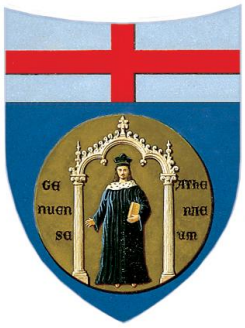

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The Role of Anaerobic Power Reserve on Exercise Tolerance: From Physiological Correlates to Performance Prediction

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Summary

ABSTRACT

Introduction

The anaerobic power reserve (APR) is defined as the difference between maximal sprint power and the power associated with $\dot{V}O_2$max and has been proposed as a simple tool to capture the athlete's anaerobic characteristics. However, its actual anaerobic role in prescribing severe exercise intensity and its predictive value for short-duration cycling and swimming performance remain unclear. Therefore, this thesis aimed to: (i) determine whether APR reduces the heterogeneity of physiological responses during $\dot{V}O_2$max-based HIIT; (ii) examine the relationship between APR and time to exhaustion across intensities; (iii) evaluate the combined predictive value of APR and critical speed for 50-, 100-, and 200-m frontcrawl performance; and (iv) explore the association between work performed above critical power, estimated using different models, and multiple anaerobic indices.

Study 1. This study examined whether APR and glycolytic power reserve (GPR) could reduce variability in HIIT responses compared with maximal aerobic power (MAP) based prescription. Twelve trained cyclists completed incremental and Wingate tests to determine MAP, APR, and GPR; then performed three randomized HIIT sessions to exhaustion (60 s work:60 s active recovery) prescribed using MAP, APR, or GPR. No significant differences in variability were observed across prescription methods for exercise tolerance, physiological, or perceptual outcomes. These findings indicate that APR- and GPR-based prescriptions do not reduce heterogeneity in HIIT tolerance or responses relative to MAP, suggesting limited ability to distinguish individual aerobic–anaerobic profiles.

Study 2. This study investigated associations between APR, time to exhaustion (Tlim), work above critical power (*W'*), and maximal accumulated oxygen deficit (MAOD) in endurance-trained male cyclists, and compared APR with a maximal power reserve (MPR) model using critical power (CP) as the lower boundary. Nineteen cyclists performed multiple trials to exhaustion at different intensities

(130 to 80% of MAP) and a Wingate test. APR and MPR correlated with most Tlim, except at lower intensities, with associations remaining only for supramaximal efforts after controlling for CP or MAP. When peak power output was controlled, only MPR remained associated with Tlim. Both reserves correlated with MAOD and *W′*, but only MPR remained related to MAOD after adjustment. Overall, power reserves were related to exercise tolerance, particularly at high intensities, but these relationships were largely driven by peak power output rather than the choice of lower boundary.

Study 3. This study examined the relationship between modified 3-minute all-out test ($3mAO_{mod}$) parameters and swimming performance over 50-, 100-, and 200-m front crawl, and evaluated the predictive accuracy of critical speed (CS) and multiple linear regression (MLR) models applying the anaerobic speed reserve (ASR) framework. Twenty-three competitive youth swimmers performed the $3mAO_{mod}$ and subsequently competed in official races. CS showed increasing correlations with race time as distance increased, whereas correlations for D′ and ASR decreased with distance. The CS model underestimated performance in the 50- and 200-m events but not in the 100-m race. In contrast, MLR models incorporating CS, D′, or ASR, and anthropometric variables accurately predicted performance across all distances. These findings support the physiological relevance of $3mAO_{mod}$ parameters and demonstrate that multivariate models provide more accurate and practical performance predictions than the CS model alone.

Study 4. This study compared the estimated curvature constant (*W′*) and actual work performed above critical power ($W_{>CP}$) across intensities using different mathematical models and examined their relationships with anaerobic indices. Twenty-one participants completed trials to exhaustion at multiple intensities and a Wingate test. Critical power and *W′* were estimated using work-time, inverse-time, and hyperbolic models. $W_{>CP}$ varied with both exercise intensity and model, decreasing at the highest intensities. Two-parameter models showed lower estimation error and stronger associations between *W′* and anaerobic markers. Regardless of the model, $W_{>CP}$ at supramaximal intensities correlated with accumulated oxygen deficit, lactate, and Wingate performance. In contrast, *W′* was

associated with anaerobic markers only in specific models. These results indicate that anaerobic characteristics cannot be fully described by a single parameter and that model selection substantially influences interpretation.

Conclusion. Overall, this thesis shows that the APR is an intuitive but physiologically incomplete construct. While APR is moderately associated with anaerobic capacity and exercise tolerance at (supra) $\dot{V}O_2max$ intensities, its prediction accuracy is limited, intensity-dependent, and largely driven by peak power output rather than by the chosen lower boundary (MAP or CP). Consequently, APR does not improve the normalization of physiological responses or tolerance during HIIT compared with conventional MAP-based prescriptions. However, when applied to short, high-intensity efforts (such as 50-200 m front crawl or cycling effort < ~5min), APR is strongly related to performance. Therefore, these findings indicate that APR is unsuitable for standardizing HIIT intensity because it cannot capture the complexity of the dynamic depletion and reconstitution of $W_{>CP}$; however, it could be used as a simple and useful tool to prescribe and predict short-duration performance.

PREFACE

The ability to sustain high-intensity exercise is fundamental to performance in a wide range of sports, from cycling to swimming. The capacity to tolerate high-intensity exercise is determined by multiple factors, among which an athlete's anaerobic characteristics play a pivotal role. While the contribution of aerobic metabolism to endurance performance is well-characterized and routinely assessed through parameters such as maximal oxygen uptake ($\dot{V}O_2max$), the quantification of an athlete's anaerobic qualities remains a significant challenge. This thesis examines a relatively recent model proposed to fill this gap: the anaerobic power reserve (APR). Defined simply as the difference between an athlete's maximal sprint power (peak power output, PPO) and their maximal aerobic power (MAP), the APR promises to provide a simple, integrative tool for athlete profiling, performance prediction, and exercise prescription. However, despite its theoretical appeal and growing popularity, the physiological validity and practical utility of the APR model remain largely unproven. This work aims to address this knowledge gap by first establishing the energetic principles underpinning the model, then, through four interconnected studies, to evaluate its ability to explain variance in performance, its attempt at normalizing training intensity, and its applicability across different sports (cycling and swimming). By doing so, this thesis seeks to move the conversation from unqualified enthusiasm to a nuanced, evidence-based understanding of what the APR model can - and cannot - offer to sport and exercise science.

SECTION 1

1. GENERAL INTRODUCTION

1.1.The Energetic Basis of Muscular Contraction

Every action of the human body requires energy, and movement is no exception. Human locomotion relies on a complex musculoskeletal system, coordinated by electrochemical signals originating from the nervous system. Within this system, the muscles play a central role: their contractions, which generate movement, are strictly dependent on the continuous supply of energy (Heckman & Enoka, 2004). However, before understanding how muscles produce movement, it is necessary to first describe their structure, moving from macroscopic to microscopic features. At the macroscopic level, skeletal muscle is organized into bundles that can be externally recognized as distinct muscle groups. Each muscle is composed of numerous fascicles, which in turn contain individual muscle fibers, the functional cellular units of contraction (Mukund & Subramaniam, 2020). Muscle fibers can be broadly classified into different types according to their contractile and metabolic properties, most commonly type I (slow-twitch, oxidative), type IIa (fast-twitch, oxidative-glycolytic), and type IIx (fast-twitch, glycolytic) (Moreno-Justicia et al., 2025). Within each fiber, the contractile machinery is arranged into myofibrils, long cylindrical structures composed of repeating units known as sarcomeres. The sarcomere is the fundamental contractile unit, consisting primarily of two main contractile proteins: actin (thin filaments) and myosin (thick filaments), whose cyclic interactions generate force. Supporting proteins, such as titin, nebulin, and desmin, contribute to the alignment, elasticity, and structural integrity of the contractile apparatus (Moreno-Justicia et al., 2025). Together, this hierarchical organization, ranging from the whole muscle to the sarcomere, provides the mechanical basis for force generation and movement. A schematic representation of the muscle architecture is shown in **Figure 1.**

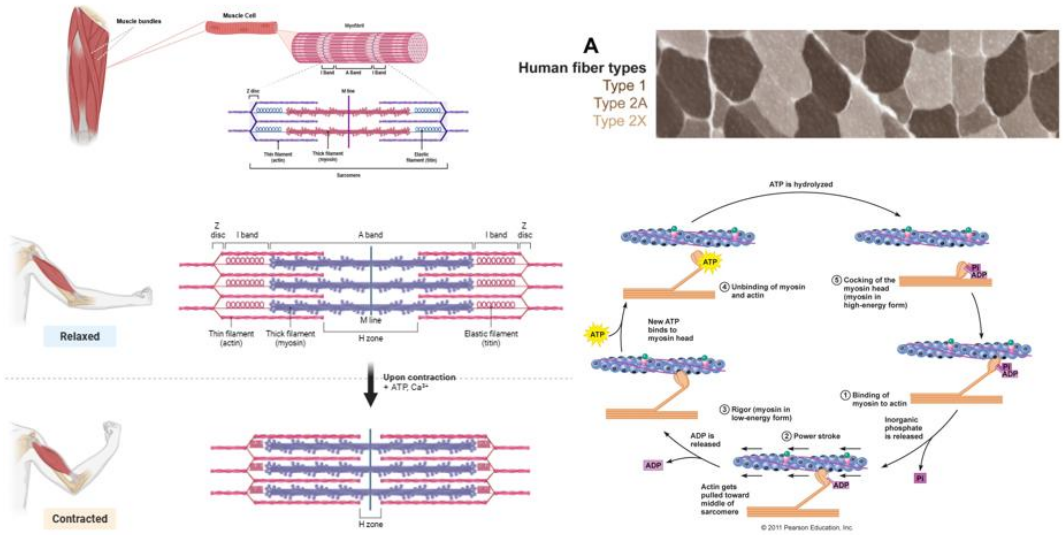

Figure 1. *Structure of skeletal muscle showing muscle bundles, fascicles, and fibers (A). Histological differences between muscle fiber type (b). Different states of the overlapping mio-proteins during contraction and relaxation (c). Basic description of the powerstroke cycle of the myosin heavy chain on the actin filament.*

1.1.1. The Role of ATP and Calcium in the Cross-Bridge Cycle

At rest, the muscle fiber is prevented from contracting. The actin filaments have binding sites for the myosin heads, but they are covered by a regulatory protein called tropomyosin, which is held in place by another complex called troponin (Fitts, 2008). When a (train of) action potential is fired by the central nervous system (CNS), through motor neurons and the neuromuscular junction, it will reach the muscle fiber (Heckman & Enoka, 2004). This signal causes the sarcoplasmic reticulum (a specialized organelle in the muscle cell) to release its stored calcium ions (Ca^{2+}) into the cytoplasm. The released Ca^{2+} binds to specific sites on the troponin complex, causing a change in the shape of the troponin complex that moves the tropomyosin protein away from the myosin-binding sites on the actin filament, thereby exposing its binding sites. This allows the myosin heads to bind to actin, forming cross-bridges (Fitts, 2008).

When an adenosine triphosphate (ATP) molecule binds to the myosin head, a decrease in myosin's affinity for actin leads to the immediate dissociation of the myosin head from the actin filament. Following detachment, the myosin head functions as an ATPase enzyme, catalyzing the hydrolysis of

bound ATP into adenosine diphosphate (ADP) and inorganic phosphate (Pi). The free energy released from this hydrolysis is captured within the myosin molecule, inducing a conformational change that cocks the lever arm of the myosin head into a high-energy, pre-power stroke state (Sundberg et al., 2018; Walklate et al., 2016). This energized myosin head then binds to a vacant binding site on the actin filament, forming a new actomyosin cross-bridge (**Figure 1**). The subsequent strong binding to actin stimulates the release of the products of hydrolysis, ADP and Pi. This release triggers the primary mechanical event of the cycle: the power stroke. During the power stroke, the myosin head undergoes a second, more drastic conformational change, pivoting on its hinge region and translating the stored chemical energy into mechanical work (Sundberg et al., 2018; Walklate et al., 2016). This action pulls the actin filament toward the center of the sarcomere, resulting in filament sliding, sarcomere shortening, and force production. Post-power stroke, the myosin head remains bound to actin in a rigor-like state, characterized by very high affinity and low energy (Fitts, 2008; Sundberg et al., 2018). The conclusion of the cycle and the prerequisite for further cycling is the binding of a new ATP molecule. Nucleotide binding to the actin-bound myosin head drastically reduces its binding affinity for actin, facilitating cross-bridge dissociation and allowing the myosin head to begin a new cycle (Fitts, 2008; Sundberg et al., 2018). Therefore, ATP serves as the substrate and energetic driver; its hydrolysis provides the free energy for the mechanical stroke, and its binding is essential for the critical dissociation of the cross-bridge to permit cycling. The perpetual demand for ATP to fuel this process underscores the critical importance of metabolic pathways.

1.1.2. Metabolic Pathways for ATP Resynthesis: Power vs. Capacity

The excitation-contraction coupling process explained above is exclusively fueled by the hydrolysis of ATP (Hargreaves & Spriet, 2020). Within the myocyte, a limited quantity of ATP is stored in the cytosol, while a larger, rapidly mobilizable reserve exists in the form of phosphocreatine (PCr), constituting the phosphagen system (Hargreaves & Spriet, 2020). This system has the highest power (the rate of ATP regeneration) among all metabolic pathways, facilitating immediate energy provision

(Hargreaves & Spriet, 2020). However, its capacity, the total amount of ATP it can produce, is severely constrained. Empirical evidence indicates that intramuscular ATP and PCr can be depleted by up to 80% following approximately 30 seconds of supramaximal exercise (Hargreaves & Spriet, 2020). To sustain ATP homeostasis beyond this brief period, cells must rely on the catabolism of energy substrates via other pathways, namely anaerobic glycolysis (independent of O_2) and oxidative phosphorylation (dependent on O_2). The anaerobic glycolysis involves the rapid breakdown of glucose or glycogen into pyruvate, with a net yield of 2 or 3 ATP per glucose molecule, respectively (Kierans & Taylor, 2024). To maintain redox balance in the absence of oxygen, pyruvate is reduced to lactate. While its power is lower than that of the phosphagen system, its capacity is significantly greater (Hirvonen et al., 1987). It serves as the predominant ATP source during high-intensity efforts lasting from ~30 seconds to ~2 minutes. The oxidative phosphorylation is conducted within the mitochondria and represents the most efficient mechanism for ATP production. It involves the complete oxidation of substrates (glycogen, glucose, fatty acids, and, to a lesser extent, amino acids) through the Krebs cycle and the electron transport chain, yielding approximately 30-33 ATP per glucose molecule (Hargreaves & Spriet, 2020). Although it possesses the highest capacity, its power, limited by the rate of oxygen delivery and mitochondrial respiration, is the lowest of the three systems. It is the primary energy pathway at rest and during prolonged, submaximal exercise (Hargreaves & Spriet, 2020).

1.2. Cellular Determinants of Athletic Performance: A Continuum

1.2.1. Anaerobic Characteristics: PCr Stores, Glycolytic Enzymes, and Type II Fibers

Extensive research has established that athletic performance across different durations (and intensities) is strongly correlated with distinct cellular and metabolic adaptations. Superior performance in short-duration, high-intensity exercise is positively associated with several anaerobic characteristics (Galvan-Alvarez et al., 2024; Medbo et al., 1988). These include a high abundance of intramuscular ATP and PCr stores, elevated activity of key glycolytic enzymes such as phosphofructokinase (PFK), and a greater proportion of type II (specifically IIa and IIx) muscle fibers (Sandford et al. 2021). For

instance, interventions that augment PCr reserves, such as creatine supplementation, have been consistently shown to enhance performance in tasks like 100-meter sprints and mean power output during 30-second all-out cycling tests (Cooper et al. 2012). Moreover, the correlation between type II fiber percentage, PFK activity, and sprint performance lasting from 1 to 30 seconds is well-documented in the literature (Cheetham et al. 1986; Galvan-Alvarez et al. 2024).

1.2.2. Aerobic Characteristics: Mitochondrial Density, Capillarization, and Oxidative Enzymes

Conversely, endurance performance is linked to a separate set of cellular adaptations oriented toward aerobic metabolism. Elite endurance athletes exhibit an elevated mitochondrial density, a greater capillary-to-fiber ratio, and an enhanced intrinsic mitochondrial respiratory capacity compared to their less-trained counterparts (Holloszy and Coyle 1984). This superior aerobic machinery facilitates efficient oxygen utilization and ATP production. Furthermore, a metabolic specialization of muscle fiber types is evident. Athletes excelling in short-duration efforts possess a higher intramuscular glycogen content to fuel glycolysis, whereas those specializing in long-distance events demonstrate increased intramuscular lipid stores, which serve as a substrate for beta-oxidation and the Krebs cycle (Van Loon et al. 2003).

Consequently, it is well-established that an athlete's performance profile is intrinsically linked to their specific cellular characteristics, whether aerobic or anaerobic. Historically, the assessment of these components and the evaluation of training-induced adaptations relied heavily on the muscle biopsy technique. This procedure is highly invasive, requiring the extraction of a small tissue sample under local anesthesia, and is associated with post-procedural discomfort. Moreover, a critical methodological limitation exists: a biopsy sample taken from a single muscle may not be representative of the entire musculature involved in a sport-specific movement (Long et al. 2023; Van de Casteele et al. 2024).

It is now evident that the microscopic characteristics of muscle cells (and beyond) fundamentally determine athletic performance across a wide range of disciplines. It is based on these factors that, over the years, researchers have endeavored to construct and refine physiological-mathematical models capable not only of describing performance itself but also of elucidating the underlying physiology, in a continuous cycle of cause and effect (Jones et al., 2010; Sandford et al., 2021).

Several protocols have been developed to evaluate such characteristics in less invasive ways. For example, the incremental test with gas exchange measurement and sprint tests with or without lactate sampling are just a few of them. It is within this context that the anaerobic power reserve model finds its application (Sandford et al., 2021). This construct, defined as the difference between the maximal power output attainable through anaerobic metabolism (such as during sprint lasting less than 3-5 seconds) and the maximal power sustainable via aerobic metabolism (measured during an incremental test), provides a practical synthesis of an athlete's physiological profile (Sandford et al., 2021). What this framework offers is a perspective on whether an athlete is more aerobically or anaerobically oriented. This knowledge can be applied in several ways. It can be used to inform the prescription of exercise intensity for specific training sessions or to help determine the sporting disciplines in which an athlete is most likely to excel. For instance, an athlete with a high APR, driven primarily by an elevated peak power output, will more likely achieve superior results in short-duration events rather than in ultra-endurance competitions. In this sense, the APR model serves as a practical, non-invasive tool, but with the advantage of being more directly applicable to athlete development and routine monitoring.

SECTION 2

2. ASSESSING THE ATHLETE'S PHYSIOLOGICAL PROFILE: FROM THE MUSCLES TO THE WHOLE BODY

2.1. The Aerobic Component: The Evolution and Principles of Ergospirometry

To circumvent the limitations of invasive techniques and to provide a more holistic evaluation of athletic performance, a suite of non-invasive performance tests and laboratory instruments has been developed. For the quantification of aerobic characteristics, a pivotal advancement was the invention of the Douglas bag, which allowed for the direct collection and subsequent analysis of expired air. This was later superseded by the development of ergospirometers (also referred to as metabolic carts), which enable the continuous, breath-by-breath measurement of pulmonary gas exchange.

The fundamental principle underlying these devices is the measurement of the difference in the fractional concentrations of oxygen (O_2) and carbon dioxide (CO_2) between inspired and expired air. By simultaneously measuring the volume of air ventilated, these systems can calculate the rate of oxygen uptake ($\dot{V}O_2$) and carbon dioxide production ($\dot{V}CO_2$) (Shephard, 2017; Ward, 2018). The calculation of $\dot{V}O_2$ is based on the following equation:

$$\dot{V}O_2 = \dot{V}_I \cdot F_IO_2 - \dot{V}_E \cdot F_EO_2$$

where $\dot{V}_I$ and $\dot{V}_E$ are the inspired and expired ventilation rates, and F_IO_2 and F_EO_2 are the fractional concentrations of O_2 in inspired and expired air, respectively.

This measured $\dot{V}O_2$ at the pulmonary level is directly proportional to oxygen utilization at the cellular level. This relationship is described by the Fick principle, which states that the total oxygen consumption of the body (or a specific organ) is equal to the product of blood flow (cardiac output, $\dot{Q}$) and the arteriovenous oxygen difference ($a\text{-}vO_{2diff}$). The principle is formulated as:

$$\dot{V}O_2 = \dot{Q} \cdot (C_aO_2 - C_vO_2)$$

Where $\dot{Q}$ represents cardiac output (L/min), C_aO_2 is the arterial oxygen content (mL O_2/100 mL blood), and C_vO_2 is the mixed venous oxygen content (mL O_2/100 mL blood). Thus, pulmonary $\dot{V}O_2$ provides an integrated measure of the entire oxygen transport chain, from ventilation and diffusion in the lungs to oxygen delivery via cardiovascular function, and finally to oxygen extraction and consumption by the mitochondria in skeletal muscle (Miyamura & Honda, 1972; Montero et al., 2015; Sun et al., 2000). The maximal rate of oxygen uptake ($\dot{V}O_2max$) measured during a graded exercise test to exhaustion remains the gold standard laboratory assessment for defining an athlete's aerobic power (Poole and Jones 2017).

2.2. Quantifying the Aerobic Characteristics: $V\dot{O}_2max$ and Metabolic Thresholds

Performance in endurance sport is largely explained by the $\dot{V}O_2max$, and therefore also by the same factors that affect it: cardiac output, oxygen transport, and utilization (Lundby et al., 2017). However has also been observed that even among athletes with similar $\dot{V}O_2max$, other characteristics contribute to superior endurance performance. The two most important among these are movement economy and the fraction of $\dot{V}O_2max$ that can be sustained for prolonged periods, which is determined by the lactate threshold (or its surrogate) (Joyner, 2017). While a detailed analysis of the factors affecting movement economy lies beyond the scope of this thesis, a deeper understanding of metabolic thresholds is essential to properly contextualize the role of exercise intensity, the associated physiological responses, the determinants of exercise tolerance, and the contribution that models such as critical power or the anaerobic power reserve can provide. Indeed, these models are designed to predict performance across a range of durations and exercise modalities and, consequently, to normalize exercise intensity across individuals with different physiological profiles (Sandford et al., 2021).

As exercise intensity increases from very low levels, blood lactate initially tends to remain relatively stable; occasionally, a slight decrease from baseline can be observed. As intensity continues to rise, lactate begins to increase (and accumulate) following an exponential function (Beaver et al., 1986; Messonnier et al., 2013). This pattern enables the identification of two key thresholds (Faude et al.,

2009; Messonnier et al., 2013). The detection and classification of lactate thresholds (LT) have been addressed using numerous methods, leading to considerable confusion in the literature (Chavez-Guevara et al., 2024; Faude et al., 2009; Galán-Rioja et al., 2020; Loat & Rhodes, 1993; Sperlich & Gronwald, 2024). Indeed, several calls have been made to avoid perpetuating this continuous proliferation of disparate terms and methodologies (Chavez-Guevara et al., 2024; Faude et al., 2009; Sperlich & Gronwald, 2024). The term "lactate threshold" generally refers to a point beyond which blood lactate exceeds a certain level. However, this "level" is not written in stone; depending on the study considered, widely different methods can be observed.

What is generally agreed upon, however, is that two distinct thresholds can be identified (Faude et al., 2009; Loat & Rhodes, 1993). These two thresholds can be determined using various approaches, including:

- Fixed blood lactate concentrations: e.g., 2 mmol/L and 4 mmol/L, often referred to as the aerobic and anaerobic thresholds (Heck et al., 1985; Jamnick et al., 2020).
- Fixed increases above baseline: e.g., baseline +0.3 mmol/L and +1.5 mmol/L (Faude et al., 2009).
- Logarithmic transformations: such as the log-log method, which identifies the point where the logarithmic relationship between blood lactate and power output (or O_2 uptake) deviates from linearity (Beaver et al., 1985).
- Visual or mathematical identification of the exponential inflection point: for example, the D-max method, which identifies the point on the lactate-power curve that yields the maximal distance from the line connecting the first and last data points (Cheng et al., 1992). This point is intended to represent the highest intensity before lactate begins to accumulate exponentially, leading eventually to exhaustion.

- Increase of no more than 1 mmol/L between the 10th and 30th minute of a constant work rate, also known as the maximal lactate steady state (MLSS) (Caen et al., 2024).

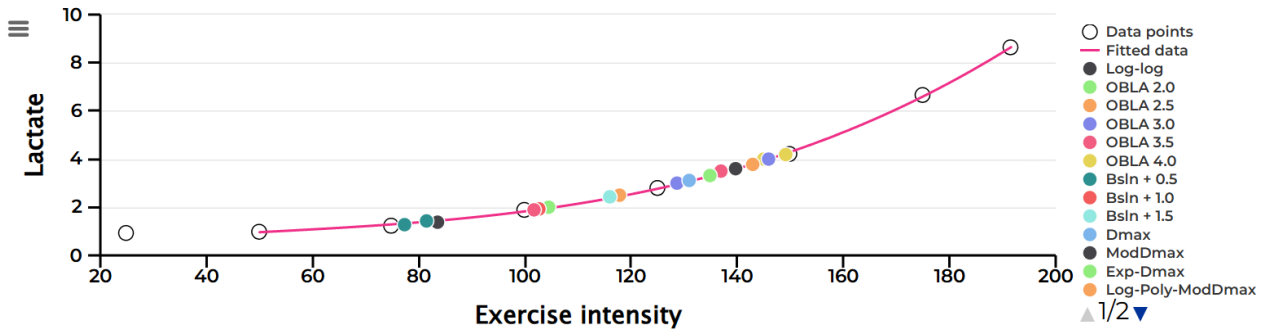

***Figure 2**. An example of the various LT that can be found in the literature. This image has been generated from simulated data using the Exphys software (https://www.exphyslab.com/lactate).*

Notably, some of these thresholds occur at a relatively low intensity (e.g., baseline + 0.3mmol/L, Log-Log, or the 2 mmol/L) and are often referred to as LT_1. While others occur at higher intensity (e.g., baseline + 1.5 mmol/L, 4mmol/L, or Dmax), closer to the MLSS (Caen et al., 2024; Faude et al., 2009; Pogliaghi et al., 2023; Smith & Jones, 2001). Because of the high number of existing parameters to distinguish intensity sustainable over a long time and those that lead to exhaustion in a relatively short time (i.e., < ~30 min), the concept of maximal metabolic steady state is gaining ground to bring together all these parameters (Iannetta et al., 2022; Mattioni Maturana et al., 2016; Van Rassel et al., 2023). The physiological responses above MMSS are characterized by a gradual drift in multiple variables: blood lactate rises continuously, minute ventilation and respiratory frequency increase, and heart rate drifts upward (Jones et al., 2019). However, the behavior of $\dot{V}O_2$ at intensities slightly above MMSS is more complex. At an intensity just above MMSS, a discernible $\dot{V}O_2$ slow component is present, but the athlete may not necessarily reach $\dot{V}O_2$max before exhaustion (De Lucas et al., 2013; Sawyer et al., 2012). Nevertheless, such intensity is unsustainable in the long term, typically leading to exhaustion within approximately 30 minutes, depending on the method used for the determination (De Lucas et al., 2013).

Parallel to the development of lactate-based thresholds, researchers observed that changes in ventilation during incremental exercise closely mirror the underlying metabolic events (Beaver et al., 1986; Wasserman et al., 1973). The first ventilatory threshold (VT_1) corresponds to the first disproportionate increase in ventilation relative to $\dot{V}O_2$, without a concomitant increase in ventilatory equivalent for CO_2 ($\dot{V}E/\dot{V}CO_2$). This threshold is closely associated with the LT_1 and marks the transition from the moderate to the heavy intensity domain (Korzeniewski & Rossiter, 2022). The second ventilatory threshold (VT_2), also known as the respiratory compensation point (RCP), occurs at higher intensities and is characterized by a systematic increase in both $\dot{V}E/\dot{V}O_2$ and $\dot{V}E/\dot{V}CO_2$, reflecting the end of isocapnic buffering and the need to compensate for metabolic acidosis through increased ventilation (Keir et al., 2022). VT_2 corresponds approximately to the maximal lactate steady state or the critical power and demarcates the boundary between the heavy and severe intensity domains (Galán-Rioja et al., 2020; Iannetta et al., 2022). Notably, in recent years, it has become more common to see the use of the term “gas exchange threshold” to identify VT_1 and “respiratory compensation point” to indicate VT_2 (Bourgois et al., 2023; Di Gennaro et al., 2025b; Iannetta et al., 2019; Iannetta et al., 2022; Keir et al., 2022).

2.2.1. Defining the Intensity Domains

Depending on whether the exercise is performed at intensities below the first or second threshold, different acute physiological responses can be observed. Below the first threshold, i.e., in the moderate intensity domain, $\dot{V}O_2$ reaches a steady state within 2-3 minutes, blood lactate remains at or below baseline levels, and exercise can be sustained for hours without progressive fatigue (Korzeniewski & Rossiter, 2022). At the cellular level, PCr stores are partially depleted but rapidly recover to a new steady state, and there is no progressive accumulation of metabolites (Korzeniewski & Rossiter, 2022).

Above the first but below the second threshold, that is, within the so-called heavy domain, $\dot{V}O_2$ still reaches a steady state, but this is delayed and requires 10-15 minutes to stabilize (Ansdell et al., 2020; Iannetta et al., 2022; Korzeniewski & Rossiter, 2022). A sustained but stable elevation in blood lactate

is observed; PCr levels are reduced; and, as for $\dot{V}O_2$, a slow component can be observed (Cannon et al., 2014). It has been shown that this intensity can be sustained for approximately 1 to 2 hours, depending on where in the heavy domain the athlete is exercising (Clark et al., 2018, 2019).

Then, above the second threshold, we encounter the severe domain. In this domain, $\dot{V}O_2$ does not reach a steady state; instead, it continues to rise until $\dot{V}O_2$max is attained, and exhaustion, soon or later, will occur (Alexander et al., 2019; Korzeniewski & Rossiter, 2022; Ozkaya et al., 2025). Blood lactate accumulates progressively, PCr is driven to very low levels, and intramuscular concentrations of H^+, ADP, and Pi increase continuously (Korzeniewski & Rossiter, 2022). Exercise tolerance in this domain is finite and inversely related to intensity, typically ranging from 2 to 30 minutes, and is strongly related to anaerobic capacity (Di Gennaro et al., 2025; Green et al., 1996; Muniz-Pumares et al., 2017). The curvature constant (W') of the critical power model is specifically designed to quantify the finite work capacity available in this domain (Drake et al., 2024), especially if the critical power is used as the boundary between heavy and severe domains.

When exercise intensity is such that exhaustion occurs before $\dot{V}O_2$max is attained, typically within less than two minutes, the resulting physiological responses differ markedly from those observed in the severe domain (Gastin, 2001). Consequently, these efforts have been classified within the so-called extreme intensity domain (Alexander et al., 2019; Iannetta et al., 2022; Ozkaya et al., 2025). In this domain, the energy demand is met predominantly (>50%) by phosphagen and glycolytic pathways (Peker et al., 2026). Neuromuscular factors become primary determinants of performance, and the metabolic perturbations are so rapid that traditional steady-state concepts no longer apply (Gastin, 2001). This explains why the standard two-parameter critical power model is not the optimal tool for describing the power-duration relationship in this domain unless the three-parameter model is employed (Vinetti et al., 2019). Conversely, this context provides a rationale for the application of the more time-efficient anaerobic power reserve model to predict performance, a point that will be developed further in the following sections (Bundle et al., 2006; Weyand et al., 2006).

2.2.2. Training Adaptations Across Intensity Domains

While no endurance athlete trains exclusively within a single intensity domain - most follow a specific intensity distribution (e.g., pyramidal, polarized, or threshold-based) - understanding the acute and chronic effects of each domain remains fundamental (Seiler & Kjerland, 2006; Stöggl & Sperlich, 2015). The physiological distinctions between intensity domains have profound implications for the adaptive responses elicited by chronic training (Filipas et al., 2022; Seiler & Kjerland, 2006; Treff et al., 2017). Higher training volume in the moderate domain is associated with greater mitochondrial content, oxidative enzyme activity (Lundby & Jacobs, 2016; Mølmen et al., 2024), and cardiac dimension (Dausin et al., 2026), with a concurrent improvement in the fractional utilization of $\dot{V}O_2$max, and $\dot{V}O_2$max itself (Filipas et al., 2022; Ingham et al., 2008; Toubekis et al., 2011). Training in the heavy domain also improves lactate kinetics and the ability to sustain higher absolute intensities, effectively shifting the lactate and ventilatory thresholds rightward (Londeree, 1997; Oliveira et al., 2025; Panascì et al., 2025; Stöggl & Sperlich, 2015), likely necessitating less training duration compared to the moderate domain (Storoschuk et al., 2025). The severe domain, where high-intensity interval training (HIIT) is typically performed, appears particularly useful for eliciting rapid and substantial increases in $\dot{V}O_2$max (Rosenblat et al., 2025). Recent work by Inglis et al. (2025) demonstrated that six weeks of severe-intensity interval training produced the largest improvements in maximal oxygen uptake, driven predominantly by central adaptations such as increased cardiac output and blood volume. Notably, extreme-domain sprint interval training (SIT), while also effective, did not produce the same magnitude of $\dot{V}O_2$max improvement as severe-intensity HIIT (Inglis et al., 2025), highlighting the distinct stimuli provided by each domain

However, comparing the effects of different domains remains methodologically challenging. A fundamental issue is the lack of a standardized framework to determine how much low-intensity volume can be considered equivalent, in terms of overall training load, to a given amount of high-

intensity work (Kowalski et al., 2025). Without such equivalence, isolating the specific contribution of each domain to long-term adaptations becomes inherently difficult.

2.3. The Challenge of Quantifying Anaerobic Metabolism

While several robust whole-body parameters exist to define the oxidative characteristics of an athlete, the same cannot be said for anaerobic metabolism. This raised considerable debate within the literature regarding which parameter should, or even could, serve as a valid proxy for an athlete's anaerobic capabilities (Sandford et al., 2021). This uncertainty about a valid and repeatable metric for assessing anaerobic characteristics inevitably complicates the evaluation of training interventions and our understanding of exercise tolerance within the severe and extreme intensity domains. If we cannot reliably measure a given parameter, how can we determine whether a training-induced improvement has actually occurred or what determines physical exhaustion? The fundamental issue is methodological: aerobic metabolism can be measured indirectly via pulmonary gas exchange, as it consumes oxygen and produces carbon dioxide; in contrast, anaerobic metabolism does not release a uniquely modified byproduct to the "external world" that can be easily quantified at the whole-body level. In fact, the contribution of anaerobic metabolism can only be inferred from bio/physiological parameters such as the blood lactate or the accumulated oxygen deficit, or through mathematical constructs such as the anaerobic power reserve or the critical power models (Green, 1995). These constructs, grounded in the physiological significance of their underlying parameters, offer a framework for delineating an athlete's profile along the aerobic-anaerobic continuum. However, the present section will focus specifically on the physiological parameters traditionally employed to define anaerobic characteristics and quantify anaerobic contribution during exercise. The mathematical models developed for this purpose, including the APR, will be discussed in detail in the following sections. The next paragraph will enumerate and explain the rationale behind several less-invasive markers and methods to characterize anaerobic capacity and power, moving beyond traditional muscle biopsies.

2.3.1. Traditional Non-Invasive Markers: Blood Lactate and its Caveats

- **Blood Lactate concentration**

One of the earliest and most widely utilized markers for inferring anaerobic glycolytic activity is blood lactate concentration. Lactate is the metabolic end-product of anaerobic glycolysis, whereby glucose is metabolized to pyruvate and subsequently reduced to lactate, regenerating NAD^+ to sustain ATP production (Brooks 2018). Numerous studies have observed correlations between peak (end-exercise) (Cheetham et al., 1986; Galvan-Alvarez et al., 2024) blood lactate levels and intramuscular markers such as PFK activity and glycogen content (Esbjörnsson-Liljedahl et al., 1999). However, these correlations are imperfect and exhibit wide confidence intervals. This is because lactate, once produced within a muscle cell, undergoes complex dynamics before appearing in the bloodstream, where it is measured. Lactate can be shuttled from its primary site of production (e.g., type IIa muscle fibers) into adjacent oxidative fibers (e.g., type I), where it is used as an oxidative substrate (Gladden 2004). Furthermore, a portion of blood lactate is cleared by the liver and kidneys via gluconeogenesis (the Cori cycle). This intricate interplay of production, clearance, and exchange means that blood lactate accumulation reflects the net outcome of these processes rather than the gross rate of anaerobic glycolysis (Gladden 2004; Brooks 2018). Consequently, the use of blood lactate as a yardstick of anaerobic lactic metabolism has been partially reconsidered. To conclude, even though lactate is often called the end-product of glycolysis, it has to be noted that it's not a waste product; indeed, it is used by various organs and tissues as an energetic provider as well as a biochemical signal for starting metabolic adjustments and/or adaptations (Gladden 2004; Brooks 2018).

- **Maximal rate of lactate accumulation ($\dot{v}La_{max}$)**

$\dot{v}La_{max}$ is defined as the maximal rate of lactate accumulation in the blood, representing the first derivative of blood lactate accumulation over time. In other words, it quantifies the maximum velocity at which lactate appears in the bloodstream. Noteworthy, this definition refers not to the total

production of lactate within the muscle, but specifically to its net appearance in the circulation. These are two distinct, albeit related, physiological phenomena. As previously stated, a significant portion of lactate produced is metabolized within the muscle itself or in other tissues before it can be measured in the blood (Gladden 2004; Brooks 2018).

A recent systematic review and prior methodological studies have extensively addressed $\dot{v}La_{max}$, aiming to identify the optimal testing protocol for eliciting its highest value (e.g., comparing 10, 15, or 30-second sprints) and to determine its construct validity as a surrogate for cellular glycolytic power (Langley et al., 2024; Meixner et al., 2024; Langley et al., 2025; Wackerhage et al., 2025). While not the central focus of this work, the most relevant aspects of $\dot{v}La_{max}$ are outlined here. The parameter is fundamentally calculated as:

$$\dot{v}La_{max} = (La_{max} - La_{bl}) / T_{exercise}$$

where La_{max} is the post-exercise peak blood lactate concentration (mmol/L), La_{bl} is the basal lactate concentration, and $T_{exercise}$ is the exercise duration in seconds. However, this basic model has been refined to account for two confounding factors:

- The initial, brief period of alactic energy contribution from ATP-PCr reserves, during which glycolytic flux is not yet maximal.
- The concurrent, although minor, aerobic energy contribution even during supramaximal exercise.

To incorporate these factors, more rigorous models have been proposed. The formula that implements the alactic phase is:

$$\dot{v}La_{max} = (La_{max} - La_{bl}) / (T_{exercise} - T_{alac})$$

where T_{alac} is the duration of the alactic phase, typically estimated to be the first 3.5 s (Langley et al. 2025).

Yang et al. (2023) proposed a further modified formula to take into account the oxidative contribution during sprint exercise. Basically, by calculating the energy contribution in percentage of alactic, lactic, and aerobic pathways using, respectively: i) the fast component of the excess post-exercise $\dot{V}O_2$ (EPOC), ii) the post-exercise peak lactate converted to L of O_2 equivalent (Zagatto et al., 2015), and iii) the O_2 consumed during the sprint, then using the percentage of oxidative pathways contribution converted into seconds to be subtracted from the exercise time, as in the following formula:

$$\dot{v}La_{max} = (La_{max} - La_{bl}) / (T_{exercise} - T_{alac} - T_{oxi})$$

where T_{oxi} is the time for which aerobic metabolism covers energy provision.

Regarding exercise modality and duration, a recent study by Langley et al. (2024) compared $\dot{v}La_{max}$ derived from 10, 15, and 30-second sprint tests. They observed that while the absolute peak lactate concentration was highest following the 30-second sprint, the most elevated $\dot{v}La_{max}$ value was obtained from the 10-second test. Furthermore, a comparative study by Quittmann et al. (2021) observed that $\dot{v}La_{max}$ is modality-specific, and in triathletes, the running $\dot{v}La_{max}$ often yields higher values than the cycling one. This is potentially due to the larger active muscle mass involved in sprint running and differences in muscle recruitment patterns, leading to a greater total and simultaneous glycolytic contribution to the energy demand. However, this higher $\dot{v}La_{max}$ is in contrast to the typical higher lactate contribution in cycling with respect to running when considering moderate and heavy domain intensity exercise (Capostagno and Bosch 2010). Therefore, further studies are needed to clarify this point.

2.3.2. Maximal Accumulated Oxygen Deficit: Concept and Methodological Constraints

For the past 50 years, the maximal accumulated oxygen deficit (MAOD) has been one of the most prominent methods for the *in vivo* assessment of anaerobic capacity in exercise physiology (Noordhof et al., 2010). A fundamental distinction must be made from the outset: MAOD is a measure of anaerobic *capacity*, representing a finite amount of energy that can be attained using anaerobic energy

sources. This contrasts with parameters like $\dot{v}La_{max}$, which provide a coarse estimate of anaerobic *power*, or the instantaneous rate of anaerobic energy usage.

The theoretical foundation of the MAOD is twofold:

1. The O_2-power output (near) linear relationship during the steady state exercise**:** during exercise intensities below the maximal metabolic steady state (MMSS), the contribution of anaerobic metabolism to the total energy yield is negligible (typically <5%) (Medbo et al. 1988). Consequently, a near-perfect linear relationship exists between the external power output (PO in Watts) and the measured oxygen uptake ($\dot{V}O_2$). By establishing this linear regression through multiple, submaximal constant work rate tests, typically spanning from ~30% of $\dot{V}O_2max$ up to the MMSS, each lasting approximately 10 minutes, one can extrapolate the predicted oxygen demand ($\dot{V}O_{2demand}$) for any given supramaximal power output.

2. The finite nature of anaerobic energy sources: during exercise above the MMSS, the aerobic system cannot meet the total energy demand. The difference between the extrapolated $\dot{V}O_{2demand}$ and the actual $\dot{V}O_2$ measured during exhaustive exercise represents the "oxygen deficit," which is quantitatively supplied by anaerobic metabolism. The MAOD is the maximal value of this accumulated deficit, achieved at the point of volitional exhaustion. This exhaustion is intended to coincide with the depletion of the free ATP-PCr, and the concurrent accumulation of intolerable levels of Pi and H^+ (also, but not only, due to anaerobic glycolysis) (Medbo 1993).

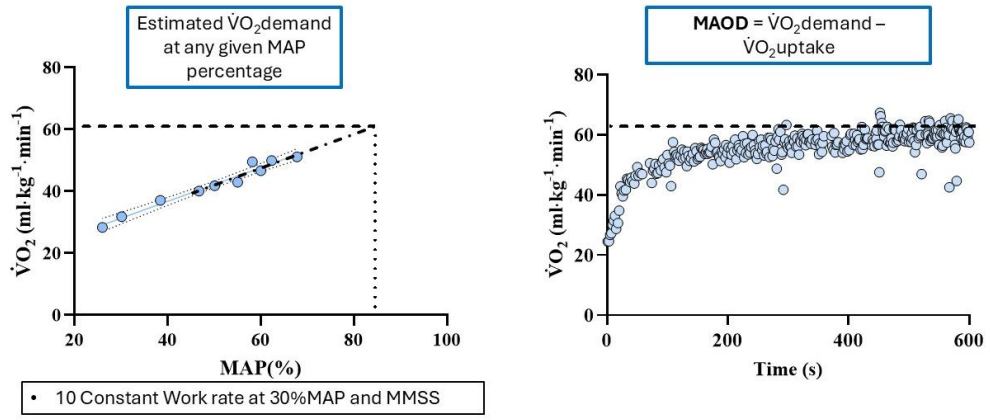

Figure 3. *Schematic representation of the MAOD method. Abbreviation: MAP, Maximal Aerobic Power; $\dot{V}O_2$, Oxygen uptake; MMSS, maximal metabolic steady state*.

Several studies highlighted how the MAOD is positively associated with exercise tolerance during time to exhaustion trials (T_{lim}) (within the severe intensity domain) when intensity is based on the maximal aerobic power (MAP) (Medbo 1993; Blondel et al., 2001; Redkva et al., 2018; Di Gennaro et al., 2025b). The reason relies on the anaerobic contribution needed to sustain intensity above the $\dot{V}O_2$max. In fact, above this intensity, such "energy reserve" gets depleted, joule by joule, at a rate proportional to the power sustained above the power associated with $\dot{V}O_2$max. Additionally, Medbo and Tabata (1993) observed that MAOD was closely related to the ATP turnover from anaerobic enzymes at the muscular level in active participants. However, it should also be noted that Green et al. (1996), who performed the same study in elite endurance cyclists, did not obtain the same results, likely indicating that the MAOD method is valid only when inter-individual differences among participants are high.

2.4. Summary of Method to Assess Anaerobic Characteristics: a Limited Usefulness

The aforementioned methodologies are widely employed in scientific literature to characterize the individual's anaerobic features. They are useful in investigating the physiological adaptations and/or adjustments that occur in response to exercise. Nonetheless, it is crucial to acknowledge their inherent limitations. These include the previously discussed approximation of cellular metabolic events. In fact,

the blood lactate measurements can only roughly estimate what occurs at the cellular level, and this constraint obviously extends to the $\dot{v}La_{max}$ value. Then, all the highlighted methodologies and parameters (MAOD included) are inherently post-exercise analytical tools. They provide invaluable insight into the physiological determinants of performance, but their retrospective nature precludes real-time application. This is because their application during exercise is constrained by methodological and practical limitations like (1) the requirement for specialized and costly laboratory equipment, such as an ergospirometer for the MAOD assessment; (2) the invasive nature of measurements like blood sampling for lactate analysis, which, despite its growing popularity in field testing, still presents logistical and compliance challenges; (3) inherent uncertainties in measurement validity and the often-weak translational link between the physiological parameter analyzed (e.g., blood lactate accumulation) and actual anaerobic performance (e.g., sprint capability); and (4) the computationally complex procedures required for parameter estimation, which hinder their integration into routine, real-world training environments. This has motivated the search for simpler, more accessible, and ready-to-use proxies for anaerobic features. The APR model, which requires only a sprint test and an incremental test, or the critical power model, which requires 2 or more time-trials (or time-to-exhaustion trials), represents such an attempt to capture the essence of anaerobic characteristics in a manner that is both physiologically meaningful and practically feasible.

SECTION 3

3. MODELLING PERFORMANCE AND EXERCISE TOLERANCE: THE POWER-DURATION RELATIONSHIPS

To address the gap in the applicability of physiological parameters within training practice, numerous options have been proposed. Among them, the relationship between power output and exercise duration is one of the most applied in both scientific and practice settings. Numerous power-duration models exist, but for the aim of this work, we will focus on the two most prevalent in contemporary literature: the critical power model and the anaerobic power reserve model. Therefore, in the following paragraph, I will discuss the rationale and the main application context of these two models, providing pros and cons of both and future perspectives.

3.1. The Critical Power Model: Principles, Physiological Correlates, and Predictive Utility

Among the mathematical models to describe the power-duration relationship, the critical power (CP) model is one of the most used (Jones et al., 2010). Originally derived from a curvilinear function (Hill 1925) (**Figure 4**), the CP model can be reformulated into a linear equation describing either the relationship between exercise time and work performed or between power output and the inverse of exercise time (Jones et al. 2010). In **Figure 5,** the mathematical behavior of these functions is illustrated with real data. The CP model relies on two key assumptions: (i) the existence of an asymptote, aka "critical power", the theoretical maximal intensity that can be sustained indefinitely, and (ii) the constancy of work performed above CP (W'), often interpreted as a measure of anaerobic capacity, and assumed to be the same for any supra-CP intensity.

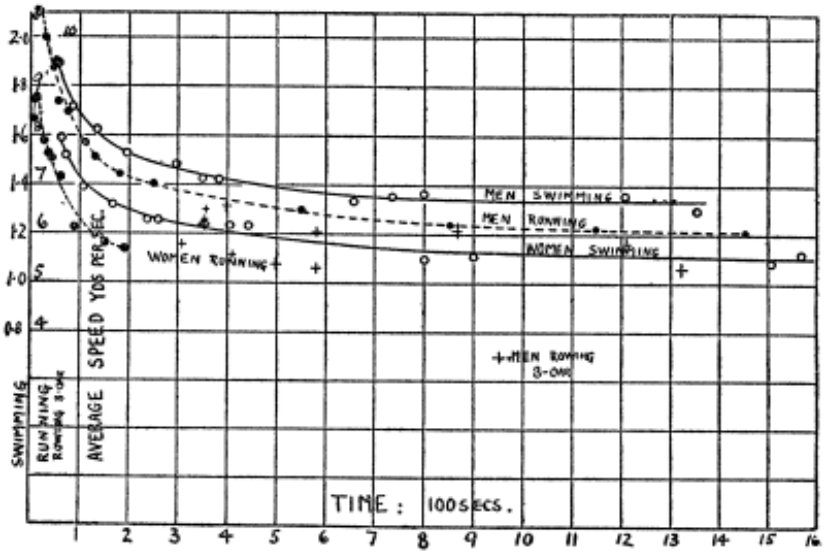

FIG. 1.—World's records for men and women swimming and running: average speed in yards per second against time in seconds. *Note.*—The scale for swimming is five times as great as for running. The observations for men rowing an eight-oar boat are on the same scale as running and are referred to later in the text.

Figure 4. *This figure is taken from the milestone paper of the Nobel laureate A.V. Hill in 1993 titled: The Physiological Basis of Athletic Records. This figure shows the hyperbolic relationship between speed and duration in different sports.*

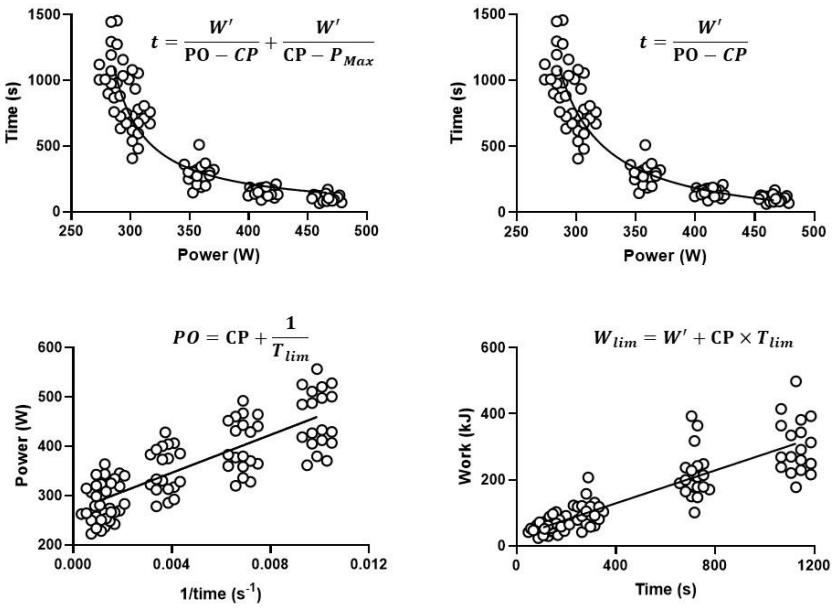

Figure 5. *This figure illustrates the relationship between power output and exercise tolerance along with their respective equations. The two graphs at the top present the critical power hyperbolic model for the 3- (on the left) and 2- (on the right) parameter models. The two graphs at the bottom represent the same data, expressed in terms of power output and the inverse of time (on the left) and in terms of work and time (on the right).*

3.1.1. Methodological Considerations in Determining CP and W'

It is important to note that while different models estimate related values for CP and *W'*, they can still present considerable disparity, with differences in CP even exceeding 30 Watts (Mattioni Maturana et al., 2018). This difference is similarly propagated to the estimates of *W'*. A further factor influencing these estimations is the duration of T_{lim} used. Protocols utilizing shorter T_{lim} durations (e.g., 2-12 minutes) typically yield higher CP estimates and lower *W'*, whereas protocols incorporating longer T_{lim} durations (e.g., 2-20 minutes) produce lower CP and higher *W'* (Mattioni Maturana et al., 2018).

A fundamental concept is that CP is intended to represent the highest metabolic rate that can be sustained indefinitely. In exercise physiology, this role was historically occupied by the maximal lactate steady state (MLSS), defined as the highest intensity at which blood lactate concentration stabilizes, typically when lactate increases by no more than 1.0 $mmol \cdot L^{-1}$ between the 10th and 30th minute of constant-work-rate exercise (Iannetta et al., 2022). Initial observations indicated that CP often overestimated MLSS, leading some authors to question the physiological validity of CP and propose it as a mathematical artifact rather than a true physiological threshold (Bergstrom et al. 2013a; Dotan 2022b; Drake et al., 2024). Subsequent methodological investigations identified two key sources for this discrepancy: first, the traditional use of shorter T_{lim} durations (up to ~12 minutes) was insufficient, and incorporating longer trials (15-20 minutes) yielded CP values more closely aligned with MLSS (Iannetta et al., 2022). Second, the standard MLSS criterion of a 1.0 $mmol \cdot L^{-1}$ of increment is arbitrary; in fact, if a less strict stability criterion of $\leq$ 1.5 $mmol \cdot L^{-1}$ increase, or if an increase $\leq$ 0.5 $mmol \cdot L^{-1}$ but during the last 10 minutes of the CWR is applied, the agreement between CP and MLSS becomes nearly perfect (Iannetta et al., 2022). Consequently, the current consensus recommends using T_{lim} durations spanning a range of 2 to 20 minutes to define a truly sustainable CP (Iannetta et al., 2022; Mattioni Maturana et al., 2016, 2018).

From a physiological perspective, CP has been validated as a marker of aerobic power, both from a local and whole-body point of view (Chorley et al., 2020; Drake et al., 2024; Vanhatalo et al., 2016).

Strong correlations have been consistently demonstrated between CP and $\dot{V}O_2$max, MLSS, and the respiratory compensation point (Chorley et al., 2020; Drake et al., 2024). Furthermore, CP is highly correlated with intramuscular determinants of oxidative function, such as citrate synthase activity, mitochondrial density and cristae surface area, capillary density, and *in vivo* measurements of muscle oxidative capacity via near-infrared spectroscopy (Caswell et al., 2024; Galán-Rioja et al., 2020; Vanhatalo et al., 2016).

3.1.2. The 2-Parameter vs. 3-Parameter Models: Addressing the Extreme-Intensity Domain

W′ (together with CP) has proven to be a significant predictor of performance in both T_{lim} and time-trials. However, its predictive efficacy is most robust for exercise durations falling within approximately 2 to 40 minutes. This leaves the so-called extreme-intensity domain (Hill et al., 2002) (< 2 min) and prolonged endurance performances outside its capacity. Studies have demonstrated that the classical two-parameter CP model systematically overestimates performance in short-duration activities (Nicolò et al., 2017; Galán-Rioja et al., 2020). This limitation likely stems from the mathematical structure of the two-parameter hyperbolic model:

$$PO = \frac{W'}{t} + CP$$

Hence, if we solve the equation for time tending to zero:

$$\lim_{t \to 0} PO = \frac{W'}{t} + CP$$

$$\lim_{t \to 0} PO = \infty + CP$$

$$\lim_{t \to 0} PO = \infty$$

where *PO* is power output, and *t* is time. As time (t) approaches zero, the term $\frac{W\prime}{t}$ approaches infinity, leading to a predicted power output that is physiologically implausible (**Figure 6**). This reflects the

model's failure to account for an athlete's finite maximum instantaneous power. To resolve this, a three-parameter CP model was introduced by Morton (1996), which incorporates a theoretical maximum power (P_{max}). This model is formulated as:

$$T_{lim} = \frac{W'}{PO - CP} + k$$

Where:

$$k = \frac{W'}{CP - P_{max}}$$

Therefore, we can rewrite the model as follows:

$$T_{lim} = \frac{W'}{PO - CP} + \frac{W'}{CP - P_{max}}$$

The addition of the third parameter, *k*, shifts the x-axis, enhancing the model's goodness-of-fit compared to the 2-parameter model. This adjustment establishes a finite, and (sometimes) plausible, ceiling for power output as *t* approaches zero. It is crucial to note, however, that this estimated P_{max} remains a theoretical ceiling derived from the model's mathematical structure. As emphasized by Morton himself, this value often lacks validity unless the model is built using (also) trials near the athlete's actual instantaneous peak power output (PPO) (Vinetti et al., 2019). Therefore, if very short trials are not included in the model, the derived P_{max} likely won't be truthful. A schematic representation of the 2 and 3-parameter model is illustrated in **Figure 6**.

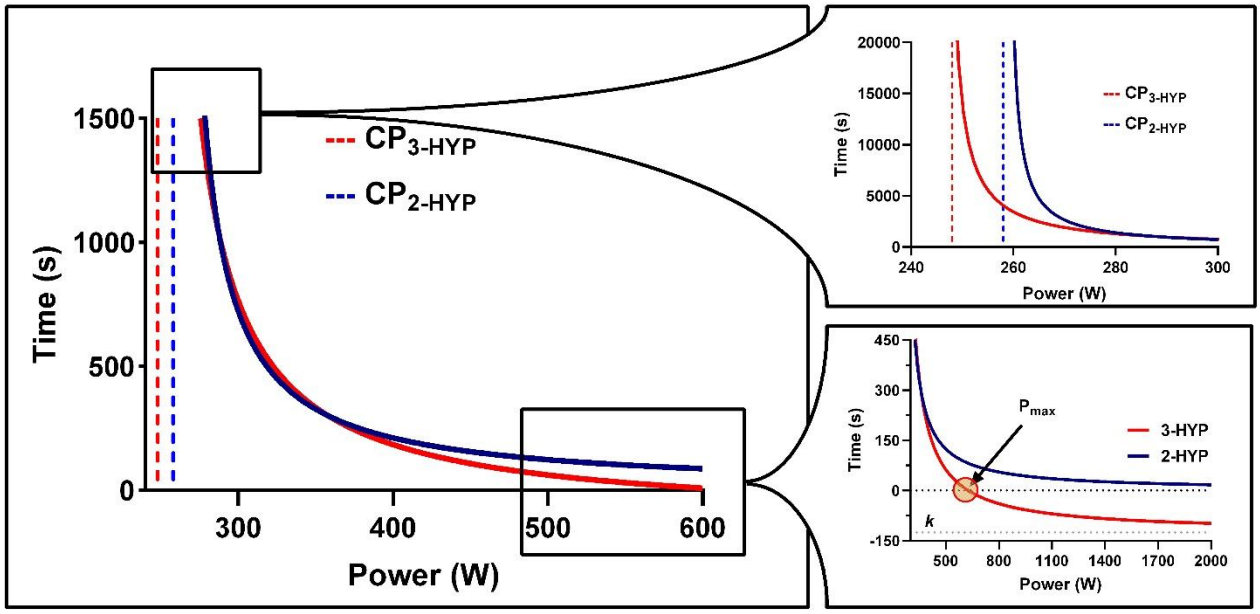

Figure 6*. The graph on the left represents the behavior of the power-duration relationship of the 2 (blue) and 3 (red)-parameter hyperbolic models. The panel at the top right is a zoom of what happens when the power output approaches critical power. The panel at the bottom right is a zoom of the models' behavior within the extreme intensity domain.*

A critical caveat for both models is that accurate prediction requires the model to be built on performance tests that are physiologically similar to the event being predicted. In essence, the most reliable way to predict a specific performance is to use a model parameterized with data from a performance of a comparable nature and duration.

In fact, some authors observed that the CP model built with the traditional approach, namely only with severe-intensity trial (intensity above CP but with T_{lim} longer than 2 min), is unable to predict performance within the extreme intensity domain accurately (Alexander et al., 2019; Pallarés et al., 2020; Drake et al., 2024). These results are also supported by data still not published by our research group, which show that even when the three-parameter model is adopted, the inclusion of a single trial within the extreme-intensity domain (together with the other trials in the severe domain) in the model is not enough to produce an accurate estimation of the work above CP ($W_{>CP}$) at those intensities, hence compromising performance predictions. Our unpublished findings suggest a fundamental reason for this inaccuracy: the introduction of the third parameter (P_{max}), while theoretically necessary to impose

an upper ceiling to the PO, concurrently increases the model's estimation uncertainty. When calibrated with a dataset clustered within the severe-intensity domain (e.g., mean T_{lim} ~9 minutes), the model must derive three independent parameters (CP, *W'*, P_{max}) from a limited number of performances. Consequently, without a sufficient number of extreme-intensity trials to properly "anchor" the P_{max}, the model can produce an excellent goodness-of-fit that is mathematically robust yet physiologically unrealistic. Notwithstanding, when several trilas within the extreme intensity domain are included, the 3-parameter model improves its prediction accuracy in the full spectrum of exercise intensity (Vinetti et al. 2019).

While clear relationships between CP and other aerobic markers from both a whole-body (e.g., $\dot{V}O_2max$) or a local perspective (e.g., mitochondrial content, citrate synthase activity) have been observed, the physiological basis of *W'* remains debated (Hill and Smith 1993; Green et al., 1994; Muniz-Pumares et al., 2017). Muniz-Pumarez et al. (2017) observed that the accumulated oxygen deficit during an exhaustive trial of approximately 3 minutes was positively correlated (r = 0.6) with $W_{>CP}$ during that trial. In line, Green et al. (1994) reported that *W'* estimated from the work–time model was correlated with intramuscular ATP changes and end-exercise blood lactate concentrations (Green et al., 1994). Yet, no associations were found with glycogen phosphorylase and phosphofructokinase activity, muscle buffering capacity (Green et al., 1994), peak blood lactate or end-exercise pH (Jenkins and Quigley 1991).

Additionally, if *W'* truly represents the finite source of anaerobic capacity, this means that under hypoxic conditions, there would be no effect, but this is not always true. In fact, has been highlighted that the percentage change of CP under hypoxia [(CP- $CP_{hypoxia}$)/CP] is strongly related to the percentage change in *W'* in hypoxia compared to normoxia (Parker Simpson et al., 2014). Valli et al. (2011) also observed that at high altitude (~5000m), even *W'* utilization is compromised; hence suggesting how the O_2 availability contributes to the full depletion of *W'*(Valli et al., 2011; Parker Simpson et al., 2014). A last but not least limitation of the CP model is the high cost in terms of time

and energy that athletes, coaches, and researchers have to spend on delineating the power-duration relationship to estimate supra-CP capabilities of an athlete. This limitation has once again driven researchers to seek faster and simpler solutions for profiling athletes' physiological characteristics and for monitoring whether training interventions are yielding the desired adaptations. Indeed, the past two decades have witnessed a significant rise in two "novel profiling approaches": i) the so-called 3-minute all-out test and ii) the anaerobic power reserve construct. Both of these frameworks promise to describe an athlete's physiological profile through methodologies that are considerably less time-consuming than conventional multi-trial time-to-exhaustion protocols. In the following paragraph, the physiological and methodological rationale underlying the 3-minute all-out test will be described, while the subsequent section will introduce the anaerobic power reserve construct.

3.1.3. The 3-Minute All-Out Test: A Practical Alternative with Limitations

The determination of CP and *W'*, through traditional methods, is prohibitively time-consuming. A solution to this problem has been the development and validation of a single-visit, 3-minute all-out test to estimate CP and *W'*, which has since been applied across various cyclic sports, including cycling, running, swimming, and rowing (Cheng et al., 2012; Mitchell et al., 2018; Saari et al., 2019; Wright, 2017). This test protocol essentially extends the concept of the 30-second Wingate test (Bar-Or 1987; Beneke et al., 2002) to a three-minute duration. The power output or velocity, plotted against time, typically displays an exponential decay that stabilizes over the final 30 seconds of the effort (Vanhatalo et al., 2007). This exponential decay in power output is typically interpreted as reflecting the progressive depletion of anaerobic energy reserves, which cannot be sufficiently replenished during the effort (Vanhatalo et al., 2007). Consequently, once anaerobic energy provision can no longer meet the total energy demand, the oxidative metabolism becomes the primary source sustaining the required energy production, albeit at a relatively lower exercise intensity (Vanhatalo et al., 2007). This stable end-value is termed as end-power (EP) or end-speed (ES) and has been validated with a high level of agreement with the CP derived from the traditional multi-trial 2-parameter model (Cheng et al., 2012;

Mitchell et al., 2018; Saari et al., 2019). However, it is important to note that the intensity corresponding to EP has been contested as true physiological steady state, in fact the sustainability of this pace resamble the same of the CP obtained with the traditional approach, that is, physiological parameters such as blood lactate, minute ventilation, and respiratory frequency fail to stabilize, and exhaustion typically occurs within 15-30 minutes (McClave et al., 2011; Bergstrom et al., 2013a; Bergstrom et al., 2013b). The work performed above this EP, often referred to as work above end power (WEP), shows only a moderate relationship with the traditionally derived *W′*. The two measures, despite not differing significantly, exhibit wide limits of agreement (LoA), complicating direct comparisons and leading to the convention of referring to the test-derived metric as WEP rather than *W′*. Furthermore, performance predictions based on the 3-minute all-out test parameters have been shown to overestimate a cyclist's performance in time-trial tasks (Nicolò et al., 2017).

A representation of the power/speed decay over time during the 3-minute all-out test is shown in **Figure 7.**

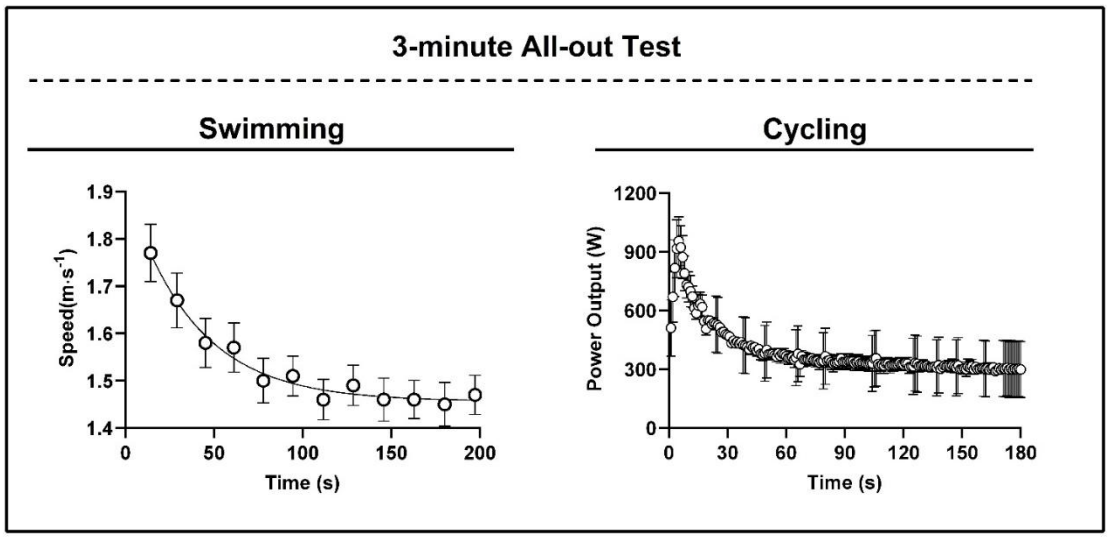

Figure 7*. This figure shows on the left the speed decay during the modified 3-minute all-out test. As can be seen from the x-axis, this test is typically longer than 3 minutes. This is because, in swimming, when using a 25-m pool, a 12 x 25m all-out test is required to estimate critical speed and finite distance capacity (for further details, please refer to (Mitchell et al., 2018)). On the right is presented the power output decay during the 3-minute all-out on a cycle-ergometer.*

Therefore, while the 3-minute all-out test provides a robust estimate of CP, its estimation of WEP is less reliable, and its performance predictions can be inflated. Furthermore, several practical limitations hinder its widespread application. These include the necessity for prior familiarization due to the test's extreme and unfamiliar nature, and its inability to provide any information on metabolic thresholds below CP, such as the gas exchange threshold. In cycling, an additional complication is the requirement to determine the optimal ergometer resistance to elicit a valid force-velocity-power profile (Dicks et al., 2016). Consequently, despite its practical appeal as a single-test solution, these complexities limit its applicability. In contrast, the traditional standard incremental exercise test with gas exchange analysis, whether using long stages (>3 minutes) or shorter stages with subsequent correction for mean response time (Iannetta et al., 2022), provides a comprehensive physiological profile, including both ventilatory thresholds and $\dot{V}O_2max$. The only issue for the incremental test is that it does not provide direct information about an athlete's anaerobic characteristics. Some authors have been attempting to use the work performed above RCP during incremental testing as a surrogate for W' (Iannetta et al., 2023) but this approach has only recently been studied, and further research is needed to assess its actual validity.

This uncertainty in measuring anaerobic characteristics has probably driven the development of the anaerobic power reserve construct (Sandford et al., 2021). In fact, this model only requires an additional sprint test over the traditional incremental test. The sprint can range in duration from 6 to 30 seconds; importantly, if the goal is simply to determine instantaneous peak power output (PPO), a 6-second sprint is sufficient (Douglas et al., 2021). Such brief sprints induce minimal fatigue and can even be performed immediately before an incremental test within a single testing session, thereby optimizing the efficiency of athlete assessment (Thron et al., 2023). Hence, the APR model integrates these two readily obtainable parameters into a single, intuitive framework. The following section will trace the origins of this concept and detail its physiological and methodological foundations.

SECTION 4

4. THE ANAEROBIC POWER RESERVE CONCEPT: BRIDGING THE GAP

Based on the preceding sections, it is evident that while the CP model provides an elegant framework for understanding endurance performance across durations, its practical application in real-world settings is challenging. Furthermore, the model offers limited insight into the physiological profile below CP and cannot accurately describe performance within the extreme-intensity domain unless it is parameterized with multiple exhaustive trials performed at or near those intensities. To address these limitations, researchers have explored simpler methods that align more closely with existing testing practices commonly employed by both scientists and coaches. The resulting construct is known as the anaerobic power or speed reserve (APR/ASR, according to the exercise involved).

4.1. Conceptual Basis of Anaerobic Power Reserve

The anaerobic power reserve framework utilizes familiar incremental exercise tests and standard sprint assessments to more straightforwardly delineate the physiological profile of an athlete. Regardless of the exercise involved, the definition is the same; in fact, it is calculated as the difference between an athlete's maximal sprint power output (PPO) and the PO associated with maximal oxygen uptake (MAP). Consequently, the APR model offers a pragmatic synthesis, requiring only two assessments that are already staples of routine testing in both laboratory and field settings: a maximal incremental exercise test and an all-out sprint test. This efficient framework enables the simultaneous identification of key parameters from a single testing battery. The incremental test provides the first and second ventilatory or lactate thresholds, $\dot{V}O_2max$, and MAP. The sprint test, in turn, yields the PPO. A schematic representation of the APR constructs is given in **Figure 8.**

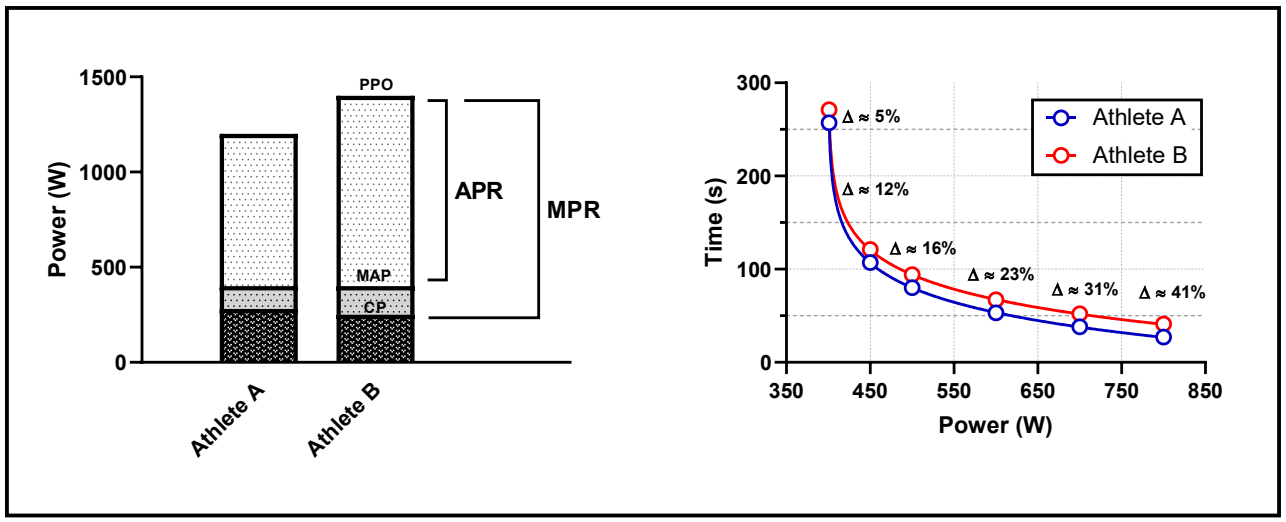

***Figura 8**. This figure shows the anaerobic power reserve (APR) of 2 athletes. MPR is the difference between the peak power output (PPO) and critical power (CP); MAP is the maximal aerobic power. On the right panel is shown the predicted time to exhaustion at the same absolute intensities for the two athletes.*

4.1.1. Early Evidence of APR as an Exercise Performance Determinant

The foundational studies on this concept originate from researchers such as Weyand and Bundle (2005; 2006; 2012). Weyand and Bundle focused on understanding the relationship between exercise tolerance and intensity for durations under three minutes, and on how the decay in power/velocity was linked to the rate of force production (Weyand and Bundle 2005; Bundle et al., 2006; Weyand et al., 2006; Bundle and Weyand 2012). They observed that power output decayed exponentially with time; for durations < 3 minutes the trend is explained by the following model:

$$PO = MAP + APR \cdot e^{-k \cdot t}$$

Where k describes the decrement in PO over time (t).

Notably, Weyand et al. (2006) observed that within a group of active students, the decay constant (k) was remarkably similar across individuals. This suggested that once the maximal aerobic and anaerobic power were known (i.e., MAP and PPO), performance could be accurately predicted (Weyand et al., 2006). They also noted that the rate of velocity decline during running was much slower than the power decline in cycling. They attributed this difference to the duty factor, that is, the proportion of time

during a stride when force is applied to the ground. When both running and cycling data were normalized for this duty factor, the rates of performance decay were comparable (**Figure 9,** taken from Weyand et al., 2006)**.**

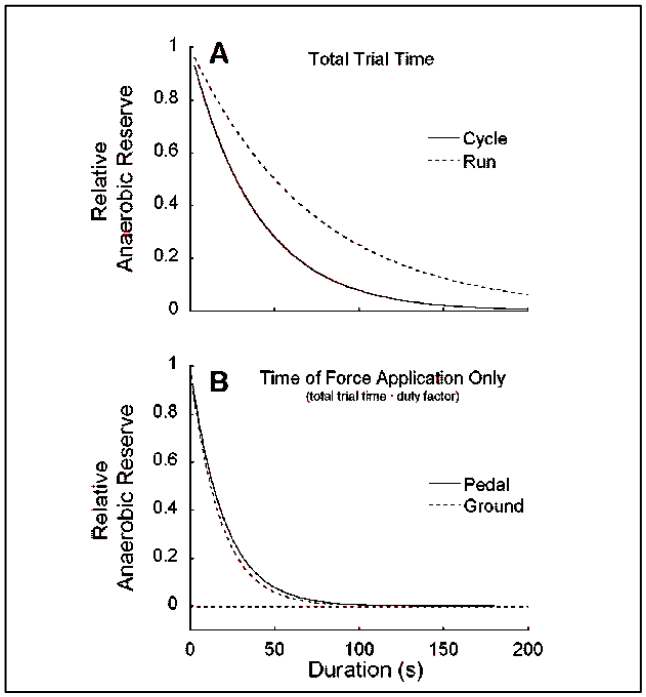

Figura 9. *Cycling power outputs and running speeds, expressed as fractions of the respective anaerobic reserves vs. total sprint trial duration (A) and vs. the cumulative time of force application to the pedals and ground (B).*

The theoretical basis of this model rests on the principle that exercise intensity, when expressed relative to an athlete's maximal aerobic capacity, fails to account for individual differences in anaerobic characteristics. Consequently, a given supramaximal intensity will elicit the utilization of a specific fraction of an athlete's total anaerobic power reserve (%APR). The model posits that the magnitude of this fraction dictates the resultant physiological stress; a higher %APR utilization is directly associated with greater metabolic perturbation and a shorter time to exhaustion. This principle is illustrated by the hypothetical scenarios of the two athletes presented in **Figure 8**. Both possess a similar aerobic capacity, but markedly different maximal power reserves (APR). Therefore, when exercising at the same absolute intensity (e.g., 150% of MAP), the athlete with a smaller MPR will be utilizing a significantly larger proportion of their anaerobic reserve and hence will reach exhaustion earlier. This

disparity theoretically results in a faster accumulation of fatigue-limiting metabolites and reduced exercise tolerance.

In this context, two critical factors should be noted: i) the performance advantage conferred by a high APR is most evident at extremely high intensities, likely within the extreme-intensity domain; ii) the underlying rationale of the APR construct parallels that of the CP model. Specifically, if we consider APR as analogous to *W'* and MAP as analogous to CP, then exercising at a fixed percentage of MAP would lead the participant with a smaller APR to reach exhaustion earlier than an athlete with a greater APR. However, while this principle holds for the CP model across the entire severe-intensity domain, the same does not appear for the APR construct, which seems to retain its validity only at intensities exceeding MAP (Di Gennaro et al., 2025b). Moreover, the empirical support for this theoretical framework is nuanced, and a critical examination of the evidence will be detailed in the following section.

The foundational work on this model and its associated decay constant (*k*) by Weyand et al. (2006) was based on data collected from a relatively small sample of healthy, active university students - a non-specialized population. Consequently, the application of their generalized *k* value to other athletic populations has been questioned (Sanders and Heijboer 2019). This prompted more recent investigations to evaluate whether using this traditional *k* is a proper choice to characterize power decline during 3-minute efforts in professional male cyclists. Sanders and Heijboer (2019) found that the traditional *k* led to less accurate results compared to an individualized *k*. However, obtaining an individualized *k* necessitates the athlete perform multiple maximal efforts of different durations, a procedure similar in demand to establishing the CP model, thereby undermining the proposed practicality and ease-of-use of the APR approach. Finally, Sanders and Heijboer (2019) utilized the maximal mean power over 3 minutes as the lower boundary for the APR construct. While this allowed for the prediction of power outputs above that intensity, it ignored all physiological events occurring below it, thereby limiting the model's overall utility.

The APR construct also seems to play a role in the fatigue developed during repeated sprint exercise (Mendez-Villanueva et al., 2008). This kind of exercis require to performe repeated bout in an all-out manner with limited recovery. It has been observed that individuals with a higher APR exhibit greater fatigability during repeated sprint tests, likely indicating that a greater reliance on anaerobic metabolism leads to more pronounced fatigue-related symptoms (Mendez-Villanueva et al., 2008). Mechanistically, Mendez-Villeneuva et al. (2008) reported that the decline in mean power output across successive bouts was correlated with a progressive reduction in EMG activity, suggesting that neuromuscular factors contribute to the greater fatigability observed in individuals with a higher anaerobic reserve.

4.1.2. $V\dot{O}_2$max and Threshold-Based Methods to Prescribe Exercise Intensity

For decades, endurance exercise intensity was commonly prescribed as a fixed percentage of maximal physiological anchors such as $\dot{V}O_2$max or Hrmax (Iannetta et al., 2020a). However, this approach has been increasingly challenged, as it fails to account for the inter-individual variability in the position of metabolic thresholds (Iannetta et al., 2020a). demonstrated that exercising at the same percentage of $\dot{V}O_2$max elicits heterogeneous metabolic responses across individuals, while (Jamnick et al., 2020) concluded that percentage-based methods have "little merit" for eliciting consistent physiological perturbations. The underlying issue is that the GET/LT_1 and MMSS occur at widely different percentages of $\dot{V}O_2$max between individuals (Iannetta et al., 2020a). Consequently, the same relative intensity may place one athlete in the heavy domain and another in the severe domain, leading to divergent acute responses and chronic adaptations (Inglis et al., 2025). This evidence has driven the shift toward threshold-based prescription anchored to individual lactate or ventilatory thresholds, which provide a physiologically meaningful foundation for delineating intensity domains and normalizing the metabolic stress across individuals.

While the shift to threshold-based prescription has improved the consistency of exercise intensity in the moderate and heavy domains, normalising intensity within the severe domain, where high-intensity

interval training (HIIT) is typically performed, remains a significant challenge (Boullosa & Abreu, 2014). The seminal work of Lansley et al. (2011) “introduced” the delta (Δ) method, prescribing intensity as a percentage of the difference between the gas exchange threshold and $\dot{V}O_2max$, aiming to account for individual variability in aerobic fitness. However, recent evidence has critically re-evaluated this approach. (Bossi et al., 2023; Bossi et al., 2024a and 2024b) demonstrated that, despite accounting for these thresholds, the Δ method still fails to normalise acute physiological responses and exercise tolerance, with inter-individual variability accounting for more than ~50% of the total variance in time to exhaustion during HIIT sessions prescribed at 70%Δ. These findings align with earlier observations by Mann et al. (2013), who emphasised that even sophisticated relative intensity methods cannot fully account for the multifactorial variability in physiological responses.

The challenge of normalising intensity is further illustrated by studies directly comparing traditional and threshold-based HIIT prescriptions. Meyler et al. (2023) compared the variability in exercise tolerance and physiological responses when HIIT was prescribed using fixed percentages of $\dot{V}O_2max$ versus intensities anchored to individual physiological thresholds (critical power and gas exchange threshold). They observed that while threshold-based prescription tended to reduce inter-individual variability in some responses, such as blood lactate and *W’* depletion, considerable heterogeneity persisted, indicating that even individually anchored intensities cannot fully eliminate differences in exercise tolerance Meyler et al. (2023). For these reasons, several attempts have been made in order to find a way to normalize exercise intensity within the exercise intensity domain and also for interval training.

One of the pioneering studies investigating the ASR/APR construct for intensity within the severe intensity domain came from Blondel et al. (2001). They evaluated the relationship between both the classic ASR and a novel approach using critical speed (CS) as the lower boundary, termed maximal speed reserve (MSR; MPR in cycling), and exercise tolerance (time to exhaustion) across intensities ranging from approximately 1 to 15 minutes. They found that both models could predict time to

exhaustion, with ASR being more accurate for supramaximal intensities and MSR for maximal and submaximal ones. However, their sample included a small (n = 10) sample of healthy students, and the standard error of the estimates remained high, suggesting caution in directly applying ASR/MSR to prescribe T_{lim} to reduce inter-individual variability. In fact, on average, the estimated standard error was between 10 and 20% of the T_{lim}.

4.1.3. The Use of APR to Prescribe Supra-$V\dot{O}_2$max Intensities

These preliminary results fostered the hypothesis that prescribing supra-$\dot{V}O_2$max exercise intensity using the APR framework could individualize training and reduce the heterogeneity in time to exhaustion and physiological responses traditionally observed with MAP-based prescriptions. An early attempt to apply this approach was made by Barnett et al. (1996). They tested both the classical APR and a novel metric called the "mean anaerobic scope" (MAS), defined as the difference between mean anaerobic power from a Wingate test and MAP. They prescribed a T_{lim} (mean duration ~2 minutes) using a percentage of MAP, a percentage of APR, and a percentage of MAS. Surprisingly, the APR method did not reduce T_{lim} variability compared to the MAP method. However, the MAS method significantly reduced variability among participants. The authors proposed that this was likely due to the MAS's closer reliance on glycolytic pathways, which are more determinant for efforts like those performed at T_{lim} intensities. On the contrary, PPO is bioenergetically farther from that effort, given that it is more influenced by phosphagen stores and neural recruitment, making it a poorer index of anaerobic capacity.

Notwithstanding, some attempts have been recently made to determine if APR/ASR could be useful for prescribing intensity during HIIT. For example, Julio et al. (2019) prescribed a short-format HIIT protocol (15 s work/15 s rest) to endurance runners and rugby players using both the traditional aerobic method (110% of Maximal Aerobic Speed, MAS) and intensities relative to ASR set at 25% and 50% of the reserve. Their findings appeared promising, indicating that the coefficients of variation for time to exhaustion were substantially lower under the ASR conditions (25% and 15%) compared to the

MAS condition (58%). This would suggest that prescribing intensity relative to ASR may reduce inter-individual variability in exercise tolerance, providing a more homogenous stimulus across the participants and likely more consistent adaptations. However, the absolute exercise intensities, and consequently the resulting T_{lim}, were not matched between conditions. The T_{lim} in the MAS condition was nearly double that in the 25% ASR condition and triple that of the 50% ASR. It is well-established that longer T_{lim} tests inherently exhibit greater variability and lower reliability compared to shorter, more intense efforts. Therefore, while the results initially support the ASR construct, the non-matched design raises the possibility that the observed reduction in variability may be attributable to the higher relative intensity and shorter test duration per se, rather than a fundamental advantage of the ASR-based prescription method (Hinckson & Hopkins, 2005; Laursen et al., 2007).

A comparable study was conducted with Australian rules football players, employing a matched-intensity design (Collison et al., 2021). The protocol consisted of 15-second work intervals interspersed with 15-second rest, prescribed at 120% of MAS and 20% of ASR. The authors reported no statistically significant reduction in Tlim variability with ASR-based prescription compared to the MAS method, although a small, non-significant effect in favor of ASR was noted ($p = 0.09$). However, a critical methodological consideration is that MAS in this study was not derived from a traditional incremental test but estimated from a 2 km time trial. This suboptimal determination of the aerobic boundary may have influenced the observed variability, potentially confounding the interpretation of the ASR model's efficacy. While one might argue that any error in MAS estimation would inherently propagate into the ASR calculation, evidence suggests that the variance in the ASR construct is predominantly determined by maximal sprinting speed (MSS), not MAS. Therefore, the primary driver of the reserve, and likely its prescription utility, may be relatively independent of minor inaccuracies in defining its lower aerobic boundary.

Similarly, Bok et al. (2023) carried out a study where 17 male physical education students performed 10 minutes of short HIIT, curiously, the same protocol as the previous two studies, 15s On/15s Off at

110% MAS and 15 or 25% of ASR. Unfortunately, despite the closeness, the intensity between 110% MAS and 15% ASR was significantly different; hence, as well as for Julio et al. (2019), any actual role of the prescription method is conflated by the different intensities. In this study, while the coefficient of variation of $V\dot{O}_2$, heart rate (HR), lactate, and rate of perceived exertion (RPE) were numerically lower under ASR prescription, none reached statistical significance. The sole significant finding was a lower variability in RPE during the 25% ASR condition. However, during this trial, only 6 out of 17 participants were able to conclude the 10 minutes of HIIT; therefore, such results are likely explained by the convergence of the RPE values toward the maximum (i.e., 20 arbitrary units [a.u.]), reflecting a ceiling effect rather than a clear superior normalization of exercise intensity by the ASR framework.

A further recent investigation in middle-distance trained adolescents examined whether physiological and perceptual responses to a short HIIT format (15-s work/15-s rest at 110% MAS) were associated with the athlete's ASR (Thron et al., 2025). The underlying hypothesis posited that when intensity is prescribed relative to an aerobic index (e.g., %MAS), athletes with a higher ASR would utilize a smaller fraction of their reserve, thereby incurring lower metabolic and neuromuscular stress. The authors were able to reject the null hypothesis. While no associations were found between the ASR profile and cardiorespiratory responses ($\dot{V}O_2$, HR), significant relationships emerged with markers of muscle stress and function. Specifically, ASR was negatively correlated with the post-exercise accumulation of creatine kinase (a biomarker of muscle damage) and positively correlated with the maintenance of countermovement jump height and the reactive strength index. These results indicate that athletes with a lower ASR experienced greater muscular disruption and a more pronounced decline in muscle contractile velocity characteristics following the HIIT session, supporting the proposed model of differential strain based on an individual's anaerobic reserve.

4.1.4. Issues Standardising the Anaerobic Power Reserve

As previously described, the APR, or its speed-based counterpart (e.g., ASR), is defined as the difference between an athlete's maximal instantaneous peak power output (typically the highest 1-

second value) and their maximal aerobic power (MAP), i.e., the power associated with maximal oxygen uptake (Sandford et al., 2021). Consequently, the APR value is inherently influenced by the methodological approaches used to determine both of its constituent parameters. While no studies have directly investigated how manipulating these methodologies affects the resulting APR, a substantial body of literature has examined how testing protocols individually influence the determination of peak power output (PPO) and MAP (Adami et al., 2013; Astorino, 2009; Astorino et al., 2005; Beltrami et al., 2014; Douglas et al., 2021; Driss & Vandewalle, 2013; Luttikholt & Jones, 2022; Niemeyer et al., 2020; Thron et al., 2023).

In cycling, PPO is dependent on pedalling cadence (Driss & Vandewalle, 2013) and can be described by the force-velocity relationship; this follows an inverted parabolic function: maximal force is exerted at very low or zero velocities, while maximal velocity is achieved under low force conditions. The optimal force-velocity combination is that which maximises power output, given that power is the product of force and velocity (PO = F × V) (Driss & Vandewalle, 2013). In cycling, linear velocity is replaced by angular velocity (i.e., pedalling cadence), while force is that applied directly to the pedals. Each athlete possesses an individual force-velocity profile; therefore, obtaining a true maximal power output requires mapping this profile to ensure that the observed peak represents the athlete's genuine maximum.

Recently, Wahl & Ji (2026) applied isokinetic sprints, where the athlete sprints at a fixed cadence for PPO determination and the subsequent APR calculation. While this method eliminates cadence dependence, prescribing the same cadence (e.g., 120 rpm) to all athletes may place some closer to their optimal cadence and others far from it. Moreover, isokinetic sprints do not exist in real-world cycling practice. Therefore, this approach could be more prone to underestimating the real APR and to having a lower ecological validity (Kordi et al., 2019; Nascimento et al., 2024). Another factor requiring standardisation is whether sprint tests are performed seated or standing, as standing allows greater recruitment of trunk and upper limb musculature, likely enabling higher power outputs (Driss &

Vandewalle, 2013). These considerations necessitate clear standardisation of whether the sprint is seated or standing, as well as the specific gear ratio (for field testing) or resistance setting (for ergometer testing). Additional factors influencing PPO determination include familiarisation, warm-up duration and intensity, passive rest preceding the test, saddle height, and whether the start is from a stationary position or a flying start, as the acceleration phase from a standstill may induce premature fatigue before peak power is reached (Driss & Vandewalle, 2013).

In running-based contexts, where maximal sprinting speed (MSS) is measured rather than power, analogous factors apply, with the notable exception that step frequency cannot be altered except by changing terrain gradient (Thron et al., 2023). However, the method of measurement itself significantly influences MSS values. Photocells, laser guns, and video analysis are not interchangeable, and the resulting speeds may differ substantially depending on the technology and protocol employed (Thron et al., 2023).

The second determinant of APR is MAP, whose protocol dependency is a well-recognised and extensively debated phenomenon in the scientific literature, alongside ongoing debates regarding its physiological validity (Astorino, 2009; Luttikholt & Jones, 2022; Niemeyer et al., 2020; Stromme et al., 1977). One of the earliest definitions, proposed by Di Prampero et al. (1986), described the power associated with $\dot{V}O_2max$ as the ratio between $\dot{V}O_2max$ and the energy cost of locomotion. Subsequently, MAP has also been defined using an approach that relies on the entire power-$\dot{V}O_2$ relationship established from multiple submaximal constant work-rate bouts. While conceptually similar to “Di Prampero's method”, this approach differs in that it is based on the full power-$\dot{V}O_2$ relationship rather than a single measurement of cycling economy (Barnett et al., 1996; Blondel et al., 2001). Another common method defines MAP as the final power output achieved during a maximal incremental test, either at the point where $\dot{V}O_2$ reaches a plateau or, more simply, as the last completed stage (Luttikholt & Jones, 2022; Sandford et al., 2021; Thron et al., 2023).

Crucially, all these methods are protocol-dependent, and a consistent pattern emerges: shorter stage durations or constant work-rate bouts yield higher MAP values (Luttikholt & Jones, 2022; Sandford et al., 2021; Thron et al., 2023). This occurs because, at intensities within the heavy domain, shorter durations do not allow sufficient time for $\dot{V}O_2$ to reach its full steady-state value, resulting in a lower measured oxygen uptake for a given power output, and consequently a lower calculated energy cost of locomotion or a shallower PO-$\dot{V}O_2$ slope. The same principle applies to the peak power attained during incremental test: shorter stages yield higher final power outputs (Adami et al., 2013; Andersson et al., 2022; Medbø & Welde, 2022; Luttikholt & Jones, 2022). The plateau-based method is further complicated by the fact that a true $\dot{V}O_2$ plateau is not always observable; in some cases, a continued rise in $\dot{V}O_2$ is evident even at exhaustion (Niemeyer et al., 2020; Poole & Jones, 2017) . Other methods used to define the lower limit of the APR/ASR have included average power over 3 minutes (Sanders & Heijboer, 2019), average speed over 2 km (Collison et al., 2021), critical speeds in swimming and running (Dalamitros et al., 2015; Kramer et al., 2021; Lanzarini et al., 2025), or critical power in cycling (Di Gennaro et al., 2025b).

The fact that both components of the APR construct are highly protocol-dependent underscores an urgent need for standardisation. Without a clear understanding of how variations in testing methodologies influence the resulting APR value, and consequently its relationship with actual performance and its underlying physiological rationale, the utility of the model as a valid and reproducible tool for athlete profiling and exercise prescription remains limited.

4.2. Synthesis of Evidence and Unresolved Questions

It is now clear that the overall picture regarding the true efficacy of the APR model for homogenizing physiological responses and exercise tolerance is more complex than it initially appeared. Resolving these uncertainties is of dual importance. Firstly, a validated APR construct could provide a practical,

intuitive, and accurate field-based tool for profiling an athlete's physiological characteristics and subsequently individualizing exercise intensity. Secondly, in a research context, it could help reduce the notorious and often prohibitive variability inherent to exhaustive exercise trials (T_{lim}), which frequently complicates their interpretation and utility. The APR concept was mainly applied in running for the practical scope to reduce inter-individual variability in physiological and perceptual responses (Blondel et al., 2001; Julio et al., 2020; Bok et al., 2023). Achieving homogeneous acute responses is a critical first step toward eliciting more consistent chronic adaptations. However, the application of this construct remains underexplored in several critical contexts. First, its utility for prescribing the HIIT commonly performed on cycle ergometers, a mainstay for both athletic and clinical populations due to its safety and practicality, has not been rigorously tested. Second, and of particular interest, are other cyclic sports where performance is mainly reliant on anaerobic metabolism. Among these, competitive swimming stands out. Short-distance events (e.g., 50m, 100m) are among the most popular and are characterized by a dominant contribution from glycolytic and phosphagen systems. Therefore, a model designed to quantify the reserve capacity between maximal sprint and aerobic power could serve as an ideal tool for athlete profiling and potentially predicting performance.

SECTION 5

5. AIM OF THE THESIS

Considering the current knowledge and research gap in the literature, the primary aim of this work was to evaluate whether the APR model can explain variance in performance and physiological responses during severe-intensity cycling, tested in both constant-work and interval-based modalities. The second aim was to extend the current knowledge by assessing the applicability of the ASR model to swimming performance, utilizing a practical 3-minute all-out test. Given that these studies employ anaerobic constructs to explain variance in predominantly aerobic performance, a third, supporting aim was to examine how the estimation of *W'*, a parameter often interpreted as anaerobic capacity, is influenced by both the mathematical model chosen and the intensity of the trials used for its determination. This study will help to better understand the inherent complexity of quantifying anaerobic capacity and will allow for better contextualisation of the results of studies focused on APR.

STUDY 1

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Maximal Aerobic Power and Anaerobic Power Reserves to Prescribe Cycling Interval Training Sessions

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Abstract

BACKGROUND: Prescribing interval training intensity can be challenging due to individual variations in physiological capacity. Traditional methods often rely on maximal aerobic power (MAP), but this may not fully capture the characteristics of different athletes. This study aimed to investigate whether alternative methods, such as anaerobic power reserve (APR) and glycolytic power reserve (GPR), could provide more individualized high-intensity interval training (HIIT) prescriptions.

METHODS: Twelve trained cyclists completed a cardiopulmonary test and Wingate test to determine MAP, APR, and GPR (mean power output during the Wingate test minus MAP). Subsequently, participants performed in a randomized order, three HIIT until-exhaustion sessions with 60-s of work and 60-s of active rest, based on APR ($HIIT_{APR}$: MAP+10% APR), GPR ($HIIT_{GPR}$: MAP+20%GPR) and MAP ($HIIT_{MAP}$: 120%MAP), respectively. Inter-individual variability in time to exhaustion, heart rate, oxygen uptake, and lactate was calculated as the root mean square of residuals and as coefficients of variation (CV).

RESULTS: Although no significant differences in inter-individual variability were observed across the three prescription methods for any of the physiological and perceptual variables ($P > 0.2$), $HIIT_{MAP}$ leads to lower inter-individual variability in time to exhaustion (CV = 21%) compared to $HIIT_{APR}$ (CV = 35%) and $HIIT_{GPR}$ (CV = 45%).

CONCLUSIONS: HIIT based on APR and GPR does not reduce inter-individual variability in physiological responses and tolerance compared with MAP-based prescription. This suggests that both APR and GPR fail to accurately differentiate between the aerobic and anaerobic characteristics of an athlete, hindering the normalization of exercise responses during HIIT.

Keywords:

exercise prescription; aerobic fitness; anaerobic fitness; acute physiological responses.

Introduction

High-intensity interval training (HIIT) is a common practice employed by endurance athletes to spend a certain amount of time near the maximal oxygen uptake (Buchheit and Laursen, 2013a). Compelling evidence suggests that when applied judiciously, these stimuli promote performance improvements and related physiological adaptations (Buchheit and Laursen, 2013a). The primary determinants of HIIT effectiveness are exercise intensity, duration, and recovery intervals (Buchheit and Laursen 2013a). These parameters should be tailored to the athlete's profile to maximize training stimulus (Laursen and Buchheit, 2019; Sandford et al., 2021; Du and Tao, 2023). Among these variables, intensity is arguably the most influential factor, and in cycling, it is typically based on the highest power attained during a maximal incremental test, often referred to as the Maximal Aerobic Power (MAP) (Barnett et al., 1996; Julio et al., 2020; Sandford et al., 2021). However, using this marker to prescribe HIIT has been observed to produce large inter-individual variability in exercise tolerance and physiological responses (Blondel et al., 2001; Julio et al., 2020; Bok et al., 2023). Previous research suggested this variability may be attributed to MAP's inability to fully capture the physiological profile of an athlete. In fact, two athletes may exhibit an identical MAP while concurrently demonstrating significant disparities in anaerobic characteristics such as the instantaneous peak power output (PPO) (Buchheit and Laursen, 2013a; Buchheit and Laursen, 2013b; Sandford et al., 2021). Therefore, adhering to the common practice of prescribing HIIT based on MAP will lead to performing the

exercise at a different percentage of anaerobic power reserve (APR), namely the difference between PPO and MAP, which will result in different exercise tolerance and physiological demands (Buchheit and Laursen, 2013a).

While this consequence has been theorized, limited research has directly investigated this concept. Some studies have investigated the role of the corresponding running parameter, the anaerobic speed reserve (ASR), in exercise prescription and its associated physiological responses (Julio et al., 2020; Collison et al., 2021; Bok et al., 2023). Notwithstanding, to our knowledge, no studies have examined the effects of prescribing HIIT relative to APR in cycling. Early research on ASR observed a positive correlation between this parameter and time to exhaustion (TTE) during continuous running tests conducted at intensities ranging from 90% to 140% of the maximal aerobic speed (MAS) (Blondel et al. 2001). Further investigations observed that during short-format HIIT, athletes with higher ASR showed lower metabolic stress (lower oxygen uptake and blood lactate accumulation) compared to those with lower ASR, thus highlighting the importance of the individual's locomotor profile (MAS + maximal sprinting speed) (Buchheit et al., 2012). More recently, a reduced variability of blood lactate accumulation and TTE during short-format HIIT(15-s work- 15-s rest) was found when exercise intensity accounted for ASR compared to MAS-based prescription (Julio et al., 2020).

Solely one study explored the application of APR to prescribe intensity during a constant supra-MAP cycling test, reporting that such exercise intensity prescription did not reduce the heterogeneity in TTE compared to MAP-based conditions (Barnett et al., 1996). Nevertheless, when intensity was based on "mean anaerobic scope", namely the difference between the mean anaerobic power achieved during the Wingate test and the MAP, there was a reduction in TTE variances. Thus, authors hypothesized that the "mean anaerobic scope" (renamed by us, as glycolytic power reserve (GPR)), which places greater emphasis on glycolytic pathways, could better account for the variability (and energy systems contribution) in TTE (Barnett et al., 1996). A more careful intensity individualization leads to more homogenous training adaptations; therefore, taking into account parameters like APR or GPR could

help to optimize performance outcomes (Du and Tao, 2023; Wang and Zhao, 2023). However, while ASR/APR appears to be useful in prescribing running (Bok et al., 2023), rowing (Wang and Zhao, 2023), and kayaking HIIT (Du and Tao, 2023), no evidence suggests that prescribing longer-format HIIT in cycling based on APR or GPR reduces inter-individual variability in exercise tolerance and physiological responses. Thus, this study aimed to compare the inter-individual variability in exercise tolerance, physiological, and perceptual responses during HIIT sessions prescribed using MAP, APR, and GPR.

Materials and methods

Participants

An *a priori* sample size calculation was conducted using the inter-individual variability in TTE quantified as the root mean square of the mean residual as one of our primary outcome measures (Collison et al. 2021). The sample size was estimated using the G*Power software (version 3.1 software, Heinrich-Heine-Universität) by applying repeated measures (within) analysis of variance (F-test) with a significance level of 0.05, and a statistical power of 80% (Faul et al., 2007). A single group with three measurements, a correlation among those of 0.80, and an effect size of 0.25 were assumed. This computation generated a desired sample size of at least 12 participants.

Twelve trained male cyclists from a local cycling team were enrolled. The inclusion criteria were: 1) maximal aerobic power of at least 3.5 $W \cdot kg^{-1}$; 2) five years of experience in cycling and a minimum of 6 hours of training per week; 3) absence of known disease or exercise limitations. Exclusion criteria were muscle or joint injuries, orthopedic problems, or any other contraindication within 6 months before the commencement of the study. The subjects were instructed to avoid any consumption of food and caffeine, 3 hours and 5 hours respectively before testing, and to refrain from strenuous exercise at least 36h before testing. They were also recommended not to change their dietary habits during the intervention period. Before the experimental protocol, all participants were fully informed about the

study aims and procedure and gave their written informed consent to participate in the investigation. The study was conducted following the ethical standards of the Declaration of Helsinki, and the protocol was approved by the Ethics Committee of Università degli Studi di Milano (n°49-2020). Participants' characteristics are presented in **Table Ia**.

Table Ia. Participants' anthropometric and physiological characteristics.

		CPET		Wingate Test		Anaerobic Reserve	
Age (y)	31 ± 6	MAP (W)	348 ± 38	PPO (W)	1035 ± 184	APR (W)	687 ± 157
Height (cm)	183 ± 6	VO_{2peak}($ml \cdot kg^{-1} \cdot min^{-1}$)	57.1 ± 8.6	MPO (W)	729 ± 95	GPR (W)	381 ± 72
Mass (kg)	77 ± 8						

***Abbreviations**: CPET, cardiopulmonary exercise test; PPO, anaerobic peak power output; MPO, anaerobic mean power output; MAP, maximal aerobic power; APR, anaerobic power reserve; GPR, glycolytic power reserve; W, Watt.*

Design

A randomized crossover design was carried out. Participants were asked to attend the laboratory on 5 different days and all experimental sessions were conducted at the same time of the day to avoid the influence of circadian rhythms. An electromagnetically braked cycle ergometer (Excalibur Sport, Lode, Groningen, Netherlands) was used, adjusted to individual anthropometry and positioning habits. During the first day, subjects underwent anthropometric measurements and were familiarized with all procedures. Then, on the second day, participants underwent a Wingate Test and a cardiopulmonary exercise test (CPET). Finally, during the following three experimental sessions, participants performed three HIITs, respectively based on the MAP ($HIIT_{MAP}$), APR ($HIIT_{APR}$), and GPR ($HIIT_{GPR}$) in a randomized and counterbalanced order.

Wingate Test

During the second visit, participants performed a warm-up consisting of 5 minutes of pedalling at 100 W, followed by three 5-s sprints at 70-90% of maximal effort, interspersed with 90-s of low-resistance pedalling. Then, 3 min of passive rest were given. The Wingate Test encompassed a 30-s maximal effort sprint initiated from a flying start, with participants maintaining a seated position on the saddle

(Bar-Or 1987). Participants were verbally encouraged to exert as much effort as possible throughout the 30-s test. The ergometer linear factor was set in order to enable athletes to achieve the optimal cadence and thus the maximal instantaneous power (PPO). Mean power output (MPO) was computed as 30-s average power output during the Wingate test.

Cardiopulmonary Exercise Test

One hour after the Wingate test, a CPET until exhaustion was conducted to determine peak oxygen uptake (VO_{2peak}) and MAP. Considering previous investigations, a crossover effect of the Wingate test on CPET has been deemed unlikely (Constantini et al., 2014; Stong et al., 2021). The CPET started with 10 min at 100 W, with subsequent increases of 25 W every 3 min until volitional exhaustion. MAP was calculated as follows: $MAP = W_{final} + (t / 180 \times 25)$ (Bentley and McNaughton, 2003). Where W_{final} was the power output of the last completed stage, and t indicated the duration of the stage that remained uncompleted due to exhaustion (Bentley and McNaughton, 2003). The selection of this CPET protocol, particularly the stage length, was primarily driven by three considerations. First, in well-trained endurance athletes, the MAP derived from this specific protocol has been shown to exhibit a strong correlation ($r = 0.94$) with endurance performance measured by a 90-minute time trial (Bentley and McNaughton, 2003). Secondly, the use of an incremental protocol with 3-minute stages is designed to reduce the relative contribution of anaerobic metabolism, thereby allowing for a more accurate assessment of aerobic capacity (Luttikholt and Jones, 2022). Finally, this protocol produces VO_{2peak} values comparable to those obtained with traditional steeper incremental tests in well-trained athletes (Bentley and McNaughton 2003). Additionally, this test has been recommended by several authors to delineate performance levels (De Pauw et al., 2013; Luttikholt and Jones, 2022). During the test, participants were instructed to pedal at a self-selected rate between 70 and 90 rpm while remaining seated. Pulmonary gas exchange was continuously collected breath-by-breath during the CPET using a wearable metabolic cart (Cosmed K5, Rome, Italy) and a mask (Hans Rudolph, INC. Shawnee, KS,

USA) with a dead space of 30 mL. The turbine was calibrated with a 3-L syringe (Cosmed, Rome, Italy). Gas analyzers were calibrated with ambient air and gas mixture (20.0% O2 and 4.0% CO2).

VO_{2peak} was taken as the highest 30-s mean and was considered to be reached when participants met the following criteria: heart rate (Hr) value >95% of predicted maximal (210– (0.65 × age), a respiratory exchange ratio >1.05, and a lactate >8.0 $mmol \cdot L^{-1}$ (Poole and Jones, 2017). Peak heart rate (Hr_{peak}) was considered the highest Hr reached. Furthermore, at the end of the test, participants' rating of perceived exertion (RPE) was assessed using Borg's scale (6-20). Two minutes after the end of CPET, blood lactate was measured ([La]) with earlobe blood samples (5 μL) using a Lactate Pro 2 (Arkray, Kyoto, Japan). APR was calculated as the difference between PPO and MAP, while the GPR was calculated as the MPO minus MAP.

High-Intensity Interval Training Sessions

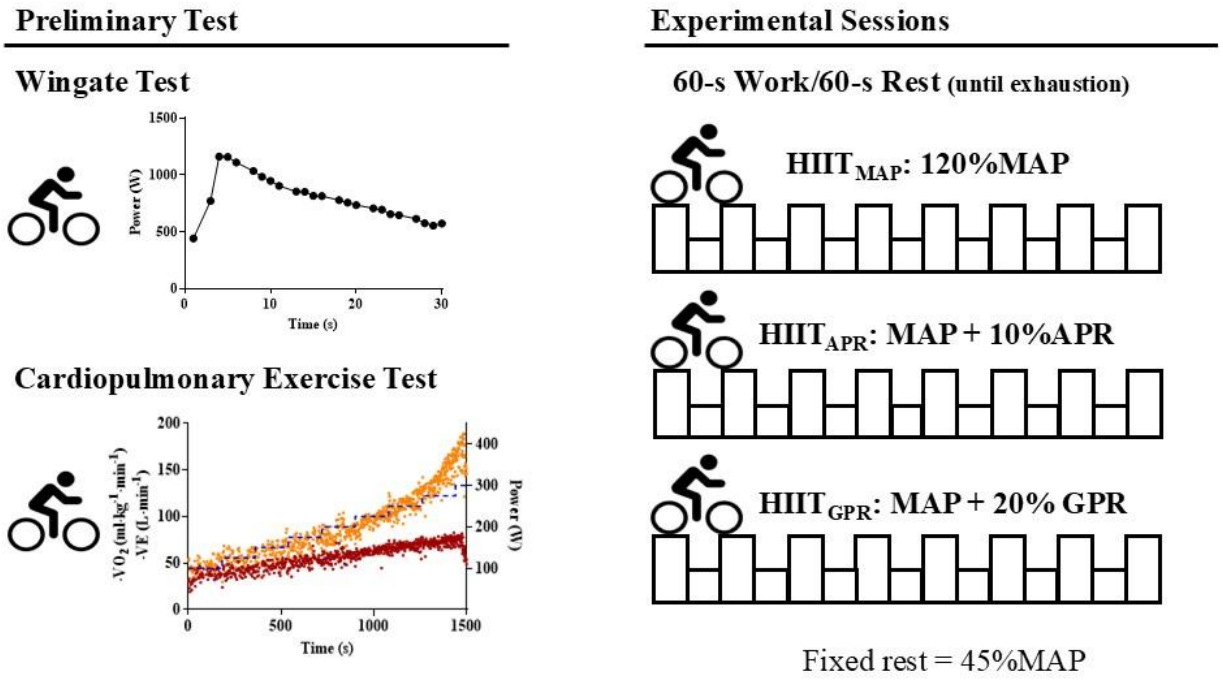
The HIIT sessions were administered to all participants on three non-consecutive days, with an inter-session interval of 72h. The HIIT consisted of repeated 60 seconds work intervals interspersed with 60 seconds of active recovery, until exhaustion. However, the HIIT protocols differed in the intensity prescription method. During $HIIT_{MAP}$, $HIIT_{APR}$ and $HIIT_{GPR}$ participants cycled to a workload of 120% MAP, Δ10% APR, and Δ20% GPR (i.e. Δ20% GPR = MAP + 20 % GPR), respectively. In all HIIT sessions, recovery between sets was performed at a workload equivalent to 45% of MAP (**Figure 1a**).

The selection of these intensities was guided by the Thibault graphical model and previous research investigating HIIT with 60s/60s intervals prescribed based on MAP/MAS (Thibault, 2003; Panissa et al. 2018). The intensities chosen for the HIIT protocol were also based on a preliminary trial involving four subjects so that they were able to achieve at least 10 minutes of work, as recommended by Buchheit and Laursen (Buchheit and Laursen 2013a). After that, an intensity equal to MAP + 10% APR and one equal to MAP + 20% GPR was adopted for the remaining experimental conditions so that the average intensity between the different HIIT protocols was similar. This approach was

necessary because a HIIT condition with significantly higher intensity would likely result in a shorter time to exhaustion, which, as previously noted, would also reduce heterogeneity in responses. Consequently, it would have been difficult to determine whether the observed reduction in the heterogeneity of physiological responses and time to exhaustion was due to the higher intensity itself or the method of intensity prescription adopted (Dotan, 2022a; Bok et al., 2023). Regarding the intensity of the recovery period, 50% of MAP was initially considered, as suggested by Buchheit and Laursen (2013a). However, following preliminary trials, to increase ecological validity, it was lowered to 45% of MAP.

Pulmonary gas exchange (breath by breath) and Hr were continuously monitored during exercise. Oxygen uptake and heart rate values were expressed as a percentage of each participant's individual VO_{2peak} (%VO_2) and Hr_{peak} (%Hr), respectively, and the last 10 seconds of each phase were averaged to generate a single value per set. Ear lobe capillary samples (5 μL) were collected every three sets during rest, and 1 min post-exhaustion, to determine [La]. RPE was assessed using Borg's scale (6-20) during each rest period. For the analyses, %VO_2, %Hr, [La], and RPE data of the third, sixth, ninth, and last intervals were retained.

Figure 1a. Experimental design.



***Abbreviations**: HIIT$_{APR}$, high-intensity interval training (HIIT) prescribed relative to anaerobic power reserve; HIIT$_{GPR}$, HIIT prescribed relative to glycolytic power reserve; HIIT$_{MAP}$, HIIT prescribed relative to maximal aerobic power.*

Statistical Analyses

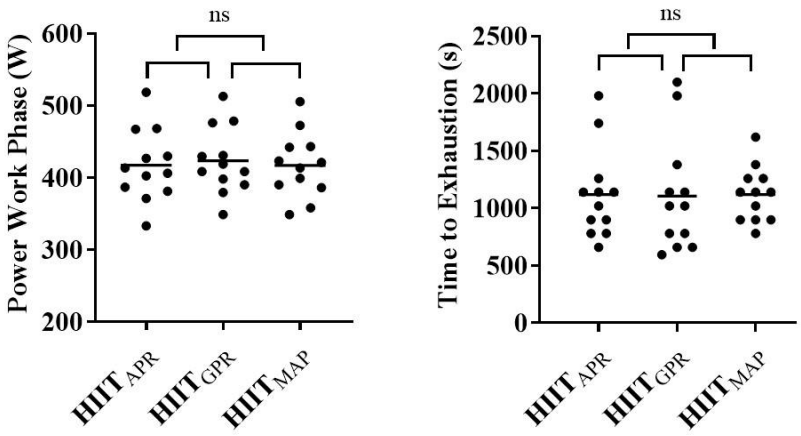
Normality and sphericity of data were tested using the Shapiro-Wilk and Mauchly's tests, respectively. Data are presented as mean ± SD. Greenhouse-Geisser correction to the degrees of freedom was applied when the assumption of sphericity was violated. Inter-individual variability in TTE, %VO_2, %Hr, and [La] was calculated as the root mean square of the residual, that is, the difference between the individual value and the mean value of a given dependent variable. An analysis of variance (ANOVA) for repeated measures (RM) was conducted to compare the TTE and the power exerted between HIIT sessions to ensure that the duration or intensity did not influence the inter-individual variability of the dependent variables. TTE residuals were compared between conditions by an RM ANOVA. Physiological, perceptual variables, and the relative residuals were compared between conditions and intervals by a two-way RM ANOVA with two within-subject factors (conditions: HIIT$_{APR}$, HIIT$_{GPR}$, and HIIT$_{MAP}$; interval: 3°,6°,9°, and last interval). Significance was set at 0.05 (two-tailed) for all analyses. Effect sizes were reported as partial eta squared (η^2_p) with cut-off points of 0.02

as small, 0.13 as medium, and 0.26 as large (Cohen 1988). Coefficients of variation have been calculated for all variables. The smallest worthwhile change proposed by Hopkins (2008) was computed when η^2_p was equal to, or greater than, 0.13 (medium effect) by multiplying the CV by 0.5 (Hopkins et al. 2009). Data were analyzed using IBM SPSS Statistics for Windows, version 26.0 (IBM Corp., Armonk, NY, USA).

Results

Data are presented as mean ± SD. Absolute performance data (i.e., TTE and PO), along with mean physiological and perceptual responses (i.e., %VO2, %Hr, La, and RPE) and residuals during the HIIT sessions, are provided in Table II and Table III. No significant differences between prescription methods in TTE ($F = 0.012$; $P = 0.988$; $\eta^2_p = 0.002$) and power output during the work phase (PO) ($F = 2.400$; $P = 0.141$; $\eta^2_p = 0.180$) were found (**Figure 2a**).

Figure 2a. Comparison of power output during the work phase (A) and time to exhaustion (B) among the high-intensity interval training sessions (HIIT).



***Abbreviations**: HIIT$_{APR}$, high-intensity interval training (HIIT) prescribed relative to anaerobic power reserve; HIIT$_{GPR}$, HIIT prescribed relative to glycolytic power reserve; HIIT$_{MAP}$, HIIT prescribed relative to maximal aerobic power; ns, not significantly different.*

CV of TTE in HIIT$_{MAP}$, HIIT$_{APR}$, and HIIT$_{GPR}$ was 21%, 35%, and 45%, respectively. Residuals in TTE were not significantly different among prescription methods ($F = 1.602$; $P = 0.225$; $\eta^2_p = 0.131$). However, HIIT$_{MAP}$ resulted in a ~52% reduction in TTE residuals compared to HIIT$_{APR}$ and a ~100% reduction compared to HIIT$_{GPR}$. These reductions exceeded the SWC threshold (46%) and thus may be deemed as practically meaningful. No significant differences among conditions in residuals of

%VO_2 (F = 1.603; P = 0.224; η^2_p = 0.120), %Hr (F = 0.668; P = 0.523; η^2_p = 0.060), [La] (F = 0.171; P = 0.751; η^2_p = 0.015) and in RPE (F = 1.658; P = 0.212; η^2_p = 0.127) were found.

No significant main effect of interval in residuals of %VO_2 (F = 1.445; P = 0.243; η^2_p = 0.121), %Hr (F = 0.831; P = 0.448; η^2_p = 0.072), [La] (F = 0.363; P = 0.702; η^2_p = 0.029) was observed, while there was a significant effect of interval in RPE residuals (F = 5.074; P = 0.046; η^2_p = 0.310). There was no significant condition × interval within residuals of %VO_2 (F = 1.554; P = 0.204; η^2_p = 0.121), %Hr (F = 0.427; P = 0.781; η^2_p = 0.043), [La] (F = 0.889; P = 0.479; η^2_p = 0.067) and RPE (F = 1.852; P = 0.181; η^2_p = 0.145).

Table IIa. Performance, average physiological and perceptual responses, and relative residual between prescription methods during HIIT sessions.

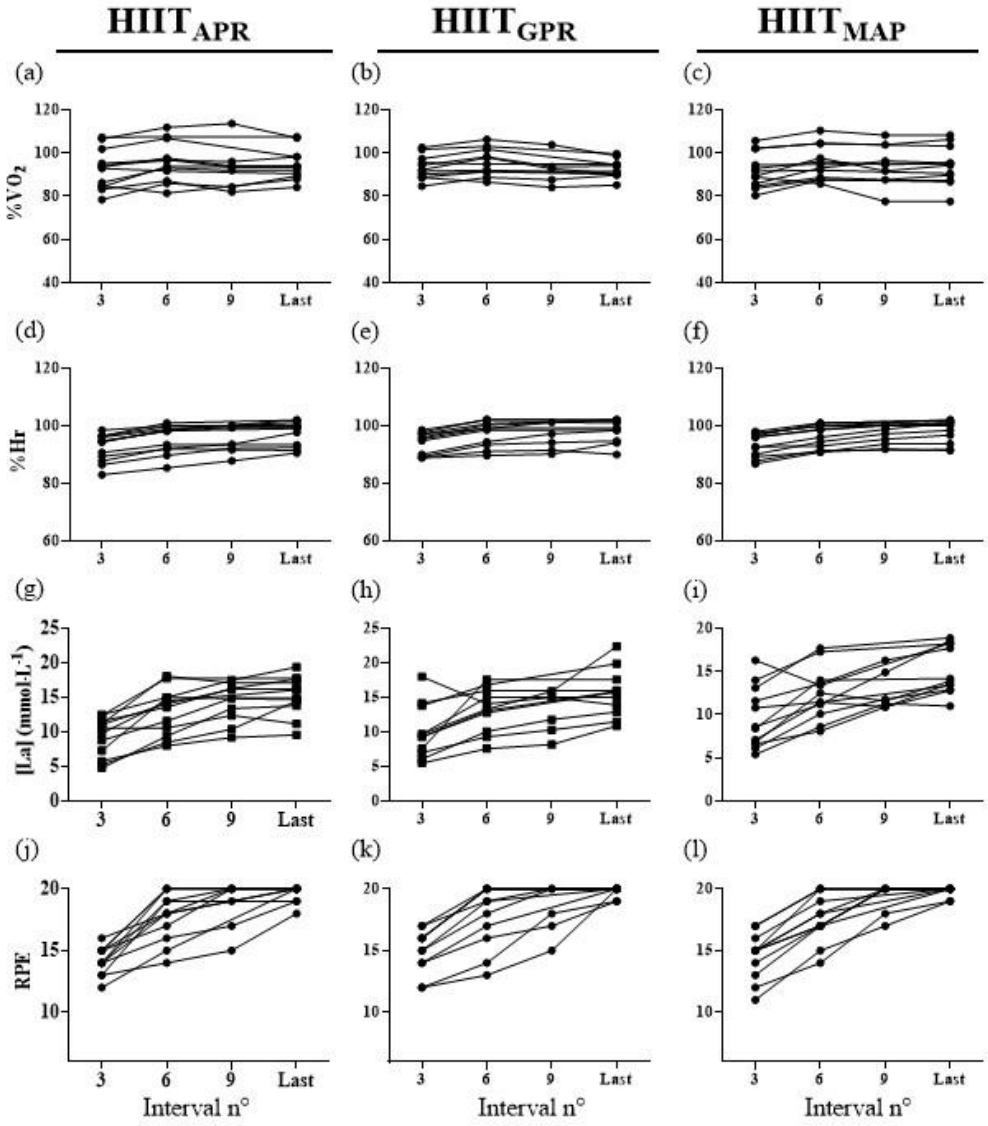
Outcome Variable	**Prescription Methods**		
	$HIIT_{APR}$	$HIIT_{GPR}$	$HIIT_{MAP}$
PO (W)	417 ± 49	426 ± 46	417 ± 45
TTE (s)	1120 ± 392	1104 ± 497	1120 ± 239
%VO_2 (%)	94.2 ± 9	93.5 ± 5.9	93.9 ± 8.5
%Hr (%)	94.9 ± 4.7	96.2 ± 4.2	96.3 ± 3.6
[La] (mmol·L^{-1})	12.9 ± 3.1	12.8 ± 3.4	12.5 ± 2.9
RPE	17.5 ± 1.5	17.6 ± 1.6	17.8 ± 1.4
		Residuals	
TTE (s)	280 ± 261	369 ± 312	183 ± 143
%VO_2 (%)	6.6 ± 5.6	4.6 ± 3.3	6.1 ± 5.1
%Hr (%)	3.9 ± 2.2	3.4 ± 2.2	3.3 ± 1.7
[La] (mmol·L^{-1})	2.4 ± 1.5	3.7 ± 2	2.4 ± 1.4
RPE	1.4 ± 1.03	1.8 ± 1.18	1.3 ± 0.9

***Abbreviations**: PO, power output of the work phase; TTE, time to exhaustion; %VO_2, O_2 uptake relative to VO_{2peak}; %Hr, heart rate relative to Hr_{peak}; [La], blood lactate concentration; RPE, rating of perceived exertion. $HIIT_{APR}$, high-intensity interval training (HIIT) prescribed relative to anaerobic power reserve; $HIIT_{GPR}$, HIIT prescribed relative to glycolytic power reserve; $HIIT_{MAP}$, HIIT prescribed relative to maximal aerobic power.*

Physiological and perceptual data at the 3°, 6°, 9°, and final intervals for each HIIT session are presented in Table III and Figure 3. No significant difference among conditions in $\%VO_2$ ($F = 0.372$; $P = 0.692$; $\eta^2_p = 0.042$), %Hr ($F = 2.267$; $P = 0.129$; $\eta^2_p = 0.182$), [La] ($F = 0.279$; $P = 0.622$; $\eta^2_p = 0.033$), and RPE ($F = 0.310$; $P = 0.735$; $\eta^2_p = 0.030$) was found. A significant effect of intervals with regard to $\%VO_2$ ($F = 9.234$; $P < 0.001$; $\eta^2_p = 0.844$), %Hr ($F = 63.836$; $P < 0.001$; $\eta^2_p = 0.863$), [La] ($F = 53.985$; $P < 0.001$; $\eta^2_p = 0.722$), and RPE ($F = 104.210$; $P < 0.001$; $\eta^2_p = 0.913$) was observed.

Finally, no significant condition × interval effect for $\%VO_2$ ($F = 1.554$; $P = 0.205$; $\eta^2_p = 0.131$), %Hr ($F = 1.921$; $P = 0.125$; $\eta^2_p = 0.157$), [La] ($F = 0.462$; $P = 0.767$; $\eta^2_p = 0.043$) and RPE ($F = 1.274$; $P = 0.302$; $\eta^2_p = 0.110$) was found (**Figure 3a**). Coefficients of variation for physiological and perceptual responses are shown in **Table IIIa**.

Figure 3a. Individual physiological and perceptual responses in O_2 uptake expressed relative to VO_{2peak} (a, b, c), heart rate expressed relative to Hr_{peak} (d, e, f), blood lactate concentration (g, h, i), and rate of perceived exertion (j, k, l).



***Abbreviations**: HIIT$_{APR}$, high-intensity interval training (HIIT) prescribed relative to anaerobic power reserve; HIIT$_{GPR}$, HIIT prescribed relative to glycolytic power reserve; HIIT$_{MAP}$, HIIT prescribed relative to maximal aerobic power.* significant main effect of intervals (P < 0.05).*

Table IIIa. Physiological, perceptual responses, and related coefficients of variation

	Interval	$HIIT_{APR}$		$HIIT_{GPR}$		$HIIT_{MAP}$	
		Mean ± SD	CV	Mean ± SD	CV	Mean ± SD	CV
$\%VO_2$	3°	92.4 ± 9.4	10.1%	93.4 ± 5.6	6.0%	91.8 ± 8.0	8.8%
	6°	95.9 ± 9.2	9.6%	95.6 ± 6.3	6.6%	95.2 ± 7.8	8.2%
	9°	93.7 ± 10.3	11.0%	92.4 ± 7.5	8.1%	94.4 ± 9.4	10.0%
	Last	94.7 ± 7.1	7.5%	92.7 ± 4.2	4.5%	94.1 ± 8.8	9.3%
%Hr	3°	92.4 ± 4.8	5.2%	94.0 ± 3.9	4.1%	93.4 ± 4.1	4.4%
	6°	95.6 ± 4.9	5.1%	97.3 ± 4.4	4.5%	96.3 ± 4.0	4.2%
	9°	94.2 ± 4.7	5.0%	94.9 ± 4.5	4.7%	97.2 ± 3.2	3.3%
	Last	97.4 ± 4.2	4.3%	98.5 ± 4.0	4.1%	98.4 ± 4.0	4.1%
[La] ($mmol \cdot L^{-1}$)	3°	9.3 ± 2.8	30%	10.0 ± 3.8	38%	9.6 ± 3.5	36.9%
	6°	13.0 ± 3.4	26%	13.3 ± 3.2	24%	12.4 ± 3.0	24.1%
	9°	14.2 ± 3.0	21%	12.2 ± 3.2	26%	12.5 ± 2.2	17.3%
	Last	15.3 ± 2.8	18%	15.6 ± 3.4	22%	15.3 ± 2.8	18.4%
RPE	3°	14.2 ± 1.1	8%	14.9 ± 1.8	12%	14.6 ± 1.8	12.6%
	6°	17.8 ± 2.0	11% *	17.8 ± 2.5	14% *	17.7 ± 1.9	10.9% *
	9°	18.7 ± 2.0	11%	18.0 ± 2.1	12%	19.2 ± 1.3	6.9%
	Last	19.7 ± 0.7	3%	19.8 ± 0.4	2%	19.8 ± 0.4	2.0%

***Abbreviations**: %VO_2, O_2 uptake relative to VO_{2peak}; %Hr, heart rate relative to Hr_{peak}; [La], blood lactate concentration; RPE, rating of perceived exertion. $HIIT_{APR}$, high-intensity interval training (HIIT) prescribed relative to anaerobic power reserve; $HIIT_{GPR}$, HIIT prescribed relative to glycolytic power reserve; $HIIT_{MAP}$, HIIT prescribed relative to maximal aerobic power; CV, coefficient of variation. * significant main effect of intervals ($P < 0.05$).*

Discussion

This study compared the inter-individual variability in time to exhaustion, physiological, and perceptual responses during HIIT sessions consisting of 60 seconds of work and 60 seconds of active rest prescribed relative to APR, GPR, and MAP in trained cyclists. The main finding was that prescribing cycling HIIT based on APR and GPR did not significantly reduce the inter-individual variability in TTE, $\%VO_2$, [La], %Hr, and RPE. Noteworthy, $HIIT_{MAP}$ leads to a lower CV, and, by extension, reduced TTE residuals compared to $HIIT_{APR}$ and $HIIT_{GPR}$. Indeed, $HIIT_{MAP}$ resulted in a substantial ~52% reduction in TTE residuals (i.e., the signal) compared to $HIIT_{APR}$ and an even more substantial ~100% reduction than the GPR approach. These reductions in TTE residuals exceeded the threshold for the smallest worthwhile change proposed by Hopkins et al. (2009), equivalent to ~46% (i.e., the noise), and thus may be deemed as practically meaningful.

Our results contrast with those of previous studies, indicating that the ASR model is effective in reducing the inter-individual variability in HIIT tolerance and physiological responses (Julio et al., 2020; Collison et al., 2021; Bok et al., 2023). For instance, Julio et al. (2020) observed that HIIT based on ASR reduced the CV in TTE and peak [La] compared to MAS-based prescription. However, their lower CV in TTE can be more a result of significantly higher intensity during $HIIT_{ASR}$ rather than the prescription method *per se*. It has been observed that shorter durations of the TTE are associated with lower variability in test results (Dotan, 2022a). This is likely attributed to the fact that longer tests require not only greater physical exertion but also a greater psychological capacity to tolerate prolonged efforts, introducing an additional confounding factor into the assessment (Dotan, 2022a).

Similarly, in running HIIT with short intervals, Collison et al. (2021) observed a slightly (although not significantly) lower variability in TTE when the prescription was based on ASR compared with MAS-based prescription. Also, Bok et al. (2023), during a running HIIT, observed a slight (not significant) reduction in inter-individual variability of physiological responses by ASR-based prescription, in contrast to our results, in which we found no significant differences in residuals related to physiological

and perceptual variables. However, it should be noted that the difference in inter-individual variability between prescription methods in previous studies always presented some uncertainty. Notwithstanding this, given the trend observed, the authors supported the rationale behind the ASR construct and suggested its use for prescribing HIIT. On the contrary, our results did not support the use of APR and GPR during longer format HIIT in cycling but rather suggest that the traditional method based on MAP likely leads to a greater reduction in TTE variability. Although the underlying reasons for these differences are unclear, several hypotheses can be proposed.

Firstly, our HIIT format and modality differ from those applied by previous studies (Julio et al., 2020; Collison et al., 2021; Bok et al., 2023). We investigated a cycling HIIT protocol, with 60 seconds of work interspersed with 60 seconds of active rest. In contrast, the abovementioned studies involved a running HIIT protocol characterized by briefer intervals, specifically 15-s of work followed by 15-s of passive rest (Julio et al., 2020; Collison et al., 2021; Bok et al., 2023). In addition, such HIIT protocols were performed at a higher intensity than ours, as also highlighted by the lower TTE values (~400s vs ~1000s) (Julio et al., 2020; Collison et al., 2021; Bok et al., 2023). Consequently, considering that the higher the intensity, the greater the contribution of the anaerobic metabolism, we could hypothesize that the APR/ASR model may be able to capture the differences in aerobic and anaerobic athlete characteristics only when the prescribed intensity is much higher than that prescribed in our research.

Additionally, in contrast to our findings, Barnett et al. (1996) reported a reduced variability in cycling TTE when intensity was based on GPR compared to the APR and MAP approaches (Barnett et al., 1996). A possible explanation could be attributed to the differences in the protocol design. Specifically, they examined a constant work rate to exhaustion with an average duration of approximately 2 minutes and 30 seconds, while our study evaluated HIIT sessions with a mean duration of ~18 min (Barnett et al., 1996). Consequently, it is plausible that the GPR, which relies on the mean power output of the 30-second Wingate test, placing greater emphasis on glycolytic pathways, may provide a more

comprehensive account of the variability observed in TTE under the exercise conditions chosen by Barnett et al. (1996) than those applied in our experimental protocol.

However, in cycling, HIIT is typically prescribed to improve aerobic power. This requires the athletes to spend a certain amount of time near 90% of their VO_{2peak} (Buchheit and Laursen, 2013a). The extent of this duration naturally depends on the athlete's level. Nonetheless, the typical time spent near VO_{2peak} spans from 10 minutes to 30 minutes (Buchheit and Laursen, 2013a). For this reason, it is important to carefully prescribe intensity to avoid premature exhaustion. Therefore, while APR/ASR may exhibit relevance at extremely high intensities, as previously suggested (Collison et al., 2021; Julio et al., 2020), we hypothesize that when prescribing intensity for HIIT to augment aerobic power in cycling, there are no clear advantages in considering APR/GPR to prescribe intensity compared to MAP. Secondly, the APR and GPR models' inability to normalize physiological responses and exercise tolerance could be attributed to the parameters employed to quantify the anaerobic reserve. Indeed, both models calculate the anaerobic/glycolytic reserve from MAP, disregarding the anaerobic contribution already required (Luttikholt & Jones, 2022; Sandford et al., 2021). A further limitation of the APR model pertains to the upper limit of the anaerobic reserve, namely the PPO achieved during the Wingate test. PPO is attained within the first few seconds of maximal effort and is primarily determined by the amount of adenosine triphosphate and phosphocreatine in muscle fibers, the firing rate of motor neurons, as well as the recruitment of motor units (Beneke et al., 2002; Driss & Vandewalle, 2013). Anaerobic glycolysis plays a smaller role in this determination (Driss & Vandewalle, 2013). Therefore, PPO is not a comprehensive indicator of an athlete's anaerobic capacity, and there is considerable uncertainty regarding its ability to accurately assess it (Minahan et al., 2007). This limitation was initially noted by Barnett et al. (1996) and has been reaffirmed by our findings. For this reason, Barnett et al. (1996) proposed normalizing TTE using a more relevant anaerobic capacity parameter, namely the GPR. However, a potential bias in GPR may be attributed to the use of

MPO as a ceiling limit for this anaerobic reserve. Indeed, MPO cannot be solely attributed to anaerobic pathways, as aerobic metabolism also plays a substantial role (Minahan et al., 2007).

Finally, this study is not without limitations. First, we investigate only a single HIIT format, therefore other combinations may yield different results. Second, a limit that pertains to our experimental approach, as well as to the APR/GPR model, is the lack of a standardization procedure for MAP determination. This dependency not only complicates the accurate assessment of a 'true' anaerobic power reserve but also underscores the need for a standardized approach to MAP assessment to facilitate cross-study comparisons. Additionally, it raises the possibility of adopting a less protocol-dependent method, such as critical power, for defining the lower boundary of the APR/GPR model. However, further research is required to evaluate the utility and validity of this approach.

Conclusions

Our findings do not support the use of APR or GPR for normalizing exercise intensity during cycling HIIT with 60-second bouts. This suggests that both APR and GPR fail to accurately differentiate between the aerobic and anaerobic characteristics of an athlete, hindering the normalization of exercise responses during HIIT. Additional research is warranted to elucidate the reasons behind the model's inefficacy in normalizing physiological and exercise tolerance responses and to explore potential connections between the APR model and the classical power-duration relationship.

STUDY 2

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Insights into Anaerobic Power Reserve On Relationships with Exercise Tolerance, Work Above Critical Power, and Accumulated Oxygen Deficit in Endurance-Trained Male Cyclists: A Pilot Study

Di Gennaro S., Panascì M., Gallo G., Filipas L., Ruggeri P., Faelli E.

Abstract

The anaerobic power reserve (APR) model seeks to account for the heterogeneity in athletes' anaerobic characteristics. However, its relationship with exercise tolerance across various durations and with anaerobic markers remains unclear. Therefore, we investigate the relationship between APR, exercise tolerance, work above critical power (W'), and maximal accumulated oxygen deficit (MAOD) in male cyclists. We further analyzed this relationship, replacing maximal aerobic power (MAP) with critical power (CP) as the lower boundary of the power reserve, defining a so-called maximal power reserve (MPR). After preliminary tests, 19 endurance-trained male cyclists performed five trials to exhaustion (Tlim) at 130%, 115%, 100%, 85%, and 80% of maximal aerobic power (MAP) and Wingate test. APR and MPR correlated with all Tlim values ($r > 0.511$, $p < 0.03$), except at 80%MAP. After fixing CP or MAP, only correlations with supramaximal Tlim remained significant ($r > 0.703$, $p < 0.002$). When PPO was fixed, only MPR correlated with Tlim at 130% and 115%MAP ($r > 0.508$, $p = 0.037$). Both APR and MPR were associated with MAOD and W′ ($r = 0.480–0.542$, $p = 0.045$), but only MPR remained significantly related to MAOD after adjusting for lower boundary ($r = 0.488$, $p = 0.040$). Our findings showed that in endurance-trained male cyclists, both power reserves relate to exercise tolerance, however their influence decreases for longer efforts. MPR exhibited a stronger link to

anaerobic capacity compared to APR. The association between exercise tolerance and APR/MPR appears largely driven by peak power output (PPO), rather than the choice of lower boundary.

Keywords:

Cycling, Modelling, Power-Duration, Critical Power, Anaerobic Power Reserve

Introduction

In the last years, the anaerobic power reserve (APR) has emerged as a potential tool to prescribe training within the severe and extreme intensity domains as well as a predictive model for sprint performance (Sandford et al., 2021). The APR is generally defined as the difference between the maximal instantaneous power output (PPO) and the last power output attained during an incremental test, often referred to as maximal aerobic power (MAP), disregarding any significant anaerobic contribution above critical power (Sandford et al., 2021). This model seeks to account for the heterogeneity in athletes' anaerobic characteristics and potentially leads to a more homogenous acute training response (Sandford et al., 2021). At the same relative intensity, prescribed as MAP percentage, athletes with higher PPO will engage a smaller fraction of their APR. This could lead to reduced metabolic stress and consequently delayed exhaustion compared to athletes with lower PPO (Buchheit & Laursen, 2013b). According to this theory, a previous study reported a strong relationship between running time to exhaustion within the severe intensity domain and anaerobic speed reserve (ASR), suggesting this parameter can be a potential proxy for anaerobic characteristics (Blondel et al., 2001). Similarly, the APR model in cycling has been shown to predict sprint performance across efforts lasting between 5 and 180 seconds in both recreational (Weyand et al., 2006) and well-trained male road cyclists (Sanders & Heijboer, 2019). However, the aforementioned approach relies on mathematical models, which can only be applied following several unconventional maximal efforts (Sanders & Heijboer, 2019; Weyand et al., 2006). Consequently, some researchers have explored whether a simpler method, such as prescribing exercise intensity based on the simple difference between PPO and MAP (i.e., APR), could account for the variability observed during exhaustion tests based on MAP (Barnett et al., 1996; Blondel et al., 2001; Boullosa & Abreu, 2014; Buchheit & Laursen, 2013b). Barnett et al. (1996) prescribed a cycling work rate based on individual APR in male athletes, trying to reduce the variability in time to exhaustion. However, this approach did not yield the expected results, suggesting instead that APR might represent an oversimplification of the metabolic and

physiological mechanisms involved in anaerobic pathways. Moreover, recent studies on male team players have reported only minimal, and never statistically significant, reductions in intersubject variability in physiological responses and exercise tolerance outcomes during interval training when using APR-based intensity prescriptions, compared to traditional methods (Bok et al., 2023; Collison et al., 2021; Julio et al., 2020). While the APR construct holds theoretical validity, its lack of physiological validation may stem from the use of MAP as its lower boundary. MAP, in addition to being protocol-dependent, also requires substantial anaerobic contributions to be sustained; thereby, this overlap complicates the isolation of a 'reserve' that clearly defines the boundary between sustainable and unsustainable power domains. Therefore, taking these factors together, some doubts arise about the actual effectiveness of APR as a prescription method to uniform exhaustion times and physiological responses during high-intensity exercise.

Currently, the most accurate method to achieve uniform exercise tolerance among athletes is to consider the individual power-duration relationship (Dotan, 2022a). This relationship enables the determination of the critical power (CP) and the finite amount of work (W') that can be performed above it (Drake et al., 2024; Jones et al., 2019). Bearing in mind the analogy between the APR model and the power-duration relationship, the former presents a potentially simpler alternative to uniform exercise stimulus compared with the more complex and demanding procedures required for CP and W' determination (Leo et al., 2022; Sanders & Heijboer, 2019). Although the APR appears to be a useful and appealing tool for exercise prescription, there is no evidence that it correlates with exercise tolerance during constant cycling within the severe-intensity domain based on MAP in male cyclists. Additionally, no research investigated the possible correlation between APR and well-established measures of anaerobic capacity, like the maximal accumulated oxygen deficit (Medbo et al., 1988; Medbø & Welde, 2022) or W' in male cyclists. Therefore, in order to assess the construct validity of the APR model, it is essential to investigate its correlation with established measures of anaerobic characteristics.

Hence, the aim of this pilot study was to investigate, in endurance-trained male cyclists: (i) the relationship between cycling exercise tolerance during severe-intensity constant work rates, MAP-based and APR;(ii) the association between APR and W';(iii) the relationship between APR and maximal accumulated oxygen deficit.

Furthermore, given the inherent protocol dependency of MAP and the high reliance on anaerobic pathways to meet energy demands at this intensity, we further analyzed this relationship using CP instead of MAP as the lower boundary of the power reserve, defining a so-called maximal power reserve (MPR).

Materials and Methods

Participants

A *priori* sample size calculation was performed through G*Power, considering a positive relationship between APR and time to exhaustion as the main outcome (Blondel et al., 2001). A bivariate correlation with a significance level set at 0.05, two tails, statistical power of 0.80, and *r* equal to 0.6 were applied. This computation generated a desired sample size of at least 17 participants. Therefore, 19 endurance-trained male cyclists were recruited (mean ± SD; age: 34 ± 9 years; height: 1.79 ± 0.07 m; mass: 74 ± 10 kg). Inclusion criteria required that participants had trained consistently for at least 6 hours per week in the last year. Any muscular or orthopedic injuries and health problems in the previous six months were considered exclusion criteria. To standardize testing conditions, participants were asked to refrain from food and caffeine at least 3-4 hours prior to each test, visit the laboratory consistently at the same time of the day (±1), avoid strenuous exercise for at least 36 hours beforehand, and keep their dietary habits unchanged throughout all the intervention period. During all visits, participants were allowed to drink water *ad libitum*. All participants were provided with a thorough explanation of the study's aim and procedures before providing written informed consent. The research adhered to the

Declaration of Helsinki, with prior approval from the Ethics Committee of Università degli Studi di Milano (University of Milan, N. 2020/49).

Design

Participants visited the laboratory seven times over four weeks. The sessions included familiarization sessions, a ramp incremental test to determine maximal oxygen uptake (VO_2max) and MAP, a Wingate test, and five severe-intensity trials to exhaustion (Tlim) to estimate CP and W'. Additionally, submaximal constant work rates (CWR) below the respiratory compensation point were conducted before each Tlim session to estimate accumulated oxygen deficit (AOD). An electromagnetically braked cycle ergometer (Excalibur Sport, Lode, Groningen, Netherlands) was used, adjusted to individual anthropometry and positioning habits. All tests, except for familiarization and the initial ramp test, were randomized across visits.

Pre-testing and familiarization

During their first visit, participants underwent anthropometric assessments and familiarization with all procedures. Body composition was estimated using a bioelectrical impedance device (Tanita BC-420 MA, Tokyo, Japan) following the manufacturer's recommendations. Height was measured using a stadiometer (Seca 217, Vogel & Halke, Hamburg, Germany). Then, participants completed a torque-velocity profile test and a Wingate test. These tests served two purposes: 1) to familiarize participants with sprinting on a stationary ergometer and 2) to determine the optimal cadence (through the ergometer linear factor) for achieving PPO during a subsequent Wingate test (Driss & Vandewalle, 2013). After thirty minutes, participants performed a familiarization ramp test and a Tlim lasting ~5 minutes. A further familiarization Tlim lasting ~15 minutes was conducted at the end of the second visit.

Ramp-incremental Test

Participants completed a ramp incremental test to determine VO_2max and the MAP following the step-ramp-step protocol proposed by Iannetta et al. (2020b). The test began with a 2-minute warm-up at 20 W, followed by a first square wave increase at 100 W lasting 6 minutes, within the moderate domain (MOD), followed by 4 minutes at 50 W. Then, an increase of ~1 W every 5 seconds (25 W every 2 minutes) started. The slope of this ramp test was chosen to be comparable to that used by one of the pivotal manuscripts on the relationship between APR and Tlim, and thus, to be able to compare the results (Blondel et al., 2001). Participants were instructed to maintain a self-selected cadence between 70 and 90 rpm throughout the test. The test ended when participants were not able to maintain their cadence above 70 rpm despite strong verbal encouragement. VO_2max was determined as the highest 30-second rolling average and was considered achieved when participants met at least two of the three of the following criteria: heart rate exceeding 95% of predicted maximal heart rate (210 – (0.65 × age)); respiratory exchange ratio exceeding 1.1; blood lactate exceeding 8.0 mmol·L^{-1} (Poole & Jones, 2017). MAP was considered the final power output achieved during the ramp test. After thirty minutes, a heavy intensity constant work rate (HVY), corresponding to 75% of the second ventilatory threshold, lasting 15 minutes was performed. Mean oxygen uptake (VO_2) of the last 2 minutes of MOD and HVY were used to left-shift the power output corresponding to gas exchange threshold (GET) and respiratory compensation point (RCP) according to the recommended procedure (Iannetta et al., 2020b).

Wingate Test

The Wingate test began with a 5-minute warm-up cycling between 50 and 100 W. Participants then performed three 4-5 second preparation sprints followed by a 5-minute passive rest period. Afterward, participants pedaled (unloaded) at 65 rpm for 10 seconds to allow a flying start. Upon completion of these 10 seconds, the linear ergometer mode was applied, and the participants were instructed to accelerate maximally and perform a 30-second sprint while maintaining a seated position (Driss &

Vandewalle, 2013). The resistance applied during the test was determined based on the torque-velocity test conducted during the familiarization session. Participants were blinded to power output and elapsed time during the test to minimize pacing strategies. PPO was considered the highest 1-second power output.

Severe Intensity Trials to Exhaustion

Five Tlim were conducted to estimate CP and W'. These trials employed a range of pre-determined power outputs corresponding to 80% (Tlim80), 85% (Tlim85), 100% (Tlim100), 115% (Tlim115), and 130% (Tlim130) of MAP. The specific power outputs were chosen to elicit a range of time-to-exhaustion durations between 2 and 20 minutes, as recently suggested (Mattioni Maturana et al., 2018). Each Tlim commenced with a 4-minute warm-up at 50 W, followed by a square wave increase to the pre-determined power output. CP and W' were modeled using the following equations (Hugh Morton, 1996):

1) Three parameters hyperbolic model: $t = W'/(PO-CP) + W'/(CP-P_{max})$

2) Two parameters hyperbolic model: $t = W'/(PO-CP)$

Where t is time to exhaustion, P_{max} is the theoretical maximal instantaneous power, W' is work (Joules), and PO is power output. Both sets of estimated values were retained for statistical analysis.

Accumulated Oxygen Deficit

Thirty minutes before each Tlim, participants performed two CWRs, 10 minutes long, separated by a 10-minute rest period. The intensity of each CWR spanned from 30% of MAP to the RCP (i.e., 30, 35, 40, 45, 50, 55, 60, 65, 70, 75% of MAP). The mean VO_2 from the final two minutes of each CWR test was used to establish the relationship between PO and VO_2. Participants were instructed to pedal at their preferred cadence (80 ± 5 rpm) and maintain the same cadence throughout the sessions. The theoretical oxygen demand for each Tlim was estimated by multiplying the extrapolated VO_2 (from the PO-VO_2 relationship) by the time to exhaustion for that specific trial. The AOD was then calculated

for each trial by subtracting the measured VO_2 consumed during the trial from the VO_2 demand area (Medbo et al. 1988; Medbø and Welde 2022). Finally, the highest AOD (MAOD) obtained from these trials was retained for further correlation analysis. The contribution of anaerobic metabolism was calculated as the ratio between the AOD and the VO_2 demand and expressed in percentage (Medbø & Welde, 2022).

Anaerobic Power Reserve and Maximal Power Reserve

APR has been calculated as PPO minus MAP. Additionally, considering that MAP, and *ipso facto* the APR, are protocol dependent, the MPR, that is, PPO minus CP, has been calculated. We specifically opted for the CP derived from the 3-parameter model ($CP_{3\text{-}hyp}$) due to its lower values compared to the 2-parameter hyperbolic model ($CP_{2\text{-}hyp}$). This selection reflects the greater sustainability of exercise intensities based on the $CP_{3\text{-}hyp}$, potentially resulting in a lower contribution from anaerobic pathways and consequently generating an MPR with less overlapping energy systems contributions (Mattioni Maturana et al., 2018).

Data Collection and Analysis

Pulmonary gas exchange was continuously monitored breath-by-breath during all testing sessions using a wearable metabolic cart (Cosmed K5, Rome, Italy) and a mask (Hans Rudolph, INC. Shawnee, KS, USA) with a dead space of 30 mL. Before the test, the ergospirometer was calibrated with a 3-L syringe (Cosmed, Rome, Italy), with ambient air and a standardized gas mixture (20.0% O2 and 4.0% CO2). The laboratory temperature was maintained at 20 ± 1 °C and relative humidity at 53 ± 4 %. Raw gas exchange was analyzed using Matlab R2023b (The Mathworks Inc., Natick, MA, USA). A custom-built script was used to remove outliers (i.e., errant breath or coughing). Subsequently, data were interpolated to a one-second time base and filtered using a low-pass forward-backward Butterworth filter (3rd order, 0.04 Hz cut-off). The VO_2 at GET during the ramp test was identified by the first non-linear increase in both ventilation (VE) and carbon dioxide output that deviated from the initial

linear rise in VO_2 (Beaver et al., 1986). The VO_2 at RCP was identified by the subsequent, distinct rise in ventilation and an initial decrease in the end-tidal partial pressure of carbon dioxide following the isocapnic buffering phase (Keir et al., 2018). Afterward, employing VO_2 of the last two minutes of MOD and HVY, the power outputs at GET and RCP were retrospectively adjusted. This adjustment was performed using a MATLAB custom-built script following the procedures outlined by Iannetta et al. (2020) to account for the VO_2 mean response time during the ramp test (Iannetta et al., 2020b).

Blood lactate concentration ([La]) was measured from earlobe samples (5 μL) using a Lactate Pro 2 (Arkray, Kyoto, Japan). Following the ramp incremental test to exhaustion, samples were obtained at 2 and 3 minutes post-exhaustion. Similarly, for the severe-intensity trials, blood was drawn 2, 3, and 4 minutes after exhaustion and the peak value was retained.

Statistical Analysis

Data normality was assessed using the Shapiro-Wilk test. Descriptive statistics are presented as mean ± standard deviation (SD). Pearson's correlation and 95% confidence intervals (CIs) were used to examine the relationship between all measured parameters with APR and MPR, and a correlation coefficient of >.1, >.3, >.5, and >.7 was considered small, moderate, large, and very large (Hopkins et al., 2009). Moreover, the standard error of estimation (SEE) has been calculated to assess the Tlim prediction accuracy of APR/MPR. Since a high APR/MPR can be obtained with a high PPO, as well as a low MAP/CP, or both, a partial correlation analysis was employed to better understand the effects of APR/MPR on exercise tolerance and other variables. The approach is therefore divided into two categories: 1) evaluating the relationship between APR/MPR and the other parameters controlling for PPO (thus evaluating the effect of APR/MPR as MAP/CP decreases); 2) evaluating the relationship between APR/MPR and the other parameters, controlling for MAP/CP (thus evaluating the effect of APR/MPR as PPO increases). A lack of correlation between APR/MPR and both CP and MAP prevented us from directly performing the latter. However, the significant relationship between PPO and both CP and MAP enabled us to employ an indirect approach to assess the influence of APR/MPR.

In particular, by performing partial correlations between PPO with other variables while fixing either CP or MAP we could isolate the contribution of APR as PPO increases. To compare the physiological and perceptual responses attained during all Tlim an analysis of variance for repeated measures was applied, and in case of significant differences between intensities, pairwise comparisons were calculated with the Bonferroni correction. La, VE, AOD, and RPE were not normally distributed, thus the Friedman test was performed. Significance was set at 0.05 (two-tailed). All analyses were performed using IBM SPSS Statistics for Windows, version 26.0 (IBM Corp. Armonk, NY, USA).

Results

Participants' characteristics are presented in **Table 1b.**

Table 1b . Participants' Characteristics

Anthropometry		**Power-Duration Relationships**	
Age (years)	34 ± 9	$CP_{3\text{-}hyp}$ (W)	249 ± 36
Height (m)	1.79 ±0.07	$W'_{3\text{-}hyp}$ (kJ)	41 ± 15
Mass (kg)	74 ± 10	P_{max} (W)	1177 ± 1124
Lean mass (kg)	63 ± 8	$CP_{2\text{-}hyp}$ (W)	257 ± 34
Body Fat (%)	13 ± 2	$W'_{2\text{-}hyp}$ (kJ)	29 ± 9
Incremental Ramp Test		**Wingate Test**	
VO_2peak ($ml \cdot kg^{-1} \cdot min^{-1}$)	61 ± 8	PPO (W)	1064 ± 190
MAP (W)	350 ± 46	**Anaerobic/Maximal Power Reserve**	
GET (W)	176 ± 32	APR (W)	714 ± 171
RCP (W)	243 ± 35	MPR (W)	815 ± 176

Notes *VO_2max, maximal oxygen uptake; MAP, maximal aerobic power; GET, gas exchange threshold; RCP, respiratory compensation point; $CP_{3\text{-}hyp}$, Critical power derived from the 3 parameters hyperbolic model; $W'_{3\text{-}hyp}$, anaerobic work capacity derived from the 3 parameters hyperbolic model; P_{max}, the theoretical maximal istantaneous power output estimated by the 3 parameters model; $CP_{2\text{-}hyp}$, Critical power derived from the 2 parameters hyperbolic model; $W'_{2\text{-}hyp}$, anaerobic work capacity derived from the 2 parameters hyperbolic model; PPO, peak power output; APR, anaerobic power reserve; MPR, maximal power reserve*.

The mean duration, PO, and physiological responses attained during each Tlim are shown in **Table 2b** and **Figure 1b**. There were no significant differences between VO_2max reached during the incremental test and VO_2peak attained during Tlims ($p > 0.49$). However, a significantly higher VO_2peak has been observed during Tlim100 compared to Tlim115 and Tlim130 ($p = 0.01$ and $p = 0.009$, respectively).

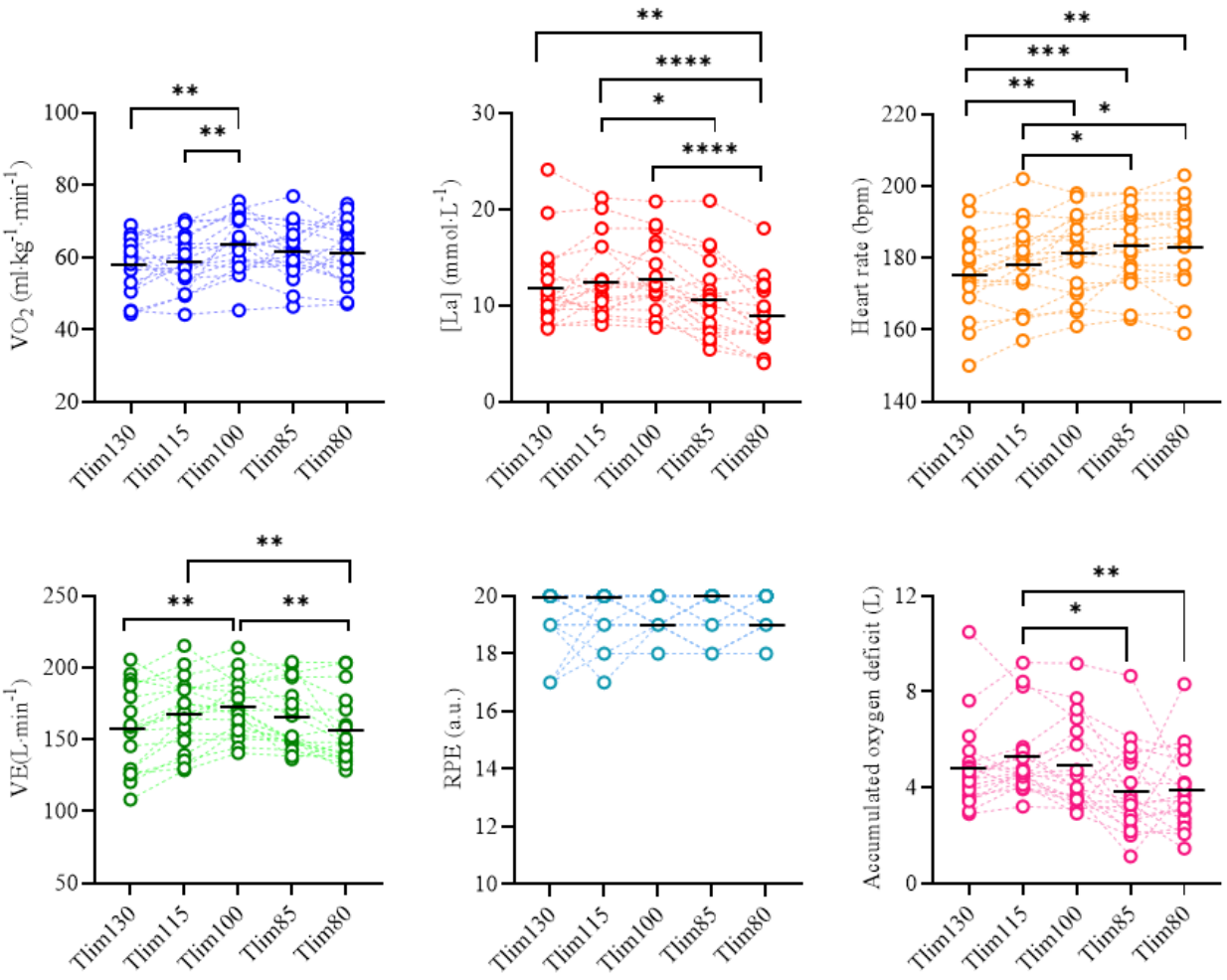
59% of participants achieved the MAOD during Tlim115, 26% during Tlim100, and 16% during Tlim130. Indeed, ANOVA revealed a significant difference in AOD among Tlims, and pairwise comparisons with Bonferroni correction indicated that AOD during Tlim115 was significantly higher than during Tlim85 and Tlim80 ($p = 0.007$ and $p = 0.045$, respectively) (**Table 2b** and **Figure 1b**).

Table 2b. Descriptive data of the time to exhaustion trials

	Tlim130	Tlim115	Tlim100	Tlim85	Tlim80
Power Output (W)	455 ± 60	403 ± 53	350 ± 46	298 ± 39	280 ± 37
Durations (s)	107 ± 26	152 ± 31	289 ± 80	717 ± 17	1126 ± 275
VO_2peak ($ml \cdot kg^{-1} \cdot min^{-1}$)	58 ± 8	59 ± 8	64 ± 9	61 ± 7	61 ± 8
Hrpeak (bpm)	175 ± 11	178 ± 11	181 ± 11	183 ± 11	183 ± 12
[La] ($mmol \cdot L^{-1}$)	11.8 ± 4.1	12.6 ± 3.9	12.8 ± 3.7	10.7 ± 4.0	8.9 ± 3.5
VEpeak ($L \cdot min^{-1}$)	157 ± 29	168 ± 25	173 ± 20	166 ± 23	156 ± 23
AOD (L)	4.4 ± 1.3	4.9 ± 1.5	4.6 ± 1.9	3.9 ± 1.8	3.6 ± 1.3
RPE (a.u.)	19.2 ± 1.1	19.3 ± 0.9	19.3 ± 0.6	19.3 ± 0.8	19.4 ± 0.6

Notes *Data are expressed as mean ± standard deviation. VO_2peak, peak oxygen uptake; Hrpeak, peak heart rate; [La], peak blood lactate concentration; VEpeak, peak ventilation; AOD, accumulated oxygen deficit; RPE, rate of perceived exertion; Tlim130, Time to exhaustion at 130% of maximal aerobic power (MAP); Tlim115, Time to exhaustion at 115% of MAP; Tlim100, Time to exhaustion at 100% of MAP; Tlim85, Time to exhaustion at 85% of MAP; Tlim80, Time to exhaustion at 80% of MAP*

Figure 1b. Physiological and perceptual responses during the time to exhaustion trials across intensities.

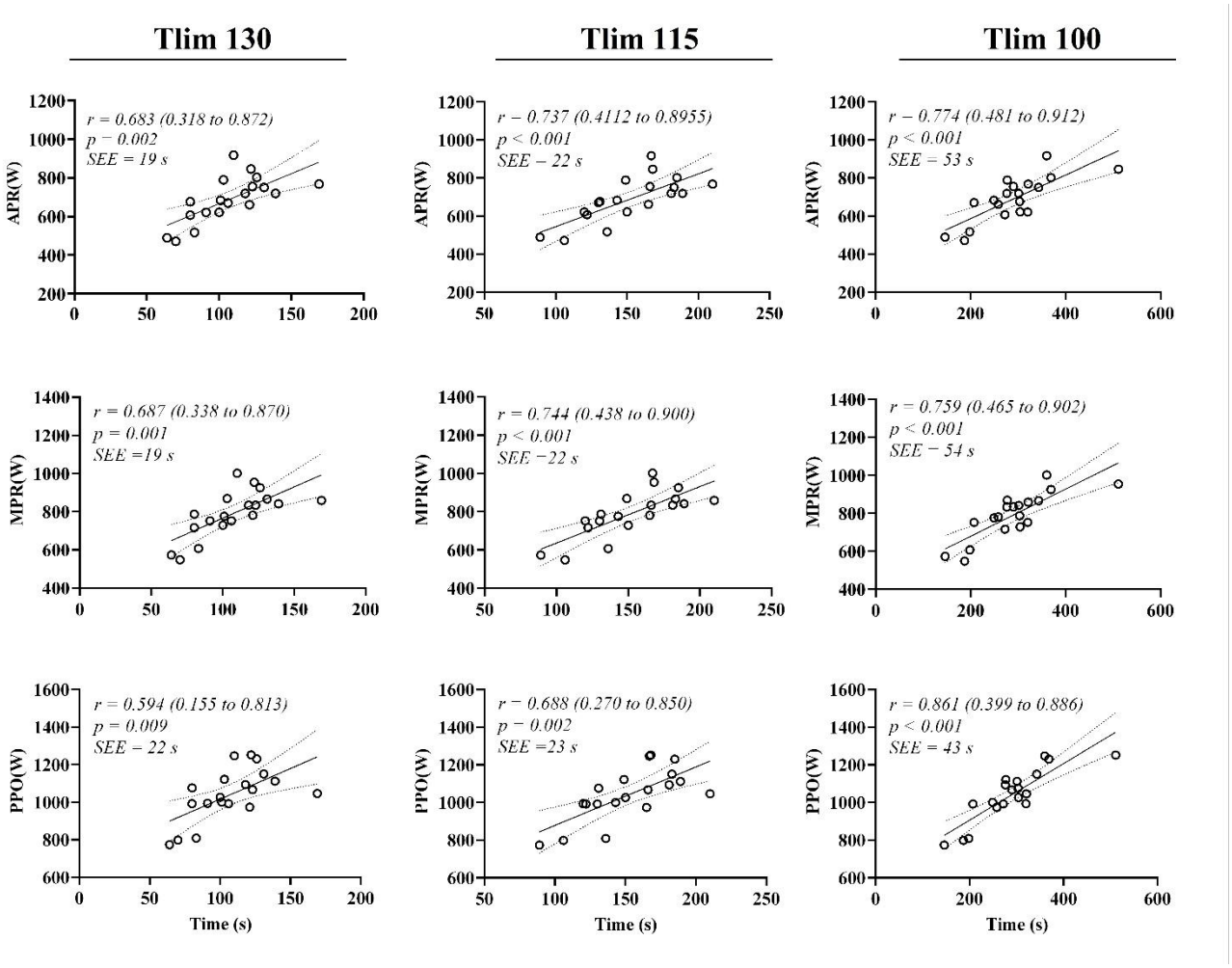


Notes *Tlim130, Time to exhaustion (Tlim) at 130% of maximal aerobic power (MAP); Tlim115, Tlim at 115% of MAP; Tlim100, Tlim at 100% of MAP; Tlim85, Tlim at 85% of MAP; Tlim80, Tlim at 80% of MAP; VO_2, oxygen uptake; [La], blood lactate concentration; VE, ventilation; RPE, rate of perceived exertion.*

APR and MPR showed a large to very large significant positive correlation with all Tlims except Tlim80, as shown in **Figures 1a** and **2b**. Notwithstanding, when APR/MPR were normalized to body mass, only the relationships with (supra)maximal Tlims remained significant (Table 3). Similarly, after controlling for CP or MAP, these relationships remain significant for Tlim130, Tlim115, and Tlim100 while becoming non-significant for Tlim85 and Tlim80. After controlling for PPO, only the relationship between MPR, Tlim130, and Tlim115 remained statistically significant (**Table 3b)**. Similarly, PPO showed a large to very large significant positive correlation with all Tlim (**Figures 2b**

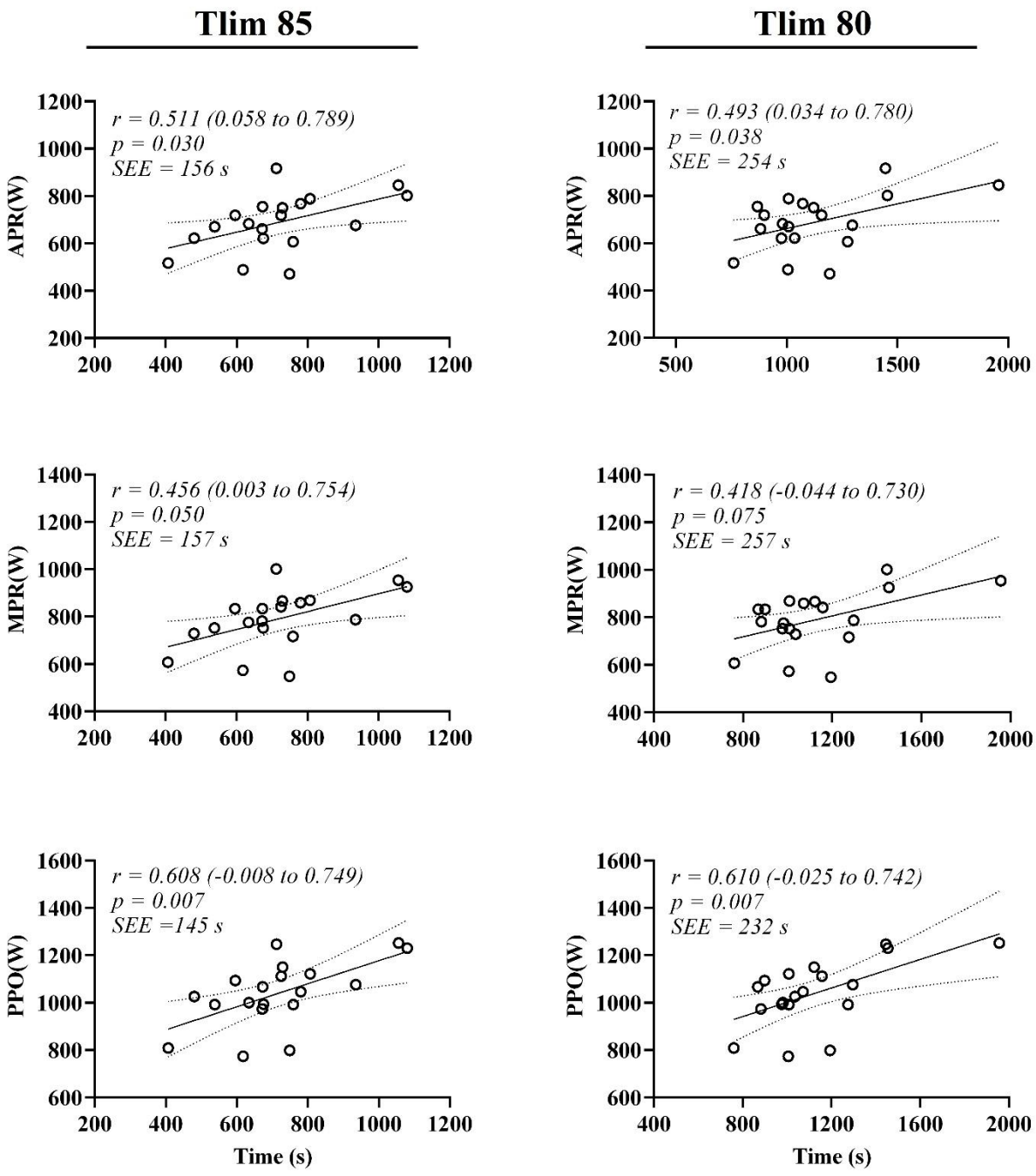
and **3b**). When expressing the data normalized to body mass, only the relationships with Tlim100 remained significant (**Table 3b**).

Figure 2b Relationships between anaerobic/maximal power reserve and time to exhaustion at 130%,115%, and 100% of maximal aerobic power.



Notes *Tlim130, Time to exhaustion (Tlim) at 130% of maximal aerobic power (MAP); Tlim115, Tlim at 115% of MAP; Tlim100, Tlim at 100% of MAP; Tlim85, Tlim at 85% of MAP; Tlim80, Tlim at 80% of MAP; APR, anaerobic power reserve; MPR, maximal power reserve. PPO, peak power output; SEE values represent the estimated standard error associated with the time estimate using APR/MPR/PPO as the independent variable.*

Figure 3b. Relationships between anaerobic/maximal power reserve and time to exhaustion at 85 and 80% of maximal aerobic power.



Notes *Tlim85, Time to exhaustion at 85% of maximal aerobic power (MAP); Tlim80, Time to exhaustion at 80% of MAP; APR, anaerobic power reserve; MPR, maximal power reserve. PPO, peak power output; SEE values represent the estimated standard error associated with the time estimate using APR/MPR/PPO as the independent variable.*

APR displayed a correlation with MAOD (r = 0.502, p = 0.029), and with W' derived by the 2-parameter hyperbolic model ($W'_{2\text{-hyp}}$) (r = 0.464, p = 0.045), but not with W' derived by the 3-

parameter model ($W'_{3\text{-hyp}}$). MPR shows significant associations with MAOD ($r = 0.542$, $p = 0.016$) and $W'_{2\text{-hyp}}$ ($r = 0.527$, $p = 0.020$) but not with $W'_{3\text{-hyp}}$. PPO was significantly related to both MAOD ($r = 0.560$, $p = 0.013$) and $W'_{2\text{-hyp}}$ ($r = 0.603$, $p = 0.006$), but it was not correlated with $W'_{3\text{-hyp}}$.

Table 3b. Pearson correlations between Anaerobic/Maximal Power Reserve normalized to body mass and Exercise Tolerance

	APR (W·kg^{-1})		**MPR (W·kg^{-1})**		**PPO (W·kg^{-1})**	
	r (95% CI)	*p*	*r (95% CI)*	*p*	*r (95% CI)*	*p*
Tlim130	0.597 (0.232 to 0.836)	0.009	0.584 (0.207 to 0.827)	0.011	0.356 (-0.111 to 0.721)	0.147
Tlim115	0.606 (0.174 to 0.858)	0.008	0.602 (0.146 to 0.853)	0.008	0.407 (-0.112 to 0.757)	0.093
Tlim100	0.622 (0.078 to 0.849)	0.006	0.643 (0.047 to 0.860)	0.004	0.594 (-0.081 to 0.844)	0.009
Tlim85	0.276 (-0.196 to 0.677)	0.257	0.264 (-0.248 to 0.676)	0.290	0.264 (-0.323 to 0.705)	0.290
Tlim80	0.284 (-0.366 to 0.681)	0.253	0.269 (-0.415 to 0.665)	0.280	0.333 (-0.369 to 0.715)	0.177

Notes *Tlim130, Time to exhaustion at 130% of maximal aerobic power (MAP); Tlim115, Time to exhaustion at 115% of MAP; Tlim100, Time to exhaustion at 100% of MAP; Tlim85, Time to exhaustion at 85% of MAP; Tlim80, Time to exhaustion at 80% of MAP; APR, anaerobic power reserve; MPR, maximal power reserve; PPO, peak power output.*

The partial correlation approach revealed that by fixing PPO, the relationships between APR, MPR, and MAOD became non-significant. While the previously non-significant correlations between APR and both $W'_{2\text{-hyp}}$ and $W'_{3\text{-hyp}}$ became significant, with negative slopes. In contrast, the association between MPR and both $W'_{2\text{-hyp}}$ and $W'_{3\text{-hyp}}$ remains non-significant. When partial correlations were employed to account for CP, relationships between PPO and MAOD remained significant, and a similar but not significant trend was observed when MAP was fixed. Finally, when CP or MAP was fixed, the relationships between PPO and $W'_{2\text{-hyp}}$ became non-significant (**Table 4b**).

Table 4b. Partial Correlations Between Anaerobic/Maximal Power Reserve, Exercise Tolerance, Anaerobic Capacity, and Work Above Critical Power

	Partial Correlation							
	APR (Controlling for PPO)		MPR (Controlling for PPO)		PPO (Controlling for CP)		PPO (controlling for MAP)	
	r (95% CI)	*p*	*r (95% CI)*	*p*	*r (95% CI)*	*p*	*r (95% CI)*	*p*
Tlim130	0.467 (-0.192 to 0.853)	0.059	0.606 (0.055 to 0.884)	0.010	0.746 (0.542 to 0.901)	<0.001	0.703 (0.406 to 0.903)	0.002
Tlim115	0.363 (-0.241 to 0.759)	0.152	0.508(-0.021 to 0.811)	0.037	0.757 (0.594 to 0.888)	<0.001	0.728 (0.505 to 0.873)	0.001
Tlim100	-0.253 (-0.637 to 0.197)	0.328	-0.151 (-0.519 to 0.276)	0.564	0.729 (0.522 to 0.916)	0.001	0.792 (0.610 to 0.930)	<0.001
Tlim85	-0.255 (-0.716 to 0.293)	0.323	-0.261 (-0.765 to 0.346)	0.312	0.348 (-0.113 to 0.656)	0.157	0.451 (0.041 to 0.779)	0.070
Tlim80	-0.333 (-0.684 to 0.026)	0.192	-0.435 (-0.768 to -0.139)	0.081	0.279 (-0.273 to 0.647)	0.263	0.428 (-0.277 to 0.774)	0.086
MAOD	-0.222 (-0.616 to 0.218)	0.376	-0.143 (-0.608 to 0.383)	0.934	0.488 (-0.033 to 0.791)	0.040	0.428 (-0.270 to 0.880)	0.076
$W'_{3\text{-hyp}}$	-0.531 (-0.770 to 0.147)	0.023	-0.335 (-0.671 to 0.052)	0.314	0.148 (-0.386 to 0.534)	0.559	-0.016 (-0.548 to 0.426)	0.949
$W'_{2\text{-hyp}}$	-0.506 (-0.860 to 0.108)	0.032	-0.269 (-0.695 to 0.141)	0.100	0.430 (-0.006 to 0.749)	0.070	0.358 (-0.062 to 0.690)	0.145

Notes *Tlim130, Time to exhaustion at 130% of maximal aerobic power (MAP); Tlim115, Time to exhaustion at 115% of MAP; Tlim100, Time to exhaustion at 100% of MAP; Tlim85, Time to exhaustion at 85% of MAP; Tlim80, Time to exhaustion at 80% of MAP; MAOD, maximal accumulated oxygen deficit; $W'_{3\text{-hyp}}$, work above crtitical powerderived from the 3 parameters hyperbolic model; $W'_{2\text{-hyp}}$, work above critical powerderived from the 2 parameters hyperbolic model; APR, anaerobic power reserve; MPR, maximal power reserve; CP, Critical power derived from the 3 parameters hyperbolic model; PPO, peak power output.*

DISCUSSION

APR/MPR and Exercise Tolerance

The present pilot study investigated the relationship between APR/MPR and exercise tolerance across various exercise intensities in endurance-trained male cyclists. We further explored potential associations between APR/MPR, anaerobic capacity, and work above critical power. Our initial findings revealed significant correlations between both APR/MPR (in absolute values) and all Tlims but Tlim80. These results align with an early study that observed a large correlation between running time to exhaustion at intensities spanning from 90% to 140% of maximal aerobic speed with both ASR and maximal speed reserve (the running counterpart of our MPR) (Blondel et al., 2001). Since a high APR/MPR can be achieved through either a high PPO or a low MAP/CP, a partial correlation approach was employed to better understand their independent contributions to exercise tolerance. Fixing PPO, only the relationship between MPR, Tlim130, and Tlim115 remained statistically significant, and all the relationships between APR and Tlims became non-significant. Fixing MAP/CP instead revealed that a higher PPO, and *ipso facto***,** higher APR/MPR were positively associated with greater exercise tolerance for Tlim100, Tlim115, and Tlim130. Moreover, positive relationships between Tlim130 and Tlim115 emerged with their corresponding AOD, indicating a greater reliance on anaerobic pathways at these intensities (see also *supplementary materials*). Collectively, these findings support the rationale for APR/MPR as a metric to assess anaerobic characteristics in endurance male cyclists, and the strong correlation between APR/MPR and supramaximal Tlims aligns with previous research demonstrating the high accuracy of the APR in predicting sprint and supramaximal efforts in healthy individuals (Weyand et al., 2006) and professional male road cyclists (Sanders & Heijboer, 2019). Additionally, these results reinforce the notion that MAP, an aerobic marker, is insufficient to explain exercise tolerance when anaerobic contributions exceed approximately 20% of the total energy demand, as seen during supra-maximal Tlims (Blondel et al., 2001). In contrast, incorporating APR/MPR, indirect markers of anaerobic characteristics, offers a more comprehensive explanation for

the observed variance in exercise tolerance. Regarding Tlim85 and Tlim80, a trend toward significant relationships highlighted how fixing MAP/CP, having a higher APR/MPR, likely leads to a greater exercise tolerance also during longer trials. This aligns with a recent study in well-trained track cyclists, in which a very large positive correlation between PPO and various maximal mean powers up to 20 minutes was observed (Ferguson et al., 2023). Nevertheless, the magnitude of these correlations decreases during sub-maximal Tlims, likely due to their lower relative anaerobic contribution (<10%).

Noteworthy, the positive association between MPR and Tlim80 became negative after controlling for PPO using partial correlations, although it did not reach statistical significance. This seemingly counterintuitive finding could be explained by the influence of this approach. By fixing PPO, athletes with higher CP would inherently have lower MPR. Consequently, the observed negative correlation might indirectly reflect the well-established principle that, for longer exercise durations, athletes with higher CP will demonstrate greater exercise tolerance when intensity is based on MAP. However, it has also been observed that for these submaximal Tlims, the body mass plays a substantial role in driving such correlation, given that when APR/MPR or PPO are normalized to body mass, the relationships become non-significant. Therefore, the correlation between APR/MPR and submaximal Tlim must be interpreted with caution.

Finally, despite these power reserves seeming to account for a substantial proportion of the variance in supramaximal Tlims, the wide confidence intervals and elevated SEE caution against relying solely on these metrics for individualized exercise prescription. Therefore, using this parameter as a yardstick for prescribing Tlims may not completely resolve the high variance classically associated with Tlims.

Pearson correlations between PPO and Tlim were also significant for all Tlims. Moreover, although it would have been more predictable that the correlation strength would decrease between PPO and Tlim as the power of Tlim decreases, we observed the strongest correlation at Tlim100. While the reason remains unclear, we propose that task familiarity may underpin this finding. Cyclists more often

perform high-intensity efforts near 100% MAP, which typically elicit exhaustion within ~5 minutes (matching Tlim100 durations). In contrast, supramaximal efforts (Tlim115 and Tlim130) are less familiar, both physiologically and psychologically, potentially introducing variability in exercise tolerance.

APR/MPR, Anaerobic Capacity, and Work Above Critical Power

MPR displayed a stronger relation with W'_{2hyp}, compared to APR. This discrepancy might be due to the neglect of anaerobic contribution by the APR approach within the power range between CP and the MAP achievement during the ramp test. In fact, this zone can be considered as a sort of anaerobic work capacity. Since exhaustion during the ramp test theoretically occurs only when the finite work capacity is depleted (beyond CP), using MAP to define the lower boundary of APR might be a less rigorous approach. Somewhat counterintuitively, when PPO's influence was fixed, the previous positive significant correlation between APR and $W'_{2\text{-}hyp}$ became negative. This can be explained by the positive correlation between $W'_{2\text{-}hyp}$ and MAP (see *supplementary materials*). Indeed, fixing PPO, athletes with higher MAP will have lower APR and simultaneously greater $W'_{2\text{-}hyp}$ and $W'_{3\text{-}hyp}$. In addition, when controlling for MAP/CP, higher APR/MPR values in our endurance male cyclist sample were not associated with higher $W'_{2\text{-}hyp}$. Therefore, these results confirm that the work capacity above CP and APR/MPR are two distinct parameters and highlight caution when interpreting APR/MPR as a measure of work that can be performed within the severe intensity domain. This result may also explain why no significant reduction in heterogeneity of exercise tolerance and physiological responses was observed in previous studies, involving male participants, when intensity is prescribed according to APR (Barnett et al., 1996; Julio et al., 2020; Collison et al., 2021; Bok et al., 2023).

Despite both APR and MPR displayed significant positive correlations with MAOD, the partial correlations revealed that these results were primarily driven by the influence of PPO. This suggests that when PPO is fixed, variations in CP or MAP do not significantly influence MAOD. Conversely,

when CP, but not MAP, is fixed, a higher PPO is likely associated with a greater MAOD. This discrepancy could again be attributed to the mixed metabolic contributions at MAP.

Finally, considering these results collectively, the divergence between MPR and APR probably stems from the latter's reliance on MAP as a lower boundary. MAP is not only inherently protocol-dependent but also represents an intensity that cannot be sustained beyond ~5 minutes due to its substantial anaerobic pathway involvement (Blondel et al., 2001). These dual limitations conflate sustainable and unsustainable domains in the "grey zone" between CP and MAP. In contrast, MPR circumvents this ambiguity by anchoring its lower boundary to CP, a well-established threshold for sustainable metabolic steady state, thereby providing a more stable and physiologically coherent model for distinguishing power domains.

The present study has some limitations. First, we enrolled exclusively male cyclists. Considering the well-documented sex-related differences in anaerobic capacity (Weber & Schneider, 2000), muscle fiber type composition (McDougall et al., 2023; Vanhatalo et al., 2016), and neuromuscular recruitment patterns (Ansdell et al., 2020; Hunter, 2016), it is plausible that female athletes may display different responses. Although males have a greater W', several studies also show greater variability in male W' than in female W', even when normalized by lean body mass and its variability is not different between sexes, several studies have reported consistent (Caswell et al., 2024) or even lower variability in W′ among females (Bourgois et al., 2023; McDougall et al., 2023). Therefore, further studies investigating sex differences surrounding the APR/MPR are needed. Second, a limited number of familiarization sessions. Due to the high physical demands placed on participants, who were required to complete seven laboratory sessions (all until exhaustion), we limited the number of Tlim familiarization sessions to two. These sessions were designed to approximate the conditions of the actual trials but may not have fully replicated them. This could explain the unexpected finding of a stronger correlation between PPO and Tlim100 compared to Tlim130. Finally, this study focused only on endurance-trained cyclists; therefore, our results cannot be broadened to other cycling disciplines involving distinct physiological

demands characterized by a higher reliance on anaerobic metabolism and explosive power output, such as track cycling.

Conclusions

This pilot investigation revealed that, in endurance-trained male cyclists, both APR and MPR are closely related to exercise tolerance at various intensities, particularly for shorter efforts (under 5 minutes). However, for longer durations, their role decreases. MPR displayed a stronger link with both mechanical and physiological measures of anaerobic capacity compared to APR. This suggests a potential bias when using MAP as the lower boundary for APR calculations. Furthermore, the association between exercise tolerance and APR/MPR seems mainly driven by the role of the PPO rather than the lower boundary used to define the power reserve. This study, however, included a single sex, thus limiting the generalizability of results and highlighting the need for future research including female cyclists.

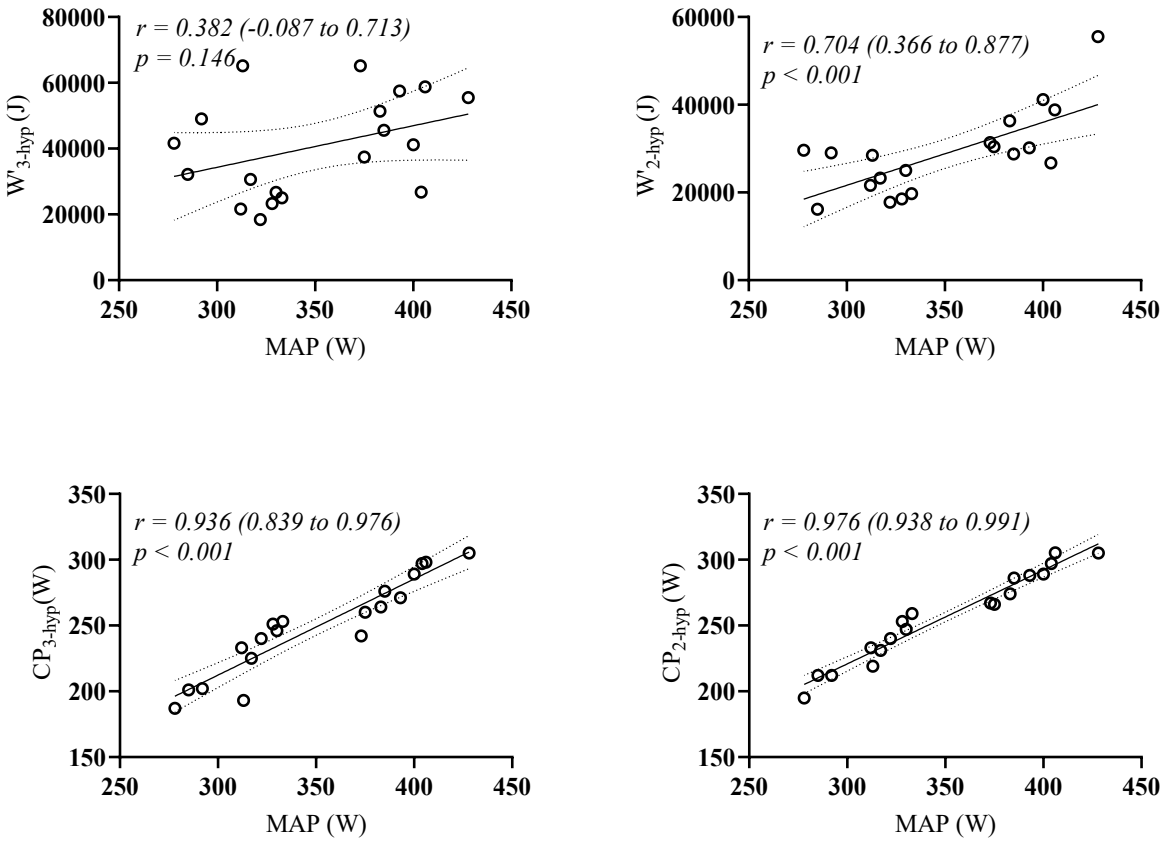
Supplementary materials

Statistical Analyses

Data normality was assessed using the Shapiro-Wilk test. Descriptive statistics are presented as mean ± standard deviation (SD). Pearson's correlation was used to examine the relationship between all measured parameters. Pearson correlations with 95% confidence intervals (CIs) were calculated. All analyses were performed using IBM SPSS Statistics for Windows, version 26.0 (IBM Corp., Armonk, NY, USA).

Results

Figure S1. Correlations between power-duration relationship parameters and maximal aerobic power.



Abbreviations: *MAP, maximal aerobic power; $W'_{2\text{-}hyp}$, anaerobic work capacity derived from the 2 parameters hyperbolic model; $CP_{2\text{-}hyp}$, Critical power derived from the 2 parameters hyperbolic model; $CP_{3\text{-}hyp}$, Critical power derived from the 3 parameters hyperbolic model; $W'_{3\text{-}hyp}$, anaerobic work capacity derived from the 3 parameters hyperbolic model. Pearson's correlation was used to examine the relationship between all measured parameters. 95% confidence intervals (CIs) were shown.*

Table S1. Correlations between power-duration relationship parameters, accumulated oxygen deficit, and exercise tolerance across intensities.

	$CP_{3\text{-hyp}}$		$W'_{3\text{-hyp}}$		$CP_{2\text{-hyp}}$		$W'_{2\text{-hyp}}$		AOD	
	r	*p*	*r*	*p*	*r*	*p*	*r*	*p*	*r*	*p*
Tlim130	-0.009	0.971	0.112	0.648	-0.020	0.935	0.394	0.095	0.724	0.001
Tlim115	0.139	0.571	0.182	0.456	0.113	0.645	0.467	0.044	0.674	0.002
Tlim100	0.578	0.009	0.500	0.029	0.584	0.009	0.698	0.001	0.288	0.231
Tlim85	0.517	0.023	0.485	0.035	0.493	0.032	0.825	<0.001	-0.039	0.875
Tlim80	0.627	0.004	0.400	0.090	0.620	0.005	0.572	0.011	-0.223	0.359
MAP	0.936	<0.001	0.382	0.146	0.976	<0.001	0.704	0.001	N/A	N/A

Abbreviations: *$CP_{3\text{-hyp}}$. Critical power derived from the 3-parameter hyperbolic model; $W'_{3\text{-hyp}}$. anaerobic work capacity derived from the 3 parameters hyperbolic model; $CP_{2\text{-hyp}}$. Critical power derived from the 2-parameter hyperbolic model; $W'_{2\text{-hyp}}$. anaerobic work capacity derived from the 2 parameters hyperbolic model; AOD. accumulated oxygen deficit. Tlim130. Time to exhaustion (Tlim) at 130% of maximal aerobic power (MAP); Tlim115. Tlim at 115% of MAP; Tlim100. Tlim at 100% of MAP; Tlim85. Tlim at 85% of MAP; Tlim80. Tlim at 80% of MAP. Pearson's correlation was used to examine the relationship between all measured parameters.*

STUDY 3

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The Modified 3-Minute All-Out Test Parameters as Predictors of 50-, 100-, and 200-m Front Crawl Official Performance in Trained Swimmers

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Abstract

In this study we: i) investigated the relationship between the modified 3-minute all-out test ($3mAO_{mod}$) parameters and swimming official performance in 50-, 100-, and 200-meter front crawl, ii) examined the predictive accuracy of critical speed (CS) model, iii) the predictive linear regression (MLR) models incorporating CS, curvature constant (D') or anaerobic speed reserve (ASR), and anthropometry. 23 young swimmers (mean ± standard deviation: age = 17.5 ± 1.9 years, mass = 61.8 ± 7.2 kg, height 1.79 ± 0.08 m) performed the $3mAO_{mod}$ and, within 6 weeks, competed in 50-, 100-, and 200-meter official races. The relationships between $3mAO_{mod}$ parameters and race times were examined using Pearson's and Spearman's correlation. Race times were predicted using the CS model and MLR, including CS, D', or ASR, sex, age, height, and body mass. Mean absolute error (*MAE*) and paired sample t-tests with Bonferroni corrections were used to compare actual and predicted results. Significance was set at $p \leq 0.05$. The correlation between CS and race time increased with the increase in race distance (r = -0.491 to -0.698) while correlations between D' or ASR with race time decreased with the increase in race distance (*rho* = -0.628 to -0.347 and -0.574 to -0.274, respectively). The CS model underestimates race times in the 50- and 200-meter races ($p \leq 0.001$, *MAE* = ±3.33 and ±5.06 s, respectively), but not in the 100-meter race ($p = 0.070$, *MAE* = ±1.72 s). Predictions from MLRs were not significantly different from actual performance in all events ($p = 0.999$, ±0.41 s < *MAE* < ±3.56 s). The $3mAO_{mod}$ parameters

are related to 50-, 100-, and 200-meter performance, supporting their physiological rationale, and when applied to the developed MLRs, can predict performance with higher accuracy compared to the CS model, and in a user-friendly manner.

Keywords:

swimming, modeling, critical speed, anaerobic speed reserve, curvature constant.

Introduction

The speed (or power) -duration relationship is widely used across various endurance sports to describe the link between the speed and the time for which that intensity can be sustained (Hill, 1993). This relationship follows a hyperbolic-like pattern, tending towards an asymptote at intensities close to the maximal lactate steady state (Iannetta et al., 2022). This relationship can be described by several models, such as the power law model, the anaerobic power reserve model, or the critical speed (CS) model (Drake et al., 2024; Hill, 1993; Sandford et al., 2021). The latter, in particular, is one of the most used and it allows the estimation of: i) CS, defined as the theoretical maximal intensity that can be sustained indefinitely and where physiological parameters such as oxygen uptake, lactate, and phospocreatine can still stabilize; ii) the so-called curvature constant (D'), which represents the distance an athlete can perform beyond CS, often interpreted as measure of anaerobic work capacity (Hill et al., 1995). In swimming contexts, considering the greater challenges in performing gas exchange and lactate analysis to delineate the physiological thresholds, the CS model, despite being indirect, offers a valid and feasible assessment of the boundary between the heavy and severe intensity domains and allows an accurate performance prediction (Hill et al., 1995).

A significant limitation of this model is that it requires the athlete to perform multiple maximal trials over various distances to estimate CS and D'. This practice is highly invasive within an athlete's training program. To overcome this problem, the so-called 3-minute all-out test was introduced, first

in cycling, then in running, and, more recently, also in swimming, where it can be applied in a 25-meter pool using a modified, intermittent-based version ($3mAO_{mod}$) (Mitchell et al., 2018). This test enables the estimation of CS and D' with high precision compared to the standard approach, with a duration of approximately 4 minutes. Additionally, the $3mAO_{mod}$ allows the determination of peak velocity (Mitchell et al., 2018) and, consequently, a recently introduced easy-to-use proxy of anaerobic characteristics, namely the anaerobic speed reserve (ASR) (Sandford et al., 2021). The ASR was originally defined as the difference between the maximal sprinting speed and the speed associated with maximal oxygen uptake (vVO_2max) (Sandford et al., 2021). However, some studies suggest that using CS to define the lower boundary provides a parameter more related to D' compared to the original approach, and it is also strongly related to exercise tolerance within the severe and extreme intensity domains (Dalamitros et al., 2015; Kramer et al., 2021; Di Gennaro et al., 2025b). Although ASR has been extensively studied in running and cycling (Sandford et al., 2021), evidence in swimming is still limited. To date, the relationship between ASR and D’ in swimming has been investigated in only one study. The researchers observed a strong correlation between them (Dalamitros et al., 2015; Kramer et al., 2021), suggesting ASR as a straightforward and practical parameter both for race time prediction and exercise intensity prescription (Dalamitros et al., 2015; Kramer et al., 2021). But it is still unclear if this parameter can be used as an alternative to D’ for swimming performance prediction.

Moreover, the validity of the 3-minute all-out test has also been contested, with evidence suggesting it overestimates both performance and the true maximal metabolic steady state across cycling and running (Bergstrom et al., 2013a; Nicolò et al., 2017). Nevertheless, CS (alone or in combination with D') can accurately predict longer performances such as 400-meter front crawl (Zacca et al., 2016). However, predictive accuracy for shorter distances spanning 50 to 200 meters remains unknown. Therefore, given this ongoing debate, coupled with the scarcity of data validating the $3mAO_{mod}$ performance prediction accuracy in swimming, we investigated the relationships between the $3mAO_{mod}$-derived parameters and 50-, 100-, and 200-meter front crawl official race times, and we

evaluated the predictive ability of the CS model. Additionally, integrating the critical influence of anthropometry in swimming performance (Lätt et al., 2010), and the parallelism between ASR and D', we also developed linear equations incorporating CS, D', or ASR, and anthropometric variables for predicting race times across the 50-, 100-, and 200-meter front-crawl distances.

Methods

Experimental Approach to the Problem

One week after a previous familiarization with the test, all participants completed the $3mAO_{mod}$ in a 25-m pool at the same time of the day (± 1h) in an indoor swimming pool with a water temperature of 27°C. The test was performed one week before the summer competition season. Anthropometric assessment in terms of barefoot subjects' stature and body mass was also collected (Seca 217, Vogel & Halke, Hamburg, Germany). Then, in the next 6 weeks, participants competed in three official front crawl events (50, 100, and 200-m) held in a 50-m swimming pool. Race times were recorded and used for correlation analyses and predictive modelling of performance.

Subjects

An *a-priori* power analysis was performed using G*Power (version 3.1.9.7; Heinrich Heine University, Düsseldorf, Germany) to estimate the required sample size. Based on data from a previous study investigating the predictive ability of the 3-minute all-out test in cycling performance (Nicolò et al., 2017), a point-biserial correlation (t-test family) with an effect size of $r = 0.60$, $\alpha = 0.05$, and statistical power = 0.80 was used. The calculations indicated a minimum sample size of 21 participants.

Twenty-three young trained swimmers (13 females and 10 males) were recruited (males: median [minimum, maximum] age = 19 [16, 20] years; mean ± standard deviation (SD), mass = 64.6 ± 8.2 kg: height 1.74 ± 0.08 m); females: age = 17.4 ± 1.4 years, mass = 59.3 ± 5.3 kg; height = 1.66 ± 0.06 m (16). The FINA points were calculated by the Federation Internationale de Natation (FINA) software (Software© 2009 by GeoLogix) (FINA points 100-m front crawl for males: 495 ± 75; for females 515

± 91). All participants regularly trained 4-5 sessions per week during the last year. None of them reported muscular or orthopedic injuries or health problems in the previous six months. To standardize testing conditions, participants were instructed to abstain from food and caffeine consumption in the 3-4 hours preceding the test and not exercise in the previous 24 hours (Tsai & Thomas, 2017). All participants received a detailed explanation of the study's objectives and procedures and provided their written informed consent (and when minors, also that of their parents). The research adhered to the Declaration of Helsinki, with prior approval from the Local Ethics Committee (University of Genova, approval n° 2024/62).

Procedures

The Modified 3-Minute All-Out Test

Before the $3mAO_{mod}$ test, swimmers completed a 10-minute low-intensity warm-up, including activation drills. Then, they engaged in a 5-minute passive rest before starting the $3mAO_{mod}$. The test was conducted according to the protocol outlined by (Mitchell et al., 2018). Participants performed 12 all-out 25-m repetitions interspersed by 5 seconds of passive rest. Athletes were instructed to attempt to surpass their personal best time for each 25-m segment. The time for each segment was measured by a member of the research team, who is also a swimming coach with ≥20 years of experience in the field, using a handheld stopwatch (3X300m Stopwatch, Finis).

Data Analysis

CS and D' were determined according to Mitchell et al. (2018). The mean speed for each 25-m segment was calculated, and subsequently, non-linear regression analysis was performed to fit an exponential decay model to the relationship between the speed of each pool ($S_{(t)}$) and the cumulative time (t) across the entire $3mAO_{mod}$: $S_{(t)} = A \cdot e^{-bt} + c$.

A and b represent weighting factors, and c is the model's asymptote. CS was defined as the average speed achieved during the two slowest 25-m segments within the final four repetitions of the $3mAO_{mod}$.

D' was calculated as follows: $(t_1 \cdot S_1) - (CS \cdot t_1) + \left(\int_{t_{12}}^{t_1} A \cdot e^{-bt} + c\right) - CS$.

Here, t_1 is the time to complete the first 25 m, and t_{12} is the time taken to complete all 12 segments. All fitting procedures were performed using MATLAB R2023b (The Mathworks Inc., Natick, MA, USA). The race times predictions using the CS model have been calculated by solving the 2-parameter using D' and CS obtained from the $3mAO_{mod}$ as follows:

$$t = \frac{Race_{dist} - D'}{CS}$$

Where t is the estimated time and race $Race_{dist}$ is the distance of the race (i.e. 50,100, or 200-m). The anaerobic speed reserve (ASR) has been calculated as the difference between the peak velocity (first pool mean velocity) and the CS (Blondel et al., 2001).

50-, 100-, 200-meter front Crawl Official Race

Within 6 weeks following the $3mAO_{mod}$ test, the participants conducted 50-, 100-, and 200-meter front crawl official races in a standard 50-m pool. Before the races, swimmers performed 15 minutes of warm-up, consisting of 5 minutes of dryland drills followed by 5 minutes of low-intensity swimming, to conclude with 5 minutes at their preferred pace, including brief sprints of 2-3 seconds (Neiva et al., 2014). From the end of the warm-up to the start of the competition, there was a time lag spanning from 10 to 30 minutes. The race time has been subsequently collected from the Italian swimming federation website (https://www.federnuoto.it/).

Statistical Analysis

The Shapiro-Wilk test was applied to assess the distribution of the variables. Descriptive statistics for normally distributed variables are presented as mean ± SD, while for the ones not normally distributed, as medians with range [minimum, maximum]. The coefficient of variation and the intraclass correlation coefficient (ICC) were calculated to assess the reliability of the $3mAO_{mod}$ parameters compared to the familiarization session (Koo & Li, 2016). Pearson correlation coefficients (r) were

calculated to determine relationships among variables. Correlation coefficients of >|0.1|, >|0.3|, >|0.5|, and >|0.7| were considered small, moderate, large, and very large (Hopkins et al., 2009). Height, ASR, and D' violated normality assumptions; consequently, Spearman's correlation was applied. Multiple linear regression (MLR) analysis was applied to predict 50-, 100-, and 200-m performance using CS, D', sex (female =1; male = 0), age, body mass, and body height as independent variables ($MLR_{D'}$). Considering the practical challenges of determining D' in the field, a further MLR analysis was conducted using ASR instead of D' (MLR_{ASR}). To assess the potential for multicollinearity, we calculated the variance inflation factor (VIF) for all independent variables, confirming that no value exceeded the threshold of 10 (Belsley et al., 2005). We also performed a variance decomposition analysis, which confirmed that no two variables exhibited a variance proportion above 0.9 for any given dimension (Belsley et al., 2005; Hair et al., 2013). Together with the R^2, the adjusted R^2 (*adj* R^2) has been calculated to account for the number of predictors. Standard error of estimation (*SEE*) and mean absolute error (*MAE*) were calculated to evaluate the prediction accuracy of the models. A paired sample t-test with Bonferroni corrections was used to compare the actual and predicted results. Mean bias and 95% limits of agreement were presented in the Bland-Altman plots to graphically show the differences between predicted and actual race times. Statistical significance was set at $\alpha = 0.05$ (two-tailed). Finally, to examine proportional bias, correlation analyses between bias and actual race times and $3mAO_{mod}$ parameters were performed. All statistical analyses were carried out using IBM SPSS Statistics for Windows, version 26.0 (IBM Corp., Armonk, NY, USA).

Results

The mean CS was 1.45 ± 0.10 $m \cdot s^{-1}$, the median D' was 15.57 [5.73, 29.92] m, and ASR was 0.32 [0.20, 0.41] $m \cdot s^{-1}$. There was no sex difference in 100m FINA points (males: 495 ± 74 vs females: 515 ± 91, $p = 0.670$). Nevertheless, males presented higher CS (1.51 ± 0.07 $m \cdot s^{-1}$ vs 1.39 ± 0.09 $m \cdot s^{-1}$, $p = 0.002$), peak velocity (1.90 ± 0.08 $m \cdot s^{-1}$ vs 1.67 ± 0.09 $m \cdot s^{-1}$, $p < 0.001$), D' (17.60 ± 7.34 m vs 11.66 ± 4.98 m, $p = 0.044$), ASR (0.39 ± 0.12 $m \cdot s^{-1}$ vs 0.28 ± 0.07 $m \cdot s^{-1}$, $p = 0.025$) compared to females.

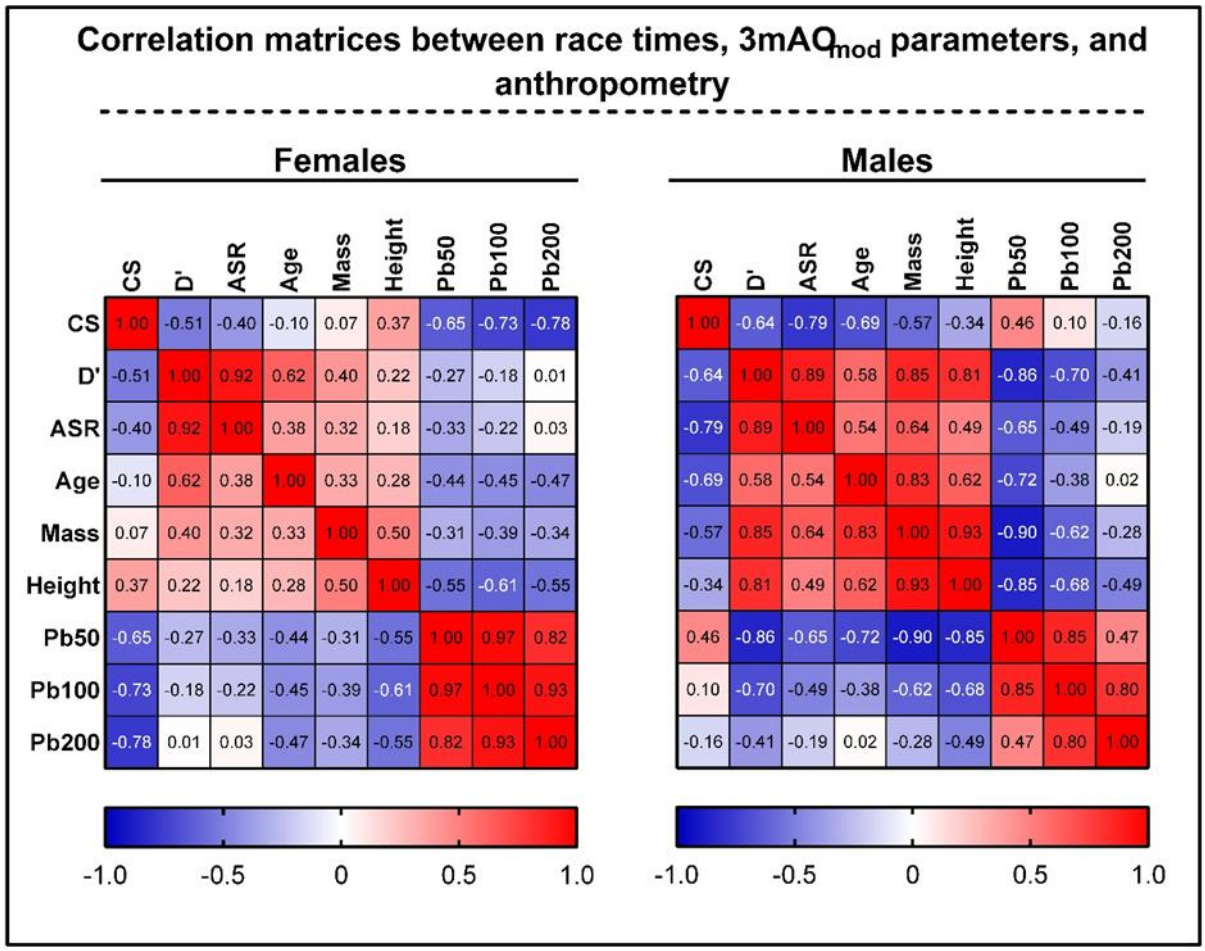
Coefficient of variation for CS, peak velocity, ASR, and D' was 1.85, 1.08, 12.18, and 14.24 %, respectively; while ICC was 0.864, 0.974, 0.809, and 0.870. Correlation analyses among race times, performance parameters, and anthropometry are shown in **Table 1c**. Additionally, sex-specific correlations are illustrated in the matrices shown in **Figure 1c.**

Table 1c. Correlation coefficients among race times, 3mAO$_{mod}$ parameters, and anthropometry.

		Pb50	Pb100	Pb200	CS	D'	ASR
CS	*r*	-0.491	-0.680	-0.698			
	p	0.017	<0.001	<0.001			
D'	*rho*	-0.628	-0.472	-0.347	-0.153		
	p	0.001	0.023	0.105	0.485		
ASR	*rho*	-0.574	-0.467	-0.274	-0.274	0.818	
	p	0.004	0.025	0.206	0.738	<0.001	
Age	*r*	-0.504	-0.315	-0.188	-0.265	0.626‡	0.436‡
	p	0.014	0.143	0.390	0.223	0.001	0.038
Body mass	*r*	0.077	0.011	-0.147	0.059	-0.727‡	-0.528‡
	p	0.727	0.961	0.503	0.789	<0.001	0.010
Body height	*rho*	-0.779	-0.718	-0.706	0.382	0.608	0.431
	p	<0.001	<0.001	0.001	0.125	0.002	0.040

Abbreviations. *CS, critical speed. D', distance performed above CS. ASR, anaerobic speed reserve. Pb50, Pb100, and Pb200 are front crawl race times in 50-,100-, and 200-m, respectively. ‡: indicates that Spearman correlation was applied.*

Figure 1c. Correlation matrices divided by sex between $3mAO_{mod}$ parameters, anthropometry, and performance variables.



***Abbreviations**: 3mAO$_{mod}$, modified 3-minute all-out test; CS, critical speed; D', distance above CS; ASR, anaerobic speed reserve; Pb50, Pb100, and Pb200 are the official race times of 50,100, and 200m front crawl races, respectively. **Note**: in the female (n=13) analyses, only r < -0.55 or > 0.55 are statistically significant; in the male (n=10) analyses, only r < -0.64 or > 0.64 are statistically significant.*

Significant correlations between actual race times and those predicted by the CS model (50-m: r = 0.858, $p < 0.001$, *SEE* = 4.570 s, *MAE* = 3.328 s, *MAE* (%) = 12.50; 100-m: r = 0.940, $p < 0.001$, *SEE* = 2.371 s, *MAE* = 1.722 s, *MAE* (%) = 2.96; 200-m: r = 0.844, $p < 0.001$, *SEE* = 6.887 s, *MAE* = 5.058 s, *MAE* (%) = 3.75) were observed. However, these predictions are significantly lower than the actual race times for 50-m ($p < 0.001$) and 200-m ($p = 0.001$) but not for 100-m (p = 0.070).

The $MLR_{D'}$ provides the following equations:

1. 50-m race time = 61.846 – 15.726 · CS – 0.195 · D' + 0.69 ·sex – 0.379 · age + 0.002 · mass – 1.31 · height (R^2 = 0.924, *adj* R^2 = 0.895*;* $p < 0.001$, *SEE* = 0.672 s, *MAE* = 0.412 s, *MAE* (%) = 1.49);
2. 100-m race time = 131.492 – 42.849 · CS – 0.46 · D' + 1.443 · sex – 0.533 · age – 3.575 · height (R^2 = 0.943, *adj* R^2 = 0.920; $p < 0.001$. *SEE* = 1.277 s, *MAE* = 0.867 s, *MAE* (%) = 1.48);
3. 200-m race time = 308.866 – 104.822 · CS – 0.967 · D' + 7.035 · sex – 0.611 · age – 0.011 · mass – 1.374 · height (R^2 = 0.796, *adj* R^2 = 0.720; $p < 0.001$, *SEE* = 5.552 s, *MAE* = 3.295 s, *MAE* (%) = 2.48).

No significant difference appears between actual and $MLR_{D'}$ predicted race times (50-m: $p = 0.999$; 100-m: $p = 0.999$; 200-m: $p = 0.999$).

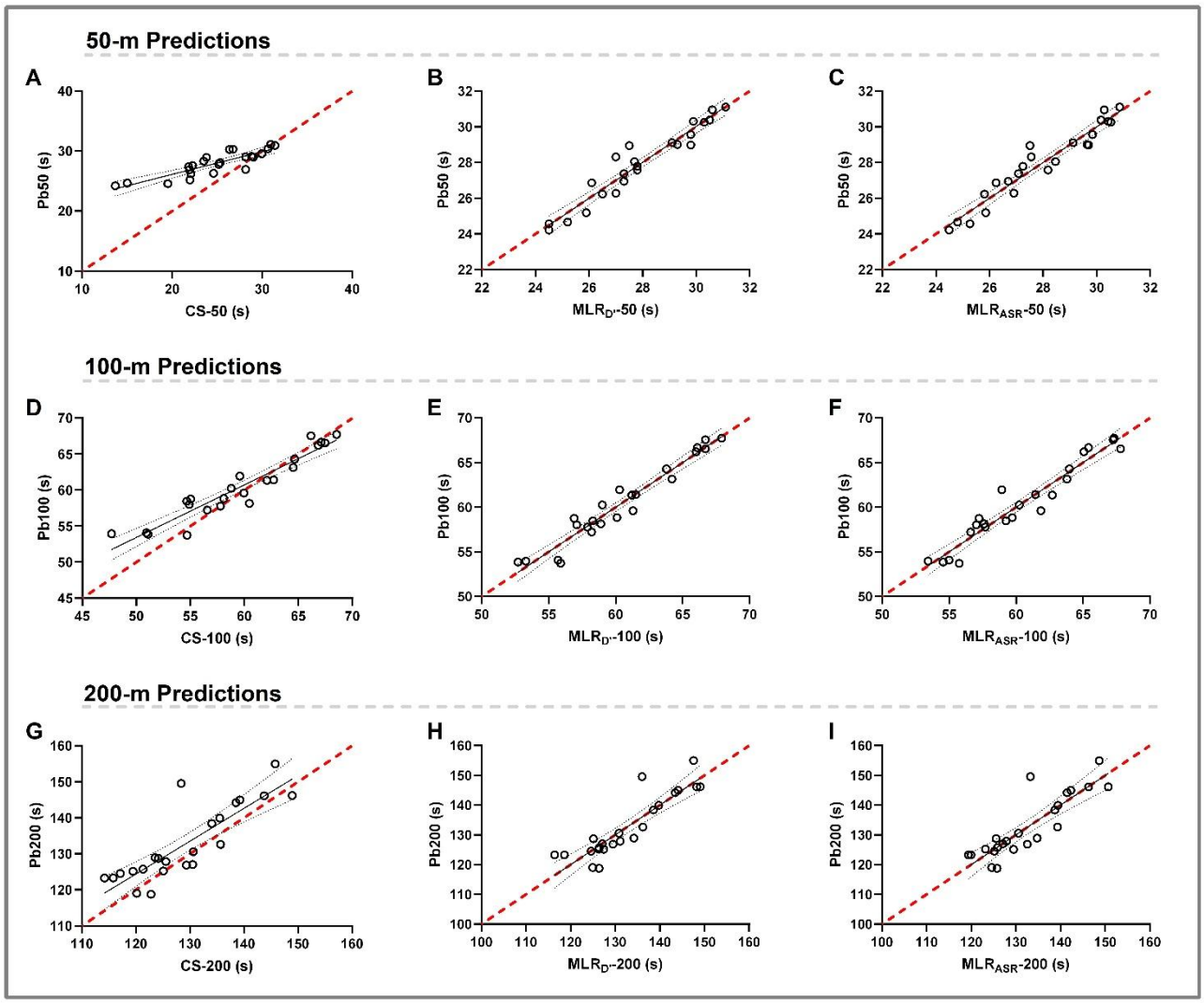
The MLR_{ASR} provides the following equations:

1. 50-m race time = 68.46 – 14.26 · CS – 9.687 ·ASR + 0.857 · sex – 0.402 · age + 0.002 · mass – 6.039 · height (R^2 = 0.924, *adj* R^2 = 0.895. $p < 0.001$, *SEE* = 0.673 s, *MAE* = 0.475 s, *MAE* (%) = 1.71);
2. 100-m race time = 146.786 – 38.553 · CS – 21.442 · ASR + 1.639 · sex – 0.596 · age + 0.001 · mass – 8.254 · height (R^2 = 0.928, *adj* R^2 = 0.901. $p < 0.001$, *SEE* = 1.419 s, *MAE* = 0.937 s, *MAE* (%) = 1.56);
3. 200-m race time = 339.121 – 90.292 · CS – 35.912 · ASR + 6.153 · sex – 0.802 · age – 0.009 · mass – 30.673 · height (R^2 = 0.755, *adj* R^2 = 0.663. $p < 0.001$, *SEE* = 6.089 s, *MAE* = 3.564 s, *MAE* (%) = 2.64).

There was no significant difference between actual and MLR_{ASR} predicted race times (50-m: $p = 0.999$; 100-m: $p = 0.999$; 200-m: $p = 0.999$).

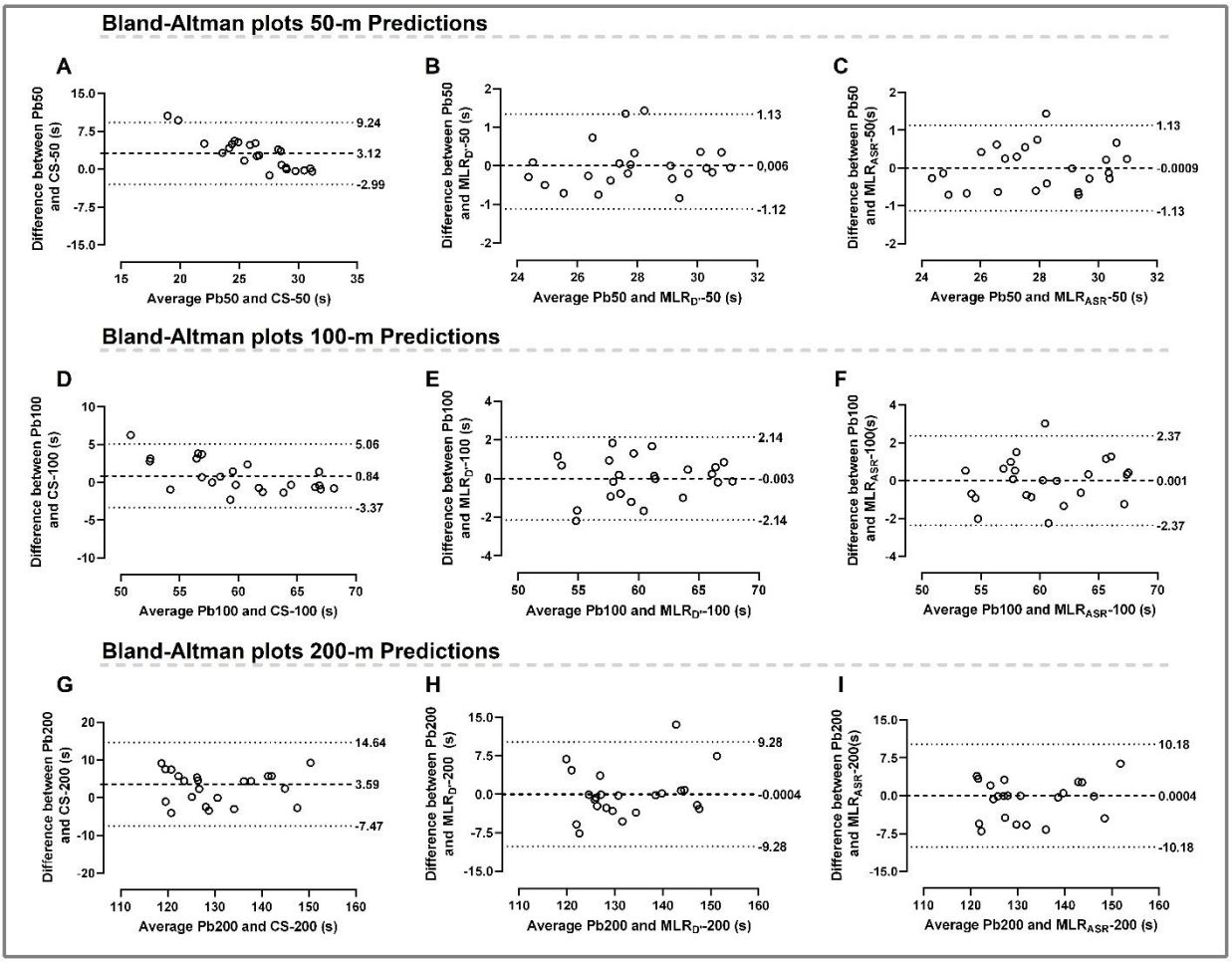
The relationships between predicted and actual race times and Bland-Altman plots are shown in **Figures 2c** and **3c**.

Figure 2c. Predicted versus actual race time.



Notes The red dashed line is the identity line (R = 1). ***Abbreviations*:** Pb50, Pb100, Pb200, front crawl race time in 50-,100-, and 200-m, respectively. CS-50, CS-100, and CS-200 are the race times predicted by the critical speed model. $MLR_{D'}$-50, $MLR_{D'}$-100, and $MLR_{D'}$-200 are the race times predicted by the multiple linear regressions (MLR) using D' (distance performed above CS) as anaerobic marker. MLR_{ASR}-50, MLR_{ASR}-100, and MLR_{ASR}-200 are the race times predicted by MLR using the anaerobic speed reserve as anaerobic marker.

Figure 3c. Bland-Altman plots of predicted vs actual race times. The dashed black line is the mean bias, and the dotted lines are the 95% confidence intervals



Abbreviations: *Pb50, Pb100, Pb200, front crawl race time in 50-,100-, and 200-m, respectively. CS-50, CS-100, and CS-200 are the race times predicted by the critical speed model. $MLR_{D'}$-50, $MLR_{D'}$-100, and $MLR_{D'}$-200 are the race times predicted by the multiple linear regressions (MLR) using D' (distance performed above CS) as anaerobic marker. MLR_{ASR}-50, MLR_{ASR}-100, and MLR_{ASR}-200 are the race times predicted by MLR using the anaerobic speed reserve as anaerobic marker.*

Table 2c. Multiple Linear Regression models divided by sex.

			Beta coefficients						*Results*						
Model	***Race***	***Sex***	**β0**	**β1**	**β2**	**β3**	**β4**	**β5**	R^2	$_{adj}R^2$	***P***	***Predicted (s)***	***SEE (s)***	***MAE (s)***	***MAE (%)***
$MLR_{D'}$	**Pb50**	**F**	58,26	-18,80	-0,2598	-0,0895	0,0410	-0,493	0,924	0,869	<0,001	28,73	0,548	0,349	1,202
		M	55,80	-12,44	-0,2204	-0,4441	0,0290	-0,398	0,901	0,777	<0,050	26,49	0,747	0,424	1,600
	Pb100	**F**	140,50	-42,48	-0,4419	-0,4637	0,0074	-3,211	0,970	0,949	<0,001	63,14	0,780	0,476	0,755
		M	145,40	-44,34	-0,6961	-1,3760	0,5793	-12,850	0,830	0,618	0,106	56,96	1,591	0,992	1,741
	Pb200	**F**	340,00	-93,72	-0,2763	-3,0080	-0,1059	-6,029	0,938	0,894	<0,001	135,98	2,772	1,773	1,291
		M	423,20	-76,81	-1,4830	-3,0000	2,7830	-161,500	0,628	0,164	0,396	126,64	7,171	4,308	3,403
MLR_{ASR}	**Pb50**	**F**	62,81	-15,64	-14,500	-0,360	0,0263	-1,907	0,931	0,881	<0,001	29,06	0,524	0,351	1,208
		M	63,11	-9,77	-6,389	-0,233	-0,0613	-6,490	0,857	0,678	0,077	26,49	0,904	0,551	2,079
	Pb100	**F**	148,20	-37,30	-25,260	-0,919	-0,0163	-5,477	0.9797	0,965	<0.001	63,06	0,651	0,445	0,705
		M	182,00	-41,35	-24,530	-0,872	0,3909	-36,130	0,742	0,420	0,220	56,91	1,940	1,222	2,145
	Pb200	**F**	344,80	-90,32	-15,300	-3,297	-0,1218	-7,561	0,939	0,895	<0.001	137,45	2,777	1,775	1,292
		M	522,00	-78,76	-58,890	-2,174	2,5290	-217,300	0,618	0,141	0,413	126,54	7,225	4,313	3,407

$$\text{Race Time} = \beta0 + \beta1 \times CS + \beta2 \times \text{D' (or ASR)} + \beta3 \times \text{Age} + \beta4 \times \text{Mass} + \beta5 \times \text{Height}$$

Abbreviations. *$MLR_{D'}$ and MLR_{ASR} are the multiple linear regression model using critical speed (CS), distance above CS (D') or anaerobic speed reserve (ASR) (respectively), age, body mass and height; Pb50, Pb100, and Pb200 are the front crawl races in 50-,100-, and 200-m, respectively; β0 is the intercept of the model; β1, β2, β3, β4, β5 are the beta coeffients which have to be multiplie to CS ($m \cdot s^{-1}$), D'(m) or ASR ($m \cdot s^{-1}$), age (years), body mass (kg) and height (m); SEE, standard error of estimatation; MAE, mean absolute error.*

Average actual and predicted race times are presented in **Table 3c.**

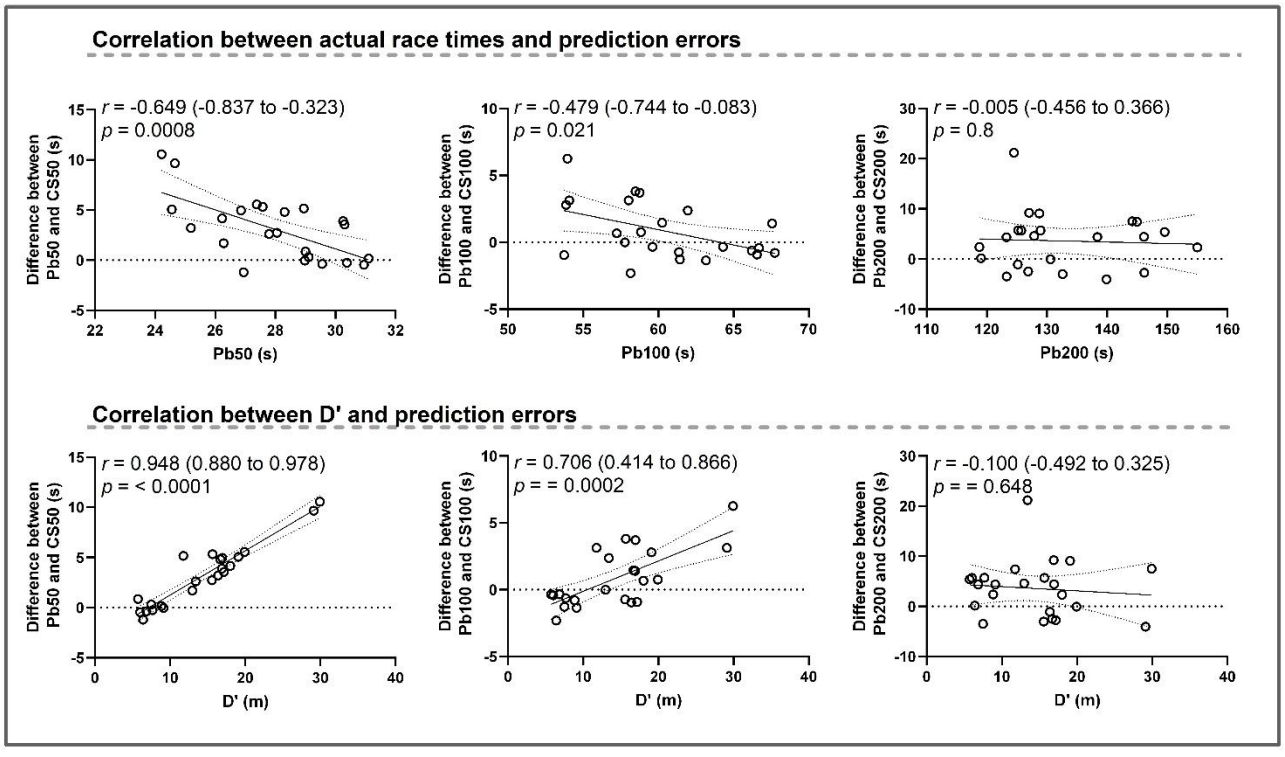
Table 3c. Average actual and predicted race times.

Race	Actual	CS model	$MLR_{D'}$	MLR_{ASR}
50-m (s)	27.94 ± 2.08	24.82 ± 4.74‡	27.94 ± 2.00	27.94 ± 2.00
100-m (s)	60.42 ± 4.52	59.58 ± 5.86	60.42 ± 4.39	60.42 ± 4.36
200-m (s)	132.69 ± 10.49	129.10 ± 9.75†	132.69 ± 9.36	132.69 ± 9.11

Abbreviations. *CS, critical speed; $MLR_{D'}$, multiple linear regression (MLR) using the curvature constant (D') and anaerobic index; MLR_{ASR}, MLR using the anaerobic speed reserve (ASR). † p<0.01; ‡ p < 0.001; significantly different from the actual race time.*

A proportional bias emerged among the CS model-predicted race times and relative bias. **Figure 4c** shows the parameters significantly related to the bias.

Figure 4c. Proportional bias examination of the critical speed model predicted results.



Abbreviations. *CS, critical speed. D', distance performed above CS. Pb50, Pb100, and Pb200, front crawl race times in 50-,100-, and 200-m, respectively. CS-50, CS-100, and CS-200 are the race times predicted by the critical speed model.*

Discussion

In this study, we investigated the relationships between the $3mAO_{mod}$ parameters (CS, D', ASR) and race times in 50-, 100-, and 200-m front crawls. Additionally, we evaluated the CS model's predictive accuracy using the $3mAO_{mod}$ parameters and developed linear equations also incorporating anthropometric measurements to predict race times. The first finding of our study relates to the relationships between the $3mAO_{mod}$ parameters and race times. In particular, an inverse relationship was observed between D' and race times in the 50- and 100-m events. Interestingly, and supporting the anaerobic underpinning meaning of D', this relationship tended to weaken as race distance increased, becoming non-significant in the 200-m event. Notwithstanding, the literature on the physiological meaning of D' is contradictory. In fact, some studies have observed correlations between D' and anaerobic indices such as blood lactate concentration or maximal accumulated oxygen deficit (Hill et al., 1995; Muniz-Pumares et al., 2017; Zagatto et al., 2019), while others found inconsistent results (Dekerle et al., 2002; Toussaint et al., 1998). This inconsistency may be due to a (partial) lack of physiological significance of this variable, as well as a high variability of its measurement due to the high difficulty of repeating maximal trials, or both (Mitchell et al., 2018; Noordhof et al., 2010). Nevertheless, our results show that athletes with higher D' values exhibit superior performance in shorter events such as 50- and 100-m front crawl, thus indirectly suggesting D' as an anaerobic index. Interestingly, when the data were analyzed separately by sex, D' was significantly associated with performance only in males, not in females. While the inferential power of this finding is limited by the reduced sample size after splitting, it suggests potential sex-specific physiological determinants of performance, even within the same event (e.g., 50, 100, or 200m). This could be partly attributable to the shorter absolute race times in males compared to females when matched for relative performance (using FINA points). Similarly to D', ASR showed a large correlation with 50-m performance, which gradually diminishes with increasing race distance and becomes non-significant in the 200-m event. Accordingly, Buchheit et al. (Buchheit et al., 2012) observed that athletes with a higher ASR

experience less metabolic stress at a given speed. Then, normalizing intensity for VO_2max, athletes with higher ASR demonstrate greater exercise tolerance, particularly during efforts lasting less than 5 minutes (Blondel et al., 2001; Di Gennaro et al., 2025b). Moreover, we observed a very large relationship between ASR and D' that is equally preserved even when stratifying the results by sex.

Potential explanations for this relationship include both mechanical and mathematical factors. From a mechanical standpoint, both parameters reflect the reserve of velocity or distance that an athlete can still swim beyond a certain threshold (Sandford et al., 2021). It is worth noting that the speed-duration relationship in swimming differs from the power-duration relationship in disciplines like cycling (Toussaint et al., 1988, 1998). This divergence arises because the energetic cost of locomotion in swimming is highly influenced by the water's drag resistance. This drag force increases quadratically with velocity, exponentially elevating the metabolic cost (Toussaint et al., 1998; Zamparo et al., 2020). Consequently, interpreting work capacity and power output above critical speed in swimming presents a greater challenge, as these measures are influenced by the distinct environment. From a mathematical perspective, the relationship between ASR and D’ could be partially explained by the fact that ASR is inherently incorporated into the equation for calculating D' (Mitchell et al., 2018). Moreover, when analyzing this relationship by sex, a pattern similar to that of D' emerged: ASR was positively associated with performance in males only and solely in the 50-meter front crawl. Even though it should be noted that these findings warrant caution due to the reduced sample size resulting from the stratification by sex.

Concerning CS, a moderate to large relationship across all distances has been observed. Contrary to the D', the strength of this correlation increased as the duration of the race increased. This pattern further supports the physiological rationale behind this parameter. Numerous previous studies have highlighted the importance of a high CS in sustaining prolonged high-intensity exercise before the onset of fatigue (Dekerle et al., 2002; Iannetta et al., 2022). This association is likely attributed to the strong correlation between CS and other physiological indicators of endurance performance, such as

the maximal lactate steady state and respiratory compensation point (Iannetta et al., 2022). An intriguing finding was the moderate correlation between CS and 50-m performance. The average 50-m race time was 27.94 seconds, so considering that the cardio-dynamic and primary phases of the oxygen kinetic typically last between one and two minutes, the energy contribution for a 50-m race is mainly supplied by anaerobic lactic and alactic pathways (Almeida et al., 2020). Therefore, while CS is correlated with 50-m race times, this does not necessarily imply a direct causal relationship. In fact, performance in this event is largely determined by technical skills and movement efficiency (Toussaint et al., 1988). However, to achieve high levels of movement economy and propulsive capacity, it is necessary to perform a high training volume, especially at low intensities, to learn and improve the movement without the detrimental effects of fatigue (Toussaint et al., 1988). This volume of low-intensity training could induce a concurrent improvement in aerobic power and technical skills, which led us to find this positive relationship between CS and 50-m performance (Mølmen et al., 2024). A further interesting result emerged from the sex-stratified analysis. CS was positively associated with performance, but in this case, in females only. Taken together with the previous finding that D' was associated with performance only in males, these results might suggest again the distinct metabolic and physiological demands of the same event on male and female swimmers.

The second aim of this study was to evaluate the predictive accuracy of the CS model applying the $3mAO_{mod}$ parameters. Our findings indicate a systematic underestimation of race times across all distances. Specifically, we observed underestimations of ~10%, ~1.4%, and ~2.7% respectively, in 50-,100-, and 200-m race times, thus suggesting this model is inaccurate for predicting such short-duration events, especially the 50-m front crawl. Although the reasons are not entirely clear, some hypotheses can be proposed. Firstly, the CS model was initially developed to explain performance over a duration spanning from 2 to 30 minutes, therefore largely outside the time domain of the 50-m (Hill, 1993). Secondly, CS and D', derived from the $3mAO_{mod}$, were validated against the traditional 2-parameter model (Mitchell et al., 2018). Previous research reported that this model tends to overestimate

performance in events lasting less than two minutes (Pallarés et al., 2020; Vinetti et al., 2019). Conversely, the 3-parameter model, which incorporates a *k*-factor representing a hypothetical maximum power output, has been shown to provide more accurate predictions for events lasting approximately 30 seconds (Pallarés et al., 2020; Vinetti et al., 2019). Considering that the 50-m performance is more power- rather than capacity-limited, the 2-parameter CS model applied in this study may not accurately predict performance.

Moreover, from the Bland-Altman plots, proportional bias emerged. Indeed, a strong inverse relationship was observed between race time and prediction error for both the 50-m and 100-m events. This indicates that the 2-parameter CS model becomes less accurate as the race times decrease. Then, a significant positive correlation was found between D' and prediction error for both the 50-m and 100-m events, suggesting that D' may be a primary source of error in these predictions. While a definitive explanation for this relationship is challenging, several factors could be considered. Firstly, D' estimated by the $3mAO_{mod}$ shows moderate to large uncertainty compared to the traditional D' estimation and is the result of an intermittent all-out test, which may overestimate the curvature constant due to the 5-second recovery after each pool (Mitchell et al., 2018). Secondly, the physiological significance of D' has been widely debated. While some researchers view this variable as synonymous with anaerobic work capacity, others contest this interpretation (Hill et al., 1995; Toussaint et al., 1998). For instance, Bundle and Weyand, (2012) highlighted that during all-out efforts like the $3mAO_{mod,}$ the power decrements (and thus also the area under the curve) may be more associated with the progressive skeletal muscle force impairment rather than the depletion of the finite anaerobic work capacity.

The third aim of this study was to develop linear equations incorporating the CS, D', or ASR and anthropometric measures to predict race time. Both multiple regression models demonstrated high accuracy. Specifically, $MLR_{D'}$ explained 92% of the variance for 50- and 100-m events and 80% for 200-m. Similarly, MLR_{ASR} explained 92%, 93%, and 76% of the variance for 50-, 100-, and 200-m

events, respectively. Additionally, Bland-Altman analysis revealed no significant bias, indicating high model accuracy. Like the CS model, accuracy decreased for the 200-m event, possibly due to participants' unfamiliarity with this distance. The improved accuracy of the $MLR_{D'}$ and MLR_{ASR} models compared to the CS model can be attributed to two primary factors. First, the inclusion of anthropometric parameters provided additional information correlated with athlete performance (Bourgois et al., 2000; Lätt et al., 2010; Zamparo et al., 2020). In fact, the present study showed a strong correlation between height and race performance across all three distances. These findings are consistent with previous studies that have observed a positive association between race time and anthropometric factors, such as height and arm span, in both adolescent and adult swimmers (Zamparo et al., 2020). Second, the regression equations were specific to the study sample. This is because the beta coefficients, generated by the regression procedures, are strictly determined to generate the model with the lowest estimated standard error for the actual race times. Finally, the sex-stratified MLRs also yielded models with low prediction errors. However, the small sample size relative to the number of predictors frequently resulted in statistically insignificant coefficients of determination. While these findings are promising, they necessitate further investigation to validate the models' predictive accuracy and to substantiate the proposed sex-based differences in the physiological demands of the swimming events studied herein.

It should be noted that this study does have some potential limitations: 1) the MLR equations provide exellent results but have been tested only against the same data used to build the same equations, therefore further investigation are needed to assess their external validity; 2) the $3mAO_{mod}$ although provides validated CS and D', does not take into accout the swimming turning phase, limiting his ecological validity; 3) in female swimmers, it was not possible to set up the preliminary test during the same phase of the menstrual cycle; 4) the use of a handheld stopwatch for data collection, while appropriate for the scope of this study, is a potential source of measurement error. Consequently, despite the good to excellent reliability observed, the results may possess a lower degree of precision

compared to those obtained with automated timing systems (e.g., photocells), which may have influenced the findings.

Practical applications

Our findings indicate the $3mAO_{mod}$ test as a practical and easy-to-administer tool for evaluating both aerobic (CS) and anaerobic (D', ASR) characteristics in swimmers in less than 4 minutes, and when used to resolve the CS model, can predict race times > 100m front crawl performance, but not the 50-m one. Instead, the MLR equations developed can accurately predict all front crawl distances investigated. In addition, the application of the ASR within the equations, while avoiding the mathematical complexity of D' calculation, retains comparable predictive accuracy, making it a practical alternative for performance prediction in field settings.

STUDY 4

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Intensity and Model-Dependent Variations in Work Above Critical Power and Its Association with Anaerobic Indices

Di Gennaro S., Hayes P., Panascì M., Ruggeri P., Faelli E.

Abstract

This study aimed to examine differences between the estimated curvature constant (*W′*) and the actual work above critical power ($W_{>CP}$) across intensities using different models and to explore correlations with anaerobic indices. After preliminary testing, 21 participants completed trials to exhaustion at 130, 115, 100, 85, and 80% of peak power of a ramp test (PO_{peak}) and the Wingate test. Critical power and *W′* were estimated by the work-time, inverse-time, 2 (2-hyp), and 3-parameter hyperbolic models. The accumulated oxygen deficit (AOD), lactate, and actual $W_{>CP}$ for each trial were assessed. Correlation analyses and repeated measures ANOVA with post-hoc comparisons were used. In all models except the inverse-time, $W_{>CP}$ at 130 and 115% of PO_{peak} was lower than at 100 and 85% (p<0.05). 2-parameter models show more consistent results with lower standard error of estimation and stronger associations between $W_{>CP}$ and *W'*. Significant correlations were observed between *W′* and both maximal AOD and [La] only for the work-time and 2-hyp models (p<0.05). However, irrespective of the model, the $W_{>CP}$ at 130 and 115% of PO_{peak} correlated with the corresponding AOD, lactate, and Wingate test results (p<0.05). In conclusion, $W_{>CP}$ is influenced by both the intensity and the model, and decreases near the extreme domain. $W_{>CP}$ at (supra)maximal intensities is linked to anaerobic markers regardless of the model, but for *W′,* this occurs only for work-time and 2-hyp models. This

suggests a single parameter cannot fully define anaerobic characteristics or explain exercise tolerance, and the model chosen strongly influences the outcome.

Keywords:

Critical Power, Anaerobic Capacity, Exercise Intensity Domains, Mathematical Modeling, Curvature constant.

Introduction

The relationship between the power output and the duration for which this power can be sustained is a cornerstone of exercise physiology. Among the mathematical models to describe this relationship, the critical power (CP) model is one of the most used (Jones et al., 2010). Originally derived from a curvilinear function, the CP model can also be reformulated into a linear equation describing either the relationship between exercise time and work performed, or between power output and the inverse of exercise time (Mattioni Maturana et al., 2018). The CP models relie on two key assumptions: (i) the existence of an asymptote, or "critical power", the theoretical maximal intensity that can be sustained indefinitely, and (ii) the constancy of work performed above CP (W'), often interpreted as a measure of anaerobic capacity, and assumed to be the same for any (supra-CP) intensity (Drake et al., 2024). However, recent findings suggest that these assumptions may not hold universally (Drake et al., 2024). Prior work indicated that trial duration affects these estimates, with shorter time-to-exhaustion (T_{lim}) trials yielding higher CP and lower W', while longer trials produce the inverse (Drake et al., 2024; Mattioni Maturana et al., 2018). This raises the question of whether the lower W' associated with shorter T_{lim} trials is simply a consequence of inflated CP estimates or whether it reflects a genuine reduction in the work that can be performed above CP ($W_{>CP}$) at higher intensities (Bergstrom et al., 2014; Mattioni Maturana et al., 2018).

Furthermore, while CP estimates generally have low standard errors of estimation (SEE), W' exhibits considerably greater variability, with SEE reaching up to 20% of its value (Mattioni Maturana et al.,

2018). This suggests that *W′*, often referred to as the "curvature constant," may not remain, *de facto*, constant across different exercise intensities. A recent study on running found that $W_{>CP}$ is higher during T_{lim} lasting 7-10 minutes, compared to shorter and longer durations (Ruiz-Alias et al., 2024). Similarly, Dekerle et al. (2015) demonstrated that reducing intensity during T_{lim} increases the work that can be completed above CP. Moreover, the traditional severe intensity domain can also be subdivided to delineate its uppermost region, termed the “extreme intensity domain” (Hill et al., 2002; Ozkaya et al., 2025). In this domain, the exhaustion occurs within ~2 minutes, $\dot{V}O_2max$ is not attained (Ozkaya et al., 2025), and *W’* is lower than that observed in the severe domain (Alexander et al., 2019). These observations challenge the assumption of *W′* as a fixed parameter and necessitate resolution of two unsolved questions: whether *W′* reductions reflect task-dependent constraints or physiological mechanisms, and if this lower *W′* at extreme intensities arises from mathematical inflation of CP or bona fide physiological limitations in the energetic pathways involved.

While clear relationships between CP and other aerobic markers from both a whole-body (e.g., $\dot{V}O_2max$) or a local perspective (e.g., mitochondrial content, citrate synthase activity) have been observed (Vanhatalo et al., 2016), the physiological basis of *W′* remains debated (Green et al., 1994; Hill and Smith, 1993; Muniz-Pumares et al., 2017). Muniz-Pumares et al. (2017) observed that the accumulated oxygen deficit, a commonly used method to assess anaerobic capacity, during an exhaustive trial of approximately 3 minutes was positively correlated with the work performed above CP during that trial. In line, Green et al. (1994) reported that *W′* estimated from the work-time model was correlated with intramuscular ATP changes and end-exercise blood lactate concentrations. Yet, no associations were found with glycogen phosphorylase and phosphofructokinase activity, muscle buffering capacity (Green et al., 1994), peak blood lactate or end-exercise pH (Jenkins and Quigley, 1991). Vandewalle et al. (1989) observed a correlation between Wingate test-derived anaerobic capacity and *W’*, but it remained unclear which of the two exhibits a stronger association with a “true” physiological anaerobic capacity. Interestingly, in all the aforementioned studies, only the work-time

model was ever applied, without analysis of other models, raising the possibility that the reported relationships are model-dependent rather than purely physiological.

Given this surrounding uncertainty, the purpose of this study was to examine the stability of $W_{>CP}$ at various intensities across different models. As complementary analyses, we examined the degree to which W', and its intensity-specific components (i.e., $W_{>CP}$ at given intensities) reflect anaerobic characteristics, using accumulated oxygen deficit, blood lactate, and Wingate-derived parameters as reference measures.

Materials and Methods

Participants

An *a priori* sample size calculation was performed using G*Power 3.1, with the correlation between *W′* and accumulated oxygen deficit (AOD) as the primary outcome. Based on Pearson correlation analysis (α = 0.05, power = 0.80, two-tailed), assuming a large effect size (r = 0.6) (Muniz-Pumares et al., 2017), at least 17 participants were required. To account for potential dropout, 21 endurance-trained athletes (19 males, 2 females) were recruited. The inclusion criteria required a $\dot{V}O_2max$>50 $mL \cdot kg^{-1} \cdot min^{-1}$ (males), >45 $mL \cdot kg^{-1} \cdot min^{-1}$ (females). Participants were regularly engaged in endurance training. Potential participants were excluded if they had any cardiovascular, metabolic, neuromuscular disorders, or lower-limb injuries within the previous 6 months that could affect their cycling performance. Participant characteristics were (mean ± SD): age = 32 ± 7 years, height = 177 ± 9 cm, body mass = 71 ± 12 kg, $\dot{V}O_2max$= 60 ± 7 $mL \cdot kg^{-1} \cdot min^{-1}$. Testing conditions were standardized by requiring participants to: (1) fast and abstain from caffeine for ≥ 3 hours pre-test; (2) attend sessions at consistent times (±1 h); (3) avoid exercise for >36 hours prior; and (4) maintain consistent dietary habits throughout the study period.

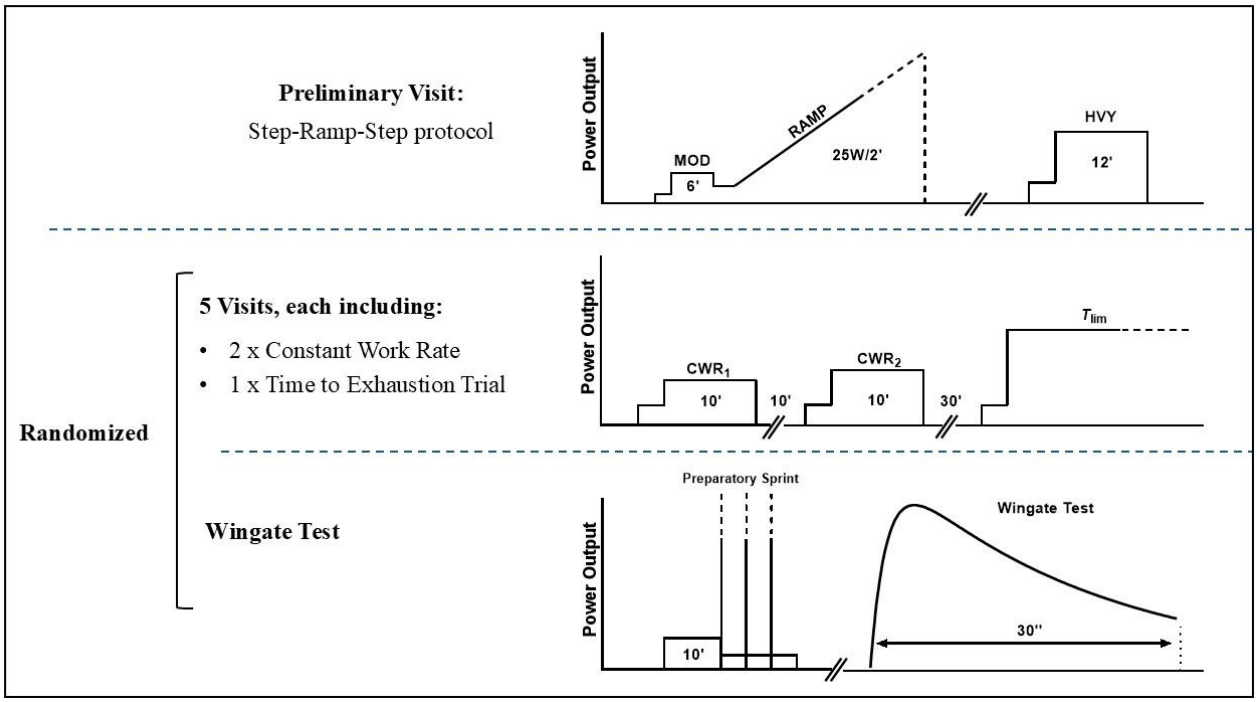
Design

Over four weeks, participants attended seven laboratory sessions and an initial familiarization session. Briefly, participants performed a step-ramp-step test (Iannetta et al., 2020b) to establish $\dot{V}O_2max$, ventilatory thresholds, and associated power output. Subsequently, in a randomized order, they conducted a Wingate test, five severe-intensity T_{lim} trials (for CP/*W′* estimation), and 2 (10 in total) submaximal constant-work-rate (CWR) bouts below the respiratory compensation point, preceding each T_{lim} session to quantify AOD. An Excalibur Sport ergometer (Lode, Netherlands) was used throughout, configured to participants' anthropometry and habitual positioning (the experimental design is shown in **Figure 1d)**.

Pre-testing and Familiarization

During the initial visit, participants completed anthropometric assessments, height via stadiometer (Seca 217); body mass and composition via bioelectrical impedance (Tanita BC-420 MA), and protocol familiarization. This included: 1) torque-velocity profiling for optimal cadence determination (through the ergometer linear factor); 2) a familiarization Wingate test; 3) a practice ramp test; and 4) a ~5-min T_{lim}.

Figure 1d. Schematic representation of the experimental design.



***Abbreviations**: MOD, moderate-intensity bout; HVY, heavy-intensity bout; CWR_1 and CWR_2, submaximal constant-work rate (2 per session, hence 10 CWR_S (all randomized, between 30 and ~75% of the peak power of the ramp test [PO_{peak}]) in total; T_{lim}, Time to exhaustion at an (randomized) intensity of 130,115, 100, 85, or 80% of PO_{peak}. Note: the familiarization trials have not been included in the figure.*

Baseline Testing: Step-Ramp-Step Test

Participants performed the step-ramp-step protocol proposed by Iannetta et al., (2020b) to determine both $\dot{V}O_2$max and the heavy-severe boundary within a single session. This was essential to ensure that the subsequent submaximal CWRs, used for AOD calculation, were performed below this threshold (Medbø & Welde, 2022). Participants began with a 2-minute warm-up (20W), a 6-minute square wave at 100W (moderate domain), and a 4-minute recovery (50W), followed by a ramp test until exhaustion (25W increment every 2 minutes). Participants kept a stable cadence between 70 and 90 rpm, and the exhaustion was confirmed when at least 2 out of the following criteria were met: heart rate exceeded 95% of predicted maximal heart rate (210 – (0.65 × age)); respiratory exchange ratio exceeded 1.1; blood lactate was above 8.0 mmol·L^{-1} (Poole & Jones, 2017). The peak power output (PO_{peak}) attained during the ramp test was retained to set the T_{lim} trials intensity subsequently. During the test, breath-by-breath pulmonary gas exchange was measured using a portable metabolic system (K5, Cosmed, Rome, Italy). Pre-test procedures included volume calibration with a 3-L syringe and gas calibration using ambient air and a reference gas mixture (20.0% O_2, 4.0% CO_2). Ambient conditions were maintained at 20 ± 1°C and 53 ± 4% relative humidity. 2 and 3 minutes post-exhaustion, earlobe blood samples (5 µL) were collected to assess blood lactate concentration (Lactate Pro 2, Arkray, Japan). Gas exchange threshold (GET) and respiratory compensation point (RCP) were identified as the first nonlinear increase in the ventilatory equivalent for oxygen and carbon dioxide (following the isocapnic buffering phase), respectively (Keir et al., 2022). After a 30-minute recovery from the ramp test, a 12-minute heavy-intensity bout (~75% of the visually detected RCP) was performed. A left-shifting of power output (PO) at GET and RCP using end-stage $\dot{V}O_2$ of the moderate and heavy-intensity bout was applied (Iannetta et al., 2020b) .30 minutes after the heavy domain bout, participants completed a second familiarization T_{lim} lasting approximately 15 minutes (~80% PO_{peak}).

Wingate Test

Participants performed a 5-minute warm-up (80 W for females and 100 W for males), concluding with three 4-5-second preparatory sprints. Thereafter, a 30-second maximal sprint in a seated position was performed from a flying start (65 rpm). Resistance was individualized using torque-velocity data collected during the familiarization visit. Both peak power (PPO; highest 1-second output) and mean PO (MPO; 30-second average) were recorded. Participants were strongly encouraged during the test and were blinded to PO and elapsed time.

Submaximal Constant Work Rate and Time-to-Exhaustion trials

Every session commenced with two 10-minute CWRs (30-75% PO_{peak}), separated by a 10-minute rest, to establish individual power-$\dot{V}O_2$ relationships for subsequent determination of accumulated oxygen deficit (AOD). Following a 30-minute recovery period, participants performed a Tlim at 80%, 85%, 100%, 115%, or 130% of PO_{peak} in a randomized order for each visit. These intensities were designed to elicit exhaustion durations of 1-20 minutes. Each T_{lim} started with a 4-minute warm-up at 50 W followed by a square-wave transition to the target intensity. Gas exchange data were collected breath by breath throughout all the visits and trials. Blood lactate concentration was assessed 2, 3, and 4 minutes post-exhaustion, with the highest value retained for analysis.

Data Analysis

The relationships between the power output and T_{lim} have been modelled for each participant using the following CP models (Mattioni Maturana et al., 2018):

- Inverse-time model: $PO = W' \cdot T_{lim} + CP$;
- Work-time model: $W_{lim} = W' + CP \cdot T_{lim}$;
- 2-parameter hyperbolic model (2-hyp): $T_{lim} = W'/(PO - CP)$;
- 3-parameter hyperbolic model (3-hyp): $T_{lim} = W'/(PO - CP) + W'/(CP - P_{max})$);

Where P_{max} is the estimated maximal instantaneous power (in watts), and W_{lim} is the work done (in Joules) in each T_{lim}. The SEE were calculated for CP and W', while root mean square errors (RMSE) were computed to assess the accuracy of the T_{lim} predictions yielded by the various CP models. $W'_{>CP}$ was computed for each T_{lim} and each model as: $W' = (PO\text{-}CP) \cdot T_{lim}$, using the model's CP and W'. To account for the influence of body mass, $W'_{>CP}$ and W' were allometrically scaled by dividing by body mass raised to the power of 0.67 (e.g., $relative\ W' = absolute\ W' \cdot body\ mass^{-0.67}$). To enable the allometric scaling of blood lactate concentration in line with W' and $W'_{>CP}$, it was necessary to convert the unit from $mmol \cdot L^{-1}$ to $mmol \cdot kg^{-0.67}$. This conversion required estimating each participant's total blood volume using the validated equations of Oberholzer et al., (2024) to calculate total lactate content, which was then normalized via allometric scaling. Raw gas exchange data were processed in MATLAB R2024b (MathWorks, Natick, MA) to eliminate anomalous breaths, interpolate data to 1-second intervals, and apply a third-order low-pass Butterworth filter (0.04 Hz cutoff) (Robergs et al., 2010). The relationship between $\dot{V}O_2$ during the last minute of each CWR and the corresponding PO has been modelled with a linear regression to estimate the theoretical O_2 demand (O_{2dem}) for any supra-$\dot{V}O_2max$ effort, and subsequently to calculate AOD as the difference between O_{2dem} and the actual O_2 consumed during each T_{lim} (Medbo et al., 1988).

Statistical Analysis

Normality was assessed using the Shapiro-Wilk test, confirmed by a further check of the quantile-quantile plot. All data were normally distributed except for lactate and AOD during T_{lim} trials at 130% of PO_{peak} and the estimated T_{lim} at 80% of PO_{peak} from the inverse-time model and 3-hyp model. To assess the stability of $W'_{>CP}$ at different intensities, a repeated-measures analysis of variance (ANOVA) was used for each model, including the estimated W'. The same analyses were performed to assess physiological responses across intensities. Furthermore, to examine the effects that these intensity-dependent variations in $W'_{>CP}$ had on the T_{lim} estimates, we compared these predictions using a two-way ANOVA with T_{lim} (predicted or actual) and intensity (130, 115, 100, 85, and 80% or PO_{peak}) as factors. The root mean square errors in percentage have been computed to assess T_{lim}'s prediction accuracy. In case of non-normally distributed variables, the Friedman test was carried out. Greenhouse-Geisser correction was applied when sphericity was violated. Effect size for ANOVA was reported as partial eta-squared (η_p^2). Where a significant main effect was identified ($p < 0.05$), pairwise comparisons were performed with Bonferroni's post-hoc corrections, and Cohen's d was calculated. Threshold values were interpreted as follows: $0.2 < |d| \leq 0.49$ = small, $0.5 \leq |d| \leq 0.79$ = moderate, $|d| \geq 0.8$ = large (Cohen, 1988). For normally distributed variables, Pearson correlations were used to examine relationships between W', $W'_{>CP}$, and anaerobic markers by accounting for the body mass using allometric scaling. The r coefficient was interpreted as follows: $r \geq 0.5$ = large, $0.3 \leq r < 0.5$ = moderate, and $r < 0.3$ = small (Hopkins et al., 2009). For non-normally distributed variables, Spearman correlation (ρ) was used, with the same interpretation thresholds. The statistical significance threshold was set at $\alpha = 0.05$ for all analyses, using two-tailed tests.

Results

Participants' characteristics and estimates of CP models are presented in **Table 1d**.

Table 1d. Participants' characteristics.

Step-Ramp-Step Test		Power Duration relationships				
			3-hyp	2-hyp	Work-Time	Inverse-Time
GET (L·min^{-1})	2.49 ± 0.6	**CP (W)**	237 ± 48	248 ± 50	252 ± 47	259 ± 48
GET (W)	174 ± 44	**CP SEE (W)**	8.9 ± 11.3	3.6 ± 2.1	4.8 ± 2.7	7.7 ± 4.1
RCP (L·min^{-1})	3.18 ± 0.68	***W'* (kJ)**	41.5 ± 15.8	25.5 ± 7.5	22 ± 7.6	19.4 ± 7.8
RCP (W)	234 ± 36	***W'* SEE (kJ)**	7.8 ± 6.8	3.4 ± 2	3.4 ± 2.1	1.7 ± 1.2
$\dot{V}O_2$max (L·min^{-1})	4.29 ± 0.89	**R^2**	0.997 ± 0.003	0.990 ± 0.01	0.999 ± 0.001	0.980 ± 0.019
$\dot{V}O_2$max (mL·kg^{-1}·min^{-1})	60 ± 7.35	**Wingate Test**				
PO_{peak} (W)	336 ± 62	**PPO (W)**	1020 ± 227			
***$W_{>RCP}$* (kJ)**	26.2 ± 10.6	**MPO (W)**	679 ± 147			

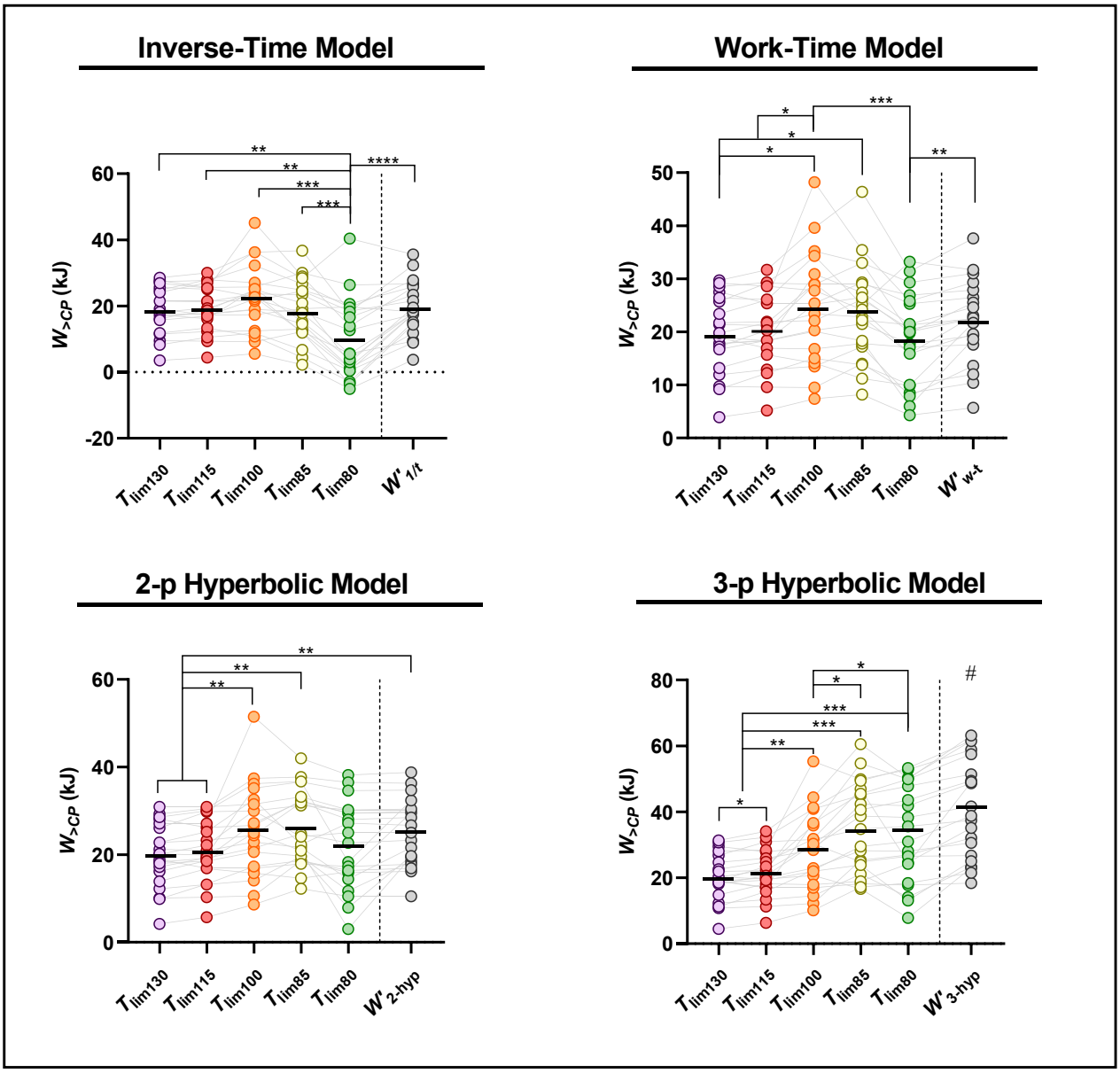
Abbreviations: *GET, gas exchange threshold; RCP, respiratory compensation point; $\dot{V}O_2$max, maximal O_2 uptake; PO_{peak}, peak power output of the ramp test; $W_{>RCP}$, work done above RCP during the ramp test; 3-p Hyp and 2-p Hyp refer to the 3-parameter and 2-parameter hyperbolic model; CP, critical power; W', curvature constant; SEE, estimated standard error; R^2, coefficient of determination; PPO and MPO are the peak (1s) and mean (over 30s) power output attained during the Wingate test, respectively.*

Differences Among $W_{>CP}$ Across Intensities and W'.

There were significant differences between the $W_{>CP}$ across intensities and with the modelled *W'* (**Figure 2d** and **Table 2d**). Post-hoc analyses (details in **Table 2d**) showed that for the inverse-time model, $W_{>CP}$ during 80% of PO_{peak} was significantly lower than in all other intensities and lower than *W'* estimated by the model. As for the work-time model, $W_{>CP}$ at 130% was significantly lower than at 100 and 85% of PO_{peak}. $W_{>CP}$ at 100% was significantly higher than at 115 and 80% $PO_{peak.}$ Regarding the 2-hyp model, $W_{>CP}$ at 130 and 115% were significantly lower compared to all other intensities (except for T_{lim} at 80%) and to the *W'* estimated. When applying the 3-hyp model, $W_{>CP}$ at 130% was lower than 115%, and both were significantly lower than $W_{>CP}$ for any lower intensities and

the modelled *W'*. $W_{>CP}$ at 85 and 80% of PO_{peak} were significantly higher than at any higher intensities except for the modelled *W',* which was significantly greater than any $W_{>CP}$.

Figure 2d. Differences among $W_{>CP}$ across intensities and W'.



Abbreviations: *$T_{lim\ 80/85/100/115/130}$ is the time to exhaustion trial at 80/85/100/115/130 % of the peak power output reached during the ramp test; $W_{>CP}$, represent the work done above critical power; $W'_{1/t}$, $W'_{w\text{-}t}$, $W'_{2\text{-}hyp}$ and $W'_{3\text{-}hyp}$ refer to the curvature constant estimated from the inverse-time model, work-time model, 2- and 3- parameter hyperbolic model, respectively; *;**;***;**** are representative of p value < 0.05; 0.01;0.001;0.0001.*

Table 2d. Repeated Measures ANOVA and paired comparisons among $W_{>CP}$ (kJ) across intensities and estimated W' (kJ).

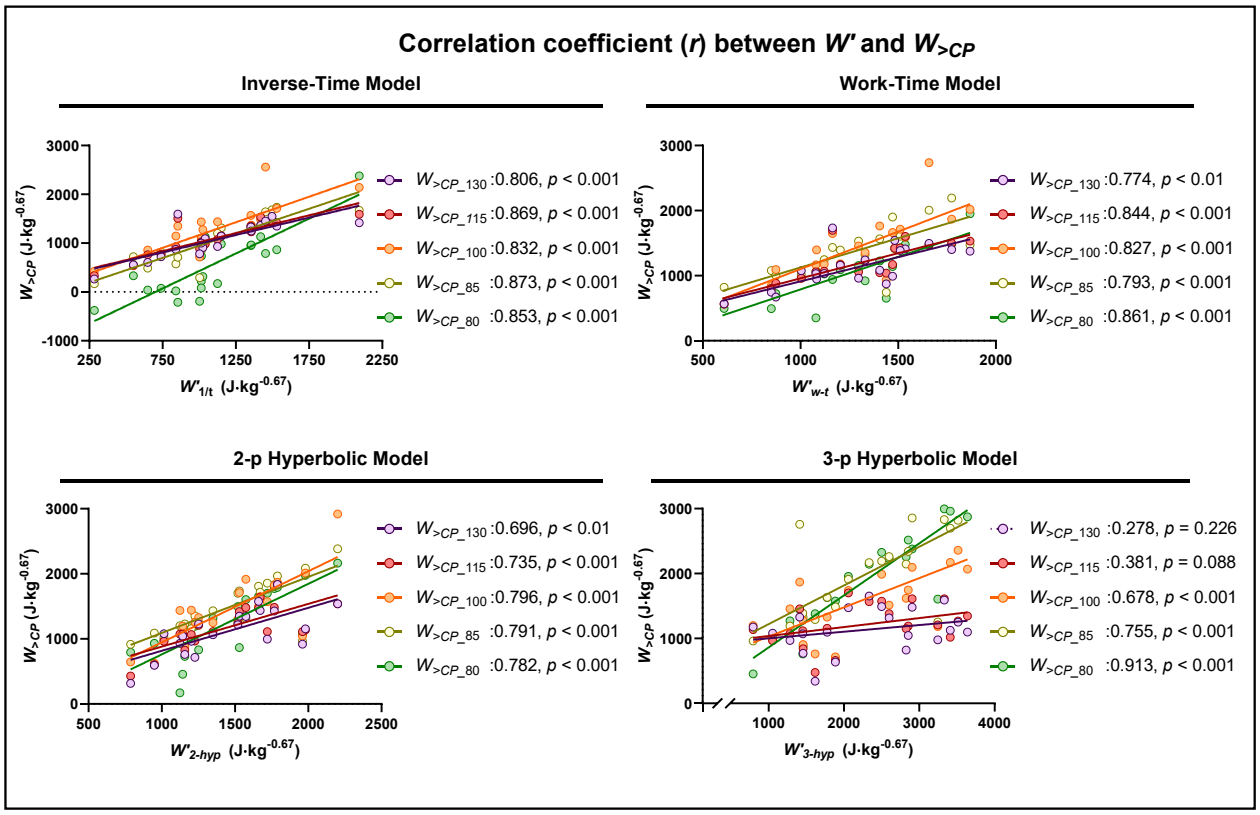
Model	ANOVA	Pairwise Comparison with Bonferroni's Correction											
		Comparison			Mean	±	SD		Mean	±	SD	$p_{corrected}$	*d*	Difference
1/Time	p<0.001 η_p^2=0.50	$W_{>CP\_130\%}$	vs.	$W_{>CP\_80\%}$	18.8	±	7.0	vs.	10.3	±	11.7	0.005	0.87	*large*
		$W_{>CP\_115\%}$			19.3	±	6.9					0.002	0.94	*large*
		$W_{>CP\_100\%}$			22.5	±	9.8					<0.001	1.13	*large*
		$W_{>CP\_85\%}$			18.7	±	9.0					<0.001	0.80	*large*
		$W_{>CP\_80\%}$	vs.	$W'_{1/t}$	10.3	±	11.7	vs.	19.4	±	7.8	<0.001	-0.92	*large*
W-T	p<0.001 η_p^2=0.37	$W_{>CP\_130\%}$	vs.	$W_{>CP\_100\%}$	19.5	±	7.1	vs.	24.6	±	10.1	0.013	-0.58	*moderate*
			vs.	$W_{>CP\_85\%}$					23.9	±	9.0	0.013	-0.55	*moderate*
		$W_{>CP\_115\%}$	vs.	$W_{>CP\_100\%}$	20.4	±	7.0	vs.	24.6	±	10.1	0.042	-0.48	*small*
		$W_{>CP\_100\%}$	vs.	$W_{>CP\_80\%}$	24.6	±	10.1	vs.	18.5	±	8.4	<0.001	0.65	*moderate*
		$W_{>CP\_85\%}$	vs.	$W_{>CP\_80\%}$	23.9	±	9.0	vs.	18.5	±	8.4	<0.001	0.62	*moderate*
		$W_{>CP\_80\%}$	vs.	$W'_{w\text{-}t}$	18.5	±	8.4	vs.	22.1	±	7.6	0.003	-0.44	*small*
2-hyp	p<0.001 η_p^2=0.34	$W_{>CP\_130\%}$	vs.	$W_{>CP\_100\%}$	19.8	±	7.2	vs.	25.5	±	10.2	0.006	-0.65	*moderate*
			vs.	$W_{>CP\_85\%}$					26.1	±	8.5	<0.001	-0.80	*large*
		$W_{>CP\_130\%}$	vs.	$W'_{2\text{-}hyp}$	19.8	±	7.2	vs.	25.5	±	7.5	<0.001	-0.78	*moderate*
		$W_{>CP\_115\%}$	vs.	$W_{>CP\_100\%}$	20.9	±	6.9	vs.	25.5	±	10.2	0.028	-0.54	*moderate*
				$W_{>CP\_85\%}$					26.1	±	8.5	0.002	-0.68	*moderate*
				$W'_{2\text{-}hyp}$					25.5	±	7.5	<0.001	-0.65	*moderate*
3-hyp	p<0.001 η_p^2=0.69	$W_{>CP\_130\%}$	vs.	$W_{>CP\_115\%}$	20.3	±	7.1	vs.	21.8	±	7.1	0.034	-0.21	*small*
				$W_{>CP\_100\%}$					28.9	±	11.6	0.001	-0.89	*large*
				$W_{>CP\_85\%}$					34.7	±	13.3	<0.001	-1.35	*large*
				$W_{>CP\_80\%}$					34.9	±	14.3	<0.001	-1.29	*large*
				$W'_{3\text{-}hyp}$					41.5	±	15.8	<0.001	-1.73	*large*
		$W_{>CP\_115\%}$	vs.	$W_{>CP\_100\%}$	21.8	±	7.1	vs.	28.9	±	11.6	0.004	-0.74	*moderate*
				$W_{>CP\_85\%}$					34.7	±	13.3	<0.001	-1.21	*large*
				$W_{>CP\_80\%}$					34.9	±	14.3	<0.001	-1.16	*large*
				$W'_{3\text{-}hyp}$					41.5	±	15.8	<0.001	-1.61	*large*
		$W_{>CP\_100\%}$	vs.	$W_{>CP\_85\%}$	28.9	±	11.6	vs.	34.7	±	13.3	0.025	-0.46	*small*
				$W_{>CP\_80\%}$					34.9	±	14.3	0.025	-0.46	*small*
				$W'_{3\text{-}hyp}$					41.5	±	15.8	<0.001	-0.91	*large*
		$W_{>CP\_85\%}$	vs.	$W'_{3\text{-}hyp}$	34.7	±	13.3	vs.	41.5	±	15.8	0.003	-0.47	*small*
		$W_{>CP\_80\%}$	vs.	$W'_{3\text{-}hyp}$	34.9	±	14.3	vs.	41.5	±	15.8	0.004	-0.44	*small*

Note: *The table presents details about the significant pairwise comparisons between work above critical power ($W_{>CP}$) at different intensities and the estimated curvature constant (W').* ***Abbreviations:*** *$W_{>CP\_80/85/100/115/130\%}$ is the work above critical power at 80/85/100/115/130 % of the peak power output reached during the ramp test; $W'_{1/t}$, $W'_{w\text{-}t}$, $W'_{2\text{-}hyp}$ and $W'_{3\text{-}hyp}$ refer to the W' estimated from the inverse-time model (1/Time), work-time model (W-T), 2 and 3- parameter hyperbolic model (2-hyp, 3-hyp), respectively.*

Correlations of W>CP across intensities and W'- allometrically scaled.

Pearson correlations revealed large correlations among *W'* estimated by all the models and the actual $W_{>CP}$ among T_{lim} trials, except for the 3-hyp in which *W'* was not correlated with $W_{>CP}$ at 130 and 115% of PO_{peak} (**Figure 3d**). Among these relationships, the lowest correlation coefficient between *W'* and the $W_{>CP}$ at different intensities always occurs at the highest intensity (130% PO_{peak}). As shown in **Figure 3d,** the range of the correlation coefficients between *W'* and $W_{>CP}$ across intensities is narrower for the work-time model, followed by the 2-hyp and inverse-time model; conversely, 3-hyp shows the widest range.

Fig. 3d Correlation plots between estimates of *W'* and $W_{>CP}$, allometrically scaled to body mass.



Abbreviations: *$W_{>CP\ 80/85/100/115/130}$ are work done above critical power (CP) the time to exhaustion trials at 80/85/100/115/130 % of the peak power output reached during the ramp test; W', curvature constant; $W'_{1/t}$, W' estimate of the inverse-time model; $W'_{w\text{-}t}$, W' estimate of the work-time model; $W'_{3\text{-}hyp}$ and $W'_{2\text{-}hyp}$ refer to the W'estimated by the 2- and 3-parameter hyperbolic model.*

Correlations among $W_{>CP}$, W', and anaerobic markers - allometrically scaled.

The analyses revealed relationships between MAOD and *W'* estimated only by the 2 parameters models ($0.546 \leq r \leq 0.591$, $p < 0.05$). There were positive associations between $W_{>CP}$ and the AOD of the corresponding trial at 130 and 115% of PO_{peak} across all models ($\rho > 0.570$ and $r > 0.658$, $p < 0.05$, respectively; details in **Table 3d**). Similar results were observed regarding [La], with positive relationships between the highest [La] of all T_{lim} trials and *W'* estimated by 2-parameter models ($r > 0.519$, $p < 0.05$). At 130 and 115% of PO_{peak}, irrespective of the model, the $W_{>CP}$ and the concomitant [La] were significantly correlated ($0.593 \leq \rho/r \leq 0.709$, $p < 0.01$; **Table 3d**). PPO was associated with the *W'* estimated by all the models except 3-hyp, and was related to the $W_{>CP}$ accumulated during T_{lim} at 130, 115, and 100% of PO_{peak}, irrespective of the model ($r > 633$, $p < 0.05$). MPO was associated with the modelled *W'* for all the models but 3-hyp ($0.570 \leq r \leq 0.702$, $p < 0.01$) and was correlated with $W_{>CP}$ accumulated at all the intensities except 80% of PO_{peak}, regardless of the model ($0.536 < r < 817$, $p < 0.05$). At 80% of PO_{peak}, this was true only for the W-T model ($r = 0.685$, $p < 0.001$).

Table 3d. Correlation among $W_{>CP}$ across intensities, *W'*, and anaerobic markers – allometric scaled

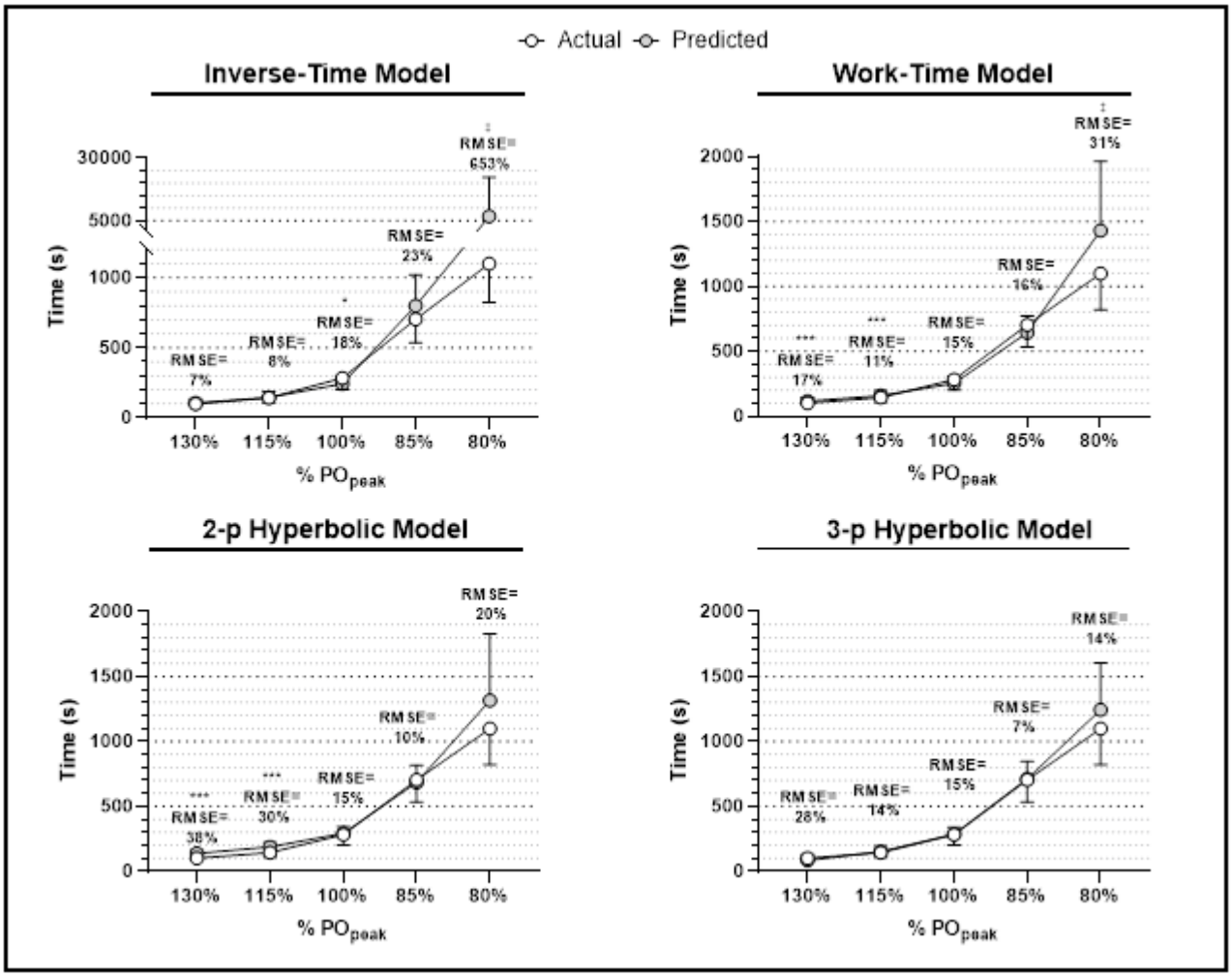
		Model	*130%*	*115%*	*100%*	*85%*	*80%*	*W'*
					(J·kg$^{-0.67}$)			
			correlation coefficient *r* or *ρ*					
AOD (mL·kg$^{-0.67}$)	**vs**	**1/Time**	0.570‡‡	0.658**	0.302	0.225	0.381	0.546*
		W-T	0.625‡‡	0.668**	0.340	0.249	0.176	0.591**
		2-hyp	0.631‡‡	0.666***	0.386	0.221	0.002	0.587**
		3-hyp	0.657‡‡	0.698***	0.459*	0.391	-0.002	0.329
[La] (mmol·kg$^{-0.67}$)	**vs**	**1/Time**	0.665‡‡	0.643**	0.547*	0.437*	0.287	0.519*
		W-T	0.709‡‡‡	0.626**	0.560*	0.408	0.248	0.574**
		2-hyp	0.706‡‡‡	0.593**	0.552*	0.444*	0.203	0.531*
		3-hyp	0.697‡‡‡	0.632**	0.541*	0.207	0.128	0.283
PPO (W·kg$^{-0.67}$)	**vs**	**1/Time**	0.744***	0.730***	0.721***	0.525*	0.301	0.590***
		W-T	0.762***	0.748***	0.733***	0.605**	0.539**	0.652**
		2-hyp	0.750***	0.736***	0.713***	0.525**	0.363	0.537**
		3-hyp	0.716***	0.633**	0.645**	0.346	0.271	0.179
MPO (W·kg$^{-0.67}$)	**vs**	**1/Time**	0.778***	0.792***	0.794***	0.614**	0.373	0.617**
		W-T	0.799***	0.817***	0.809***	0.740***	0.685***	0.702***
		2-hyp	0.781***	0.794***	0.771***	0.599**	0.420	0.570**
		3-hyp	0.736***	0.689***	0.747***	0.536*	0.477*	0.384

Abbreviations: *80/85/100/115/130 % of the peak power output reached during the ramp test; W', curvature constant; AOD, accumulated O_2 deficit; [La], peak of blood lactate concentration of the respective T_{lim}; PPO, peak power output (1s) during the Wingate test; MPO, mean power output over the 30s of the Wingate test.* ***Note****: when analyzing the relation between W' and AOD or [La], the maximal AOD or the highest [La] achieved throughout all the trials has been used.*;**;***;*** *are representative of p value < 0.05; 0.01;0.001. ‡, ‡‡ are used for non-parametric analyses with p value <0.05 and 0.01, respectively.*

Physiological responses and time to exhaustion prediction

The mean T_{lim} at 130, 115, 100, 85, and 80% of PO_{peak} was, respectively, 100 ± 29s, 144 ± 36s, 283 ± 82s, 704 ± 169s, 1098.6 ± 276s. Actual and predicted T_{lim} are presented in **Figure 3d**.

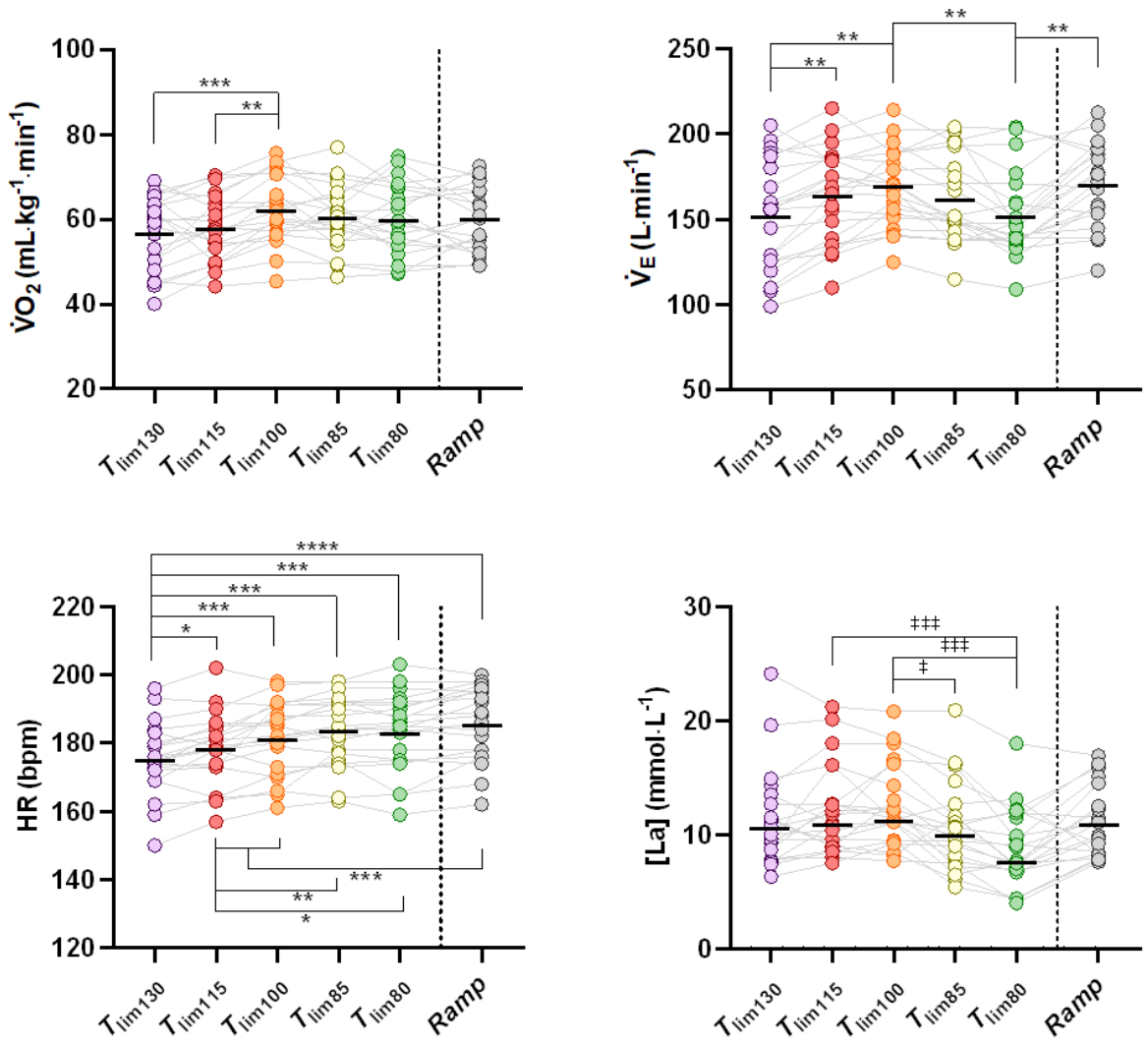
Figure 3d. Actual and predicted time to exhaustion trials.



Abbreviations: *PO_{peak}, peak power output reached during the ramp test; RMSE, root mean square error; *,**,*** are representative of p value < 0.05, 0.01,0.001.*

Physiological responses of T_{lim} and ramp tests in terms of O_2 uptake, minute ventilation, heart rate, and lactate are shown in **Figure 4d**. Peak O_2 uptake was significantly lower at 130 and 115% compared to 100% of PO_{peak} (56.80 and 58.00 vs 62.32 $ml \cdot kg^{-1} \cdot min^{-1}$; $p < 0.01$). A parallel behaviour appears for the peak HR throughout T_{lim} trials ($p < 0.05$). $\dot{V}_E$ was significantly higher at 100% of PO_{peak} compared to 130, 115, and 80% ($p < 0.01$). On the contrary, peak [La] was lower at 85 and 80% of PO_{peak} compared to the higher intensities ($p < 0.05$).

Figure 4d. Peak physiological responses during T_{lim} trials and ramp test.



Abbreviations: *$T_{lim\ 80/85/100/115/130}$ is the time to exhaustion trial at 80/85/100/115/130 % of the peak power output reached during the ramp test; Ramp represents the incremental ramp test; $\dot{V}O_2$, O_2 uptake; $\dot{V}_E$, minute ventilation; HR, heart rate; [La], blood lactate concentration. *;**;***;**** are rapresentative of p value < 0.05; 0.01;0.001;0.0001. ‡, ‡‡ are used for non-parametric analyses with p value <0.05 and 0.001, respectively.*

Discussion

The primary finding of this investigation is that $W_{>CP}$ is not constant but is instead highly dependent on both exercise intensity and the mathematical model used for its estimation. We observed that for all the models (except the inverse-time model), $W_{>CP}$ at 130 and 115% PO_{peak} was significantly lower than at 100 and 85% (for the 3-parameter model, this was also the case at 80% PO_{peak}). Also suggesting that the classically observed low W' when estimated from short T_{lim} trials is not only the result of an overestimated CP but also of a real decrease in the ability to accumulate work above it when approaching the extreme domain. For the linear models, a lower $W_{>CP}$ is also observed at 80% of PO_{peak}, likely because the CP estimates from these models are typically higher and often very close to the pre-set 80% of PO_{peak}, significantly slowing down the accumulation of work above CP. Our results align with the growing body of evidence questioning the constancy of the W'. Like those of Alexander et al. (2019) who observed lower W' within the extreme intensity domain than for the severe domain. Similarly, even considering just the severe domain, lowering the power output (but still above CP) during a T_{lim} leads to accumulating more $W_{>CP}$ (Dekerle et al., 2015; Welburn et al., 2025). Together, these findings suggest that exhaustion does not always result from the depletion of W', and that a single parameter cannot comprehensively explain the variability in T_{lim} across the entire severe intensity domain.

The physiological basis for the reduction in $W_{>CP}$ at 115% and 130% of PO_{peak} remains to be fully elucidated, but our data can provide some hypotheses. These intensities can be categorized within the extreme intensity domain (Ozkaya et al., 2025), where non-oxidative energy contribution is a determinant. In fact, in the current study, AOD and [La] responses were positively correlated with $W_{>CP}$ (regardless of the model), and the anaerobic contribution was approximately 48% and 40% of the total energetic demand. Additionally, the peak $\dot{V}O_2$ achieved at 130% and 115% of PO_{peak} was significantly lower compared to that at 100% of PO_{peak} (90 and 92%, respectively). Together, these factors suggest that the observed reduction in $W_{>CP}$ may be linked to a limit in the rate of anaerobic

energy transfer at these high power outputs. In particular, the early intracellular accumulation of H^+ can slow down the phosphofructokinase-1 (PFK-1), the rate-limiting enzyme of glycolysis, and therefore reduce the ability to accumulate $W_{>CP}$ (Spriet, 1991). This explanation is further corroborated by the previously observed associations between PPO and MPO and PFK-1 concentration (Green et al., 1996; Medbo et al., 1988), supporting our correlations between $W_{>CP}$ at (supra) maximal intensities and PPO, MPO, AOD, and [La]. A further hypothetical physiological explanation for the reduction in $W_{>CP}$ near/within the severe-intensity domain may relate to the kinetics of oxygen uptake and its full utilization. Indeed, our data show that a greater work above CP is accumulated only in trials where $\dot{V}O_2$max is actually achieved. This observation is somewhat in line with current literature demonstrating that interventions which accelerate the primary phase of $\dot{V}O_2$ kinetics, such as priming exercise, increase W' without altering CP (Burnley et al., 2011; Goulding et al., 2023). Conversely, a strong reduction in O_2 availability, as at high altitudes (~5000m), has been shown to reduce W' (Valli et al., 2011). Mechanistically, the pronounced reduction in O_2 availability forces a greater reliance on anaerobic pathways already at rest and during the initial phase of exercise (Medbo et al., 1988). This early and sustained dependence on anaerobic metabolism may prematurely accelerate the inhibition of PFK-1. Consequently, this could limit the accumulation of $W_{>CP}$ and ultimately prevent the attainment of $\dot{V}O_2$max. Notably, these two latter phenomena, limited $W_{>CP}$ accumulation and the inability to reach VO_2max, mirror the defining characteristics of exercise performed within the extreme-intensity domain under normoxic conditions (Alexander et al., 2019; Ozkaya et al., 2025). Unfortunately, our experimental design precludes a valid analysis of $\dot{V}O_2$ kinetics due to the CWR trials preceding each $T_{lim,}$ and the heterogeneity observed in $T_{lim,}$ which would make it difficult, if not impossible, to interpret the kinetic parameters consistently. Consequently, further studies are needed to confirm or refute the hypotheses just proposed regarding the relationship between $W_{>CP}$ depletion and $\dot{V}O_2$ kinetics.

Interestingly, this intensity-dependent variation in $W_{>CP}$ was most pronounced for the 3-parameter hyperbolic model, which also showed W' poorly related to $W_{>CP}$ at extreme intensities (not significant

at 130 and 115% of PO_{peak}) and even higher than any measured $W_{>CP}$. This model was originally formulated by introducing a third parameter (P_{max}) to impose an upper ceiling on power output, thereby addressing the limitation of the 2-parameter model. However, our results suggest this adjustment comes at a cost of greater estimation uncertainty. The model estimates three independent parameters (CP, W', P_{max}) from a limited number of trials (five in this study), likely leading to wide confidence intervals, as can be observed from the high SEE of CP and W'. Additionally, Vinetti et al. (2019) observed that only when three trials in the extreme domain are included in the power-duration relationship profiling, the estimate of P_{max} is accompanied by less variability than traditional approaches in which trials in the extreme domain are not (or only to a limited extent) included (Hugh Morton, 1996). In our case, the intensity spectrum applied elicited exhaustion between ~100 seconds to ~20 minutes, hence optimized to fit the longer-duration T_{lim} better, as evidenced by both the stronger correlation between W' and $W_{>CP}$ at 85% and 80% of PO_{peak} and the greater prediction accuracy of those T_{lim} compared to the 2-parameter models. In particular, the 3-parameter model shows lower prediction accuracy at 130 and 115% of PO_{peak} (RMSE = 28 and 14%, respectively) compared to linear models (~12 and ~10%). On the contrary, at 85 and 80% of PO_{peak}, the predictive accuracy of the 3-parameter model substantially outperforms linear models (RMSE ~10% vs. >20%). However, despite excellent goodness-of-fit statistics (R^2 = 0.997), W' estimated from the 3-parameter model is larger than any measurable $W_{>CP}$, and for each intensity, there was a greater variability between the respective $W_{>CP}$, as can be inferred from both the SEE of the model (7.8 kJ for 3-Hyp vs. an average of 2.8 kJ of the 2 parameters models, **Table 1**) and the magnitude of the difference in terms of effect size (i.e., Cohen's *d*, **Table 2**). Unfortunately, a definitive explanation is not possible, but two hypothesis could explain this result: 1) the moderating role of P_{max}: in fact, in the three-parameter model, exercise tolerance is not determined solely by CP and W', but also by P_{max}; 2) W' estimated by this model tends to show the weakest correlations with the anaerobic parameters used as a reference in this study.

Regarding the linear models, we observed more consistent relationships between W' estimates and the measured $W_{>CP}$. However, this was not the case for intensity approaching CP (80% of PO_{peak}). This was especially true for the inverse-time model, where we noted a reduction in measured $W_{>CP}$ alongside a dramatic loss of predictive accuracy: T_{lim} was overestimated by approximately threefold, and the RMSE surged from a reasonable 23% (at 85% of PO_{peak}) to an unreasonable 653% at (80% of PO_{peak}). We attribute this result to the model's known tendency to overestimate the heavy-severe boundary (Mattioni Maturana et al., 2018), which in some participants fell very close (or even higher) to 80% of PO_{peak}. In essence, if CP represents a true sustainable asymptote, the model's flaw is its assumption that work done above CP is constant across all supra-asymptotic intensities.

The choice of the model applied also influences the correlation between W', mechanical, and physiological markers of anaerobic features. In fact, the 2-parameter model resulted in W' being more strongly correlated with MAOD and peak [La] compared to the 3-hyp model. Moreover, PPO and MPO were also more strongly related to W' and $W_{>CP}$ at (supra)maximal intensities for 2-parameter models. Therefore, our data indicates that the chosen model is not a neutral decision but fundamentally alters the physiological meaning of the estimated W'. Unfortunately, these findings pose a challenge for selecting a model in future research. While all the 2-parameter models in our study produced more plausible W' estimates (given the lower SEE and greater association with AOD and [La]), in previous investigations, it is the 3-parameter model that yields a CP value that aligns more closely with established end physiological benchmarks like the maximal lactate steady state or the corrected respiratory compensation point (Caen et al., 2024; Iannetta et al., 2022). Researchers are therefore left to prioritize which parameter, W' or CP, is more critical for their specific investigation.

Some limitations of the present study should be considered when interpreting the results. Firstly, the CP model parameters were derived from T_{lim} trials. While this is a standard approach, it raises the question of whether the observed intensity-dependent dynamics of $W_{>CP}$ would be replicated using a self-paced time-trial protocol. Secondly, the physiological correlates of W' and $W_{>CP}$ were investigated

using indirect and non-invasive anaerobic indices (e.g., AOD, [La], and Wingate-test-derived parameters). Although these are widely accepted measures, they provide only an inferential link to the underlying intracellular anaerobic function. Consequently, the precise mechanistic relationship between $W_{>CP}$ and the true physiological limit of anaerobic energy production remains to be fully elucidated. Third, the sample was mainly characterized by males. This prevents a robust sex-based analysis and limits the generalizability of our findings. Finally, a methodological consideration should be noted regarding the analysis of $W_{>CP}$. The consistently higher SEE for W' compared to CP across all models underscores how the curvature constant is a parameter of inherently greater statistical variability. While one could theoretically fix W' and calculate a corresponding "intensity-dependent CP" (i.e., $CP_{130\%} = PO_{130\%} - W' / T_{lim}$), such a method would propagate the high uncertainty/variability of W' into the derived values, complicating their interpretation. Therefore, our decision to calculate $W_{>CP}$ by fixing the more stable CP estimate for each model was deliberate. It ensures that the observed intensity-dependent variations in $W_{>CP}$ are more likely to reflect genuine physiological dynamics rather than the amplification of estimation noise from the less reliable parameter.

In conclusion, this study demonstrates that $W_{>CP}$ is not constant but is instead highly dependent on both exercise intensity and the mathematical model used, diminishing significantly as intensity approaches the extreme domain. The reason behind this lower $W_{>CP}$ likely stems from the concurrent high anaerobic contribution and the inability to reach the $\dot{V}O_2max$ quickly enough. At maximal and supramaximal intensity, $W_{>CP}$ was always associated with all the anaerobic markers tested herein, regardless of the model. However, only the 2-parameter models provide estimates of W' more physiologically grounded and mathematically consistent, as shown by their association with MAOD and peak [La]. The observed variations in $W_{>CP}$ also impact the accuracy of T_{lim} predictions, demonstrating that model selection must be intensity-specific. Our data indicate that, when a traditional profiling of the power-duration relationship with five T_{lim}, with a duration between 100 seconds and 20 minutes, linear models provide superior prediction accuracy for intensities near the extreme domain,

whereas hyperbolic models are more accurate for lower intensities. Consequently, no single model is universally superior; the optimal choice is contingent upon the specific experimental question and the intensity range under investigation. Furthermore, from a physiological perspective, the interpretation of W' as a pure anaerobic parameter is inherently model-dependent. The significant influence of the model on its estimated value challenges the notion that W' represents a fixed, intrinsic physiological capacity, and future investigations should take into account this factor.

SECTION 6

6.1 Main Findings and Final Considerations

Throughout these studies, we have investigated the role of the anaerobic power reserve (APR) model in exercise performance, tolerance, and intensity prescription across cycling and swimming. By examining different formulations of the construct, varying its lower (e.g., MAP vs. CP) and upper (PPO vs. MPO) boundaries, we provide new insights into its limitations and potential applications.

The findings from study 1 highlighted that using APR to prescribe cycling HIIT intensity did not yield more homogeneous VO_2, Hr, [La], RPE responses, or times to exhaustion compared to the conventional MAP-based approach (Di Gennaro et al., 2025a). Unfortunately, the use of the GPR methods (MPO-MAP), which previously showed promise in reducing heterogeneity during constant work rate to exhaustion (Barnett et al., 1996) does not lead to more homogeneous physiological responses in our HIIT format. The results from study 1 are, however, in contrast with other studies (Bok et al., 2023; Collison et al., 2021; Julio et al., 2020) that used higher relative intensities. In fact, they observed a marginal, though non-significant, reduction in variability.

Several factors may explain the discrepancies between our findings and those of previous studies. First, in the study by Julio et al. (2020), the intensity prescribed using ASR was significantly higher than that prescribed using MAS, resulting in substantially shorter times to exhaustion. Consequently, the marginally lower variability observed in the ASR condition may be attributable to a ceiling effect, where convergence toward maximal values artificially reduces variance, rather than to a genuine advantage of the prescription method itself, as could have occurred in the study of Bok et al. (2023). Second, in the investigation by Collison et al. (2021), although exercise intensity was matched between conditions, MAS was estimated from a 2 km time trial rather than determined directly from an incremental test. This approach provides an estimate of the true maximal aerobic speed, and it is therefore plausible that the observed reduction in variability with ASR prescription simply reflects the

greater imprecision inherent in the MAS estimation. One might argue that any error in MAS determination would propagate into the ASR calculation. However, as demonstrated in study 2, the upper boundary (PPO or maximal sprinting speed) plays a substantially larger role than the lower boundary in determining the APR/ASR. Thus, even if MAS is poorly estimated, the impact on ASR may be mitigated by the dominant contribution of the sprint component (Di Gennaro et al., 2025b; Thron et al., 2023, 2025).

In recent years, a more sophisticated model than APR has been developed to address the challenge of normalizing HIIT intensity, namely the *W′* balance model. Unlike APR, which provides a fixed, one-dimensional reserve, the *W′* balance model conceptualizes *W′* as a dynamically depleting and replenishing "energy reservoir." Its core mechanics are governed by two key processes: 1) the depletion of *W′* when exercising above CP, and 2) its reconstitution during recovery phases below CP, a process characterized by a time constant (τ) that is itself inversely related to the difference between CP and the recovery intensity. Despite its greater physiological detail and complexity, this model still cannot fully predict exercise tolerance during HIIT (Bourgois et al., 2023; Caen et al., 2019; Lievens et al., 2021; Skiba et al., 2014; Welburn et al., 2025). Therefore, if even the *W′* balance model shows a certain degree of uncertainty, it is unlikely that a simple tool such as the APR could significantly reduce inter-subject variability in physiological responses and exercise tolerance.

Several other studies have attempted to normalise HIIT intensity using different approaches, such as the delta method, the percentage of *W′* depletion, or intensities anchored to critical power (Bossi et al., 2023; Bossi et al., 2024a; Bossi et al., 2024b; Meyler et al., 2023; Wang & Zhao, 2023). However, regardless of the approach, the effect of the prescription method, when present, appears to be consistently small, and none have dramatically reduced inter-individual variability (Bossi et al., 2024a; Collison et al., 2021). This is likely because intra-individual variability has been shown to play a non-negligible role, although it does not fully account for the larger inter-individual differences observed (Bossi et al., 2024a).

Prescribing intensity based on lactate thresholds has been shown to reduce inter-individual variability in blood lactate responses during constant work-rate exercise at moderate and vigorous intensities (Nuuttila et al., 2026). Similarly, time-to-exhaustion trials prescribed using the reserve between mean Wingate power and maximal aerobic power (i.e., the "mean anaerobic scope") have demonstrated reduced inter-subject variability in time to exhaustion (Barnett et al., 1996). However, it remains uncertain whether a method that successfully normalises performance outcomes can also normalise physiological responses, or vice versa.

During HIIT sessions, the use of self-paced exercise has been shown to reduce both intra- and inter-individual variability, allowing all recruited athletes to complete the prescribed training session without premature exhaustion (Bossi et al., 2024a). Similarly, repeated-sprint training performed in an all-out manner appears to elicit more uniform adaptive responses compared to traditional prescription methods, such as those based on a fixed percentage of MAP (Peng et al., 2025; Tongwu et al., 2025). Two possible explanations are: i) that by self-regulating pacing, or simply by giving maximal effort during each bout (as in sprint training), the influence of methodological errors, such as inaccuracies in the determination of ventilatory or lactate thresholds, or in the estimation of *W'*, is minimised; ii) the athlete by self-pacing or exercising in an all-out manner intrinsically (and perhaps unconsciously) take into account the the daily physiological and perforamance variability. Speculatively, however, the self-paced approach may be better suited to experienced athletes, who possess a well-developed sense of effort and pacing, rather than to novices or sedentary individuals, who may lack the ability to regulate intensity effectively.

Study 4 provides some insights for understanding the limitations of *W′* as a predictive parameter and, therefore, as a method to normalize exercise intensity. Contrary to the theoretical assumption that *W′* represents a constant capacity across the entire severe domain, our data revealed that the work actually performed above CP is intensity-dependent. $W_{>CP}$ was lower at intensities approaching both the upper boundary of the severe domain (i.e., near and within the extreme intensity domain) and the lower

boundary adjacent to the heavy domain, especially when W' was estimated using the two-parameter models.

Given the inherent complexity of prescribing intensity for HIIT, it becomes more understandable why the APR construct failed to reduce inter-individual variability in study 1. However, a critical insight emerged from comparing the results of study 1 with the existing literature on similar investigations, as well as with the foundational work of Weyand and Bundle (Weyand et al., 2006; Weyand & Bundle, 2005) and the early observations of Blondel et al. (2001): the relationship between APR and exercise performance appears to be intensity-dependent. In fact, by solving the original APR model developed by Weyand et al. (2006) considering time as an independent variable, it was possible to observe that the advantage of having a high APR is present, but that by reducing the intensity to that of $V\dot{O}_2max$ alone, its contribution became minimal (**Figure 7**). Moreover, it is logical to hypothesize that a reference performance such as instantaneous peak power output, which is strongly correlated with APR, would exhibit a stronger association with performances of similar duration (i.e., very short efforts) than with those that are temporally distant. Yet, upon reviewing the literature, we realised that no data were available on the relationship between APR and time-to-exhaustion in cycling, nor on how exercise intensity modulates this relationship.

This gap in the literature provided the rationale for study 2, which investigated the association between APR (and its variant MPR) and time-to-exhaustion across a range of intensities spanning from 130% to 85% of MAP. We observed a moderate to strong relationship between APR models and T_{lim} at $V\dot{O}_2max$ intensity and above, while its correlation with lower intensity was small and not always significant. However, also emerges that this portion of variance explained is, though, accompanied by large standard errors of estimation, which in turn leads to a low T_{lim} prediction accuracy. In this context, a very recent study has demonstrated that the APR model, when combined with its aerobic reserve component, defined as the difference between MAP and the power output at the LT, can predict performance across a remarkably wide range of durations, from 30 seconds to one hour (Wahl & Ji,

2026). However, the authors employed an isokinetic sprint test at a fixed cadence (120 rpm) to determine PPO, a method which, as discussed in *Section 4.1.4,* is quite impractical in real-world cycling contexts. Second, their profiling protocol was conducted over two separate days, which somewhat diminishes the purported convenience of the APR framework as a single-session assessment tool.

In study 2 to test the construct validity of the APR concept, we also investigated the relationship between APR and indices of anaerobic capacity, like MAOD and *W'*, and how the relationships between them are affected by the lower boundary chosen (i.e., MAP for APR or CP for MPR) and the upper boundary. We observed a slightly greater strength of association between MAOD (and *W'*) and MPR compared to MAOD and APR. This is likely because the APR, using the MAP as lower threshold, includes a higher anaerobic contribution compared to the MPR, which sets the CP as lower edge (Di Gennaro et al., 2025b). Furthermore, by applying partial correlation analyses, controlling once for the lower boundary and once for the upper boundary, we observed that, in trained male cyclists, PPO plays a more dominant role than the lower boundary in determining both APR and MPR. Therefore, if the PPO testing protocol is not adequately standardised, the resulting measurement error may propagate into the APR calculation, compromising its accuracy as a predictive tool. This also raises some concern about the comparability between studies because, to date, the PPO for subsequent APR calculation has been performed as a fixed resistance (Du & Tao, 2023; Tongwu et al., 2025), fixed cadence (Wahl & Ji, 2026) and by the estimated optimal cadence to reach the highest PPO attainable (Di Gennaro et al., 2025a; Di Gennaro et al., 2025b). While it remains unclear which method yields the most accurate determination of PPO, the practical utility of the APR model in field settings ultimately depends on the replicability of the chosen protocol. Therefore, the "best" method may be defined not by theoretical precision alone, but by its feasibility and consistency in real-world conditions.

Prompted by the insight from study 2 that APR's predictive value might be greater for shorter, higher-intensity efforts, we applied the analogous ASR model to a new context: competitive swimming (study 3). In the 50m, 100m, and 200m front crawl events, we found that ASR has a moderate to large relationship with 50- and 100m performance but not with the 200-m event. This result, again, underscores the previously discussed intensity-dependent importance of the APR/ASR construct. Interestingly, in this context, ASR showed a stronger correlation with D' (the swimming analog of *W'*) compared to the association between APR and *W'* found in study 2. This is likely because in study 2, *W'* was derived from a traditional power-duration relationship, while in study 3, D' and the ASR were both derived from the same (modified) 3-minute all-out test, and the D' calculation from this test is based on the ASR itself. Concurrently, the correlation between ASR and D' seen in study 3 aligns with that of Lanzarini et al. (2025), who observed the same trend in running when using the traditional 2-parameter, but not the 3-parameter CP model. To fully interpret these findings, it is necessary to consider the integrated results of studies 2 and 4.

In study 2, a stronger correlation was observed between *W'* and APR when using the 2-parameter hyperbolic model compared to the 3-parameter version. Study 4 provided insight by demonstrating that the estimation of *W'* is highly dependent on both the mathematical model chosen and the intensity of the trials used for its determination. Specifically, it showed that linear models yield *W'* estimates strongly related to mechanical and physiological markers of anaerobic capacity (e.g., MAOD, lactate, Wingate test parameters) compared to hyperbolic models. Furthermore, it confirmed that the correlation between any anaerobic marker and the actual work done above CP is intensity-dependent, being stronger for trials at higher, supra-maximal intensities. Accordingly, the stronger ASR-D' correlation reported by (Lanzarini et al., 2025) is likely attributable to the use of higher intensity trials for the CS/D' determination and to the model selection. In fact, similarly to us, Lanzarini et al. (2025) observed significant correlations between ASR and D' estimated by the 2-parameter model, but not for the 3-parameter model.

6.2 Future perspective

The information provided by this thesis points to clear priorities for future research. First, there is an urgent need to establish consensus on the standardisation of both PPO and MAP determination. Without clearly defined and universally applicable protocols, APR values remain poorly comparable across studies and settings, limiting the model's utility as a robust scientific and practical tool. Second, the applicability of the APR construct should be explored across a broader range of sports and performance durations. While the present thesis focused on relatively short-duration events (cycling (HIIT) and swimming races up to 200 m), the development of non-linear regression models incorporating APR could extend their predictive capacity to longer durations and would allow researchers to prescribe T_{lim} intensities that lead to lower inter-individual variability with just a single preliminary visit.

Third, once robust standardisation is achieved, a further line of inquiry could investigate whether athletes with contrasting APR profiles, i.e., "power-oriented" (high APR) versus "endurance-oriented" (low APR), exhibit parallel differences in muscle fibre type composition and, crucially, whether their adaptive responses to training diverge (Thron et al., 2026). Such knowledge could have strong implications for early talent identification and for the design of individualised training programmes that align with an athlete's inherent physiological predispositions, thereby maximising long-term athletic development.

6.3 Conclusion

The findings presented in this thesis contribute to a growing body of literature examining the APR model. Although the construct was first proposed over three decades ago (Barnett et al., 1996), it has only recently garnered greater attention in both research and applied settings. The APR model results in an intuitive but physiologically incomplete construct. While it moderately correlates with aspects of anaerobic capacity (i.e., accumulated oxygen deficit and curvature constant), this relationship is

inconsistent, method- and model-dependent, and primarily driven by PPO rather than the choice of lower aerobic boundary (i.e., CP or MAP). Second, its practical application for normalising HIIT intensity is not supported by our evidence, as it fails to reduce inter-individual variability more effectively than traditional methods. Despite these limitations, consistent with the current literature, the model retains utility as a practical tool for predicting short-duration performance (<5 min) in both cycling and swimming, and for providing a coarse but accessible profile of an athlete's position along the aerobic-anaerobic continuum.

SECTION 7

Extra publications during candidature

Int J Sports Physiol Perform 2023 Aug 3;18(10):1189-1195. doi: 10.1123/ijspp.2023-0103.

Efficacy of Resisted Sled Sprint Training Compared to Unresisted Sprint Training on Acceleration and Sprint Performance in Rugby Players: an 8-Week Randomized Controlled Trial

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* The author(s) equally contribute to this work

Abstract

Purpose: To compare the effects between resisted sled sprint training (RSS) and unresisted sprint training (URS) on sprint and acceleration performance, vertical jump and maximal strength during an eight-week period of pre-season training. ***Methods:*** Twenty-six recreational active rugby players were randomly divided into either RSS or URS training groups and then performed 8 weeks of training, 2 sessions/week sprint-specific training program. The RSS group performed sprints by towing a sled overloaded with 12.6 % of body mass for 2 of the 3 sets of 3 × 20-m sprints, plus one set was carried out with unresisted modality. The URS groups performed 3 sets of 3 × 20-m unresisted sprints. The measures of 10-m and 30-m sprint times, vertical jump and 3-RM squat tests were performed at baseline and after eight weeks. ***Results:*** 10-m and 30-m sprint times ($p < 0.05$ and $\eta^2_p > 0.44$) improved significantly more in RSS than in URS. Both groups improved significantly in vertical jump and 3-RM squat tests, however, no significant differences ($p > 0.1$ and $\eta^2_p < 0.11$) between groups were found. ***Conclusions:*** Our findings indicate that an 8-week program of resisted sled sprint training is more effective than unresisted sprint training for enhancing sprint time performance in male recreational active rugby players. In addition, these data suggest that a sled overload corresponding to

12.6% of body mass can induce positive effects on both acceleration and speed performance in recreational active rugby players.

Front Physiol. 2023 Dec 8:14:1310319. doi: 10.3389/fphys.2023.1310319.

Fat oxidation rates and cardiorespiratory responses during exercise in different subject populations with post-acute sequelae of SARS-CoV-2 infection: a comparison with normative percentile values

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Abstract

Introduction: Post-acute sequelae of SARS-CoV-2 infection (PASC) presents a spectrum of symptoms following acute COVID-19, with exercise intolerance being a prevalent manifestation likely linked to disrupted oxygen metabolism and mitochondrial function. This study aims to assess maximal fat oxidation (MFO) and exercise intensity at MFO (FATmax) in distinct PASC subject groups and compare these findings with normative data. **Methods:** Eight male subjects with PASC were involved in this study. The participants were divided into two groups: "endurance-trained" subjects (VO_2max > 55 mL/min/kg) and "recreationally active" subjects (VO_2max < 55 mL/min/kg). Each subject performed a graded exercise test until maximal oxygen consumption (VO_2max) to measure fat oxidation. Subsequently, MFO was assessed, and FATmax was calculated as the ratio between ˙VO2 at MFO and ˙VO2 max. **Results:** The MFO and FATmax of "endurance-trained" subjects were 0.85, 0.89, 0.71, and 0.42 and 68%, 69%, 64%, and 53%, respectively. Three out of four subjects showed both MFO and FATmax values placed over the 80th percentile of normative data. The MFO and FATmax of "recreationally active" subjects were 0.34, 0.27, 0.35, and 0.38 and 47%, 39%, 43%, and 41%, respectively. All MFO and FATmax values of those subjects placed below the 20th percentile or between the 20th and 40th percentile. **Discussion:** Significant differences in MFO and FATmax values between 'endurance-trained' and "recreationally active" subjects suggest that

specific endurance training, rather than simply an active lifestyle, may provide protective effects against alterations in mitochondrial function during exercise in subjects with PASC.

Cryobiology 2024 Dec:117:105158. doi: 10.1016/j.cryobiol.2024.105158. Epub 2024 Nov 12.

Effects of cryo-facial mask on running performance in amateur middle-distance runners

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Abstract

The excess heat accumulated during exercise can lead to stress-induced fatigue, possibly impairing athletic performance. Various precooling techniques have been applied to enhance thermal comfort, reduce perception of effort, and improve endurance. In this randomized crossover study, twelve male amateur middle-distance runners (age: 33.69 ± 5.9 years; body mass: 71.9 ± 4.4 kg; height: 178.4 ± 5.6 cm; VO_2peak: 63.3 ± 5.6 mL/min/kg) wore a facial cooling mask before a time-to-exhaustion (TTE) test on a treadmill, under cryostimulation or control conditions. The running performance comprised also two constant load trials, one conducted before and another after wearing the mask, both performed at the velocity of the first ventilatory threshold. Under cryostimulation condition, the TTE was 13 % higher than the control condition ($p = 0.0049$; $d = -0.19$) with a significant main effect of time for both ratings of perceived exertion ($F1, 22 = 50.10$; $p < 0.0001$; $\eta 2\ p = 0.69$) and heart rate ($F1, 22 = 31.53$; $p < 0.0001$; $\eta 2\ p = 0.59$). A significant interaction "condition × time" was found for facial skin temperature ($F2, 44 = 36.93$; $p < 0.0001$; $\eta 2p = 0.63$) and for heart rate during the constant load trial after wearing the mask ($F1, 22 = 5.90$; $p = 0.0238$; $\eta 2p = 0.21$). The localized cryostimulation provided by the mask lowered the skin temperature on the face, potentially mitigating the negative effects of heat stress during running. Incorporating the cryo-facial mask as part of a pre-exercise routine for runners may offer a practical and convenient method to optimize performance and enhance overall training outcomes.

Int J Sports Physiol Perform. 2024 Nov 28;20(1):99-108. doi: 10.1123/ijspp.2023-0468.

Moderate-Duration Dynamic Stretching During Warm-up Improves Running Economy and Running Performance in Recreational Distance Runners

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Abstract

Purpose: The purpose of this study was to investigate, in distance runners, the acute effects of moderate durations (60 s per leg) of static (SS) and dynamic stretching (DS) on running economy (RE) and performance. **Methods:** Twelve recreational runners completed a randomized crossover design. Initially, the second ventilatory threshold (VT2) and the speed associated with the maximal oxygen uptake (VO2max) (vVO2max) were determined through an incremental test. Then, participants completed submaximal continuous-running (75%VT2 and 85%VT2) and running-until-exhaustion (vVO2max) tests preceded by 3 warm-ups: running plus SS or DS (SS or DS conditions) and running without stretching (NS condition). The SS and DS conditions consisted of 5 minutes of running plus 10 minutes of SS or DS, respectively, and the NS condition consisted of 15 minutes of running without stretching. RE at 75%VT2 and 85%VT2, time to exhaustion, and total running distance were evaluated. Rating of perceived exertion was also assessed. **Results:** Running economy at 75%VT2 resulted significantly better in the DS than in the NS ($P < .001$) and in the SS ($P < .05$). Time to exhaustion and total running distance were significantly improved in DS compared with NS ($P < .001$) and SS ($P < .01$). No differences in rating of perceived exertion among conditions were found. **Conclusions:** Our results showed that, in recreational distance runners, a pre-exercise moderate-duration bout of DS improved RE and enhanced total running distance and time to exhaustion, whereas 60 seconds of SS did not induce significant improvements. Overall, our study demonstrates the

effectiveness of moderate DS durations in optimizing RE and performance parameters, showing that such effects depend on the stretching modality used.

Int J Sports Physiol Perform. 2025 Mar 14;20(5):700-708. doi: 10.1123/ijspp.2024-0133.

Effects of Moderate- Versus Mixed-Intensity Rowing Training on Physiological Responses and Performance in Highly Trained Adolescent Rowers: A Pilot Study

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Abstract

Purpose: In rowing, the effectiveness of adding high-intensity interval training (HIIT) to moderate-intensity continuous training (MICT) within the weekly training program on physiological adaptations and performance is still unclear. This study compared the effects of HIIT plus MICT (MIXED) versus MICT alone on physiological/metabolic responses and performance in adolescents. **Methods:** Twelve highly trained adolescent rowers (age: 15.7 [0.5] y) were divided into 2 groups: MIXED and MICT. Before and after a 7-week intervention period, rowers underwent an incremental step test to determine peak oxygen uptake (VO2peak), power at VO2peak (WVO2peak), power corresponding to a lactate concentration of 2 and 4 mmol·L-1, power output at lactate threshold, oxygen uptake at the second lactate threshold (VO2LT), and peak oxygen pulse. Training load from TRIMP was also measured. The training intervention consisted of 7 sessions per week including 2 "off-water," 3 "on-water," and 2 resistance-training sessions. The "on-water" and resistance-training sessions were the same for both groups, while during "off-water" sessions, the MIXED group performed HIIT (4 × 4 min at 85% WVO2peak) and the MICT group performed moderate-intensity training (80 min at 70% WVO2peak). **Results:** Statistical analysis showed that in the MIXED group, VO2LT was significantly increased and training load from TRIMP was significantly reduced ($P < .00001$) compared with the MICT group ($P = .008$). Both groups similarly improved VO2peak, peak oxygen pulse, WVO2peak, power output at lactate threshold, and power corresponding to a lactate concentration of 2 and 4 mmol·L-1.

Conclusions: Our findings showed that, in adolescent rowers, MIXED training enhanced VO2LT, thus indicating HIIT as a valid and time-efficient addition to traditional MICT. However, given that adolescents were examined, data should be interpreted with caution, as training and/or growth/maturation may have contributed to performance changes.

SECTION 8

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Attestation of Authorship

I hereby declare that the work contained in this thesis has not been previously submitted either in whole or in part to qualify for any other academic award. I also certify that the thesis is my own work carried out during my candidature and that any assistance that I have received in my research work and in the preparation of this thesis has been acknowledged.

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