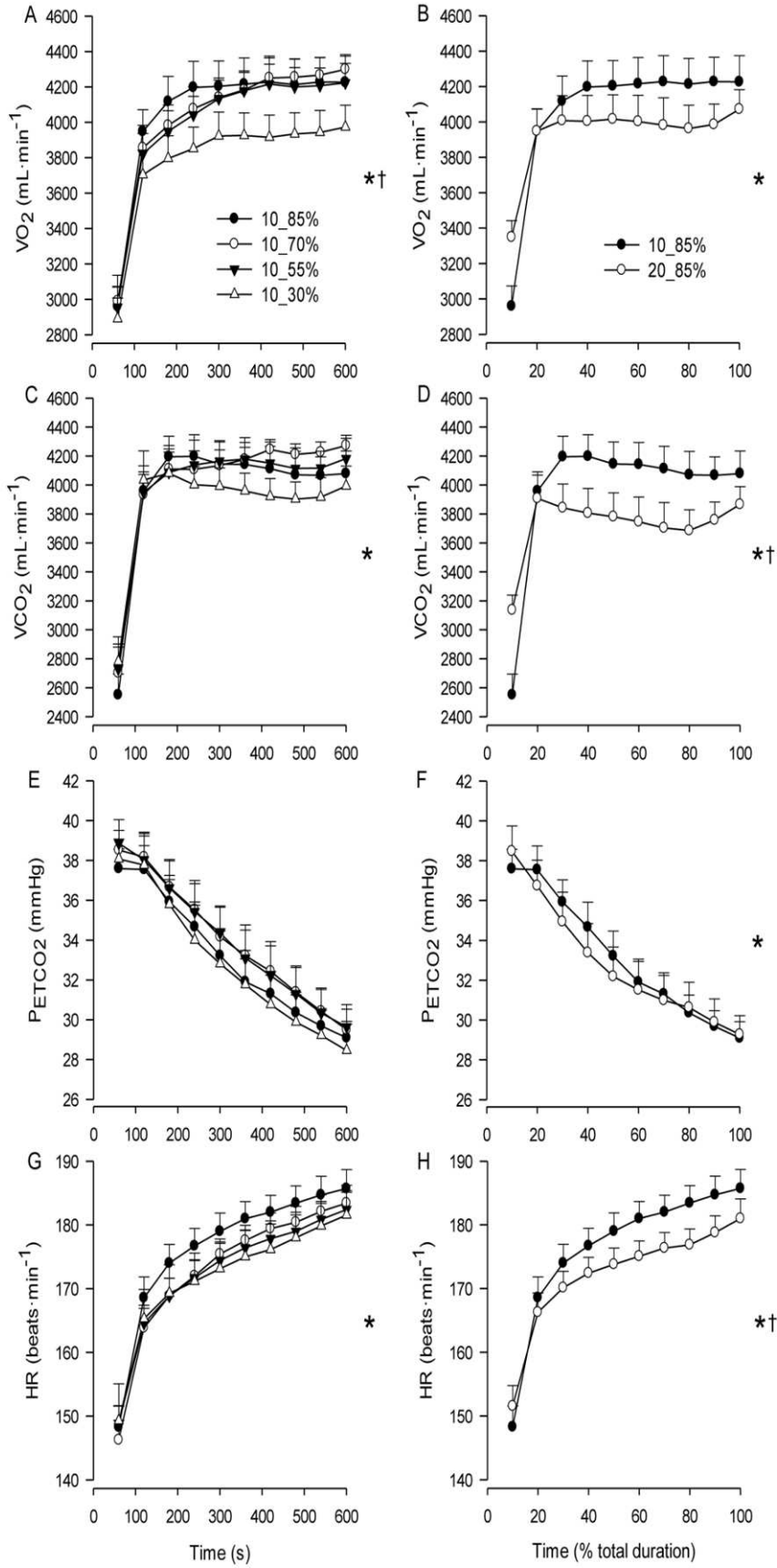


Figure 6. Effect of recovery intensity manipulation (A) and exercise duration manipulation (B) on La^- for 10_85% (closed circles), 10_70% (open circles, panel A), 10_55% (closed triangles), 10_30% (open triangles) and 20_85% (open circles, panel B). Data are mean \pm SE.

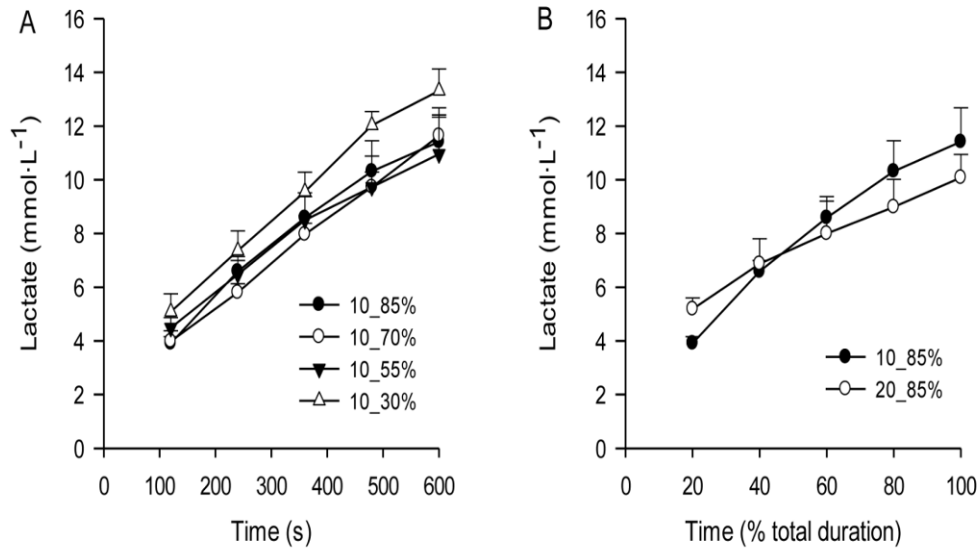


Figure 7. Effect of recovery intensity manipulation (A) and exercise duration manipulation (B) on the correlation between RPE and f_R , RPE and \dot{V}_E and RPE and V_T for 10_85% (closed circles), 10_70% (open circles), 10_55% (closed triangles) and 10_30% (open triangles).

Each symbol represents the mean value of all participants at each time point.

