Use of heart rate for estimating the moderate-to-heavy intensity transition during prolonged exercise

by

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Research Questions

What effect does acute prolonged moderate-intensity exercise have on the power output observed at the moderate-to-heavy intensity transition in cyclists and triathletes?

Is the heart rate associated with the moderate-to-heavy intensity transition preserved over-time during prolonged exercise?

Abstract

The moderate-to-heavy intensity transition is commonly measured in individual athletes for purposes of training intensity regulation and load monitoring. The moderate-to-heavy intensity transition is typically expressed using an external work rate metric, such as power output, and an internal work rate metric, such as heart rate. These assessments are often performed in well-rested athletes; however, data from these assessments are used to regulate intensity during prolonged exercise where progressive physiological changes occur over-time. The degree to which these changes occur may vary depending on the so-called 'durability' of the individual athlete. In the context of exercise, durability refers to the time of onset and magnitude of any deterioration in physiological profiling characteristics over-time during prolonged exercise. The acute effect of prolonged exercise on the moderate-to-heavy intensity transition power output and associated heart rate has not been established. Therefore, the aim of this research was to determine the effect of prolonged exercise on moderate-to-heavy intensity transition power output and heart rate. It was hypothesised that the power output at the moderate-to-heavy intensity transition would decrease as a result of acute prolonged exercise, but that proportional cardiovascular drift would occur such that the heart rate associated with the transition would be preserved over-time. Fourteen endurance-trained cyclists and triathletes took part in the present investigation (13 males, 1 female, $\dot{V}O_2$peak 59.9 ± 6.8 $mL \cdot kg^{-1} \cdot min^{-1}$, training volume 9 ± 3 $h \cdot week^{-1}$), which consisted of two laboratory visits. Following a characterisation trial in which the power output at the first ventilatory threshold (VT_1) was estimated, participants undertook a five-stage incremental step test to determine the power output and heart rate at the moderate-to-heavy intensity transition before and after two hours of cycling at 90% of the estimated power output at VT_1. In line with the stated hypothesis, power output at the moderate-to-heavy intensity transition significantly decreased following acute prolonged exercise when determined using expired gases (VT_1, 217 ± 42 W vs. 196 ± 42 W, $P < 0.0001$) and blood lactate concentrations (LoglogLT, 212 ± 47 W vs. 190 ± 47 W, $P < 0.0035$). This was attributable to loss of efficiency (VT_1, -8 ± 10 W; LoglogLT, -7 ± 9 W) and rates of metabolic energy expenditure (VT_1, -14 ± 11 W; LoglogLT, -15 ± 22 W) at the transition. However, in contrast to the hypothesis, the heart rate associated with the moderate-to-heavy intensity transition increased following acute prolonged exercise (VT_1, 142 ± 9 beats.min^{-1} vs. 151 ± 12 beats.min^{-1}, $P < 0.001$; LoglogLT, 140 ± 13 beats.min^{-1} vs. 150 ± 15 beats.min^{-1}, $P < 0.006$). These results demonstrate that the external work output at the moderate-to-heavy intensity transition decreases during prolonged exercise, and that heart rate at the moderate-to-heavy intensity transition increases during prolonged exercise. Therefore, individual assessments of athlete 'durability' are warranted.

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Attestation of Authorship

I hereby declare that this submission is my own work and that, to the best of my knowledge and belief, it contains no material previously published or written by another person (except where explicitly defined in the acknowledgements), nor material which to a substantial extent has been submitted for the award of any other degree or diploma of a university or other institution of higher learning.

Julian Stevenson

Co-authored works

- Julian, 80%; conceived and designed the research, conducted experiments and collected the data, analysed the data, drafted the manuscript.
- Ed, 15%; conceived and designed the research, assisted with data collection, read and approved the manuscript.
- Andrew, 5%; assisted with the conception of the research, read and approved the manuscript.

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Introduction

Endurance athletes commonly perform physiological assessments for estimating the external work outputs that demarcate the boundaries between exercise intensity domains (Maturana et al., 2017). These intensity domain transitions are salient parameters for regulating training and competition intensities, monitoring training load and monitoring adaptations to training (Sanders et al., 2020). The exercise intensity domains are characterised by distinct physiological responses. Exercise in the moderate-intensity domain is characterised by low and stable blood lactate concentrations (Burnley & Jones, 2018), as well as minimal autonomic stress (Seiler et al., 2007). The heavy-intensity domain is characterised by a delayed steady-state in blood lactate concentration that occurs above baseline, whereas in the severe-intensity domain a metabolic steady-state is not achievable and whole-body oxygen consumption ($\dot{V}O_2$), blood lactate concentration, and muscle [H^+] and [Pi] progressively increase, whilst muscle [phosphocreatine] progressively decreases until the limit of tolerance is reached (Black et al., 2017; Jones et al., 2008; Poole et al., 1988; Vanhatalo et al., 2016).

Observational studies have revealed many endurance athletes perform large training volumes in the moderate-intensity domain (Billat et al., 2001, Tonnessen et al., 2014, Mujika, 2017). This may be because exercise in this domain requires only a short recovery time and consequently facilitates a high overall training volume, which may be desirable for elite endurance athletes (Seiler, 2010). If the moderate-to-heavy intensity transition is assessed as 200 W, this information can be used by coaches seeking to prescribe large overall training volumes, as exercise below this transition elicits low autonomic stress (Seiler et al., 2007). Indeed, there is some experimental evidence that training programmes including large volumes of moderate-intensity exercise combined with small volumes of specific heavy and severe intensity exercise produce desirable adaptive responses (Neal et al., 2013; Stöggl & Sperlich, 2014). This approach has been referred to as 'polarised training' (Stöggl & Sperlich, 2014) and therefore requires coaches to identify where the intensity domain transitions exist to programme training according to the desired training intensity distribution. The heavy-to-severe intensity transition represents the highest work rate that can be sustained without a progressive loss of homeostasis (Jones et al., 2008). Specific interval training sessions can be programmed using knowledge of the heavy-severe transition, as work done above the heavy-to-severe transition disturbs muscle metabolic homeostasis (Jones et al., 2008) and triggers specific adaptive signalling cascades (Fiorenza et al., 2018). Accordingly, knowledge of the work rates at the intensity domain transitions are useful for training intensity regulation and training load monitoring.

An under-studied effect of prolonged exercise is the likely reduction in external work rates observed at these intensity transitions over-time. Amongst many other physiological changes, prolonged exercise induces a progressive reduction in muscle glycogen content and increase in circulating non-esterified fatty acid concentration (Hearris et al., 2018), a progressive increase in core and muscle temperatures (Alhadad et al., 2019), and possible progressive dehydration (Goulet, 2012). These

physiological changes may plausibly cause a reduction in the work outputs at which the intensity domain transitions occur. A series of recent studies reported a reduction in the power output at the heavy-to-severe intensity transition following prolonged heavy-intensity cycling (Clark et al., 2018; Clark et al., 2019a; Clark et al., 2019b). The first study (Clark et al., 2018) used a repeated measures design and had, on separate occasions, fifteen cyclists complete a 3-minute all-out test before and after 2 h of heavy-intensity exercise to determine changes in end-test power (EP). A 3-minute all-out cycling test is a maximal, unpaced effort in which participants are instructed and strongly encouraged to maintain the highest possible cadence at all times throughout the test on a cycle ergometer in a linear mode (Burnley et al., 2006). Participants are not informed of the elapsed time to avoid pacing (Vanhatalo et al., 2007). End-test power (EP) is determined as the average power output during the final 30 seconds of the test (Burnley et al., 2006). The EP has been shown to not be significantly different from, and highly correlated with, the heavy-to-severe intensity transition power output (Vanhatalo et al., 2007). The authors reported that EP (282 ± 52 vs. 306 ± 56 W; $P < 0.01$) was significantly decreased after 2 h of heavy-intensity exercise, and therefore that the heavy-to-severe intensity transition power output was reduced following prolonged exercise (Clark et al., 2018). These findings were replicated in a similar, subsequent study involving fourteen cyclists, with the addition of a muscle biopsy before and after the 2 h of heavy-intensity exercise (Clark et al., 2019a). Again, the fatigued EP was ~11% lower than rested EP. As the reduction in muscle glycogen content was not significantly correlated with the changes in EP ($r = 0.19$; $P > 0.05$), the authors therefore concluded that the prolonged exercise-induced reduction in heavy-to-severe intensity transition power was not mechanistically related to changes in muscle glycogen content, although caution was acknowledged given the associational nature of the data (Clark et al., 2019a). The final study of the series investigated the influence of carbohydrate ingestion on EP, and changes in EP before and after a period of 40-min, 80-min, and 2 h of heavy-intensity exercise (Clark et al., 2019b). Sixteen participants ingested a placebo beverage during 40-min, 80-min, and 2 h of heavy intensity cycling prior to a 3-minute all-out test. On another occasion, a CHO supplement was ingested (60 $g \cdot h^{-1}$) during the two hours of heavy-intensity cycling prior to a 3-minute all-out test. In the placebo condition, EP was significantly lowered by 9% after 120 min, but not after 40 min or 80 min. CHO ingestion nullified the reduction of EP following 2 h of heavy-intensity exercise. The pooled data show that muscle glycogen at rest was correlated with control EP, and that muscle glycogen after 2 h of heavy-intensity exercise was correlated with fatigued EP ($r = 0.43$; $P < 0.05$). The change in EP following 2 h of heavy-intensity exercise was not correlated with the change in muscle glycogen. However, the percent change in muscle glycogen following 2 h of heavy-intensity exercise was correlated with the absolute change in work done above EP. These data suggest the heavy-to-severe intensity transition power output is sensitive to CHO availability at specific timepoints, and these associational data may therefore indicate a role for muscle glycogen availability in the heavy-to-severe intensity domain transition power output when fresh, and after prolonged exercise.

Less is understood regarding the moderate-to-heavy intensity transition, with the effects of prolonged exercise on this transition yet to be studied. Plausibly, external work output at the moderate-to-

heavy intensity domain transition may also decline with prolonged exercise due to progressive depletion of endogenous carbohydrate stores. A recently posited mechanism linking muscle glycogen availability to fatigue is the localisation hypothesis, which suggests the subcellular localisation of glycogen, and its specific localised depletion, may impair excitation-contraction coupling, and therefore the contractile function of specific individual muscle fibres (Ørtenblad and Nielsen, 2015; Ørtenblad et al., 2013). In support, fatigue resistance (Nielsen et al., 2009) and measures of tetanic Ca^{2+} handling (Nielsen et al., 2014, Ortenblad et al., 2011) have been correlated with intramyofibrillar glycogen content. Importantly, intramyofibrillar glycogen stores are depleted during exercise at a relatively faster rate than the intermyofibrillar and sub-sarcolemmal stores (Marchand et al., 2007; Nielsen et al., 2011). Therefore, following prolonged exercise, intramyofibrillar glycogen depletion-induced impairment of specific muscle fibres may therefore increase the mechanical and metabolic burden of a given external work rate on the now smaller number of active, fully functional fibres. Speculatively, this could in turn increase the fibre-specific work rate at a given external power output, reducing the external work rate achieved across the muscle as a whole at the moderate-to-heavy intensity transition following prolonged exercise that induces significant intramyofibrillar glycogen depletion.

Cycling gross efficiency is proven to be a crucial determinant of cycling performance (Hopker et al., 2013). Gross efficiency is defined as the percentage of total energy expenditure that is translated into mechanical power (Moseley & Jeukendrup, 2001). Studies have shown a reduction in gross cycling efficiency with prolonged moderate-intensity exercise (Hopker et al., 2017; Passfield & Doust, 2000). Theoretically, with a reduction in gross cycling efficiency over-time during prolonged exercise, the rate of metabolic energy expenditure associated with the moderate-to-heavy transition would produce less external work output. Therefore, even if the rate of metabolic energy expenditure associated with the moderate-to-heavy transition is maintained over-time during a bout of prolonged exercise, the associated external power output would decrease. The contributions made by changes in gross cycling efficiency and rates of metabolic energy expenditure achieved at the moderate-to-heavy intensity transition to prolonged exercise-induced changes in the external power output at the moderate-to-heavy transition have not been quantified. These contributions could be quantified by measurement of the metabolic energy expenditure vs. external power output relationship, and identification of the rate of metabolic energy expenditure associated with the moderate-to-heavy intensity transition, before and after prolonged exercise.

Given the previously observed reductions in heavy-to-severe intensity transition power output with prolonged exercise (Clark et al., 2018, Clark et al., 2019a, Clark et al., 2019b), and the possibility that moderate-to-heavy intensity transition power output may follow a similar pattern with prolonged exercise, the use of well-rested assessments of external work output at intensity domain transitions to regulate training intensity could result in inadvertent drift into a higher intensity domain during prolonged exercise (Maunder et al., 2021). A common strategy employed by endurance athletes and coaches that may combat this possibility is the use of heart rate monitoring to regulate training intensity (Benson & Connolly, 2019). Heart rate can be

easily and accurately measured non-invasively during exercise (Achten & Jeukendrup, 2003), and routine physiological profiling assessments may express intensity domain transitions as associated heart rate values as well as external work outputs (Maunder et al., 2021). Progressive increases in heart rate at given external work rates can occur during prolonged exercise, even when performed in temperate environments; this is referred to as cardiovascular drift (Coyle & Gonzalez-Alonso, 2001). Cardiovascular drift can therefore alter the heart rate vs. power output relationship drastically (Jeukendrup & Diemen, 1998). Accordingly, if an athlete was using heart rate for training intensity regulation and cardiovascular drift was occurring, they may reduce their power output to remain at the target heart rate. Surprisingly, the degree to which cardiovascular drift during prolonged exercise reflects shifts in exercise intensity domain transitions has not been established. Understanding the relationship between cardiovascular drift and acute exercise-induced changes in the external work outputs at intensity domain transitions has implications for whether using heart rates observed at these intensity transitions during typical well-rested assessments is a suitable strategy for training intensity regulation during prolonged exercise.

To my knowledge, no study has investigated the effect of prolonged moderate-intensity exercise on the moderate-to-heavy intensity transition power output and heart rate. The aim of the present study was therefore to determine the acute effects of prolonged exercise on the power output and heart rate associated with the moderate-to-heavy intensity transition in cyclists. I hypothesised that the moderate-to-heavy intensity transition power output would decrease as a result of acute prolonged exercise, but that proportional cardiovascular drift would occur such that the associated heart rate would be maintained. This study has implications for understanding best practices for training intensity regulation during prolonged exercise.

Methods

Subjects

Fourteen endurance-trained cyclists and triathletes took part in the present investigation (13 males, 1 female, age 34 ± 10, height 178.1 ± 5.6 cm, mass 71.2 ± 6 kg, $\dot{V}O_2$peak 59.9 ± 6.8 $mL \cdot kg^{-1} \cdot min^{-1}$, HRmax 183 ± 10 $beats.min^{-1}$, training volume 9 ± 3 $h \cdot week^{-1}$). *A priori* sample size estimation indicated that a total sample size of 15 was required to detect a large magnitude (ES = 0.7) reduction in power output at the moderate-to-heavy intensity transition with 80% statistical power using the G*Power software package (Faul et al., 2009). A large magnitude effect size was used for this calculation based on previous studies showing the effect of prolonged exercise on the heavy-to-severe intensity transition (Clark et al., 2018). A one-tailed test was used as it is implausible that the moderate-to-heavy intensity power output would increase as a result of acute prolonged exercise. All participants understood the information presented about this research project and provided informed consent to take part. Data collection was interrupted by a COVID-19 lockdown and one participant dropped out. All participants were free of recent (<3 months) musculoskeletal injury and chronic disease and habitually training >5 $h \cdot week^{-1}$ in cycling-based endurance sports. This study was performed in accordance with the standards of the Declaration of Helsinki, 2013, and the Auckland University of Technology Ethics Committee approved all procedures (21/253).

Study design

A cross-sectional design was used in the present investigation. Participants visited the laboratory on two occasions in the morning after an overnight fast, approximately one week apart. The initial assessment was used to estimate power output at the first ventilatory threshold (VT_1) and peak oxygen uptake ($\dot{V}O_2$peak). In the experimental trial, participants completed an individualised, incremental test designed to accurately identify the power output and heart rate associated with the moderate-to-heavy intensity transition, before and after a two-hour bout of cycling at 90% of the power output estimated at VT_1 in the initial assessment. The order of the visits was not randomised as the initial assessment was used to define the parameters of the experimental trial.

Initial assessment

Participants initially reported to the laboratory for an incremental cycling test. Participants arrived after a 10-hour overnight fast having refrained from vigorous exercise for 24 hours and having ingested ~1 L of plain water ~2 h beforehand. These pre-trial procedures were necessary to ensure the participants were adequately rested and hydrated for each assessment, as well as for within- and between-participant standardisation. Height and body mass was first measured.

Cycling subsequently commenced on an electromagnetically-braked ergometer (Excalibur Sport, Lode BV, Groningen, NET) at 95 W, and the power output initially increased by 35 W every 3 minutes. Expired gases were collected continuously using indirect calorimetry (TrueOne 2400, ParvoMedics, UT, USA). This was calibrated for volume and gas content using a 3 L syringe and standard gas (~16% oxygen and ~4% carbon dioxide). Although the gold standard of indirect calorimetry is considered to be the Douglas bag technique (Betts & Thompson, 2012), the practicalities of using the Douglas bags made them not favourable for the present work. The Douglas bag technique does not allow for detection of changes in expired gas parameters in short time-intervals *within* stages, such as the ventilatory equivalents used to detect ventilatory thresholds (see below). Automated gas analysers, in contrast, allow for near-instantaneous analysis of expired gas parameters. The ParvoMedics TrueOne 2400 has demonstrated accurate and reliable outcomes for the measurement of gas exchange variables (Crouter et al., 2006). The TrueOne 2400 was not significantly different from the Douglas bag method at rest or during cycling at 50, 100, 150, 200, and 250 W for measurements of $\dot{V}_E$, $\dot{V}O_2$, or $\dot{V}CO_2$ (Crouter et al., 2006). Heart rate was measured continuously using a chest-strap heart rate monitor (Tickr, Wahoo Fitness, Atlanta, USA).

Once the respiratory exchange ratio exceeded 1.0 and clear signs of increased $\dot{V}E{\cdot}\dot{V}O_2^{-1}$ emerged, power output was increased by 35 W every minute until task failure. The $\dot{V}O_2$peak was identified as the highest 15-s average $\dot{V}O_2$, and VT_1 was identified as the $\dot{V}O_2$ at which a systematic rise in $\dot{V}E{\cdot}\dot{V}O_2^{-1}$ occurred. This $\dot{V}O_2$ was converted to a power output by linear fit of the power output vs. $\dot{V}O_2$ relationship, using the last minute of $\dot{V}O_2$ data from each 3-min stage.

The last minute of expired gas data in each 3-min stage was used to determine whole-body fat oxidation rates through standard calculations (Jeukendrup & Wallis, 2005 Eq. 1). The highest observed rate of whole-body fat oxidation was accepted as the peak fat oxidation rate (PFO) (Achten et al., 2002). The PFO measured during fasted, incremental exercise is a useful metric for capturing an athlete's capacity for fat oxidation during exercise, as evidenced by the extra variation explained by its addition to models of endurance performance in recent work (Maunder et al., 2022). Substrate oxidation was calculated using the Jeukendrup & Wallis equation (Jeukendrup & Wallis, 2005 Eq. 1).

$$\text{Whole body fat oxidation rate } (g{\cdot}min^{-1}) = 1.695 \times \dot{V}O_2 - 1.701 \times \dot{V}CO_2$$

Eq. 1 where $\dot{V}O_2$ and $\dot{V}O_2$ are in $L{\cdot}min^{-1}$.

Experimental trial

Participants arrived for the experimental trial after a 10-hour overnight fast, having refrained from vigorous exercise for 24 hours, and having ingested ~1 L of plain water ~2 h beforehand. Following measurement of body mass, the experimental trial commenced on the same electromagnetically-braked

ergometer as the initial assessment with a 5-min warm-up at 100 W, followed by a five-stage incremental assessment to determine the power output and heart rate at the moderate-to-heavy intensity transition. The incremental test began with 4-min at 50 W below the previously estimated VT_1 power output, and power output increased by 25 W per increment.

Expired gases and heart rate was measured continuously (TrueOne 2400, ParvoMedics, UT, USA; Tickr, Wahoo Fitness, Atlanta, USA), and a fingerprick capillary blood sample was obtained in the last 30-s of each increment for measurement of blood lactate concentration using an automated analyser (Lactate Pro 2, Arkray). These data were used to quantify the power output and heart rate at the moderate-to-heavy intensity transition prior to prolonged exercise (PRE, see Data analysis section below). Participants then cycled for 5 min at 100 W, and then at 90% of the previously estimated power output at VT_1 for 2 h. Heart rate was recorded throughout, and participants consumed plain water *ad libitum*. Expired gases were collected for 4 min, every 15 min.

Following the two-hour constant-work rate phase, participants again cycled for 5 minutes at 100 W before repeating the five-step incremental exercise assessment. These data were used to quantify the power output and heart rate at the moderate-heavy intensity transition following prolonged exercise (POST). Sweat loss was also assessed by measurement of pre- and post-exercise body mass, and water consumption. Total water consumption was recorded by measuring the mass of the bottle before and after use and refilling, and was then added to changes in body mass in order to calculate total sweat loss. If participants used the toilet during the trial, body mass was recorded before and after and accounted for in sweat loss calculations.

Estimation of the moderate-to-heavy intensity transition

The PRE and POST moderate-to-heavy intensity transitions were estimated using expired gas and blood lactate data. Specifically, using expired gas data, the moderate-to-heavy intensity transitions in the PRE and POST assessments of the experimental trial were estimated using the VT_1 method in accordance with the procedures described above for the initial assessment. The VT_1 is defined as the rate of oxygen consumption at which the ventilatory equivalent for oxygen ($\dot{V}_E \cdot \dot{V}O_2^{-1}$) began to systematically increase. A rising ventilatory equivalent for oxygen likely reflects accumulation of blood lactate and changes in blood pH, which occur as a result of the increased glycolytic flux characteristic of the heavy intensity domain. Changes in blood pH stimulate peripheral chemoreceptors, driving disproportionate increases in ventilation and hence an increase in $\dot{V}_E \cdot \dot{V}O_2^{-1}$. The $\dot{V}O_2$ at VT_1 was converted to a power output by linear fit of the power output vs. $\dot{V}O_2$ relationship, using the last minute of $\dot{V}O_2$ data from each of the five 4-min stages. Power output was then matched with a heart rate value by linear fit of the power output vs. heart rate relationship, using the average heart rate during the last minute of each 4-min stage. The VT_1 power output was then converted to a rate of whole-body energy expenditure by linear fit of the whole-body energy expenditure vs.

power output relationship. The whole-body rate of energy expenditure was calculated for each power output in the incremental assessment using the average $\dot{V}O_2$ and $\dot{V}CO_2$ in the last minute of each of the five 4-min stages with a stoichiometric equation (Jeukendrup & Wallis, 2005, Eq. 2).

Whole body rate of energy expenditure ($kcal \cdot min^{-1}$) = 0.550 x $\dot{V}CO_2$ - 4.471 x $\dot{V}O_2$

Eq. 2 where $\dot{V}O_2$ and $\dot{V}O_2$ are in $L \cdot min^{-1}$.

Using blood lactate data, the PRE and POST moderate-to-heavy intensity transitions were estimated using the LoglogLT method. The LoglogLT method models a blood lactate concentration vs. power output curve using two segments, and the intersection point of the two lines with the lowest residuals sum of squares is taken as the moderate-to-heavy intensity transition (Beaver et al., 1995, Jamnick et al., 2018). Blood lactate concentrations were measured using the Lactate Pro 2 (Lactate Pro 2, Arkray). The results from a study of five portable analysers (including the Lactate Pro 2 used in the present study) indicated that no single portable analyser is both highly accurate and reliable throughout the range of ~1-23 $mmol \cdot L^{-1}$, although most were in close accord of each other (Bonaventura et al., 2015). All portable analysers had a tendency to underestimate at high concentrations ~15-23 $mmol \cdot L^{-1}$ (-2.16 $mmol \cdot L^{-1}$). From 245 samples, the Lactate Pro 2 had a small positive bias at resting concentrations ~1.0-2.0 $mmol \cdot L^{-1}$ (0.32 $mmol \cdot L^{-1}$), and relatively low bias at high lactate concentrations (Bonaventura et al., 2015). The Lactate Pro 2 is therefore sufficiently reliable within the range of lactate concentrations measured in this study. The LoglogLT power output in the PRE and POST assessments were converted to heart rate, $\dot{V}O_2$, and whole-body rate of energy expenditure values by linear fit of the relationships between these values and power output, as per above.

In order to quantify the proportion of prolonged exercise-induced changes in moderate-to-heavy intensity transition power output associated with changes in gross cycling efficiency and changes in rates of metabolic energy expenditure achieved at the moderate-to-heavy transition, rates of energy expenditure observed at VT_1 and LoglogLT in the *POST* assessment were converted to a power output value using linear regression of the power output vs. energy expenditure relationship for each participant in the *PRE* assessment (denoted as $POST_{EE}PRE_{Eff}$). The $POST_{EE}PRE_{Eff}$ therefore indicates the power output that the rate of metabolic energy expenditure observed at the moderate-to-heavy transition in the POST assessment would have achieved with the level of gross cycling efficiency in the PRE assessment. Accordingly, the proportion of prolonged exercise-induced changes in VT_1 and LoglogLT power output associated with changes in efficiency and rates of metabolic energy expenditure achieved at the transition was calculated using the below equation.

Contribution of change in efficiency = POST - $POST_{EE}PRE_{Eff}$

Contribution of change in metabolic energy expenditure = $POST_{EE}PRE_{Eff}$ – PRE

Eq. 3 where PRE = power output at the moderate-to-heavy transition pre-prolonged exercise, POST = power output at the moderate-to-heavy transition post-prolonged exercise, and $POST_{EE}PRE_{Eff}$ = power output that would be produced in the PRE assessment using the rate of metabolic energy expenditure observed at the moderate-to-heavy transition in the POST assessment.

Statistical analysis

Data are presented as mean ± standard deviation (SD), unless otherwise stated. Normality of data distributions were assessed using the Shapiro-Wilk test, which is appropriate for small sample sizes (Mahibbur Rahman & Govindarajulu, 2010). The effect of prolonged exercise on moderate-to-heavy intensity transition power output, heart rate, $\dot{V}O_2$, and rate of energy expenditure was assessed using paired t-tests (or the non-parametric equivalent Wilcoxon test). Relationships between PRE to POST changes in moderate-to-heavy transition power output and PFO, $\dot{V}O_2$peak, sweat loss, and dehydration were assessed using Pearson's (r) or Spearman's rank-order (r_s) correlation coefficients, depending on normality, and expressed with 95% confidence intervals. Changes in heart rate and whole-body energy expenditure over-time during the two-hour constant-work rate phase were analysed using repeated measures one-way analyses of variance. Whole-body fat oxidation rates during the first three stages of the PRE and POST incremental tests were compared using a mixed model analysis of variance due to missing data-points. Variance was located post-hoc using Holm-Bonferroni stepwise correction. Analyses were performed in GraphPad Prism Version 9.3.1. Significance was inferred when $P \leq 0.05$.

Results

Constant-work rate phase

The estimated power output at VT_1 in the initial assessment was 216 ± 45 W. Consequently, the two-hour constant-work rate phase in the experimental trial was completed at 194 ± 41 W. The heart rate during the two-hour constant-work rate phase significantly increased ($P < 0.0001$, Figure 1a). The rate of whole-body energy expenditure did not increase during the two-hour constant-work rate phase, although this approached significance ($P = 0.07$, Figure 1b).

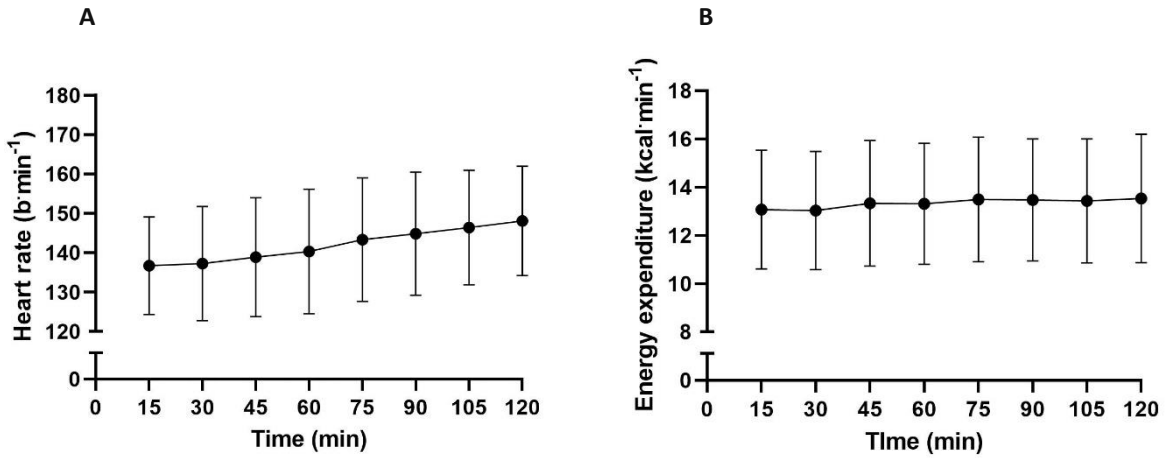


Figure 1 – Heart rate and whole-body energy expenditure during the two-hour constant-work rate phase.

Moderate-to-heavy intensity transition

The power output at VT_1 (217 ± 42 W vs. 196 ± 42 W, $P < 0.0001$, Figure 2a) and LoglogLT (212 ± 47 W vs. 190 ± 47 W, $P < 0.0035$, Figure 2b) significantly decreased from PRE to POST. There were no significant associations between the magnitude of PRE to POST changes in moderate-to-heavy intensity transition power output and selected outcome measures in this study (Table 1).

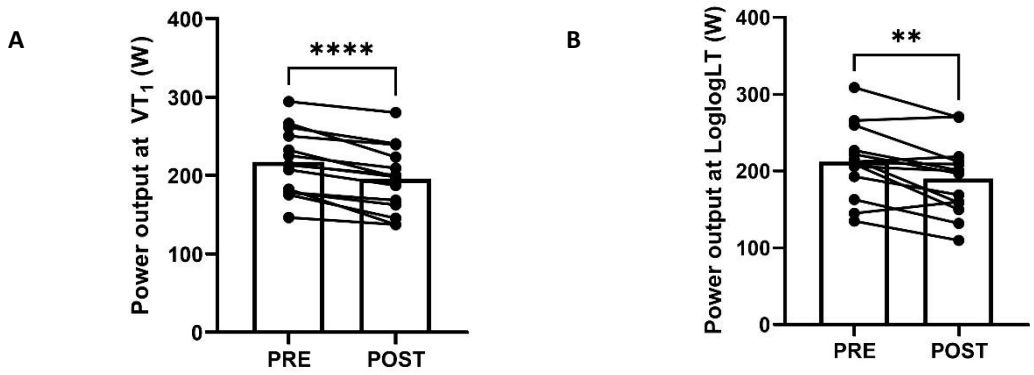


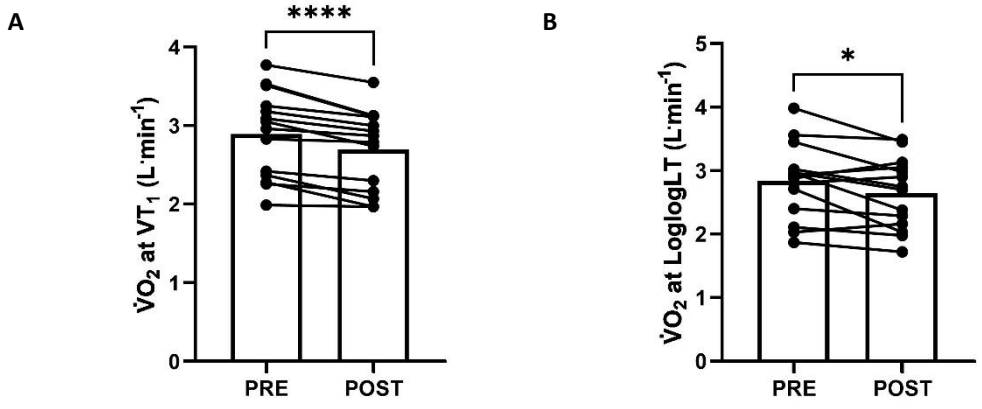
Figure 2 – Power output at the moderate-to-heavy intensity transition in the PRE and POST assessment determined by (a) the first ventilatory threshold (VT_1) and (b) blood lactate concentrations (LoglogLT). Bars indicate mean values and lines indicate individual responses. ** denotes $P \leq 0.01$, **** denotes $P \leq 0.0001$.

Table 1 - Bivariate associations between durability of the moderate-to-heavy transition (Δ VT_1 and Δ LoglogLT) and outcome measures in this study. Data are reported as Pearson's product-moment (r) or Spearman's rank-order (r_s) correlation coefficients (95% confidence intervals), with accompanying P-values.

	Δ VT_1 (W)	Δ LoglogLT (W)
PFO ($g \cdot min^{-1}$)	r_s = -0.13 (-0.63, 0.44) *P* = 0.66	r = 0.37 (-0.20, 0.75) *P* = 0.19
$\dot{V}O_2$peak ($mL \cdot kg^{-1} \cdot min^{-1}$)	r_s = -0.23 (-0.69, 0.36) *P* = 0.43	r = 0.10 (-0.45, 0.60) *P* = 0.73
$\dot{V}O_2$peak ($L \cdot min^{-1}$)	r_s = -0.35 (-0.75, 0.24) *P* = 0.22	r = -0.01 (-0.54, 0.52) *P* = 0.98
Sweat loss (L)	r_s = -0.17 (-0.71, 0.49) *P* = 0.61	r = 0.01 (-0.59, 0.61) *P* = 0.97
Dehydration (% of BM)	r_s = 0.43 (-0.25, 0.82) *P* = 0.19	r = 0.53 (-0.10, 0.86) *P* = 0.09

Abbreviations: Δ LoglogLT = prolonged exercise-induced change in the lactate threshold power output, BM = body mass, PFO = peak fat oxidation rate observed in the initial assessment, $\dot{V}O_2$peak = peak oxygen uptake, and Δ VT_1 = prolonged exercise-induced change in the first ventilatory threshold power output.

The $\dot{V}O_2$ at VT_1 (2.89 ± 0.55 $L \cdot min^{-1}$ vs. 2.69 ± 0.51 $L \cdot min^{-1}$, *P* < 0.0001, Figure 3a) and LoglogLT (2.83 ± 0.59 $L \cdot min^{-1}$ vs. 2.64 ± 0.56 $L \cdot min^{-1}$, *P* < 0.0296, Figure 3b) significantly decreased from PRE to POST. The rate of energy expenditure at VT_1 (14.4 ± 2.7 $kcal \cdot min^{-1}$ vs. 13.5 ± 2.7 $kcal \cdot min^{-1}$, *P* < 0.0002, Figure 3c) and LoglogLT (14.1 ± 2.9 $kcal \cdot min^{-1}$ vs. 13.2 ± 3 $kcal \cdot min^{-1}$, *P* < 0.0246, Figure 3d) significantly decreased from PRE to POST. The decrease in power output at VT_1 and LoglogLT following prolonged exercise was attributable to decreased efficiency and rates of metabolic energy expenditure at the transition (Figure 3e). The relative contribution made by decreased efficiency and rates of metabolic energy expenditure to the decrease in power output at the moderate-to-heavy intensity transition was not significantly different (VT1, *P* = 0.18; LoglogLT, *P* = 0.28).



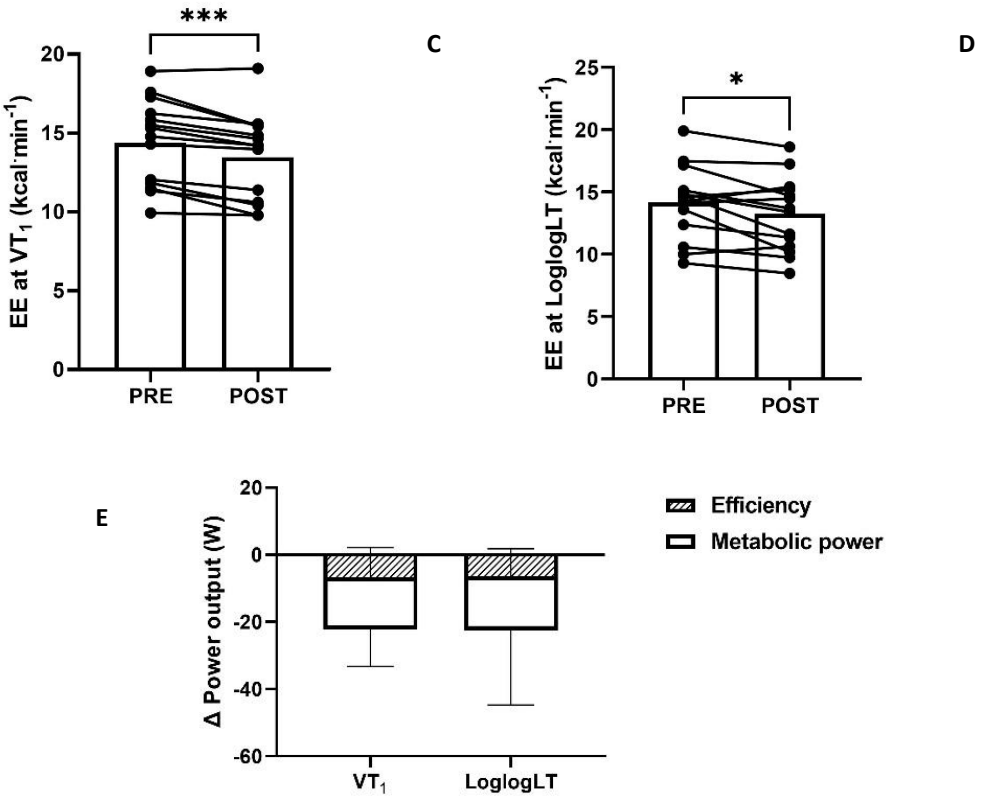


Figure 3 – The $\dot{V}O_2$ and energy expenditure (EE) at the moderate-to-heavy intensity transition in the PRE and POST assessment determined by (a and c) expired gases (VT_1) and (b and d) blood lactate concentrations (LoglogLT). The contributions to prolonged exercise-induced changes in moderate-to-heavy intensity transition power output made by loss of efficiency and metabolic energy expenditure at the transition is shown in (e). Bars indicate mean values and lines indicate individual responses. * denotes $P \leq 0.05$, **** denotes $P \leq 0.0001$.

There was an effect of prolonged exercise on heart rate at the moderate-to-heavy intensity transition. The heart rate at VT_1 (142 ± 9 beats.min^{-1} vs. 151 ± 12 beats.min^{-1}, $P < 0.001$, Figure 4a) and LoglogLT (140 ± 13 beats.min^{-1} vs. 150 ± 15 beats.min^{-1}, $P < 0.006$, Figure 4b) significantly increased from PRE to POST.

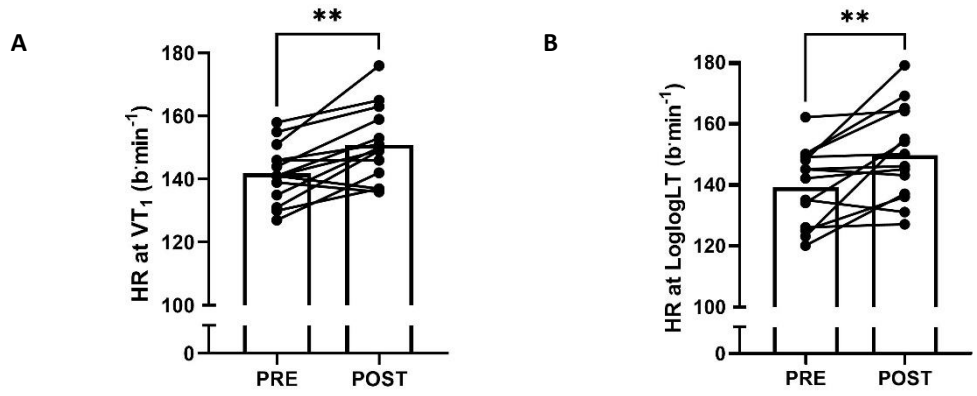


Figure 4 – The heart rate (HR) at the moderate-to-heavy intensity transition in the PRE and POST assessment determined by (a) expired gases (VT_1) and (b) blood lactate concentrations (LoglogLT). Bars indicate mean values and lines indicate individual responses. ** denotes $P \leq 0.01$.

There was an effect of prolonged exercise ($P = 0.04$), and prolonged exercise by intensity interaction ($P = 0.002$), on whole-body fat oxidation rates during the PRE and POST assessments. Specifically, whole-body fat oxidation rates were greater in the POST vs. PRE assessment during the first and second steps (Figure 5).

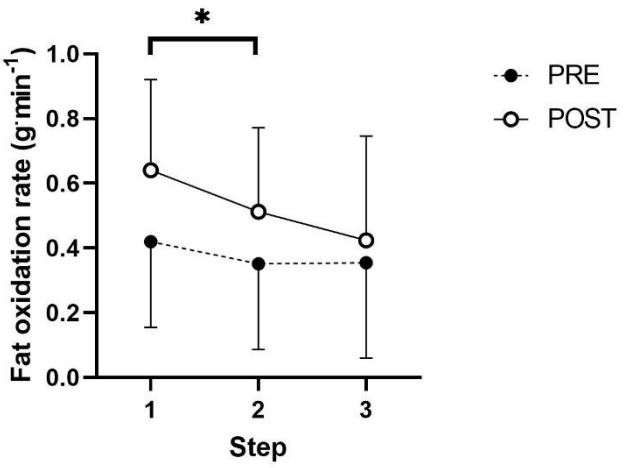


Figure 5 – The whole-body fat oxidation rate ($g \cdot min^{-1}$) in PRE and POST in steps 1, 2, and 3. Circles indicate means and error bars indicate standard deviation. * denotes $P \leq 0.05$ from PRE to POST in steps 1 and 2.

Discussion

The aim of this study was to investigate the effects of two-hours of moderate-intensity cycling on the moderate-to-heavy intensity transition power output and heart rate. In line with the stated hypothesis, power output at the moderate-to-heavy intensity transition significantly decreased following acute prolonged exercise (Figure 2). Cardiovascular drift occurred within the two-hour constant-work rate phase, as heart rate significantly increased (Figure 1a). However, in contrast to the hypothesis, the heart rate associated with the moderate-to-heavy intensity transition increased following acute prolonged exercise (Figure 4). These data have implications for athlete profiling, training load monitoring, and training programming, and indicate that the 'durability' of the moderate-to-heavy intensity transition warrants attention at the individual level.

The observed reduction in moderate-to-heavy intensity transition power output following acute prolonged exercise was accounted for by both reduced efficiency and reduced rates of metabolic energy expenditure at the transition (Figure 3e). The proportion of the prolonged exercise-induced reduction in moderate-to-heavy intensity transition power output associated with loss of efficiency and rates of metabolic energy expenditure at the transition was not significantly different (Figure 3e). Loss of efficiency was demonstrated during the two-hour constant-work rate phase, as whole-body rates of energy expenditure at the fixed power output increased over-time, albeit not significantly (P = 0.07, Figure 1b). Decreased gross cycling efficiency has previously been observed over-time during prolonged exercise, plausibly due to progressive mitochondrial and/or contractile inefficiency (Hopker et al., 2017), and has been linked to impaired performance (Passfield & Doust, 2000).

The remainder of the reduction in moderate-to-heavy intensity transition power output was accounted for by decreased rates of metabolic energy expenditure at the transition (Figure 3e). This is demonstrated by the reduction in rates of energy expenditure as well as $\dot{V}O_2$ at the moderate-to-heavy intensity transition from PRE to POST (Figure 3a-d). Plausibly, the observed reduction in energy expenditure at the moderate-to-heavy intensity transition with prolonged exercise may be at least partially attributable to decreased endogenous carbohydrate availability. It is understood that carbohydrate availability modulates muscle excitability and contractile function (Black et al., 2017). It is likely that the development of peripheral fatigue within the moderate-intensity domain is related to depletion of muscle glycogen and impairment in neuromuscular excitability and transmission (Chin & Allen, 1997; Gejl et al., 2014). Specifically, localised glycogen depletion in the intramyofibrillar compartment may have contributed to the prolonged exercise-induced reduction in metabolic energy expenditure at the moderate-to-heavy intensity transition. Intramyofibrillar glycogen depletion has been linked to impaired excitation-contraction coupling, which manifests as reduced Ca^{2+} release from the sarcoplasmic reticulum under neural innervation (Ørtenblad and Nielsen, 2015; Ørtenblad et al., 2013). Therefore, intramyofibrillar glycogen depletion during the prolonged exercise of the current study may have diminished the function of specific, active individual muscle fibres (Ørtenblad and Nielsen, 2015; Ørtenblad et al., 2013). Indeed, evidence for depletion of endogenous carbohydrate availability is provided by the observed increase in whole-body fat oxidation rates from PRE to

POST (Figure 5), given the autoregulatory nature of muscle glycogen metabolism (Hargreaves et al., 1985). In turn, impaired contractile activity of specific muscle fibres due to intramyofibrillar glycogen depletion may have increased the metabolic burden that a given power output placed on the smaller number of active, fully functional fibres. However, as muscle glycogen depletion, and more specifically compartmental muscle glycogen depletion, was not measured in this study, this mechanism remains speculative and could be interrogated in future work.

Indeed, the reduction in moderate-to-heavy intensity transition power output was not significantly associated with PFO (Table 1). The PFO is a marker of an individual's capacity for fat oxidation during exercise (Maunder et al., 2018), meaning that having a greater capacity to oxidise fatty acids in a fresh state during exercise was not related to 'durability' of the power output at the moderate-to-heavy intensity transition. If the muscle glycogen depletion were the primary mechanism behind the observed prolonged exercise-induced reduction in moderate-to-heavy intensity transition power output, one might have predicted that possessing a greater capacity to oxidise fatty acids during exercise would have mitigated this decline. The absence of a relationship between PFO and the magnitude of the reduction in moderate-to-heavy intensity transition power output therefore appears to counter this proposed mechanism. However, PFO may not accurately reflect the degree of glycogen depletion induced by the prolonged exercise, and therefore future work may consider replicating the design of the present study, but with measurements of muscle glycogen availability in order to test this hypothesis.

These proposed effects of glycogen depletion on the moderate-to-heavy transition may have been exacerbated by the exercise of the present study being conducted after an overnight fast and without carbohydrate intake during exercise, and may plausibly therefore be lessened in training and competition scenarios in which exercise is performed postprandially and with carbohydrate feeding. Additionally, the importance of glycogen availability for durability of the moderate-to-heavy transition could be further explored through repetition of the present protocol with experimental manipulation of pre-exercise glycogen availability through exercise and/or nutrition interventions.

The cardiovascular drift that occurred during the two-hour constant-work rate phase increased by heart rate by 8.2 ± 2.7 % from 15 min to 120 min (Figure 1a). A plausible explanation for this may be prolonged exercise-induced increases in core temperature and therefore a progressive increase in cutaneous blood flow, as well as progressive dehydration and consequently reduced stroke volume (Coyle & Gonzalez-Alonso, 2001). More importantly, in contrast to my hypothesis, it was observed that the heart rate associated with the moderate-to-heavy intensity transition significantly increased from PRE to POST (VT_1, 6.3 ± 5.8 %; LogLogLT, 7.1 ± 9.0 %, Figure 4). These data demonstrate that the cardiovascular drift that occurred with acute prolonged exercise was proportionally larger than the downward drift in the power output associated with the moderate-to-heavy intensity transition, and therefore that the heart rate associated with the moderate-to-heavy intensity transition is not preserved over-time during acute prolonged exercise.

Practical applications

This study has several important practical applications for endurance sport. Firstly, as the power output at the moderate-to-heavy intensity transition decreased following acute prolonged exercise, these data suggest that using a well-rested assessment of power output at the moderate-to-heavy intensity transition for programming prolonged exercise risks inadvertent drift from the moderate into the heavy intensity domain. For example, if an athlete's moderate-to-heavy intensity transition power output was initially assessed at 200 W, but decreased to 180 W after two hours, cycling at 190 W after two hours will become heavy-intensity exercise. This may have implications for training periodisation; specifically, drift into the heavy domain may extend the recovery required after sessions intended to be of moderate intensity (Seiler et al., 2007; Stanley et al., 2013). Additionally, in this example 190 W is not the same physiological intensity when fresh compared to after multiple hours of exercise, ergo training load models may need to consider accounting for the durability of intensity domain transitions in order to better quantify training load (Van Erp et al., 2019; Sanders et al., 2017; Halson, 2014).

These results also suggest use of well-rested assessments of the heart rate at the moderate-to-heavy intensity transition to prescribe prolonged exercise may also be misleading. For example, maintaining the same heart rate with prolonged exercise based on an initial assessment of 150 beats.min^{-1} at the transition may risk 'undertraining' or downward drift within the moderate-intensity domain, if the heart rate associated with the transition increases from 150 beats.min^{-1} after multiple hours of exercise.

Lastly, in the present study there was inter-individual variation in the degree of reduction in moderate-to-heavy intensity transition power output with prolonged exercise (VT_1 power reduction ranging from ~9-44 W, Figure 2). This suggests that the durability of the moderate-to-heavy intensity transition is not a uniform characteristic between-athletes, and thus that profiling prolonged exercise-induced changes in moderate-to-heavy intensity transition power output at the individual level may be useful for capturing an endurance athlete's capabilities. Future work may consider exploring the implications of this characteristic for endurance performance.

Conclusion

In conclusion, the present investigation demonstrated two-hours of constant-load, moderate-intensity cycling significantly reduced the power output observed at the moderate-to-heavy intensity transition. The heart rate associated with this transition increased as a result of prolonged exercise. Therefore, it may be valuable for endurance athletes to measure these effects at an individual level in order to refine physiological profiling, training prescription, and load monitoring.

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Appendices

Appendix I: Participant Information Sheet

Date Information Sheet Produced:

30th June, 2021

Project Title

Durability of the lactate threshold in endurance-trained cyclists and triathletes

An Invitation

Kia ora! My name is Julian Stevenson, I am a Master of Sport, Exercise, and Health student at AUT. I am supervised by Dr. Ed Maunder, who is an applied exercise physiologist and lecturer at AUT. I am delighted to extend you an invitation to participate in my research project taking place at AUT Millennium. My research is interested in assessing how the lactate threshold changes within a long-bout of cycling. This research is being conducted as part of my Masters thesis.

What is the purpose of this research?

The purpose of this study is to investigate how the power output at the lactate threshold responds to a long-bout of cycling. In endurance sport, we typically measure the power output at the lactate threshold when fresh and rested. However, we do not know if this power at lactate threshold is maintained over the course of the long duration training sessions we perform. We also want to determine if changes in heart rate during exercise reflect changes in the lactate threshold. The findings of this research may be used for academic publications and presentations, and will be used in my Masters thesis.

How was I identified and why am I being invited to participate in this research?

You have been identified, and so are being invited to participate, after responding to my recruitment advertisement. You are a 20–50 year-old endurance-trained cyclist or triathlete athlete taking part in cycling and/or triathlon events, habitually training >5 hours per week. We also require participants to have a VO_2max of >50 mL/kg/min, and we will assess this in the first visit to the laboratory. Lastly, I am looking for athletes who have not had a recent (<3 months) musculoskeletal injury, are free of chronic disease, and not a student or client of Dr. Maunder's, or client of mine.

How do I agree to participate in this research?

In order to accept my invitation to participate, you will need to get in touch by email (juliandavidstevenson@gmail.com) to arrange your first visit to AUT Millennium. You will first have the opportunity to ask questions about the study and have them answered. If you are still keen to take part in the study, you will be given an informed consent form and then asked to complete a health screening questionnaire to ensure your suitability. If suitable, I will then ask you to perform an incremental exercise test in our laboratory.

Your participation in this research is voluntary (it is your choice) and whether or not you choose to participate will neither advantage nor disadvantage you. You are able to withdraw from the study at any time, including after having completed the first study visit. If you choose to withdraw from the study, then you will be offered the choice between having any data that is identifiable as belonging to you removed or allowing it to continue to be used. However, once the findings have been produced, removal of your data may not be possible.

What will happen in this research?

As part of this study, we will ask you to report to our laboratory at AUT Millennium on the North Shore on two occasions, approximately one week apart, both first thing in the morning after an overnight fast. As mentioned above, the first visit will be for an incremental exercise test or VO_2max test to determine your aerobic fitness, physiological thresholds, and carbohydrate and fat metabolism during exercise performed

on a lode ergometer. It is important that you are well-rested for this visit, having refrained from vigorous exercise for 24 hours. We also ask that you arrive for this visit after an overnight fast, having had nothing to eat or drink other than water for 10 hours, and having had ~1 litre of water to drink ~2 hours before coming in. In this visit we will assess if your VO2max meets our inclusion criteria (>50 mL/kg/min). If it doesn't, you will not be able to continue in the study, but can still get a study report if you would like one.

In the second visit, approximately one week later, we will ask you to again return to the laboratory after an ~10-hour overnight fast, having refrained from vigorous exercise for 24 hours, and having had ~1 litre of water to drink ~2 hours before coming in. We will first perform a lactate threshold test, which involves cycling for 4 min at progressively increasing power outputs. With a 5-min warm-up and 5-min cool-down, this component of the trial will last ~30 minutes. During this time, you will be breathing through a snorkel-type device such that we can collect all the air you breathe out. I will also prick your finger using a lancet a total of five times and use a very small amount of blood for its lactate concentration. You will then ride at a steady power output for two hours, and this specific power output will only be of moderate-intensity and based on the results of your incremental exercise test. During this time, you will be able to drink water, but not eat. Following the two hours of steady riding, we will repeat the lactate threshold test, exactly as described.

What are the discomforts and risks?

As you are a trained cyclist, the discomforts and risk associated will be minimal. The exercise itself will not be anything you do not do on a regular basis. The finger-prick capillary blood sampling is unlikely to cause any discomfort at all, and is a very safe procedure. This is the same procedure that diabetic individuals would use to measure their blood sugar at home. You are welcome to rest, eat, and drink in one of our consultation rooms after the exercise tests if you would like to.

How will these discomforts and risks be alleviated?

Both exercise sessions will be supervised and monitored. Whilst we do not expect any discomfort or pain, sessions can be ceased at any time for any reason by either the researcher or the participant.

What are the benefits?

At the individual level this research will provide valuable information to the participants. As you are an endurance athlete who regularly trains and competes, it will be advantageous to better understand your own unique physiology and performance. Participants have the opportunity to get advice on their training and the latest science from the researchers, and will be provided with an individualised written report on how the information generated from their participation in the study can be integrated into your training going forward. For us as researchers, I hope to publish the results of this study in an academic journal.

What compensation is available for injury or negligence?

In the unlikely event of a physical injury as a result of your participation in this study, rehabilitation and compensation for injury by accident may be available from the Accident Compensation Corporation, providing the incident details satisfy the requirements of the law and the Corporation's regulations.

How will my privacy be protected?

Your data will remain confidential at all times. Upon enrolment into this study, you will be given a randomised number which will be used to identify any data associated with you. Therefore, your name or any other identifier will not be connected to any of the data obtained.

What are the costs of participating in this research?

Participating in this study will require ~5 hours spread over the two visits. The first visit will last ~1 hour, and the second visit ~4 hours.

What opportunity do I have to consider this invitation?

You have up to one month to decide to participate in this study.

Will I receive feedback on the results of this research?

Each participant will be provided with their individual data and results, with an explanation of how this study affected them at the individual level. This information will also be sent via email.

What do I do if I have concerns about this research?

Any concerns regarding the nature of this project should be notified in the first instance to the Project Supervisor, *Ed Maunder, ed.maunder@aut.ac.nz, +64 2108094015*

Concerns regarding the conduct of the research should be notified to the Executive Secretary of AUTEC, *ethics@aut.ac.nz* , (+649) 921 9999 ext 6038.

Whom do I contact for further information about this research?

Please keep this Information Sheet and a copy of the Consent Form for your future reference. You are also able to contact the research team as follows:

Researcher Contact Details:

Julian Stevenson, juliandavidstevenson@gmail.com

Approved by the Auckland University of Technology Ethics Committee on *type the date final ethics approval was granted*, AUTEC Reference number *type the reference number*.

Appendix II: Consent Form

Project Supervisor: ***Dr. Ed Maunder***

Researcher: ***Julian Stevenson***

- ○ I have read and understood the information provided about this research project in the Information Sheet dated 30th June 2021.
- ○ I have had an opportunity to ask questions and to have them answered.
- ○ I understand that taking part in this study is voluntary (my choice) and that I may withdraw from the study at any time without being disadvantaged in any way.
- ○ I understand that if I withdraw from the study then I will be offered the choice between having any data or tissue that is identifiable as belonging to me removed or allowing it to continue to be used. However, once the findings have been produced, removal of my data may not be possible.
- ○ I am not suffering from heart disease, high blood pressure, any respiratory condition (mild asthma excluded), any illness or injury that impairs my physical performance, or any infection.
- ○ I understand that if my VO2max, as measured in the first visit, does not meet the inclusion criteria (>50 mL/kg/min), I will not be able to continue in the study.
- ○ I am not a student or client of Dr. Maunder's or Julian's.
- ○ I agree to provide capillary blood samples.
- ○ I understand that my data will be destroyed 10 years after completion of the study.
- ○ I agree to take part in this research.
- ○ I wish to receive a summary of the research findings (please tick one): Yes○ No○
- ○ I wish to have my capillary blood samples returned to me in accordance with right 7 (9) of the *Code of Health and Disability Services Consumers' Rights* (please tick one): Yes○ No○

Participant's signature: .....

Participant's name: .....

Participant email : .....

Date: .....

Approved by the Auckland University of Technology Ethics Committee on type the date on which the final approval was granted AUTEC Reference number type the AUTEC reference number.

Appendix III: Pre-trial screening questionnaire

1. What is your sex?
    a. ……..
2. What is your age?
    a. ……..
3. Are you currently advised not to undertake vigorous exercise?
    a. ……..
4. Do you suffer from any chronic disease impacting for maximal exercise? E.g. Cardiovascular disease, kidney disease, etc.
    a. ……..
5. Are you taking medication for blood pressure or your heart?
    a. ……..
6. Do you feel pain in your chest when you undertake physical activity?
    a. ……..
7. In the last month have you had pain in your chest when not doing exercise?
    a. ……..
8. Do you ever lose balance because of dizziness or do you ever lose consciousness?
    a. ……..
9. Have you had a feverish illness in the last month?
    a. ……..
10. Do you currently have a musculoskeletal injury?
    a. ……..
11. Approximately how many hours per week do you normally train (e.g. over the last ~8 weeks)?
    a. ……..
12. How would you classify your current performance level?
    a. Recreational (train but do not participate in competition)
    b. Amateur (enter races but do not expect to win)
    c. High-level amateur (qualify and compete at National Championship level)
    d. Elite non-professional (qualify and compete at the international level)
    e. Professional
13. What is your preferred endurance event (e.g. Olympic-distance triathlon, road cycling, etc)?
    a. ……..

Signed (participant):

…… Date: ……..

Signed (researcher):

…… Date: ……..