Estimating the maximal metabolic steady state using critical power: assessment of the adequacy of different models

by

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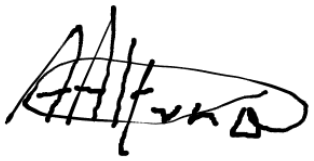
Declaration

No part of this thesis has been submitted in support of an application for any degree or other qualification of the University of Kent, or any other University or Institution of learning.

Signed



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zaituztet, bai egoera onetan bai txarretan. Eman zenezaketen laguntza osoa eman didazue nire helburuak erdiets ditzadan. Ezin da gurasoengandik gehiagorik eskatu.

Abstract

It has been advocated that critical power (CP) should be considered the gold standard to determine the maximal metabolic steady state (MMSS). However, the choice of the model affects the estimation of CP, previous research reporting differences of up to 28% between the lowest and highest CP estimates. The purpose of this thesis was to investigate which of the models, exponential (CP_{exp}), 3-parameter hyperbolic ($CP_{3\text{-}hyp}$), 2-parameter hyperbolic ($CP_{2\text{-}hyp}$), linear (CP_{linear}), and inverse of time ($CP_{1/time}$), estimates MMSS best, defined by the maximal intensity at which an oxygen uptake ($\dot{V}O_2$) steady state is still achievable. Eleven male participants (Age: 31 ± 11 years, Body mass: 70.5 ± 5.6 kg) performed three time-trials (12-, 6-, and 3-min long) to determine CP from the five models. On three subsequent visits, participants cycled for 30-min, or until task failure, at the CP estimated by each model. CP_{exp} estimated the highest CP (303 ± 69 W), followed by $CP_{1/time}$ (272 ± 66 W), CP_{linear} (270 ± 64 W), $CP_{2\text{-}hyp}$ (266 ± 65 W) and $CP_{3\text{-}hyp}$ (262 ± 63 W). $\dot{V}O_2$ stabilised at a significantly lower value than peak $\dot{V}O_2$ ($\dot{V}O_{2peak}$) during exercise at CP_{linear}, $CP_{2\text{-}hyp}$, and $CP_{3\text{-}hyp}$ (94 ± 5%, P = 0.041; 87 ± 4%, P < 0.001; 86 ± 4%, P < 0.001, respectively). CP_{linear} had a mixed individual response, 7 out of 11 participants failing to attain a $\dot{V}O_2$ steady state. $\dot{V}O_2$ stabilisation was not significantly different to $\dot{V}O_{2peak}$ during exercise at CP_{exp} and $CP_{1/time}$ (98 ± 2%, P = 1.000; 94 ± 6%, P = 0.130, respectively). Rate of perceived exertion significantly increased over time during exercise at $CP_{1/time}$ (P < 0.001) and CP_{linear} (P = 0.006) but was unchanged between minute 15 and end-exercise during $CP_{2\text{-}hyp}$ (P = 0.762) and $CP_{3\text{-}hyp}$ (P = 0.569). Lactate increased significantly in the last 10, 15, and 20 minutes of the exercise for all models. No model had an

increase of ≤ 1 mmol · L^{-1} from minute 10 to 30. These results suggest that $CP_{2\text{-hyp}}$ or $CP_{3\text{-hyp}}$ should be favoured when CP is used to assess MMSS.

Keywords: threshold; oxygen uptake; lactate; endurance

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Abbreviations

^{31}P-MRS	Phosphorus magnetic resonance
ATP	Adenosine triphosphate
ADP	Adenosine diphosphate
AMP	Adenosine monophosphate
ANOVA	Analysis of variance
AWC	Anaerobic work capacity
CP	Critical power
$CP_{1/time}$	Inverse of time critical power linear model
$CP_{2\text{-}hyp}$	2-parameter hyperbolic critical power model
$CP_{3\text{-}hyp}$	3-parameter hyperbolic critical power model
CP_{exp}	Exponential critical power model
CP_{linear}	Work-time linear critical power model
CS	Critical speed

CT	Critical torque
GET	Gas exchange threshold
HCO_3^-	Bicarbonate or hydrogencarbonate
IAT	Individual anaerobic threshold
LDH	Lactate dehydrogenase
log	Logarithm
LT	Lactate threshold
MAP	Maximal aerobic power
MLSS	Maximal lactate steady state
MMSS	Maximal metabolic steady state
PCr	Phosphocreatine
PFK	Phosphofructokinase
pH	Potential hydrogen
P_i	Inorganic phosphate
$p\dot{V}O_{2max}$	Power at $\dot{V}O_{2max}$

RCP	Respiratory compensation point
RER	Respiratory exchange ratio
$s\dot{V}O_{2max}$	Speed at $\dot{V}O_{2max}$
TT	Time-trial
$\dot{V}CO_2$	Carbon dioxide production
$\dot{V}O_2$	Oxygen uptake
$\dot{V}O_{2max}$	Maximal oxygen uptake
$\dot{V}O_{2max}$	Peak oxygen uptake
VT_1	First ventilatory threshold
VT_2	Second ventilatory threshold
W'	Work done above critical power

1. Introduction

Endurance events are often characterised by athletes striving to complete a given distance in the shortest possible time. Such a performance is determined by a number of factors (Coyle 1999), with critical power (CP) being regarded as a key endurance performance determinant (Poole *et al.* 2016).

The importance of CP relies on the fact that it separates sustainable from nonsustainable exercise. Below CP, metabolic markers such as oxygen uptake ($\dot{V}O_2$), muscle and blood lactate, inorganic phosphate (P_i), pH, and phosphocreatine (PCr) reach a steady state; above CP, such markers fail to achieve a steady state and they reach maximal or minimal values that lead to exercise intolerance (Poole *et al.* 1988; Jones *et al.* 2008; Vanhatalo *et al.* 2016; Black *et al.* 2017). Therefore, CP marks one of the most important fatigue thresholds that influences endurance performance (Craig *et al.* 2019).

CP is obtained by determining the horizontal asymptote of the power-duration relationship, by fitting the CP model to 3 or more maximal efforts lasting 2 to 15 min (Vanhatalo, Jones and Burnley 2011). When adequate methodology is used for CP estimation, CP has repeatedly shown to differentiate steady state from nonsteady state exercise (Poole *et al.* 1988; Jones *et al.* 2008; Burnley, Vanhatalo and Jones 2012; Vanhatalo *et al.* 2016; Black *et al.* 2017; Nixon *et al.* 2021). Given that, CP has been considered to be the gold standard to estimate the maximal metabolic steady state (MMSS) (Jones *et al.* 2019b).

However, various models exist to estimate CP, and they provide different CP estimates (Gaesser *et al.* 1995; Bull *et al.* 2000; Bergstrom *et al.* 2014; Mattioni Maturana *et al.* 2018). Given the difference in the CP estimates, it can be expected the models estimating higher CP may provide a CP estimate that is in an exercise intensity domain where a steady state is not achieved, while the models estimating the lower CP may provide a CP estimate that is in an exercise intensity domain where a steady state is achieved. If CP is to be considered the gold standard for MMSS estimation but different CP models estimate different CP values, potentially in different exercise intensity domains, which of the models should be considered the standard for MMSS estimation?

To date, no research has been conducted comparing the physiological responses to exercise at the CP obtained from the different CP models. Given that gap in the literature, the aim of this thesis is to assess which of the CP models is the most adequate model to obtain a CP estimate that adequately represents MMSS.

2. Literature review

2.1 Physiological determinants of endurance performance

"Endurance" has been defined as the capacity to sustain a given speed or work rate for the longest possible time (Jones 2006). However, such a definition can be applied to a vastly different spectrum of durations. For the purposes of this thesis, events or activities lasting longer than 2 minutes (up to several hours) will be considered as endurance events or activities. When exercise is conducted for 2 minutes or longer, oxidative mechanisms are the primary source of energy supply (Jones 2006; Brooks, Fahey and Baldwin 2019). Endurance exercise relies on the aerobic resynthesis of adenosine triphosphate (ATP). Thus, in this thesis, the focus will be on the determinants of oxidative energy production as they are the primary source of energy supply for endurance events. "Performance" will be considered as the ability to complete a given amount of work in the minimum amount of time possible or the ability to sustain a work rate for the maximum possible duration (Coyle 1999). Therefore, "endurance performance" refers to the ability to complete an activity longer than 2 minutes at the maximum possible work rate.

Several factors determine endurance performance; however, three factors (Figure 2.1) are usually considered as the principal determinants: maximal oxygen uptake ($\dot{V}O_{2max}$), gross mechanical efficiency, and fractional utilization of $\dot{V}O_{2max}$ (Coyle *et al*. 1988; Coyle 1995, 1999; Jones 2006; Joyner and Coyle 2008). Although those three factors are the main determinants of endurance performance, nonoxidative energy sources (*i.e.*, phosphagen system and nonoxidative glycolysis) also affect endurance performance, to a lesser extent. The shorter the event is in the spectrum of duration, the larger the contribution of nonoxidative energy sources is.

The three determining factors of endurance performance are considered *functional* abilities (Coyle 1995; Coyle 1999). They are called "functional" because they are a function of fundamental physiological properties, but they are not fundamental physiological properties themselves. Those abilities combined turn into *performance* abilities such as power or speed. Likewise, *functional* abilities are the product of *morphological* abilities. The *functional* abilities and the impact of *morphological* abilities on them will be analysed below.

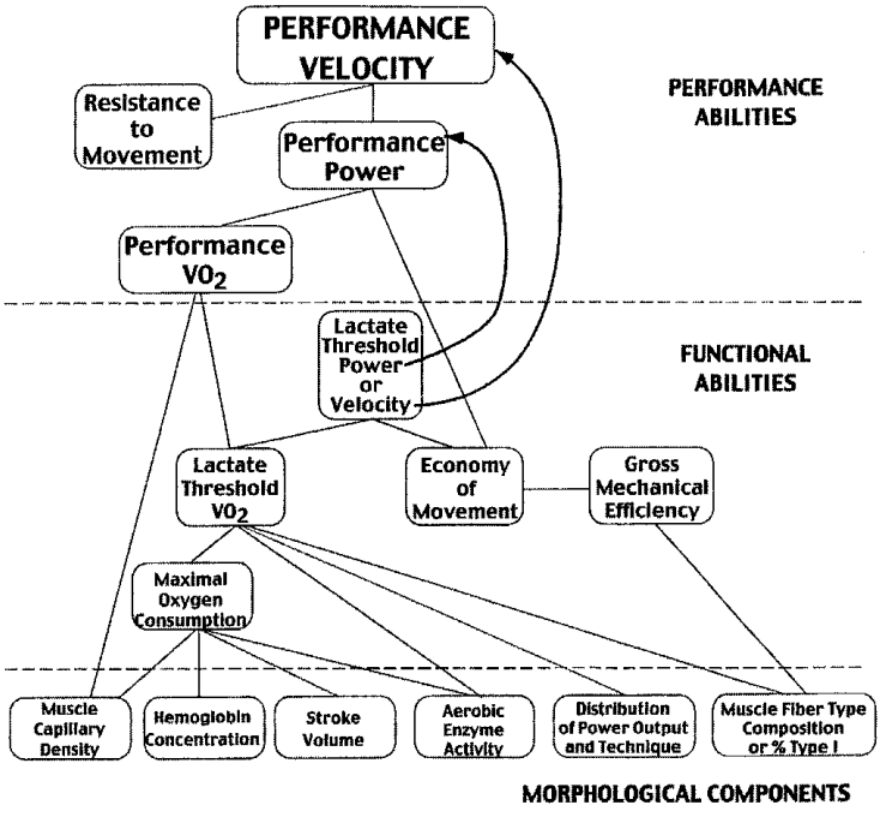

Figure 2.1: Endurance performance determinants divided into morphological components or abilities, functional abilities, and performance abilities (Coyle 1999).

2.1.1 $\dot{V}O_{2max}$

$\dot{V}O_{2max}$ is the maximal oxygen uptake, which sets the upper limit of oxidative energy production. $\dot{V}O_{2max}$ has been described as a good measure of cardiovascular fitness, it has been correlated to endurance performance, and it is common to see the highest values in elite athletes (Jones 2006). However, the highest $\dot{V}O_{2max}$ values are usually found in athletes specializing in the shorter end of the duration spectrum of endurance events. Given that the power associated with $\dot{V}O_{2max}$ ($p\dot{V}O_{2max}$), or speed ($s\dot{V}O_{2max}$) in the case of running, is sustainable for approximately 5 minutes (Billat *et al.* 1996), it is to be expected that events lasting around 5 minutes are better correlated with $\dot{V}O_{2max}$ when compared to longer events. In longer events, oxygen consumption values lower than $\dot{V}O_{2max}$ are sustained.

$\dot{V}O_{2max}$ can be divided into its components in the form of the Fick equation:

$$\dot{V}O_2 = (f_h)(V_s)(a - v)O_2$$

where:

f_h = HR = frequency of the heart or heart rate

V_s = SV = stroke volume

$(a - v)O_2$ = arteriovenous oxygen difference; the difference in oxygen content between the arteries and veins

When the components are maximized, they result in $\dot{V}O_{2max}$. That is, an athlete's $\dot{V}O_{2max}$ will be equal to the product of his maximal heart rate, maximal stroke volume, and maximal arteriovenous oxygen difference if the maximum values can be sustained simultaneously.

The primary factor affecting $\dot{V}O_{2max}$ is stroke volume (Coyle 1995; Bassett and Howley 2000). It is thought that the ability to deliver oxygen is more important than the ability of the mitochondria to consume oxygen (Holloszy and Coyle 2016). However, even if stroke volume is considered the main factor limiting $\dot{V}O_{2max}$, it is not the only factor. The ability to deliver oxygen is affected by haemoglobin concentration. An increased haemoglobin concentration increases $\dot{V}O_{2max}$ while a decreased concentration reduces $\dot{V}O_{2max}$ (Ekblom, Wilson and Astrand 1976). Another factor contributing to oxygen delivery is capillary density. An increase in capillary density maintains or elongates mean transit time. That causes a greater ability to deliver oxygen by maintaining oxygen extraction at higher rates of muscle blood flow (Saltin 1985).

At the periphery, mitochondrial content, mitochondrial enzyme activity and myoglobin also affect $\dot{V}O_{2max}$, but by increasing the consumption of oxygen by the mitochondria (Honig, Connett and Gayeski 1992; Holloszy and Coyle 2016). However, its effects in $\dot{V}O_{2max}$ are rather low when compared to stroke volume. Yet, it improves endurance performance by increasing the fat oxidation rate.

Changes in morphological abilities will lead to a greater ability to supply energy by oxidative mechanisms, which will lead to better endurance performance. However, the changes in performance from a higher $\dot{V}O_{2max}$ are more prevalent in shorter endurance events rather than longer endurance events.

2.1.2 Gross mechanical efficiency

Among the endurance performance determinants, gross efficiency probably is the least studied and understood factor (Jobson *et al.* 2012). While exercising, the body converts chemical energy into mechanical energy to perform external work. Via the oxidative system, mitochondria produce ATP, the chemical intermediate to power muscle contractions, from oxygen and substrates such as glucose and fatty acids. ATP then provides the chemical energy to allow muscle contractions, producing mechanical energy, which can then complete external work.

However, all the chemical energy liberated in the body is not turned into mechanical work. Hence, there is a discrepancy between the energy liberated and the completed work. The ratio of external work to liberated energy is known as efficiency, and, in the case of cycling, where external work can be calculated, gross mechanical efficiency. The formula to calculate efficiency is as follows:

$$\text{Gross mechanical efficiency} = \frac{\text{external work}}{\text{metabolic energy cost}}$$

External work can be calculated in cycling with the use of power measuring devices, while the metabolic energy cost can be calculated via indirect calorimetry with gas exchange measuring equipment. Therefore, performance abilities are the product of the combination of $\dot{V}O_2$ (metabolic energy cost) and gross mechanical efficiency. The product of $\dot{V}O_2$ and gross mechanical efficiency is external work, and external work divided by time is work rate or power. Therefore, the best combination of $\dot{V}O_2$ and gross mechanical efficiency would lead to the better performance ability. For example, there may be two athletes of identical height and weight,

athlete “A” and athlete “B”, having a different $\dot{V}O_2$ for a given duration (1 hour). Athlete A has a $\dot{V}O_2$ of 4.5 L · min^{-1}, and athlete B has a $\dot{V}O_2$ of 4.2 L · min^{-1}. By looking at $\dot{V}O_2$ only, it would be inferred that athlete A is a better athlete and that they would perform better than Athlete B during an event lasting 1 hour. However, if athlete A had a gross efficiency of 21% but athlete B had a gross efficiency of 24%, athlete B would be most likely to perform better during an endurance event. Using Lusk’s formula (Lusk 1924) and assuming a gas exchange ratio (RER) of 1.00, the product of 4.5 L · min^{-1} is 1583.75 J · s^{-1}, while the product of 4.2 L · min^{-1} is 1478.52 J · s^{-1}. When gross efficiency is considered, the external work rate or power is 333 W for athlete A and 355 W for athlete B. Thus, despite athlete A having the higher $\dot{V}O_2$, athlete B would have a better performance due to a better combination of $\dot{V}O_2$ and gross mechanical efficiency.

While there is a considerable body of evidence on the limiting factors of $\dot{V}O_{2max}$, the picture is less clear regarding gross mechanical efficiency. However, gross mechanical efficiency seems to respond more markedly to training than $\dot{V}O_{2max}$ (Hopker *et al.* 2012), and it has been shown to improve with the introduction of high-intensity training (Hopker *et al*. 2010). Among the morphological factors that affect gross mechanical efficiency, muscle fibre type composition was initially identified (Horowitz, Sidossis and Coyle 1994). Type I fibres have been described as more efficient fibres. Therefore, a greater percentage of Type I fibres would lead to a greater gross mechanical efficiency. However, Hopker *et al.* (2013) cast doubt on the hypothesis that muscle fibre type determines efficiency.

2.1.3 Fractional utilisation of $\dot{V}O_{2max}$

Even if the combination of $\dot{V}O_{2max}$ and gross mechanical efficiency produces a performance ability, power, it does not fully explain endurance performance. As mentioned, endurance events last more than 2 minutes, up to several hours. In the shorter end of the spectrum, the combination of $\dot{V}O_{2max}$ and gross mechanical efficiency are the main performance determinants due to exercise being done near $\dot{V}O_{2max}$, in combination with nonoxidative energy production. A world-class runner running 1 mile to 3 kilometres will spend the whole distance (after reaching $\dot{V}O_{2max}$) at or near $\dot{V}O_{2max}$ due to the short duration of the event. Similarly, a track cyclist doing a 4000-meter individual pursuit will spend the whole distance (after reaching $\dot{V}O_{2max}$) at or near $\dot{V}O_{2max}$. In those cases, or any case in which the duration is near 5 minutes, the best combination of $\dot{V}O_{2max}$ and gross mechanical efficiency will yield the best performance, tactics and nonoxidative energy production aside.

During longer endurance events (>5 minutes), $\dot{V}O_{2max}$ cannot be sustained for the whole duration. In those cases, the fractional utilization of $\dot{V}O_{2max}$ will play a factor in performance. While in the shorter events stroke volume and the determinants of gross mechanical efficiency will mostly determine performance, in longer events mitochondrial content, enzymes and peripheral factors will gain relevance. Those factors will affect the lactate threshold (LT), which serves as a reference point for fractional utilisation.

A study comparing $\dot{V}O_{2max}$ and gross mechanical efficiency with performance (Støren *et al.* 2013) found that LT had the best correlation with a 15-kilometre time-trial (TT). However, maximal aerobic power (MAP), obtained from an incremental test, was also correlated with TT

performance, but to a lower degree. Given that LT was measured as power output and that power output is the product of internal energy production (*i.e.*, $\dot{V}O_2$) and gross efficiency, comparing the correlation of LT to the correlation of MAP (also measured as power output) with performance is fairer. Furthermore, in elite athletes, improvements in performance have been associated with LT rather than $\dot{V}O_{2max}$ (Jones 2006). In the case study, Jones found that $\dot{V}O_{2max}$ remained stable throughout the career of the athlete, while LT increased from 14–15 km · h^{-1} to 17.5–18.5 km · h^{-1}. These results suggest that fractional utilization is a key performance factor in events lasting longer than ~20 minutes. It is a better performance predictor than $\dot{V}O_{2max}$, gross mechanical efficiency, or the combination of both, MAP. Thus, LT and fractional utilisation prove to be important determinants of endurance performance for any athlete competing or participating in events over 20 minutes. In the following section, LT and other thresholds at which fractional utilisation is measured will be covered.

2.2 Physiological thresholds

The rationale behind measuring fractional utilisation at LT or other thresholds is that they refer to points that establish a boundary between different domains of physiological control (Poole *et al.* 2020). When crossing such a threshold, the metabolic behaviour changes. The importance behind that is that thresholds demarcate the sustainability of an effort. If we transition from sustainable to nonsustainable exercise, the performance implications are clear: exhaustion time will decrease rapidly as fatigue is accelerated. Thus, independently from $\dot{V}O_{2max}$, thresholds will

affect how sustainable a bout of exercise will be. As shown in Figure 2.2, in general, two thresholds are differentiated in endurance exercise.

The first threshold differentiates moderate and heavy exercise. Moderate exercise is characterised by resting-like lactate concentration and steady $\dot{V}O_2$ ~2 minutes after the onset of exercise (Barstow *et al.* 1994; Black *et al.* 2017). Crossing the first threshold and staying below the second threshold, thus exercising in the heavy exercise intensity domain, will cause an increase in lactate from resting levels and the development of the $\dot{V}O_2$ slow component, a continual increase in $\dot{V}O_2$ without a change in external load (Barstow and Mole 1991; Black *et al.* 2017). However, heavy exercise is sustainable as lactate and $\dot{V}O_2$ achieve a steady state. In contrast, the second threshold differentiates sustainable from nonsustainable exercise, and in doing so, the heavy and severe exercise intensity domains. Severe exercise, exercise above the second threshold, is characterized by an increase in lactate and $\dot{V}O_2$ that does not achieve a steady state (Poole *et al.* 1988).

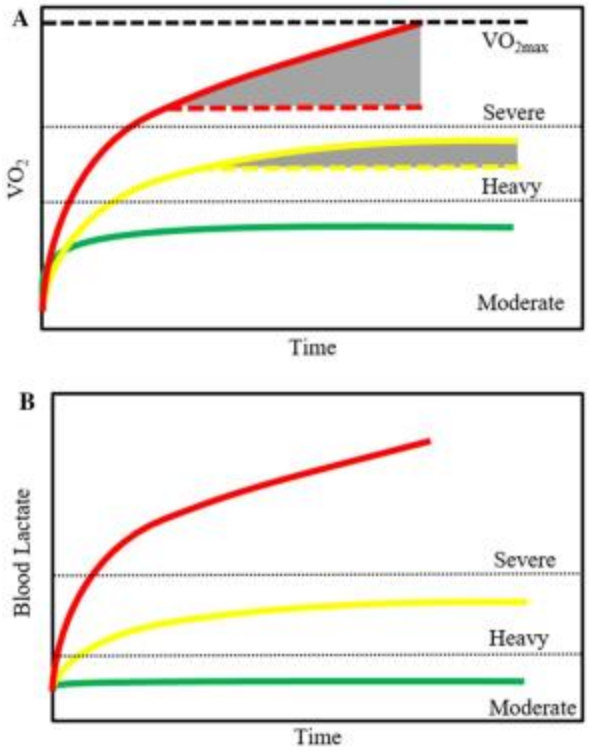

Figure 2.2: $\dot{V}O_2$ (panel A) and blood lactate (panel B) responses to exercise in the different intensity domains. The green line represents moderate exercise, the yellow line heavy exercise, and the red line severe exercise. The lower dashed line represents the first threshold and the upper dashed line represents the second threshold. **A** $\dot{V}O_2$ is steady soon after exercise onset at an elevated level during moderate exercise. During heavy exercise, $\dot{V}O_2$ reaches a level at which it would stabilise during moderate exercise, the yellow dashed line, but it keeps increasing slowly due to the slow component, grey area, until it reaches an elevated steady state. During severe exercise, the slow component also occurs but it does not stabilise and $\dot{V}O_2$ reaches $\dot{V}O_{2max}$. **B** Blood lactate stays at resting levels during moderate exercise. During heavy exercise, it elevates from resting levels and stabilises at an elevated level. During severe exercise, blood lactate fails to stabilise and keeps increasing until exhaustion (Jamnick *et al.* 2020).

2.2.1 Origins of the threshold concept

The threshold concept can be traced back to the early studies of Hill (1925). By the analysis of athletic records, Hill identified a hyperbolic relationship between speed and time. When plotted

speed against time of athletic records over various distances, the relationship tended towards a horizontal asymptote (Figure 2.3).

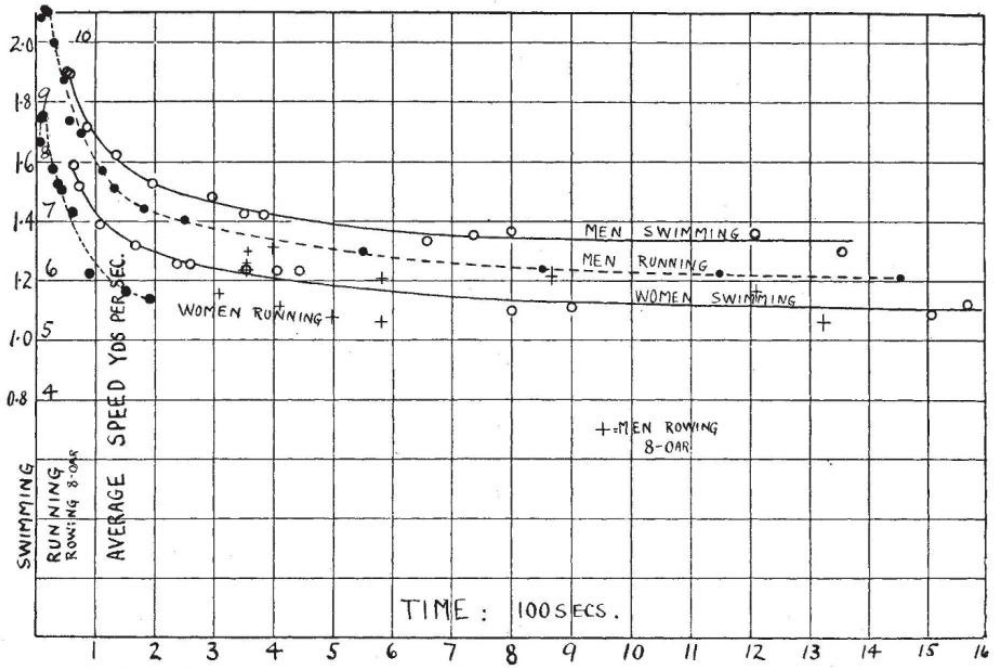

Figure 2.3: The speed-duration relationship of athletic records. As duration increases, the curve tends towards a horizontal speed asymptote (Hill 1925).

According to Hill, the fatigue above the horizontal asymptote was caused by the "initial factor" in fatigue. That kind of fatigue was said to be due to the oxygen debt. When an exercise task needs to be completed as quickly as possible, Hill thought that the time to complete it would depend on the maximal oxygen uptake and the maximal oxygen debt, both providing energy via different sources. The energy debt would be an additional amount of energy coming from the formation of lactic acid. Lactic acid would be oxidised later, after exercise ceases or intensity is decreased. Thus, exercise above that horizontal asymptote would depend on the maximal oxidative capacity and the maximal oxygen debt produced by lactic acid formation. However, even if the speed against time tended towards a horizontal asymptote, speed plotted against the

logarithm of time, where longer durations appeared compressed to cover up to 100 miles, the fall was continued throughout its length. For Hill, that further fall beyond the asymptote was due to a different kind of fatigue. That fatigue would be due to the "exhaustion of the material of the muscle" or "incidental disturbances". The speed at the asymptote marks a physiological transition. Despite not naming it "threshold", Hill's description mimics the definition of "threshold"; after the flattening of the curve, a different domain of physiological control occurs. This first notion of an exercise threshold was found by analysing an external measure or output: speed.

In a similar line, Owles (1930) identified a critical metabolic level above which blood lactate increased. As opposed to Hill, this threshold was identified by analysing an internal measure: lactate. He tied that increase in blood lactate to a local deficiency in oxygen of the working muscles. That lack of oxygen would cause an increase in lactate. However, that threshold was different to Hill's threshold. Owles threshold marked a demarcation from baseline levels. That is, a threshold demarcating intensities that have resting-like physiological behaviour from nonresting-like physiological behaviour. Hill's threshold, on the other hand, demarcates sustainable intensities from nonsustainable intensities. Hill's and Owles' thresholds will set the two different types of thresholds, conceptually, that will be reproduced in various ways.

2.2.2 The first threshold

Following the idea of a first threshold characterised by transitioning to a nonbaseline-like behaviour, Wasserman and McIlroy coined the term "anaerobic threshold" (Wasserman and

McIlroy 1964; Wasserman *et al.* 1973). Given that there was an intensity above which lactate started to increase, Wasserman and McIlroy hypothesized a noninvasive method to measure the first threshold based on gas exchange data. Based on the finding that the fermentation of lactic acid was buffered by bicarbonate (HCO_3^-), they proposed that the buffering would cause an increase in expired CO_2 ($\dot{V}CO_2$). During exercise, $\dot{V}O_2$ increases linearly coupled with intensity to provide the energy requirements of the increased work rate via aerobic means. $\dot{V}CO_2$ is coupled with $\dot{V}O_2$ in that increase until there is a demarcation, which is also characterised by an increase in RER against $\dot{V}O_2$ (Figure 2.4). Such demarcation, in Wasserman's view, was due to exhaled CO_2 from lactic acid buffering via HCO_3^-. That demarcation point was identified as the anaerobic threshold. The crossing of the anaerobic threshold was thought to cause a transition to anaerobic metabolism due to a lack of oxygen in the muscle (a hypothesis dating back to Hill's work (Hill, Long and Lupton 1924)), hence the use of the word "anaerobic".

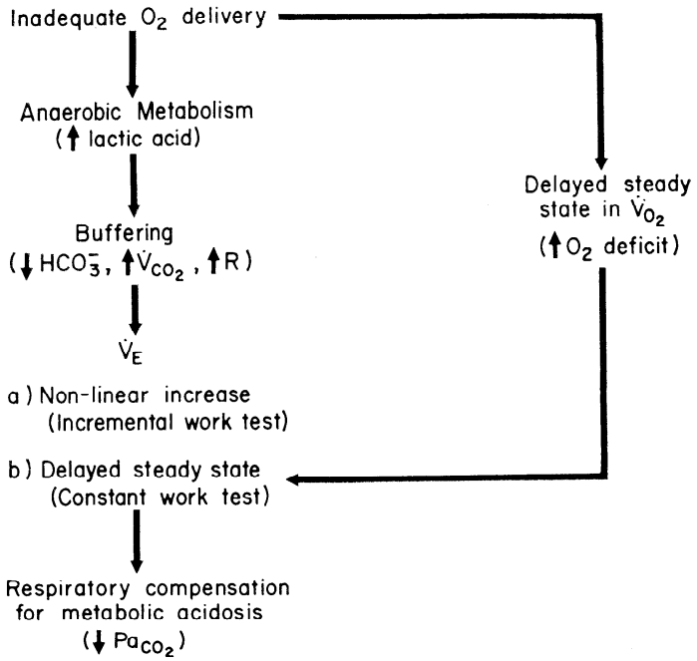

Figure 2.4: The physiological mechanism behind the anaerobic threshold (Wasserman *et al.* 1973).

Different names and methods were developed to refer to and identify the anaerobic threshold. Hollman proposed the "point of optimal ventilatory efficiency" during a conference in 1959, which he covers in a later publication (Hollmann 1985). The point of optimal ventilatory efficiency was defined as the point at which ventilation increased nonlinearly while $\dot{V}O_2$ kept increasing linearly. The point of optimal ventilatory efficiency has later been called the first ventilatory threshold (VT_1) (McLellan 1985). The greater level of ventilation is tied to the increase in CO_2, as the rise in CO_2 causes a rise in CO_2 partial pressure, which stimulates an increase in ventilation mediated by the carotid bodies (Wasserman *et al.* 1975). However, the methods listed above require visual inspection to identify the threshold. In order to overcome that, Orr *et al*. (1982) developed a linear regression method to identify the anaerobic threshold. The first breakpoint in the modelled ventilation against $\dot{V}O_2$ was identified as the anaerobic threshold or VT_1 (Figure 2.5). Similarly, Beaver, Wasserman and Whipp (1986) developed the V-slope method. The change in slope of the modelled $\dot{V}CO_2$ against $\dot{V}O_2$ marks the anaerobic threshold (Figure 2.6).

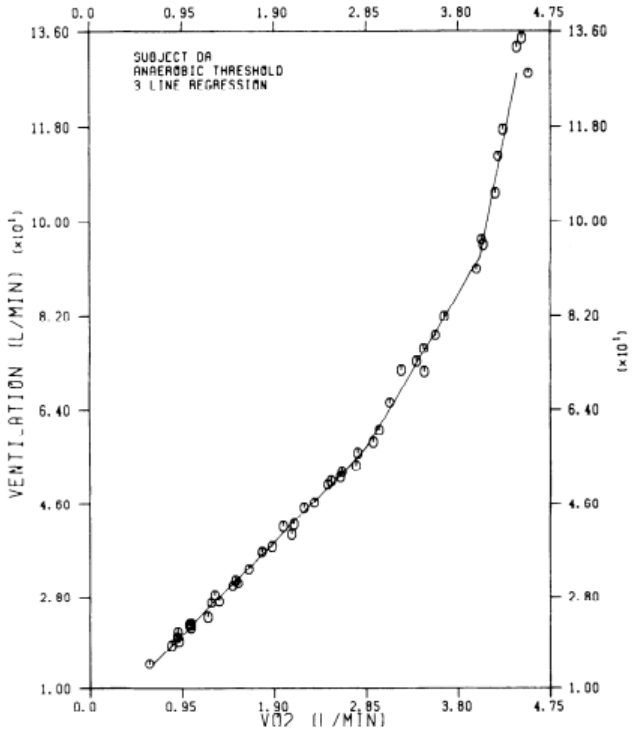

Figure 2.5: Ventilation plotted against $\dot{V}O_2$. Linear regression is performed and the first breakpoint represents the anaerobic threshold (Orr *et al.* 1982).

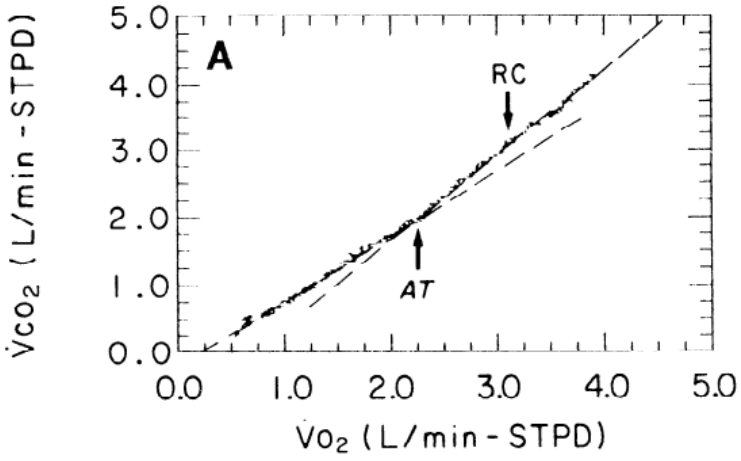

Figure 2.6: $\dot{V}CO_2$ plotted against $\dot{V}O_2$. The intersection of the two lines established by linear regression marks the anaerobic threshold (Beaver, Wasserman and Whipp 1986).

Constant load tests also showed differences in oxygen uptake kinetics between intensities below the anaerobic threshold and above it (Whipp and Wasserman 1972). Below the anaerobic threshold, $\dot{V}O_2$ remains constant after the initial rise. However, above the anaerobic threshold, $\dot{V}O_2$ keeps increasing slowly until it achieves a delayed steady state. That rise is called the "slow component". More recently, the term "gas exchange threshold" (GET) has been used to refer to the first threshold based on gas exchange data (Yoshida 1987; Yoshida *et al.* 1989; Jamnick *et al.* 2020).

Lactate-based methods to identify the first threshold have remained similar to Owles', despite an increase in terms to refer to the same phenomenon. Described as the point above which blood lactate increases from resting values, it is now commonly referred to as the "lactate threshold" (Black *et al.* 2017). The first use of the term "lactate threshold" can be traced back to the late 1970s (Pendergast, Cerretelli and Rennie 1979), despite it being decades later than its initial characterisation. A more thorough analysis of the term came by Yoshida (Yoshida 1987), setting the definition of an increase in lactate from baseline values. His group also provided empirical evidence of the correlation between the lactate threshold and the gas exchange threshold (Yoshida *et al.* 1989), giving credibility to the underlying mechanism outlined by Wasserman and McIlroy. Applied to the same definition, Brooks (1985) named the lactate threshold the "lactate break point", and Hofmann *et al.* (1997) named it the "first lactate turn point".

To avoid the visual identification of the lactate threshold, Beaver, Wasserman and Whipp (1985) developed the "log-log model" to identify the lactate threshold. The method consists of plotting lactate against $\dot{V}O_2$ in a logarithmic scale for both the *y* and *x* axes (Figure 2.7). When

plotted, a transition from a slow increase to a rapid increase is seen. The intersection of the two phases, represented by linear regression, marks the lactate threshold. Other objective methods have been developed, such as an increase of 0.5 mmol · L^{-1} from baseline levels (Zoladz, Rademaker and Sargeant 1995), an increase of 1.0 mmol · L^{-1} from baseline levels (Coyle 2005), or a fixed lactate value of 2.0 mmol · L^{-1} (Mader *et al.* 1976). Although there is no research comparing the validity of the different methods to separate intensity domains, given the high correlation and prediction similarity between the visually inspected lactate threshold, the lactate threshold defined by an increase of 0.5 mmol · L^{-1} from baseline levels, and the log-log method, the three of them seem appropriate to establish the first threshold (Davis *et al.* 2007).

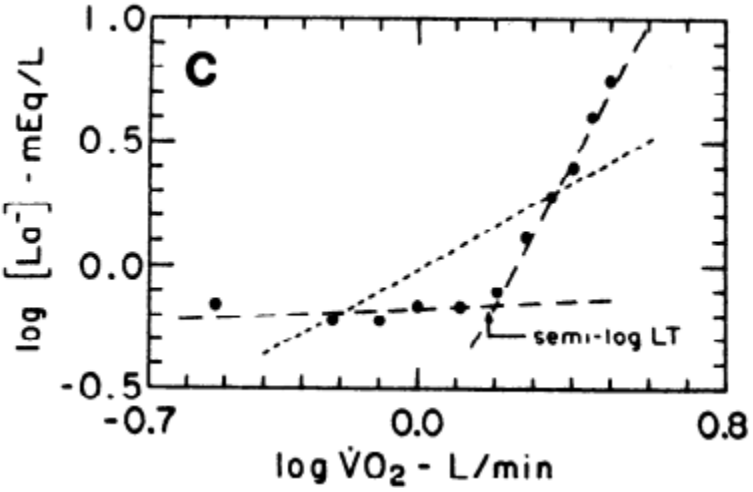

Figure 2.7: Log lactate plotted against log $\dot{V}O_2$ with the log-log model being the intersection of the two long-dash lines. "semi-log LT" is not marking the intersection but the LT determined by using a logarithmic scale for lactate; it is marked to show the difference between the semi-log LT and the log-log LT (Beaver, Wasserman and Whipp 1985).

Given the proposed causal relationship between the lactate and gas exchange thresholds, they should provide similar estimations. In fact, similar values have been found (Yoshida *et al.* 1989; Pallarés *et al.* 2016). However, the differences between them, even if not big, suggest that their interchangeable use may not be completely adequate, and that further research should be done to identify if they separate intensity domains adequately.

The lactate-based and gas exchange-based thresholds intend to represent the same physiological phenomenon by different means. Their physiological significance relies on their ability to represent the transition from baseline or resting-like behaviour to nonresting-like behaviour, by the loss of baseline homeostasis. This transition, even if it has a well-documented physiological basis, has several problems.

First, there is a terminological problem. The first threshold, initially coined the "anaerobic threshold", has been called the "aerobic threshold", and "anaerobic threshold" has also been used to refer to the second threshold (Kindermann, Simon and Keul 1979). The terminological choice was made because above the first threshold and below the second threshold lactate is steady, which suggests no additional anaerobic contribution (Mader *et al.* 1976). Thus, they chose to call the first threshold the "aerobic threshold" and the second threshold the "anaerobic threshold". On top of that, different terms have been used to refer to the same phenomenon (see Table 2.1). The use of "lactate threshold" for the lactate-based threshold and the use of "gas exchange threshold" for the gas exchange-based threshold is advisable given their historical development, current use, and the physiological phenomenon they represent.

Table 2.1: Terms used to refer to the first threshold

First threshold
Anaerobic threshold
Point of optimal ventilatory efficiency
GET
VT_1
Lactate threshold
Lactate break point
First lactate turn point
Aerobic threshold

Secondly, there is a problem in the use of the term "anaerobic threshold" for the physiological phenomenon it refers to. The mechanism behind the anaerobic threshold has been subject to many critics (Jones and Ehrsam 1982; Brooks 1985; Yoshida 1987; Hopker, Jobson and Pandit 2011; Poole *et al.* 2020). The main critique has been that there is no hypoxic state in the muscle above the anaerobic threshold and that the increase in lactate above the anaerobic threshold is due to an inability of the mitochondria to oxidise lactate at that rate (for a detailed analysis, see Poole *et al.* 2020).

However, even if the term "anaerobic threshold" is not appropriate to refer to the first threshold, the first threshold still separates resting-like behaviour from nonresting-like behaviour due to lactate values being above baseline and the appearance of the $\dot{V}O_2$ slow component, among other physiological phenomena. The first threshold separates different physiological domains, *i.e.*, the moderate from the heavy intensity domain, which has practical implications for training prescription on top of physiological validity.

2.2.3 The second threshold

With the emergence of lactate and gas thresholds, a second threshold was identified in the late 70s. The first characterisation of this second threshold was done by Mader and colleagues, who named it the "aerob-anaerobic threshold" (Mader *et al.* 1976; Mader and Hollmann 1977). Its physiological significance relied on the fact that it sets the upper limit of purely aerobically supplied energy. Below the aerob-anaerobic threshold, lactate achieves a steady state; above it, lactate fails to stabilise and increases until exercise cessation. To identify the aerob-anaerobic threshold during an incremental test, they set a lactate value of 4 mmol · L^{-1}. However, later work from the same group set the current standard for measuring the aerob-anaerobic threshold, which has been named the "maximal lactate steady state" (MLSS) since then (Heck *et al.* 1985). The criteria to determine MLSS is an increase of less than 1 mmol · L^{-1} in the last 20 minutes of a constant-load 30-minute bout of exercise (Figure 2.8). Given the differences in lactate values at MLSS, this method has been the standard instead of the set 4 mmol · L^{-1} value achieved during an incremental test (Beneke and Von Duvillard 1996).

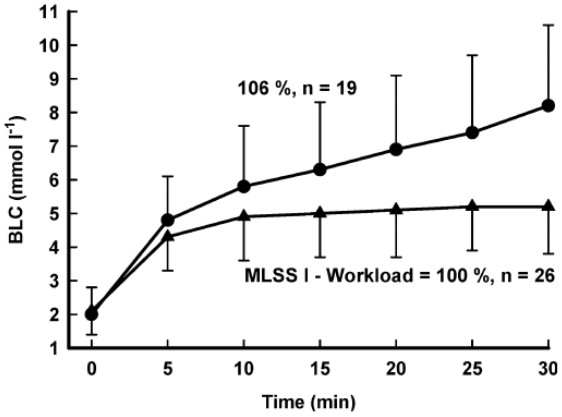

Figure 2.8: Blood lactate plotted against time. The triangles represent blood lactate at a workload equating to MLSS. A steady state in blood lactate is achieved, as opposed to exercising at 106% of MLSS where blood lactate keeps rising (Beneke 2003).

Similarly to the first threshold, a gas exchange-based threshold was also identified for the second threshold (Beaver, Wasserman and Whipp 1986). The second gas exchanged-based threshold was named the "respiratory compensation point" (RCP) or second ventilatory threshold (VT_2) (McLellan 1985). It is identified by a demarcation between $\dot{V}CO_2$ and ventilation in which ventilation is increasing more rapidly than $\dot{V}CO_2$ (Figure 2.9). This hyperventilation is thought to be caused by metabolic acidosis, and hyperventilation is a mechanism to overcome such acidosis. Meyer *et al.* (2004) tested that hypothesis by injecting sodium bicarbonate intravenously to subjects exercising on an ergometer. Their conclusion was that they proved the causal relationship between lactic acidosis and hyperventilation, thus, they claimed to set the physiological basis of the RCP, which had not been set yet. However, their results do not corroborate their claims. At the point of injection, pH was elevated and remained above 7.37 throughout the incremental test. There was a delay in RCP, but RCP still happened even if the blood pH was near resting or above resting values. Thus, the causal relationship was not shown. There is no strong physiological basis to RCP, and given that it occurs at intensities well above MLSS and other methods to determine the second threshold (Dekerle *et al.* 2003; Nakamura *et al.* 2009; Caen *et al.* 2018; Galán-Rioja *et al.* 2020), it appears to be a threshold without any clear physiological reference, other than an increase in ventilation.

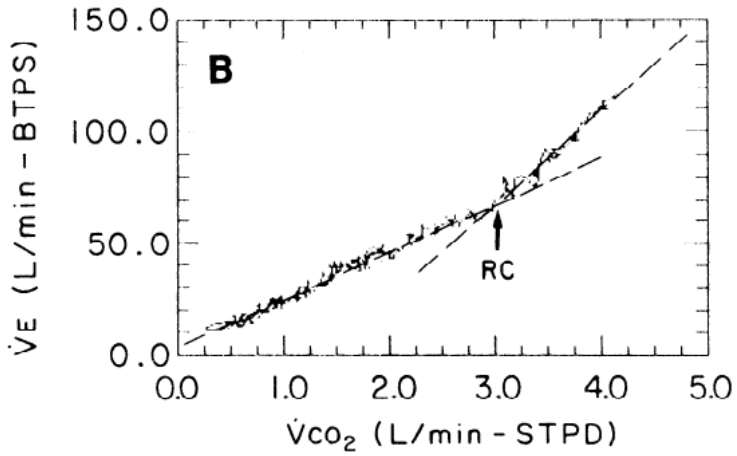

Figure 2.9: Ventilation plotted against $\dot{V}CO_2$. An increase in ventilation relative to $\dot{V}CO_2$, identified by a breaking point of the two lines established by linear regression, marks RCP (Beaver, Wasserman and Whipp 1986).

Given the issues related to RCP and the robust physiological basis of MLSS, MLSS has been considered the standard for identifying the maximal metabolic rate at which energy is supplied solely by aerobic means and a steady state can be maintained (Beneke 2003; Billat *et al.* 2003).

However, the effort required to properly identify MLSS, which requires four to five laboratory visits, has led to the development of single-visit testing protocols that aim to estimate MLSS. As an example, the individual anaerobic threshold (IAT) was developed with that aim (Stegmann, Kindermann and Schnabel 1981). An individual's lactate response to incremental exercise is plotted and the point of equilibrium between maximal lactate elimination and diffusion is the IAT (Figure 2.10)

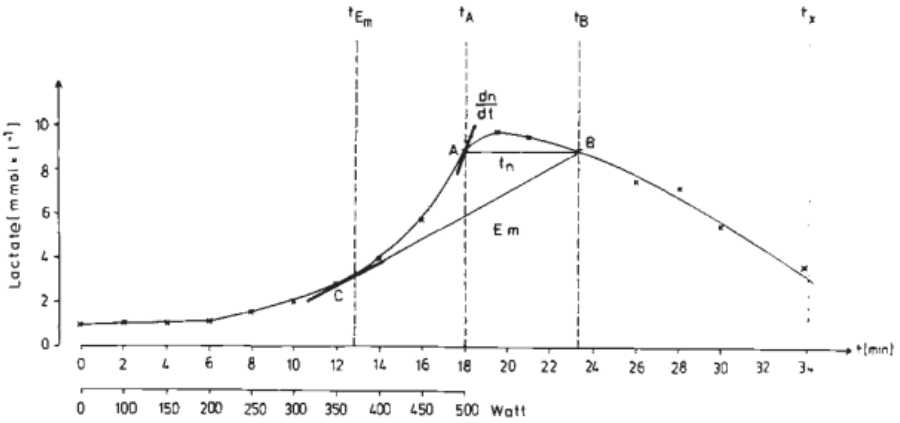

Figure 2.10: Lactate plotted against time during an incremental test. The dashed vertical line t_{En} marks IAT, as it is the contact point of the maximal elimination rate tangent.

IAT estimations of MLSS have proven not to be accurate (Beneke 1995), which undermines the basis upon which IAT is created: the maximal intensity at which lactate elimination and diffusion are at equilibrium. Further protocols have been developed to estimate MLSS by using incremental protocols, most of them providing values other than MLSS; a thorough comparison can be found in Jamnick *et al.* (2018). The condition of any validation study of IAT or any other threshold estimated by incremental protocols is that MLSS truly represents the maximal metabolic steady state (MMSS). There is not a clear definition of MMSS in the literature, although it is characterised by being the highest metabolic rate at which physiological homeostasis is achievable, as marked by the attainment of a steady state in $\dot{V}O_2$ at submaximal levels, stable PCr levels, stable pH, stable P_i levels, and stable muscle lactate levels (Jones *et al.* 2019b). However, for the purposes of this thesis, MMSS will be operationally defined as the maximal intensity at which a submaximal steady state in $\dot{V}O_2$ is still achievable, as per Nixon *et al.* (2021). If any of the proposed thresholds to characterise the second threshold (Table 2.2) is compared against MLSS to assess its validity, MLSS needs to represent MMSS.

This has been regarded to be the case, as shown earlier, but recent evidence is casting doubt on whether MLSS is the best indicator of MMSS (Bräuer and Smekal 2020; Nixon *et al.* 2021).

Table 2.2: Terms used to refer to the second threshold

Second Threshold
Aerob-anaerobic threshold
Maximal lactate steady state
Respiratory compensation point
VT_2
Individual anaerobic threshold
Anaerobic threshold

The critique of MLSS as an adequate marker of MMSS, gathered in a paper from Jones *et al.* (2019b), has two lines of argument. The first line criticises the methodology of MLSS determination. As previously pointed out, MLSS is the highest intensity at which there is an increase in blood lactate of less than 1 mmol $\cdot$ L^{-1} in the last 20 minutes of a 30-minute exercise bout. One of the issues with this methodology is the achievement of a steady state later than 10 minutes into the bout of exercise. Given that only two datapoints, the 10-minute and 30-minute datapoints, are considered for the determination of MLSS, the initial rise in lactate could take longer than 10 minutes and start to stabilise at 15 minutes. In such a case, that intensity would be above MLSS despite achieving a steady state after 15 minutes. Another methodological issue is that the measured MLSS will necessarily be below the actual MLSS. Given that the protocol requires to increase the intensity of each 30-minute bout by 10-30 W (Beneke 2003), MLSS will always be between the determined MLSS and the next step, with an average error of 5-15 W. A final methodological concern is the error of blood lactate measuring equipment. Commonly used

hand-held lactate analysers have shown to have an error of 0.2-0.5 mmol · L^{-1} when compared to a criterion lactate analyser, affecting the determination of power output at different lactate thresholds (Bonaventura *et al.* 2014). They also found that biological variation could affect lactate values up to ~50%, in line with previous data from Saunders *et al.* (2004).

The second line of argument criticises the adequacy of blood lactate as a marker of the metabolic state of the body and working muscles. In the 80s it was shown how there was a discrepancy between blood lactate and muscle lactate levels (Tesch, Daniels and Sharp 1982). These discrepancies are due to the differences between lactate production in the muscle, lactate efflux from the muscle to the blood, and lactate uptake from the blood (Stainsby and Brooks 1990). Lactate produced in the muscle is transported to the blood, increasing linearly with work rate up to a release of 4.5 mmol · L^{-1}, after which it levels off (Jorfeldt, Juhlin-Dannfelt and Karlsson 1978). The fate of that lactate is to be oxidised by the liver, heart, and, mainly, oxidative muscle tissue (Stainsby and Brooks 1990). This evidence leads to viewing blood lactate levels as a marker that does not align with muscle lactate levels, which makes blood lactate an inadequate marker of the muscle metabolic state. However, if the MLSS achieves stable muscle lactate levels, it could still be adequate for assessing MMSS. In a paper from Vanhatalo *et al.* (2016), they showed that, at an intensity exceeding MLSS, muscle lactate levels were still steady (Figure 2.11). That intensity caused an increase in blood lactate from minute 10 to minute 24 of 1.8 mmol · L^{-1}, which would likely be more if the exercise bout was prolonged to 30 minutes. Additionally, muscle PCr and pH remained stable.

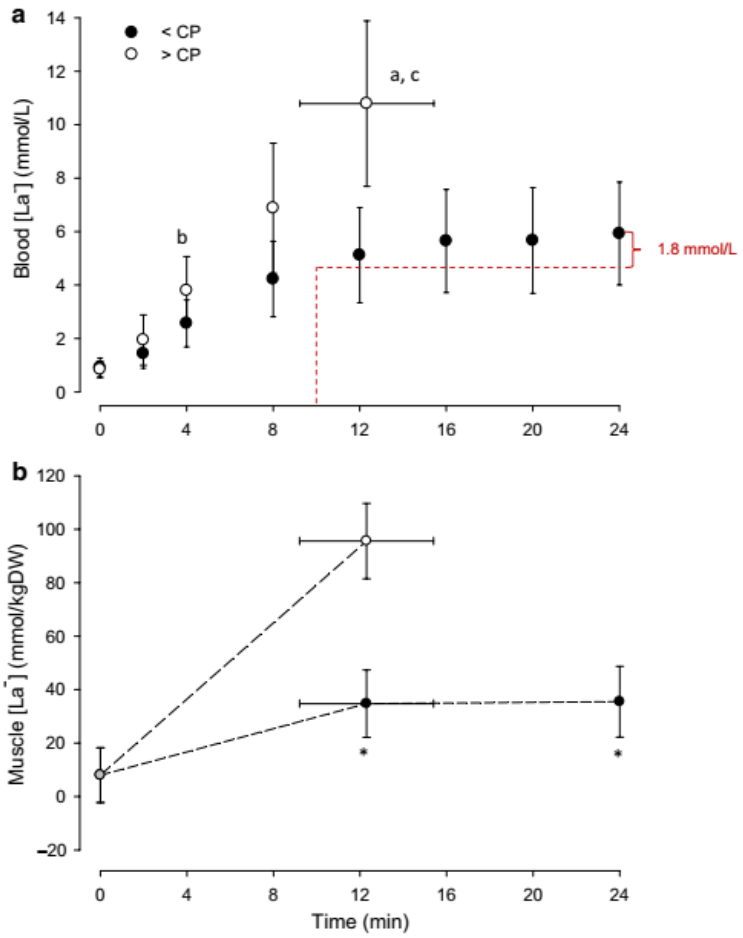

Figure 2.11: Plot "a" shows blood lactate plotted against time during a trial above MLSS (filled black circles). There is an increase of more than 1 mmol · L^{-1}. However, in plot "b" it can be seen that muscle lactate levels remained steady although exercise was above MLSS (Jones *et al.* 2019a).

A second indicator of blood lactate not being an adequate marker of the metabolic state is the achievement of a steady $\dot{V}O_2$ at intensities exceeding MLSS (Bräuer and Smekal 2020; Nixon *et al.* 2021). A stable $\dot{V}O_2$ is characteristic of the heavy intensity domain (Burnley and Jones 2007). When intensity exceeds the heavy intensity domain, a steady state is no longer achievable and $\dot{V}O_2$ keeps increasing until $\dot{V}O_{2max}$ if the exercise is prolonged sufficiently. MLSS, as a marker of MMSS, would mark the upper limit of the heavy intensity domain. However, if $\dot{V}O_2$ is still in a steady state above MLSS, at least one indicator of the heavy intensity domain is still

present above MLSS. In fact, this has been reported several times (Mattioni Maturana *et al.* 2016; Iannetta *et al.* 2018; Bräuer and Smekal 2020). All three studies made participants exercise at MLSS and 10 W above MLSS. Visual inspection of the data from Mattioni Maturana *et al.* (2016) and Iannetta *et al.* (2018) shows a steady state in $\dot{V}O_2$ for both MLSS and exercise 10 W above MLSS. Bräuer and Smekal modelled the $\dot{V}O_2$ kinetics and quantified the steadiness of $\dot{V}O_2$ by measuring the increase in $\dot{V}O_2$ in phase III of the $\dot{V}O_2$ kinetics. However, it could be argued that Bräuer and Smekal's modelling is incorrect given that they use a 3-component exponential model and subsequently apply the time points and phases erroneously. Their phase I comprises both phase I and phase II of the $\dot{V}O_2$ kinetics, their phase II comprises the early part of phase III, and their phase III comprises the middle and later part of phase III. Thus, the marker they use to assess steady state, the increase rate in phase III, does not comprise the whole phase three. It cuts the early part of phase III, where the biggest increase in $\dot{V}O_2$ happens. However, given that the later part of phase III is assessed to identify a steady state and that Bräuer and Smekal's included the later part of phase III in their analysis, their conclusions are not affected.

The validity of blood lactate to reflect the metabolic state is hindered due to its discrepancy with intramuscular markers such as muscle lactate, PCr, pH and the $\dot{V}O_2$ kinetics. Given that, the validity of MLSS to reflect MMSS is also hindered. In order to establish a marker of MMSS and overcome the limitations of MLSS, Jones *et al.* (2019b) propose critical power as the gold-standard.

2.3 The mathematical modelling of MMSS: critical power

Unlike the previously seen lactate-based and gas exchange-based thresholds, CP does not rely on any physiological measurement for its determination. CP relies solely on performance data: power in the case of cycling or rowing, and speed in the case of running. Based on that data, a mathematical model of the power-duration relationship can be produced. The power-duration relationship represents a hyperbolic form, providing two parameters: CP and W′. CP is the horizontal power asymptote of the curve (when power is represented in the *y* axis and time in the *x* axis), while W′ is the curvature constant, representing the work done above CP. Originally, W′ was referred to as the anaerobic work capacity (AWC). However, the use of W′ has been favoured as it just represents the work done above CP, without implying that the work reserve is anaerobic. The assessment of CP and W′ does not necessarily need the use of laboratory equipment, which makes its practical use easier. In this section, its history, physiological basis, adequacy to represent MMSS, and limitations will be analysed.

2.3.1 Historical development of CP

CP has a relatively long history, its concept dating back to Hill's work in the 1920s (Hill 1925). Although he did not model the data from athletic records, the graphical representation and the identification of a horizontal asymptote towards which speed tends mimic the CP model. In fact, the description of the horizontal asymptote as a level that demarcates different types of fatigue is similar to the description of CP as the threshold between the heavy intensity domain and the severe intensity domain (Poole *et al.* 2016). However, the origin of CP and its associated model

is credited to Monod and Scherrer (1965). Monod and Scherrer analysed the relationship between time and muscle contractions. Their analysis showed a hyperbolic relationship between force and duration, which they represented graphically, introducing CP as the force rate or power towards which contractions tend as duration increases. Monod and Scherrer's model was based on the work from Rohmert (1960) who also represented the same relationship graphically, showing the same asymptote and curve. Even though he analysed the same phenomenon, Rohmert did not introduce the term "critical power" or provide the mathematical formula which would persist to date. Monod and Scherrer provided the formula of the power-duration relationship, which is as follows:

$$t_{lim} = \frac{a}{P - b}$$

where t_{lim} equals the duration for which a given power can be sustained, a is the energetic reserve, P is the maximal power for which duration is calculated, and b is CP. In the graphical representation b or CP sets the vertical asymptote (when power is represented in the *x* axis and time in the *y* axis) and a or W′ sets the curvature constant. The relationship can also be presented in a linear form, with the following formula:

$$W_{lim} = a + b \cdot t_{lim}$$

where W_{lim} replaces P as the maximal work that can be done for a given duration. In this case, in the graphical representation, a or W′ sets the *y* axis intercept of the line, and b or critical power sets the slope of the line. From the work of Monod and Scherrer (1965), two problematic statements have persisted throughout the literature: "When the imposed power is inferior or

equal to the critical power, it is evident from the former equation . . . that **exhaustion cannot occur**" and "*The critical power* of a muscle (or a muscular group) corresponds to the maximum rate it can keep up **for a very long time without fatigue**" These two statements and the idea that CP is an intensity that can be sustained without fatigue will be reasons for criticising the CP model as proposed by Monod and Scherrer (Morton 2006; Gorostiaga, Sánchez-Medina and Garcia-Tabar 2021).

The application of the CP model to whole-body exercise started in the 80s by Moritani *et al.* (1981). Participants exercised at several power outputs until task failure. The work achieved for each duration was plotted against time and represented linearly, using the same formula as Monod and Scherrer. From these data, CP and the energy reserve were calculated with a good model fit (R^2 ranging from 0.982 to 0.998 ($P < 0.01$). In addition, Moritani *et al.* (1981) correlated CP to the anaerobic threshold ($r = 0.907$, $P < 0.01$). That correlation supported the idea that CP represented the maximal intensity at which fatigue does not occur. However, the data from Moritani *et al.* (1981) have been criticised for not properly assessing CP given its closeness to the anaerobic threshold (Jones *et al.* 2010). Moritani *et al.* (1981) used the original definition of the anaerobic threshold: an increase in ventilation and $\dot{V}CO_2$ compared to $\dot{V}O_2$, seemingly representing the first threshold. Given that CP separates the heavy intensity domain from the severe intensity domain, and, therefore, represents the MMSS or second threshold, it should be at a higher intensity than the anaerobic threshold or first threshold. However, the protocol established by Moritani *et al.* (1981) has been maintained to calculate CP (Vanhatalo, Jones and Burnley 2011). Three or more maximal trials in the severe intensity domain are performed, ranging from 2 to 15 minutes. The CP model is then fitted, linearly or nonlinearly, to the obtained results from the maximal trials, and a CP estimate and a W′ estimate are obtained.

An early review of the work done on CP by Hill (Hill 1993), and later developed by Morton (2006) proposed the underpinning assumptions on which the CP model is established. The assumptions are as follows: first, energy supply has only two components, aerobic and anaerobic; second, the aerobic supply is rate limited (CP) but not limited in capacity ("exhaustion cannot occur"); third, the energy reserve or W′ is limited in capacity but not in rate; fourth, exercise is ceased when W′ is depleted. Implicitly, it is also assumed that: CP is available at the onset of exercise and remains available indefinitely; the model applies in the power domain $CP < P < \infty$; and in the time domain $0 < t < \infty$. However, this assumption leads to evident errors such as the ability to produce infinite power when duration tends to zero, or the infinite t_{lim} at CP.

In order to overcome the problem of power tending to infinite when time tends to zero, Morton introduced the 3-parameter model (Morton 1996). The 3-parameter model sets the maximum value that power can have, avoiding the vertical asymptote (when representing power in the *y* axis and time in the *x* axis). This affects the fourth assumption given that exercise can cease before the depletion of W′ (*i.e.*, at t = 1 exercise ceases near maximum power instead of near-infinite where it should cease if all W′ was depleted). It also affects the determination of both CP and W′ given that it sets CP below and W′ above the 2-parameter model. This causes an estimation of CP closer to MLSS which has led certain researchers to believe that the 3-parameter model provides a more adequate estimate of CP than the 2-parameter model (Billat *et al.* 2003). The mathematical formula for the 3-parameter model is:

$$t_{lim} = \frac{W'}{P - CP} - \frac{W'}{CP - P_{max}}$$

where t_{lim} is the duration for which maximal power is calculated, P is the power for which the longest sustainable time is calculated, and P_{max} is the maximal peak power that can be achieved.

Attempts to overcome the issue of the horizontal CP asymptote have also been made, so that the model fits long-duration data where power falls below CP (Puchowicz, Baker and Clarke 2020). The model developed by Puchowicz, Baker and Clarke, the omni-domain power-duration model, is based on the 2-parameter CP model but it limits the available W′ at short durations using an exponential model and reduces power below CP as duration increases by integrating a log-linear model. The consequent model is described by the following equations:

$$P = \frac{W'}{t_{lim}}\left(1 - e^{-t_{lim}\frac{P_{max}-CP}{W'}}\right) + CP;\ t_{lim} \leq TCP_{max}$$

$$P = \frac{W'}{t_{lim}}\left(1 - e^{-t_{lim}\frac{P_{max}-CP}{W'}}\right) + CP - A\ \cdot Ln\left(\frac{t_{lim}}{tCP_{max}}\right);\ t_{lim} > TCP_{max}$$

where P is the maximal power that can be sustained for a given duration, t_{lim} is the longest duration for which P can be sustained, P_{max} is the maximal power, and tCP_{max} represents the time to task failure at CP, which is set at 1800 seconds or 30 minutes. This can be adjusted to the individual's time to task failure at CP. The mean percentage residuals from the model fits were 4 ± 1%, a seemingly well-fitting model that overcomes two of the main issues with the theoretical assumptions of the original CP model.

In addition to the hyperbolic representation of the power-duration relationship to determine CP, other methods have also been used. One such method is the exponential model (Hopkins *et al.* 1989). The exponential model predicts power better for shorter durations (from 0

to 180 seconds), partly because it limits the maximal power that can be achieved. In addition, the use of an exponential decay appears to better fit the decay in power output for short durations. Given that it is an exponential model, it uses the exponent tau (τ), and it does not provide a W′ estimate:

$$P = CP + (P_{max} - CP)\, e^{-\frac{t_{lim}}{\tau}}$$

A method not using several trials to establish the power-duration relationship is the 3-minute all-out test (Vanhatalo, Doust and Burnley 2007). By completing a single bout of exercise, arguably the most time-efficient method to estimate CP, Vanhatalo, Doust and Burnley were able to achieve comparable results to a regular CP test. The test consists of pedalling against a fixed resistance as hard as possible constantly for 3 minutes. The start is at the maximal peak power, and power decays as the participants fatigue. Towards the end of the 3 minutes, power stabilises, and the mean power of the last 30 seconds is taken as the CP estimate. The basis of the test is that W′ will be emptied leading to the sustained end-test power being equivalent to CP. However, the test seems to underestimate W′, leading to shorter duration predictions for a given power output. The methodology for this particular test is important, given that different ergometer settings do not produce valid CP estimates (Karsten *et al.* 2014). The test's ecological validity can also be undermined given the difficulty to complete it out on the road; performing it out on the road can be dangerous due to the severity of the effort, and it would be difficult to replicate as a fixed resistance for that length of time is difficult to find (*i.e.*, straight road with consistent pavement, gradient, and wind).

Despite the issues of the CP model with a vertical asymptote and CP being sustainable infinitely, these are mathematical consequences of the model and do not need to be considered as physiological phenomena. An attempt to explain all physiological phenomena with a mathematical model is likely to be a failure. Instead, being aware of the limits and applicability of a certain model is more appropriate. For instance, the applicability of the CP model is within the severe intensity domain, ranging intensities of 2 to 15 minutes, or even up to 30 minutes (Vanhatalo, Jones and Burnley 2011). Restraining its use for power/speed and duration prediction within that range will likely provide valid results. On the other hand, trying to apply it to considerably longer or shorter durations will likely lead to erroneous estimations of CP. However, given that the focus of this thesis is to assess whether CP is a valid measure of MMSS, it is not of importance whether it can predict the power output for a 1-minute maximal effort, or whether it can be sustained indefinitely. If CP can provide an adequate estimate of MMSS and separate the heavy intensity domain from the severe intensity domain, it is useful enough.

2.3.2 The validity of CP to estimate MMSS

Given that the CP model is solely based on performance data such as power or speed, the model itself does not give a physiological justification of the phenomena described. In order to do that, empirical data of the physiological response when exercising at, above, and below CP are needed.

There are differences in several metabolites and physiological parameters when exercising over MMSS against below MMSS. Intramuscularly, in the severe intensity domain, a

continuous loss of homeostasis occurs where muscle lactate, P_i, and H^+ levels increase, and PCr and pH levels decrease to maximal and minimal levels, respectively (Jones *et al.* 2008; Black *et al.* 2017). Moreover, no steady state in blood lactate and K^+ levels is seen, coupled with an increase in $\dot{V}O_2$ until reaching maximal values. On the contrary, in the heavy intensity domain, the homeostatic milieu is perturbed and values different to baseline values are achieved, but a delayed steady state is achieved in intramuscular parameters, blood lactate, and $\dot{V}O_2$. Thus, if CP is a valid estimate of MMSS it should differentiate those two physiological states.

An early study analysing one of the factors that differentiate the heavy intensity domain from the severe intensity domain, $\dot{V}O_2$, found that exercising at CP leads to a steady state, while exercise ~15 W above CP did not (Poole *et al.* 1988). After estimating CP with data from 5 maximal trials, cyclists exercised for 24 minutes, or until task failure, at CP and ~15 W above critical power. Visual inspection shows steady $\dot{V}O_2$ at critical power, while a steady state is not achieved above CP (Figure 2.12). In addition, task failure occurred near 20 minutes for the exercise over CP. Blood lactate had a larger increase when exercising above CP, but a steady state was not achieved exercising at CP, showing a discrepancy between $\dot{V}O_2$ and blood lactate. pH of arterialised venous blood remained stable at CP, while it decreased above CP. These data suggest that, despite no intramuscular analysis, there was a metabolic steady state at CP while there was not above CP, despite increasing lactate levels. Later work making participants exercise ~10% above and below CP has shown similar $\dot{V}O_2$, blood lactate, and pH results (Vanhatalo *et al.* 2016; Black *et al.* 2017). Vanhatalo *et al.* (2016) found steady $\dot{V}O_2$ below CP, while blood lactate was not steady. When exercising above CP neither $\dot{V}O_2$ nor lactate were steady. Black *et al.* (2017) displayed similar results but blood lactate was steady below CP in

their study. Nixon *et al.* (2021) also found that exercise above critical speed (CS) (~0.4 km · h^{-1} over CS) did not achieve a $\dot{V}O_2$ steady state but exercise below CS (~0.5 km · h^{-1} below CS) achieved a $\dot{V}O_2$ steady state (Figure 2.13)

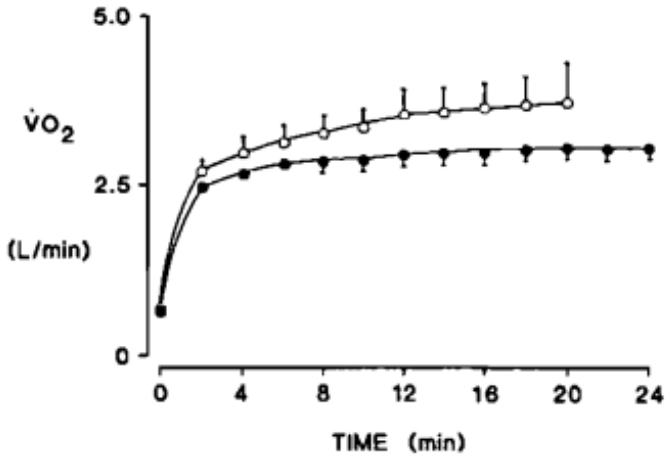

Figure 2.12: Oxygen uptake from 0 min to 24 min at CP (black filled circles) and above CP (white filled circles). A steady state is attained at CP but not above CP (Poole *et al*. 1988).

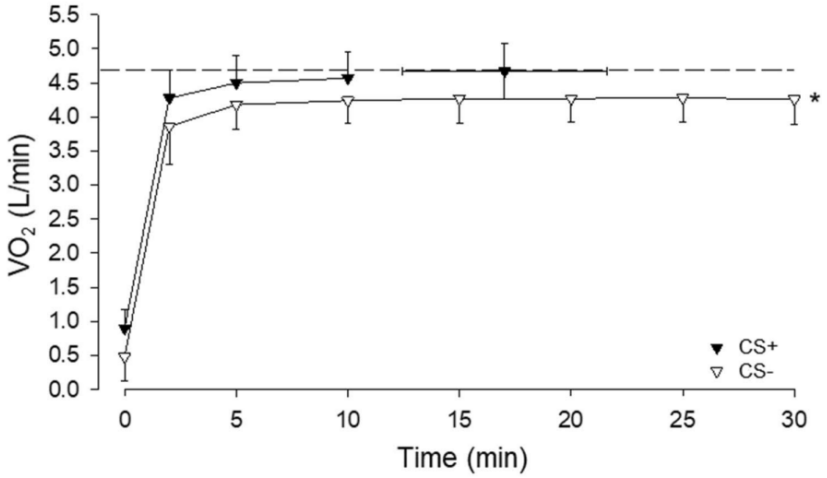

Figure 2.13: Oxygen uptake below CS (white triangles) and above CS (black filled triangles). The dashed line indicates $\dot{V}O_{2peak}$. *End-exercise $\dot{V}O_2$ significantly different from $\dot{V}O_{2peak}$ (Nixon *et al*. 2021).

The analysis of intramuscular metabolites and parameters also provides evidence of CP as a demarcation point of the heavy intensity domain and severe intensity domain. Jones *et al.*

(2008) asked participants to exercise 10% above and below CP for 20 minutes or until task failure while they estimated PCr, P_i, and pH in the contracting quadriceps muscle by ^{31}P-MRS. After an initial decrease, PCr and pH remained stable throughout. P_i increased initially but then stayed stable throughout. Exercise above CP caused a continuous increase of P_i and a continuous decrease of PCr and pH, until task failure. Muscle biopsies led to the same results for Vanhatalo *et al.* (2016) and Black *et al*. (2017). In both studies, muscle lactate was measured, and they found stable muscle lactate below CP against nonstable muscle lactate above CP. Final values of muscle lactate were more elevated above CP than below CP in both studies.

Neuromuscular data also support the findings mentioned above. Burnley, Vanhatalo and Jones (2012) analysed peripheral and central fatigue of isometric contractions (3 seconds of contraction and 2 seconds of rest) above and below the critical torque (CT). They found significant differences in peripheral and global fatigue when doing contractions below and above CT; contractions 10% below CT had a 4 to 5 times smaller reduction in maximal voluntary contractions than above CT. Although these data come from isometric contractions and it analyses CT instead of CP, there are similar data showing discrepancies in neuromuscular fatigue when exercising below and above CP (Black *et al.* 2017). Black *et al.* (2017) showed an increase in neural drive coupled with a decrease in M-wave amplitude and M-wave area during severe intensity exercise. A decrease in muscle compound action potential with an increase in neural drive led to the conclusion that task failure was not caused by central fatigue in the severe intensity domain. The same results did not occur in the heavy intensity domain, suggesting more complex (and different) causes of fatigue in that domain.

The $\dot{V}O_2$, intramuscular metabolite, and neuromuscular data provide solid evidence of the adequacy of CP to demarcate the heavy intensity domain from the severe intensity domain. Although the intramuscular and neuromuscular data are based on intensities of ~10% below and above CP, they represented an intensity above MLSS that still showed a steady state in all markers other than blood lactate. Given the limitations of blood lactate outlined in the previous section, it can be assumed that an intensity above MLSS, maybe CP, is valid for MMSS. The $\dot{V}O_2$ data from exercising exactly at CP support that assumption. However, the lack of a blood lactate steady state may necessitate a revision of how CP has been defined. When given a physiological definition of CP, it has been repeatedly defined as the maximal intensity at which energy is supplied solely oxidatively (*i. e.*: Morton 2006; Poole *et al.* 2016; Jones and Vanhatalo 2017; Jones *et al.* 2019b). For that to be the case, lactate appearance in the blood and removal need to be at equilibrium. If not, part of the energy coming from glycolysis, produced nonoxidatively, is not being oxidised by other muscle tissue, the liver or the heart. This leads to a net excess of energy produced nonoxidatively. However, this seems not to affect the muscular milieu, given the steady state of PCr, pH, and muscle lactate. It does not affect the whole-body physiological state either, given the stability of $\dot{V}O_2$. Nevertheless, the steadiness in $\dot{V}O_2$ suggests that CP represents the upper limit of steady oxidative metabolism, even if part of the overall energy production comes from nonoxidative sources which are not reoxidised. Intensities above CP lead to an oxidative energy supply that fails to stabilise and keeps increasing until maximal values. Thus, physiologically, CP may represent the upper limit of steady oxidative energy supply, MMSS, and the demarcation point between the heavy and severe domains.

Given that CP appears to be a valid measure of MMSS, and that it is predominantly oxidative even if not wholly, it should be correlated with basic physiological factors that affect

oxidative capacity. The evidence is scarce, but there are strong correlations between CP and morphological factors that affect oxidative energy production. Two independent groups showed a correlation between type I (oxidative) fibres and CP ($r = 0.67$, $P = 0.025$; and $r = 0.79$, $P = 0.001$) (Vanhatalo *et al.* 2016; Mitchell *et al.* 2018). Mitchell *et al.* (2018) found an even stronger correlation when CP was compared to the number of capillary contacts with type I fibres ($r = 0.94$, $P < 0.001$). Even if the evidence is scarce, CP seems to have a strong correlation with key morphological attributes of oxidative energy production.

W′ has traditionally been linked to the anaerobic production of energy (a combination of energy produced via glycolysis and high-energy phosphate breakdown), hence the term "anaerobic work capacity" used initially (Poole *et al.* 2016). Since then it has been shown that W′ is correlated with the amplitude of the slow component, a parameter of the $\dot{V}O_2$ kinetics (Vanhatalo *et al.* 2011). The amplitude of the severe intensity domain will likely affect W′. If CP sets the lower limit of the severe intensity domain but it is at a submaximal $\dot{V}O_2$ (it can be as low as 80% (Poole *et al.* 1988)), part of W′ will be the energy produced by the difference between $\dot{V}O_2$ at CP and $\dot{V}O_{2max}$. However, it is also correlated with measures of anaerobic capacity (Nebelsick-Gullett *et al.* 1988), and it is affected by the availability of nonoxidative energy substrates (Miura *et al.* 1999; Miura *et al.* 2000). Muscle strength and size also affect W′ (Kordi, Menzies and Parker Simpson 2018). W′ is most likely affected by both nonoxidative energy production and energy production by the difference between $\dot{V}O_2$ at CP and $\dot{V}O_{2max}$. Given the contribution of both anaerobic and aerobic energy sources, the term W′ is favourable over AWC as it does not imply that the energy source is anaerobic.

Due to the aerobic basis of CP, it has been regarded as a more adequate performance index for endurance exercise than the LT and $\dot{V}O_{2max}$ (Craig *et al.* 2019). For that to be the case, CP should be correlated with performance in a similar or better way than LT. In fact, CP has been correlated and has been shown to predict cycling time-trial performance (Black *et al.* 2014). CP showed a greater correlation ($r = - 0.83$, $P < 0.01$) with 16.1-km time-trial performance than ramp incremental test peak power ($r = - 0.75$, $P < 0.05$), RCP ($r = - 0.68$, $P < 0.05$), and GET ($r = - 0.21$).

However, CP and CS have also been criticised and their ability to represent MMSS has been put in doubt (Gorostiaga, Sánchez-Medina and Garcia-Tabar 2021). The authors designed a study where they estimated CS from running race performances of elite athletes. A first analysis was done using data from the 10 best runners, males and females, who completed the 1500-m, 3000-m, 5000-m, and 10000-m races in the same season. For these athletes, they estimated two different CS, one using all the race times, and another one using all the race times but the 10000-m race time. When all race times were used for CS estimation, the mean CS was estimated at 5.19 $m \cdot s^{-1}$. When the 10000-m race time was excluded, the mean CS was estimated at 5.45 $m \cdot s^{-1}$. In both cases, CS was estimated at a similar percentage of the speed of the longest race used for CP estimation. A second analysis was done using world record races ranging from (1) 1000 to 5000-m, (2) 1000 to 10000-m, (3) 1000-m to the half marathon, and (4) 1000-m to the marathon race. CS for (1) was 6.38 $m \cdot s^{-1}$, 6.22 $m \cdot s^{-1}$ for (2), 6.05 $m \cdot s^{-1}$ for (3), and 5.77 $m \cdot s^{-1}$ for (4).

Given the difference in CS estimates when different trial lengths are used, Gorostiaga, Sánchez-Medina and Garcia-Tabar (2021) build the following argument against the use of

CP/CS for MMSS: (a) the definition of CP, an intensity that can be sustained for a very long time without fatigue, has never been demonstrated empirically and it has been shown that exhaustion occurs within 24-65 minutes (Hill 1993); (b) CP occurs at different relative intensities (near the first threshold (Moritani *et al.* 1981), near the second threshold (Poole *et al.* 1988), and above the second threshold (Mattioni Maturana *et al.* 2016)); (c) CP/CS changes depending on the duration of the trials used; given (a), (b), and (c), the CP/CS model is a purely mathematical artefact and is not a physiological feature. The argument can be divided into two main objections: (a) represents a conceptual objection, and (b) and (c) represent a methodological objection.

The conceptual objection is clear and is sound: Monod and Scherrer (1965) stated that CP was an intensity at which fatigue will not occur and they stated that it could be sustained for a very long time. As the authors explain, there are no data to support that claim and, in fact, there are data to suggest the opposite (Hill 1993). However, the original conception of CP has clearly evolved. CP is no longer thought to be an intensity at which fatigue does not occur, but an intensity at which a steady state is still possible and an intensity that demarcates severe from heavy exercise (Jones *et al.* 2019b). It is not an intensity at which fatigue does not occur, but an intensity that demarcates different types of fatigue (Black *et al.* 2017). In fact, proponents of CP currently do not maintain the original concept of CP (Hill 1993; Jones *et al.* 2010; Vanhatalo, Jones and Burnley 2011; Poole *et al.* 2016; Jones and Vanhatalo 2017; Burnley and Jones 2018). As data are gathered and the understanding of a phenomenon is increased, it is normal for the conceptual representation of that phenomenon to evolve. In the case of CP, the initial concept has evolved towards a concept that is based on the data collected since its origin. Given that it has been shown that fatigue occurs at CP and that it cannot be sustained for a very long time, the current concept of CP is not the original one. Even if the original concept of CP was not correct,

we do not need to disregard the phenomenon of CP itself, but change our understanding around it to create a concept that is consistent with the data. Thus, the fact that the original concept has been proven wrong does not necessarily lead to discrediting the CP phenomenon itself.

(b) and (c) can be seen as the same methodological objection as (b) is a product of (c): different lengths used to estimate CP/CS lead to different estimates, which will cause CP/CS to be at a different relative intensity. It is true that different trial lengths lead to different CP estimates, as shown by Gorostiaga, Sánchez-Medina and Garcia-Tabar (2021) and by Mattioni Maturana *et al.* (2018). However, the second analysis by Gorostiaga, Sánchez-Medina and Garcia-Tabar using world records is not completely adequate, as they are using data from different athletes, the athletes setting each world record, to calculate a single CS. Although CS and CP apply across people and species, they are attributes of an individual, not a cluster of individuals. Thus, an assessment of a single CS should be done for a given individual, as Gorostiaga, Sánchez-Medina and Garcia-Tabar did in their first analysis, but not different people as they did in the second analysis. However, their point still stands given the data of their first analysis and the data from Mattioni Maturana *et al.* (2018). The differences across CS estimates when different lengths are used leads Gorostiaga, Sánchez-Medina and Garcia-Tabar to conclude that the choice of trial length for CS estimation is arbitrary. However, the opposite can be argued. When the length of the trial is carefully chosen, CP has repeatedly shown to differentiate intensity domains and steady state exercise from nonsteady state exercise (Poole *et al.* 1988; Burnley, Vanhatalo and Jones 2012; Vanhatalo *et al.* 2016; Black *et al.* 2017; Nixon *et al.* 2021). The choice of trials ranging from 2 min to 15 min is done so that the CP estimate represents MMSS and not some other physiological phenomenon. That is not an arbitrary choice, but a tested and validated choice to apply CP for MMSS estimation.

It is true that CP is a mathematical model, and without any context or standardised methodology, it is a purely mathematical artefact. However, that does not mean that it cannot represent a given physiological phenomenon: MMSS. In fact, it does it adequately, as shown previously, when its application is limited to its domain of validity, *i.e.*, the severe intensity domain. The evolution of the concept associated with CP, and the standardisation of the methodology enables the use of the mathematical model as a physiological tool. Thus, the evolution of the concept and the intentional choice of trial length is not something to be criticised, but something to be approved as it enables physiological assessments that would not be possible without the CP model.

The differences in physiological, intramuscular, and neuromuscular responses below and above CP support the use of CP as an estimator of MMSS over other indicators of the second threshold. Its oxidative physiological foundations and its ability to predict performance make it a key parameter for the assessment of endurance ability and changes in performance throughout a season or over different seasons. However, its measurement requires of methodological considerations so that the estimations fulfil their purpose: identification of MMSS and prediction of performance in the severe intensity domain.

2.3.3 Methodological considerations for CP testing

The estimation of CP and W′ by maximal trials in the severe intensity domain is mainly affected by three factors: length of the trials, number of trials, and choice of the model.

There is a consensus on what the length of the trials should be or what the range of durations should be. Trials lasting 2 to 15 minutes have been regarded as adequate for CP estimation, the shortest and longest being at least 5 minutes apart (Hill 1993; Vanhatalo, Jones and Burnley 2011; Jones and Vanhatalo 2017; Jones *et al.* 2019b). Durations shorter than 2 and longer than 15 minutes may impair the full depletion of W′ and $\dot{V}O_{2max}$ may not be achieved. Thus, a broad range of durations between 2 minutes and 15 minutes will give the best hyperbolic fit.

The number of trials may differ depending on the aim of the testing. A minimum of 3 and up to 5 trials are used (Vanhatalo, Jones and Burnley 2011). Although 2 trials are sufficient to establish CP and W′ any small variation in one of the two trials will have a greater effect on the estimations given the lack of degrees of freedom. Adding a third trial adds a degree of freedom, provides confidence intervals, and, most importantly in practical applications, reduces the reliance on any single trial. However, the confidence intervals with three trials are wide, and adding a fourth trial reduces the confidence interval significantly, while a fifth trial reduces it further. For example, if a cyclist has done three maximal trials lasting 3, 6, and 12 minutes and produced 412, 375, and 348 W, respectively, the CP estimate will be 326 W and the 95% confidence interval will be 277 W to 379 W. The addition of a 10-minute trial, with a produced power output of 353 W, results in a CP of 326 W and a 95% confidence interval of 314 W to 338 W, much smaller than the confidence interval of the estimation based on three trials. The additional trial provides greater confidence in the estimates but is more time demanding. Karsten *et al.* (2017) have shown that a single-day CP testing protocol with 30-minute recoveries in between has proven to be a valid way to estimate CP. This provides a practical benefit over doing 4 or more trials, as it allows estimating CP in a single day. However, it will lead to a wider

than desired confidence interval. Therefore, there is a trade-off between the required time and the confidence of the estimate, and different settings will favour one or the other. In a practical setting, in which two or more days of testing can interrupt an entire week of training, the single-day protocol will most likely be favoured. However, in certain research settings, the tighter confidence interval provided by 4 or 5 trials will be favoured.

Besides the number of trials and duration, the fixed parameter of the trial can also be different. The fixed parameter can be either time or power (Karsten *et al.* 2018). When time is the fixed parameter, a time-trial is completed aiming to achieve the highest mean power throughout the fixed time period. When power is fixed, sustaining the power for the longest period of time possible is the goal. Both methods provide similar results (Karsten *et al.* 2018). The benefit of using time as a fixed parameter is that it is more time-efficient, and trial end-point is known meaning it is not as impacted by participant levels of motivation and decision making over trial termination. To set the power of the time to task failure tests, a previous ramp test is needed. The maximal power of the test is used as a reference to then do time to task failure tests at percentages of it. In the field, time is the fixed parameter due to the need of using an ergometer to maintain power constant throughout a trial. The results obtained in the field are applicable to the laboratory, and *vice versa* (Karsten *et al.* 2013).

Finally, the choice of the model also affects CP and W′ determination. The 5 models, as mentioned above, are as follows:

(1) CP_{linear}: $W_{lim} = W' + CP \cdot t_{lim}$

(2) $CP_{1/time}$: $P = W' \cdot \frac{1}{t_{lim}} + CP$

(3) $CP_{2\text{-}hyp}$: $t_{lim} = \frac{w'}{P-CP}$

(4) $CP_{3\text{-}hyp}$: $t_{lim} = \frac{W'}{P-CP} - \frac{W'}{P_{max}-CP}$

(5) CP_{exp}: $P = CP + (P_{max} - CP)\, e^{-\frac{t_{lim}}{\tau}}$

(1) is the linear model, CP_{linear}; (2) is the inverse of time linear model, $CP_{1/time}$; (3) is the 2-parameter hyperbolic model, $CP_{2\text{-}hyp}$; (4) is the 3-parameter hyperbolic model, $CP_{3\text{-}hyp}$; and (5) is the exponential model, CP_{exp}.

CP_{linear}, $CP_{1/time}$, and $CP_{2\text{-}hyp}$ are mathematically equivalent models. Equation (1), can also be:

$$W_{lim} = P \cdot t_{lim}$$

given that total work is the multiplication of power and time, equation (1) and the equation above are equivalent. By replacing W_{lim} in one of the two equations with the equal to W_{lim} of the other equation, the following equation is achieved:

$$P \cdot t_{lim} = W' + CP \cdot t_{lim}$$

next, when divided by t_{lim}, equation (2) is achieved:

$$P = W' \cdot \frac{1}{t_{lim}} + CP$$

In equation (1), CP is the slope and W′ is the *y* axis intercept, while in (2) CP is the *y* axis intercept and W′ is the slope. When CP is subtracted:

$$P - CP = W' \cdot \frac{1}{t_{lim}}$$

then, when multiplying by t_{lim}:

$$(P - CP) \cdot t_{lim} = W'$$

when divided by P – CP, equation (3) is achieved:

$$t_{lim} = \frac{w'}{P - CP}$$

$CP_{3\text{-}hyp}$ adds P_{max} to avoid the vertical asymptote when duration tends to zero, and CP_{exp} does not provide a W′ estimate and instead of being a hyperbolic relation between power and duration, it is an exponential relation.

The graphical representation of the five models can be seen in Figure 2.14 which have been fitted with the same data set obtained from three maximal trials. It can be seen how the same data can lead to different CP and W′ estimates, depending on the model used, despite the mathematical equivalence of some of the models.

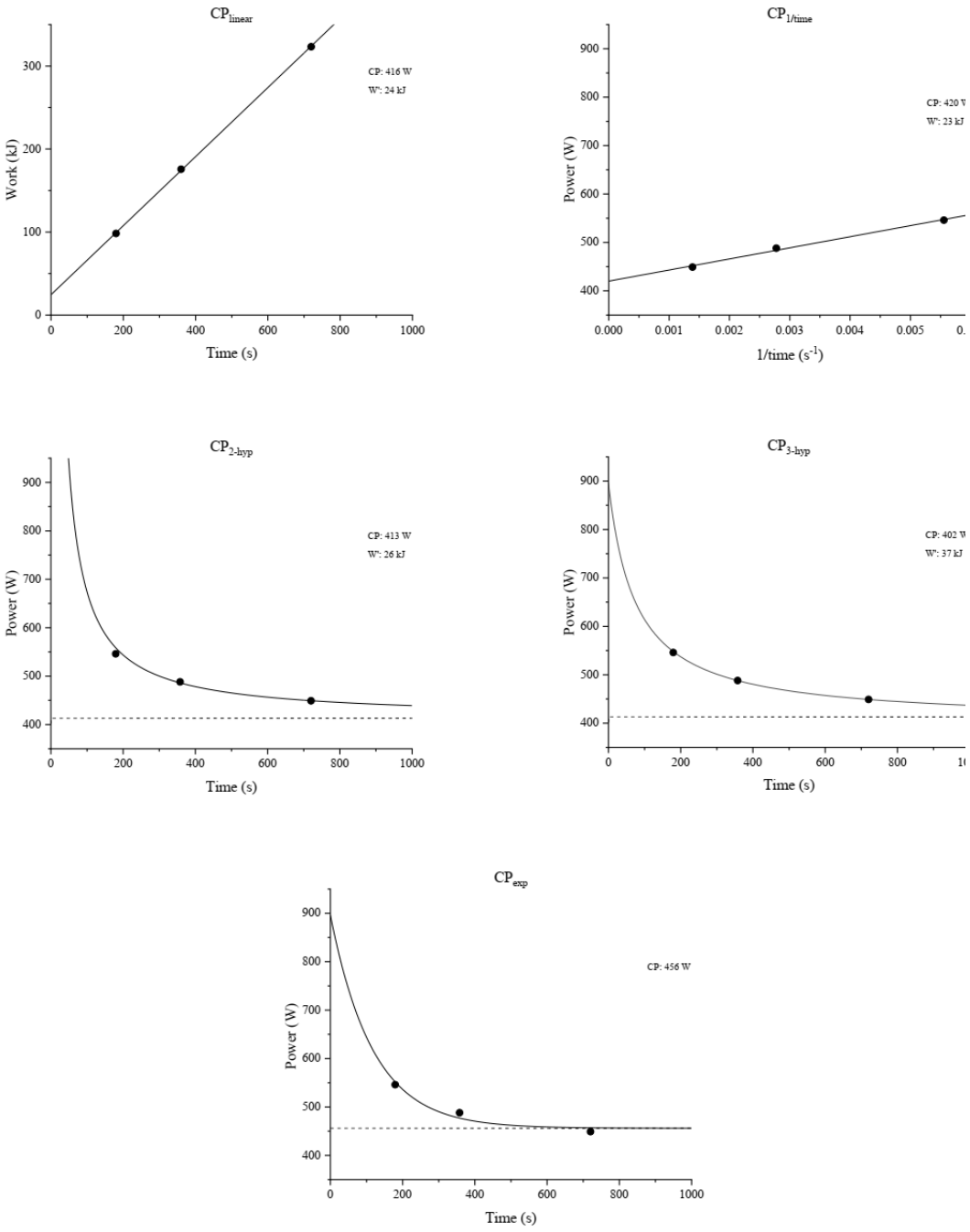

Figure 2.14: CP_{linear} represented in the top left corner. The y axis intercept is W′ and the slope is CP. $CP_{1/time}$ is represented in the top right corner. CP is the y axis intercept and W′ is the slope. $CP_{2\text{-}hyp}$ and $CP_{3\text{-}hyp}$ are represented in the middle, CP being the horizontal power asymptote (dashed line) and W′ the curvature constant. CP_{exp} is represented at the bottom, the dashed line being CP. The black filled circles are the data points obtained from time-trials.

The same phenomena have been observed in research, where the CP and W′ estimates from different models have differed despite using the same data for each model (Gaesser *et al.* 1995; Bull *et al.* 2000; Bergstrom *et al.* 2014; Mattioni Maturana *et al.* 2018). The first of such studies comparing the estimation of CP and W′ by Gaesser *et al.* (1995) fitted the model with 5-7 time to task failure tests based on power outputs relative to the maximal power output achieved in an incremental test. The time to task failure tests fell between 1 and 20 minutes. CP_{exp} gave the highest mean CP estimate (242 W), followed by $CP_{1/time}$ (237 W), CP_{linear} (224 W), $CP_{2\text{-}hyp}$ (215 W), and $CP_{3\text{-}hyp}$ (195 W). This shows a wide range of values that would affect training prescription and proper demarcation of intensity domains. Later data from Bull *et al.* (2000) showed similar results (CP_{exp} 212 W, $CP_{1/time}$ 208 W, CP_{linear} 196 W, $CP_{2\text{-}hyp}$ 192 W, and $CP_{3\text{-}hyp}$ 180 W). The models were fitted using data from time to task failure tests, set at power outputs relative to the maximal power output of an incremental test, and ranged from ~1 to, at least, to 10 minutes. Bergstrom *et al.* (2014) also fitted the models to data from task to time failure tests. The intensity was based on the maximal power of an incremental test and the difference between GET and the maximal power. The duration of the time to task failure tests was between ~3 and 15 minutes. The differences between models were smaller than from previous studies (CP_{exp} 198 W, $CP_{1/time}$ 184 W, CP_{linear} 181 W, $CP_{2\text{-}hyp}$ 176 W, and $CP_{3\text{-}hyp}$ 174 W). Mattioni Maturana *et al.* (2018) did a more exhaustive analysis, including the differences between models, number of trials, and duration of trials. In the table below (Table 2.3) a comparison of the obtained CP and W′ can be seen. The numbers 1 to 5 denote the different trials used to estimate CP and W′. Trial 1 is performed at 110% of the peak power of an incremental test. Trial 2 is performed at 95%, 3 at 80%, 4 at 75%, and 5 at 72%. The mean times to task failure were as follows: 1 was 1.7 min, 2 was 3.2 min, 3 was 7.1 min, 4 was 12.5 min, and 5 was 19.4 min. The differences between

models when using 5 trials were smaller than in previous studies, with a difference of 3% between $CP_{3\text{-}hyp}$ and $CP_{1/time}$, as opposed to 6-22% found in other studies (Gaesser *et al.* 1995; Bull *et al.* 2000; Bergstrom *et al.* 2014). CP_{exp} remained higher than the other models no matter what the number of trials used was. Similar results have been found for CS (Housh *et al.* 2001)

Table 2.3: CP parameter estimates for different models using different combinations of time to exhaustion trials. Numbers on the top first and second row identify the number of trials and their corresponding durations, respectively (Mattioni Maturana *et al.* 2018)

	5	4		3				2			
		1,2,3,4	2,3,4,5	1,2,3	1,3,5	2,3,4	3,4,5	1,2	1,5	3,4	4,5
CP_{exp}											
CP	275 ± 47	281 ± 47	270 ± 46								
SEE	5.7 ± 2.1	8.0 ± 2.8	2.9 ± 1.9								
$CP_{3\text{-}hyp}$											
CP	252 ± 44	250 ± 41	250 ± 43								
SEE	3.4 ± 1.8	5.9 ± 5.8	4.5 ± 4.6								
W'	23.1 ± 7.6	25.8 ± 12.6	24.9 ± 10.0								
SEE	3.7 ± 2.0	5.9 ± 6.3	6.3 ± 7.3								
$CP_{2\text{-}hyp}$											
CP	253 ± 44	256 ± 42	253 ± 44	263 ± 43	254 ± 43	256 ± 42	252 ± 44				
SEE	1.6 ± 1.1	2.8 ± 2.2	2.3 ± 1.7	5.0 ± 6.9	1.6 ± 1.5	3.4 ± 3.2	2.6 ± 2.6				
W'	20.3 ± 5.9	18.7 ± 6.7	20.1 ± 6.0	16.4 ± 5.7	19.8 ± 7.0	19.2 ± 7.1	21.2 ± 6.5				
SEE	1.7 ± 1.0	1.9 ± 1.4	2.2 ± 1.5	1.6 ± 2.0	1.8 ± 1.5	2.3 ± 2.1	2.6 ± 2.3				
CP_{linear}											
CP	256 ± 45	259 ± 44	255 ± 45	265 ± 47	256 ± 45	256 ± 45	253 ± 44	272 ± 50	257 ± 45	252 ± 46	251 ± 44
SEE	2.3 ± 1.1	3.9 ± 2.7	2.4 ± 1.4	6.1 ± 6.5	3.1 ± 2.5	4.2 ± 3.5	2.4 ± 2.1				
W'	17.9 ± 5.7	17.1 ± 5.7	19.2 ± 5.8	15.9 ± 5.7	17.7 ± 5.9	18.7 ± 5.9	21.0 ± 7.1	14.8 ± 6.5	16.3 ± 5.9	21.7 ± 8.8	22.3 ± 7.6
SEE	1.5 ± 0.8	1.7 ± 1.1	1.8 ± 1.0	1.6 ± 1.5	2.4 ± 2.0	2.1 ± 1.8	2.0 ± 1.7				
$CP_{1/time}$											
CP	261 ± 45	263 ± 45	256 ± 45	268 ± 47	260 ± 45	257 ± 45	253 ± 45	272 ± 50	257 ± 45	252 ± 46	251 ± 44
SEE	4.4 ± 2.4	5.8 ± 3.4	3.1 ± 1.9	7.9 ± 5.6	4.6 ± 3.5	4.4 ± 3.6	2.4 ± 1.8				
W'	16.1 ± 6.0	15.8 ± 5.9	18.4 ± 5.6	15.2 ± 6.0	16.0 ± 5.9	18.2 ± 5.7	21.1 ± 7.6	14.8 ± 6.5	16.3 ± 5.9	21.7 ± 8.8	22.3 ± 7.6
SEE	0.8 ± 0.4	1.0 ± 0.6	1.0 ± 0.6	1.1 ± 0.8	0.8 ± 0.6	1.3 ± 1.0	1.5 ± 1.2				

The discrepancy between models suggests a limitation of the CP model. If CP is assumed to be the gold-standard for MMSS, but if its estimations can vary up to 22% excluding the exponential model, which model is the most adequate for estimating MMSS?

Several suggestions of which model is most adequate have been made. Early in the 2000s, Billat *et al.* (2003) recommended the use of $CP_{3\text{-}hyp}$, because it was closer than $CP_{2\text{-}hyp}$ to MLSS. However, that recommendation relies on the validity of MLSS to reflect MMSS, which is far from clear as explained above. Other researchers, particularly Jones and colleagues, the advocates of CP as the MMSS, have proposed to use the model with the best fit (Jones *et al.* 2019b). This, although intuitive, has no empirical evidence, and, given the necessity of empirical evidence to verify any physiological aspects of CP, more evidence is needed to justify the use of one model over the other. Given the reliability of estimates despite changes in trial duration, Mattioni Maturana *et al.* (2018) recommend the use of $CP_{3\text{-}hyp}$, or $CP_{2\text{-}hyp}$ when a trial longer than 10 minutes is used. Neither reasons given by Billat *et al.* (2003), Jones *et al.* (2019b), or Mattioni Maturana *et al.* (2018) are sufficient to justify the choice of any model to calculate CP for MMSS estimation due to the necessity to give empirical evidence to support any physiological claim around the CP model.

Lastly, participants need to be motivated and willing to do the maximal effort possible in every single trial. A lack of will or motivation during all trials will underestimate CP and W′ due to its effort-dependent nature. Unlike all the lactate-based and gas exchange-based thresholds that we have seen in the previous section, CP relies on the maximal effort of every trial for proper assessment. Even worse than a lack of will for all the trials can be a lack of will for some of the trials and not others. For example, if a cyclist is setting his CP by doing time-fixed time-trials, lasting 12-, 6-, and 3-minutes long, a strong will in the 12-minute trial and a lack of motivation in the 3-minute trial will cause an overestimation of CP, leading to setting training intensity zones wrong, and training too hard when intending to do threshold training. All other trials equal, a decrease in power of 10% in the 3-minute trial would lead to CP being 4% higher,

or 338 W in the case of the cyclist with a CP of 326 W mentioned earlier. In the opposite scenario, a strong will in the 3-minute trial and a lack of motivation in the 12-minute trial will lead to an underestimation of CP, leading to training easier than expected, and not hitting high enough intensities when doing high-intensity interval training. All other trials equal, a decrease in power of 10% in the 12-minute trial would lead to CP being 11% lower, or 289 W in the case of that same cyclist. A review on the effects of psychology on endurance performance showed that mental fatigue is likely to decrease endurance performance, and that the use of self-talk, imagery, and goal setting is beneficial for endurance performance (McCormick, Meijen and Marcora 2015). Thus, motivation and a strong will in the day or days of testing, evenly distributing that will, the use of psychological strategies that improve performance, and the avoidance of mental fatigue in testing days are necessary for an accurate estimation of CP.

To appropriately determine CP, performing three to five trials lasting 2 to 15 minutes, and making sure that there is no mental fatigue and that effective psychological strategies are implemented is needed (Salam, Marcora and Hopker 2018). The choice of the model remains an open question that needs to be answered either by measuring individually whether exercise at the CP estimate of a certain model causes a steady state, or by conducting research analysing the effects of the different models on physiological parameters.

2.4 Aim of the thesis and hypothesis

CP has been shown to be near the transition from the heavy intensity domain to the severe intensity domain and may as such be considered as a good indicator of MMSS. However, there has been no standardisation on which modes should be used for CP estimation, probably due to a lack of data comparing the mathematical models. Therefore, the aim of this thesis is to analyse the $\dot{V}O_2$, blood lactate, and perceptual responses when exercising at the estimated CP by CP_{linear}, $CP_{1/time}$, $CP_{2\text{-}hyp}$, $CP_{3\text{-}hyp}$, and CP_{exp}, and identify which of them, if any, provides the best estimate of MMSS.

$H1_0$: There will be no difference in the $\dot{V}O_2$, blood lactate, and perceptual responses between the different models used to estimate CP.

$H1_1$: There will be differences in the $\dot{V}O_2$, blood lactate, and perceptual responses between the different models used to estimate CP.

$H2_0$: There will be no differences between models in their ability to estimate MMSS.

$H2_1$: There will be differences between models in their ability to estimate MMSS.

3. Methods

3.1 Participants

Twelve healthy participants (mean (± SD): age: 31 ± 11 years, height: 1.78 ± 0.03 m, body mass: 70.5 ± 5.6 kg, $\dot{V}O_{2peak}$: 4.10 ± 0.70 L · min^{-1}, relative $\dot{V}O_{2peak}$: 58 ± 8 mL · kg^{-1} · min^{-1}) were recruited for the study. The participants were trained cyclists, with at least two years of cycling training experience and trained three times per week or more at the time of recruitment. One participant was excluded from the data analysis due to improper completion of time-trials to estimate CP. All participants provided informed consent and the study was approved by the University of Kent School of Sport and Exercise Sciences Research Ethics Advisory Group (Prop 07_2020_21) and was conducted in alignment with the declaration of Helsinki.

3.2 Study design

Participants visited the laboratory on four occasions separated by a minimum of 48 h. Each visit was done at the same time of the day (± 1 h). The participants avoided heavy exercise in the 48 h prior to the visits, alcohol, and caffeine in the 24 h prior to the visit, and were advised to eat and drink as they would approaching an important race or event. During the first visit, participants completed fixed duration exercise trials of 12, 6 and 3 minutes in order to estimate CP (see *Estimation of Critical Power* below). During visits 2, 3, and 4 participants completed steady state trials at CP estimated from the five mathematical models (see *Determination of Physiological*

Responses below). All trials were conducted on a Cyclus 2 ergometer (RBM elektronik-automation GmbH, Leipzig, Germany). The participants' own bikes were fitted to the ergometer which was pre-programmed to run the exercise protocols. Power output data were recorded continuously throughout the exercising portions of the visits within the Cyclus 2 ergometer. Heart rate was also recorded continuously throughout all trials (Polar H10, Polar Electro Oy, Kempele, Finland). Expired gases were measured continuously throughout all exercise trials on a breath-by-breath basis using a metabolic cart (Metalyzer 3B; CORTEX Biophysik GmbH, Leipzig, Germany). Prior to all testing, the Cortex analyser was calibrated using a two-point gas calibration method using ambient air and known concentrations of O_2 (17%) and carbon dioxide (CO_2; 5%). The flow sensor and turbine were calibrated using a 3-litre syringe (Hans Rudolph Inc. Kansas, USA).

Estimation of Critical Power

During the first visit, the participants' height and body mass (seca GmbH & Co. KG., Hamburg, Germany) were measured. Participants subsequently warmed up for 15 minutes at 150 W. After the warm-up, the participants completed a 6-s all-out sprint to obtain their maximal power. After the sprint, the participants completed 5 minutes of active recovery prior to undertaking the maximal trials for CP estimation. The maximal trials were duration-clamped, and the participants were asked to self-pace the time-trial to achieve the maximal mean power for the duration of the trial. The participants received strong verbal encouragement during the time-trials. The time-trials were 12-minute, 6-minute, and 3-minute long, and there was a 30-minute recovery in

between trials (Karsten *et al*. 2017). The first 10 minutes of the recovery were done actively on the bike, the middle 10 minutes were passive rest sited, and the last 10 minutes were active recovery on the bike again before starting the next trial. Finally, the participants cooled down for 10 minutes on the bike after the completion of the last trial. This single-day protocol has shown to be valid for CP estimation (Karsten *et al.* 2017). Gas exchange was measured breath by breath 5 minutes before and during the time-trials. Blood lactate was measured (Biosen C-line, EKF diagnostics, Cardiff, United Kingdom) immediately before the start of the time-trial and immediately after the completion of the time-trial. Heart rate was measured continuously throughout the whole exercise protocol. RPE using the Borg Scale (Borg 1982) was measured at the end of each time-trial. The acceptance criteria for the CP determination trials were less than a 5% inter-trial variance in $\dot{V}O_{2peak}$, even pacing without major changes in power (by visual inspection), a peak RER of >1.05, and no significant inter-trial variance in peak RER and blood lactate concentration (Midgley *et al.* 2009). A diagram of the protocol is shown in Figure 3.1.

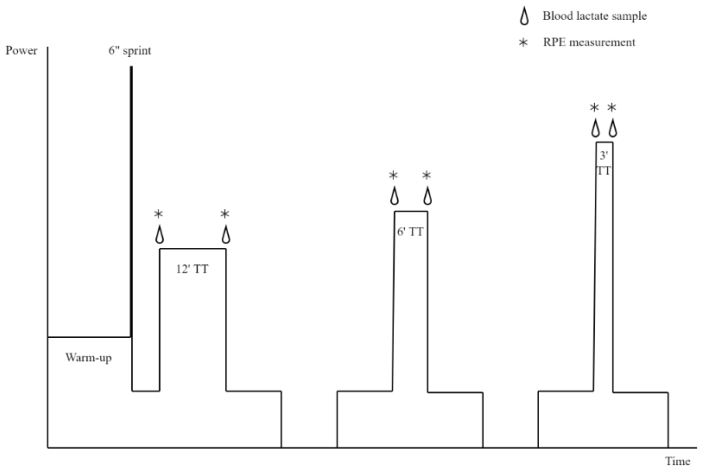

Figure 3.1: Exercise protocol of visit 1. A 15-min warm-up was followed by a 6-s sprint, a 5-min recovery, a 12-min TT, a 30-min recovery, a 6-min TT, a 30-min recovery, a 3-min TT, and a 10-min cool-down. Lactate samples were taken and RPE was measured before the start and after the end of each TT.

From the data obtained in the first visit, CP and W′ were calculated using the following 5 formulas:

CP_{linear}: $W_{lim} = W' + CP \cdot t_{lim}$

$CP_{1/time}$: $P = W' \cdot \frac{1}{t_{lim}} + CP$

$CP_{2\text{-}hyp}$: $t_{lim} = \frac{w'}{P-CP}$

$CP_{3\text{-}hyp}$: $t_{lim} = \frac{W'}{P-CP} - \frac{W'}{P_{max}-CP}$

CP_{exp}: $P = CP + (P_{max} - CP)\, e^{-\frac{t_{lim}}{\tau}}$

CP_{linear} and $CP_{1/time}$ were fitted using the data from the three time-trials by linear regression. $CP_{2\text{-}hyp}$ was fitted using the data from the three time-trials by nonlinear regression, minimising the sum of the squared residuals. For $CP_{3\text{-}hyp}$ and CP_{exp}, P_{max} was set empirically from the 6-s sprint data instead of obtained from fitting the model, as it would need an additional fourth visit to obtain the three parameters; measuring P_{max} instead of obtaining it from model fitting is more desirable (Vinetti *et al.* 2019). Then, from the P_{max} and time-trial data, the models were fitted by nonlinear regression, minimising the sum of the squared residuals.

The peak $\dot{V}O_2$ attained during the maximal trials to estimate CP was defined as the participant's $\dot{V}O_{2peak}$ and was subsequently used for the data analysis, as the peak $\dot{V}O_2$ attained during maximal trials in the severe domain lead to $\dot{V}O_{2max}$ (Hill and Ferguson 1999).

Determination of physiological responses at CP estimates

During visits 2, 3, and 4, the participants started by warming up for 15 minutes at 150 W. They completed a 30-minute trial, or until exhaustion, at each of the CP estimates (5 trials in total) in random order. After the warm-up, the participants cycled for 30 minutes at the estimated CP by one of the models. During visits 2 and 3, the participants rested for 30 minutes prior to completing another 30-minute bout at the estimated CP from one of the remaining models (Figure 3.2). By the end of visit 3, the participants completed trials at 4 of the 5 CP estimates. Thus, during visit 4, the participants cycled only once at the remaining CP estimate (Figure 3.3). The participants cooled down for 10 minutes after the completion of the last 30-minute bout at CP of the visit. Gas exchange was measured breath by breath 5 minutes before and during the 30-minute trials at CP. Blood lactate samples were taken every 5 minutes after the start of the 30-minute CP trial until the end of the trial. If the trial lasted less than 30 minutes, a blood lactate sample was taken upon exhaustion. HR was measured continuously throughout the exercise trial. RPE was measured every 5 minutes after the start of the 30-minute CP trial until the end of the trial. If the trial lasted less than 30 minutes, an RPE sample was taken upon exhaustion.

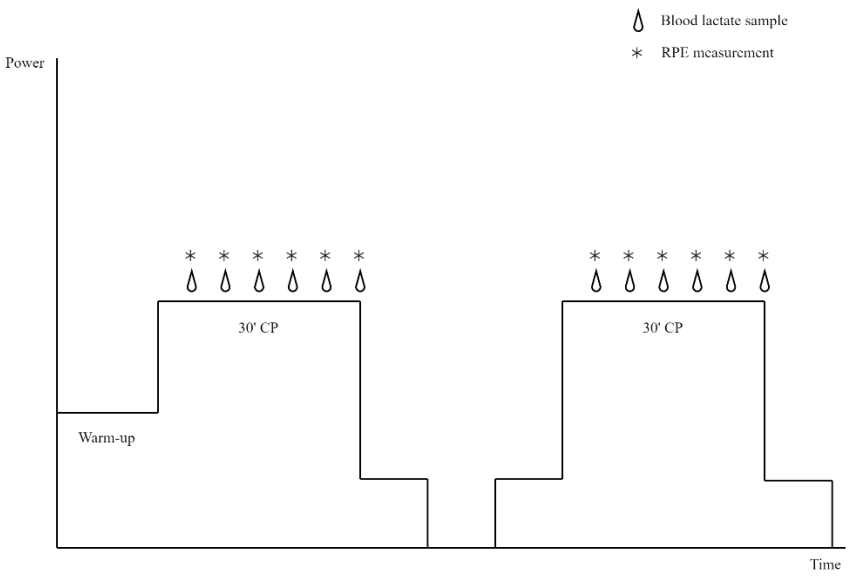

Figure 3.2: Exercise protocol of visits 2 and 3. A 15-min warm-up was followed by 30 min at one of the CP estimates, a 30-min recovery, 30 min at one of the remaining CP estimates, and a 10-min cool-down. Lactate samples were taken and RPE was measured every 5 min after the start of the 30-min CP trials.

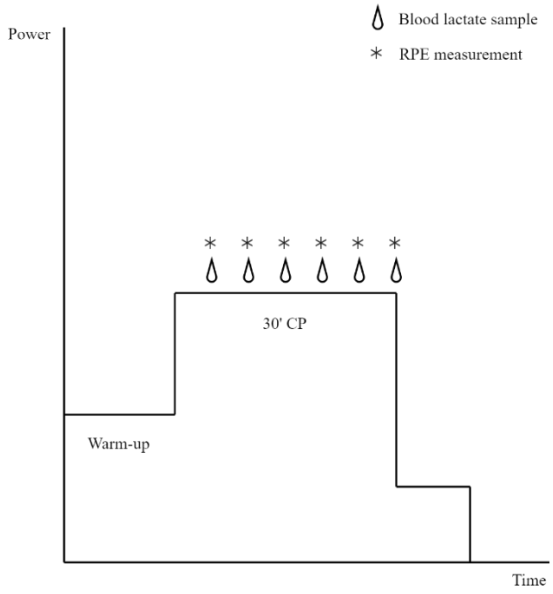

Figure 3.3: Exercise protocol of visit 4. A 15-min warm-up was followed by 30 min at the remaining CP estimate, and a 10-min cool-down. Lactate samples were taken and RPE was measured every 5 min after the start of the 30-min CP trial.

3.3 Statistical methods

Prior to statistical analysis, visual inspection of Q-Q plots and Shapiro-Wilks's statistics were used to check whether data were normally distributed. One-way repeated measures ANOVA was performed to assess differences between peak values of $\dot{V}O_2$ attained during the CP determination trials and the end $\dot{V}O_2$ from exercise at each CP model estimate; the time at which fatigue occurred prior to target duration from exercise at each CP model estimate; end RPE from exercise at each CP model estimate. Two-way repeated measures ANOVA across model and time (5 models x 6 timepoints) was performed to assess differences between $\dot{V}O_2$, blood lactate, and RPE. The criterion of $P < 0.05$ was used for the detection of significance in all cases. The point of $\dot{V}O_2$ stabilisation was visually determined and linear regression was applied to the subsequent $\dot{V}O_2$ datapoints to verify that the slope of $\dot{V}O_2$ was not different to zero. Pairwise comparisons were conducted using Bonferroni adjustments where main effects and interactions were significant. Data are presented as individual values or mean ± SD (unless specified otherwise). Statistical analyses were conducted using IBM SPSS Statistics 28 (IBM, Armonk, New York, USA).

4. Results

4.1 CP and W′ estimates

CP was estimated at 270 ± 64 W using CP_{linear}, 272 ± 66 W using $CP_{1/time}$, 266 ± 65 W using $CP_{2\text{-}hyp}$, 262 ± 63 W using $CP_{3\text{-}hyp}$, and 303 ± 69 W using CP_{exp}. W′ was estimated at 19.0 ± 1.9 kJ using CP_{linear}, 17.9 ± 1.7 kJ using $CP_{1/time}$, 20.3 ± 2.1 kJ using $CP_{2\text{-}hyp}$, and 24.7 ± 2.3 kJ using $CP_{3\text{-}hyp}$ (see Table 4.1). CP estimates from CP_{linear} and $CP_{1/time}$ were not significantly different ($P = 0.384$). All models were significantly lower than CP_{exp} ($P < 0.001$). CP_{linear} was significantly higher than $CP_{2\text{-}hyp}$ ($P = 0.011$) and $CP_{3\text{-}hyp}$ ($P < 0.001$). $CP_{1/time}$ was significantly higher than $CP_{2\text{-}hyp}$ ($P < 0.001$) and $CP_{3\text{-}hyp}$ ($P < 0.001$). $CP_{2\text{-}hyp}$ was significantly higher than $CP_{3\text{-}hyp}$ ($P < 0.001$). For 1 subject, $CP_{1/time}$ was the best-fitting model. For the other 10 subjects, $CP_{3\text{-}hyp}$ was the best fitting model.

Table 4.1**:** CP and standard error of the estimate expressed as %, W′ and the standard error of the estimate expressed as %, and the total error of the model

	CP (W)	**CoV (%)**	**W′ (kJ)**	**CoV (%)**	**Total error (%)**
CP_{linear}	270 ± 64	1.6 ± 1.2	19.0 ± 1.9	10.11 ± 6.2	11.71 ± 7.4
$CP_{1/time}$	272 ± 66	2.1 ± 1.6	17.9 ± 1.7	8.4 ± 5.5	10.5 ± 7.1
$CP_{2\text{-}hyp}$	266 ± 65	1.2 ± 0.9	20.3 ± 2.1	9.5 ± 5.6	10.7 ± 6.5
$CP_{3\text{-}hyp}$	262 ± 63	1.0 ± 1.0	24.7 ± 2.3	7.2 ± 5.6	8.2 ± 6.6
CP_{exp}	303 ± 69	4.4 ± 1.2	–	–	–

4.2 $\dot{V}O_2$ response to exercising at the different CP estimates

$\dot{V}O_2$ reached a plateau for the five models (see Figure 4.1). There were no significant increases in $\dot{V}O_2$ after stabilisation for any of the models (P = 1.000). While exercising at the CP estimate of CP_{exp}, the plateau was reached significantly faster than during all other conditions (P < 0.001 – P = 0.010) but not $CP_{3\text{-}hyp}$ (P = 0.122). The $\dot{V}O_2$ at which a plateau was achieved was not significantly different to the subject's $\dot{V}O_{2peak}$, attained during the maximal trials for CP estimation, for CP_{exp} (P = 1.000) and $CP_{1/time}$ (P = 0.130). The $\dot{V}O_2$ at which a plateau was achieved was significantly lower than the subject's $\dot{V}O_{2peak}$ for CP_{linear} (P = 0.041), $CP_{2\text{-}hyp}$ (P < 0.001), and $CP_{3\text{-}hyp}$ (P < 0.001). $\dot{V}O_2$ relative to $\dot{V}O_{2peak}$ was 94 ± 5% for CP_{linear}, 94 ± 6% for $CP_{1/time}$, 87 ± 4% for $CP_{2\text{-}hyp}$, 86 ± 4% for $CP_{3\text{-}hyp}$, and 98 ± 2% for CP_{exp} (Table 4.2).

Table 4.2**:** Time for $\dot{V}O_2$ to stabilise, percentage of $\dot{V}O_{2peak}$ at which $\dot{V}O_2$ stabilises, and point of fatigue for each model

	VO_2 stabilisation time (min)	**Percentage of VO_{2peak} (%)**	**Point of fatigue (min)**
CP_{linear}	14.1 ± 4.0	94 ± 5[a]	25.7 ± 3.8
$CP_{1/time}$	13.0 ± 3.3	94 ± 6	23.1 ± 4.7[b]
$CP_{2\text{-}hyp}$	11.7 ± 2.6	87 ± 4[a]	29.5 ±1.5
$CP_{3\text{-}hyp}$	11.1 ± 4.2	86 ± 4[a]	30.0 ± 0.0
CP_{exp}	6.2 ± 1.8	98 ± 2	9.8 ± 2.6[b]

[a] Significantly lower than $\dot{V}O_{2peak}$. [b] Significantly shorter than 30 minutes.

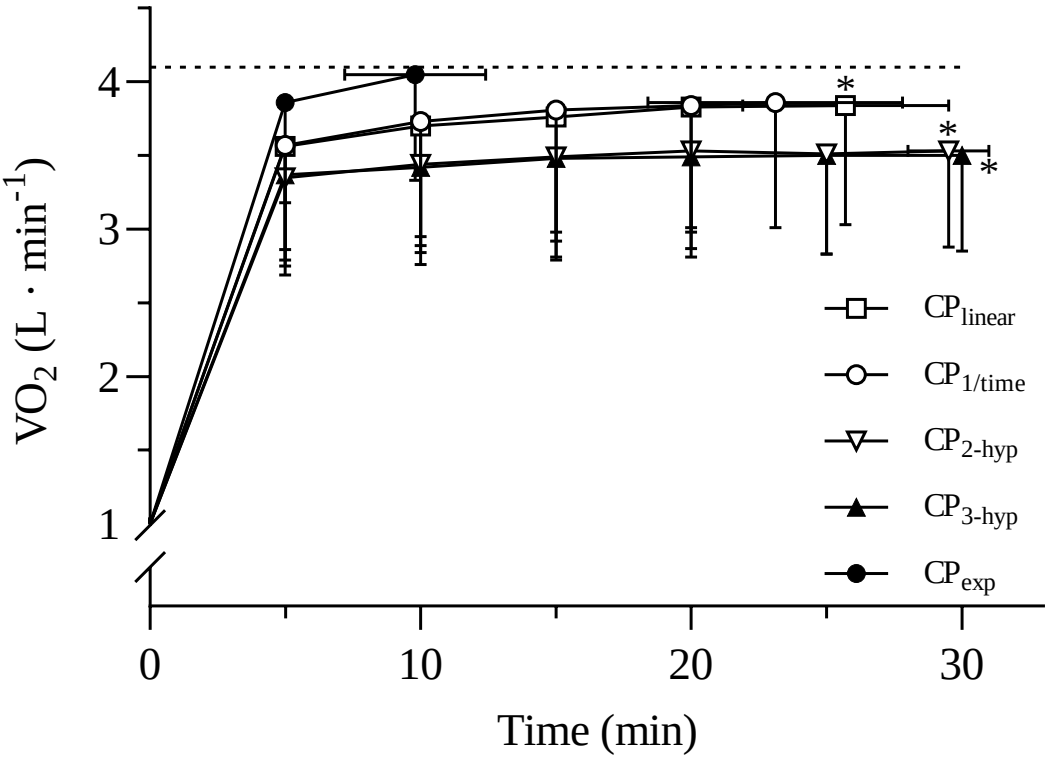

Figure 4.1: $\dot{V}O_2$ response to exercise at CP_{linear}, $CP_{1/time}$, $CP_{2\text{-}hyp}$, $CP_{3\text{-}hyp}$, and CP_{exp}. $\dot{V}O_2$ did not change significantly after stabilisation. The dashed line represents $\dot{V}O_{2peak}$. *End-exercise $\dot{V}O_2$ significantly lower than $\dot{V}O_{2peak}$.

4.3 Lactate response to exercising at the different CP estimates

Blood lactate increased significantly from minute 10 to the end of exercise for CP_{linear} ($P < 0.001$), $CP_{1/time}$ ($P < 0.001$), $CP_{2\text{-}hyp}$ ($P = 0.011$), and $CP_{3\text{-}hyp}$ ($P = 0.004$; see figure 4.2). Blood lactate increased significantly from minute 15 to the end of exercise for CP_{linear} ($P < 0.001$), $CP_{1/time}$ ($P < 0.001$), $CP_{2\text{-}hyp}$ ($P = 0.020$), and $CP_{3\text{-}hyp}$ ($P = 0.046$). Blood lactate increased significantly from minute 20 to the end of exercise for CP_{linear} ($P < 0.001$), $CP_{1/time}$ ($P < 0.001$), $CP_{2\text{-}hyp}$ ($P = 0.008$), and $CP_{3\text{-}hyp}$ ($P = 0.011$). From minute 10 to the end of exercise

lactate increased by 4.1 ± 2.0 mmol · L^{-1} for CP_{linear}, 4.2 ± 1.5 mmol · L^{-1} for $CP_{1/time}$, 2.2 ± 1.3 mmol · L^{-1} for $CP_{2\text{-}hyp}$, 1.8 ± 0.4 mmol · L^{-1} for $CP_{3\text{-}hyp}$. Two participants had an increase of less than 1 mmol · L^{-1} in the last 20 minutes of the 30-minute trial for CP_{linear}, $CP_{1/time}$, $CP_{2\text{-}hyp}$, and $CP_{3\text{-}hyp}$. The other 9 participants had an increase of more than 1 mmol · L^{-1} for all models.

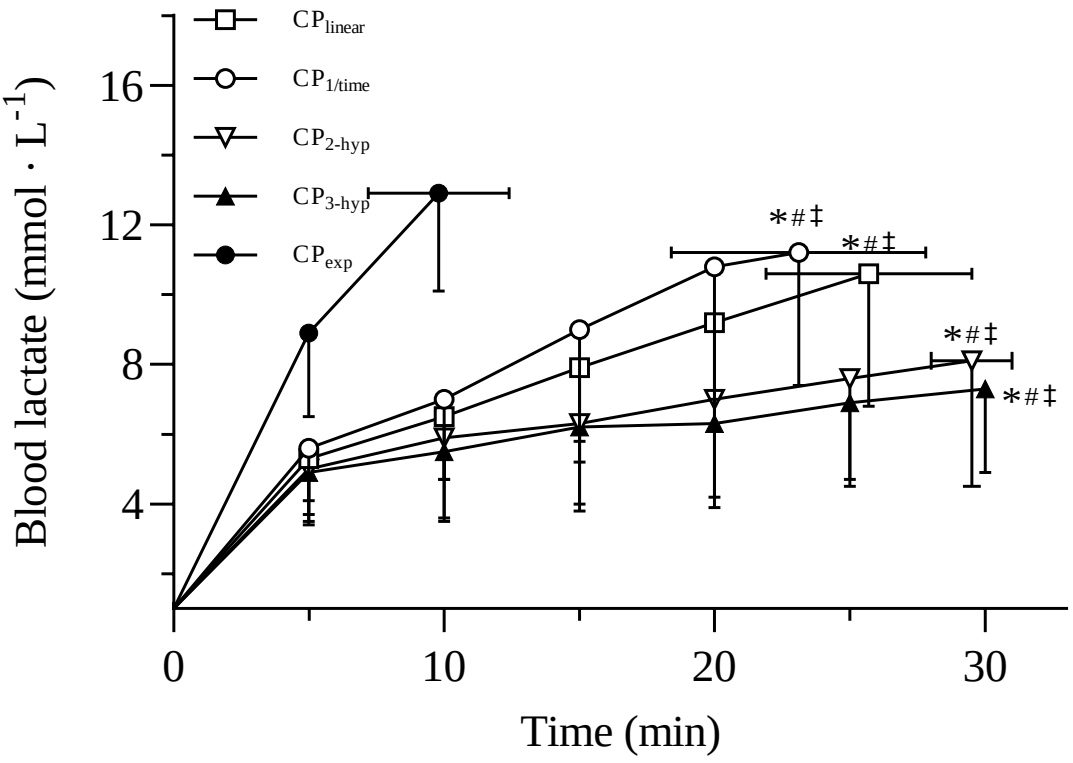

Figure 4.2: Blood lactate response to exercise at CP_{linear}, $CP_{1/time}$, $CP_{2\text{-}hyp}$, $CP_{3\text{-}hyp}$, and CP_{exp}. *Significant increase in lactate from minute 10 to end of exercise. #Significant increase in lactate from minute 15 to end of exercise. ‡Significant increase in lactate from minute 20 to end of exercise.

4.4 Perceptual response to exercising at the different CP estimates

RPE did not significantly increase from minute 10 to the end of exercise when exercising at $CP_{2\text{-}hyp}$ ($P = 0.060$) and $CP_{3\text{-}hyp}$ ($P = 0.115$). RPE increased significantly from minute 15 to the end of exercise during exercise at the CP_{linear} ($P < 0.001$) and $CP_{1/time}$ ($P < 0.001$). RPE did not significantly increase from minute 15 to the end of exercise when exercising at $CP_{2\text{-}hyp}$ ($P = 0.762$) and $CP_{3\text{-}hyp}$ ($P = 0.569$). RPE increased significantly from minute 15 to the end of exercise during exercise at the CP_{linear} ($P < 0.001$) and $CP_{1/time}$ ($P < 0.001$). RPE did not significantly increase from minute 20 to the end of exercise when exercising at $CP_{2\text{-}hyp}$ ($P = 1.000$) and $CP_{3\text{-}hyp}$ ($P = 1.000$). RPE increased significantly from minute 20 to the end of exercise during exercise at the CP_{linear} ($P < 0.001$) and $CP_{1/time}$ ($P < 0.001$). End-exercise RPE was significantly lower than maximal exertion (20 on the scale of 6 to 20) when exercising at $CP_{2\text{-}hyp}$ ($P < 0.001$) and $CP_{3\text{-}hyp}$ ($P < 0.001$). RPE was not significantly different to 20 at the end of exercise for CP_{linear} ($P = 0.574$), $CP_{1/time}$ ($P = 1.000$), and CP_{exp} ($P = 1.000$) (Figure 4.3).

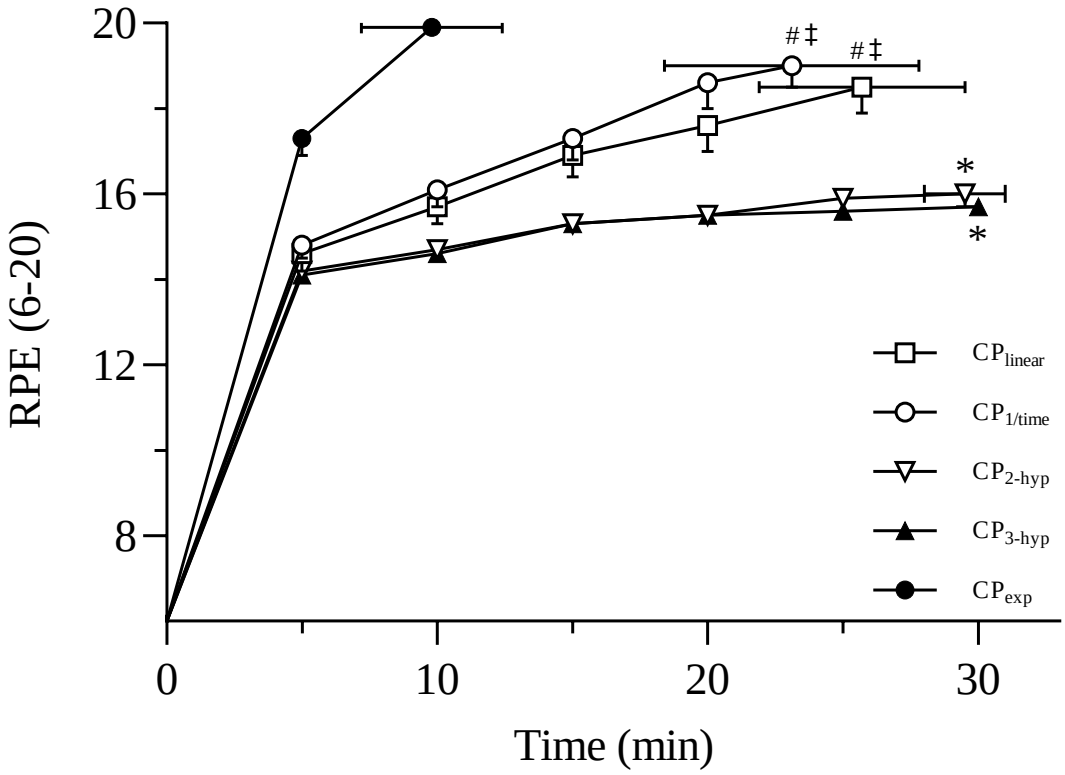

Figure 4.3: RPE when exercising at CP_{linear}, $CP_{1/time}$, $CP_{2\text{-}hyp}$, $CP_{3\text{-}hyp}$, and CP_{exp}. *Significantly lower than maximal perceived exertion (20). #Significant increase from minute 15 to end of exercise. ‡Significant increase from minute 20 to end of exercise.

5. Discussion

5.1 Main findings

The main findings of this study were that exercise at $CP_{2\text{-}hyp}$ and $CP_{3\text{-}hyp}$ resulted in attainment of a submaximal $\dot{V}O_2$ and RPE steady state, thus providing an adequate estimate of MMSS. Exercise at CP_{linear} resulted in the attainment of a submaximal $\dot{V}O_2$ steady state at a group level, but RPE failed to attain a steady state and reached maximal levels by the end of exercise. Exercise $CP_{1/time}$ and CP_{exp} failed to achieve a submaximal $\dot{V}O_2$ and RPE steady state and both parameters reached maximal levels. All models failed to reach a steady state in blood lactate levels.

5.2 Differences in CP estimation between models

Findings of the current study demonstrate that CP_{exp} produces estimates that are ~30 W higher than other models. These findings are also supported by those of Mattioni Maturana *et al.* (2018), albeit with this research suggesting ~20 W higher estimate. These differences between the studies can be attributed to the different methods used to establish P_{max} in the CP_{exp} and $CP_{3\text{-}hyp}$ models. Mattioni Maturana *et al.* (2018) obtained P_{max} from the model fitting using 4 or more trials, thus, providing an estimate of P_{max}. In the current study, P_{max} was obtained empirically from a performed maximal sprint. Mattioni Maturana *et al.* (2018) found a difference of ~10 W between $CP_{3\text{-}hyp}$ and $CP_{1/time}$, very close to the difference of 10 W found in the current study,

even though the P_{max} setting method was different between the studies. The differences between $CP_{2\text{-}hyp}$ and $CP_{3\text{-}hyp}$ were similar in both studies, ~3 W were found by Mattioni Maturana *et al.* (2018) and 4 W here. When the duration of the trials of Mattioni Maturana *et al.* (2018) was closest to the current study, CP_{linear}, $CP_{1/time}$, and $CP_{2\text{-}hyp}$ were almost identical, $CP_{1/time}$ being 1 W higher than the other two models. In the current study, the differences were slightly larger, with $CP_{1/time}$ being 2 W higher than CP_{linear} and 6 W higher than $CP_{2\text{-}hyp}$.

The differences between models found in both the current study and that of Mattioni Maturana *et al*. (2018) may pose a question as to whether the differences between models are significant when CP_{exp} is excluded, even if previous research has shown greater differences in CP estimates between models (Gaesser *et al.* 1995; Bull *et al.* 2000; Bergstrom *et al.* 2014). Statistically, there were significant differences in our study. All models were statistically different from each other, apart from CP_{linear} and $CP_{1/time}$, when Bonferroni adjustment was applied for pairwise comparison. However, when pairwise comparison was performed with the Fisher Least Significant Difference method all models were significantly different, CP_{linear} and $CP_{1/time}$ included. However, that is a testament to the variation, but not the magnitude of the variation. When the confidence interval is considered, all models are within the confidence interval of the other models. Thus, can the small differences between the models be noticeable? When the lowest estimate, obtained from $CP_{3\text{-}hyp}$, is compared to the highest, obtained from $CP_{1/time}$, the difference is 10 W, both in the current study and that from Mattioni Maturana *et al.* (2018). When put into context, the difference is likely important from an applied perspective. For the following example, the formula below will be used:

$$P = (1 - \frac{\text{Loss}_{dt}}{100})^{-1} \cdot [(9.8067 \cdot M \cdot [\sin(\arctan(\frac{G}{100})) + C_{rr} \cdot \cos(\arctan(\frac{G}{100}))]) + (0.5 \cdot C_d \cdot A \cdot \text{Rho} \cdot S^2)] \cdot S$$

where P is power output, Loss_{dt} is the drivetrain loss as a percentage, M is the system mass, C_{rr} is the coefficient of rolling resistance, G is the percentage gradient of the road, C_d is the drag coefficient, A is the area, Rho is the air density, and S is speed. For the calculations, a system mass (cyclist, bike, and all equipment combined) of 72 kg, a C_{rr} of 0.005 (Grappe *et al.* 1999), a CdA of 0.350 m^2 (Crouch *et al.* 2017), and a Rho of 1.226 $kg \cdot m^3$ will be assumed.

If the 10 W difference between models is applied in the context of a typical wattage for a professional cyclist contending the overall win of a grand tour race like the Tour de France, 400 W (Van Erp *et al.* 2020), the difference is 15 W. At that power, a Grand Tour contender would climb Alpe d'Huez (13.2 km and 8.1% grade), a recurrent Tour de France climb, in 38 min 19 s. If that rider was able to produce 15 W more, he would climb it in 37 min 11s, 1 min 8s faster. The last time the Tour de France finished at the Alpe d'Huez, 53 s were the difference between the winner of the stage and the 10th finisher. Even if 10 W seems like a small difference, it is a noticeable difference and can be the difference between winning or being dropped and finishing 10th in a stage of the Tour de France. Thus, apart from being statistically significantly different, the models produce estimates that are different in practical terms too; the magnitude of difference between them could result in target power outputs that may not produce a sustainable effort during a climb, leading to a loss of more than 1 minute in a climb such as that of Alpe d'Huez.

Other studies have also shown differences in CP estimates between models; Bull *et al.* (2000) found a difference of 28 W between $CP_{3\text{-hyp}}$ and $CP_{1/time}$, for CP estimations around 200 W, which are larger differences for a sample with a considerably lower CP than the current study. The reasons for the larger differences in Bull *et al.*'s study (2000) can be due to the lower fitness level of the participants and the length of the CP determination trials. The shortest CP determination trial in Bull *et al.*'s study (2000) lasted between 0.6 min and 2.2 min, depending on the participant. The longest trial lasted between 9.0 min and 35.8 min, depending on the participant. Thus, there were participants with CP determination trials outside of the recommended length of 2 to 15 min (Vanhatalo, Jones and Burnley 2011). The inadequate trial length and variation in trial length between participants may have led to an inadequate assessment of CP and increased error, which leads to a bigger difference between models. Gaesser *et al.* (1995) found even bigger differences. The difference between $CP_{3\text{-hyp}}$ and $CP_{1/time}$ was 42 W in their study. Gaesser *et al.* (1995) also had participants with a lower fitness level, which could lead to inappropriate completion of the maximal trials. The CP determination trial length was inappropriate too, as the shortest trial lasted ~1 min and the longest ~20 min, with a participant for whom the longest trial lasted 28 min. Bergstrom *et al.* (2014) also found big differences, with a separation of 22 W between $CP_{3\text{-hyp}}$ and $CP_{1/time}$. Similar to Gaesser *et al.* (1995) and Bull *et al.* (2000), their participants' fitness level was lower, with a CP around 180 W, but the length of the trials was in norm with the current recommendations. These significant differences in CP estimates between models seen in the literature certainly show that the different models would lead to different physiological responses at the same supposed threshold point. It can be seen that, when the length of time is appropriate and when the participants are trained, the differences in the CP estimates are diminished, as shown by Mattioni Maturana *et al.*

(2018) and the current study. However, even if the differences are smaller, they remain both statistically and practically significant. Although Gaesser *et al.* (1995), Bull *et al.* (2000), Bergstrom *et al.* (2014), and Mattioni Maturana *et al.* (2018) analysed the differences in CP estimates from the different models, they did not analyse whether exercise at the different CP estimates produced different physiological responses.

5.3 Physiological responses to exercise at CP

5.3.1 $\dot{V}O_2$ response

In the $\dot{V}O_2$ plot, Figure 4.1, three groups of responses can be seen. First, $CP_{2\text{-}hyp}$ and $CP_{3\text{-}hyp}$ had a similar response. Exercise at the CP estimate from both models caused a $\dot{V}O_2$ plateau near 3.55 L · min^{-1}, at a similar time point. After reaching the plateau, neither of them had any further increase in $\dot{V}O_2$ and the level of the plateau was significantly lower than $\dot{V}O_{2peak}$, attained during the maximal trials for CP estimation, for both models. Secondly, CP_{linear} and $CP_{1/time}$ also had a similar response to each other. A plateau was reached at 3.85 L · min^{-1} for both, higher than $CP_{2\text{-}hyp}$ and $CP_{3\text{-}hyp}$, and at a similar time point, both later than $CP_{2\text{-}hyp}$ and $CP_{3\text{-}hyp}$. For CP_{linear} and $CP_{1/time}$ there were no further increases in $\dot{V}O_2$ after the stabilisation of $\dot{V}O_2$. $CP_{1/time}$ was not significantly lower than $\dot{V}O_{2peak}$ but CP_{linear} was significantly lower than $\dot{V}O_{2peak}$. Lastly, CP_{exp} shows a different behaviour to the previous two groups, as it reaches a plateau in $\dot{V}O_2$ earlier, at a higher fraction of $\dot{V}O_{2peak}$. The time at which fatigue occurred is also markedly shorter during exercise at CP_{exp} than in the other four models.

The behaviour shown in Figure 4.1 may lead to erroneous interpretation. CP_{linear} and $CP_{1/time}$ both show a steady state that is maintained for 10-15 min at a submaximal level, which can lead to thinking that a submaximal steady state was achieved. The reason for that is that there was certain heterogeneity in the results. Three participants had a submaximal steady state when exercising at $CP_{1/time}$ and 4 participants had a submaximal steady state when exercising at CP_{linear}. The rest of the participants reached $\dot{V}O_{2peak}$ and had to stop soon after reaching $\dot{V}O_{2peak}$, which resulted in the mean $\dot{V}O_2$ data being suggestive of a steady state at submaximal values. Instead, the mean data need to be seen as the product of two distinct $\dot{V}O_2$ responses: one of a submaximal steady state and another one of maximal $\dot{V}O_2$ sustained for a short time (this can be graphically seen in Figure 5.1). Most likely, the difference of 1 subject in achieving a steady state during CP_{linear} but not $CP_{1/time}$ leads to end $\dot{V}O_2$ being considerably lower than $\dot{V}O_{2peak}$ for CP_{linear} but not $CP_{1/time}$.

It also needs to be noted that despite all models reaching a $\dot{V}O_2$ plateau, the aetiology of the plateau was not the same for all models. In the case of CP_{exp}, the plateau is clearly reached due to reaching the maximal oxygen uptake capacity of the subject. This can also be seen in the time at which fatigue occurred, as it is close to the stabilisation time. In the case of CP_{linear} and $CP_{1/time}$, there is a combination of achieving a plateau due to reaching $\dot{V}O_{2peak}$ and achieving a submaximal steady state. For the 4 and 3 participants to reach a submaximal steady state during CP_{linear} and $CP_{1/time}$, respectively, the aetiology of the steady $\dot{V}O_2$ was different from the remaining 7 and 8 participants who achieved a plateau in $\dot{V}O_2$ when they reached $\dot{V}O_{2peak}$. On the other hand, the steady state achieved by all participants during exercise at $CP_{2\text{-}hyp}$ and $CP_{3\text{-}hyp}$ was due to reaching a submaximal steady state. The different aetiology of the steady $\dot{V}O_2$ may be

explained by the exercise intensity domain within which the participants were cycling. While cycling at CP_{exp}, all participants were likely in the severe intensity domain, which fails to achieve a submaximal $\dot{V}O_2$ and $\dot{V}O_2$ keeps increasing until maximal levels are reached (Poole *et al.* 1988; Vanhatalo *et al.* 2016; Black *et al.* 2017). For the 7 participants exercising at CP_{linear} and 8 participants exercising at $CP_{1/time}$, this was also the likely scenario. However, all the participants exercising at $CP_{2\text{-}hyp}$ and $CP_{3\text{-}hyp}$, and 4 participants exercising at CP_{linear} and 3 participants exercising at $CP_{1/time}$ were exercising in the heavy intensity domain. Exercise in the heavy intensity domain is characterised by achieving a steady state in $\dot{V}O_2$ at a submaximal level (Poole *et al.* 1988; Vanhatalo *et al.* 2016; Black *et al.* 2017). It also needs to be noted that identifying no further increase in $\dot{V}O_2$ does not necessarily mean that the exercise is in the heavy domain, as $\dot{V}O_{2max}$ can be sustained for up to 14 minutes during constant load exercise (Morton and Billat 2000). Thus, it is possible to achieve a plateau in $\dot{V}O_2$ at $\dot{V}O_{2max}$ and then sustain that $\dot{V}O_2$ without any further increase for several minutes. To know whether a submaximal steady state is achieved and, consequently, exercise is in the heavy domain, the $\dot{V}O_{2peak}$ of a subject needs to be known to rule out the attainment of a $\dot{V}O_2$ plateau at maximal levels instead of a submaximal steady state.

The data from the current study cannot be directly compared to other studies, as no other studies, to the author's knowledge, have measured $\dot{V}O_2$ during exercise at the CP estimates from the different CP models.

Data from the current study can be compared to others investigating the $\dot{V}O_2$ response while exercising at CP or near CP (Poole *et al.* 1988; Brickley, Doust and Williams 2002; Vanhatalo *et al.* 2016; Black *et al.* 2017; Nixon *et al.* 2021). There are mixed results on the $\dot{V}O_2$

response at CP, with some studies showing a behaviour corresponding to the heavy intensity domain (Poole *et al.* 1988) and others showing a behaviour corresponding to the severe intensity domain (Brickley, Doust and Williams 2002). Poole, Gardner and Whipp (1988) found a $\dot{V}O_2$ response characteristic of the heavy intensity domain, as $\dot{V}O_2$ reached a submaximal steady state. That response was similar to the response found in the current study when exercise was conducted at $CP_{2\text{-}hyp}$ and $CP_{3\text{-}hyp}$. It needs to be noted that Poole, Gardner and Whipp used $CP_{1/time}$, so their results do not align directly with the results of the current study. The duration of the CP determination trials was not provided in the study by Poole, Gardner and Whipp, although they stated that the duration of the shortest trial was longer than 1 minute. It cannot be precisely known whether a difference in the length of the trials could explain the discrepancy between the results. On the other hand, Brickley, Doust and Williams (2002) did not find a steady state exercising at CP and concluded that it did not show a typical response of the heavy intensity domain when using $CP_{1/time}$, which aligns with the results of the present study.

When $\dot{V}O_2$ data from studies comparing exercise below and above CP are considered, studies show similar results in the $\dot{V}O_2$ response (Vanhatalo *et al.* 2016; Black *et al.* 2017; Nixon *et al.* 2021). All three studies found a steady state in $\dot{V}O_2$ below CP and a nonsteady state in $\dot{V}O_2$ above CP, or CS in the case of Nixon *et al.* (2021). The three studies followed the current guidelines for CP estimation, ensuring better reliability of the CP estimates when compared to the earlier studies. The model used for CP determination was the model that yielded the least standard error of the estimate of CP and W′ combined. The difference between the trial below CP and above CP of the studies amounts to more than the difference between $CP_{3\text{-}hyp}$ and $CP_{1/time}$ of the current study. Thus, no matter which model used, the power output in the trial below CP

was lower than $CP_{3\text{-hyp}}$ and the power output above CP was higher than $CP_{1/time}$. Thus, a direct comparison with the current study is difficult to make.

5.2.2 Lactate response

Contrary to $\dot{V}O_2$, the lactate response was not as different between the models. The rate of increase was greater the higher the CP estimate of the model, although all of the models failed to achieve a steady state. The explanation for lactate not being steady while $\dot{V}O_2$ was steady is not clear. Below MMSS, the produced lactate is oxidised by the mitochondria or other tissues in the body (Brooks 2018). Above MMSS, lactate does not achieve a steady state, pH levels decrease, and PCr concentration decreases leading to termination of exercise (Poole *et al.* 1988; Vanhatalo *et al.* 2016; Black *et al.* 2017). However, it has been shown that muscle lactate and blood lactate do not always have the same behaviour (Vanhatalo *et al.* 2016). The discrepancy between muscle and blood lactate is due to the differences between lactate production in the muscle, lactate efflux from the muscle to the blood, and lactate uptake from the blood (Stainsby and Brooks 1990). Thus, failing to achieve stable blood lactate levels at CP, as seen in the current study, does not necessarily mean that muscle lactate homeostasis was disturbed.

The current results are consistent with previous data showing a nonsteady lactate response to exercise at CP (Poole *et al.* 1988; Brickley, Doust and Williams 2002; Vanhatalo *et al.* 2016; Galán-Rioja *et al.* 2020; Nixon *et al.* 2021), and support the notion that CP represents an intensity that is higher than MLSS (Galán-Rioja *et al.* 2020). Further, these results suggest that CP sits at an intensity at which energy contribution is not solely derived aerobically given

the glycolytic activity yielding an increase in blood lactate. That does not mean, necessarily, that CP does not represent MMSS. In this thesis, MMSS is determined by analysing whether a $\dot{V}O_2$ steady state is achieved rather than a blood lactate steady state, due to its ability to better represent the physiological state of the body (Jones *et al.* 2019b; Bräuer and Smekal 2020; Nixon *et al.* 2021). A lack of a steady state in blood lactate is not a sufficient condition to reject MMSS. During constant workload exercise, it is possible for $\dot{V}O_2$ to be steady, muscle lactate to be steady, but blood lactate to increase due to different efflux and uptake rates (Vanhatalo *et al.* 2016). Thus it may be possible for CP to represent MMSS, where energy production is steady, despite not being produced solely by oxidative means (Jones *et al.* 2008). However, testing this hypothesis would require an analysis of muscle lactate production which was not possible in the study.

5.2.3 Perceptual response

RPE shows two markedly different responses. Exercise using $CP_{2\text{-}hyp}$ and $CP_{3\text{-}hyp}$ both showed submaximal RPE values and a steady state achieved in the second half of the trials. In contrast, CP_{linear}, $CP_{1/time}$, and CP_{exp} failed to achieve a steady state and reached maximal values. These marked differences in responses align with the differences seen in $\dot{V}O_2$ data.

The perception of effort is a complex psychophysiological measure that is regulated by motor drive, afferent feedback, prior experience, awareness, and motivation (Abbiss *et al.* 2015). Those factors are heavily affected by changes in homeostasis, especially by increased proton accumulation and fall in pH (Noble *et al.* 1983; Robertson *et al.* 1986; Mense 2009). The relation

between motor output and RPE is due to an efference copy of the motor command being sent to sensory areas of the brain, which generates perceptions associated with motor output (Enoka and Stuart 1992; Duncan, Al-Nakeeb and Scurr 2006; Christensen *et al.* 2007; de Morree, Klein and Marcora 2012). A greater central motor command leads to a higher RPE (de Morree, Klein and Marcora 2012).

Given the interaction between the homeostatic state of the body and RPE, the increasing levels of RPE until maximal values during exercise at CP_{linear}, $CP_{1/time}$, and CP_{exp} are likely due to a progressive loss of homeostasis. Such a loss of homeostasis is present during exercise in the severe intensity domain (Black *et al.* 2017; Jamnick *et al.* 2020). On the other hand, the steady state in RPE achieved during exercise at $CP_{2\text{-}hyp}$ and $CP_{3\text{-}hyp}$ could reflect maintenance of homeostatic control, which is present during exercise in the heavy intensity domain (Black *et al.* 2017; Jamnick *et al.* 2020).

The differences in the perceptual responses during exercise at the different CP models suggest a difference in the homeostatic control of the body. Such a difference is likely due to exercise being in different exercise intensity domains during CP_{linear}, $CP_{1/time}$, and CP_{exp} on the one hand, and $CP_{2\text{-}hyp}$ and $CP_{3\text{-}hyp}$ on the other hand.

5.3 Adequacy of CP to estimate MMSS

To assess whether CP adequately reflects MMSS, there is a reliance on the measured variables during constant load exercise at the CP estimate. If the measured variables show a steady state

while exercising at CP, then it can be considered that CP represents MMSS. However, such an assessment also relies on the validity of the measured variables to adequately reflect what the physiological state of the body is. In the current study, $\dot{V}O_2$, blood lactate, and RPE were measured. RPE, certainly, is not a metabolic parameter, but it is tied to the metabolic processes of the body (Borg 1982). On the other hand, $\dot{V}O_2$ and blood lactate are parameters related to metabolic processes.

Given the perceptual and $\dot{V}O_2$ responses, it could be argued that the current study was able to identify CP as a marker of the MMSS state. These data support previous evidence providing physiological validity to CP as the boundary between the heavy and severe intensity domains (Poole *et al.* 1988; Vanhatalo *et al.* 2016; Black *et al.* 2017; Nixon *et al.* 2021). However, it is important to note that not all models used in the current study were equal in their adequacy to estimate MMSS. The physiological responses were markedly different between models. $CP_{2\text{-}hyp}$ and $CP_{3\text{-}hyp}$ showed good adequacy to represent MMSS with stable $\dot{V}O_2$ and perceptual response. Arguably, $CP_{2\text{-}hyp}$ provided the adequate estimate of MMSS given that its CP estimate was higher than $CP_{3\text{-}hyp}$. Thus, $CP_{2\text{-}hyp}$ provided the MMSS estimate and $CP_{3\text{-}hyp}$ provided an estimate for an intensity that leads to a steady state, but not the maximal intensity at which a steady state is still achievable. On the other hand, CP_{exp}, $CP_{1/time}$, and CP_{linear} were inadequate to estimate MMSS given a physiological response characteristic of the severe intensity domain, *i.e.*, an intensity greater than MMSS. Despite $\dot{V}O_2$ at the end of exercise being significantly lower than $\dot{V}O_{2peak}$ during exercise at CP_{linear}, the high proportion of participants that reached $\dot{V}O_{2peak}$ at the end of exercise, and the perceptual response, suggests that CP_{linear}

overestimated MMSS. These data show that the choice of the model is of great importance when CP is used to estimate MMSS.

It could be argued that both $CP_{2\text{-hyp}}$ and $CP_{3\text{-hyp}}$ might underestimate MMSS, given that there could still be an intensity marginally higher which would show a steady state. However, it is not feasible to test such an argument as single watt increments would be needed, which would require several days or weeks to complete. In addition, daily variation in performance and the margin of error of the equipment would likely account for more than the difference from one intensity to the next. What this study has shown is that it is possible to identify an intensity higher than MLSS, *i.e.*, $CP_{2\text{-hyp}}$ and $CP_{3\text{-hyp}}$, where a metabolic steady state is still possible, and that it applies across multiple individuals. In addition, the data show that it is still possible to achieve steady state like $\dot{V}O_2$ and perceptual responses at an intensity exceeding MLSS, despite a continuous rise in blood lactate. This adds to the data that suggests MLSS underestimates MMSS (Bräuer and Smekal 2020; Nixon *et al.* 2021).

The conclusions of the current study are in contrast to those of Bull *et al.* (2008) who suggest that CS is not adequate for determination of the MMSS. Bull *et al*. (2008) used the original concept of CP, *i.e.*, an intensity that can be sustained for a very long time without fatigue, to assess the adequacy of CS for MMSS estimation. As more than half of their participants fatigued before 60 minutes, Bull *et al.* (2008) concluded that CS was not adequate for MMSS estimation. However, as explained in section 2.3.2, the current understanding of CP/CS is different to the original conception, and the original concept should not be a criterion to assess the validity of CP/CS for MMSS estimation.

The current data suggest that the criterion used for model selection by other researchers may not be adequate (Vanhatalo *et al.* 2016; Black *et al.* 2017; Nixon *et al.* 2021). The criterion often used for model selection is to choose the CP model with the lowest error in the CP and W′ estimates. In order to logically disprove that argument, it is needed that the model with the lowest error does not represent MMSS, that is, for the precedent to be true but the consequent to be false. For the sake of comparing the current data to those from Vanhatalo *et al.* (2016), Black *et al.* (2017), and Nixon *et al.* (2021), only the mathematically equivalent models, *i.e.*, CP_{linear}, $CP_{1/time}$, and $CP_{2\text{-}hyp}$ will be considered. The current study found that in more than half of the participants (7) the best fitting model did not represent MMSS. Thus, it could be argued that the model with the lowest error should not automatically be used to estimate MMSS.

The current study showed that, at a group level, there were significant differences between models in their adequacy to reflect MMSS. However, there were also individual differences in each model's adequacy to reflect MMSS; *i.e.*, some models provided a CP estimate that caused a steady state in certain individuals but not in others. The physiological response at CP_{exp} was consistent throughout all participants, leading to the conclusion that CP_{exp} overestimates MMSS. All participants had the same response at $CP_{2\text{-}hyp}$ and $CP_{3\text{-}hyp}$ too, a response characteristic of the heavy intensity domain. However, CP_{linear} and $CP_{1/time}$ had mixed results. Specifically, 4 participants displayed a steady $\dot{V}O_2$ and perceptual response at CP_{linear} and 3 participants displayed a steady $\dot{V}O_2$ and perceptual response at $CP_{1/time}$. In these instances, given that CP_{linear} and $CP_{1/time}$ were at a higher power output than $CP_{2\text{-}hyp}$ and $CP_{3\text{-}hyp}$, it could be suggested that they provided more adequate estimates of MMSS. In Figure 5.1 the different $\dot{V}O_2$ responses of two participants while exercising at $CP_{1/time}$ can be seen. As it can be seen from the figure, it is important to consider the individual $\dot{V}O_2$ response while exercising at the CP estimate

of each model due to the individual variability in physiological responses for a given model. However, given the resource and time requirements, the use of $CP_{2\text{-}hyp}$ or $CP_{3\text{-}hyp}$ for MMSS can be used given their ability to estimate MMSS across individuals, as found in the current study.

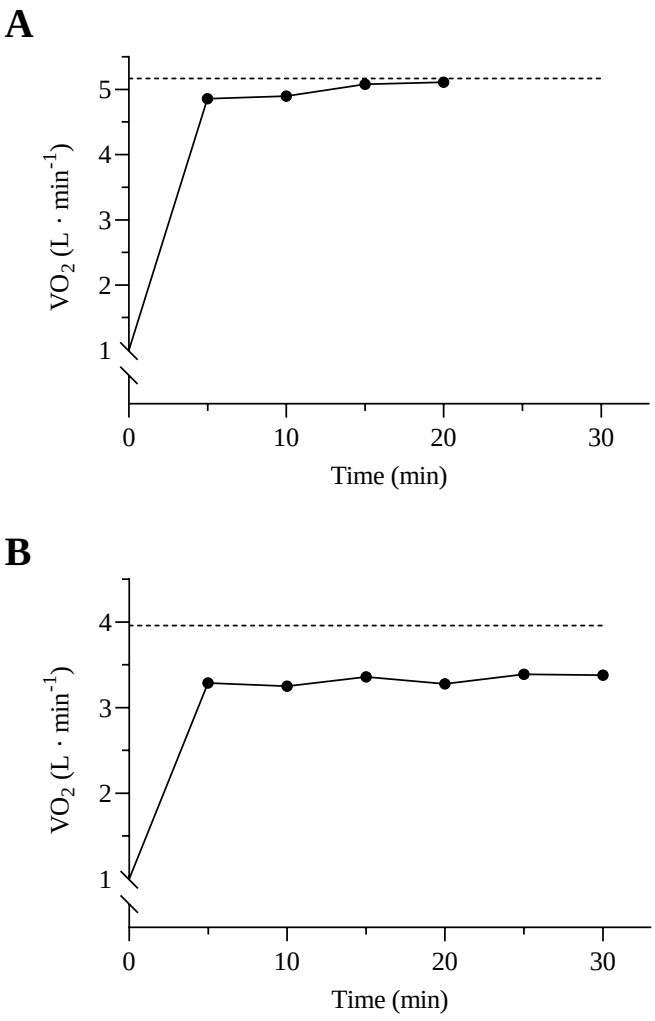

Figure 5.1: $\dot{V}O_2$ response of two different participants at $CP_{1/time}$. In panel A, it can be seen that the participant had a $\dot{V}O_2$ response characteristic of the severe intensity domain. A submaximal steady state was not achieved and $\dot{V}O_2$ reached maximal values. In contrast, in panel B, it can be seen that a participant reached a steady $\dot{V}O_2$ and that the response was characteristic of the heavy intensity domain.

Despite the validity of blood lactate as a metabolic marker being questioned earlier in this thesis, its instability in conjunction with a stable $\dot{V}O_2$ can provide an indication that CP is within a phase transition from the heavy to the severe intensity domains (Pethick, Winter and Burnley 2020). Exercise in such a phase transition has a combination of the behaviour of the heavy and severe intensity domains. Stable blood lactate levels are characteristic of the heavy intensity domain while increasing lactate levels are characteristic of the severe intensity domain (Jamnick *et al.* 2020). In the current study, $CP_{2\text{-}hyp}$ and $CP_{3\text{-}hyp}$ showed a $\dot{V}O_2$ behaviour characteristic of the heavy intensity domain, but lactate showed a behaviour of the severe intensity domain. Thus, the overall physiological response was a combination of the behaviour characteristic of both the heavy and severe intensity domains, supporting the suggestion that CP occurs within the phase transition zone. Nevertheless, data from the current study indicate a marked change in physiological responses from one model to the other, despite less than 10 W difference in cycling power output between them. As a consequence, CP might appear as a marked a threshold rather than a transition phase. It is important to note that the current study did not aim to test the phase transition hypothesis, so it is not possible to make a firm conclusion on whether CP marked a threshold or a phase transition.

5.4 Limitations

First, the protocol we used for CP testing was a single-day protocol. Despite being a valid protocol (Karsten *et al.* 2017), different results may occur when CP is tested across multiple days with a single maximal trial each day. Given that in research a multiple-day protocol has been commonly used (Poole *et al.* 1988; Jones *et al.* 2008; Vanhatalo, Jones and Burnley 2011;

Vanhatalo *et al.* 2016), current findings may not be fully transferable. In addition, the maximal trials for CP determination in the current study were time-trials, as opposed to time to exhaustion trials as used in some of the more seminal studies (Poole *et al.* 1988; Jones *et al.* 2008; Black *et al.* 2017; Mattioni Maturana *et al.* 2018). Both methods may lead to slightly different results. The duration of the trials also needs to be considered, as trials of different durations can affect the results (Mattioni Maturana *et al.* 2018).

Two exercise trials were performed in visits 2 and 3. In the second trial, it is possible that residual fatigue from the first trial may have affected the results. There could also have been alterations to $\dot{V}O_2$ kinetics. However, during pilot testing, there were no significant differences evident in $\dot{V}O_2$ kinetics or the perception responses of the participants, and there was no indication of any performance decrement from the first to the second trial. The randomisation of the trials also intended to mitigate the potential order effects in the data.

The $\dot{V}O_{2peak}$ measure was taken from the time-trials performed for CP determination. The desirable method for $\dot{V}O_{2peak}$ measurement is a ramp test where workload is increased linearly until exhaustion with the attainment of a $\dot{V}O_2$ plateau. $\dot{V}O_{2peak}$ was determined from the time-trials to limit the total amount of visits due to uncertainty around whether the study could continue until the end without interruptions. Given that one of the criteria for MMSS determination was the obtention of a steady submaximal $\dot{V}O_2$, and that whether it was submaximal was assessed based on $\dot{V}O_{2peak}$, a differently set $\dot{V}O_{2peak}$ could lead to different results. However, maximal time-trials in the severe intensity domain should lead to the participant's $\dot{V}O_{2max}$.

Additionally, the markers measured in the current study do not fully cover the metabolic processes occurring in the body. Analysis of intramuscular markers such as PCr and muscle lactate could lead to additional data that may not be consistent with the conclusions of this thesis.

Lastly, the study presented in this thesis was conducted during the COVID-19 pandemic. As a result, the duration of data collection was reduced as much as possible, without critically reducing the quality of the study, to avoid a potential interruption of data collection as a result of government restrictions. If conducted under normal circumstances, an additional initial visit would have been conducted to identify the lactate threshold and true $\dot{V}O_{2max}$ of the participants via an incremental test, as well as performing the 5 trials at CP in separate visits instead of twice per visit.

5.5 Practical recommendations

This thesis has analysed the adequacy of different CP models to assess MMSS, a key performance parameter. A correct assessment of CP is useful for training prescription (Jamnick *et al.* 2020), performance modelling and pacing (Pettitt 2016; Kirby *et al.* 2021). Given the results from this thesis, the following recommendations can be made:

- For CP determination, the use of 3 to 5 maximal trials that are 2 to 15 minutes in duration is needed, with at least one short trial lasting ~3 minutes and another long trial lasting ~12 minutes.

- A single-day CP testing protocol can be successfully used to determine MMSS. 3 maximal trials separated by 30 minutes of recovery and lasting 12, 5 to 7, and 3 minutes are recommended.
- The use of $CP_{2\text{-}hyp}$ is recommended to determine MMSS. Adding a maximal sprint to set P_{max} allows fitting $CP_{3\text{-}hyp}$, which will provide a similar result to $CP_{2\text{-}hyp}$ with a slightly lower error.

5.6 Conclusion

In this thesis, novel data have been presented on the adequacy of each CP model to assess MMSS. CP_{linear}, $CP_{1/time}$, and CP_{exp} have shown to be inadequate for MMSS estimation. In contrast, $CP_{2\text{-}hyp}$ and $CP_{3\text{-}hyp}$ have shown to be adequate for MMSS estimation across a sample of cyclists as shown by the attainment of a steady state $\dot{V}O_2$ and RPE. Additionally, MMSS estimation can be done in a single day, using the $CP_{2\text{-}hyp}$ and $CP_{3\text{-}hyp}$ models, which can be applied without interfering with the training process. Further research investigating intra-muscular metabolic markers may provide additional data to guide the choice of a CP model to estimate MMSS.

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