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Neuromuscular and physiological variables evolve independently when running immediately after cycling

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Abstract

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Disciplines

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NEUROMUSCULAR AND PHYSIOLOGICAL VARIABLES EVOLVE
INDEPENDENTLY WHEN RUNNING IMMEDIATELY AFTER CYCLING

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Keywords: Cycle-run; EMG; Cycling; Running; Transition; Triathlon

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Abstract

During the early period of running after cycling, EMG patterns of the leg are modified in only some highly trained triathletes. The majority of studies have analysed muscle EMG patterns at arbitrary, predetermined time points. The purpose of this study was to examine changes to EMG patterns of the lower limb at physiologically determined times during the cycle-run transition period to better investigate neuromuscular adaptations. Six highly trained triathletes completed a 10 min isolated run (IR), 30 min of rest, then a 20 min cycling procedure, before a 10 min transition run (C-R). Surface EMG activity of eight lower limb muscles was recorded, normalised and quantified at four time points. Oxygen uptake and heart rate values were also collected. Across all muscles, mean (\pm SD) EMG patterns, demonstrated significant levels of reproducibility for each participant at all four time points ($\alpha < 0.05$; $r = 0.52 - 0.97$). Mean EMG patterns during C-R correlated highly with the IR patterns ($\alpha < 0.05$). These results show that EMG patterns during subsequent running are not significantly affected by prior cycling. However, variability of muscle recruitment activity does appear to increase during C-R transition when compared to IR.

Keywords: Cycle-run; EMG; Cycling; Running; Transition; Triathlon

1. Introduction

Successful performance in triathlon is largely dependent on the ability of an athlete to overcome the complications of transitioning between disciplines, the most crucial of which is the cycle-run transition (C-R)(Millet & Vleck, 2000). The C-R transition is

5 defined as the period from the last kilometre of the cycle leg to the end of the first kilometre of the run (Millet & Vleck, 2000), and is considered an overall performance determinant (Bonacci, Green, et al., 2010). Therefore, understanding and limiting cycling-induced changes to an athlete's running ability both biomechanically and physiologically is of significant importance (Millet & Vleck, 2000). Previous research

10 has established the impaired effects of prior cycling on subsequent running economy performance among a variety of triathlete populations (Hue, Le Gallais, Chollet, Boussana, & Prefaut, 1998; Millet & Bentley, 2004). In particular, within the first minutes of running after cycling oxygen uptake ($\dot{V}O_2$), breathing frequency, minute ventilation and heart rate (HR) are all elevated compared to during isolated running

15 (IR) (Guezennec, Vallier, Bigard, & Durey, 1996; Hausswirth, Bigard, Berthelot, Thomaidis, & Guezennec, 1996; Millet & Vleck, 2000). Furthermore, alterations to muscle recruitment activity may influence running economy (Bonacci, Chapman, Blanch, & Vicenzino, 2009) and indirectly $\dot{V}O_2$ when running after cycling (Bonacci, Green, et al., 2010). In their study, muscle recruitment activity was altered

20 in seven of 15 moderately trained triathletes, accompanied with clinically significant alterations to $\dot{V}O_2$ during C-R exercise. They concluded that cycling related muscle recruitment changes are linked with alterations to running economy during subsequent running. However, this group (Bonacci, Saunders, Alexander, Blanch, & Vicenzino, 2011) later suggested that prior low or high intensity cycling did not influence

25 neuromuscular control or running economy in seven elite international triathletes. A

relationship between muscle recruitment activity and $\dot{V}O_2$ when running after cycling is far from conclusive, and likely dependent upon an athlete's experience and training history (Hauswirth, Bigard, & Guezennec, 1997). However, past studies have established a reasonable link between muscle recruitment activity and metabolic cost during constant load exercise (Moore, Jones, & Dixon, 2014; Saunders et al., 2000). Therefore, understanding changes in muscle recruitment activity, resulting from prior cycling exercise, may potentially assist in identifying changes and adaptations of early phase changes in $\dot{V}O_2$ during the subsequent C-R phase within triathlete populations. Previous studies looking at changes to muscle recruitment activity during the C-R phase have used arbitrary time points (i.e. 1 min, 2 min and 3 min), rather than times based upon individual physiological variables. The challenge is to recognise where within the transition period muscle recruitment activity and $\dot{V}O_2$ changes occur and if they are the result of prior cycling in the absence of fatigue. Therefore, the purpose of this study was to analyse muscle recruitment patterns during the C-R phase, compared to IR, at specific time points based upon individually calculated physiological variables representing physiological adaptation. We were also interested in understanding if and how EMG patterns quantified at those specific time points reflected changes in those physiological variables.

2. Methods

2.1. Subjects

Six trained, competitive triathletes were selected to participate in this study. All had experienced Australian National level and/or International Triathlon Union (ITU) level competition and had gained this experience in at least the year preceding testing.

Age, physical characteristics, competitive experience, training distances and sessions
50 and personal records are given in Table 1.

2.2. Protocol

A controlled single-group laboratory-based investigation was conducted to compare
muscle recruitment activity, $\dot{V}O_2$ and HR values during submaximal intensity IR and
C-R exercise. The protocol used has been specifically developed, using moderate
55 intensity cycling and running in order to analyse neuromuscular changes when
running after cycling and has been shown to be highly repeatable providing a robust
baseline measure of neuromuscular control during running without causing undue
fatigue (Chapman et al., 2009).

All subjects completed a 10 min IR, followed by 30 min of rest before completing a
60 20 min variable cadence cycle bout and a 30 min transition run. Participants had to
transition between cycling and running in less than 60 s. All running tests were
conducted on a treadmill (grade = 0%, Landice, Randolph, USA) to allow comparison
with past research of a similar nature (Bonacci, Blanch, Chapman, & Vicenzino,
2010; Bonacci et al., 2011). Treadmill speed was reached within the first 20 s of
65 commencing running and remained constant for the entirety of IR and C-R conditions.
Prior to experimental testing a standardised five minute warm-up run was completed.
During this period participants were asked to self-select a running speed that would be
manageable and non-fatiguing for 30 min of running. This self-selected speed was
used as the treadmill speed for both the IR and C-R for that individual. Following the
70 warm-up exercise, participants were required to rest in a seated position to allow their
HR to recover to baseline values. The resting period between warm-up and testing
exercise was 10 ± 0.6 min. Participants performed the variable cadence cycling

protocol using their personal road bikes mounted on a stationary magnetic cycle ergometer (Tacx Satori Trainer, Tacx, Netherlands). Cadence was controlled using an
75 ANT+ bike computer compatible with the speed/cadence monitors on the participant's bicycles. During the first five-minutes and final three-minutes the participants cycled at an individually preferred cadence. Four cadence blocks of three-minute duration – (1) individually preferred cadence, 55-60 rpm, 75-80 rpm and 95-100 rpm – were randomly ordered between the 6th to 17th min. During the variable
80 cadence cycling protocol the participants were required to sustain a level of intensity consistent with an RPE of 14.

2.3. Data acquisition

2.3.1. EMG

Electromyographic activity of the gluteus medius (GM), biceps femoris (BF), vastus
85 medialis (VM), vastus lateralis (VL), rectus femoris (RF), gastrocnemius medialis (MG), gastrocnemius lateralis (LG) and tibialis anterior (TA) was recorded from the left leg in all participants. This current study focused on muscles of the thigh and leg due to their functional importance to both cycling and running performance during triathlon (Chapman, Vicenzino, Blanch, & Hodges, 2008; Manninen & Kallinen,
90 1996). Further, evidence suggests that atypical muscle recruitment patterns and musculoskeletal injury of the lower limb are potentially related (Chapman, Vicenzino, Blanch, & Hodges, 2008; Cowan, Hodges, Bennell, & Crossley, 2002). Preparation of each EMG skin site included shaving, mildly abrading and cleansing with isopropyl alcohol swabs according to the “Standards of Reporting EMG Data”
95 (Electromyography and Kinesiology, 1997). Additionally, pre-gelled bipolar Ag/AgCl 1 mm parallel-bar surface EMG electrodes (fixed inter-electrode distance of 10 mm)

(Delsys[®], USA) were anatomically positioned on the mid-belly of each muscle, with the electrode bars situated perpendicular to the direction of muscle fibres of each muscle in accordance with procedures outlined by the European Surface EMG for the

100 Non-Invasive Assessment of Muscles (SENIAM) to minimise crosstalk. A ground reference Dermatode[®] (American Imex, Irvine, USA) electrode was positioned on the left lateral malleolus. EMG signals were recorded using an eight channel Delsys[®] Bagnoli[™] EMG System (Delsys[®], USA). EMG measurements were recorded at a sampling frequency of 1,000 Hz and digitised by a 16-bit Analog-to-digital converter.

105 The bipolar signal was amplified (input impedance > 1 M Ω) and band-pass filtered between 10 and 500 Hz with a mode rejection ratio of 110 dB, gain of 305 and maximum noise of 1.6 μ V and a second order Butterworth filter was applied to the data to remove contamination from movement artefacts before being full-wave rectified, DC offset. EMG data were integrated into subject specific time bins.

110 Rectified EMG data were exported in an embedded VICON c3d file and stored for computer processing.

2.3.2. $\dot{V}O_2$ and HR

Ventilatory data, including total $\dot{V}O_2$, were measured continuously and recorded at breath-by-breath rate using a metabolic gas analysis cart (Parvo TrueMax 2400,

115 Parvomedics, USA). Prior to testing standing-resting $\dot{V}O_2$ was measured over a five-minute period to provide a comparative level of baseline $\dot{V}O_2$ values.

Participants wore a nose clip and breathed through a low-dead space, minimal resistant mouthpiece that was secured via a capillary line attached to the mouthpiece, to the volume transducer. Starting $\dot{V}O_2$ was recorded as the first breath after the

120 onset of exercise. Beat-by-beat HR was measured using a short-distance telemetry

Polar Interface module synchronised to a Polar heart rate monitor chest unit (Polar Electro, Port Washington, N.Y., USA) during both running conditions.

2.4. Data processing

2.4.1. EMG

125 All data processing and analysis was performed off-line using a commercial software
package (MATLAB 6.1, The MathWorks Inc., Natick, MA, 2000). EMG data were time
standardised to 100 points for each stride, from heel strike to heel strike, during
the final minute (10th min) of the IR and for 20 strides at the respective C-R exercise time
points. EMG amplitudes were expressed as a percentage of the peak record EMG
130 amplitude for the respective muscle, in accordance with previous procedures
(Bonacci, Blanch, et al., 2010). All EMG traces were visually screened and data of
inadequate quality (i.e. traces containing high levels of artefact, that could not be
adequately removed by signal filters) were excluded from analysis, as previously
recommended (Bonacci, Blanch, et al., 2010). Analysed EMG traces were reported
135 using indices of: (i) the pattern of muscle recruitment and movement indicated by
EMG traces; (ii) mean EMG amplitude for the duration of each stride during
respective time periods; (iii) peak EMG amplitude for each muscle and (iv)
coefficient of variation (CV).

2.4.2. $\dot{V}O_2$

140 Breath-by-breath $\dot{V}O_2$ data from all tests were screened to exclude errant breaths. A
single-phase, logarithm model proposed by Stupnicki et al., (2010) was used to
calculate theoretical halftime ($t_{1/2}$) to steady-state using a remodelled logarithm

(Equation 1). The logarithm was reconstructed using net $\dot{V}O_2$ (x_i) values and with a calculated peak submaximal $\dot{V}O_2$ value (x_m).

145
$$\text{Logit} = \log [(x_i)/(x_m - x_i)]$$

$$\text{Logit} = \log (x_i) - \log(x_m - x_i)$$

(1)

Mean response time (MRT) was considered as the time required to achieve ~63% of steady-state (Chatterjee et al., 2013) and was calculated using Eq. (2) as reported by

150 Whipp (1971).

$$\text{MRT} = [t * \Delta \dot{V}O_2 - \Sigma \dot{V}O_2] / \Delta \dot{V}O_2$$

(2)

Steady-state $\dot{V}O_2$ was recorded at 180 s (3rd minute) after exercise onset, based upon physiological steady-state being achieved after 2.5 to 3 min in young healthy adults

155 (Pringle et al., 2003; Xu & Rhodes, 1999). Therefore, this study considered the trained participants as having achieved steady-state by 180 s of constant velocity running, using a $\pm 5\%$ range prediction calculated from steady-state $\dot{V}O_2$. Mean $\dot{V}O_2$ at the 10th minute was calculated as the average between the 9th and 10th minute and was used to compare with the 180 s (steady-state) values to ensure that

160 $\dot{V}O_2$ did not drift under exercising conditions. Rate constants (k) were calculated using Eq. (3) (Whipp, 1971), and used to determine potential transient changes in $\dot{V}O_2$ and the relative effectiveness of the cardiopulmonary and metabolic systems adaptation after the onset of the IR and C-R conditions.

$$k = \dot{V}O_{2 \text{ (steady-state)}} / \dot{V}O_2 \text{ deficit}$$

2.5 Statistical analysis

Individual mean EMG traces for respective $t_{1/2}$, MRT and 180 s time points during the C-R condition were reconstructed from 20 strides at each time interval (ten strides prior and ten strides post each time interval) and were compared with their respective mean IR data collected from the final minute of running. Traces were deemed significantly different between the IR and C-R conditions if the mean EMG trace during C-R condition timeframes exceeded 10% of mean IR trace (Bonacci, Green, et al., 2010). Mean variation of EMG waveforms between IR and C-R EMG traces were assessed via calculation of the CV for all four time points. The CV was calculated as the root mean square of the standard deviation over the period of a stride divided by the mean collection waveform over the 20 stride sampling period (Winter & Yack, 1987). Group main effects were compared by differences retested using analysis of variance (ANOVA) with Bonferroni adjusted pairwise multiple comparisons of within-individual differences at each point. Comparisons of mean and peak EMG amplitudes between time points were made using repeated measures ANOVA for all individual participants.

Mean $\dot{V}O_2$ and k values recorded during the IR and C-R ($t_{1/2}$, MRT and 180 s) conditions were compared using paired t -tests. Mean steady-state $\dot{V}O_2$ values were also correlated using a Pearson's correlation of coefficient (r) to determine similarities between the conditions. Paired t -tests were used to identify differences between group mean HR across the respective time points and k values for the IR and C-R conditions.

Statistical analysis was carried out using IBM SPSS 21 (IBM Corporation, Armonk, NY), with statistical significance set at $\alpha < 0.05$. Descriptive data are reported as

190 mean \pm standard deviation (SD) and data in the figures are presented as mean \pm standard error (SE).

3. Results

Throughout testing the participants maintained a mean running speed of 14.1 ± 0.8 km/h. Fig. 1 displays the mean (\pm SD) processed EMG activity of the documented

195 muscles for all the participants. While the mean EMG traces were consistently similar ($r = 0.52-0.97$; $\alpha < 0.05$) across all time points for the respective muscles, a trend of increased variability in the C-R $t_{1/2}$, MRT and 180 s sample traces was visible (Figure 1). Table 2 shows the CV (\pm SD) for the population mean EMG traces outlining the increased inconsistency in muscle activity during C-R strides. Mean and peak EMG
200 amplitude across all individual traces showed no differences between the IR and the C-R conditions.

Oxygen uptake values corresponding to the IR and C-R sampling times are presented Table 3 and mean $\dot{V}O_2$ traces to 180 s are evident in Figure 2. Mean starting $\dot{V}O_2$ was significantly higher at the onset of the C-R ($\alpha = 0.02$). Mean $t_{1/2}$ and MRT values were significantly lower during the C-R, compared to IR ($\alpha < 0.01$). These results corresponded with a significantly faster k value during the C-R ($\alpha = 0.00$). Mean O_2 at 180 s and at the 10th minute of running were not different between conditions. Correlated mean steady-state $\dot{V}O_2$ values proved to be significantly similar ($r = 0.77$; $\alpha = 0.03$).

210 Heart rate values are presented in Table 4 and mean HR traces are presented in Figure 3. At 180 s and the 10th minute HR was not different between conditions. Mean

starting HR was significantly elevated at the onset of the C-R condition ($\alpha < 0.01$). Mean $t_{1/2}$ and MRT values for HR were not different between the IR and C-R and were reflective of no change in the mean k value.

215 **4. Discussion**

The purpose of this study was to compare muscle recruitment activity at physiologically determined time points during the C-R transition and compare these to IR recruitment patterns in highly competitive triathletes. The main findings were that compared to IR, prior moderate-intensity cycling did not significantly impact
220 mean EMG activity or adversely affect steady-state $\dot{V}O_2$ and HR values during subsequent running during the C-R condition. However, variability of muscle recruitment activity was noticeably higher at all-time points during the C-R condition (Fig 1.).

These results suggest that muscle recruitment activity is preserved in highly
225 competitive triathletes during running after cycling at a relatively moderate-intensity. This finding is consistent with previous investigations concluding that cycling has no adverse effect on subsequent running mechanics (Quigley, 1996) or muscle recruitment patterns during running after cycling (Bonacci, Blanch, et al., 2010; Bonacci et al., 2011). However, our findings are in contrast to previous studies
230 reporting that muscle recruitment was altered after cycling in 36% of highly trained triathletes (Chapman, Vicenzino, Blanch, Dowlan, & Hodges, 2008) and 53% of moderately trained ones (Bonacci, Green, et al., 2010). We did however observe an increase in muscle recruitment variability during running after cycling that is
consistent with the results of Chapman et al. (2008). These authors further reported
235 that changes to muscle recruitment persisted for the duration of the 30 min transition

run. Our results similarly demonstrated an evident increase in muscle recruitment variability across all sampling time points during the C-R conditions.

The minimal individual effects of prior cycling on muscle recruitment activity and metabolic cost of subsequent running were unexpected and are contrary to previous findings that demonstrated significant and occasionally large individual effects when running after cycling that were absent when presenting group data (Bonacci, Green, et al., 2010), signifying adaptations are highly individually-specific. However, past research has confirmed that experienced or highly trained triathletes tend to display less mechanical and performance decrements as opposed to their lesser-experienced peers (Millet, Millet, & Candau, 2001; Millet, Millet, Hofmann, & Candau, 2000). Compared to elite or highly trained triathletes, middle level or moderately trained triathletes have previously displayed greater vertical displacement, acceleration and deceleration of their centre of mass (Millet et al., 2001) and greater change to muscle recruitment and sagittal plane kinematics (Bonacci, Green, et al., 2010; Chapman, Vicenzino, Blanch, Dowlan, et al., 2008). Alternatively, the absence of real change in muscle recruitment activity may be a result of a training effect. Previously, Bonacci et al. (2011) has discussed a potential training effect present among highly trained triathletes as an explanation for the lack of alteration in muscle recruitment during running after a prior high intensity cycling bout; proposing the idea that a training effect or adaptation minimising or negating substantial alterations to muscle recruitment during running after cycling is plausible. Additionally, the relative moderate intensity and short duration of the prior cycling exercise, during this study may also have influenced the lack of individual change in muscle activity. However, previous research has employed both low and high intensity cycling protocols prior to running and have reported little or no significant change to muscle recruitment

patterns (Bonacci et al., 2011). Similar evidence has stated that prior submaximal or moderate intensity exercise is not sufficient to influence subsequent physiological variables, including the speeding of $\dot{V}O_2$ (Gerbino, Ward, & Whipp, 1996; Jones et al., 2008). Our results show that $\dot{V}O_2$ values at $t_{1/2}$ and MRT are significantly
265 reduced in time (Table 3) and are likely due to the elevated metabolic starting point during the C-R condition. These results do reflect similar studies where prior cycling is reported to influence $\dot{V}O_2$ during subsequent running (Hauswirth et al., 1997; Hue et al., 1998; Millet et al., 2000). More specifically, reductions to ventilatory thresholds have been reported to impair subsequent running performance in well-
270 trained triathletes following prior swimming and cycling exercise (De Vito, Bernardi, Sproviero, & Figura, 1995). Furthermore, the k would suggest that the increase in O_2 was also faster during the C-R condition, indicating that steady-state could have been reached earlier following prior moderate intensity cycling exercise. The cause of the changes observed during the first minute of the C-R condition are not fully
275 understood, but may have resulted from an enhanced muscle oxygen supply (DeLorey, Kowalchuk, Heenan, Dumanoir, & Paterson, 2007), an increase in blood flow (Krustrup, Gonzalez-Alonso, Quistorff, & Bangsbo, 2001) and/or improved oxidative enzyme activity (Sahlin, Sorensen, Gladden, Rossiter, & Pedersen, 2005). In contrast, HR values were not influenced by prior exercise.

280 In conclusion, moderate intensity prior cycling had minimal impact on muscle recruitment activity and HR values during the C-R transition period. However, $\dot{V}O_2$ values do appear to be affected during this time, although prior cycling did not affect the overall $\dot{V}O_2$ at steady-state during subsequent running. These results suggest that muscle recruitment activity and $\dot{V}O_2$ during the C-R transition do not share any
285 meaningful link. This is in contrast to previous research that suggests a relationship

between changes in muscle recruitment and $\dot{V}O_2$ during exercise following prior exercise (Barker, Trebilcock, Breese, Jones, & Armstrong, 2014; Layec et al., 2009), particularly an increase in muscle recruitment activity correlated with increase in $\dot{V}O_2$ (Burnley, Doust, Ball, & Jones, 2002; Saunders et al., 2000). These studies all employed high-intensity exercise protocols, whereas the current study opted for self-regulated moderate intensity exercise and this may explain the differences between the results of the current study and previous ones. Nullifying the effects of fatigue was a primary concern in this study. In addition, the use of a moderate intensity protocol was intended to ensure a near-linear exponential increase in $\dot{V}O_2$ and HR variables at the onset of exercise to steady-state. This was important for the IR condition, allowing us to record stable $t_{1/2}$ and mean response time variables. Participants were required to reach a steady-state level of functioning in order to segment EMG traces based upon physiological variables, rather than arbitrary time points, and this was achievable using the current moderate intensity protocol. Adopting a higher intensity testing protocol may not have guaranteed that subsequent running motor recruitment patterns were influenced by accumulative fatigue. Furthermore, the necessary achievement of steady-state could have been affected by cardiac drift (Jeukendrup & Diemen, 1998) or delayed steady-state (Barstow, Jones, Nguyen, & Casaburi, 1996) if a high intensity exercise protocol was used.

It is recommended that future research analysing the C-R transition period employ higher intensity testing protocols to more closely replicating racing demands. Also, the addition of a run-run condition may further help determine the impact of prior cycling on subsequent running performance, specifically during the C-R transition period.

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Tables

Table 1

Age, physical characteristics, competitive experience, training distances/sessions^a and personal records (PR).

	Age (yrs)	Mas s (kg)	Heig ht (cm)	Experie nce (yrs)	Cycle (km/w k)	Run (km/w k)	Sessio ns (km/w k)	10 km PR (min)	OT PR (h)
Triathle tes <i>n</i> = 6	24.8 (±7.6)	69.1 (±6.3)	178.4 (±7.2)	4.4 (±1.1)	296.7 (±72.3)	55.4 (±11.4)	10.2 (±1.2)	32.3 (±1.3)	1 h 53 (±2.2)

^a training distances/sessions recorded during the three months prior to testing.

Table 2

Mean CV (± SD) for EMG traces

	GM	BF	VM	VL	RF	MG	LG	TA
IR	21.3 ± 5.1	17.8 ± 4.6	20.3 ± 6.0	18.2 ± 4.1	23.2 ± 9.4	19.5 ± 6.0	22.7 ± 4.1	20.5 ± 5.0
C-R	44.3 ± 9.8	40.5 ± 17.2	41.3 ± 7.6	40.2 ± 4.9	41.5 ± 7.4	53.0 ± 7.5	50.0 ± 5.7	40.8 ± 11.9
t _{1/2}	45.3 ± 8.4	43.7 ± 14.9	44.5 ± 7.9	43.0 ± 7.8	42.7 ± 9.6	54.2 ± 7.5	51.0 ± 5.8	44.7 ± 13.3
MRT	46.2 ± 9.5	41.7 ± 11.7	42.3 ± 7.3	46.0 ± 10.8	44.2 ± 8.7	56.8 ± 6.9	52.3 ± 8.7	39.7 ± 11.1
C-R	180 s							

Table 3

$\dot{V}O_2$ (ml.kg⁻¹.min⁻¹) values

$\dot{V}O_2$	$\dot{V}O_2^a$	t _{1/2} ^a	MRT ^a	<i>k</i> ^a	Steady-state	10 th min
IR	4.8 ± 0.7	68.0 ± 9.1	127.1 ± 7.5	0.5 ± 0.0	34.8 ± 4.0	34.8 ± 4.0
C-R	11.7 ± 0.7	50.1 ± 5.7	57.1 ± 13.9	0.9 ± 0.1	35.9 ± 3.9	35.9 ± 4.2

^a Significant difference ($\alpha < 0.05$) between IR and C-R condition. t_{1/2} and MRT are shown in seconds (s).

Table 4

Mean HR (bpm) values

HR	St HR ^a	t _{1/2}	MRT	<i>k</i>	Steady-state	10 th min
IR	68 ± 10	43.8 ± 10.5	79.4 ± 19.6	0.8 ± 0.3	144 ± 12	144 ± 13

C-R 106 ± 15 48.4 ± 7.6 87.6 ± 15.2 0.7 ± 0.1 150 ± 14 151 ± 14

^a Significant difference ($\alpha < 0.05$) between IR and C-R condition. $t_{1/2}$ and MRT are shown in seconds (s).

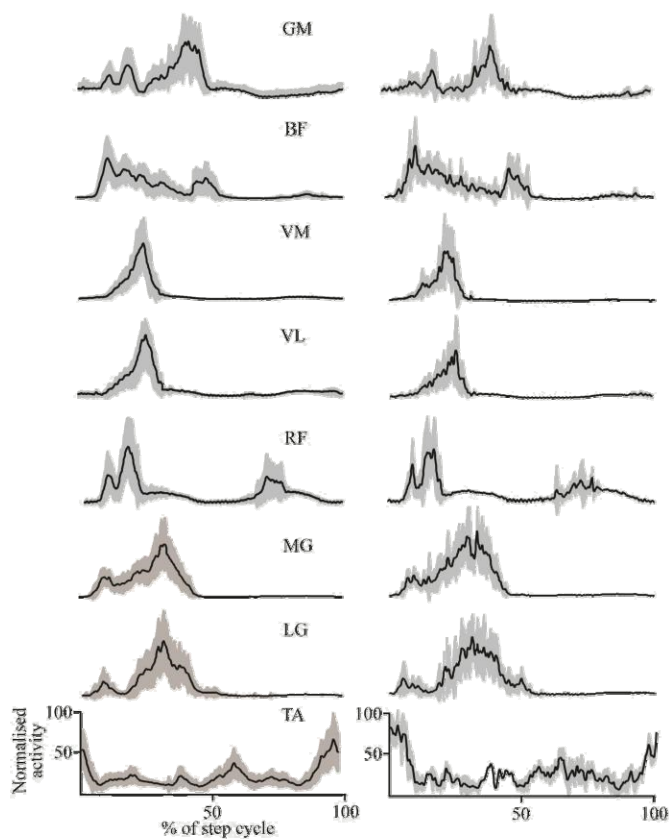
Captions to Illustrations/Figures

Figure 1: Plotted are the mean (\pm SD) EMG traces for all sampling periods. Normalised EMG activity was plotted as a percentage of the maximal recorded EMG in the y-axis and as a percentage of the step (gait) cycle on the x-axis. Mean waveforms (solid black line) were highly similar across each sampling period and variability of EMG traces are represented by the SD (grey shading).

Figure 2: Mean (\pm SE) $\dot{V}O_2$ traces plotted for the IR (●) and C-R (○) conditions from running onset to 180 s.

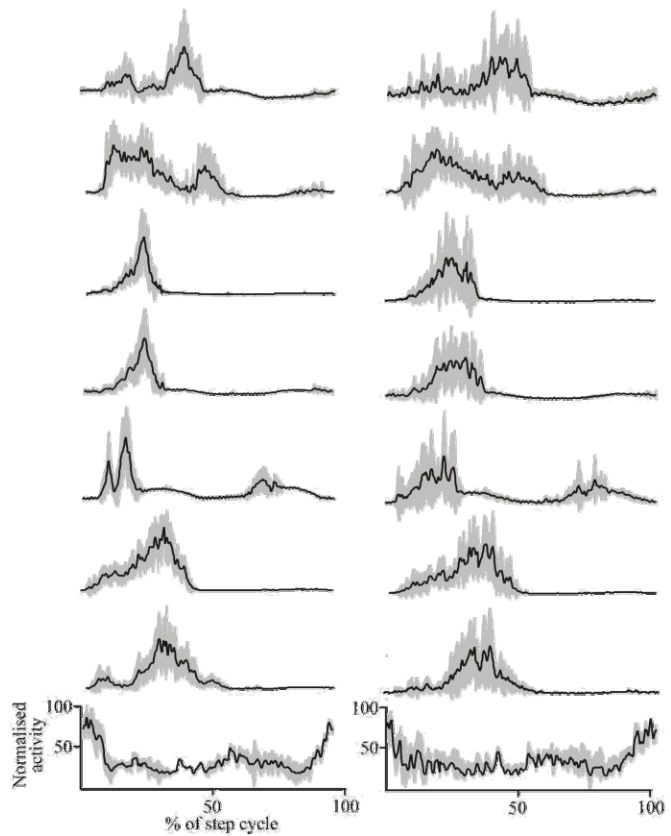
Figure 3: Mean (\pm SE) $\dot{V}O_2$ traces plotted for the IR (●) and C-R (○) conditions from running onset to 180 s.

a. IR

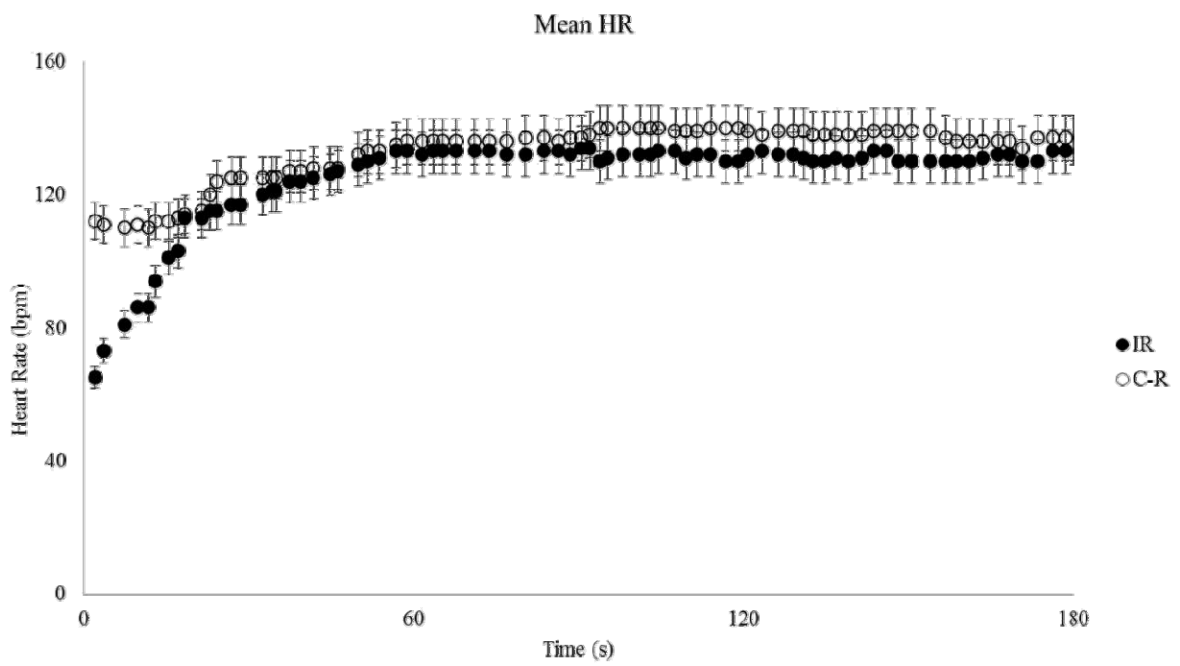
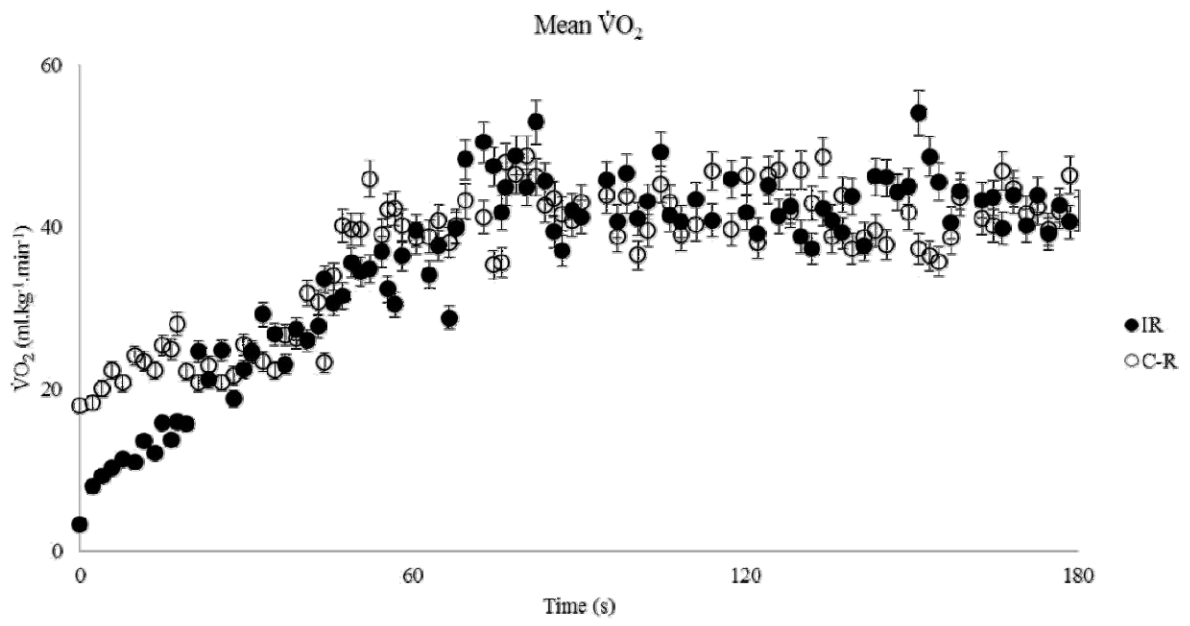


b. $t_{1/2}$

c. MRT



d. 180 s



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