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Decrements in Cycling Performance are Dependent on the Intensity and Duration of Prior Exercise

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Decrements in Cycling Performance are Dependent on the Intensity and Duration of Prior Exercise

by

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Abstract

The maximal lactate steady state (MLSS) is a physiological model that can be used to delineate the heavy- from the severe-intensity domain of exercise. The importance of this threshold has been repeatedly demonstrated, as it differentiates intensities that result in stable physiological responses from those that progress towards maximal values. To understand the implications that exercise performed at this intensity has on subsequent performance, a recent study demonstrated that time-to-exhaustion (TTE) performance is reduced following prior exercise performed at the power output (PO) corresponding to MLSS ($MLSS_p$) and reduced even further following a small (i.e., 10 watt (W)) increase above $MLSS_p$. Therefore, the purpose of this thesis was to determine whether submaximal intensities (i.e., sub-$MLSS_p$) as well as different durations at $MLSS_p$ would have any impact on subsequent TTE performance, within the severe-intensity domain. The results from this thesis demonstrated curvilinear and linear reductions in TTE performance following increasse in the intensity and duration of the preceding bout of exercise, respectively. These results highlight that the capacity within the severe-intensity domain is dependent on both the intensity and duration of a prior bout of exercise. Moreover, these findings also illustrate the importance of properly determining thresholds and the exercise intensity domains on an individual basis, as small changes in the PO surrounding MLSS have detrimental effects on performance. Lastly, this thesis also highlighted the importance of perceptual responses during exercise. Rating of perceived exertion (RPE) was associated with the reduction in TTE performance for both the intensity and duration conditions, indicating that the capacity within the severe-intensity domain can be estimated by RPE assessed immediately prior to each TTE trial.

Preface

This thesis is original work by the author, Madison Fullerton, and is based on the following scientific manuscript.

Chapter Three of this thesis was submitted as Fullerton, M. M., Passfield, L., MacInnis, M. J., Iannetta, D. & Murias, J. M. Prior exercise impairs subsequent performance in an intensity- and duration- dependent manner. *J Sport Health Sci.*

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List of Symbols, Abbreviations and Nomenclature

Abbreviation	Definition
$[La^-]_b$	Blood Lactate Concentration
[PCr]	Concentration of Phosphocreatine
$[P_i]$	Phosphate Ions
ATP	Adenosine triphosphate
CP	Critical Power
$Delta_{50}$	PO associated with the halfway point between LT and $MLSS_p$
f_R	Breathing Frequency
GET	Gas Exchange Threshold
H^+	Hydrogen Ions
HR	Heart Rate
LT	Lactate Threshold
$LT_{-5\%}$	PO associated with 5 % below LT
MLSS	Maximal Lactate Steady State
$MLSS_{+5\%}$	PO associated with 5 % above $MLSS_p$
$MLSS_{15}$	15 min at the PO corresponding to $MLSS_p$
$MLSS_{45}$	45 min at the PO corresponding to $MLSS_p$
$MLSS_{-5\%}$	PO associated with 5 % below $MLSS_p$
$MLSS_p$	PO associated with $MLSS_p$
O_2	Oxygen
PCO_2	Partial Pressure of CO2
PO	Power Output
PO_2	Partial Pressure of O_2
PPO	Peak Power Output
RCP	Respiratory Compensation Point
RER	Respiratory Exchange Ratio
RPE	Rating of Perceived Exertion
TTE	Time-to-exhaustion
TTE_b	Baseline TTE
TTE_{CP}	TTE at CP
$TTE_{Delta50}$	TTE following $Delta_{50}$
$TTE_{LT-5\%}$	TTE following $LT_{-5\%}$
$TTE_{MLSS+5\%}$	TTE following $MLSS_{+5\%}$
TTE_{MLSS15}	TTE following $MLSS_{15}$
TTE_{MLSS45}	TTE following $MLSS_{45}$
$TTE_{MLSS-5\%}$	TTE following $MLSS_{-5\%}$
TTE_{MLSSp}	TTE following $MLSS_p$
$\dot{V}CO_2$	Carbon Dioxide Output
$\dot{V}_E$	Pulmonary Minute Ventilation
$\dot{V}O_2$	Pulmonary Oxygen Uptake
$\dot{V}O_{2max}$	Maximal Oxygen Uptake
$\dot{V}O_{2peak}$	Peak Oxygen Uptake
W	Watt

Epigraph

Surround yourself with the dreamers and the doers, the believers and thinkers, but most of all, surround yourself with those who see the greatness within you, even when you don't see it yourself.

Edmund Lee

Chapter One: Introduction

The intensity domain schema was first introduced by Whipp and Mahler (1980), who differentiated three domains of exercise intensity, moderate, heavy, and severe (1). Since the work of these two physiologists, it has been suggested that the domains can be demarcated by the gas exchange threshold (GET) or the first lactate threshold (LT) (2, 3) and critical power (CP) or the maximal lactate steady state (MLSS) (4, 5) (Figure 1.1). Since 1980, the physiological responses within each intensity domain have been characterized by many researchers, such that moderate-, heavy-, and severe-intensity exercise is commonly prescribed at 40-59 %, 60-84 %, and > 85 % of maximal oxygen consumption ($\dot{V}O_{2max}$) (6).

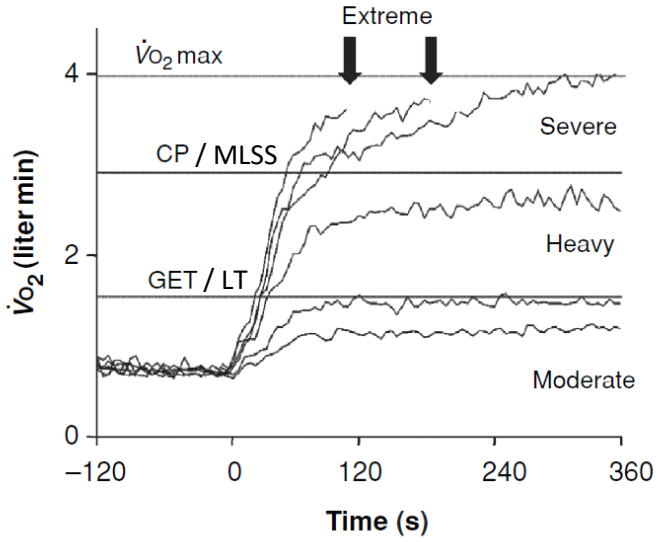

Figure 1.1. The moderate-, heavy-, and severe-intensity domains can be separated by the gas exchange and lactate thresholds (GET and LT, respectively) and the critical power (CP) or maximal lactate steady state (MLSS). Adapted from Wilkerson et al., 2004 (7) and Poole et al., 2012 (8).

The demarcation between the heavy- and severe-intensity domains (i.e., CP and MLSS) is especially important, as this intensity is suggested to delineate stable from unstable physiological responses, such as the response in oxygen consumption ($\dot{V}O_2$) and blood lactate

concentration ($[La^-]_b$) (9, 10). Therefore, from herein, MLSS and CP may be referred to as the "critical intensity of exercise." Specifically, the physiological model of MLSS has traditionally been proposed as the highest intensity of exercise where the production of $[La^-]_b$ is matched by its rate of removal (11). Although the importance of MLSS and CP is well-established (9, 11), the implications that prior exercise at this intensity has on subsequent performance is under researched, specifically pertaining to the MLSS model.

Recently it was demonstrated that prior exercise performed at the power output (PO) corresponding to MLSS ($MLSS_p$) significantly reduced subsequent time-to-exhaustion (TTE) performance (10). Moreover, this study also illustrated a further reduction in TTE following exercise performed only 10 watts (W) above $MLSS_p$. These results highlight the importance of properly determining MLSS, as small adjustments in the PO surrounding this intensity have detrimental effects on subsequent performance. By accurately determining MLSS, and therefore, the exercise intensity domains on an individual basis, the metabolic stimulus can be standardized amongst individuals when prescribing exercise. Furthermore, it is unknown whether prior exercise performed at intensities within each domain, specifically at sub-MLSS intensities, has any effect on subsequent performance and serves as the main purpose of this thesis.

The following chapter contains a literature review summarizing the current understanding of the intensity domain schema and the critical intensity of exercise, as both concepts pertain to exercise performance. The third chapter contains a manuscript that was submitted to the Journal of Sport and Health Science in July of 2020 and is currently under revision. Lastly, a concluding chapter addressing the limitations, future directions, and practical application of the thesis.

Chapter Two: Literature Review

2.1 The Exercise Intensity Domain Schema and Related Thresholds

2.1.1 The moderate-intensity domain

Exercise performed below LT can be defined as in the moderate-intensity domain, where exercise is sustainable for upwards of 4 hours (12). During the commencement of exercise within this domain, $\dot{V}O_2$ rises monoexponentially and achieves steady state within 2 to 3 min (13–15). Similarly, due to the increased metabolic demand at the onset of exercise, $[La^-]_b$ rises initially but is quickly oxidized and returned to baseline values where steady state is maintained (14). It has also been shown that within the first couple of minutes of moderate-intensity exercise, new steady state values are achieved for pH and the concentration of phosphocreatine ([PCr]) and phosphate ions ($[P_i]$) (2, 16). Even when exercise is performed to exhaustion, it has been shown that substrates and metabolites, such as [PCr] and the concentration of hydrogen ions ($[H^+]$), do not reach critical levels (2, 17). Moreover, corresponding with exercise intolerance, it has been suggested that the development of peripheral fatigue within the moderate-intensity domain may be related to the depletion of muscle glycogen (2). Briefly, neuromuscular fatigue during exercise can be defined as a reduction in a muscles ability to produce force or power (18). Specifically, central and peripheral mechanisms of fatigue may be related to a reduction in neural activation of the muscle (18) and an impairment in the contractile properties of the muscle (19), respectively.

During submaximal exercise (i.e., below $MLSS_p$), oxidative phosphorylation almost exclusively supports the energy required for a given bout of exercise (11). One way of evaluating substrate metabolism during exercise is by assessing the respiratory exchange ratio (RER). RER can be used to estimate whole body substrate metabolism by evaluating the ratio between carbon

dioxide production ($\dot{V}CO_2$) and $\dot{V}O_2$ (20). Typically, a RER value of 0.7 would be indicative of fat metabolism, whereas a RER of 1.0 would highlight that substrate metabolism is dominated by the breakdown of carbohydrates. Below LT, the oxidation of fats (plasma free fatty acids and intramuscular triglycerides) and carbohydrates (blood glucose and muscle glycogen) both contribute significantly to substrate metabolism, as indicated by an average RER value of 0.85 (20, 21). Although carbohydrate metabolism becomes progressively important as the intensity of exercise increases, during moderate-intensity exercise (i.e., below LT), total fat oxidation is at its highest as a fuel source, and it is especially important for prolonged durations within this domain (20–22).

2.1.2 The gas exchange and lactate threshold

Both LT and GET can be used to demarcate the threshold that separates the moderate- from the heavy-intensity domain. This threshold represents an important turning-point, as the $\dot{V}O_2$ slow component begins to develop (17, 23), $[La^-]_b$ remains elevated above baseline (24), there is an increased but stable production of metabolites (2), and a greater energy transfer from blood glucose and muscle glycogen (21).

The LT and GET are independent measures determined using either $[La^-]_b$ or ventilatory responses during an incremental exercise test, respectively. LT is typically established during a step-incremental exercise test (25), where on average, each stage is performed for 3 min and $[La^-]_b$ is assessed at the end of each stage prior to an increase in PO (i.e., 25 W per stage). Through this procedure, it is suggested that LT can be defined as the first sustained rise in $[La^-]_b$ above baseline (24). Despite the ambiguity of the prior definition, Faude et al. (24), indicated that LT has also been defined by several researchers as “the first significant/ marked/ systematic/ non-linear/ sharp/ abrupt/ sustained increase in $[La^-]_b$ above baseline” (26–36). Although the method

outlined above has traditionally been used to define LT, it has been suggested that GET may be used to estimate the $\dot{V}O_2$ at LT (37).

In order to estimate LT from GET, gas exchange and ventilatory responses from an incremental exercise test are plotted against $\dot{V}O_2$. The $\dot{V}O_2$ at GET can be identified as the point at which $\dot{V}CO_2$ begins to increase disproportionately in relation to $\dot{V}O_2$ (37) (Figure 2.1), with a systematic rise in the ventilation ($\dot{V}_E$)/$\dot{V}O_2$ ventilatory equivalent and end-tidal partial pressure of oxygen (PO_2), whereas the $\dot{V}_E/\dot{V}CO_2$ ventilatory equivalent and end-tidal partial pressure of carbon dioxide (PCO_2) remain stable (37) (Figure 2.1). Once GET is established, it has been suggested that the $\dot{V}O_2$ at LT can be estimated from the determined GET as a result of the relationship between $[La^-]_b$ and $\dot{V}CO_2$ (37). To explain further, during an incremental exercise test, a sustained increase in $[La^-]_b$ above baseline results in a disproportionate increase in $\dot{V}CO_2$ compared to $\dot{V}O_2$. The increased $\dot{V}CO_2$ response, which is measurable in the breath (i.e., metabolic cart), may be indicative of the buffering of excess H^+ ions by bicarbonate (37).

In this thesis, the term LT (as derived from the GET) was used to define the demarcation between the moderate- and heavy- intensity domains.

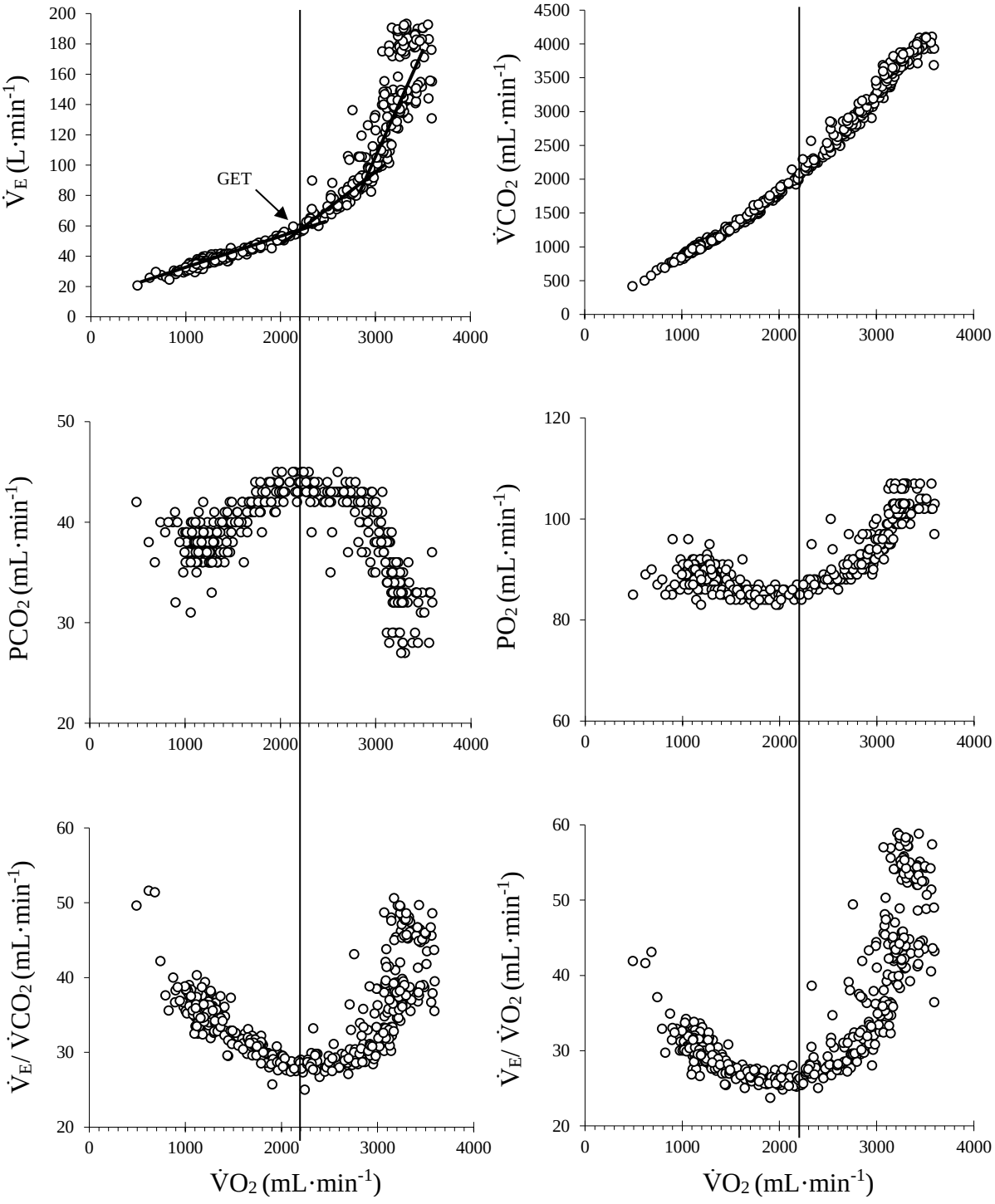

Figure 2.1 Raw ventilatory and gas exchange variables from a ramp-incremental exercise test for the determination of GET. Each variable is plotted against oxygen uptake ($\dot{V}O_2$), and includes ventilation ($\dot{V}_E$), carbon dioxide production ($\dot{V}CO_2$), end-tidal partial pressure of carbon dioxide (PCO_2) and oxygen (PO_2), and $\dot{V}_E/\dot{V}CO_2$ and $\dot{V}_E/\dot{V}O_2$ ventilatory equivalents.

2.1.3 The heavy-intensity domain

In between LT and MLSS lies the heavy-intensity domain. Contrary to exercise performed below LT, within the heavy-intensity domain there is a marked delay in the attainment of a steady state $\dot{V}O_2$ and $[La^-]_b$ response (38–40), with the ceiling of this domain defined by the PO that elicits the highest but stable $[La^-]_b$ (i.e., MLSS) (11). The significant delay in the achievement of a steady state $\dot{V}O_2$, by as long as 15 min, is due to the development of a slow component in the $\dot{V}O_2$ response (13, 15, 17, 23, 39–41).

The $\dot{V}O_2$ slow component has been suggested to represent a sustained alteration in muscle and blood acid base status (17). Specifically, until steady state $\dot{V}O_2$ is achieved, there is a greater contribution from anaerobic energy sources to the energy transfer as the magnitude of the increase in intensity progresses (17, 39, 42). It has also been suggested that a continual reduction in muscle [PCr] (41), a progressive increase in motor unit recruitment (43), and an increased adenosine triphosphate (ATP) and oxygen (O_2) cost for power production in fatigued muscle fibres (17) contribute to the $\dot{V}O_2$ slow component.

Numerous studies have shown that relative to moderate-intensity exercise, there are greater metabolic perturbations associated with exercise performed within the heavy-intensity domain (2, 10, 16, 17, 44). Although over time these metabolic perturbations ultimately result in the achievement of new steady state values, they may be associated with the development of fatigue and ultimately a reduced ability to perform when the duration of exercise increases (2, 10, 17, 44). These metabolic perturbations may include significant reductions in [PCr], pH, and stored glycogen and increases in $[La^-]_b$, all of which have been shown to coincide with exercise intolerance within the heavy-intensity domain (2). Moreover, during prolonged submaximal exercise performed within the heavy-intensity domain, it has been suggested that the progressive

depletion of muscle glycogen significantly contributes to a reduction in performance capabilities (45, 46).

As mentioned, as the intensity of exercise increases so does the utilization of carbohydrates for substrate metabolism. The increased reliance on carbohydrates is a result of the insufficiency of fat metabolism at higher intensities, as fats are not able to be oxidized at sufficiently high rates (21, 22). Although fats may not be the main fuel source during heavy-intensity exercise, both plasma free fatty acids and intramuscular triglycerides contribute significantly to total fat oxidation within this domain (47, 48). At the ceiling of the heavy-intensity domain, represented by MLSS, RER values are close to 1.0, indicating that carbohydrate metabolism is dominating energy turnover, specifically the breakdown and utilization of muscle glycogen (11, 20, 49).

2.1.4 The 'critical intensity': The maximal lactate steady state and critical power

The $MLSS_p$ can be defined as the highest intensity of exercise where $[La^-]_b$ production and oxidation are in equilibrium, resulting in a steady-state response (11, 50–53). On average, exercise performed at $MLSS_p$ can be sustained for ~ 60 min (44). When viewed as a threshold, MLSS and other concepts such as CP, respiratory compensation point (RCP), and anaerobic threshold, have all been suggested to demarcate the heavy- and severe-intensity domains (4, 51, 54, 55). Theoretically, at MLSS, oxidative metabolism primarily accounts for the total energy required, without a considerable contribution from anaerobic resources (24). Exercise performed above this PO results in a disproportionate increase in the glycolytic rate and the progressive accumulation of $[La^-]_b$ (10, 11).

Moreover, the determination of MLSS is relatively laborious, as participants are required to visit the laboratory on numerous occasions in order to accurately determine $MLSS_p$ (56, 57).

In 1985, a group of physiologists defined MLSS as the change in $[La^-]_b$ of less than or equal to 1 $mmol \cdot L^{-1}$ from the 10^{th} to 30^{th} min of exercise during a 30 min constant-PO cycle ergometry test (56). This criterion outlined by Heck et al. (56), is the most commonly accepted approach for determining MLSS (5, 10, 24, 50, 53, 58–60).

Another approach to determine the critical intensity of exercise is by evaluating CP. In this model, CP represents the division between intensities that can be sustained for long periods of time from those that result in exercise intolerance (61). Coinciding with these intensities, CP has been shown to separate stable from unstable physiological responses, such as $[La^-]_b$ and $\dot{V}O_2$ (9). Briefly, the most common method for determining CP involves 4 to 5 constant-PO trials performed to exhaustion, with the goal of having an equal distribution of TTE trials ranging from 1 to 20 min in duration (62, 63). The TTE trials are then fitted, most commonly, with a two-parameter hyperbolic model for the determination of CP. Another important outcome measure of the CP model is the concept known as W' (62). W' represents the fixed amount of work that can be performed above CP before reaching exercise intolerance (64). Moreover, W' notionally represents the finite amount of energy available from high energy phosphates and it is believed to be linked to anaerobic metabolism (65, 66).

Many physiologists believe that MLSS and CP both represent the highest intensity of sustainable exercise (5, 53, 60). However, numerous studies have reported that CP over-estimates the PO at MLSS (5, 10, 11, 59, 60, 67–69), and in a couple of cases by as much as an average of 20 W (59, 60). The typical measurement error with the model predicted CP is approximately 5 % (55). The discrepancy in the PO at MLSS and CP may be a direct result of the different methods of determination (63), as outlined above. Moreover, a disadvantage of the CP model is that no physiological measures are obtained during the determination of CP, such as

$[La^-]_b$ (69–72). Therefore, this mathematical model may be used to predict a physiological state without evaluating any physiological responses during the determination of the critical intensity of exercise.

Like CP, MLSS is determined experimentally and is not without a certain degree of measurement error. With MLSS one major source of error is the experimental change in PO between determination trials. For example, if using a 10 W increment between $MLSS_p$ determination trials, $MLSS_p$ may be underestimated by as much as 9 W (10). Therefore, the true $MLSS_p$ may occur at any PO within the chosen increment range. Another limitation of this model is that typically $MLSS_p$ is solely defined by a few $[La^-]_b$ measures. There can be measurement error when using analyzers to evaluate $[La^-]_b$ and therefore, when adopting the traditional criteria of a change in $[La^-]_b$ of less than or equal to 1 $mmol \cdot L^{-1}$, it is possible that $MLSS_p$ may be over- or underestimated (3). Moreover, exercise performed at intensities within the heavy- and severe-intensity domain may result in haemoconcentration as a result of shifts in bodily fluid (i.e., dehydration), another factor that may affect $[La^-]_b$ values during exercise (3).

MLSS and CP are not without limitations, and independently, both models are widely accepted and challenged by many physiologists. However, for the purpose of this thesis MLSS was chosen to delineate the heavy- from the severe-intensity domain, as the MLSS model provides physiological evidence (i.e., $[La^-]_b$) for establishing the highest PO of sustainable exercise.

2.1.5 The severe-intensity domain

Exercise conducted above MLSS/CP can be defined as being in the severe-intensity domain. Within the heavy-intensity domain, steady state $\dot{V}O_2$ is delayed but still achieved; however, within the severe-intensity domain, steady state $\dot{V}O_2$ is not reached, as it is suggested

that the progressive development of the $\dot{V}O_2$ slow component leads to the attainment of $\dot{V}O_{2max}$ (4, 7, 40, 73).

In the severe-intensity domain, the $\dot{V}O_2$ slow component is reflective of the changes in the metabolic status of the muscles (16, 74), such as the progressive reduction in [PCr] and the increased inefficiency of energy turnover (39, 42). Therefore, exercise intolerance within this domain has been suggested to be related to the attainment of a critical muscle metabolic milieu (2) and from the perspective of the CP model, the depletion of W' (9, 65, 75). The muscle metabolic milieu can be characterized by the attainment of consistently low values of muscle [PCr] and pH and high $[La^-]_b$ (2). Regardless of the intensity or the duration sustained within the severe-intensity domain, it has been suggested that the achievement of maximal exercise intolerance coincides with the attainment of a consistent muscle metabolic milieu (i.e., critically low concentrations of substrates and high levels of metabolites, respectively) (2). Moreover, in order to compensate for the development of peripheral fatigue (i.e., increased muscle metabolic perturbations), there may be a greater contribution of central neural mechanisms, such as muscle fibre recruitment during severe-intensity exercise (2).

Like heavy-intensity exercise, the utilization of carbohydrates for substrate metabolism is crucial in order to perform exercise within the severe-intensity domain. However, unlike the heavy-intensity domain, fat oxidation contributes even less to energy turnover during severe-intensity exercise. Therefore, to meet the energy demands and to prolong the duration of exercise within this domain, the breakdown and utilization of stored muscle glycogen is critical (21). As a result of an increased glycolytic flux above MLSS/CP, $[La^-]_b$ production exceeds oxidation and begins to accumulate (21, 76). Therefore, coinciding with exercise intolerance, maximal $[La^-]_b$ values are commonly achieved during severe-intensity exercise (14).

2.2 Current Understanding of Exercise Performed at the Critical Intensity of Exercise

2.2.1 Physiological responses to exercise performed at and above MLSS or CP

Over the past few decades, the physiological responses during exercise at MLSS and CP have been characterized by many researchers. Poole et al. (4) in 1988, was the first to demonstrate that a small increase in PO above CP (CP + 5 % of peak power output (PPO)) resulted in the progressive increase in $\dot{V}O_2$, $[La^-]_b$, and $\dot{V}E$, compared to exercising at CP, where steady-state values were observed for $\dot{V}O_2$ and $[La^-]_b$. Since the work of Poole et al. (4), researchers have further evaluated the physiological responses to exercise performed at and above MLSS or CP. It is well-understood that during exercise at MLSS, $\dot{V}O_2$, $[La^-]_b$, $\dot{V}CO_2$, and RER all attain steady state (10, 24, 67, 77), whereas, heart rate (HR) and $\dot{V}_E$ increase significantly and do not reach steady state values (24, 50). Contrarily, when exercise is performed slightly above MLSS or CP, non-linear increases in $\dot{V}O_2$, $[La^-]_b$, HR, and $\dot{V}_E$, are all key physiological responses to exercise above this threshold (4, 9, 10, 51, 53, 59, 72, 78). Specifically, unsteady responses in $[La^-]_b$ and $\dot{V}_E$ are defining characteristics of constant-PO exercise performed above MLSS compared to at MLSS (10).

2.2.2 Performance related consequences of prior exercise

As a result of the profound differences in the physiological responses to exercise performed at and above the critical intensity of exercise, physiologists have begun to evaluate the implications that this intensity and intensities within the different domains have on subsequent performance. Specifically, in whole body exercise (i.e., cycling), most of the research assessing prior exercise, although small, has focused on the CP model.

Numerous studies by Clark et al. (79–81) have demonstrated that prolonged constant-PO exercise, within the heavy-intensity domain, has detrimental effects on the model predicted CP

as well as the work capacity within the severe-intensity domain. Specifically, following 2 hours of heavy-intensity cycling, CP and W' were both reduced by ~ 8 and ~ 20 %, respectively, compared to baseline values (79). Moreover, previous studies have also illustrated that prior exercise performed above CP (i.e., severe-intensity domain) reduces subsequent TTE performed at CP (71) as well as TTE performance within the severe-intensity domain (78). For intensities below the critical intensity of exercise, although we may have a better understanding of the ramifications associated with prior heavy-intensity exercise, little is known about the implications surrounding exercise performed within the moderate-intensity domain. A study by Passfield and Doust (82) was the first to explore the performance related consequences associated with prior moderate-intensity exercise. On two separate occasions participants cycled at a moderate intensity for 60 min, followed by either a 5 min performance trial or a 30 s sprint test. The authors found that prior moderate-intensity exercise reduced the 5-min performance PO as well as the peak and mean PO recorded during the 30 s sprint. Collectively, these studies indicate that regardless of the intensity domain in which a prior bout of exercise is performed, subsequent performance is significantly impacted.

Most of the research evaluating prior exercise, and its effects on subsequent performance, has done so using CP. However, only one study has used the physiological model of MLSS to evaluate exercise performed at this intensity and slightly above on TTE performance. A recent study by Iannetta et al. (10), was the first to evaluate the performances related consequences associated with prior exercise performed at $MLSS_p$ and 10 W above $MLSS_p$. Each condition ($MLSS_p$ and $MLSS_p$ + 10 W) was performed for 30 min and was followed by a TTE trial. The authors found that compared to a baseline TTE trial, TTE was reduced by ~ 37 and ~ 65 % following constant-PO cycling at $MLSS_p$ and 10 W above, respectively. This study highlighted

that there are implications associated with exercise performed at $MLSS_p$ and demonstrates that a small increase in PO above this workload (by as little as 10 W) has major detrimental effects on subsequent performance capabilities. However, a limitation of this study was that the participants were given a 3 min recovery period between the completion of each 30 min condition and the subsequent TTE trial. Therefore, it is possible that the results of the TTE trials may not fully be reflective of the metabolic perturbations associated with the prior bout of exercise. Moreover, the authors only evaluated one intensity in addition to $MLSS_p$ (i.e., MLSS + 10 W), therefore it is unknown whether TTE performance is affected by prior submaximal exercise (i.e., sub-$MLSS_p$). The work of Iannetta et al. (10) demonstrated the importance of properly determining the exercise intensity domains, as even small changes in intensity become relevant when approaching the critical intensity of exercise. These results also have important implications for exercise prescription based on the exercise intensity domain schema.

Taken together each of these studies indicate that to varying effects, prior exercise does indeed impact subsequent performance. However, each of these studies utilized different types of exercise (i.e., continuous vs. intermittent) and evaluated different outcome measures for assessing performance (i.e., TTE, time trials, and 30 s sprints). Therefore, the performance related consequences resulting from prior exercise performed at different intensities is inconclusive. Furthermore, no study has evaluated or characterized the impact that prior exercise performed at intensities within each intensity domain, for a standardized duration, has on subsequent performance. Moreover, as demonstrated by Iannetta et al. (10), 30 min at $MLSS_p$ significantly reduces TTE; however, based on this evidence it is unknown if manipulating the duration of exercise at $MLSS_p$ (i.e., durations shorter and longer than 30 min) has any impact on subsequent performance.

2.3 Perceptual Responses During Exercise

2.3.1 Rating of perceived exertion

Perception of effort can be evaluated during exercise through psycho-physical assessment, such as rating of perceived exertion (RPE). During exercise, RPE can be described as an individual's perception of how hard and strenuous a physical task is (83, 84). In 1970 Gunnar Borg created the 6 – 20 RPE scale, with 7 representing 'very, very light' and 19 representing 'very, very hard' perceived exertion (85). Although the 6 – 20 scale is the most common RPE scale used in a variety of settings (86), the scale was later modified (i.e., CR 0 – 10) for simplicity and for the proper interpretation of the scales' increments by the general population (85). Some physiologists believe that perceived exertion, independently, is the best indicator of physical stress during exercise and as a result of the reliability of RPE scales and the simplicity of the assessment, they can be used in place of other methods, such as monitoring HR, during exercise (85, 86).

Historically, RPE is believed to be reflective of the integration between signals from the muscles and joints, the cardiovascular and respiratory system, as well as the central nervous system (85). Moreover, RPE is also thought to be influenced by factors such as an individual's motivation, fear, memory of previous exercise experience, stress, pain, and discomfort (87). As a result of these different factors, it is especially important to administer the scale properly (i.e., consistent description of the RPE scale and consistent phrasing each time the scale is administered), so that participants are not influenced by the test administrator's translation or description of the RPE scale (86).

Since the original work of Borg, many researchers have tried to understand the mechanisms that govern RPE during exercise. Different models have been proposed to explain

the peripheral and central components that may determine RPE, such as the afferent feedback model of perceived exertion and the corollary discharge model of perceived exertion, respectively (83). Briefly, the afferent feedback model of perceived exertion is based on the premise that during exercise afferent signals from the working muscles provide feedback to the central nervous system regarding homeostatic disturbances experienced within the muscles, directly influencing RPE (73, 83, 86). Contrary to this model, the corollary discharge model of perceived exertion proposes that RPE is centrally governed by efferent neural signals, where an efferent copy of the motor command is sent directly to sensory areas of the brain in order to help assist in the perception of effort (83, 86). Moreover, it has been suggested that both afferent feedback and corollary discharge influence perception of effort during exercise, however, the role of afferent feedback in impacting RPE is continuously debated by many physiologists (18, 86, 87).

The pattern of the RPE response during incremental and constant-PO exercise is well-established. Specific to exercise performed on a cycle ergometer, Borg's RPE scales were designed so that RPE would increase linearly with an increase in PO and HR (88). During incremental exercise, RPE has been shown to follow a similar pattern to various physiological variables such as, $[La^-]_b$ (85, 89, 90), HR (89, 91), $\dot{V}O_2$ (91), and $\dot{V}_E$ (92).

Numerous studies have also demonstrated that during constant-PO exercise, RPE increases linearly with the duration to exhaustion (84, 92–95). Specifically, Pires et al. (93) evaluated the slope of the increase in RPE during constant-PO exercise performed to exhaustion at intensities corresponding to the moderate-, heavy-, and severe-intensity domains. The main finding from this study was that the slope of the increase in RPE during constant-PO exercise in each intensity domain accurately predicted the point of exercise exhaustion. Moreover, Pires et

al. (93) also demonstrated an association between RPE and $[La^-]_b$ during each constant-PO condition performed within the heavy- and severe intensity domains. Similarly, previous research has also shown that, like $[La^-]_b$, RPE is closely related to the responses in $\dot{V}O_2$, HR, $\dot{V}_E$, and breathing frequency (f_R) during constant-PO exercise (96–98). Interestingly, it has been shown that compared to traditionally monitored physiological variables, such as HR, f_R is the best correlate of RPE during exercise (90, 99). The close relationship between RPE and f_R is suggested to be a result of the sensitivity and rapidness of the f_R response during the onset and offset of exercise (99).

As evidenced, the relationship between RPE and various physiological variables as well as the duration to exercise exhaustion is well-researched. However, given that RPE may be used to predict the duration to task-failure during constant-PO exercise, whether RPE at the end of a bout of exercise corresponds to the duration to exhaustion in a subsequent bout is unknown.

2.4 Research Objectives and Hypotheses

2.4.1 Research objectives

The effects of prior exercise performed at $MLSS_p$ and 10 W above on subsequent performance is known (10). Similarly, the duration to exercise exhaustion at $MLSS_p$ and within each intensity domain is also known (2, 44). However, exercise performed at submaximal intensities (i.e., sub-$MLSS_p$) within the moderate- and heavy-intensity domains and their impact on subsequent performance is unknown. Therefore, the **primary objective** of this thesis was to characterize the changes in TTE performance following constant-PO exercise at a range of specific, domain-derived intensities. The **secondary objective** was to determine whether exercise performed for different durations at $MLSS_p$ has a further effect on subsequent TTE performance. Lastly, the **tertiary objective** was to evaluate whether perceptual (i.e., RPE) and/or

physiological (i.e., $[La^-]_b$) responses, assessed at the end of each constant-PO condition, correspond to the duration to exercise exhaustion during a subsequent TTE trial.

2.4.2 Research hypotheses

Based on the significant impact that exercise performed at $MLSS_p$ and 10 W above has on subsequent TTE performance (10), we hypothesized that constant-PO cycling for 30 min at submaximal intensities, would have detrimental effects on subsequent TTE performance. The physiological implications associated with exercise exhaustion in each intensity domain are well-understood (2); therefore, we hypothesized that as the constant-PO increased (from the moderate-, to the heavy-, and to the severe-intensity domains) the reduction in TTE would become progressively pronounced. Similarly, based on the finding that 30 min at $MLSS_p$ reduces TTE performance by ~ 37 % (10), we hypothesized that following an increase in the duration performed at $MLSS_p$, there would be a progressive reduction in TTE performance. Lastly, based on the linearity between RPE and exercise intolerance, as well as the relationship between RPE and $[La^-]_b$ (93, 96), we hypothesized that both RPE and $[La^-]_b$ evaluated at the end of each constant-PO condition would be indicative of the duration to exhaustion in each subsequent TTE trial.

Chapter Three: A study on the Reduction in Cycling Performance Following Prior Exercise

Prior exercise impairs subsequent performance in an intensity- and duration-dependent manner

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3.1 Abstract

Introduction: The duration to task-failure during exercise performed within each intensity domain is well understood. However, the effects that exercise in each domain has on subsequent performance is unknown. We tested the hypothesis that prior exercise, performed at different intensities or durations, would result in the progressive reductions in subsequent time-to-exhaustion (TTE), following an increase in intensity or duration. Moreover, we hypothesized that the reductions in TTE may be estimated using rating of perceived exertion (RPE), assessed immediately prior to each TTE trial. **Methods:** Eleven individuals (6 men, 5 women, 28±8 yrs) completed the following tests on a cycle ergometer: *i*) a ramp-incremental test, *ii*) a baseline TTE trial performed at 80% of peak power (TTE_b), *iii*) five 30-min constant-PO rides at 5% below lactate threshold ($LT_{-5\%}$), halfway between LT and the maximal lactate steady state ($MLSS_p$) ($Delta_{50}$), 5% below $MLSS_p$ ($MLSS_{-5\%}$), $MLSS_p$, and 5% above $MLSS_p$ ($MLSS_{+5\%}$), and *iv*) 15- and 45-min rides at $MLSS_p$ ($MLSS_{15}$ and $MLSS_{45}$, respectively). Each constant-PO ride was immediately followed by a TTE trial. **Results:** Compared to TTE_b, there were 8.0±24.1, 23.6±20.2, 41.0±14.8, 52.2±18.9, and 75.4±7.4% reduction in TTE following $LT_{-5\%}$, $Delta_{50}$, $MLSS_{-5\%}$, $MLSS_p$, and $MLSS_{+5\%}$, respectively. Following $MLSS_{15}$ and $MLSS_{45}$, there were 29.0±20.1 and 69.4±19.6% reductions in TTE, respectively (P <0.05). The percent reduction in TTE was strongly correlated with RPE when the intensity or duration of the prior condition were manipulated (r= -0.93±0.07 and r = -0.79±0.41, respectively). **Conclusion:** Subsequent TTE performance is reduced following exercise within the moderate- and heavy-intensity domains. Furthermore, RPE assessed at the end of a bout of exercise is related to the duration to task-failure in a subsequent maximal effort.

Key Words: Performance, exercise intensity domains, RPE, maximal lactate steady state

3.2 Introduction

The lactate threshold (LT) and the maximal lactate steady state (MLSS) or critical power (CP) partition the exercise intensity spectrum into three domains of intensity: moderate (<LT), heavy (>LT but <MLSS/CP), and severe (>MLSS/CP) (43). The physiological responses to exercise performed in each of these domains are highly predictable (2), such that this intensity domain "schema" represents a useful tool to prescribe and "normalize" the exercise intensity (i.e., metabolic stimulus) amongst individuals (12).

From this perspective, the metabolic perturbations and the neuromuscular responses associated with exercise performed within each intensity domain have been characterized (2, 17, 100). Briefly, short-duration exercise within the moderate-intensity domain, typically corresponding with ~60% of maximum O_2 uptake ($\dot{V}O_{2max}$), does not evoke major metabolic and neuromuscular disturbances (2, 17). In contrast, during exercise within the heavy- (~70-85% of $\dot{V}O_{2max}$) and severe-intensity domains (>85% of $\dot{V}O_{2max}$), these perturbations become pronounced (2, 10, 17, 44, 101, 102), with the effects of neuromuscular fatigue beginning to accumulate rapidly after the onset of the exercise and progressing in a manner similar to that of the $\dot{V}O_2$ slow-component (100). Despite these observations, it is poorly understood how exercise performed at specific domain-derived intensities and durations affects subsequent exercise capacity within the severe-intensity domain (as measured by a time-to-exhaustion (TTE) trial). We have previously demonstrated that 30-min of cycling at the power output (PO) corresponding to MLSS ($MLSS_p$) and at 10 W above MLSS reduced subsequent TTE performance by ~40% and ~65%, respectively, in relation to a control TTE trial with no prior exercise (10). Additionally, Clark et al. (79) have shown that two hours of cycling within the heavy-intensity domain reduced the PO associated with predicted CP by ~8% and the capacity to perform work

above CP by ~20%. Collectively, these observations suggest that exercise performed below MLSS/CP can impair subsequent performance, and that small increases in sustained intensity exacerbate this impairment. Building from these observations, it could be possible to hypothesize a progressive impairment in the capacity to perform exercise as the intensity, or duration (for a given workload) of exercise increases. To our knowledge, no study has evaluated the changes in TTE performance following systematic and progressive changes in domain-derived intensities and durations in the same group of individuals. Investigating the changes in TTE is important as it could shed light on the effect that intensity and duration have on subsequent performance and be informative for coaches and practitioners when prescribing exercise and predicting the exercise capacity within the severe-intensity domain.

In addition to the physiological responses discussed above, perceptual responses may also be related to subsequent exercise capacity within the severe-intensity domain (84, 103). For example, Pires et al. (93) demonstrated that the slope of the increase in the ratings of perceived exertion (RPE) during constant-PO exercise accurately predicted the time of task-failure. Therefore, it is possible that the RPE measured immediately prior to a maximal effort may predict the reduction in TTE performance.

The objectives of this study were to: (*i*) assess whether a reduction in TTE performance occurs following prior exercise performed at specific domain-derived intensities and different durations at $MLSS_p$; and (*ii*) determine whether physiological measures and RPE immediately prior to a bout of maximal exercise were associated with reduced TTE performance. We hypothesized that: (*i*) as the intensity of exercise for a given duration and the duration of exercise for a given intensity increased, there would be a progressive reduction in TTE performance; and

(*ii*) the impairment in TTE would be related to both physiological and perceptual responses (i.e., RPE) during the prior exercise condition.

3.3 Methods

3.3.1 Participants

Eleven recreationally active and well-trained men (n=6) and women (n=5) (28±8 yrs, 67.9±9.0 kg, 175.4±8.5 cm), who took part in endurance-based exercise at least three times per week voluntarily participated in this study. Participants were non-smokers, non-obese (BMI ≤ 30 kg/m^2) and were not undergoing any medical treatment. Prior to testing, each participant completed the CSEP Physical Activity Readiness Questionnaire (PAR-Q+) and provided written informed consent. All procedures were approved by the Conjoint Health and Research Ethics Board of the University of Calgary, in compliance with the latest version of the Declaration of Helsinki.

3.3.2 Experimental design

All participants completed all testing procedures on an electromagnetically braked cycle ergometer (Velotron; RacerMate, Seattle, WA), in an environmentally controlled room (temperature: 19-20° C; humidity: 50-60%), at a similar time of the day (±30 min). The visits to the laboratory included, *i*) one ramp-incremental test to exhaustion, *ii*) five 30-min constant-PO rides at 5% below LT ($LT_{-5\%}$), halfway between LT and $MLSS_p$ ($Delta_{50}$), 5% below $MLSS_p$ ($MLSS_{-5\%}$), $MLSS_p$, and 5% above $MLSS_p$ ($MLSS_{+5\%}$). Each of these rides was immediately followed by a TTE trial (i.e., $TTE_{LT-5\%}$, $TTE_{Delta50}$, $TTE_{MLSS-5\%}$, TTE_{MLSSp}, and $TTE_{MLSS+5\%}$, respectively), *iii*) 15- and 45-min constant-PO rides at $MLSS_p$ immediately followed by a TTE trial (TTE_{MLSS15} and TTE_{MLSS45}, respectively), and *iv*) a baseline TTE trial (TTE_b), serving as a

"control" condition, only preceded by a 4-min 50 W warm-up. All tests were performed over the course of five consecutive weeks with a minimum of 48 hours between each visit (see below for order of visits). For each testing session, participants were able to view their cycling cadence but were blinded to the condition they were performing, the PO and the elapsed time. Moreover, participants were not provided with any feedback related to their performance before concluding the study. Prior to each visit, participants were instructed to avoid the consumption of food and caffeinated beverages for at least 2 and 8 hours, respectively, and to abstain from vigorous physical activity for 24 hours. Participants were also asked to maintain a similar diet throughout the course of the study, self-recording what they ate the day before and of each laboratory visit.

Ramp-incremental test. On the first visit to the laboratory, participants completed a ramp-incremental test to exhaustion. The session began with a 4-min baseline at 20 W, followed by a moderate step-transition (6 min at 100 W) to facilitate the accurate computation of the $\dot{V}O_2$ mean response time (MRT) of the ramp-incremental test (104). Immediately following the moderate step-transition, participants cycled for 4 min at 50 W leading to the incremental portion of the test (25 $W \cdot min^{-1}$ for women (1 W every 2.4 s) and 30 $W \cdot min^{-1}$ (1 W every 2 s) for men). The test was stopped once participants reached volitional exhaustion or when the cycling cadence dropped below 70 rpm for longer than ten consecutive seconds, despite strong verbal encouragement.

Constant-PO trials. The second laboratory visit consisted of cycling for 30 min at each participant's predicted $MLSS_p$. $MLSS_p$ prediction was based on a previously published mathematical model (see Iannetta et al. (57)). $MLSS_p$ was established as the highest PO where blood lactate concentration ($[La^-]_b$) between the 10^{th} and 30^{th} minute differed by no more than 1 $mmol \cdot L^{-1}$ (49). When the $[La^-]_b$ between the 10^{th} and 30^{th} minute differed by greater or less than

(or equal to) 1 mmol·L^{-1}, the PO was decreased or increased by 5%, respectively, for the subsequent visit. Upon the determination of $MLSS_p$ and the completion of the corresponding confirmation ride (i.e., $MLSS_{+5\%}$ and/or $MLSS_{-5\%}$), the remaining conditions were performed in a randomized order and consisted of cycling either at a constant-PO for 30 min at $LT_{-5\%}$, $Delta_{50}$, $MLSS_{-5\%}$, for 15 or 45 min at $MLSS_p$, or TTE_b. For all conditions, the constant-PO rides were initiated after a 4-min baseline warm-up at 50 W. During these rides, participants were instructed to cycle consistently at their preferred cadence determined during the ramp-incremental test.

Time-to-exhaustion trials. Each constant-PO condition was followed by a TTE trial in the severe-intensity domain. 10 s before the end of the constant-PO ride, participants were informed that the TTE trial was about to begin. The PO for this TTE trial corresponded to 80% of the peak PO (PO_{peak}) from the ramp-incremental test. The TTE_b condition was proceeded by a 4-min warm-up at 50 W only and conducted randomly after determination of $MLSS_p$. All TTE trials were stopped when participants reached volitional exhaustion, or their cadence dropped below 70 rpm for 10 s.

3.3.3 Data collection

Breath-by-breath gas-exchanges and ventilatory variables were measured continuously during each testing session with a metabolic cart (Quark CPET, Cosmed, Rome, Italy). Calibration was performed according to the manufacturer's instructions with a 3L syringe and a standard gas mixture (16% O_2, 5% CO_2, and balance N_2). For measures of $[La^-]_b$, a 20 µL finger-prick blood sample was collected into a capillary tube and immediately analysed (EKF Biosen C-Line Analyzer, EKF Diagnostics, Barleben, Germany). For each condition, $[La^-]_b$ was assessed during the 4-min warm-up phase, every 5-min of each constant-PO ride, and immediately after each TTE trial. At the 10^{th} and 30^{th} min of the constant-PO rides, three blood samples were taken

in rapid succession, and the two closest measures were averaged for subsequent analyses. The 0-10 Borg scale (85) was used to measure RPE at the same time points as the $[La^-]_b$ measurements. Participants were familiarized with the RPE scale during the first visit to the laboratory (i.e., ramp-incremental test). The RPE scale was evaluated by asking each participant "how hard are you working?", with "0" representing effort equivalent to "sitting on the couch watching TV" and "10" representing "maximal effort, the hardest task you have ever done before."

3.3.4 Data Analyses

Ventilatory and gas-exchange variables. For each ramp-incremental test, the raw ventilatory and gas-exchange variables were plotted against $\dot{V}O_2$ to estimate LT (37). LT corresponded to the point at which carbon dioxide output ($\dot{V}CO_2$) began to increase out of proportion in relation to $\dot{V}O_2$, coincidentally with a systematic rise in the ventilation ($\dot{V}_E$)/$\dot{V}O_2$ ventilatory equivalent and end-tidal PO_2, whilst the $\dot{V}_E/\dot{V}CO_2$ ventilatory equivalent and end-tidal PCO_2 were stable (37). To accurately identify the PO associated with the estimated LT, the ramp-$\dot{V}O_2$ was corrected by the MRT to account for the muscle-lung transit time delay and the kinetics of $\dot{V}O_2$ at ramp-onset (104).

After the raw ventilatory and gas-exchange variables were edited on the basis of $\dot{V}O_2$ (105), peak $\dot{V}O_2$ ($\dot{V}O_{2peak}$) was computed from the highest 30-s rolling average during the ramp-incremental test. The same strategy was employed to determine $\dot{V}O_{2peak}$, as well as the peak $\dot{V}_E$, respiratory exchange rate (RER), breathing frequency (f_R), and heart rate response during each TTE trial. PO_{peak} corresponded to the PO value at the end of the ramp-incremental test. To compute the average $\dot{V}O_2$, $\dot{V}_E$, f_R, and RER response during each constant-PO ride, the last 5 min of each condition were averaged.

Total Work. The total mechanical work for each constant-PO condition, including each TTE trial, was calculated as follows.

Total Work (kJ) = time (s) x power output (W) / 1000

Data and Statistical Analyses

Data are presented as means ± SD, and all statistical analyses were performed using SPSS version 25 (SPSS, IBM, Chicago, IL). For the five 30-min constant-PO conditions, a repeated-measures ANOVA was used to compare: *i*) the absolute duration of each TTE trial, *ii*) the reduction in each TTE trial, expressed as a percentage of TTE_b; *iii*) $\dot{V}O_2$, $\dot{V}_E$, $[La^-]_b$, and RPE responses at the 30th minute of each constant-PO condition; *iv*) the average $\dot{V}O_2$, $\dot{V}_E$, f_R, RER responses during each constant-PO condition; and *v*) the total work performed during each constant-PO condition and subsequent TTE trial. The same comparisons were used to evaluate responses for the three durations of cycling at $MLSS_p$, and for the comparison of $\dot{V}O_2$ and $\dot{V}_E$ at iso-time in these rides (i.e., 15th minute of $MLSS_{15}$, $MLSS_p$, and $MLSS_{45}$). Prior to performing the repeated-measures ANOVA, the data were evaluated for normality and sphericity of the variance. For all repeated-measures ANOVA, when a main effect was found a Bonferroni post hoc was performed and statistical significance was accepted at $\alpha < 0.05$.

For each individual, the Pearson's correlation coefficient was used to assess the relationship between the percent reduction in TTE performance and, *i)* RPE, *ii)* $[La^-]_b$, and *iii)* $\dot{V}_E$, measured at the end of each constant-PO condition, and, *iv)* the constant-PO duration at $MLSS_p$. The Pearson's correlation coefficient was also used to evaluate the association between RPE and $\dot{V}_E$ at the end of each constant-PO condition.

3.4 Results

The average PO_{peak} at the end of the ramp-incremental cycling test was 336±59 W and the derived 80% of PO_{peak} used for the TTE trials was 269±47 W. Table 3.1 presents the average for each constant-PO condition, as well as the corresponding ventilatory and gas exchange responses.

Profiles of $\dot{V}O_2$ as well as of $\dot{V}_E$, $[La^-]_b$, and RPE during each constant-PO condition are displayed in Figure 3.1. During each 30-min trial, the $\dot{V}O_2$ and $\dot{V}_E$ responses at the 30th minute of each exercise intensity were all significantly different from each other ($P < 0.05$) (Figure 3.1, A and C, respectively). No difference was found in the $\dot{V}O_2$ at the end of each $MLSS_p$ condition of different duration ($P > 0.05$) (Figure 3.1, B). The $\dot{V}_E$ at the end of $MLSS_{15}$ was lower than both $MLSS_p$ and $MLSS_{45}$ rides ($P < 0.05$), with no difference found between $MLSS_p$ and $MLSS_{45}$ ($P > 0.05$) (Figure 3.1, D). Moreover, no difference was found at iso-time for $\dot{V}O_2$ or $\dot{V}_E$ ($P > 0.05$), (i.e., assessed at the 15th minute of $MLSS_{15}$, $MLSS_p$, and $MLSS_{45}$). As the intensity of each constant-PO ride increased up to $MLSS_p$, progressively higher but stable $[La^-]_b$ responses were observed ($P < 0.05$) (Figure 3.1, E). A disproportionate increase in $[La^-]_b$ occurred during $MLSS_{+5\%}$ compared to all other conditions ($P < 0.05$). $[La^-]_b$ values recorded at the end of $MLSS_{15,}$ $MLSS_p$, and $MLSS_{45}$ were not different from one another ($P > 0.05$) (Figure 3.1, F). The RPE responses throughout each constant-PO condition are shown in Figure 3.1 (G and H). Peak ventilatory and gas exchange, heart rate, $[La^-]_b$, and perceptual responses during the ramp-incremental and each TTE trial are shown in Table 3.2. The percent reduction of each TTE trial with respect to TTE_b is shown in Figure 3.2 (A to D). The mean reduction in TTE performance after each constant-PO ride follows a downward-curvilinear pattern (Figure 3.2, C). In contrast, a linear reduction in TTE was found following different exercise durations at $MLSS_p$ (Figure 3.2,

D). The total amount of work performed during each constant-PO condition and TTE trial are shown in Figure 3.2 (E and F).

The relationship between the percent reduction in the different TTE trials and $[La^-]_b$ and RPE recorded immediately prior to each TTE trial is presented in Figure 3.3. The percent reduction, for each individual, in TTE with increasing intensity of the constant-PO rides was strongly correlated to the progressive increase in $[La^-]_b$ (r= -0.93±0.04) and RPE (r= -0.93±0.07) (Figure 3, A and C). However, for the $MLSS_{15}$, $MLSS_p$, and $MLSS_{45}$ conditions (Figure 3.3, B and D), the reduction in TTE performance was correlated with RPE (r = -0.79±0.41) but not $[La^-]_b$ (r = -0.21±0.69). The percent reduction in TTE performance with increasing intensity and duration of the constant-PO rides, for each individual, was strongly correlated to the progressive increase in $\dot{V}_E$ (r = -0.94±0.06 and -0.79±0.22, respectively). The relationship between $\dot{V}_E$ and RPE, immediately prior to each TTE trial, was strongly correlated following an increase in intensity of the constant-PO rides (r = 0.96±0.03), whereas a moderate correlation was found following an increase in the duration at $MLSS_p$ (r = 0.68±0.27).

3.5 Discussion

The current investigation shows that prior exercise performed at a range of specific, domain-derived intensities and a range of durations progressively impairs subsequent exercise capacity within the severe-intensity domain, and that this impairment is predictable as it is strongly correlated to RPE measured prior to TTE performance.

3.5.1 Reduction in time-to-exhaustion performance following exercise at different intensities and durations.

Following 30 min of cycling at a constant-PO there was a progressively increasing impairment in TTE performance with increasing exercise intensity ($LT_{-5\%}$:~ 8% , $Delta_{50}$: ~ 24%,

$MLSS_{-5\%}$: ~ 41%, and $MLSS_p$: ~ 52%, $MLSS_{+5\%}$: ~75 %) (Figure 3.2, A). When these distinct reductions in TTE performance were plotted against each constant-PO expressed as a percentage of $MLSS_p$, a downward exponential pattern was observed (Figure 3.2, C). Findings from this study show that all intensities and durations of exercise investigated herein impaired subsequent TTE performance in the severe-intensity domain. Surprisingly, this observation is true even following 30 min of exercise in the moderate-intensity domain, within which exercise-induced depletion of substrates and accumulation of metabolites is likely minimal (2). Moreover, manipulating the duration of the exercise also had marked effects on TTE performance (Figure 3.2, B). Indeed, as the duration of exercise at $MLSS_p$ was increased (from 15, to 30, and to 45 min), the TTE performance was reduced in a seemingly linear pattern (from ~ 29%, to ~ 52%, and to ~ 69 %) (Figure 3.2, B and D). This finding demonstrates that changes in intensity and duration pose different challenges that contribute differently in the reduction of subsequent TTE performance.

During submaximal exercise (in the moderate- and heavy-intensity domain), the amplitude of the changes in intramuscular substrates and metabolites from baseline become progressively greater with increasing metabolic rates (16, 41). However, while in the moderate- and heavy-intensity domains the metabolic milieu remains relatively stable, exercise within the severe-intensity domain results in an inexorable fall in substrate availability (i.e., [PCr]) and accumulation of metabolites (i.e., [P_i], [ADP], and [H^+]) (2, 16, 41, 106, 107), such that, independently of the external workload (i.e., PO), exercise performed within the severe-intensity domain typically culminates with the attainment of a consistent intramuscular metabolic milieu (61). Therefore, the progressive reduction in TTE performance might be explained by the fact that various TTE trials were initiated from progressively higher levels of metabolic perturbation

leading to the attainment of this “critical” metabolic milieu being reached earlier. Interestingly, the combined mechanical work expressed during the 30-min constant-PO rides and subsequent TTE trials were not different across conditions, except for $LT_{-5\%}$ (Figure 3.2, E), suggesting an inextricable link between the level of metabolic perturbations in the heavy-intensity domain and exercise capacity within the severe-intensity domain.

The pattern of decrease in TTE performance was exponential (Figure 3.2, C), suggesting that slight increases of prior intensity can lead to disproportionate reductions of exercise capacity within the severe-intensity domain. This behaviour is somewhat consistent with the exponential nature of the power-duration curve, as increases in PO in the severe-intensity domain have disproportionate effects on tolerable durations of exercise compared to those in the other domains (i.e., moderate and heavy) (55). Regardless, Passfield and Doust (82) demonstrated that cycling efficiency declines during sustained exercise within the moderate-intensity domain and that this decline is highly correlated with subsequent severe-intensity performance. Similarly, Cannon et al. (17) and more recently Keir et al. (100) have reported a close association between the progression of the $\dot{V}O_2$ slow component and the time course of peripheral fatigue, demonstrating a link between the development of metabolic inefficiency and the capacity of the muscle to sustain the required levels of force. These studies could suggest that, as these metabolic inefficiencies become progressively greater with increasing metabolic rates, they could contribute to the disproportionate impairment of exercise capacity in the severe-intensity domain.

When considering the different mechanisms that may contribute to the reduction of TTE performance the potential role of muscle glycogen should also be considered. At the onset of exercise muscle glycogen begins to decline, and as the intensity of exercise increases its rate of

depletion increases (21, 22). However, based on the durations evaluated in this study it is unlikely that the levels of glycogen depletion attained at the end of each constant-PO ride were sufficiently low to directly influence subsequent TTE performance (21). Regardless, given the wide range of exercise intensities and durations studied, it is possible that the mechanisms responsible for the reductions in TTE performance should be considered fluid and variable. Collectively, the findings of the present study indicate that the effects of intensity and duration should be considered separately when comparing or prescribing varying exercise bouts or evaluating their impact on subsequent performance. Specifically, it appears that an increase in intensity of prior exercise has a greater detrimental consequence than an increase in duration (at least within the times tested in this study). As discussed herein, the curvilinear reduction in TTE reflects the greater physiological stress of an increase in intensity, and it may offer partial support to the observation that varying the intensity and duration of exercise training can alter the magnitude of adaptation in $\dot{V}O_{2max}$ (108).

3.5.2 Lactate and perceptual responses: relationship with time-to-exhaustion performance.

An interesting finding of the present study is the relationship between $[La^-]_b$ and RPE, assessed immediately prior to each TTE trial, and the reduction in exercise capacity within the severe-intensity domain. As the intensity of the constant-PO trials increased, the percent reduction in TTE performance was also strongly correlated with both the progressive increase in $[La^-]_b$ and RPE (Figure 3.3, A and C). The progressive increase in $[La^-]_b$ at the end of each exercise intensity condition (from $LT_{-5\%}$ to $MLSS_{+5\%}$) is indicative of an increasing glycolytic flux and the accumulation of metabolites (76) (Figure 3.1, E). However, although there were strong associations between both $[La^-]_b$ and RPE and TTE performance for the 30-min exercise bouts (performed at different intensities), the decline in TTE performance following

progressively longer rides (at $MLSS_p$) provide a different perspective. At the end of each duration condition performed at $MLSS_p$, the reduction in TTE performance was associated with RPE (Figure 3.3, D) but was not with $[La^-]_b$ (Figure 3.3, B). This lack of relationship would indicate that a given $[La^-]_b$ is not predictive of reduction in subsequent TTE performance (Figure 3.3, B).

Several studies have shown that RPE during constant-PO exercise increases linearly with the duration to task-failure (84, 92–95). The present study shows that RPE at the end of each constant-PO ride (regardless of intensity or duration) negatively correlates with reductions in TTE performance. It has been suggested that RPE is reflective of the integration of a diverse range of peripheral and central feedback and feedforward responses to exercise (18, 84, 87). As RPE responses increase with increasing exercise intensities and durations, it is not surprising that levels of RPE strongly predict reductions in TTE performance. In addition to this, $\dot{V}_E$, a response argued to be affected by central and peripheral responses (18, 109–111), has been shown to influence RPE, especially as the intensity of exercise increases (91). Consequently, several studies have established a strong relationship between RPE and $\dot{V}_E$ during exercise (91, 96–98). Consistent with these findings, we found a strong association between RPE and $\dot{V}_E$ at the end of each constant-PO condition, regardless of its intensity and duration. Moreover, $\dot{V}_E$ at the end of each constant-PO condition was strongly correlated with the reduction in subsequent TTE performance. Taken together, these data indicate a role for both peripheral and central mechanisms affecting subsequent exercise capacity within the severe-intensity domain.

3.6 Methodological Considerations

To demarcate the boundary between the heavy- and severe-intensity domains and, thus, to normalize the intensity across participants, we used the traditional MLSS approach. While we

acknowledge that this approach might have resulted in an underestimation of the heavy to severe boundary (3), at the same time we justify this choice because in the present study we needed to normalize intensity against a set of replicable physiological criteria. Regardless, the fact that participants were unable to perform the TTE trial following $MLSS_{+5\%}$ for much longer than 1 min, in addition to the unstable $[La^-]_b$ responses, suggest that participants were indeed in the lower boundaries of the severe-intensity domain. Alternatively, we could have adopted the CP model and used specific fractions of the estimated PO at CP. However, considering that the CP model also presents some level of uncertainty in the prediction of this boundary (3, 63, 70), this choice would not have guaranteed an additional layer of accuracy. Regardless, the purpose of the present study was to evaluate the evolution of TTE performance following constant-PO exercise performed at different intensities and durations, thus the choice of the physiological marker separating the heavy- and the severe-intensity domains, while important, should not have represented a confounding factor.

3.7 Conclusion

This study characterized the effects of prior exercise performed at different intensities and durations on subsequent TTE performance. We found a strong correlation between the reduction in TTE performance following constant-PO exercise across intensity domains and a range of durations of preceding exercise. Notably, when a bout of exercise was performed at sub-MLSS intensities, subsequent performance was reduced. The current study also highlighted that RPE is relate to the exercise capacity within the severe-intensity domain.

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Authors' contributions

MMF participated in the design of the study, contributed to data collection and data analysis; DI participated in the design of the study and data analysis; MJM contributed to data analysis; LP and JM participated in the design of the study. All authors contributed to the manuscript writing. All authors have read and approved the final version of the manuscript.

Competing interests

The authors declare that they have no competing interests.

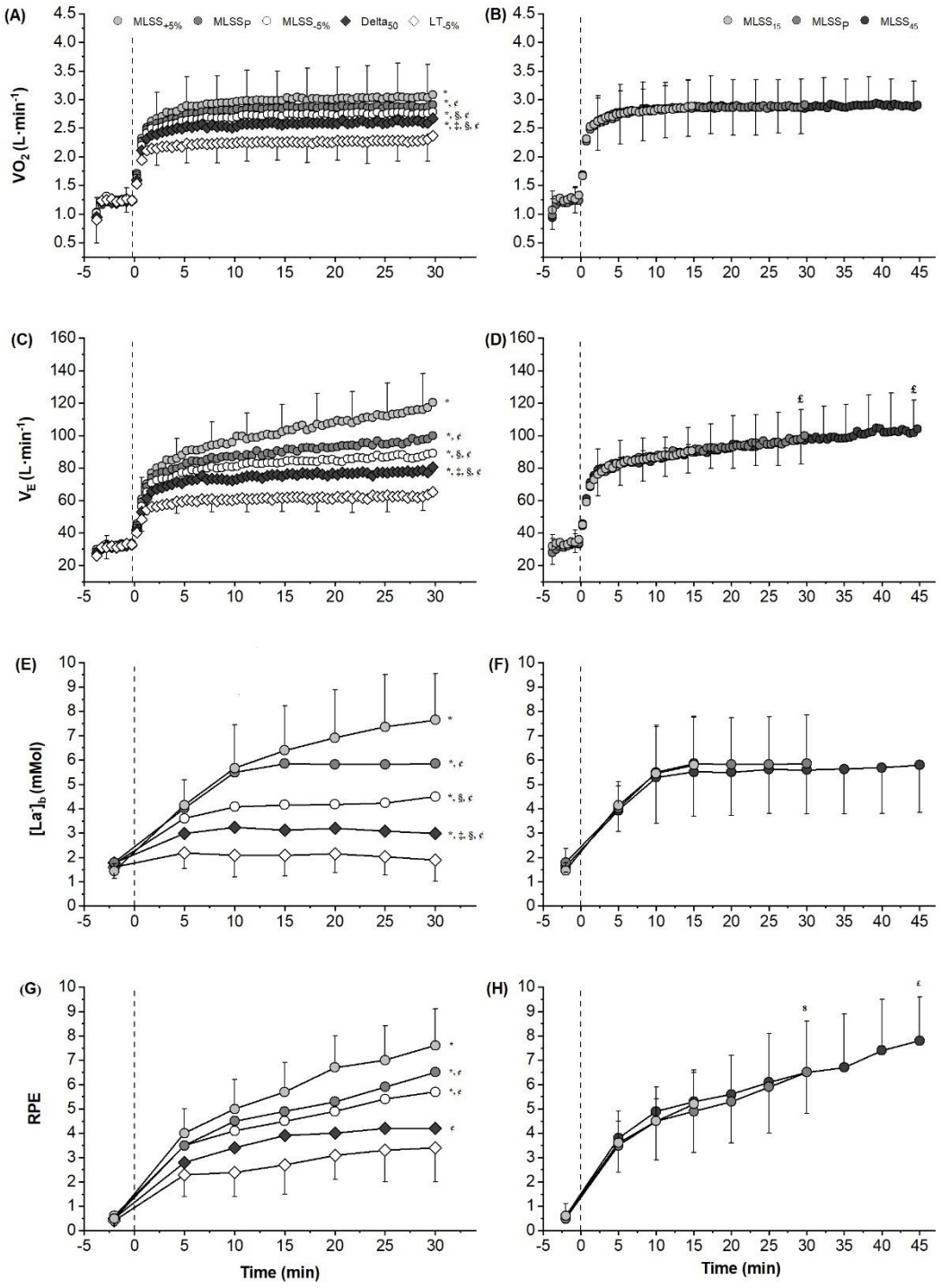

Figure 3.1 Group mean data (with SD bars) (n=11) displaying $\dot{V}O_2$ (A, B), $\dot{V}_E$ (C, D), $[La^-]_b$ (E, F), RPE (G, H) during 30-min constant-PO rides at $MLSS_{+5\%}$ (light grey circles), $MLSS_p$ (grey circles), $MLSS_{-5\%}$ (white circles), $Delta_{50}$ (dark grey diamonds), $LT_{-5\%}$ (white diamonds), and

during $MLSS_{15}$ (light grey circles) and $MLSS_{45}$ (dark grey circles). For each variable, the average of the last 2 min of each condition were compared.
* Different from $TTE_{LT-5\%}$
‡ Different from $TTE_{MLSS-5\%}$
§ Different from TTE_{MLSSp}
¢ Different from $TTE_{MLSS+5\%}$
£ Different from TTE_{MLSS15}
∞ Different from TTE_{MLSS45}
($P < 0.05$)

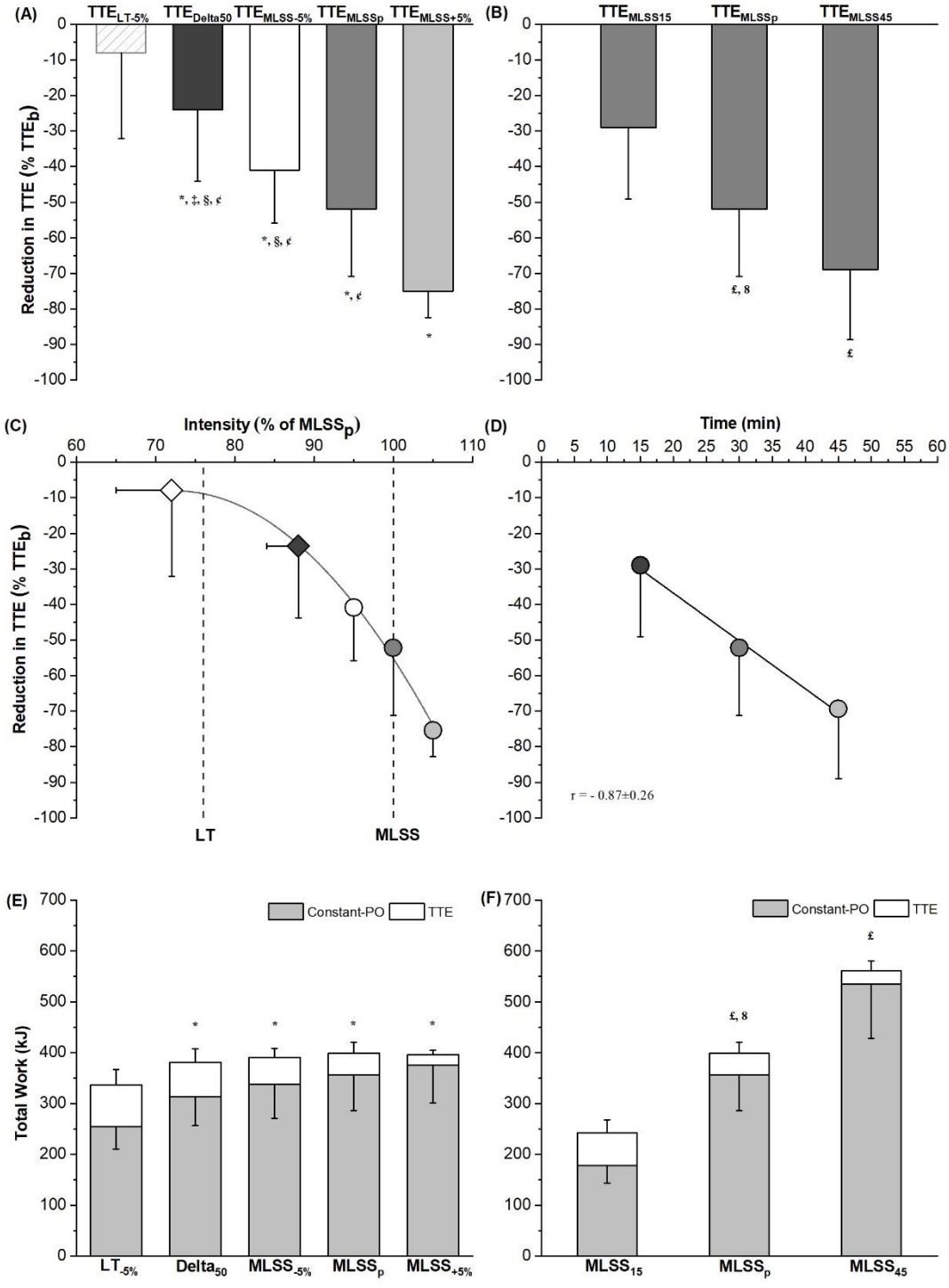

Figure 3.2 Group mean data (with SD bars) (n=11) displaying the reduction in TTE performance expressed as a percentage of TTE_b (A, B), the reduction in TTE performance and each 30-min intensity expressed as a percentage of $MLSS_p$ (C) and each exercise duration at $MLSS_p$ (D), and the total amount of work performed during each constant-PO and TTE trial (E, F). Correlations

are based on the individual data (D) and a second order polynomial was fitted to the percent reduction in TTE performance compared with each 30-min PO expressed as a percentage of $MLSS_p$ (C).
* Different from $TTE_{LT-5\%}$
‡ Different from $TTE_{MLSS-5\%}$
§ Different from TTE_{MLSSp}
¢ Different from $TTE_{MLSS+5\%}$
£ Different from TTE_{MLSS15}
∞ Different from TTE_{MLSS45}
($P < 0.05$).

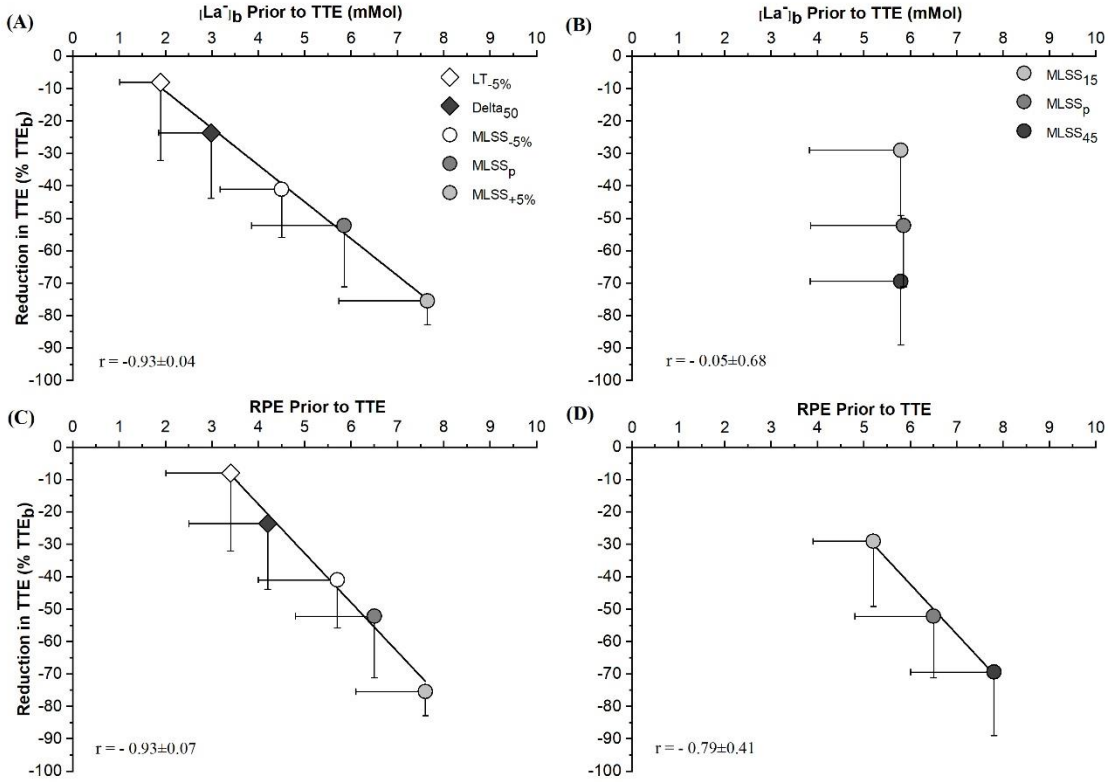

Figure 3.3 Group mean data (with SD bars) (n=11) displaying the relationship between the reduction in TTE performance expressed as a percentage of TTE_b, and $[La^-]_b$ (A, B) and RPE (C, D) evaluated immediately prior to the commencement of each TTE trial. Correlations are based on the individual data.

Table 3.1 The average PO, ventilatory and gas exchange variables during the last 5-min of each constant-PO condition.

Variable	$LT_{-5\%}$	$Delta_{50}$	$MLSS_{-5\%}$	$MLSS_p$	$MLSS_{+5\%}$
PO (W)	142±25	174±31$^{*, ‡, §, ¢}$	188±38$^{*, §, ¢}$	198±39$^{*, ¢}$	208±41*
$\dot{V}O_2$ (L·min^{-1})	2.29±0.33	2.62±0.32$^{*, ‡, §, ¢}$	2.76±0.40$^{*, §, ¢}$	2.88±0.44$^{*, ¢}$	3.04±0.56*
RER	0.91±0.03	0.91±0.03	0.94±0.06	0.94±0.04	0.93±0.04
$\dot{V}E$ (L·min^{-1})	63±7	78±9$^{*, ‡, §, ¢}$	87±11$^{*, §, ¢}$	96±13$^{*, ¢}$	115±20*
f_R (breath·min^{-1})	32.3±5.4	38.7±6.8$^{*, ¢}$	41.4±9.3$^{*, ¢}$	44.2±10.1$^{*, ¢,}$	52.9±8.5*
Variable	$MLSS_{15}$	$MLSS_p$	$MLSS_{45}$		
PO (W)	198±39	198±39	198±39		
$\dot{V}O_2$ (L·min^{-1})	2.84±0.41	2.88±0.44	2.90±0.47		
RER	0.94±0.05	0.94±0.04	0.93±0.05		
$\dot{V}E$ (L·min^{-1})	88±10	96±13$^{£}$	103±23$^{£}$		
f_R (breath·min^{-1})	39.8±8.2	44.2±10.1$^{£}$	49.9±12.1$^{£}$		

* Different from $LT_{-5\%}$
‡ Different from $MLSS_{-5\%}$
§ Different from $MLSS_p$
$^{¢}$ Different from $MLSS_{+5\%}$
$^{£}$ Different from $MLSS_{15}$
$P < 0.05$

Table 3.2 Peak ventilatory and gas exchange, heart rate, blood lactate, and perceptual responses during the ramp-incremental test and each TTE trial.

Variable	Ramp-incremental	TTE_b	$TTE_{LT-5\%}$	$TTE_{Delta50}$	$TTE_{MLSS-5\%}$	TTE_{MLSSp}	$TTE_{MLSS+5\%}$
TTE duration (s)	-	330±52	300±74 †, ‡, §, ¢	251±70 €, §, ¢	194±55 €, ¢	155±62 €, ¢	80±26 €
$\dot{V}O_{2peak}$ ($L \cdot min^{-1}$)	3.39±0.16	3.48±0.18	3.45±0.21	3.37±0.16	3.36±0.15	3.28±0.16	3.22±0.18
$\dot{V}E$ ($L \cdot min^{-1}$)	160±29	155±28	151±31	151±32	147±33	147±31	141±27
HR (bpm)	181±7	174±6	177±5	178±8	180±5	178±11	179±5
$[La^-]_b$	11.2±2.0	10.6±2.5	10.2±3.1	9.3±2.9	9.1±1.9	9.5±2.6	9.4±3.0
RPE	8.9±1.1	9.2±1.1	9.3±0.8	9.6±0.9	9.5±0.9	9.5±0.7	9.7±0.5

Variable	Ramp-incremental	TTE_b	TTE_{MLSS15}	TTE_{MLSSp}	TTE_{MLSS45}
TTE duration (s)	-	330±52	234±69 €, §, ∞	155±62 €, ∞	101±66 €
$\dot{V}O_{2peak}$ ($L \cdot min^{-1}$)	3.39±0.16	3.48±0.18	3.46±0.17	3.28±0.16	3.15±0.15
$\dot{V}E$ ($L \cdot min^{-1}$)	160±29	155±28	154±31	147±31	131±25
HR (bpm)	181±7	174±6	175±7	178±11	178±6
$[La^-]_b$	11.2±2.0	10.6±2.5	9.3±2.9	9.5±2.6	7.1±2.5
RPE	8.9±1.1	9.2±1.1	9.6±0.7	9.5±0.7	9.5±0.9

TTE duration (s) results:
€ Different from TTE_b
† Different from $TTE_{Delta50}$
‡ Different from $TTE_{MLSS-5\%}$
§ Different from TTE_{MLSSp}
¢ Different from $TTE_{MLSS+5\%}$
∞ Different from TTE_{MLSS45}
$P < 0.05$

Chapter Four: Conclusion

As outlined in Chapter Two, the physiological responses to exercise performed within each intensity domain and at the different thresholds have been characterized by many physiologists. Similarly, the critical intensity of exercise (i.e., MLSS/CP) is continuously researched and evaluated, as it delineates stable from unstable physiological responses and concomitantly, sustainable from unsustainable intensities of exercise. Although we are starting to have a better understanding of how different physiological variables respond to exercise performed at different intensities and for different durations, the implications that these intensities and durations have on subsequent performance capabilities are largely unknown. However, the recent work of Iannetta et al. (10), was the first to demonstrate that prior exercise performed at $MLSS_p$ and slightly above this PO had detrimental effects on subsequent TTE performance. Therefore, based on these findings, the goal of this thesis was to characterize the pattern of change in TTE performance following exercise performed in each intensity domain, specifically at sub-MLSS intensities, and following different durations at $MLSS_p$.

As presented in Chapter Three, this investigation demonstrated that TTE performance is reduced in a curvilinear and linear fashion following either an increase in constant-PO intensity or duration, respectively. Specifically, these findings demonstrate that even submaximal exercise, performed below $MLSS_p$, results in the reduction in TTE performance. Based on what we know about the metabolic perturbations associated with exercise performed to exhaustion in each intensity domain (2), we can only speculate on the potential mechanisms that may govern the observed reductions in TTE performance. Therefore, as presented in Chapter Three, it is possible that the reductions in TTE performance were influenced by the fluidity of the metabolic milieu attained during each prior bout of exercise and that the metabolic milieu may be both

intensity and duration dependent, as manipulating both variables resulted in significant reductions in TTE. However, the metabolic milieu is only one perspective that may explain the observed reductions in TTE performance, and therefore, it possible that other mechanisms and models of fatigue may explain the observed reductions in TTE performance, such as the critical threshold of peripheral fatigue and the sensory tolerance limit (18).

The novel findings from this thesis are that (*i*) TTE performance were reduced following submaximal exercise, (*ii*) increasing the duration of exercise at $MLSS_p$ resulted in a linear reduction in subsequent TTE performance, and (*iii*) RPE evaluated at the end of each constant-PO condition is related to the exercise capacity within the severe-intensity domain.

4.1 Study Limitations and Methodological Considerations

Commonly, the methodology of MLSS determination posses as a potential limitation for any research involving the MLSS model. First, as mentioned in Chapter Two, one of the largest sources of error with MLSS is the PO increments used between determination trials. The study presented in Chapter Three utilized a 5 % increment for the change in PO when determining the PO at MLSS. Therefore, in this thesis, MLSS could have been underestimated by as little as 7 W or as much as 13 W, for a participant whose $MLSS_p$ was 150 W and 280 W, respectively. Another limitation for determining MLSS could have been the criteria used to establish a stable $[La^-]_b$ response. Based on the most commonly used criteria, MLSS was defined as the change in $[La^-]_b$ of less than or equal to 1 $mmol \cdot L^{-1}$ from the 10^{th} to 30^{th} min of exercise (56). However, recent evidence suggests that $[La^-]_b$ may still be adjusting during the 10^{th} min of exercise, where the attainment of a stable $[La^-]_b$ response may not be noticeable until the 15^{th} min (3). Therefore, in this thesis MLSS could have been underestimated, as it was defined by the $[La^-]_b$ response from the 10^{th} to 30^{th} min, not 15^{th} to 30^{th}. It is important to note that $[La^-]_b$ is highly sensitive to

changes in nutrition and hydration status (112), therefore, during MLSS determination it is important to conduct testing at a similar time of day and to control for diet, as $[La^-]_b$ is subject to day-to-day variability. In this thesis, participants were asked to maintain a similar diet before each laboratory visit, which was monitored by participants self-recording what they ate the day prior and before testing.

Apart from the limitations surrounding the methodology of MLSS determination, another limitation that should be addressed is the possibility of a training effect. Participants were required to attend the laboratory on nine separate occasions, and in order to minimize any training stimulus, they were tested on average of two times per week. However, the participants were highly active and were accustomed to the intensity and duration that each visit required. Therefore, the conditions presented in this thesis did not necessarily pose as an extra stimulus for improvements in fitness. Moreover, $MLSS_p$ was always determined first, including a verification ride ($MLSS_{+5\%}$), and the remaining visits were performed in a randomized order. Therefore, two of the "harder" conditions were always performed first, and if they were to invoke a training stimulus, the results of the later conditions (i.e., the subsequent TTE trials) may have been performed for longer durations compared to if they were evaluated first. Furthermore, a limitation of this thesis is that no post-study measures were performed to assess whether participants trained through this study, such as performing a second ramp-incremental test and re-testing each participant's $MLSS_p$.

4.2 Future Directions

The results of this thesis have contributed to understanding how prior constant-PO exercise, performed at specific, domain-derived intensities and for different durations at $MLSS_p$, effects subsequent performance capabilities. Although the goal of this study was to characterize

the pattern of decrease in TTE performance following an increase in either intensity or duration, the mechanisms responsible for these changes have not been investigated. As presented in Chapter Three, based on our understanding of the metabolic perturbations associated with exercise in each intensity domain (2), we proposed that during each constant-PO condition there was an attainment of a new metabolic milieu that may have contributed to the observed reductions in TTE performance. However, further research is required to quantify the conditions of the metabolic milieu during bouts of exercise performed for a standardized duration, within each intensity domain.

From a neuromuscular point of view, the reductions in TTE performance may raise questions about the peripheral and central mechanisms of fatigue. Therefore, the lack of neuromuscular assessment is a limitation of the thesis and an area for future investigations. Neuromuscular assessment (i.e., maximal voluntary contraction, resting twitch force, superimposed twitch, etc.) could have been performed at the end of each constant-PO condition in order to better understand and quantify the implications associated with exercise performed at $MLSS_p$ and within each intensity domain. Apart from characterizing peripheral and central components of fatigue, future investigations should explore different concepts such as the critical threshold of peripheral fatigue and the sensory tolerance limit (18). The sensory tolerance limit may provide information on whole body or global fatigue during exercise and it has been suggested that RPE can be used to measurement the proximity to an individual's sensory tolerance limit. The sensory tolerance limit relies on afferent feedback from different sensory areas within the body, such as the locomotor muscles directly involved in the exercise as well as respiratory muscle feedback (18).

Lastly, to add to the small body of evidence on the effects of prior exercise on subsequent performance, future investigations should examine different outcome measures of endurance performance, such as time trials. In this thesis, the TTE trials could have been replaced by time trials in order to evaluate the total amount of work that could be performed in a set amount of time. Although TTE and time trials are both considered to be accurate and reliable ways of measuring endurance performance (84), time trials may provide a different perspective on the results of this thesis as they pertain to performance.

4.3 Practical Application for Endurance Sport

The results from this thesis may provide useful information into pacing strategies and multistage endurance events. First, the total work results (Figure 3.2) indicate no difference in the total amount of work that could be completed during each 30 min constant-PO condition (performed within the heavy- and severe-intensity domains) and the corresponding subsequent TTE trials. As mentioned, these results indicate a close relationship between exercise performed within both the heavy- and severe-intensity domains. However, these results also highlight that although an athlete who paces slightly below their MLSS might complete the same total amount of work as an athlete who paces slightly above their MLSS, it would take them longer to do so and therefore, they would not win the race. However, this finding is not novel as it is well-understood that an athlete who can sustain a faster pace than their competitors is more likely to succeed in finishing first.

Another way of interpreting the findings from this thesis is by considering the implications that these results have for multistage events such as triathlon. We found that prior exercise varying in intensity effects an individual's ability to complete a subsequent bout of exercise. Regarding triathlon, these results highlight the importance of properly pacing during

each stage of the competition (swim, bike, run) in order to optimize performance during each subsequent stage. This may be supported by previous research which has shown that prior exercise focusing on one set of locomotor muscles (i.e., exercise of the arms) affects the ability to perform a subsequent bout of exercise in a different group of locomotor muscles (i.e., muscles involved in leg cycling) (113, 114). However, further research is required to evaluate the implications surrounding pacing during multistage competitions such as triathlon.

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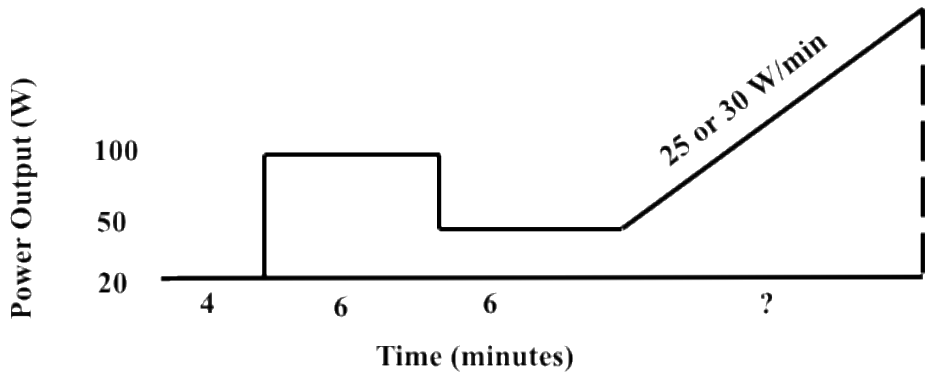
Appendices

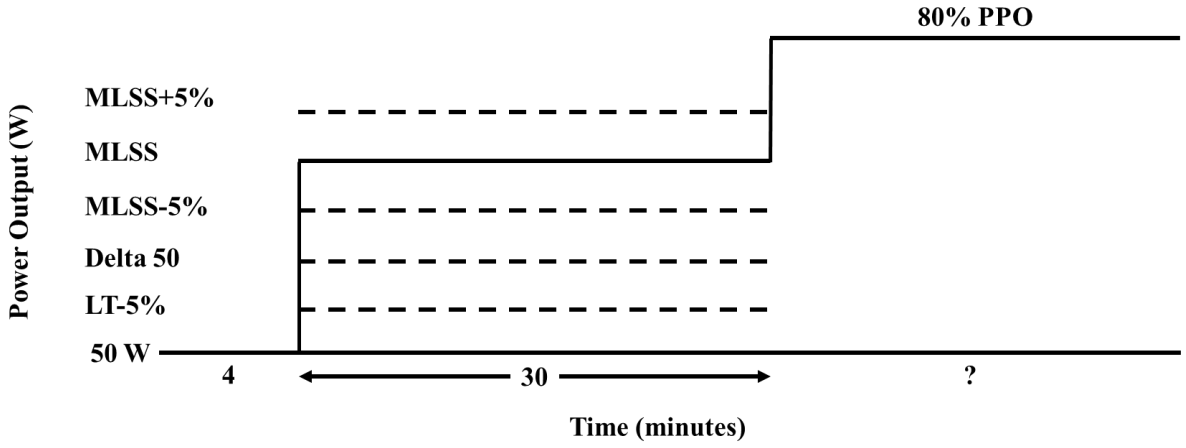
Appendix A: Schematic of the Protocol Presented in Chapter Three

Ramp-Incremental Exercise Test



Constant-PO Conditions

Intensity



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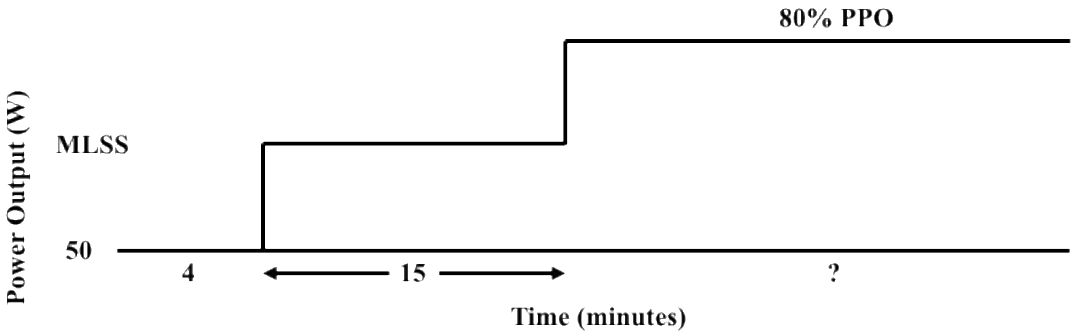
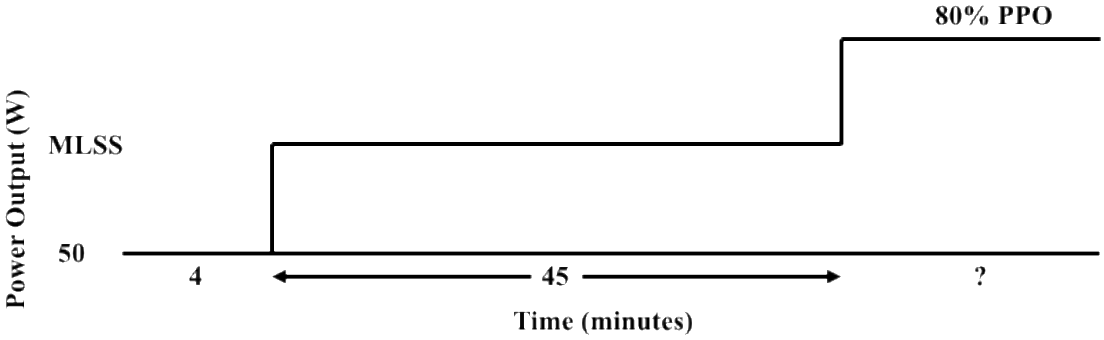
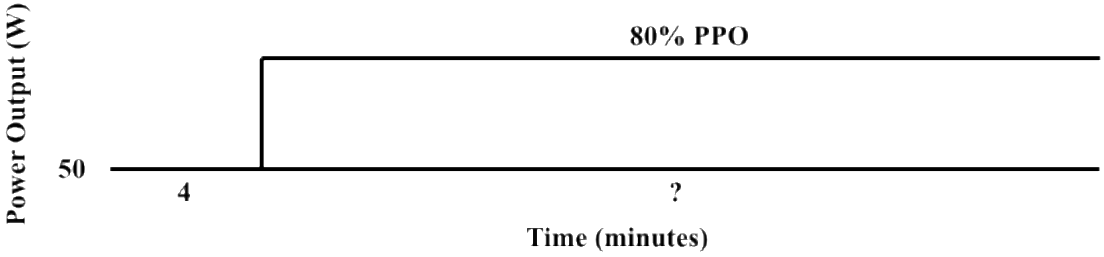




Baseline TTE Trial



Appendix B: Permission to Reuse Any Published Material

Chapter One: Permission from Respiratory Physiology & Neurobiology and from Comprehensive Physiology to adapt Figure 1.1 is attached below:

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Appendix C: Letter of Consent

Letter of Consent

TITLE: The effects of cycling at intensities above and below the maximal lactate steady-state on subsequent time-to-exhaustion performance trials.

SPONSOR: Natural Sciences and Engineering Research Council of Canada (NSERC).

INVESTIGATORS:

Principal investigator
[redacted]
Office phone: [redacted]
Email: [redacted]

Co-investigators
[redacted]
Email: [redacted]

This consent form is only part of the process of informed consent. It should give you a basic idea of what the research is about and what your participation will involve. If you would like more details about something mentioned here, or information not included here, please ask. Take the time to read this carefully and to understand any accompanying information. You will receive a copy of this form.

BACKGROUND

You are being invited to participate in a study that examines how cycling at different exercise intensities affects subsequent exercise performance, and to better understand why these changes in performance occur. The maximal lactate steady-state (MLSS) is highest intensity of exercise where the aerobic energy system dominates, and many of the body's systems are in a steady-state (i.e. oxygen consumption (VO_2), blood lactate). Above MLSS the intensity of exercise places a greater demand on the body's, resulting in large increases in VO_2 and blood lactate that stop you from exercising. Therefore, MLSS is a threshold that is used to improve endurance performance and to properly prescribe exercise. Exercise performance at different intensities will be assessed by determining your MLSS, evaluating your maximal oxygen consumption (VO_{2max}), and by measuring your ability to endure a high intensity of exercise. This study is the first to look into understanding what happens to exercise performance following exercise at varying intensities above and below MLSS.

WHAT IS THE PURPOSE OF THE STUDY?

To examine the effects of cycling at a constant power output at various exercise intensities above and below the maximal lactate steady-state on subsequent exercise performance, measured through a series of time to exhaustion trials.

WHAT WOULD I HAVE TO DO?

To be part of the study, the following inclusion criteria must be followed: healthy, non-smoker, non-obese, between the ages of 18 to 45, and not taking medications known to affect cardiovascular or hemodynamic responses to exercise (e.g., β-blockers, anti-inflammatories, anti-coagulants, etc.). You will be required to pass the Physical Activity Readiness Questionnaire (PAR-Q+) and to meet the following baseline values: resting heart rate <100 bpm, resting systolic blood pressure <144 mmHg, and resting diastolic blood pressure <90 mmHg. Moreover, you will be required to be a recreationally-active to well-trained as we need you to be able to exercise for relatively long periods of time (i.e., 30-45 min).

Once accepted into the study, you will be required to come to the laboratory for approximately 13 sessions that are each about 1.5 hour in duration. Total time commitment will be approximately 19.5 hours over an estimated 4 to 6-week period.

On the first visit to the laboratory, you will complete a progressive exercise test (VO_{2max} test) until you will be physically unable to continue exercising because the intensity is either too high or too uncomfortable. The exercise will consist of cycling on a stationary bicycle (cycle ergometer) while in the upright, seated position. The test will begin with the exercise intensity being very light (very little resistance). After several minutes the exercise intensity will gradually increase until you are unable to continue because of exhaustion, or until you wish to stop. Although the incremental part of the test should last ~10-15 min, this visit should last approximately 1 hour, as explanation of the study and preparation for the test will require some extra time. Additionally, this visit will be used as a guideline for the bike tests in the subsequent visits.

On the second to fourth visits, you will perform a 30-minute cycling trial at a predetermined intensity. During the trial, your breathing parameters and muscle oxygenation will be continuously monitored, as well as blood lactate and your perceived exertion measures will be taken every 5 minutes. Each subsequent visit you will perform the same protocol, but at a slightly higher or lower intensity (+/- 10W), depending on whether steady-state (no significant change in blood lactate and breathing parameters) was observed. These visits will last approximately an hour, and will allow us to identify the highest intensity that your aerobic system can support MLSS. Immediately after these trials you will perform a time-to-exhaustion ride at an intensity greater than that you were previously riding at (i.e., 85% of the highest values during the VO_{2max} test). These trials should last approximately 3-5 min. Should MLSS not be determined in the second and third visit, a fourth visit will be required.

The fifth to thirteenth visits, will occur in a randomized order and will consist of eight different sessions. On separate laboratory visits you will perform a baseline time-to-exhaustion ride, two 30-minute cycling trials at two different intensities above your MLSS (determined during the

second to fourth visits), and three 30-minute cycling trials at different intensities below your MLSS. You will also cycle for 15 and 45 minutes at your MLSS. Immediately after each trial, you will perform a time-to-exhaustion ride. During all eight trials, your breathing parameters, blood lactate, and muscle oxygenation will be continuously monitored.

Measurements: All measurement techniques are used as routine measures in our laboratory. During each of the exercise tests on the stationary bicycle you will be required to wear a mask connected to a volume turbine so that the amount of oxygen that you breathe in and out and your breathing rates can be measured. This will enable us to measure the amount of oxygen that you consume at rest and during exercise. Masks and turbines are disinfected before each test. Similarly, during each of the exercise tests, the oxygen in the thigh muscles will be continuously measured non-invasively using near-infrared spectroscopy (NIRS). NIRS send light into a specific location of your leg muscles (the probe will be placed on your leg approximately midway between your hip and your knee) and measures the amount of light coming out at another location. The amount of light that is reflected and detected by the NIRS probe is used to measure muscle oxygenation. A small piece of equipment (the NIRS probe) will be placed on your upper leg. It will be secured with tape, covered to prevent light from entering or leaving the area, and bound with elastic bandage to minimize movement. Heart rate (how many times per minute your heart beats), stroke volume (how much blood your heart pumps every time it beats), and cardiac output (how much blood your heart pumps in a minute) will be continuously monitored by electrodes placed on the left chest, right chest, and left side under your ribs. After the ramp incremental test, throughout the MLSS tests, and immediately post all TTE trials, lactate will be evaluated by drawing a small blood sample through a small finger prick puncture using a lancet. A drop of blood will be then immediately placed in a testing tube, which is placed into a lactate analyzer, which displays blood lactate concentration results within 60 seconds. This is similar to blood glucose measurement conducted by diabetic patients on their own fingertips.

WHAT ARE THE RISKS?

All exercise carries a minimal risk and may be uncomfortable if you are unfit or not used to doing exercise. In healthy adults who have no signs or symptoms, which may contraindicate exercise, there is no reason to expect any harmful effects of exercise protocols in the moderate to severe intensity exercise domains. To minimize any potential risks during maximal testing, heart rate will be monitored throughout the test and a certified exercise physiologist will be present to ensure clinical competence. There might be some minor discomfort during the exercise testing. You may experience increased awareness of breathing, muscle pain and/or fatigue, increased sweating, or a general feeling of fatigue or nausea, all of which are not unexpected consequences of exercise.
You may experience some minor discomfort from wearing the breathing mask necessary for VO_2 measurements, and by having the NIRS probes secured to your leg during the exercise period. These sensations often become less noticeable with time during the tests. The placement of electrodes and the NIRS probe might require shaving, and removal of electrodes may cause some discomfort. Additionally, mild discomfort from finger prick blood samples might also be experienced. All blood lactate samples will be taken by a trained administrator.

If you have any pre-existing medical conditions, it is essential that you inform the researchers prior to testing, as your participation may carry additional risks. Note that no studies known to date have shown NIRS to have harmful effects on healthy populations. All testing procedures will only be conducted when a lab technician or research assistant that is certified in CPR is present. In the case of an emergency, 911 will be called using a telephone available in the testing laboratory. An automatic external defibrillator is also available within the testing building.

WILL I BENEFIT IF I TAKE PART?

This is a basic physiology study and, as such, there will be no direct benefits received because of participating in the study. If you are interested, the rationale for conducting the research and theory and significance of each of the tests will be explained, as will your individual results from each of the tests. You will also have the opportunity to learn about and better understand your physiological responses to an exercise situation. You are encouraged to ask questions regarding the purpose of the study, specific measures or outcomes of your exercise test, or overall findings and conclusions from this research study.

DO I HAVE TO PARTICIPATE?

Your participation in this research project is entirely voluntary. You can withdraw anytime just by sending an email to [redacted] or by expressing this desire verbally to the investigators.

You might be withdrawn from the study for the following reasons:
Changes in your status so that you do not fit within the admission criteria for this study.
You cannot complete all testing sessions within the proposed period of the study.
You are not able to comply with the instructions prior to each testing session.

If new information becomes available that might affect your willingness to participate in the study, you will be informed as soon as possible.

WHAT ELSE DOES MY PARTICIPATION INVOLVE?

Your participation in this study does not involve anything else beyond what is specified on this informed consent.

WILL I BE PAID FOR PARTICIPATING, OR DO I HAVE TO PAY FOR ANYTHING?

You will not be paid for your participation in this research project. If you are required to pay for parking, a value of $8 cash will be reimbursed to you at the end of the visit to the lab.

WILL MY RECORDS BE KEPT PRIVATE?

Information obtained during this research project is confidential. Nobody except the researchers will have access to your personal information. Your records are listed according to an identification number rather than by your name. Published reports resulting from this study will

not identify you by name. Data will be stored indefinitely as information may be used in combination with other research projects. The data collected for this study and if used in future studies will only be identifiable by the participant identification number that is assigned to you. All data identifiers (i.e. your name) will be destroyed upon the completion of the testing sessions. Thus, your right to privacy will be retained. If you require it, you will be given a summary of your results and the average results for all participants in this study.

IF I SUFFER A RESEARCH-RELATED INJURY, WILL I BE COMPENSATED?

In the event that you suffer injury as a result of participating in this research, no compensation will be provided to you by the University of Calgary, or the Researchers. You still have your legal rights. Nothing said in this consent form alters your right to seek damages.

SIGNATURES

Your signature on this form indicates that you have understood to your satisfaction the information regarding your participation in the research project and agree to participate as a participant. In no way does this waive your legal rights nor release the investigators or involved institutions from their legal and professional responsibilities. You are free to withdraw from the study at any time without consequences. If you have further questions concerning matters related to this research, please contact:

[redacted]

If you have any questions concerning your rights as a possible participant in this research, please contact the Chair, Conjoint Health Research Ethics Board, University of Calgary at 403-220-7990.

Participant's Name	Signature and Date
Investigator/Delegate's Name	Signature and Date
Witness' Name	Signature and Date

The University of Calgary Conjoint Health Research Ethics Board has approved this research study.

A signed copy of this consent form has been given to you to keep for your records and reference.