The Kinetics of the
Work Capacity Above Critical Power

Submitted by Philip Friere Skiba to the University of Exeter as a thesis for the degree of Doctor of Philosophy in Sport and Health Sciences, June 2014.

I certify that all material in this thesis which is not my own work has been identified and that no material has previously been submitted and approved for the award of a degree by this or any other University.

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Abstract:

The critical power (CP) model includes two constants: the CP and the W′ [P = W′ / t) + CP]. The W′ is the finite work capacity available above CP. Power output above CP results in depletion of the W′; complete depletion of the W′ results in exhaustion. It is possible to model the charge and discharge of the W′ during intermittent exercise using a novel integrating model (the W'_{BAL} model), and to generate a function describing a curvilinear relationship between time constants of reconstitution of the W′ in terms of the difference between recovery power and CP (D_{CP}) ($r^2 = 0.77$). The depletion of the W′ as predicted by the W'_{BAL} model during intermittent exercise is linearly related to the rise in $\dot{V}O_2$ above exercise baseline ($r^2 = 0.82 – 0.96$).

During intermittent exercise, the W'_{BAL} model is generally robust with respect to the length of work and recovery interval, yielding a mean under-prediction of the W'_{BAL} of only -1.6 ± 1.1 kJ. The amount of W′ remaining after a period of intermittent exercise correlates with the difference between the subject's $\dot{V}O_2$ at that time ($\dot{V}O_{2START}$) and $\dot{V}O_{2PEAK}$ (D_{VO2}) ($r = 0.79$, $p < 0.01$). Moreover, the W'_{BAL} model also performs well in the field, permitting accurate estimation of the point at which an athlete becomes exhausted during hard training or competition (mean W'_{BAL} at exhaustion = 0.5 ± 1.3 kJ (95% CI = 0 – 0.9 kJ). The W'_{BAL} model meets the mathematical criteria of an excellent diagnostic test for exhaustion (area under ROC curve = 0.91).

^{31}P magnetic resonance spectroscopy during single leg extensor exercise revealed a correlation between the recovery of the W'_{BAL} model and recovery of creatine phosphate ([PCr]) after a bout of exhaustive single leg extensor exercise ($r = 0.99$, $p < 0.01$). The W'_{BAL} model also accurately predicted recovery of the W' in this setting ($r = 0.97$, $p < 0.05$). However, a complete understanding of the relationship between the depletion and recovery of [PCr] and the depletion and recovery of the W' remains elusive. Muscle carnosine content is curvilinearly related to the rate of W'_{BAL} recovery, with higher muscle carnosine associated with faster recovery, with implications for muscle buffering capacity and calcium handling.

The W'_{BAL} model may be recast in the form of a differential equation, permitting definition of the time constant of recovery of the W'_{BAL} in terms of the subject's known W' and the D_{CP}. This permits the scaling of the model to different muscle groups or exercise modalities. Moreover, modifications to this mathematical form may help explain some of the variability noted in the model in earlier studies, suggesting novel avenues of research. However, the present formulation of the W'_{BAL} model is mathematically robust and represents an important addition to the scientific armamentarium, which may aid the understanding the physiology of human performance.

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Symbols and Abbreviations

P	Power
W	Watt
CP	Critical Power; asymptote of the power-duration relationship
$\mathbf{W'}$	"W-Prime"; curvature constant of the power-duration relationship
$\mathbf{W'_{BAL}}$	Amount or balance of W' remaining
$\mathbf{T_{LIM}}$	Time limit of tolerance
CS	Critical Speed
$\mathbf{D'}$	"D-Prime"; curvature constant of speed-duration relationship
AWC	Anaerobic work capacity
WEP	Work done above end power (3-minute all-out test), synonymous with W'
EP	End test power (3-minute all-out test), synonymous with CP
$\boldsymbol{\tau}$	tau; time constant or characteristic time, time (s) for a response to reach 63% of maximum
$\boldsymbol{\tau_{W'}}$	Tau-W'; time constant of recovery of the W'
$\mathbf{D_{CP}}$	Difference between recovery power and CP
CWR	Constant work rate
$\dot{\mathbf{V}}\mathbf{O_2}$	Volume of oxygen uptake
$\dot{\mathbf{V}}\mathbf{O_{2MAX}}$	Maximum oxygen uptake
$\dot{\mathbf{V}}\mathbf{O_{2PEAK}}$	Peak oxygen uptake
$\dot{\mathbf{V}}\mathbf{O_{2START}}$	$\dot{V}O_2$ at the end of intermittent exercise (c.f. Chapter 5)

D_{VO2}	Difference between $\dot{V}O_{2START}$ and the $\dot{V}O_{2PEAK}$ recorded at the end of CWR (c.f. Chapter 5)
GET	Gas exchange threshold
LT	Lactate threshold
MLSS	Maximal lactate steady state
OBLA	Onset of blood lactate accumulation
^{31}P-MRS	^{31}P Magnetic resonance spectroscopy
^{1}H-MRS	^{1}H Magnetic resonance spectroscopy
PCr	Creatine phosphate
$D_{[PCr]}$	Difference between [PCr] at the end of recovery and [PCr] at the time of exhaustion
[X]	Concentration of substance X (i.e. K^+, PCr, etc)
P_i	Inorganic phosphate
Ca^{2+}	Calcium
K^+	Potassium

Declaration, Communications and Publications

The material contained within this thesis is the original work of the author, and was conducted and written by the author. The following communications and publications are a direct consequence of this work.

2014

Skiba PF, Clarke DC, Vanhatalo A, Jones A. Validation of a Novel Intermittent W′ Model for Cycling Using Field Data. *Int J Sports Physiol Perform*. February 2014. In press / E-Pub ahead of print.

Skiba, Philip Friere, Sarah Jackman, David Clarke, Anni Vanhatalo and Andrew M. Jones. Effect of Work & Recovery Durations on W′ Reconstitution during Intermittent Exercise. *Med Sci Sports Exerc*. January 2014. In press / E-Pub ahead of print.

2013

Noordhof DA, **Skiba PF**, de Koning JJ. Determining anaerobic capacity in sporting activities. *Int J Sports Physiol Perform*. 2013 Sep;8(5):475-82. Review.

Chidnok W, Fulford J, Bailey SJ, Dimenna FJ, **Skiba PF**, Vanhatalo A, Jones AM. Muscle metabolic determinants of exercise tolerance following exhaustion: relationship to the "critical power". *J Appl Physiol*. 115(2):243-50. July 2013

Chidnok W, DiMenna FJ, Fulford J, Bailey SJ, **Skiba PF**, Vanhatalo A, Jones AM. Muscle metabolic responses during high-intensity intermittent exercise measured by (31)P-MRS: relationship to the critical power concept. *AmJ Physiol Regul Integr Comp Physiol*. 2013 Nov 1;305(9):R1085-92.

Clarke DC, **Skiba PF**. Rationale and resources for teaching the mathematical modelling of athletic training and performance. *Adv Physiol Educ*. 37(2):134-52. June 2013.

Wylie LJ, Kelly J, Bailey SJ, Blackwell JR, **Skiba PF**, Winyard PG, Jeukendrup AE, Vanhatalo A, Jones AM. Beetroot juice and exercise: pharmacodynamic and dose-response relationships. *J Appl Physiol*. May 2, 2013. [Epub ahead of print]

Clarke, David and **Philip Friere Skiba**. Athletic Performance Engineering. IAP. Massachusetts Institute of Technology. January 19-20, 2013.

Jones, Andrew M, Anni Vanhatalo, David Poole and **Philip Friere Skiba**. Critical Power: Cardiovascular and Muscle Metabolic Determinants of Oxygen Uptake. Symposium. *ACSM Annual Meeting, Indianapolis, IN*. May 30, 2013.

Skiba, Philip Friere. Performance Engineering: A Legal Approach for the Elite Athlete. *ESPN / MIT Sloan Sports Analytics Conference Presented by CNN*. Boston, MA. March 1, 2013.

2012

Skiba, Philip Friere, Weerapong Chidnok, Anni Vanhatalo and Andrew M. Jones. Modelling the expenditure and reconstitution of work capacity above critical power. *Med Sci Sports Exerc.* 44(8):1526-32. August 2012.

Skiba, Philip Friere. The Fatigued Athlete: An Overview From Math To Medicine. *Keynote lecture, AOASM Annual Conference*. April 2012.

2011

Skiba, Philip Friere, Weerapong Chidnok, Anni Vanhatalo and Andrew M. Jones. Modelling the charge / discharge status of the W' during intermittent exercise. *Med Sci Sports Exerc*. 43(5):141. May 2011. Abstract.

Skiba, Philip Friere and Andrew M. Jones. Commentary on: VIEWPOINT: Michael J. Joyner, Jonatan R. Ruiz, and Alejandro Lucia. The Two-Hour Marathon: Who and When? *J Appl Physiol*, January 2011. (Letter)

2010

Clarke, David C. and **Philip Friere Skiba**. Rationale & Resources for a Course Module in Athletic Performance Engineering. *Biomedical Engineering Society Annual Meeting*. October 9, 2010.

Skiba, Philip Friere. 'Aerobic' and 'Anaerobic': Understanding Modern Exercise Physiology. Invited lecture, AOASM Annual Conference. May 2010.

Skiba, Philip Friere. Putting It All Together: The Synthesis of Physiology and Human Performance. Invited lecture, AOASM Annual Conference. May 2010.

Foreword and Acknowledgements:

In Michael Frayn's award-winning play *Copenhagen*, we witness young Werner Heisenberg in a lively discussion with his mentor Neils Bohr on the nascent mathematics of quantum mechanics.

> **Heisenberg:** What something means is what it means in mathematics.
>
> **Bohr:** You think that so long as the mathematics works out, the sense doesn't matter.
>
> **Heisenberg:** Mathematics *is* sense! That's what sense is!

Both of my long-suffering thesis advisors will confirm that we three have had very similar conversations many times over the course of my studies in Exeter. I make no attempt to draw parallels between ourselves and the titans of nuclear physics using the above quotation. Rather, I'm fond of the Heisenberg – Bohr dynamic because I have believed, from earliest childhood (and often to the chagrin of my instructors), that the great mysteries of biology may be best understood though mathematics. However, this world-view becomes particularly difficult in light of the uncertainties and measurement difficulties inherent in biological systems. We have often found ourselves perched on the very narrow intersection of that which is mathematically *defensible* and physiologically *plausible*, yet unacceptably *speculative*.

Modelling is a dangerous business, and not simply because of our difficulties in data collection. Professor Manfred Eigen once wrote, "A theory has only the alternative of being right or wrong. A model has a third possibility: it may be right, but irrelevant." This is an important point, and one often lost on the mathematically minded. Our goal must not be a quantitatively perfect, yet intellectually gauche formulation. This sentiment was best described during a lecture to the Royal Society by Prof. Samuel Karlin. "The purpose of models is not to fit the data," he said, "But to sharpen the questions."

This thesis began as an effort to develop useful mathematical tools to assist the training and performance of athletes. However, the beauty of the mathematics lies not merely in the possibility of optimizing human performance (though this is a noble aim), but in our ability to interrogate these models with respect to the underlying physiology. The critical power model appears to apply across kingdom, phylum and class of animal life (68, 90, 91, 149). These observations suggest a highly conserved and organized physiological process, and perhaps a unifying principle of bioenergetics. In short, it is something worth understanding for its own sake. It is my hope that this work will provide a mathematical and conceptual framework that may move this process of understanding forward.

As intimated above, this was not a solitary endeavor. I must first thank my advisors, Dr. Vanhatalo and Professor Jones for their good counsel throughout this process. I came as a math-minded sports physician, convinced that a biological mystery would yield to my calculations. I leave as a physiologist who (with some sadness) understands that certain biological questions may not be answerable to the level of mathematical precision I would like. I am indebted to both of my advisors for allowing me the academic latitude to discover this in my own time.

When I arrived in Exeter, I did not fully appreciate the integration of the team at the College of Life and Environmental Science. I am grateful to the colleagues and friends I have made during my time here. It is a rare place indeed where such a large collection of scientists and support staff are so interested in mutual success. It was never difficult to find subjects, collaborators, or helping hands. Perhaps more importantly, when personal tragedy struck, this same group was willing to rally around and offer support to an American who was a long way from home. Although it seems my ultimate destiny will be found in the United States, I say without hesitation that the opportunity to work with the good men and women in Exeter has been the greatest good fortune and honour of my academic career. I look forward to our continued friendship and collaboration in the years to come.

Not all of my important collaborators are at the University of Exeter. I have been privileged to collaborate with Dr. David Clarke of the Massachusetts Institute of Technology (now of Simon Fraser University), and Dr. Dionne Noordhof and Professor Jos de Koning of the MOVE Institute Amsterdam. It would also be remiss of me not to acknowledge the importance of the mathematical input and criticism offered by Kevin Joubert (particularly with respect to the integration in Chapter 8), as well as Dr. Andy Froncioni. Sports scientists who both work with elite athletes and understand mathematics are a relative rarity, and I am fortunate to have had the opportunity to work with colleagues of such high professional standard.

I must also remark on my family. I was born with both insatiable curiosity and (perhaps pathological) enthusiasm for discovery, but my need to understand has been incubated in a milieu of people who stressed the importance of academics. I had a mother who taught me to read, and a father who ensured his university texts were readily available to my young mind. I had grandparents and a great-grandfather who instilled in me a fundamental belief in education above all else. I had an uncle who dragged me out of bed in the dark of the night to view lunar eclipses and meteor showers so that I would experience science first hand. When it became clear that the scientific and philosophical questions I had were without answers, this same group (not to mention my wife) supported my decision to clear off to a laboratory halfway around the world to discover them myself. My career as a physician and scientist has been a team effort from birth. The credit for this thesis is as much theirs as it is mine; any errors are mine alone.

As has been made clear above, I was blessed with both the opportunity and support to succeed in my professional life. Despite these same advantages, my brother Michael was foiled by personal challenges that proved insurmountable. It often occurs to me that our positions might well have been reversed, had I lived his life. This body of work is dedicated to his memory.

"What we observe is not nature itself, but nature exposed to our method of questioning."

-Professor Werner Heisenberg (1901-1976)
Nobel Laureate (Physics)

"I found England was a heavenly place for me. I don't care who else finds it difficult, but to me, it's heaven."

-Willie "Drive 'Em Down" Hall (d.1930)
American Blues Musician

I. Introduction

1.0 Conceptual Framework and Basic Mathematics

Throughout history, scientists have used the study of the kinetics of natural phenomena as a means to understand basic underlying laws of nature. For example, we may consider the apocryphal stories of Sir Isaac Newton and the apple, or of Galileo dropping a variety of cannonballs from the Tower of Pisa. In more modern times, we may consider the mathematical rules governing enzyme activity in solution (158), or of the utilization of oxygen (109). In each case, valuable discoveries were made by carefully considering the way natural systems change over time. In fact, entire systems of mathematics (the calculus) were developed specifically for use in describing such changes (175, 215).

One of the first applications of the calculus was in understanding the relationship between work and power. *Work* is most easily understood as a force multiplied by the distance over which it acts. The SI unit of work is the joule (J). *Power*, in contrast, is a measure of work *rate*, or work divided by time. The SI unit is the watt (W), equal to 1 $J \cdot s^{-1}$. In pictorial terms, given a graph of power over time, we may learn the work done by calculating the area under a particular part of the curve though the process of integration.

In modern sport, work and power have been used in a variety of ways to facilitate an understanding of the response of the human body to exercise using mathematical models (65). Two mathematical constructs in particular offer complementary information about physical performance capacity: the Critical Power (CP) model (164, 165, 239) and the Banister impulse-response (IR) model (18, 20, 23, 65). The CP model describes the relationship between work rates (i.e. power or velocity) and the durations for which an individual can sustain them during constant-work exercise. The IR model describes the dynamics by which an individual's performance capacity changes over time as a function of training. In other words, the CP model tells us *what* the athlete may be capable of, whilst the IR model tells us *when* they may be capable of it. Both models elegantly abstract the underlying physiology, and both can accurately fit performance data. The former will be the primary subject of this dissertation.

1.1 Exercise tolerance

The "threshold" phenomenon

Athletes are familiar with the concept of a threshold phenomenon with respect to perceived exertion and performance. Indeed, popular sports training literature is rife with references to some level of effort that represents a "red line", above which fatigue rapidly ensues (73, 117, 205). That lay people often (and certainly erroneously) attribute this threshold phenomenon to singular physiological processes such as blood lactate accumulation is immaterial. As a practical matter, athletes rapidly learn to respect this perceptual cue or consign themselves to premature exhaustion and suboptimal performance (205).

When first developed, the CP model represented a means of codifying this threshold phenomenon without the outright invocation of a discrete physiological mechanism (164). It simply offers an asymptote known as the CP, based upon a curve plotted through an athlete's time limit of tolerance at a variety work rates (power or velocity) (Figure 1.0). Below this asymptote, the athlete may maintain the selected work rate for "a long time" (164). If the athlete attempts a work rate above this asymptote, they begin an inexorable path towards exhaustion. Thus, the model is extremely practical. However, in recent years it has also become clear that the parameters of the model have important physiological implications (85, 133, 134, 173, 185). The CP model therefore sits at the crucial intersection of basic biological principles and human experience, and is worth considering in detail.

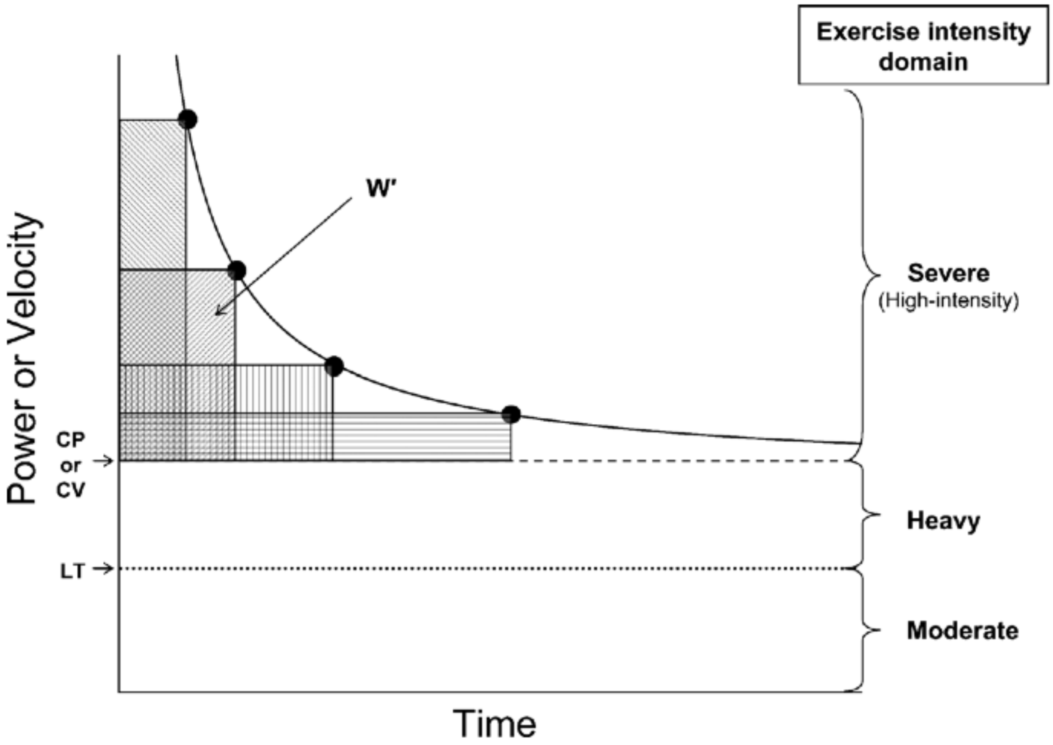


Figure 1.0: *Schematic representation of the power-duration relationship codified by the CP model. Dots indicate time limit of tolerance at a particular work rate. Dashed line indicates asymptote or CP. Hatched boxes are of equivalent area, termed the W'. Of note, the W' remains constant irrespective of the rate of discharge. It can be expended quickly or slowly, but the amount of energy available does not change. Right axis denotes "domains" of exercise tolerance used in the present work. The "extreme" domain is omitted for clarity. See text for details. Data reprinted from (130), with permission.*

Chapter 2: Literature Review

2.0 $\dot{V}O_2$ kinetics as a defining feature of exercise tolerance

In the early part of the 20th century, the advent of gas analysis offered the opportunity to directly record the inner workings of the human during exercise. Hill and Lupton (106) made observations indicating that there existed physiological states that accompanied exercise which were fundamentally stable or unstable. (Arguably, Krogh and Lindhard (141) may have first recorded similar phenomena even earlier, in 1913 (c.f. Fig. 7), though the extent to which they understood this is unclear). Hill and Lupton differentiated these as "moderate" and "severe" exercise intensities (106), or as "steady" and "not steady" (107, 110). It would not be until the latter half of the 20th century that higher resolution methods of monitoring $\dot{V}O_2$ emerged. This permitted the development of two schemata for the definition of exercise intensity, based upon the kinetics of the $\dot{V}O_2$ response to physical work (129, 183, 185, 234, 242).

In these schemata, the $\dot{V}O_2$ response has been divided into four analogous 'domains': *moderate, heavy, severe and extreme* (Figure 2.0) (93, 112, 185, 244), or *moderate, heavy, very-heavy and severe* (174, 234, 242). The demarcation between *moderate* and *heavy* is the gas exchange threshold (GET) or lactate threshold (LT) (235, 236), and between *heavy* and *severe* (or *heavy* and *very-heavy*), the CP (43, 112, 127, 130, 185, 225, 234).

In the first (*moderate*) domain, after a small initial rise in $\dot{V}O_2$ (termed the cardiodynamic phase (241)), the $\dot{V}O_2$ profile is well fit by a monoexponential function, suggesting first-order linear control dynamics (235, 236, 238, 240-242). A defining feature of this domain is that the rate constant of the relationship is independent of the eventual steady-state $\dot{V}O_2$ (237), which occurs without concomitant metabolic acidosis (232, 233, 236). This is due to the maintenance of an equilibrium between the rate of consumption of ATP in working myocytes and the rate of production of ATP via oxidative phosphorylation.

The second (*heavy*) domain is characterized by the superimposition of a second, slower exponential rise in $\dot{V}O_2$ (the $\dot{V}O_{2sc}$), which changes as a function of time, rather than work rate alone (99, 152, 177, 235, 236); that is, $\dot{V}O_2$ manifests an increase despite a constant work rate. This $\dot{V}O_{2sc}$ becomes manifest approximately 90-120 s after exercise commences (43, 235), although there has been some controversy on this point (214). The $\dot{V}O_{2sc}$ may take 10 *minutes* or more to stabilize (25, 177, 235, 242). It is also possible to observe an increased (but stable) arterial [lactate] and [H^+], along with intramuscular [PCr], [P_i], and pH (134, 135).

In the third (*severe* or *very-heavy*) domain, the $\dot{V}O_{2SC}$ does not stabilize, despite a constant mechanical power output (43, 93, 127, 128, 130, 177, 185, 235, 236) (Figure 2.0). These domains are characterized by a progressive loss of metabolic homeostasis; $\dot{V}O_2$, [Pi], and arterial [lactate] exhibit steady increases, whilst [PCr] and pH steadily decrease until the subject achieves $\dot{V}O_{2MAX}$ and becomes exhausted soon after (174, 234, 242). The effect of the $\dot{V}O_{2SC}$ is substantial, having been demonstrated to contribute more than 1 L / min to the overall $\dot{V}O_2$ response (41, 177, 237).

The fourth domain is defined slightly differently between the schemas. The *extreme* domain comprises power outputs where the subject becomes exhausted before achieving $\dot{V}O_{2MAX}$ (112). The analogous *severe* domain includes power outputs where the ATP requirement is in excess of that at $\dot{V}O_{2MAX}$ (174, 234, 242).

Excellent contemporary reviews of $\dot{V}O_2$ kinetics are available (129, 183, 191). For the purposes of agreement with the present author's published work, the *moderate / heavy / severe / extreme* schema (112, 185) will be used in this thesis.

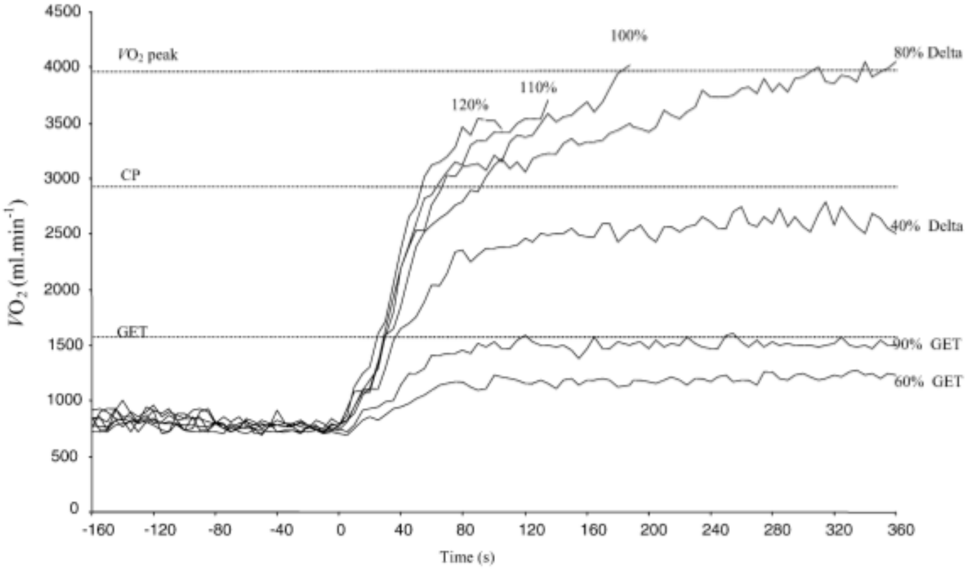


Figure 2.0: *$\dot{V}O_2$ response in the moderate, heavy, severe and extreme domains. 60% GET and 90% GET indicate exercise was undertaken at those respective percentages of*

the power output that elicits the GET. 40% and 80% Delta indicate that exercise was undertaken at those respective percentages of the difference between GET and $\dot{V}O_{2PEAK}$*. 100%, 110% and 120% indicate exercise undertaken at those respective percentages of* $\dot{V}O_{2PEAK}$*. Data reprinted from (244), with permission.*

As discussed in the preceding paragraphs, the well-documented instability of the pulmonary $\dot{V}O_2$ response with respect to time in the severe domain does not come in isolation. Rather, other broad markers of stress also rise as a subject approaches $\dot{V}O_{2MAX}$, sometimes to characteristic maxima. In addition to the behaviour of blood lactate (93, 185), epinephrine and norepinephrine also exhibit an inexorable rise (95, 184, 185). Integrated electromyogram activity exhibits a similar characteristic pattern (228). Of paramount interest to the present work is the observation that a generalized physiological steady state *cannot* be attained within the severe domain. Moreover, within the severe domain, there exists an apparent symmetry between the work rate a subject can tolerate for a particular period of time, and the time it takes the subject to achieve $\dot{V}O_{2MAX}$ (173, 185, 244) (Figure 2.1). This suggests that basic physiological principles may govern the observed power-duration relationship of human performance (173).

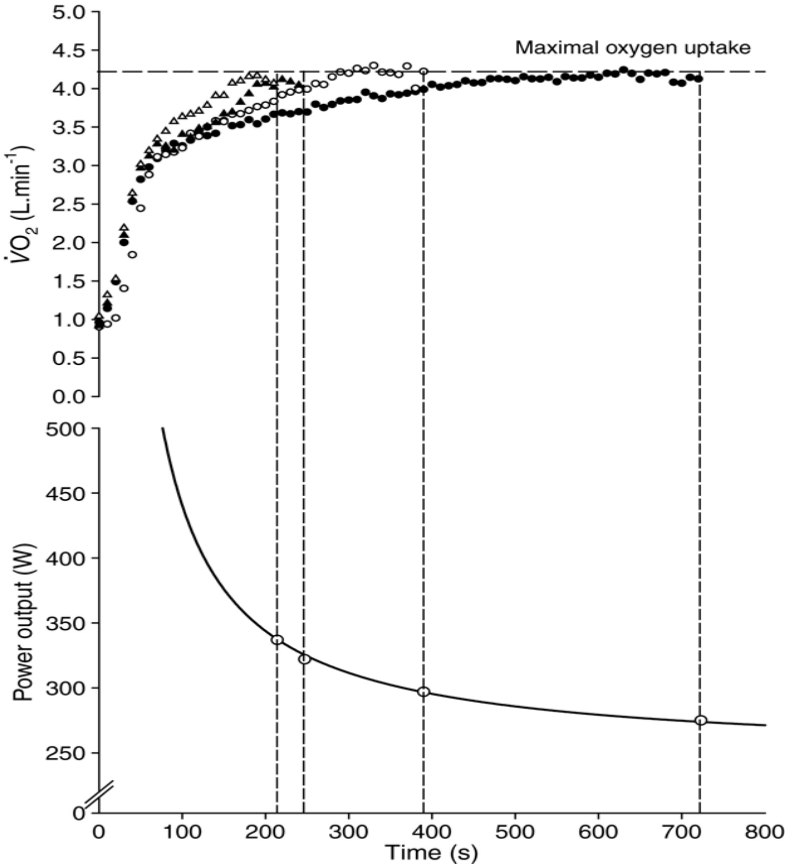


Figure 2.1: *Correlation between time limit at a particular work rate and pulmonary* $\dot{V}O_2$ *for a particular subject. Note that exercise termination appears to coincide with the attainment of* $\dot{V}O_{2MAX}$. *Data reprinted from (43), with permission.*

Mechanistic bases and implications of the $\dot{V}O_{2sc}$

A working understanding of the $\dot{V}O_{2sc}$ is crucial to fully appreciate the link between physiology and performance. Given this, the historical treatment of the $\dot{V}O_{2sc}$ is extremely interesting, describing a sort of cognitive dissonance within respiratory physiology (128). For the majority of the past century, $\dot{V}O_2$ kinetics were widely accepted to be best fit by a simple exponential model, irrespective of exercise intensity (104, 106). The $\dot{V}O_2$ response would come to be understood to be largely representative of a first-order linear system, determined chiefly by the mitochondrial creatine kinase reaction (129, 138, 235, 242). However, data existed from the early 20th century that indicated a simple monoexponential model might not be appropriate for all work rates. Hill and Lupton attempted to explain away an increase in $\dot{V}O_2$ after several minutes of running in subject "H" as the consequence of an inefficiency resulting from a blistered foot (110). Jones et al. (128) have discussed other (perhaps better-known) examples of the observation and apparent dismissal of the $\dot{V}O_{2sc}$ (13, 14).

By the 1970's, work had begun in earnest to better characterize and understand the $\dot{V}O_{2sc}$, with Whipp and Wasserman noting the possibility of a fast and slow exponential component (236). A number of putative mechanisms were proposed to explain the $\dot{V}O_{2sc}$ (lactate accumulation, muscle temperature, the work of remote muscles, among others) (99). A significant step forward came from Poole et al. in 1991 (184), who demonstrated that more than 80% of the $\dot{V}O_{2sc}$ must arise within the exercising muscle mass. Indeed, this was first directly visualized by Rossiter et al. (194) using ^{31}P-MRS, who observed a "slow component" of [PCr] utilization. In the mid-1990's, Barstow et al. (24) would present indirect evidence that the $\dot{V}O_{2sc}$ was correlated with the type II fibre pool. This work would inform the biopsy studies of Peter Krustrup and his colleagues in Copenhagen, who demonstrated a fundamental linkage between muscle fibre recruitment and the development of the slow component (143, 144, 147) in a series of elegant studies. These data indicated that the $\dot{V}O_{2sc}$ is associated with additional fibre recruitment (144), that glycogen depletion of the type I fibre pool resulted in an increased type II fibre activity and $\dot{V}O_2$ (145), and that selective neuromuscular blockade of the type I fibre pool resulted in enhanced type II fibre activation (143). It is advisable to avoid over-interpretation of this limited selection of studies, owing to evidence (in both exercising humans and in-situ canine preparations) that the $\dot{V}O_{2sc}$ may *also* be in part the result of a loss of efficiency in fibres that have already been recruited (56, 228, 246). That is, progressive recruitment of muscle fibres is not strictly required to observe a slow component. It remains unclear the extent to which we can apportion parts of the $\dot{V}O_{2sc}$ to these potentially interrelated mechanisms.

It is possible to synthesize the above studies and thus develop a conceptual framework linking the recruitment of discrete muscle fibre pools to the kinetic observations made of the pulmonary $\dot{V}O_{2sc}$ (24, 128, 243). It is tempting to hypothesize that that the slow component, fibre recruitment, and the power-duration relationship may be mechanistically linked (173, 228). That is: one consequence of exercise in the severe domain is the progressive fatigue and successive recruitment of higher order (e.g. type II) muscle fibres (3, 101, 144, 157, 202, 242) (Figure 2.2). This may result in a progressive metabolic instability due to the basic biochemical and respiratory properties of the type II fibres (100, 200, 229), which may be collectively observed as both the inexorable rise in pulmonary $\dot{V}O_2$ and limit of exercise tolerance in the severe domain (Figure 1.2) (43, 157, 173). This framework has certain implications in terms of the character and control mechanisms of the $\dot{V}O_{2sc}$ (242). Rather than describing the mass recruitment of a uniform metabolic "compartment", the $\dot{V}O_{2sc}$ may well be representative of the summative response of a number of smaller compartments (242, 243) (Figure 2.2). This response may differ based upon the precise nature of the exercise task undertaken.

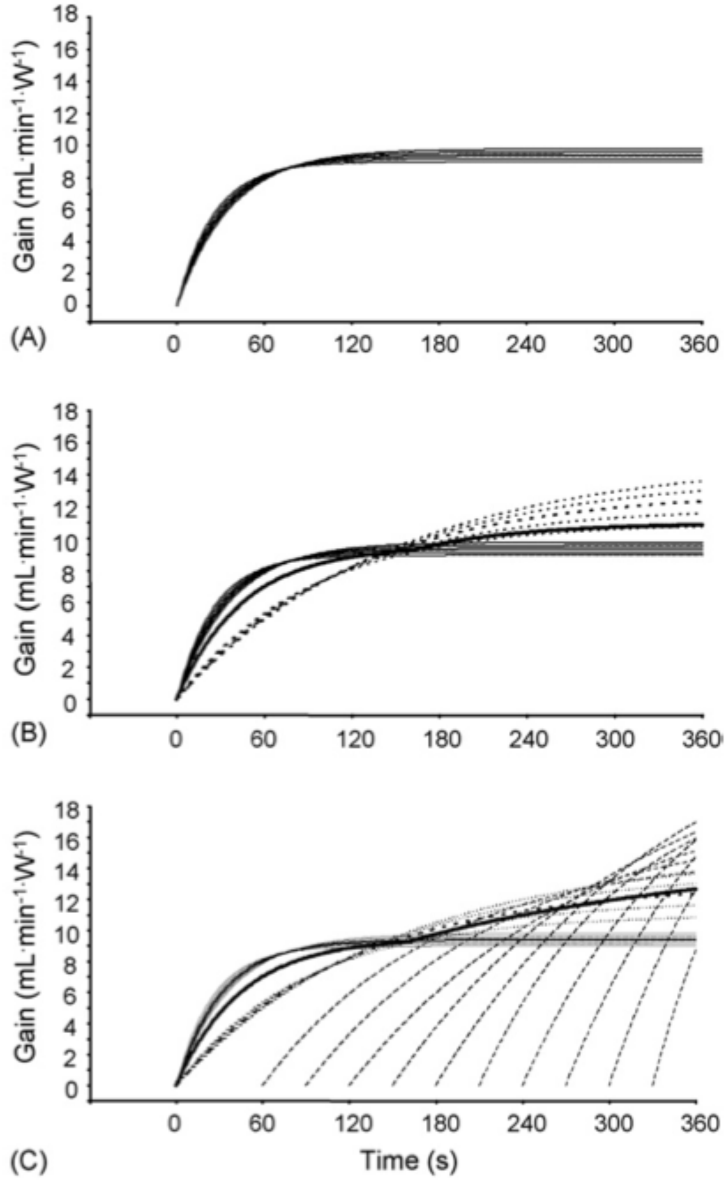


Figure 2.2: *Theoretical model presented by Wilkerson and Jones (243), reprinted with permission. Panel A: Fibres low in the recruitment hierarchy yield group kinetics reminiscent of the* $\dot{V}O_2$ *response in the moderate domain. Panel B: The recruitment of higher order fibres result in kinetics reflective of the heavy domain. Panel C: The progressive recruitment of additional higher order motor units in the severe domain result in a response that does not stabilize.*

Constant work rate versus intermittent exercise

A considerable portion of the literature base of the field of $\dot{V}O_2$ kinetics concerns constant work rate exercise, or step changes between constant work rates (129, 183, 191). Indeed, the typical mathematical approach to understanding system response involves imposing step transitions in system input (i.e. external work) and careful study of the resultant output (i.e. the pulmonary $\dot{V}O_2$ signal) (129, 203). Whilst this simplifies the modelling process to a certain extent, many areas of human performance involve significant discontinuities in system input (e.g. a triathlete who may change sports, and change work rate frequently within a sport). This also presents certain challenges in light of the aforementioned functional discontinuities in physiological response (i.e. the exercise intensity domains). However, it remains important to study a system under conditions most similar to the way it is used.

Several authors have studied the pulmonary $\dot{V}O_2$ signal during intermittent exercise in a way that may inform the present work. Astrand et al. (11) examined ergometer exercise, utilizing equal work and recovery durations of 0.5, 1, 2, or 3 minutes. It was noted that short work intervals (1 minute or less) appeared to amount to a submaximal physiological stress. However, longer intervals resulted in considerable lactate accumulation and a higher oxygen requirement, and feelings of extreme exertion on the part of the subject (11). The authors reported some surprise at discovering the low lactate values elicited by the short work intervals, and proposed an interesting explanation: that myoglobin at the level of the muscle served as a sort of buffer, liberating enough oxygen to cover the short work requirement aerobically (11). Astrand et al. furthered this hypothesis in a follow up work (12), also concluding that both the length of the *work and recovery period* were of considerable importance in determining lactate accumulation (though emphasis was placed on the work period), depending upon the experimental condition. The authors calculated that approximately 0.43 L of oxygen must have been available at the level of the working muscle at the time an interval began (11), which would essentially cover the entire cost of the work in the case of short intervals. However, the authors also had some difficulty when attempting to calculate the amount of oxygen bound to myohemoglobin in their experimental subject. They noted an extant deficit of approximately 50%(11). Similar conclusions were reached by Christensen et al. (64), who used a wider variety of work and recovery durations.

The advent of muscle biopsy techniques permitted a more mechanistic understanding of the physiology of intermittent exercise. Essén (81) examined 15 s work and recovery intervals over a period of 60 minutes using a cycle ergometer. The work intensity was fixed at an intensity that elicited $\dot{V}O_{2MAX}$ (notably, without reference to the precise protocol used to determine $\dot{V}O_{2MAX}$); group mean work interval power output was 299 W (81). This was compared to CWR exercise at a (group mean) power output of 157W in order to ensure that the two conditions resulted in the same mean power output over the course of an hour long test session (81). Essén reported that the $\dot{V}O_2$ response was similar between the two experimental conditions (approximately 50-60% of $\dot{V}O_{2MAX}$) (81). Of interest, while plasma lactate rose in a similar fashion in the initial part of both conditions, it remained elevated in the intermittent condition whilst exhibiting a steady decline in the continuous condition. Analysis of biopsy specimens indicated greater glycogen utilisation in the continuous work condition, and greater oxidative metabolism and increased lipid contribution in the intermittent condition (81).

Interpretation of the above classical studies with reference to the modern understanding of CP, $\dot{V}O_2$ kinetics and the intensity domains can be challenging. However, contemporary work has attempted modern kinetic analysis of intermittent exercise. Founded in part on the work of Astrand and Essén, Turner et al. (219) examined oxygen uptake during intermittent cycling exercise at 120% of peak ramp power during the "work" interval, and 20 W during the "recovery" interval. Work and recovery durations were varied such that the recovery duration was always double that of the work duration (10 s : 20 s, 30 s : 60 s, 60 s : 120 s, and 90 s : 180 s respectively). They reported an association between blood lactate profiles and pulmonary $\dot{V}O_2$ kinetics; with 10 s : 20 s yielding a 'moderate' response, and 30 s : 60 s yielding a 'heavy' response (i.e. a $\dot{V}O_{2sc}$ emerged that seemed to stabilise). The 60 s : 120 s and 90 s 180 s conditions both resembled a 'severe' response (i.e. a $\dot{V}O_{2sc}$ emerged that did not appear to stabilise). The authors proposed that (219):

> *"…the association of $\dot{V}O_2$ kinetics and blood lactate accumulation profiles may provide a functionally rigorous classification of intermittent exercise intensity, as in the case for constant load exercise."*

Whilst it may be tempting to accede to this conceptual framework, there are several shortcomings in Turner et al. (219) that preclude direct application to the work in this thesis. First, a subsequent study involving one of the authors (Ward; (84)) has demonstrated that kinetics of lactate clearance are considerably slower than those of $\dot{V}O_2$ recovery ($t_{½}$ = 1366 ± 799 s vs. 74 ± 2 s, respectively) or those of the W′ (234 ± 32 s).

Given these kinetic differences, there may exist combinations of work and / or recovery intensity, duration and time that significantly dissociate $\dot{V}O_2$ and plasma lactate. Measures of plasma lactate may lack sufficient resolution to inform the rigorous modelling process undertaken in the subsequent chapters of this thesis.

There are other more methodological concerns raised by Turner et al. (219). For example, the work intervals were normalized to peak ramp power output (i.e. a multiple of power at $\dot{V}O_{2PEAK}$) rather than the CP (219), an approach which at least one of the authors (Ward; (242)) previously (and correctly) argued against, stating that exercise intensity should be defined by a common physiological profile (242). Moreover, work and recovery duration were not manipulated independently. This complicates interpretation of the results, as work and recovery duration have independent effects (12).

Finally, although the subjects in Turner et al. (219) executed work intervals at a power output likely falling in the severe domain, no attempt was made to account for the CP or the work executed above CP (i.e. Figure 1.0). Given the importance of the power-duration relationship in describing exercise in the severe domain, such an approach could yield valuable information. This necessitates the utilisation of a mathematically rigorous analysis of exercise tolerance. One of the best models in this regard is the CP model, which serves as a foundation of the present work.

2.1 Foundations of the Critical Power model

The intersection of biology and performance

Although the CP model was primarily devised as a mathematical tool to understand human performance, it soon became clear that the parameters of the model might have bona-fide physiological interpretations (85, 133, 134, 173, 185). Somewhat separately, certain mathematical modifications have been made to the CP model to account for observations with respect to maximal power output or performance during intermittent exercise. Alternative approaches have also been developed to identify the model parameters. These topics will be reviewed in the following sections.

Definition and history

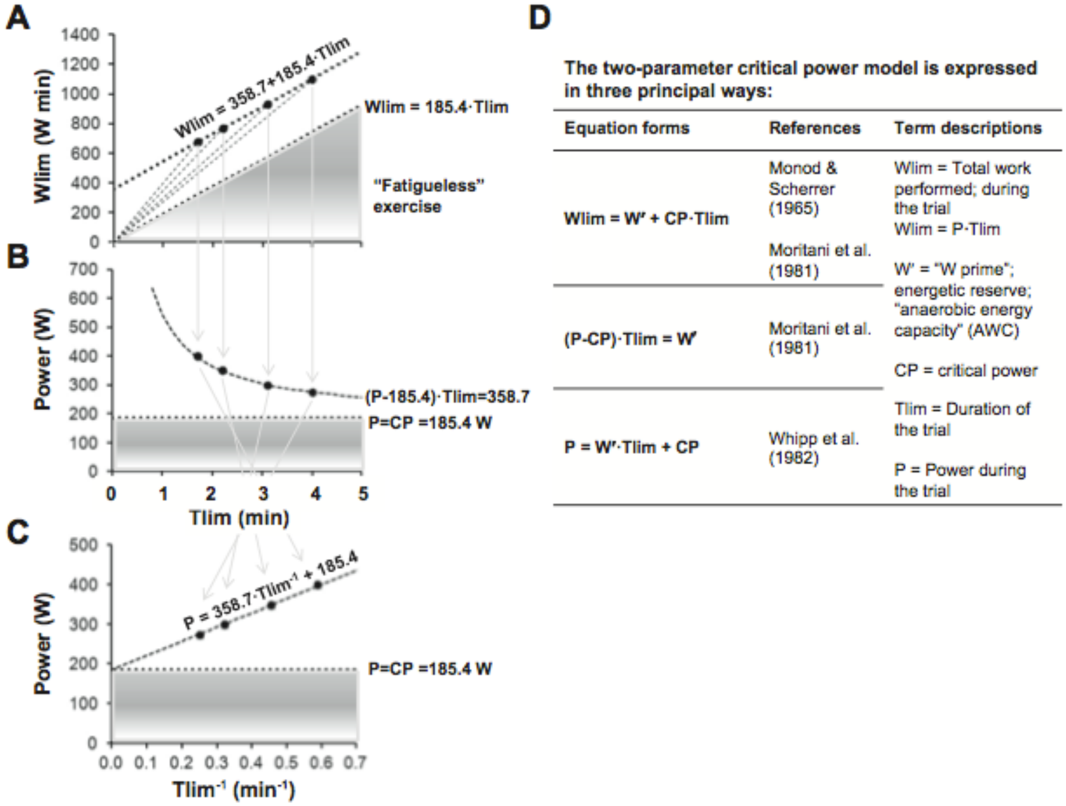
The CP model describes the capacity of an individual to sustain particular work rates as a function of time. In this way, the model summarizes the relationship between exercise intensity and duration for an individual. The historical context of the CP model has been reviewed in detail elsewhere (33, 130, 167). Briefly, a hyperbolic relationship between work rate and time was first suggested by Hill in 1925 (108), who plotted velocity vs. time for world records in swimming and running over various distances. Monod and Scherrer observed a similar hyperbolic relationship in their studies of work rate vs. sustainable duration in skeletal muscle for synergistic small muscle mass exercise, codifying this relationship mathematically in 1965 (164). They also defined the term “critical power” as the power that can be sustained without fatigue for a “very long time”. In the early 1980s, Moritani et al. and Whipp et al. extended this concept to whole-body exercise by having human subjects exercise to exhaustion at different work rates on a cycle ergometer (165, 239). Whereas Moritani used the formalism of Monod and Scherrer, Whipp et al. fit a linearized two-parameter CP model to their data (239). Since those initial studies, the CP model has been applied in a variety of settings and to diverse types of subjects to evaluate muscular performance (45, 130). In particular, the model has been applied to several sports in addition to cycling including running (115), swimming (230) and rowing (137).

Equation derivation & assumptions

Monod and Scherrer devised the CP model by combining the equation for power (power = work/time) with the observed linear relationship between the amount of work done and the duration of tests to exhaustion performed at different work rates (164). The model features two parameters, CP and W′, which are related according to the following equation:

$$W' = (P - CP)t \qquad \textbf{Eq. 2.0}$$

where P is the power and t is the duration for which that power was sustained (239). Note that for sports such as swimming or running, P and CP can be expressed as speed (S) and critical speed (CS), respectively, and the W′ expressed as distance (D′) rather than energy. Figure 1.0 presents the three algebraic forms of the 2-parameter critical power model.



The two-parameter critical power model is expressed in three principal ways:

Equation forms	References	Term descriptions
Wlim = W' + CP·Tlim	Monod & Scherrer (1965) Moritani et al. (1981)	Wlim = Total work performed; during the trial Wlim = P·Tlim
(P-CP)·Tlim = W'	Moritani et al. (1981)	W' = "W prime"; energetic reserve; "anaerobic energy capacity" (AWC) CP = critical power
P = W'·Tlim + CP	Whipp et al. (1982)	Tlim = Duration of the trial P = Power during the trial

Fig. 2.3: *Definitions and descriptions of the three principal forms of the two-parameter critical power model. The data are those of a representative subject ("M.P.") from the Moritani et al. (165) study. The units of energy are expressed as Watts · minute in keeping with the convention used by Moritani et al. (165), but W= is usually expressed in units of joules. The grey-shaded regions on each plot indicate work rates less than critical power (CP), which implies that they would not cause exhaustion ("fatigueless exercise"). A: the linear relationship between the total mechanical work done (W_{lim}) by synergistic muscle groups during constant power trials and the time limit of tolerance of those trials (T_{lim}). The slopes of the dashed lines between the origin and the data points are equal to the mean power of the trials. B: the hyperbolic form of the CP model, which is derived from the first equation by substituting power (P) and T_{lim} for W_{lim}. C: the*

linearized form of the CP model, which is derived from the hyperbolic form by solving for P. Data reprinted from (65), with permission.

Morton has succinctly catalogued the explicit and implicit assumptions of the CP model (167). The four principal assumptions are as follows: 1) Power output is a function of two energy sources, termed aerobic and anaerobic (164, 165); 2) Aerobic energy is unlimited in capacity (i.e., one could exercise at an intensity at or below CP for infinite duration) but is limited in the rate at which it can be converted into power (94, 165); 3) anaerobic energy is unlimited in rate of conversion (i.e., maximal power output or speed is infinite) but is limited in capacity (185, 239); and 4) exhaustion occurs when W′ is fully depleted (167). Each of these assumptions is physiologically imprecise but the model is nevertheless useful for modelling the power-duration relationship for maximal exercise lasting from approximately 2 to ~20-40 min, i.e. within the severe domain of exercise intensity (92, 119, 120, 156, 170, 185, 239). These and other assumptions are further are discussed in the following subsection on limitations.

2.2 Physiological basis of the parameters

W′ and CP are the empirical parameters in the CP model. CP is the maximal work rate that can *theoretically* be performed for infinite duration and corresponds to the maximal aerobic power sustainable without drawing upon W′ (164, 165, 211). W′, originally but perhaps inaccurately called the anaerobic work capacity (AWC), represents the amount of energy available for work at power outputs above CP (88, 92, 185). During exercise at a power above CP, there is a clear and progressive loss of metabolic homeostasis. As discussed previously, $\dot{V}O_2$ and blood lactate concentration attain steady values in response to exercise below CP whereas exercising above CP leads to the eventual attainment of $\dot{V}O_{2MAX}$ and to inexorable blood lactate accumulation (130, 185). At the level of the muscle, Jones et al. observed steady levels of creatine phosphate (PCr), inorganic phosphate (P_i) and pH through twenty minutes of leg extension exercise at a work rate ~10% below CP (134) (Figure 2.4). In contrast, a work rate 10% above CP resulted in continually decreasing [PCr] and pH and increasing [P_i] until exhaustion was reached at ~14.7 minutes (134) (Figure 2.4). Interestingly, Vanhatalo et al. demonstrated that different work rates above CP resulted in almost identical PCr and pH levels at exhaustion (225).

Thus, CP appears to be a true physiologic “threshold” phenomenon that reflects metabolic disturbance in the working muscle mass, and can reasonably be called an “aerobic” parameter (94, 164, 165, 173). It corresponds to a power output that exists between those corresponding to the GET (analogous with lactate threshold) and

$\dot{V}O_{2MAX}$ (130, 185). Of these three parameters, the CP is most useful for predicting performance in endurance events, such as time trial performance in cycling (38, 210). It may be slightly higher than the power corresponding to the maximal lactate steady state (MLSS) (187, 209, 231), but is also highly correlated to the MLSS (187), which is a predictor of performance for exercise lasting 30-60 min (32, 35). It may be that the MLSS and CP are actually representative of the same basic phenomenon, with the difference between them due to difficulty in measuring the MLSS or precise test protocol used. However, CP is more accessibly estimated than MLSS because its measurement does not require invasive measurements and is easily executed. Importantly, the CP has been well established as a marker of “aerobic fitness”, responding positively to both endurance and interval training (94, 120, 121, 186, 222), and negatively to resistance training (36).

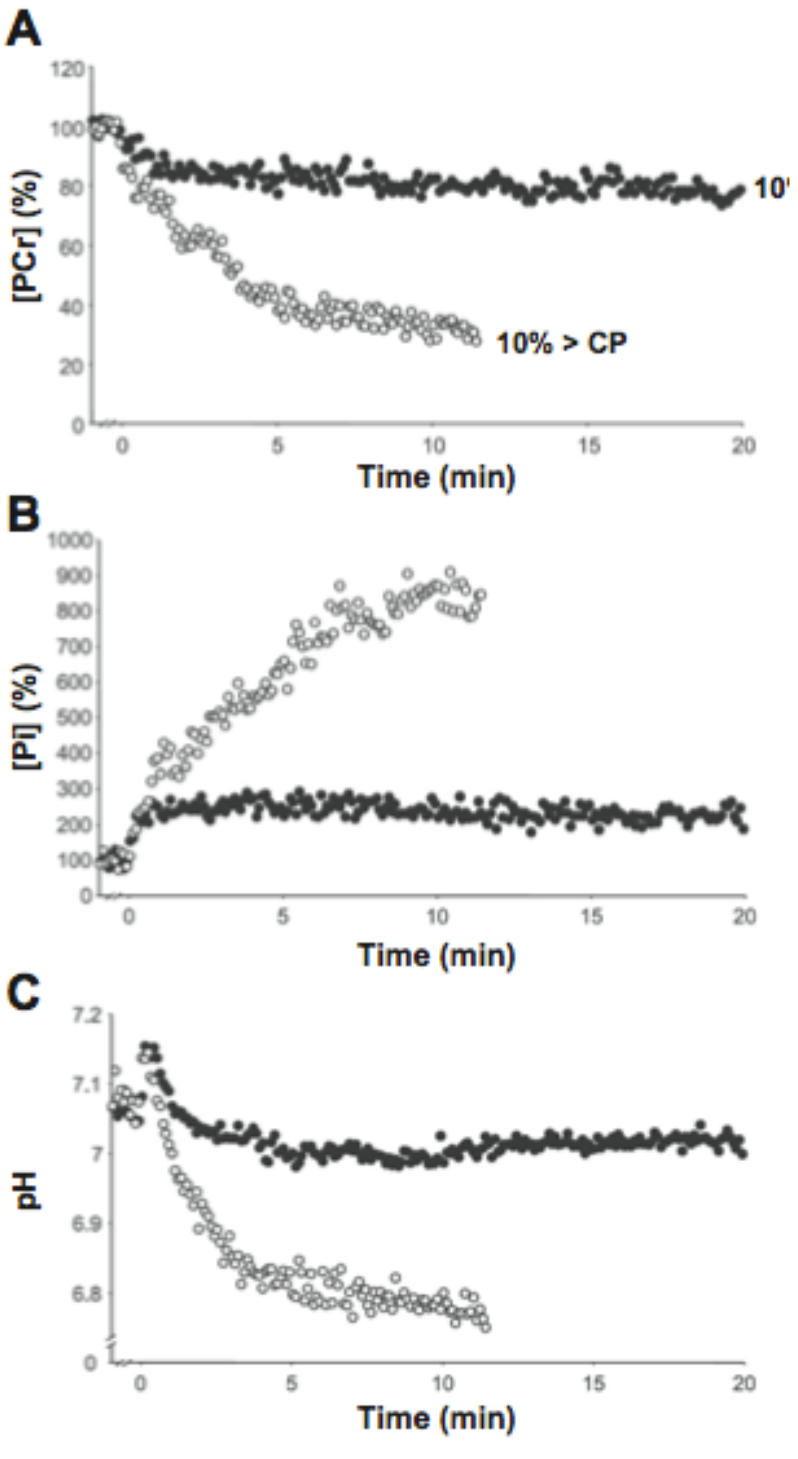


Fig. 2.4: *The physiology of CP. Workloads slightly above CP lead to a loss of metabolic homeostasis, whereas workloads slightly below CP do not. Phosphocreatine (PCr; A), and concentrations* P_i *concentrations (B), and pH (C) concentrations in quadriceps*

muscle were estimated using ^{31}P *magnetic resonance spectroscopy during dynamic exercise above and below CP. Note the shorter duration of the >10% CP trial in which exhaustion was achieved. Data reprinted from (134), with permission.*

The physiological basis of the W′ is less clear. Attempts to specifically characterize the underlying physiological determinants of the W′ have not been wholly satisfactory and it may not be possible to ascribe the W′ to any single physiological variable (130). Indeed, the traditional interpretation of W′ as a fixed "anaerobic work capacity" seems dated in light of work that demonstrated decreased W′ during exposure to hyperoxic gas or with training, and an inverse relationship between CP and W′ (120, 186, 222, 225). However, the picture is confused by data indicating no effect of hypoxia on the W′ (165, 239), though the former study is an abstract and the latter tested the effects of hypoxia on only two subjects. There also exists some work with respect to the W′ and interventions traditionally thought to affect anaerobic exercise performance. The W′ been reported to increase with creatine supplementation (161), and decrease with glycogen depletion (162).

As noted above (and visualised in Figures 2.1 and 2.4), is clear that several physiological variables trend towards what would appear to be lower (e.g. pH, [PCr]) or upper limits ($[P_i]$, $\dot{V}O_2$) as the W′ is depleted (61-63, 130, 134, 185, 225). Other recent work correlated the recovery of the W′ with the "slow" portion of the recovery of $\dot{V}O_2$ (84). In this context, and in light of the sometimes contradictory findings in the paragraph above, it becomes necessarily difficult to assign relative importance to any single physiological variable over any other in terms of 'causing' the exhaustion that is concomitant with the depletion of the W′. However, irrespective of the physiology involved, the W′ is very useful because it represents a robust, performance-related parameter (89, 120, 122, 131, 210). Discharge of the W′ begins when the subject exceeds CP, and is replenished with a $t_{1/2}$ of approximately 3.5 min during passive (e.g. unloaded cycling) recovery (84).

2.3 Peripheral heterogeneity

Human muscles represents fundamentally heterogeneous structures (200). There exist a variety of fibre types (I, IIA, IIX), which differ both biochemically and electrophysiologically (200). For example, the resting membrane potential is more negative in fast than in slow fibres (196, 197). [PCr] depletion is unevenly distributed among fibre types, with type II fibres achieving a deeper depletion than type I fibres (28, 136). This is not necessarily surprising, as the rate of ATP hydrolysis is proportional to power output, which differs based on fibre type (212). Moreover, fibre types and sizes are inhomogenously distributed both within and between muscles (125, 150, 151, 153, 181).

Of particular importance to the present work is evidence that there are differences in perfusion of different fibre types (9, 204), suggesting that particular fibre types and motor units may therefore exist in unique local environments. This has been observed in-vivo in the exercising human via several modalities. For example, Rossiter et al. (192) observed splitting of the P_i peak using ^{31}P-MRS, which could be indicative regions of the muscle that exist in a different pH milieu, although interpretation of this data is not necessarily straightforward as Rossiter’s group utilised a large (12.7 cm) receiving coil that may have captured signal from entirely different muscles. However, other groups have reported similar findings (118). Using positron emission tomography (PET), Mizuno et al. (163) demonstrated flow heterogeneity in human muscle both at rest and during recovery from exhaustive exercise, and that anatomical differences exist, with distal sites showing less perfusion and O_2 consumption than proximal sites. Utilising NIRS, Koga et al. (139) demonstrated regional differences in O_2 consumption and delivery during exercise. Moreover, it has become clear from studies in both animal and human subjects that these heterogeneities are also dependent upon intensity of exercise (67, 68, 140).

Systemically, some of the PET and NIRS observations may follow in part from anatomical considerations. For example, the *rectus femoris* (along with *vastus lateralis*) is primarily perfused by branches of the lateral circumflex branch of the femoral artery (98). In contrast, the *vastus medialis* receives blood from (from proximal to distal) the lateral circumflex artery, perforating branches of the deep femoral artery, and the superior genicular branch of the popliteal artery (98). Understanding system flow is complicated because the flow rates may be centrally limited (i.e. by flow in the femoral artery in the proximal portion of the lower limb), interdependent (i.e. based on steal phenomena between proximal and distal arterial branches of a common trunk), and involve areas of shared 'watershed' (i.e. there exist portions of muscle which receive blood from multiple sources).

Collectively, the above demonstrate that phenomena that may seem superficially well-organised and mathematically uniform (e.g. the pulmonary $\dot{V}O_2$ signal and power-duration relationship) are likely to be based on an inherently complex foundation. From a modelling perspective, we must always keep in mind that we are using mathematical abstractions to understand overall system behaviour, and should avoid the temptation to specifically ascribe too much physiology to specific model parameters.

2.4 Central versus peripheral factors

Whilst much of the present work focuses upon the physiology of the periphery, the behaviour of this periphery is in part the result of central direction (i.e. the brain and spinal cord). During exercise of sufficient intensity, the declining capacity of a muscle or group of muscles to produce force is the result of both peripheral (e.g. the biochemical factors noted previously) and central factors (e.g. reductions of central motor drive, subject motivation) (3). As might be expected, these systems are interdependent. For example, some authors have reported that the process of central fatigue is intimately related to the projection of muscle afferents to the central nervous system (6); (96) (c.f. Figure 20). A blockade of nociceptive feedback to the central nervous system results might be expected to effect alterations in pacing strategy and improve performance (1, 7, 8). Indeed, it is possible to modulate performance by using a variety of analgesics (8, 154). However, afferent feedback to the central nervous system and subsequent modulation of pacing or effort cannot be the sole limiter of endurance exercise performance. For example, it has been reported that spinal reflexes may be directly modulated after prolonged treadmill running, suggesting that parts of the fatigue cascade are not necessarily mediated supraspinally (189).

Amann (6) has proposed an “individual critical threshold”, which may be the result of an increase in firing frequency of Group III / IV afferents, resulting in a limitation of peripheral fatigue development (6, 45, 96). It is worth considering this proposal in light of work from Burnley et al. (45). In this study, subjects were required to perform intermittent (isometric) quadriceps contractions (2 s “on”, 1 s “off”) for up to 1 hour. In the first 5 trials, the subjects executed the “on” segments at an intensity of 35 to 55% of maximal voluntary contraction (MVC) (45). By recording time to task failure (3 successive contractions of 5 nm below target torque), it was possible to construct a critical torque (CT) curve (analogous to the CP curve). In the last 2 trials, subjects executed the “on” segments at 10 or 20% below the CT. In all cases, the subjects performed an MVC with doublet stimulation at the end of each minute (45).

Burnley at el (45) made several crucial observations. Below CT, MVC torque declined little, and after 60 minutes, all subjects save one were still able to execute MVC’s significantly above the target torque. This was coupled with a slight increase in EMG signal. In all trials above CT, MVC and doublet torque fell until they were equal to target torque at task failure. Moreover, the EMG signal at failure was equal to that required to generate an MVC. These data are compatible with Amann et al. (6). Whilst rates of fatigue below CT could not be predicted from rates of fatigue above CT, the data reported by Burnley et al. (45) clearly suggest the presence of central fatigue in both the sub-CT and supra-CT trials (e.g. a reduction in voluntary activation and maximal EMG signal). That is, subjects could not drive the quadriceps as hard at the end of the trials as they could at the beginning.

Importantly, the data of Burnley et al. (45) indicate the presence of both peripheral and central elements in the fatigue cascade and task failure during exercise above CT, and by extension CP. Thus, the power-duration relationship as codified by the CP model implicitly accounts for both peripheral and central fatigue.

2.5 Practical implementation

CP and W′ are traditionally estimated by having the athlete perform a series of constant work rate (CWR) trials to exhaustion, and fitting these data using regression techniques (Figure 2.5A). Several practical issues arise with this approach including the choice of durations and the amount of rest between tests (37, 119, 211). With regard to the latter, if the tests are performed on the same day then sufficient recovery is needed to fully restore W′, which implies a lengthy session because W′ is recharged on the timescale of minutes (84, 85). These issues can be resolved by performing the tests on different days. However, doing so introduces the potential confounder of training or learning effects and it can be cumbersome to perform the tests over multiple days (211). Finally, regardless of the timing of the tests, they should be performed in random order to promote statistical independence between the data points and to eliminate possible confounders introduced by the order of the tests.

To address the shortcomings of the multiple test approach, a 3-minute all-out sprint test has been developed to estimate CP and W′ (Figure 2.5B) (42, 223). In this test, the subject exercises maximally from the start and maintains the effort throughout the test;

there is no pacing. This is a stringent requirement because of the prolonged discomfort involved, and the subject must be highly motivated and should not receive feedback during the test in order to execute it properly. The power output reaches a maximum within a few seconds and then progressively declines as W′ depletes (Figure 2.5B). By ~2.5 min, W′ depletes completely and the power output stabilizes near CP. Therefore, CP is estimated directly as the end-test power, which is calculated as the mean power in the final 30 s of the test, and W′ is estimated by integrating the area bounded by the power profile and a horizontal line at end-test power (Figure 2.5B). The validity of the 3-min all-out test has been supported by high correlations of the CP and W′ estimates from the 3-min test to those independently estimated using the traditional protocol (223). Due to the appeal of estimating CP model parameters in a single test, the 3-min all-out test has attracted considerable interest and has recently been adapted for running (180) and rowing (58).

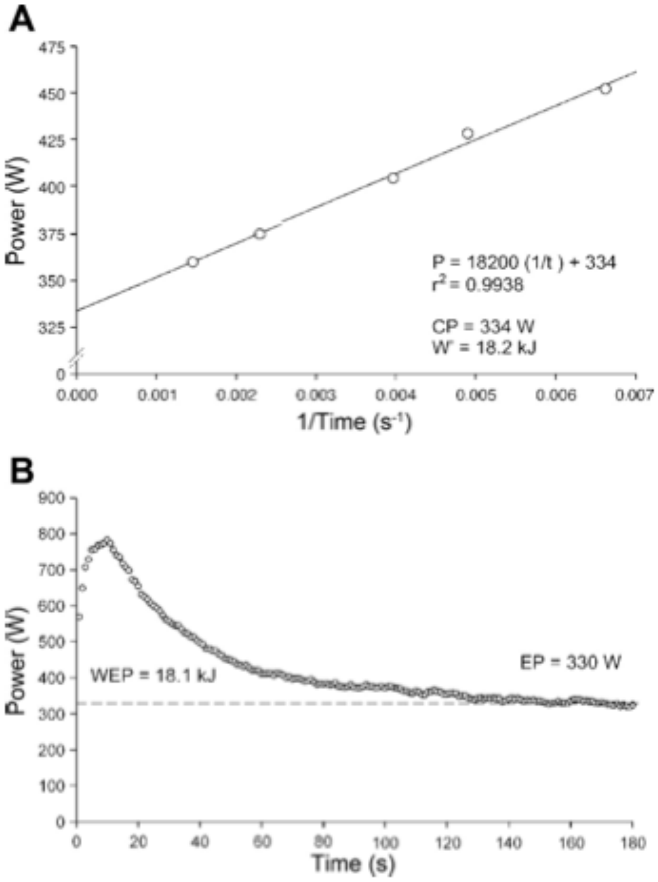


Fig. 2.5: *Fitting the CP model. A: linear regression of power on duration is most commonly done using the linearized form of the CP model, in which the line of best fit is found through the method of least squares. B: CP parameters can be estimated using a 3-min all-out test. The mean power over the final 30 s of the test (the "end-test" power) closely correlates with the CP estimated using the standard protocol. The area bounded by the power-time curve and the horizontal line defined by the end-test power is equal to W'. EP, end-test power; WEP, work done above EP. Data reprinted from (223), with permission.*

Whilst the concept of the 3-min all-out test is appealing, some problems in its practical application have been reported (30, 155). For example, McClave et al. (155) reported that the 3-min all-out test may significantly overestimate CP in elite cyclists. However, it is important to look at the precise experimental conditions of the studies conducted. McClave et al. (155) conducted the test on a RacerMate ergometer dependent upon the interface between a bicycle tire and a roller, not the Lode ergometer used in the original studies (42, 223), and did not conduct a constant ramp exercise test for the determination of the GET, as was done in the original studies (0.5 $W \cdot sec^{-1}$) (42, 223). Finally, 'validation' was carried out by having the subjects ride at the CP as determined by their 3-min all-out test (155). This is statistically indefensible as any measurement has an associated error. A more robust study design would have validated some percentage above and below the estimated CP (c.f. Burnley et al. (42)) Similarly, Bergstrom et al. (29, 30) reported that the 3-min all-out test overestimated CP. Although the 3-minute all-out test has been reported to be sensitive to the manipulation of cadence (224), Bergstrom et al. (29, 30) fixed pedal cadence at 70 (rather than at the cyclists preferred cadence), and also conducted the required incremental exercise test with 2 minutes stages, rather than a constant ramp protocol (30).

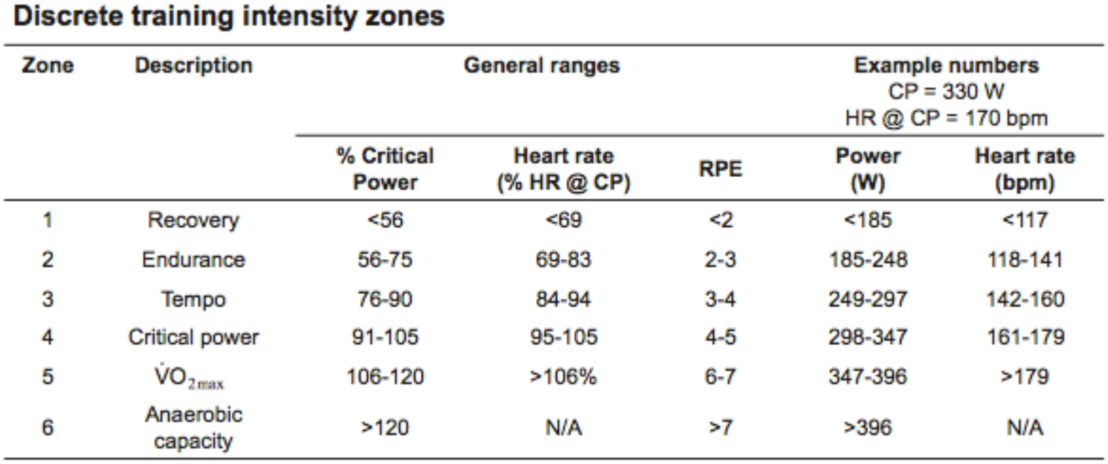
Collectively, the apparent differences between the findings of Burnley et al. and Vanhatalo et al. (42, 223) and those of McClave et al. (155) and Bergstrom et al. (29, 30) may, in part, underscore the importance of executing an experimental protocol exactly, rather than a problem with the 3-min all-out test (or any particular subject type) *per se*. It is noteworthy that other investigators (126), who applied the protocol of Burnley et al. and Vanhatalo et al. (42, 223) more strictly, found that the 3-min all-out test yielded reliable estimates of the CP. This said, it would be unwise to wholly discount the findings of McClave et al. (155). The present author is also co-author of a forthcoming manuscript detailing some problems with the 3-min all-out test in elite track cyclists. Part of this may be due to the difficulty in defeating the inherent pacing strategies elite athletes have developed over the course of many years of practice.

2.6 Conceptual benefits & practical applications

The CP model provides a physiologically sound language to express several of the qualitative sensations and observations of coaches and athletes. First, often athletes will speak of “blowing up” or “dying” when exhaustion was reached. This sensation may be more accurately stated as the depletion of the W′. Second, an observation that can be explained by the CP model is the variable abilities of athletes to excel at shorter duration events or to “go all day”, with the former likely exhibiting high W′ relative to their CP and the latter vice versa. Finally, as previously noted, athletes and coaches often refer to the nebulous “threshold” to describe the dividing line between intensities that can be sustained for a long time versus those that cannot. Physiologically, this dividing line is associated with the CP or MLSS. However, the term “threshold” is imprecise and is often confused with lactate threshold or with anaerobic threshold. Lactate threshold may be defined as the intensity of exercise eliciting a 1 mM increase in blood lactate above resting levels (70) and is less than the intensity corresponding to MLSS or onset of blood lactate accumulation (OBLA, 4 mmol / L). Use of the term anaerobic threshold can be somewhat confusing for athletes and coaches because a variety of energy systems contribute to supplying energy for exercise as intensity increases (e.g. although glycogenolysis rises with increasing work rate during CWR exercise, ‘aerobic’ energy production also continues). Indeed, the (now classic) exhaustive discussion between Brooks and Davis (40, 74) on the semantics, biochemistry, and physiology associated with this topic demonstrates that the term ‘anaerobic’ is to a certain extent loaded, even among expert physiologists. While the MLSS terminology is accurate, thinking in terms of MLSS encourages the erroneous notion that fatigue is caused by lactic acid when in fact lactate is merely a by-product of the biochemical mechanisms responsible for energy supply during exercise. In contrast, CP is a bona fide physiological threshold, full depletion of W′ corresponds to exhaustion and does not invoke lactate as a causal mechanism in fatigue. Therefore, CP should be the preferred terminology over thresholds that are defined solely on the basis of the blood lactate concentration.

The CP model serves as a tool for devising optimal pacing and tactical strategies in athletic competition. With regards to pacing, theoretically optimal strategies have been proposed using the CP model (16, 130) that could inform sports such as swimming or kayaking. The CP model could also inform running race tactics. One could estimate the CS and D′ values of his or her competitors from recent results and use these numbers to suggest the best tactical approach for any particular athlete. For instance, a 10k runner with a superior CS would be well advised to take the lead early, forcing his or her competitors to expend their limited D′ in pursuit. Likewise, another athlete with a high D′ but relatively limited CS would be advised to get to the front and attempt to dictate a slower pace, preserving his or her superior D′ for a finishing sprint (89, 131).

Table 2.0: *Discrete training intensity zones defined as the percentage of CP, RPE or HR as commonly used for coaching purposes (205). These zones facilitate communication between the athlete and coach with respect to workout expectations. The example numbers on the right were calculated from the subject's CP from Fig. 2.2. The heart rate [HR; in beats/min (bpm)] at CP was assumed.* $\dot{V}O_{2MAX}$*, maximal* O_2 *consumption; RPE, rating of perceived exertion; N/A, not applicable. Reprinted from (65), with permission.*



Discrete training intensity zones

Zone	Description	General ranges			Example numbers CP = 330 W HR @ CP = 170 bpm	
		% Critical Power	Heart rate (% HR @ CP)	RPE	Power (W)	Heart rate (bpm)
1	Recovery	<56	<69	<2	<185	<117
2	Endurance	56-75	69-83	2-3	185-248	118-141
3	Tempo	76-90	84-94	3-4	249-297	142-160
4	Critical power	91-105	95-105	4-5	298-347	161-179
5	$\dot{V}O_{2\max}$	106-120	>106%	6-7	347-396	>179
6	Anaerobic capacity	>120	N/A	>7	>396	N/A

The CP model provides a basis for prescribing individualized workout intensities during training (130). Workout intensities are commonly subdivided into discrete zones corresponding to different physiological events or states (Table 2.0) (205). Furthermore, a coach constructing a severe-intensity interval workout could use the CP model for intermittent exercise (which is described in the subsection below on modifications to the CP model) to determine the interval durations and work and rest intensities that would result in depleted W′ at the end of the session, thus optimizing the quality of the workout.

2.7 Limitations

As stated above, the CP model relies on four principal assumptions that contravene known physiology. Here I will address the inaccuracies of each assumption in the same order that they were presented above:

1) Three energy-producing pathways contribute to power output, namely high-energy phosphate, glycolysis and oxidative phosphorylation (167). Thus, parsing the energetics of the power-duration curve into singular 'aerobic' and 'anaerobic' terms is necessarily an oversimplification. It is conceivable that a more detailed model could be developed. However, there is a substantial risk of over-parameterization given that athletes are unlikely to be willing to submit to many more test sessions (i.e. testing is stressful, and interferes with regular training sessions).
2) Power continues to decline below the asymptote defined by CP given enough time, i.e. CP cannot truly represent a "fatigueless task". As noted previously, the applicability of the CP model extends to exercise lasting from about 2 min to 20 or 40 min in most people (92, 167, 185), but up to 60 min in some individuals (113).
3) Power output using W′ is finite because mechanical and physiological limits exist to how fast or powerfully one can sprint (167). In other words, as t_{lim} approaches zero, the two-parameter CP model predicts power outputs that are unreasonably large. This is perhaps best visualized in plots of 3-min all-out test data (Figure 2.5) (222, 223).
4) W′ need not be completely depleted at exhaustion (167). In constant-power trials, the subject ceases exercise when he or she cannot maintain the required power output. However, W′ may not be fully depleted because if the stipulated power output was

reduced to a level still above CP but less than the original power, exercise can continue, at least for a short time (45, 63, 66). Therefore, the maximal power output is a function of the remaining W′.

Despite these limitations, the two-parameter CP model is remarkably robust when applied to exercise within the severe domain (61, 92, 186, 226, 239), and is attractive due to both its relative mathematical simplicity and the accuracy with which it may define the lower limit of the severe domain (112, 134, 185, 186). From a practical perspective (see *General Methods*) the two-parameter CP model has seen wide acceptance by coaches and athletes (5, 205), making it an important tool in the translation of laboratory science to practical implementation. For these reasons, the two-parameter model was selected as the basis of the present work.

2.8 Modifications to the CP model

The three-parameter model

To address the limitations stemming from the assumptions of the two-parameter CP model, Morton created a three-parameter CP model (166). The three-parameter model addresses the assumptions that maximal power output is infinite and that exhaustion occurs when W′ is depleted. Morton's modification was to relax the requirement of the two-parameter model that an asymptote exist at t = 0, which caused P to unrealistically approach infinity as t approaches 0 (166). His modification is expressed mathematically as follows:

$$t = \frac{W'}{(P - CP)} + k \text{ , } (k < 0) \qquad \textbf{Eq. 2.1}$$

where k is the asymptote and assumes a negative value. Because the maximal power possible (P_{max}) can only occur for instantaneous time (i.e., time to exhaustion = 0), it implies that:

$$t = \frac{W'}{(P - CP)} + \frac{W'}{(CP - P_{\max})} \qquad \textbf{Eq. 2.2}$$

Morton further assumed that the maximal achievable power output during a bout of exercise depends on the amount of remaining W′. Through additional reasoning and mathematics, he recovered the above equation except that the interpretation of P_{max} changed to be the "maximal instantaneous power" and was shown to be a linear function of the remaining W′ (166). Therefore, with this form of the CP model, the assumption that W′ is fully depleted at exhaustion is changed to the more realistic assumption that exhaustion occurs when P_{max} is less than the desired power output. This has been demonstrated to be physiologically plausible in the setting of whole body exercise and single leg extension exercise (63, 66).

Whilst the 3-parameter model attempts to address one physiological extreme (i.e. above the limit of the severe domain), it is important to realize that it does not affect the opposite extreme. The 3-paramter model still exhibits an asymptote, meaning that there should exist a power output that may be sustained indefinitely.

The CP model as applied to intermittent exercise

Morton and Billat (168) extended the two-parameter CP model to intermittent exercise, which is valuable as many modes of human activity require periods of physical exertion interspersed with periods of relative rest or recovery. For the first time, there existed the possibility of making comparisons between model behaviour and the temporal characteristics of physiological markers (i.e. $\dot{V}O_2$) during variable work rate exercise (60). The model is stated mathematically as follows:

$$t = n(t_w + t_r) + \frac{W' - n[(P_w - CP)t_w - (CP - P_r)t_r]}{P_w - CP} \qquad \textbf{Eq 2.3}$$

where t = total endurance time, n = number of intervals, t_w and t_r are the durations of the work and recovery phases in each interval, respectively, and P_w and P_r are the power outputs during the work and rest phases, respectively (168). Note that proper behaviour of the model requires the following constraints (168):

$$0 \le P_r < CP < P_w < CP + {}^{W'}\!/_{t} \qquad \textbf{Eq. 2.4}$$

Importantly, this model assumes that the W′ is depleted at a rate of $\frac{P_w - CP}{s}$, and recovered at a rate of $\frac{CP - P_r}{s}$.

The intermittent CP model was an important innovation in practical athlete training. Whilst the standard CP model is useful for the prediction of continuous exercise performance in the severe domain, athletes generally execute training above CP or CV as a series of intervals with defined parameters for work rate, as well as work and recovery durations (73, 199, 201). The intermittent model permits the calculation of the maximum number of repetitions an athlete is capable of given the session ‘prescription’. Thus, customized workouts may be devised based upon the particular fitness and physiology of the athlete. However, this model does have some important shortcomings, which limit its utility. These will be discussed in the following section.

2.9 Conceptual framework

To fully appreciate the implications of a quantitative system, it can be helpful to conceptualize the relevant mathematics in terms of everyday macroscopic phenomena. The present state of understanding of the W′ and CP may be reimagined in terms of a tub or tank (Figure 2.6). In this example, the W′ is represented by a vessel that can be emptied by a drain of variable size (i.e., depending on how far above CP exercise occurs) and refilled by a tap with an adjustable flow (where the maximum flow rate is representative of CP). The level of water in the vessel at any time is the amount of the W′ available for use.

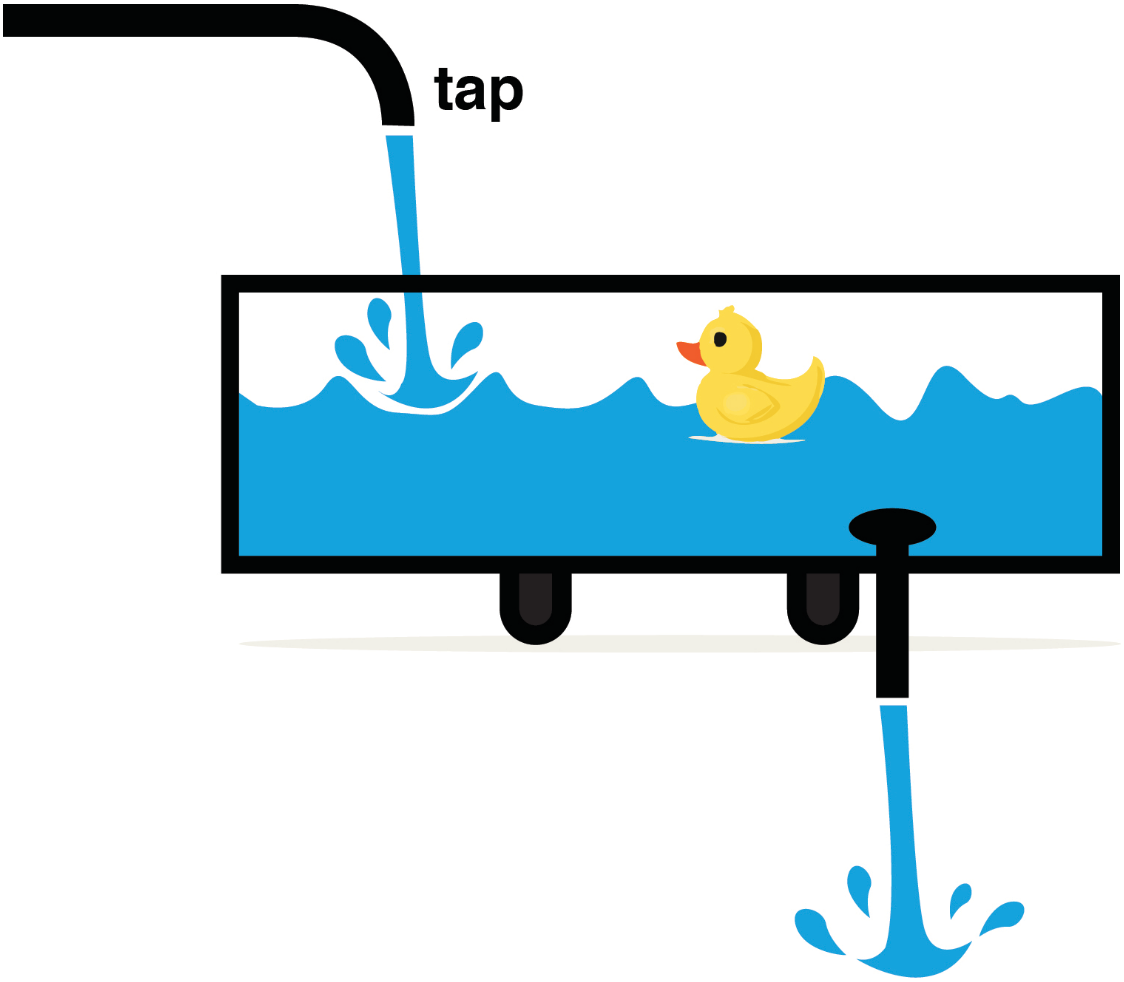


Figure 2.6: *Conceptualization of the CP model. The volume of water in the tub as measured by the height of the rubber duck is indicative of the W' available for use. It is emptied by a drain of variable size (depending upon how hard the athlete exercises), and is refilled by a tap of variable rate, limited by CP. A subject becomes exhausted when the tub is emptied. This schema is not dissimilar to Morton's hydraulic model (167), with the exception that the tap rate in the Morton model is fixed at CP.*

This conceptual system has several important mathematical properties that are worthy of discussion. Firstly, when the drain rate exceeds the maximum tap rate, the volume in the tub begins to decrease (Figure 2.7A). The rate of decrease is constant and linear with respect to time. Second, when the tap rate exceeds the drain rate, the volume in the tub begins to rise (Figure 2.7B), and the change in volume is linear with respect to time. Finally, a curvilinear relationship between the differences between the tap rate and drain rate and the predicted time constant of refill would be expected (Figure 2.7C). That is, a very large difference between the tap rate and drain rate should result in a fast time constant, which slows curvilinearly as the difference approaches zero.

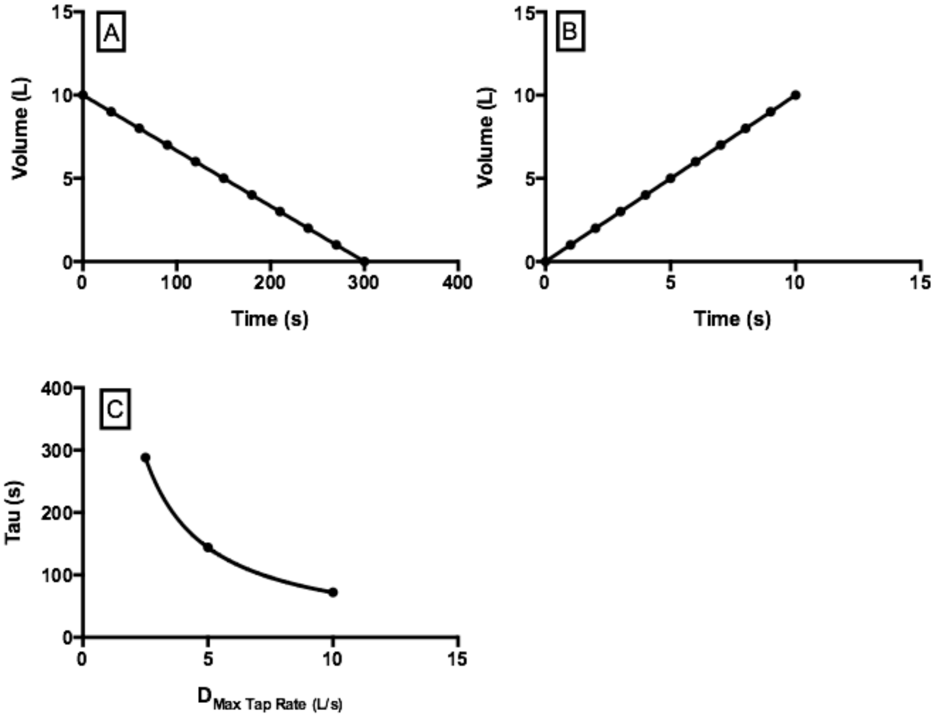


Figure 2.7: *Graphical depiction of expected W' behaviour given the constraints of the tub model. Panel A = volume in tub when drain rate is faster than fill rate, Panel B = volume when fill rate is faster than drain rate. Panel C: Kinetic relationship between difference in tap rate and drain rate and expected time constant.*

The Morton and Billat (168) formulation for intermittent exercise was initially developed to analyse running data, in particular interval workouts. It has also been recently applied in cycling ergometry (60). However, whilst the intermittent model makes assumptions which are mathematically plausible (i.e. linear discharge and recovery of the W′), and which are reasonable in the context of the tank analogy above, recent results indicate that this may be an oversimplification of a more complex system. For example, Ferguson et al. (84) have recently reported that the W′ may recover in a curvilinear fashion, with an interpolated time constant of 336 s. This is of particular concern to athletes and their advisors. Without an accurate estimation of the recovery rate, it becomes impossible to accurately calculate the amount of W′ remaining at any point in a workout or race simulation. The assumption of linear W′ recovery kinetics may therefore represent a significant shortcoming in the intermittent CP model. Moreover, whilst pace or power during running or ergometer workouts can be easily dictated (i.e. the ergometer or treadmill can be pre-programmed), athletes who train in the field do not have this luxury. For example, power output on a bicycle is sensitive to wind direction and drafting, road grade, and traffic conditions.

It would be advantageous to formulate a continuous mathematical function that could evaluate the W′ “on the fly”. Such a construct would facilitate athlete training and permit coaching staff to provide more accurate analysis and advice. From a scientific perspective, given the way in which the 2-parameter CP model has been used to interrogate muscle physiology, it is possible that a kinetically-correct model could be very useful as a tool to further probe the physiological determinants of the W′ in a variety of intermittent exercise modes.

2.9.1 Aims

The primary purpose of this thesis was the development of a novel model of the W′ for intermittent exercise using integral calculus. Such a model would conform to recently reported kinetic behaviour (84), and would accurately predict exhaustion during intermittent exercise under a wide variety of circumstances. As physiological phenomena often exhibit highly characteristic courses of onset and decay (e.g. the fundamental and slow components of $\dot{V}O_2$ kinetics (128, 129, 177, 236)), a secondary goal was to evaluate the model's utility in investigating the control processes underlying the W′ through comparison to observed physiological phenomena.

Specific aims

1) The development of a novel, continuous integrating model of the W′ which takes into account the observed kinetics of the W′ (84), and which can be compared to physiological markers known to correlate with the W′ (e.g. the slow component of $\dot{V}O_2$ kinetics).
2) The interrogation of this model with respect to variability in work and recovery duration, and the correlation of changes in model behaviour with changes in $\dot{V}O_2$.
3) The practical application of the model to athlete pacing during stochastic exercise in training and in competition.

4) The extension of the model to different modes of exercise, such that it could be used in different sports, and the characterization of the model with respect to muscle metabolic responses using ^{31}P MRS and ^{1}H MRS.

Hypotheses Tested:

1) Study 1 (Chapter 4)
    a. That it is possible to estimate the balance of W′ remaining during intermittent exercise by integrating the amount of W′ expended, which recovers exponentially when the power output falls below CP;
    b. That the rate of recovery of the W′ during intermittent exercise should be curvilinearly related to the difference between recovery power and CP;
    c. That the depletion of the W′ during intermittent exercise should correlate with the rise in $\dot{\mathrm{V}}\mathrm{O}_2$ (most likely representative of the $\dot{\mathrm{V}}\mathrm{O}_{2\mathrm{SC}}$) noted during intermittent exercise in the severe domain;
2) Study 2 (Chapter 5)
    a. That the model should be robust to variations in work and recovery duration;
    b. That amount of W′ remaining after a period of intermittent exercise should correlated with the difference between the $\dot{\mathrm{V}}\mathrm{O}_2$ at that time and $\dot{\mathrm{V}}\mathrm{O}_{2\mathrm{MAX}}$;
3) Study 3 (Chapter 6)
    a. That the model should be able to accurately predict complete depletion of the W′ and concomitant exhaustion during stochastic exercise.
4) Study 4 (Chapter 7)

a. That the model should be transferrable to small muscle mass exercise;
b. That recovery of the W′ should correlate with the recovery of intramuscular [PCr], pH or [P_i] as assessed by ^{31}P-MRS and possibly carnosine concentration as assessed by ^{1}H-MRS;

Chapter 3: General Methods

3.0 General experimental procedures

All exercise tests were conducted in a climate-controlled exercise physiology laboratory at sea level. All experimental procedures were approved by the University of Exeter Research Ethics Committee prior to any subject recruitment or data collection.

Subjects:

All of the subjects were members of the university community. The subjects were recreational athletes, but were not highly trained, and were in good health. In general, the subjects were already familiar with laboratory exercise testing procedures, owing to a background in sports science and frequent participation in laboratory experiments. Subjects were instructed to arrive at the laboratory in a rested and fully hydrated state, at least 3 h postprandial. They were also asked to avoid strenuous exercise in the 24 h preceding each testing session and to refrain from caffeine and alcohol for 3 h before each test. Subjects were always tested at approximately the same time of day to avoid diurnal variation in performance.

Informed Consent:

After the experimental procedures, associated risks, and potential benefits of the study protocol had been explained to the subjects both verbally and in writing, they were required to give their written informed consent to participate. The subjects were assured that their anonymity would be preserved, but that their data would be published and possibly presented at scientific conferences. Subjects were also informed that they were free to withdraw from an experimental study at any time and for any reason or for none at all, without any disadvantage.

Health and Safety:

To ensure the health and wellbeing of all study participants, testing procedures and laboratory conditions strictly conformed to the health and safety guidelines established by the University of Exeter Department of Sport and Health Sciences. All work surfaces were disinfected with dilute Virkon disinfectant before and after each subject was tested. All respiratory apparatus was disinfected according to the manufacturer's guidelines.

3.1 Testing and measurement procedures

Descriptive data

Easy study participants height and mass were measured and recorded along with gender and age prior to study commencement. In experiments employing cycle ergometry, the peak power output, $\dot{V}O_{2max}$, and GET were determined at the outset of the respective study.

Cycle ergometry

All laboratory-based cycling was performed on an electronically braked cycle ergometer (Lode Excalibur Sport, Groningen, The Netherlands). Saddle and handlebar heights and fore / aft position were adjusted to maximise the comfort of the subject, and were recorded such that these dimensions could be precisely replicated in all subsequent testing.

The ergometer has the ability to apply a variety of work rate forcing functions. For the purposes of this thesis, three modes were used:

1) Step mode: Permits near-instantaneous changes in work rate (1,000 $W \cdot s^{-1}$) in a stepwise manner, i.e. from one constant work rate to another. The work rate is independent of cadence. This function was used for all intermittent exercise tests.
2) Ramp mode: Permits a constant (i.e. linear) increase in work rate at a predetermined ramp-rate for a predetermined duration. The work rate remains independent of cadence from 25 to 180 RPM. This function was used for all ramp-exercise protocols.
3) Linear mode: Imposes work rate based upon subject cadence according to the equation.

$$Linear\ factor = \frac{Power\ output}{Cadence^2} \qquad \textbf{Eq. 3.0}$$

This mode was used for the 3-min all-out exercise testing.

The ergometer was regularly calibrated and serviced in accordance with the manufacturer's recommendations. Dynamic calibration was carried out several times annually utilizing the Lode Calibrator 2000 provided by the manufacturer.

Ramp testing

In studies 1 and 2, the first visit to the laboratory involved an incremental ramp exercise test to the limit of tolerance. These tests required 2 minutes of pedalling at 20 W, followed by the imposition of a power ramp rate of 0.5 $W \cdot sec^{-1}$. Subjects were instructed to hold a self-selected cadence. The test was terminated when pedal cadence fell by 5 RPM despite vigorous verbal encouragement. The peak power achieved was recorded. At the conclusion of the test, the subject was permitted a "cool-down" period at 20 W at a self-selected cadence until they wished to get off the ergometer. This was less than 3 minutes in all cases. Subjects were monitored for several minutes afterwards in a seated or supine position until they felt ready to leave the laboratory.

3-minute all-out test for CP and W'

Subjects began with 3 min of cycling at 20 W, followed by a 3 min all-out effort (223). 10 s prior to the conclusion of the 3 min period, the subject was encouraged to increase their cadence to approximately 110 to 120 rpm. At the conclusion of the 3 min period, the ergometer was switched to linear mode. Equation 3.0 was used to set the linear factor (i.e. the 'gearing') of the ergometer such that the subject would attain a power output halfway between the power at GET and peak ramp test power upon reaching their preferred cadence. Subjects were instructed to sprint as quickly as possible whilst remaining on the saddle, and were continuously and vigorously encouraged to maintain a cadence as high as possible through the 3-min all-out effort. In order to prevent pacing, the subjects were not provided with any verbal or visual feedback regarding how much time had elapsed or how much time they had remaining. At the conclusion of the test, the subject was permitted a "cool-down" period at 20 W at a self-selected cadence until they wished to get off the ergometer. This was less than 3 minutes in all cases. Subjects were monitored for several minutes afterwards in a seated position until they felt ready to leave the laboratory. Ergometer data was downloaded in Excel format. The CP was estimated as the mean power for the final 30 s of the all-out test, and the W′ as the power-time integral above the EP during the all-out test.

Single-legged knee-extension ergometry

The ergometer was constructed in house, and has been described in detail elsewhere (44, 134). The apparatus consists of a nylon frame, which fits onto the bed as a series of arches placed over the subject's lower extremities. A base unit is placed at the end of the bed. The subject's right foot was connected to a rope that runs over the top of the frame to the base unit. Pulleys on the base unit allow the rope to be attached to nonmagnetic weights. The amount of weight can be varied to set the desired load. The pulley system was also attached to a small shaft encoder (type BDK-06, Baumer Electronics, Swindon, UK), which allowed the collection of a computerized record of the distance the weights were lifted. The subjects lifted and lowered the weight over a distance of approximately 0.22 m.

During each trial, the subjects were required to maintain a rhythm of 40 extensions per minute in time with visual and / or audio cues. During testing outside the MRI machine, the subject was cued audibly. Whilst in the MRI machine, subjects were cued both audibly and visually via a video display. Subjects were given strong vocal encouragement throughout the assigned task. Work was calculated by the Newtonian equation $m \times g \times h$; where m = mass, $g = 9.81$ m/s^2, and h = the displacement of the mass lifted by the subject.

Exercise tolerance

In the case of cycle ergometry, exhaustion was defined as the inability to maintain self-selected cadence (defined as a drop of ≥5 rpm) for greater than 5 s despite strong vocal encouragement. In the case of single leg extension ergometry, exhaustion was defined as the inability to maintain synchronization with audio-visual cues for ≥5 s or inability to complete a full leg extension, despite strong vocal encouragement, whichever came first.

Pulmonary gas exchange and data processing

During all sessions involving cycle ergometry, pulmonary gas exchange was measured breath-by-breath with continuous sampling via capillary line, utilising a commercially available metabolic cart system (Jaeger Oxycon Pro, Hoechberg, Germany). Gasses were analysed via the supplied differential paramagnetic O_2 sensor and infrared absorption CO_2 sensor. The analysers were calibrated prior to each test with gases of known concentration (4% CO_2, 16% O_2, 80% N_2). The supplied impeller turbine assembly (Jaeger Triple V) was fitted with a volume transducer, and was calibrated before each test using a 3L syringe (Hans Rudolph, MO) using several rates within the physiological range (i.e. between approximately 20 and 50 strokes per minute). Delay between the flow and concentration sensors was automatically accounted for by the CPU of the Oxycon Pro. Sensor calibrations were repeated after each test to check for drift, and to prepare the apparatus for the following test, as other subjects were often conducted sequentially.

Subjects wore a nose clip and breathed through a low dead space (90 mL), low resistance (0.75 mmHg.L^{-1}.s^{-1} at 15 L.s^{-1}) mouthpiece, which was supported by adjustable wire headgear to maximise comfort. $\dot{V}O_2$, $\dot{V}CO_2$ and minute ventilation were calculated using standard formulae (27) and were displayed on screen. Subjects were oriented such that they were unable to view the screen whilst being tested. Following each test, the data files were exported in text format for later analysis.

^{31}P Magnetic resonance spectroscopy

^{31}P-MRS was performed in the University of Exeter Magnetic Resonance Research Centre (Exeter, UK) with a 1.5-T superconducting MR scanner (Intera, Philips). Participants were positioned within the scanner, head and torso first, and in a prone position. This resulted in the distal portion of the lower extremity protruding from the bore of the magnet. A 6 cm ^{31}P transmit/receive surface coil was placed within the scanner bed and positioned such that the subjects' right *rectus femoris* muscle was centred directly over it.

Survey images were initially acquired to determine that the muscle was positioned correctly relative to the coil. Several preacquisition steps were then carried out to optimize the signal from the muscle under investigation. An automatic shimming protocol was undertaken using the proton signal of muscle water. This was done within a volume that defined the quadriceps, in order to optimize the homogeneity of the local magnetic field, thereby leading to maximum signal collection. The volume was slightly different between subjects owing to different leg size between individuals. Tuning and matching of the coil were subsequently performed to maximize energy transfer between the coil and the muscle.

To ensure that scanning took place at the same point of muscle contraction, thereby ensuring the muscle was at a consistent distance from the coil at the time of data sampling, the subject was audibly cued via an audible tone. The subject was also visually cued via a display consisting of two vertical bars, one that moved at a constant rate with a frequency of 0.67 Hz and one that monitored foot movement via a sensor within the pulley to which they were connected. The subject endeavoured to match the movements of these two bars.

Before exercise, during exercise, and during recovery, data were acquired every 12 s, with a spectral width of 1500 Hz. The subsequent spectra were quantified via peak fitting, with the assumption of prior knowledge, using the jMRUI (version 2) software package and the AMARES fitting algorithm (221). Spectra were fitted with the assumption that P_i, PCr, α-ATP (2 peaks, amplitude ratio 1:1), γ-ATP (2 peaks, amplitude ratio 1:1), β-ATP

(3 peaks, amplitude ratio 1:2:1), and phosphodiester peaks were present. In all cases, relative amplitudes were corrected for partial saturation due to the repetition time relative to the longitudinal relaxation time (T1). Intracellular pH was calculated using the chemical shift of the P_i spectral peak relative to the PCr peak (217).

^{1}H Magnetic resonance spectroscopy

^{1}H spectroscopy was undertaken with a 4 element, wrap around coil. A voxel was selected in the right *rectus femoris* at approximately mid-thigh (at the same location as ^{31}P had been undertaken) of dimensions 20x30x50 mm, at which location point-resolved spectroscopy (PRESS) was undertaken. 96 measures were averaged, with a repetition time (TR) = 2000 ms, echo time (TE) = 31 ms, 1024 data points and spectral bandwidth = 1200 Hz. Water and carnosine peak areas were calculated within jMRUI (V4) software. Carnosine values were expressed as peak size relative to the water peak having taken into account respective T1 and T2 (transverse relaxation) times.

3.2 Modelling procedures

$\dot{V}O_2$

The breath-by-breath $\dot{V}O_2$ data collected during exercise testing were reviewed to exclude errant breaths resulting from sighing, coughing or swallowing. Values lying >4 SD from the local mean were removed. The remaining data were subsequently linearly interpolated to provide second-by-second values. The specific uses and analysis of this data in Studies 3 and 4 is detailed in the respective chapters

$\dot{V}O_{2MAX}$, $\dot{V}O_{2PEAK}$, *and Gas Exchange Threshold (GET)*

$\dot{V}O_{2MAX}$ was determined by inspection of the filtered and interpolated breath-by-breath $\dot{V}O_2$ data in order to ascertain the presence of a plateau phenomenon, i.e. a lack of meaningful rise in pulmonary $\dot{V}O_2$ despite a continued increase in imposed work rate (57, 114, 160, 218). It was recorded as the average $\dot{V}O_2$ during the final 30 s of exercise (57). In general, the recorded $\dot{V}O_{2MAX}$ values were in good agreement with the $\dot{V}O_{2PEAK}$ recorded in the subsequent 3-min all-out test.

$\dot{V}O_{2PEAK}$ was defined as the highest 30-s moving average value calculated from the filtered and interpolated breath-by-breath $\dot{V}O_2$ data without reference to the presence or absence of a plateau phenomenon.

The GET was determined by averaging the breath-by-breath $\dot{V}O_2$ data from the incremental ramp cycling tests into 10-s bins. GET was estimated as the first disproportionate increase in $\dot{V}CO_2$ as determined by v-slope analysis of individual plots of $\dot{V}CO_2$ vs. $\dot{V}O_2$ (26), as this was the method originally used by Burnley et al. (42) and Vanhatalo et al. (223) to set up the 3-min all-out test.

PCr

The [PCr] recovery time constant $\tau_{[PCr]}$was determined by fitting a single exponential function to the [PCr] recovery (Graphpad Prism, Graphpad Software, San Diego, California, USA).

$$[PCR] = [PCR]_{EE} + (1 - [PCR]_{EE}) \cdot (1 - e^{-K \cdot t}) \qquad \textbf{Eq. 3.0}$$

where concentrations are expressed as a fraction of resting, $[PCR]_{EE}$ is end-exercise [PCr], *K* is the rate constant and *t* is time in seconds.

Power-Duration

It has been noted that the various possible algebraic formulations of the 2-parameter CP model are mathematically, but not necessarily statistically identical (92). However, both Poole et al. (186) and Gaesser et al. (92) have noted that when linear correlation coefficients are high, parameter estimates are virtually identical. Thus, for the purposes of this thesis, the decision was made to use the linearized work-time formulation of the CP model first published by Monod and Scherrer (164) in studies 3 and 4:

$$Work = CP(T_{lim}) + W' \qquad \textbf{Eq. 3.1}$$

A secondary concern was practical in nature. Common coaching practice (5, 205) often involves calculating the CP and W′ by plotting work expended against time for different tests and then calculating a linear regression line using Microsoft Excel. As one of the aims of this thesis was the creation of a practical modelling tool for coaches and athletes in the field, it is pedagogically helpful to use a formulation already familiar to the population.

W'_{BAL} modelling by integration

In Chapter 4, the general form of a novel equation for calculating the balance of W′ remaining at any time during an intermittent exercise session (the W'_{BAL} model) is introduced.

$$W'_{BAL} = W' - \int_0^t W'_{EXP} \cdot e^{\frac{-(t-u)}{\tau_{W'}}} \cdot du \qquad \textbf{Eq. 3.2}$$

Where W' equals the subject's known W′ as calculated from the 2-parameter CP model, W'_{EXP} is equal to the expended W′, (*t-u*) is equal to the time in seconds between segments of the exercise session that resulted in a depletion of W′, and $\tau_{W'}$ is the time constant of the reconstitution of the W′. In other words, the amount of W′ remaining at any time *t* is equal to the difference between the known W′ and the total sum of the joules of the W′ expended before time *t* in the exercise session, each joule of which is being recharged exponentially.

Of note, the W'_{BAL} model assumes a first-order kinetic relationship with respect to the recovery of the W′. This is not meant to imply *certainty* with respect to these kinetics, as there is almost no data available on the mathematics that may govern the process of W′ recovery. Having only 3 data points, Ferguson et al. avoided curve fitting entirely, though the data appears curvilinear in nature (see Chapter 2)(84). The present model assumes the simplest possible exponential mathematics, as a more complex model would present problems of parameterisation, and in any case would be difficult to justify as the model is fit to just one point: the time at which the subject reaches exhaustion.

With respect to the calculation of the W'_{BAL} model, it is important to carefully consider the behaviour of the integral, which is not necessarily intuitive. The process of integration takes into account the sum of the entirety of the data being processed. The exponential term and associated time constant dictate that there is always some “recovery” of the W′ going on, even when there is a net *depletion* of the W′ observed. For example, let us assume that a subject with a CP of 200 W decides to exercise at 235 W for a single second. The subject has thus expended 35 J of W′. Let us assume he then carries on for an additional second. Our intuition tells us that he will have now expended a total sum of 70 J of W′; 35 J for each second. The integral suggests something different; that the subject began “recovering” some tiny fraction of the W′ in the time between the first and second seconds. That is, at the end of second two, the sum of W′ expended is 35 J plus the *remainder* of the 35 J expended in the first second (assuming $\tau_{W'}$ = 380s, 34.9 J), for a running balance of 69.9 J. After 60 s, the total W′ expended would be approximately 2.0 kJ, rather than 2.1 kJ. The W′ is *depleting* with each second. However, it is not depleting quite as quickly as might be *expected*. During recovery, the integral behaves precisely as we expect an exponential recovery to behave. That is, the extant sum begins recovering according to the specified $\tau_{W'}$ at the moment the power falls below CP. Given the standard errors typically associated with the determination of the W′ are often an order of magnitude more, we may consider this a computational ‘quirk’ of the model. However, there may exist some physiological importance to this particular model behaviour (see *General Discussion*, Chapter 8).

The equation was implemented in a spreadsheet in Microsoft Excel, and was iterated on a second-by-second basis. If the subject was exercising at a power output less than CP, a zero was entered into the equation for that second (i.e. no joules of W′ were expended). If the subject was exercising above CP, the number entered that second was equal to the difference between the CP and the power output (i.e. the number of joules of W′ expended that second).

Statistical methods

All statistical analyses were carried out using SPSS (SPSS ver. 20, IBM Corporation, Armonk, NY) or GraphPad Prism (Graphpad Prism, Graphpad Software, San Diego, California, USA). Specific statistical tests and software are discussed in the individual chapters in which they were used. Statistical significance was accepted at the $P < 0.05$ level. All data are presented as mean ± SD unless otherwise indicated in the individual experimental chapters.

Chapter 4: Modelling the Expenditure and Reconstitution of Work Capacity Above Critical Power

Modeling the Expenditure and Reconstitution of Work Capacity above Critical Power

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ABSTRACT

SKIBA, P. F., W. CHIDNOK, A. VANHATALO, and A. M. JONES. Modeling the Expenditure and Reconstitution of Work Capacity above Critical Power. *Med. Sci. Sports Exerc.*, Vol. 44, No. 8, pp. 1526–1532, 2012. **Purpose:** The critical power (CP) model includes two constants: the CP and the W' [$P = (W' / t) + \mathrm{CP}$]. The W' is the finite work capacity available above CP. Power output above CP results in depletion of the W' complete depletion of the W' results in exhaustion. Monitoring the W' may be valuable to athletes during training and competition. Our purpose was to develop a function describing the dynamic state of the W' during intermittent exercise. **Methods:** After determination of $\dot{V}O_{2max}$, CP, and W', seven subjects completed four separate exercise tests on a cycle ergometer on different days. Each protocol comprised a set of intervals: 60 s at a severe power output, followed by 30-s recovery at a lower prescribed power output. The intervals were repeated until exhaustion. These data were entered into a continuous equation predicting balance of W' remaining, assuming exponential reconstitution of the W'. The time constant was varied by an iterative process until the remaining modeled $W' = 0$ at the point of exhaustion. **Results:** The time constants of W' recharge were negatively correlated with the difference between sub-CP recovery power and CP. The relationship was best fit by an exponential ($r^2 = 0.77$). The model-predicted W' balance correlated with the temporal course of the rise in $\dot{V}O_2$ ($r^2 = 0.82$–0.96). The model accurately predicted exhaustion of the W' in a competitive cyclist during a road race. **Conclusions:** We have developed a function to track the dynamic state of the W' during intermittent exercise. This may have important implications for the planning and real-time monitoring of athletic performance.

Key Words: CRITICAL POWER, W', MODELING, PERFORMANCE PREDICTION

The curvilinear relationship between work rate and performance time for a sporting event involving whole-body exercise was first noted by Hill (18) in 1925. However, a formal mathematical framework for the fatigue of synergistic muscle groups was not developed until Monod and Scherrer (30) presented the critical power (CP) model in 1965. The equation is hyperbolic and consists of two parameters: CP and the W' (22):

$$P = (W'/t) + \mathrm{CP} \qquad [1]$$

In this model, P is equal to power output, and t is equal to time to exhaustion at that power output. For sports such as swimming or running, P and CP can be substituted with speed and critical speed (CS), respectively, and the W' can be substituted with distance.

The CP represents an asymptote, a power output that could *theoretically* be maintained indefinitely on the basis of principally "aerobic" metabolism. The CP is unlimited in capacity but limited in rate (22). It is an important predictor of endurance exercise performance (34). In contrast, the W' represents a finite work capacity (J) available to the athlete once he or she attempts a power output above CP, i.e., it is theoretically unlimited in rate but limited in capacity (22). The CP equation also implies that the W' (power–time integral > CP) remains constant regardless of the rate of its discharge.

The W' is of substantial importance to athletic performance because complete depletion of the W' results in the inability to perform supra-CP exercise (for review, see Jones et al. [22] and Vanhatalo et al. [37]). Knowledge of both the CP and W' can assist in performance optimization (14,22), particularly in situations where the athlete is required to make surges in power output above CP. However, it has been difficult to apply the power–duration relationship in real time because there has traditionally been no way of tracking power output and thus W' expenditure during competition. This has changed with the advent of the on-bicycle power meter (e.g., SRM, Colorado Springs, CO; Saris PowerTap, Saris, Madison, WI [3,17]), which could theoretically permit dynamic modeling of W' utilization during an exercise task given an appropriate mathematical framework.

Despite the ease with which the CP and W' can be calculated, the precise physiological determinants of the W' remain uncertain (12,22,38). The CP defines the boundary between the heavy and severe exercise intensity domains

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(7,19,20,22,32,36). Interestingly, the slow component of pulmonary O_2 uptake ($\dot{V}O_2$) in the severe domain has been demonstrated to be related to the W' during both constant work rate and "all-out" exercise (7,13,23,38). Although any intrinsic relationship between the W' and $\dot{V}O_2$ kinetics is at odds with the traditional interpretation of the W' as a fixed "anaerobic" work capacity (29,30), Vanhatalo et al. (36) have recently reported that hyperoxia both reduces the W' and increases the CP during knee extensor exercise. This suggests that the CP and W' are interrelated and that the nature of the W' might need to be reconsidered (7).

The aforementioned studies have only examined "all-out" exercise or constant work rate exercise in the severe domain. However, many endurance athletic competitions require frequent changes in power, with surges above CP and periods of recovery below it. Many studies indicate a correlation between maximal $\dot{V}O_2$ ($\dot{V}O_{2max}$) and the ability to repeat sprint exercise (1,4,11) and between the primary (phase II) time constant of $\dot{V}O_2$ kinetics and the ability to repeat sprint exercise (10). However, despite the probability that the ability to perform and sustain intermittent severe exercise is related to the charge/discharge state of the W', these studies have not addressed a possible dynamic temporal relationship between $\dot{V}O_2$ and the W'.

The primary purpose of the present investigation was to develop a dynamic model that tracks the discharge and recharge of the W' during severe intermittent exercise. We also investigated the possibility of a link between $\dot{V}O_2$ kinetics and the discharge of the W'. Finally, we wished to consider whether such a model might be useful in the analysis of real-world bicycle race power data.

MATHEMATICAL FRAMEWORK

Morton and Billat (31) presented a novel model that permitted the application of the CP model to intermittent exercise:

$$t = n(t_w + t_r) + W' - n[(P_w - \mathrm{CP})t_w - (\mathrm{CP} - P_r)t_r] \,/\, (P_w - \mathrm{CP}) \qquad [2]$$

where t is equal to total endurance time, P_w and P_r are equal to the work and rest interval power, and T_w and T_r are equal to the work and rest interval time. This model was successfully applied to intermittent cycling exercise by Chidnok et al. (8). However, although this model makes assumptions that are mathematically plausible (i.e., linear kinetics of W' discharge and recharge), it has been recently reported that the W' may actually be reconstituted in a *curvilinear* manner, with a calculated $t_{1/2}$ of approximately 234 s (assuming exponential recovery, time constant = 336 s) for cycle ergometer exercise (12). This suggests the possibility of developing a simplified continuous function that would account for the depletion and reconstitution kinetics of the W' during intermittent exercise.

Assuming that 1) the expenditure of the W' begins the moment a subject exceeds CP, 2) the reconstitution of the W' begins the moment the subject falls below CP, and 3) the reconstitution of the W' follows a predictable exponential time course, it is possible to formulate an equation describing the balance of W' remaining at any given time during an exercise session (W'_{bal}) where some amount of W' was expended.

$$W'_{bal} = W' - \int_0^t (W'_{exp})(e^{-(t-u)/\tau_{W'}}) \qquad [3]$$

where W' equals the subject's known W' as calculated from the two-parameter CP model, W'_{exp} is equal to the expended W', $(t - u)$ is equal to the time in seconds between segments of the exercise session that resulted in a depletion of W', and $\tau_{W'}$ is the time constant of the reconstitution of the W'. In other words, the amount of W' remaining at any time t is equal to the difference between the known W' and the total sum of the joules of the W' expended before time t in the exercise session, each joule of which is being recharged exponentially during recovery < CP (Fig. 1).

METHODS

Protocol. Full details of the experimental procedures are given in the companion article (8). Briefly, seven healthy males (mean ± SD: age = 26 ± 5 yr, height = 1.79 ± 0.06 m, body mass = 81 ± 6 kg) volunteered to participate in this study. The subjects were recreational athletes but were not highly trained. They were familiar with laboratory exercise testing procedures, having previously participated in studies using similar procedures in our laboratory. The study was approved by the University of Exeter Research Ethics Committee. After the experimental procedures, associated risks, and potential benefits of the study protocol had been explained to the subjects, they were required to give their written informed consent to participate. Subjects were instructed to arrive at the laboratory in a rested and fully hydrated state and at least 3 h postprandial. They were also asked to avoid strenuous exercise in the 24 h preceding each testing session and were asked to refrain from caffeine and

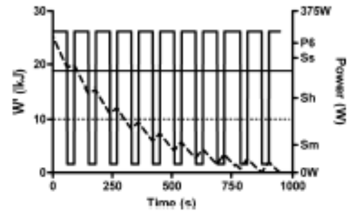

FIGURE 1—Model of W'_{bal} (*dashed line*) and power output (*continuous line*) for a representative subject in the S_{10} trial. The *dotted and horizontal solid lines* indicate the subject's GET and CP, respectively. *Hash marks* on the right axis indicate power outputs for the S_S, S_H, and S_M recovery trials. In this example, the subject exercises in the severe domain for 60 s and recovers at 20 W, repeating the process until exhaustion.

TABLE 1. Physiological data for each subject and group mean ± SD.

Subject	W' (kJ)	CP (W)	$\dot{V}O_{2max}$ (L·min^{-1})
1	28	211	3.93
2	25.4	220	4.14
3	22.3	213	4.21
4	21.6	351	5.59
5	18.2	277	4.25
6	17.6	187	3.19
7	14.3	221	3.39
Mean	21.1	240	4.1
SD	4.7	56	0.778

alcohol for 3 h before each test. All tests were performed at the same time of day (±2 h) at sea level in an air-conditioned laboratory at 20°C. At least 48 h separated each test.

The gas exchange threshold (GET) and $\dot{V}O_{2max}$ were estimated for each subject from data collected on a Lode cycle ergometer (Lode Excalibur Sport, Groningen, The Netherlands) during a standard ramp incremental protocol (30 W·min^{-1}). After an advance familiarization trial, the subject's CP and W' were determined using a 3-min all-out test (35). The CP was determined as the mean power output during the final 30 s of the test, and the W' was estimated as the power–time integral above the CP. In subsequent visits, the subjects performed a constant work rate trial to exhaustion in the severe domain and four intermittent exercise trials to exhaustion. In each case, the intermittent exercise consisted of 60-s work intervals at the power output predicted to result in exhaustion in 6 min (P_6; equation 1) + 50% of the difference between P_6 and the athlete's CP and 30-s recovery intervals at a predetermined intensity (Fig. 1). The recovery intervals were devised as follows:

1. 20 W (S_{20}).
2. Moderate-intensity recovery (S_M) at a power output of 90% of the GET.
3. Heavy-intensity recovery (S_H) at a power output of GET + 50% of the difference between the GET and CP.
4. Severe-intensity recovery (S_S) at a power output equal to P_6 − 50% of the difference between the CP and P_6.

During all tests, pulmonary gas exchange was measured breath by breath (Jaeger Oxycon Pro; Hoechberg, Germany) with subjects wearing a nose clip and breathing through a low dead space (90 mL), low-resistance (0.75 mm Hg·L^{-1}s^{-1} at 15 L·s^{-1}) mouthpiece, and impeller turbine assembly (Jaeger Triple V). The analyzer was calibrated before each test with gases of known concentration, and the turbine volume transducer was calibrated using a 3-L syringe (Hans Rudolph, Shawnee, KS). $\dot{V}O_2$, carbon dioxide output, and minute ventilation were calculated using standard formulae (2).

Analyses. The work/time data from intermittent bouts 1–4 were fit to equation 3 by inputting the number of joules expended above CP each second. The time constant was varied by an iterative process until modeled $W'_{bal} = 0$ at the time of exhaustion. Derived time constants were then plotted against the difference between recovery power and CP (D_{CP}).

The breath-by-breath $\dot{V}O_2$ data collected during each of the work bouts were processed to exclude errant breaths, and values lying >4 SDs from the local mean $\dot{V}O_2$ were removed. These data were then linearly interpolated to provide second-by-second data. $\dot{V}O_{2baseline}$ was defined as the mean $\dot{V}O_2$ measured during the final 90 s of unloaded cycling before the onset of the protocol, whereas the work interval $\dot{V}O_2$ was defined as the mean $\dot{V}O_2$ measured during the entire 60-s work interval. This was plotted against the modeled end W'_{exp} for each corresponding interval. Regression analysis was performed using computer software (GraphPad Prism; GraphPad Software, San Diego, CA). The relationship between $\tau_{W'}$ and CP was assessed by linear regression. The relationship between $\tau_{W'}$ and D_{CP} was assessed by both linear and nonlinear regression. Significance was accepted at the $P < 0.05$ level, and data are reported as mean ± SD.

RESULTS

The W', CP, and $\dot{V}O_{2max}$ for each of the subjects are reported in Table 1.

$\tau_{W'}$ was inversely correlated with CP in the S_M condition ($r^2 = 0.64$, $P = 0.03$). There was a strong trend toward a significant inverse correlation in the S_{20} and S_H conditions ($r^2 = 0.53$, $P = 0.06$ and $r^2 = 0.48$, $P = 0.08$), suggesting a relationship between $\tau_{W'}$ and CP irrespective of sub-CP intensity domain. There was no correlation in the S_S condition ($r^2 = 0.07$, $P = 0.56$).

$\tau_{W'}$ was inversely correlated with D_{CP} in the S_{20}, S_M, and S_H trials ($r^2 = 0.67$, $P < 0.0001$). There was no linear correlation when recovery power exceeded CP (S_S trial) ($r^2 = 0.05$, $P = 0.55$). Above CP, $\tau_{W'}$ increased to nonphysiological values, indicating no net recharge of the W' and merely a slightly lower rate of depletion during the recovery interval. The $\tau_{W'}$-versus-D_{CP} data were best fit by an exponential regression of the form $y = ae^{(-kx)} + b$,

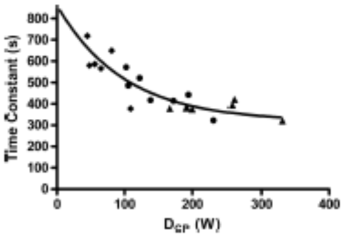

FIGURE 2—Graphical depiction of time constant of W' reconstitution ($\tau_{W'}$) as a function of D_{CP}. Individual recovery levels are represented by a common symbol, where S_{20} = *triangles*, S_M = *circles*, and S_H = *diamonds*. Note that the overall relationship could also be well described by a bilinear fit.

TABLE 2. Calculated time constants of W' repletion ($\tau_{W'}$) for each recovery condition for each subject and group mean ± SD.

	$\tau_{W'}$ (s)			
Subject	S_{20}	S_M	S_H	S_S
1	381	570	719	11,873
2	375	415	566	30,758
3	380	519	586	1319
4	321	321	377	596
5	380	412	649	1395
6	379	484	580	1635
7	421	441	569	1816
Mean	377	452	578	7056
SD	29	81	105	11,169

yielding a close correlation for the S_{20}, S_M, and S_H trials ($r^2 = 0.77$; SE: $a = 86.11$, $k = 0.004$, $b = 61.8$) (Fig. 2). The precise equation as determined by nonlinear regression (GraphPad Prism; GraphPad Software) would be written as follows:

$$\tau_{W'} = 546e^{(-0.01D_{CP})} + 316 \qquad [4]$$

The mean $\tau_{W'}$ for the S_{20} trial was 377 ± 29 s. The majority of the W' repletion time constants clustered near 380 s during the S_{20} trial. There was greater variation in the S_M (452 ± 81 s) and S_H conditions (580 ± 105 s) (Table 2). The S_S condition yielded an average time constant of 7056 ± 11,969 s.

Modeled W' depletion was strongly related to the rise in VO$_2$ above baseline during each successive interval in the severe domain ($r^2 = 0.82$–0.96, $P < 0.0002$–0.0049) (Figs. 3A–C).

Equation 3 was also used to analyze preexisting data collected from a well-trained cyclist participating in a mass start race to determine whether model-predicted depletion of the W' was coincident with athlete exhaustion. $\tau_{W'}$ was estimated using equation 4, using the mean of all power values less than CP (75 W) as the recovery power for calculating D_{CP}. D_{CP} was held constant for the purposes of the simulation. The model demonstrated that the cyclist was forced to reduce power output below CP (227 W) as the calculated W' balance fell below 1.5 kJ (Fig. 4). After executing two major attacks during the race, the athlete was forced to retire after 55.4 min.

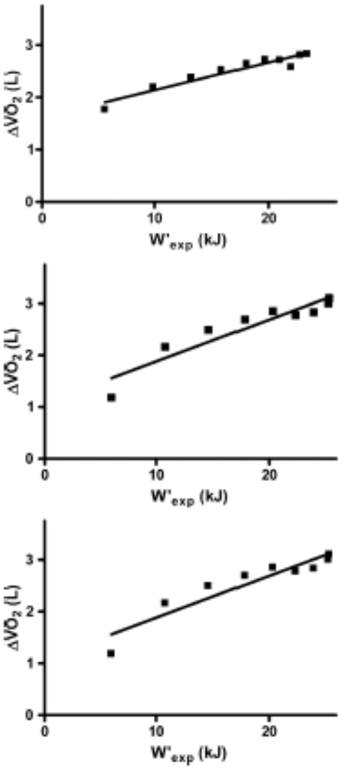

FIGURE 3—Modeled W' expended versus increase in $\dot{V}O_2$ above CP during intermittent exercise for a representative subject (subject 2). ***Top panel*****: 20-W recovery ($r^2 = 0.91$).** ***Middle panel*****: moderate recovery ($r^2 = 0.87$).** ***Bottom panel*****: heavy recovery ($r^2 = 0.88$).**

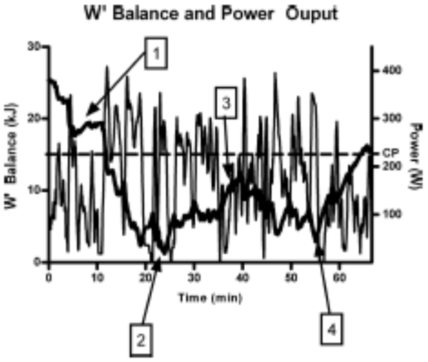

FIGURE 4—Modeled W' expended (*heavy solid line*) and athlete power output (*thin solid line*). The athlete's CP (227 W) is denoted by the *dashed line*. Peak power output was 409 W. *Numbers* indicate important points as race unfolds. 1: athlete establishes position in pack. 2: athlete has attacked but has severely depleted the W', forcing recovery. 3: athlete attacks again. 4: athlete again depletes W' and is forced to withdraw from race as lead pack escapes.

DISCUSSION

This is the first study to mathematically characterize the discharge and reconstitution kinetics of the W' during intermittent exercise over a range of recovery power outputs.

The $\tau_{W'}$ was negatively correlated with D_{CP} and was well fit by an exponential function for all power outputs below CP (Fig. 3). A likely explanation for the inverse correlation between $\tau_{W'}$ and D_{CP} is the presence of a smaller "oxidative reserve" with increasing recovery power output. In other words, the smaller the difference between the $\dot{V}O_2$ required to maintain the recovery power and the VO_2 at CP, the smaller the capacity to reconstitute the W'. As expected, the modeled time constants became unreasonably large when the recovery interval power output exceeded CP, indicating that no recharge of the W' occurred within the 30 s of "recovery" time permitted.

The mean $\tau_{W'}$ for the S_{20} trial is compatible with results presented previously (12). The $\tau_{W'}$ seemed to cluster between 370 and 380 s during the S_{20} trial for most of the subjects, with one $\tau_{W'}$ of 320 s for a subject (subject 4), who had the highest CP of the group. The greater variation in the time constants calculated in the S_M and S_H conditions and the observation that there was only a trend toward correlation between CP and $\tau_{W'}$ in the S_H condition and no correlation in the S_S suggest that the process of W' repletion may become more complex with increasing recovery power. With this in mind, it is interesting to note that the relationship between $\tau_{W'}$ and D_{CP} is better fit by an exponential than a linear regression, and it is also possible that the relationship is bilinear. This suggests that the reconstitution of the W' may be related to different physiological factors with increasing recovery power output. This would help explain the variability of the relationship between CP and $\tau_{W'}$ in the different recovery conditions. Further investigations with respect to the mechanisms underpinning the W' should consider this possibility.

The correlation between the rise in $\dot{V}O_2$ during intermittent exercise and the calculated net discharge of the W' is most interesting, particularly in light of the fact that it is difficult to perform conventional $\dot{V}O_2$ modeling because of the short work and rest durations (Figs. 3A–C). The progressive loss of efficiency noted with increasing repetitions is most likely representative of the $\dot{V}O_2$ slow component (VO_{2sc}) that has been described for constant work rate exercise above GET (21,25,26). In this context, these data lend support to previous findings that link the W' to $\dot{V}O_2$ kinetics. Not only does it seem that the W' is related to the "size" of the severe domain (7,22,36,38), but also, its expenditure seems to correlate well with the *temporal course* of the rise in $\dot{V}O_2$ in the severe domain during intermittent exercise. Because the$\dot{V}O_{2sc}$ has been linked to the recruitment of Type II fibers and the development of fatigue (21), this may suggest the possibility that the W' is related to such recruitment.

It has been suggested that CP may differentiate exercise intensities that are principally limited by the availability of glycogen (below CP) from other mediators of fatigue (34). Indeed, time to exhaustion above CP is correlated with time to attain $\dot{V}O_{2max}$ (19). This may be explained by the observation that discharge of the W' is associated with a depletion of muscle phosphocreatine (PCr) stores (22,24). In turn, this may explain the progressive rise in $\dot{V}O_2$ as the fall in PCr predicts increased stimuli to mitochondrial respiration (33). It has been proposed (24,32,36) that the depletion of the W' may reflect the predictable rate of PCr degradation and/or increase in other metabolites toward some limiting value, which would coincide with exhaustion. However, data indicating a definitive *causative* relationship are lacking.

There is other evidence suggesting a relationship between [PCr] and the W' from biopsy studies (5,6). For example, restoration of [PCr] after a 30-s sprint was highly correlated with recovery of peak power output, 6- and 10-s maximal power output, and maximal pedal speed after 1.5 and 3 min of recovery. This suggests a much shorter time constant than that reported for W' reconstitution. However, a "plateau" in the recovery pattern has also been noted (6). Assuming exponential reconstitution, 30-s sprint power output recovered with a time constant of approximately 333 s (6), close to the 377-s average time constant calculated in this study for the S_{20} condition and closer still to the 336 s extrapolated from a previous investigation (12). It has been suggested that this "plateau" in 30-s sprint power recovery reflects fatigue of the fast-twitch fiber pool because of inherently slower PCr recovery kinetics (6). We may also come to this supposition independently in light of recent work linking the $\dot{V}O_{2sc}$ (and thus the slow component of PCr on-kinetics) with the recruitment of Type II muscle fibers (25–27). In addition, disproportionate perfusion of predominantly glycolytic (fast twitch) muscle regions has been reported when rats were exercised above CS (9). Because the CP construct seems highly conserved among vertebrates (9,15,28) and even arthropods (16), there is reason to believe that similar phenomena and mechanisms might be observed in humans.

Taken together, the above suggests that the W' may be primarily representative of the recruitment of (and relative fatigue state of) a separate "compartment" of the exercising muscle mass, i.e., the Type II fiber pool. The present model can therefore be rewritten with two components (i.e., in a bottom-up approach) to satisfy the following conditions:

1. Depletion of the W' begins the instant the subject exceeds CP, and some portion of the W' comes from two separate compartments that are representative of the Type I and Type II fiber pools, both of which are activated simultaneously.
2. The portions describing compartment I and compartment II have different time constants to reflect different rates of W' repletion during recovery.
3. The absolute contribution to the W' of the two compartments is different, either because of purely energetic considerations or because of the absolute size of the compartment providing the work. In either (or both) case(s), there must be a gain term in addition to the exponential term.

4. The sum of W' expended by I and II at exhaustion must equal the known W'.

In the general case, we would write the new equation as follows:

$$W'_{\mathrm{bal}} = W' - \int_0^t \left(k_1 e^{-(t-u)/\tau_1} + k_2 e^{-(t-u)/\tau_2}\right) w(u)\,du \qquad [5]$$

where k_1 and k_2 are gain terms and τ_1 and τ_2 are the time constants for the two different compartments.

The benefit of using the simpler model tested in this work is that it can be calculated through noninvasive, easily performed tests such as the 3-min all-out test to determine CP and W' and the time constants derived using regression equation 3. If more detailed information is required, the athlete needs only to perform intermittent severe work to exhaustion at several different recovery power outputs such as in the present study to enable the calculation of a personalized regression model. Such a process may be more important in highly trained athletes: we noted an outlier in our data set (subject 4) with a high $\dot{V}O_{2max}$ (>5 L·min^{-1}), whose time constant of W' repletion did not change from the S_{20} to the S_M condition. It is possible that highly aerobically fit individuals differ from recreationally active individuals in terms of W' recovery.

Practical applications. Because this model accounts for both the expenditure and repletion of the W', it permits the possibility of intracompetition performance management. We retrospectively analyzed power meter records from a competitive amateur cyclist who collected the data during a road race (Fig. 4). Using a time constant calculated by the use of equation 4 (440 s), we found that the athlete was forced to reduce power output because of the perception of impending exhaustion any time the calculated W' balance approached 1.5 kJ. This simulation is limited in that the $\tau_{W'}$ will, in fact, vary according to the instantaneous D_{CP}. Despite this limitation, the model performed adequately for the purposes of this simulation. However, future studies should investigate the use of a variable $\tau_{W'}$ to take into account variable power output. The present data do suggest a novel technological application: the equations could be programmed into an on-bike power monitoring device for cycling (e.g., SRM or PowerTap). This would permit the athlete knowledge of the real-time state of the W' and thus help inform important decisions, e.g., the optimal amount of drafting and recovery in advance of a sprint. Similarly, adapting the equation to CS and distance would allow it to be programmed into a wrist-worn GPS or accelerometer devices for use in running races.

CONCLUSIONS

The principal novel finding of this work is the development of a simplified, continuous equation that describes the dynamic state of the W' during intermittent exercise. This model may be of significant practical value to competitive athletes who are interested in managing and optimizing training and racing performances.

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The results of the present study do not constitute endorsement by the American College of Sports Medicine.

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Chapter 5: Effect of Work & Recovery Durations on W′ Reconstitution During Intermittent Exercise

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Effect of Work and Recovery Durations on W' Reconstitution during Intermittent Exercise

PHILIP F. SKIBA[1], SARAH JACKMAN[1], DAVID CLARKE[2], ANNI VANHATALO[1], and ANDREW M. JONES[1]

[1]*Sport and Health Sciences, College of Life and Environmental Sciences, St. Luke's Campus, University of Exeter, Exeter, Devon, UNITED KINGDOM; and* [2]*Department of Biomedical Physiology and Kinesiology, Simon Fraser University, Burnaby, BC, CANADA*

ABSTRACT

SKIBA, P. F., S. JACKMAN, D. CLARKE, A. VANHATALO, and A. M. JONES. Effect of Work and Recovery Durations on W' Reconstitution during Intermittent Exercise. *Med. Sci. Sports Exerc.*, Vol. 46, No. 7, pp. 1433–1440, 2014. **Purpose**: We recently presented an integrating model of the curvature constant of the hyperbolic power–time relationship (W') that permits the calculation of the W' balance (W'_{BAL}) remaining at any time during intermittent exercise. Although a relationship between recovery power and the rate of W' recovery was demonstrated, the effect of the length of work or recovery intervals remains unclear. **Methods**: After determining $\dot{V}O_{2max}$, critical power, and W', 11 subjects completed six separate exercise tests on a cycle ergometer on different days, and in random order. Tests consisted of a period of intermittent severe-intensity exercise until the subject depleted approximately 50% of their predicted W'_{BAL}, followed by a constant work rate (CWR) exercise bout until exhaustion. Work rates were kept constant between trials; however, either work or recovery durations during intermittent exercise were varied. The actual W' measured during the CWR (W'_{ACT}) was compared with the amount of W' predicted to be available by the W'_{BAL} model. **Results**: Although some differences between W'_{BAL} and W'_{ACT} were noted, these amounted to only -1.6 ± 1.1 kJ when averaged across all conditions. The W'_{ACT} was linearly correlated with the difference between $\dot{V}O_2$ at the start of CWR and $\dot{V}O_{2max}$ ($r = 0.79$, $P < 0.01$). **Conclusions**: The W'_{BAL} model provided a generally robust prediction of CWR W'. There may exist a physiological optimum formulation of work and recovery intervals such that baseline $\dot{V}O_2$ can be minimized, leading to an enhancement of subsequent exercise tolerance. These results may have important implications for athletic training and racing. **Key Words:** CRITICAL POWER, W', $\dot{V}O_2$, INTERMITTENT EXERCISE, INTERVAL TRAINING

The critical power (CP) concept has profound implications for the modeling of human performance (14,16). It consists of two parameters: the CP and the W'. The CP represents a power output below which it is possible to maintain steady-state exercise and above which the time to exhaustion becomes highly predictable. The W' represents a finite amount of energy available for work performed in excess of the CP:

$$P = \frac{W'}{T_{\mathrm{lim}}} + \mathrm{CP}, \qquad [1]$$

where T_{lim} is the time to exhaustion at any power (P) in excess of the CP. Although several limitations of this formulation have been noted (14), the model is useful in that it represents a robust mathematical representation of human performance.

The depletion and reconstitution of the W' during exercise is of paramount interest to athletes. For example, the balance of W' remaining (W'_{BAL}) at any point in a race necessarily determines the frequency, duration, and intensity of surges above CP an athlete may make to escape a competitor, or to close a gap (13). Recently, Skiba et al. (22) presented a novel integrating model of the W' that permits the calculation of the W'_{BAL} at any time t during intermittent exercise.

$$W'_{\mathrm{BAL}} = W' - \int_0^t W'_{\mathrm{exp}} e^{-\frac{(t-u)}{\tau_{W'}}} dt. \qquad [2]$$

In this formulation, W'_{exp} is equal to the expended W', $(t - u)$ is equal to the time in seconds between segments of the exercise session that resulted in a depletion of W', and $\tau_{W'}$ is the time constant of the reconstitution of the W'. Thus, the W'_{BAL} at any point during a training session or race is the difference between the known W' and the total W' expended before time t in the exercise session, which is being recharged exponentially when power falls below CP (22).

It is assumed that the W' recovers with an exponential time course during intermittent large muscle mass exercise (equation 2) (11,22). Moreover, $\tau_{W'}$ seems to vary as a curvilinear

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function of the difference (D_{CP}) between recovery P and the CP, suggesting a highly organized underlying control process (22):

$$\tau_{W'} = 546e^{(-0.01D_{CP})} + 316, \qquad [3]$$

where the numerical constants are arbitrary parameters fit to the previously reported data set; in particular, 316 W seems to represent an asymptote beyond which a larger D_{CP} does not further speed recovery. This recovery schema may be conceptualized in several ways. Noodhof et al. (20) recently offered the example of a vessel of water (W'), which may be filled by a tap (aerobic metabolism) and emptied by a drain of variable size (supra-CP work rate). In such a case, the rate of refill would be curvilinearly related to the difference between the fill rate and the drain rate.

A robust model of the depletion and reconstitution of the W' provides an opportunity to test how different performance scenarios may affect W' kinetics. Given the importance of the W' as a performance indicator, this might have valuable real-world applications for athletes. In particular, investigating whether (and how) different work and recovery intervals alter the $\tau_{W'}$ will be important in refining the intermittent model of W' presented previously (22) with consequent practical applications for athletic training and the tactics used by athletes during competition.

Thus far, there have been no attempts to study the effects of intermittent protocols as conditioning or "priming" exercise on subsequent exercise energetics. This question is of considerable importance from a performance perspective, as foot and bicycle races often involve a series of surges in pace before a final, protracted effort in the finale. Indeed, road and track cyclists are often observed to limit the length of a "pull" while leading a group of riders, preferring to divide the work among the group. As there have been some data suggesting a linkage between the W' and the $\dot{V}O_2$ "slow component" ($\dot{V}O_{2SC}$) (22,27), it is possible that there exists a physiologically optimum formulation of intermittent exercise, which may minimize the development of the $\dot{V}O_{2SC}$ and thus permit an increase in time to exhaustion.

The primary purpose of this investigation was to test the robustness of the W'_{BAL} model using a variety of work and recovery durations during intermittent exercise performed before an exhaustive constant work rate (CWR) exercise bout. We hypothesized that the W'_{BAL} model would accurately predict the W' remaining and therefore available for use during this subsequent bout of CWR exercise. We also hypothesized that there would be a positive relationship between the difference between the $\dot{V}O_2$ immediately preceding the start of the CWR bout and the $\dot{V}O_{2peak}$ ($D_{\dot{V}O_2}$) and the W' remaining for CWR exercise.

METHODS

Five healthy males (mean ± SD: age = 27.4 ± 6 yr, height = 1.84 ± 0.08 m, body mass = 85.2 ± 18 kg) and six healthy females (mean ± SD: age = 25.2 ± 1.6 yr, height = 1.67 ± 0.12 m, body mass = 65.3 ± 12.7 kg) volunteered to participate in this study. The subjects were recreational athletes but were not highly trained. All subjects were familiar with laboratory exercise testing procedures. The study was approved by the University of Exeter Research Ethics Committee. After the experimental procedures, associated risks, and potential benefits of the study protocol had been explained to the subjects, they were required to give their written informed consent to participate. Subjects were instructed to arrive at the laboratory in a rested and fully hydrated state, at least 3 h postprandial. They were also asked to avoid strenuous exercise in the 24 h preceding each testing session and to refrain from caffeine and alcohol for 3 h before each test. All tests were performed at the same time of day (±2 h) at sea level in an air-conditioned laboratory at 20°C. At least 48 h separated each test, with the main experiment being completed within a 2-wk period.

All testing was carried out using the same Lode Excalibur Sport (Lode, Groningen NL). The gas exchange threshold and the maximum oxygen uptake ($\dot{V}O_{2max}$) were estimated for each subject from data collected during a standard ramp incremental protocol (30 W·min^{-1}). After an advance familiarization trial, the subject's CP and W' were estimated using a 3-min all-out test as previously described (24). The CP was taken as the mean power output for the final 30 s of the test, and the W' was estimated as the power–time integral above the CP (21).

In each of six subsequent visits, the subjects performed intermittent exercise, followed by a CWR exercise bout until exhaustion (Fig. 1). For ease of comparison with previous work from our laboratory (9,22), the work rates for both the "on" interval of the intermittent exercise and the CWR portion of each trial (P_{EXP}) were calculated as that predicted to result in exhaustion in 6 min (P_6; equation 1) + 50% of the difference between P_6 and the CP. The "off" or recovery

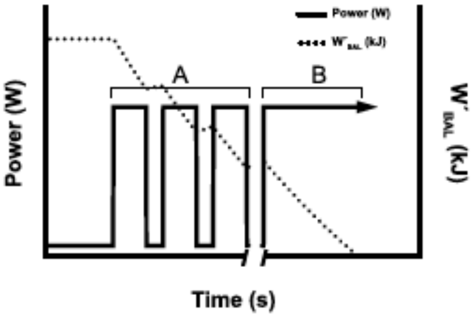

FIGURE 1—Schematic representation of the experimental protocol. The solid line represents power output, whereas the dotted line represents the W'_{BAL}. Each experimental session involved a period of intermittent exercise (A), followed by a period of CWR exercise to exhaustion (B). The length of the work durations (60, 40, or 20 s) or the recovery durations (30, 20, 10, or 5 s) were varied in the different experimental sessions. In each case, we compared the W'_{BAL} predicted to be available at the start of CWR to the W' actually measured during B.

intervals were performed at 20 W in all cases, again in keeping with previous publications (9,22). The ergometer was set for the fastest possible change in load, which is reported by the manufacturer to be nearly instantaneous, and which we observed to be less than 0.5 s during testing.

The pattern of the intermittent work was different in each of the six visits, with the duration of either the work intervals or the recovery intervals being manipulated. In three of the trials, the work interval duration was varied (60, 40, or 20 s) while maintaining a recovery interval of 30 s (trials 60–30, 40–30, and 20–30, respectively). In the other three trials, a work interval duration of 20 s was maintained, followed by a recovery interval duration of 20, 10, or 5 s (trials 20–20, 20–10, and 20–5, respectively). In each case, the intermittent exercise portion of the protocol was designed to result in an approximately 50% depletion of the W' by using equation 3 (22).

The CWR portion of each trial began immediately after the final recovery interval (Fig. 1) and was continued until the subject's cadence fell by more than 5 rpm below the subject's self-selected cadence despite vigorous verbal encouragement ($T_{\lim}$). All subjects completed all trials in a randomized order. After the completion of the main protocol, subjects returned to the laboratory to complete another 3-min all-out test to determine any potential training effect.

$\dot{V}O_2$ data collection and modeling. During all sessions, pulmonary gas exchange was measured breath by breath (Jaeger Oxycon Pro, Hoechberg, Germany) with subjects wearing a nose clip and breathing through a low dead space (90 mL), low resistance (0.75 mm Hg·L^{-1}·s^{-1} at 15 L·s^{-1}) mouthpiece and impeller turbine assembly (Jaeger Triple V). The analyzer was calibrated before each test with gases of known concentration, and the turbine volume transducer was calibrated using a 3-L syringe (Hans Rudolph, Kansas, MO). $\dot{V}O_2$, carbon dioxide output, and minute ventilation were calculated using standard formulae (1).

The breath-by-breath $\dot{V}O_2$ data collected during exercise testing were reviewed to exclude errant breaths resulting from sighing, coughing, or swallowing. Values lying >4 SD from the local mean were removed. The remaining data were subsequently linearly interpolated to provide second-by-second values. $\dot{V}O_{2start}$ was defined as the mean $\dot{V}O_2$ calculated for the 30 s immediately preceding the CWR bout. $\dot{V}O_{2peak}$ was defined as the mean $\dot{V}O_2$ calculated for the final 30 s of exercise in each CWR bout. $D_{\dot{V}O_2}$ was calculated as the difference between $\dot{V}O_{2start}$ and $\dot{V}O_{2peak}$.

Analyses. The power data from all sessions were fit to equation 2. $\tau_{W'}$ was varied by an iterative process until the W'_{BAL} at the time CWR began was equal to the amount of W' actually measured to be available during the CWR portion of the trial (W'_{ACT}). W'_{ACT} was calculated as the sum of the work performed in excess of the CP, assuming a constant CP. W'_{BAL} and W'_{ACT}, as well as the predicted and actual $T_{\lim}$ were compared using repeated-measures ANOVA (SPSS ver. 20; IBM Corporation, Armonk, NY). Repeated-measures ANOVA was also used to compare any differences in $\dot{V}O_{2start}$ between trials. The Pearson product moment correlation coefficient was used to test the relationship between the $D_{\dot{V}O_2}$ and the W'_{ACT}. Paired-samples t-test were used to compare the CP, W', and maximum 30-s $\dot{V}O_2$ measured during the 3-min all-out test before and after the main experiment. Statistical significance was accepted at the $P = 0.05$ level, and data are reported as group mean ± SD.

RESULTS

Variable work interval trials. The group mean $\tau_{W'}$ fell considerably (i.e., the kinetics were faster) as the work interval duration was reduced from 60 to 40 to 20 s (conditions 60–30, 40–30, and 20–30; Table 1 and Fig. 2), indicating that subjects recovered more quickly than predicted as work duration was shortened. This led to a relative underprediction of $T_{\lim}$ and W'_{ACT} (Table 1 and Fig. 3). The difference between W'_{BAL} and W'_{ACT} was not significant in the 60–30 or 40–30 bout but reached significance in the 20–30 bout ($P < 0.01$). The relationship between the work interval duration and the W'_{ACT} underprediction was linear ($r = 0.99$, $P < 0.05$). The comparison of the predicted and measured $T_{\lim}$ yielded a significant difference in both the 40–30 and the 20–30 conditions ($P < 0.01$; Table 1).

Variable recovery interval trials. Decreasing the recovery interval duration from 30 to 20 s resulted in an additional reduction of the $\tau_{W'}$ (Table 1 and Fig. 2), indicating a faster than expected recovery. This resulted in a relative underprediction of W'_{ACT} (Table 1 and Fig. 3). The differences between W'_{BAL} and W'_{ACT} were statistically

TABLE 1. Mean trial values ± SD for predicted and actual W, predicted and actual $T_{\lim}$, calculated $\tau_{W'}$, $\dot{V}O_{2start}$ and $\dot{V}O_{2peak}$.

Trial	W_{pred} (kJ)	W_{act} (kJ)	Diff (kJ)	$T_{\lim\ Pred}$ (s)	$T_{\lim\ Act}$ (s)	Diff (s)	$\tau_{W'}$ (s)	$\dot{V}O_{2start}$ (mL·min^{-1})	$\dot{V}O_{2peak}$ (mL·min^{-1})
20–5	5.28 ± 2.49	6.03 ± 3.03	−0.75 ± 1.7^{d}	84 ± 22	96 ± 37	−12 ± 29^{c}	337 ± 150	3333 ± 828^{c,d}	3493 ± 968
20–10	5.49 ± 2.46	8.27 ± 4.43	−2.78 ± 2.6^{a,e}	92 ± 28	138 ± 59	−46 ± 40^{a,c}	234 ± 119	2910 ± 700^{c,e}	3484 ± 939
20–20	6.96 ± 2.81	9.73 ± 3.88	−2.77 ± 1.9^{a}	112 ± 17	159 ± 39	−46 ± 51^{a}	212 ± 114	2414 ± 567^{e}	3449 ± 925
20–30	7.74 ± 2.99	9.60 ± 4.0	−1.86 ± 1.7^{a,b,e}	125 ± 16	155 ± 29	−29 ± 23^{a,b}	263 ± 91	2229 ± 554^{b,e}	3454 ± 949
40–30	7.79 ± 2.87	8.98 ± 3.67	−1.19 ± 2.02	133 ± 20	145 ± 36	−13 ± 38^{a}	302 ± 145	2524 ± 687^{b}	3437 ± 972
60–30	7.13 ± 2.80	7.35 ± 3.07	−0.22 ± 1.68	121 ± 13	121 ± 32	0 ± 30	403 ± 164	2753 ± 674	3436 ± 945
Mean	6.76 ± 1.13	8.34 ± 1.42	−1.58 ±1.06	114 ±119	148 ±14	27 ±19	274 ± 69	2693 ± 395	3459 ± 17

Key indicates significant differences.
[a] Significantly different from predicted.
[b] Significantly different from trial 60–30.
[c] Significantly different from trial 20–30.
[d] Significantly different from trial 20–10.
[e] Significantly different from trial 20–5.

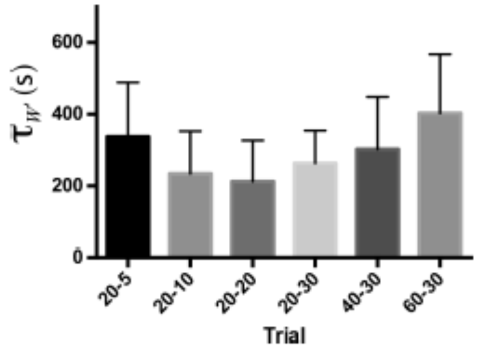

FIGURE 2—Group mean ± SD time constants of W' recovery extrapolated using equation 1 and solved for $W' = 0$ at time of exhaustion. An optimum may exist in the area of the 20–20 trial.

significant in the 20–20 ($P < 0.01$) and 20–10 bouts ($P < 0.01$). There was no significant difference between W'_{BAL} and W'_{ACT} in the 20–5 bout. A similar pattern was noted for the comparison of the predicted and measured T_{lim} (Table 1). These underpredictions were also statistically different from one another in several cases (Table 1).

$\dot{V}O_2$ analysis. Group mean $\dot{V}O_2$ data are presented in Figures 4a–4d. A "sawtooth" pattern for $\dot{V}O_2$ during work and recovery was evident in the 60–30 and 40–30 conditions but was lost in the 20–30 condition, where the trace resembled a slow curvilinear rise in $\dot{V}O_2$. There was a significant difference between the $\dot{V}O_{2start}$ recorded in the 60–30, 40–30, and 20–30 trials ($P < 0.05$), with a trend toward a linear fall in $\dot{V}O_{2start}$ as the work interval was reduced from 60 to 20 s ($r = 0.99$, $P = 0.07$; Fig. 4a). There was no significant difference between trials in the $\dot{V}O_{2peak}$ recorded.

The group mean pattern of the work and recovery interval $\dot{V}O_2$ resembled a slow curvilinear rise in the 20–20, 20–10, and 20–5 trials (Figs. 4c and 4d). There was a significant difference in $\dot{V}O_{2start}$ between the 20–30 and the 20–10 and 20–5 trials ($P < 0.05$) (Table 1). There was also a significant difference between the $\dot{V}O_{2start}$ for the 20–10 and 20–5 trials. The $\dot{V}O_{2start}$ for the 20–5 trial was significantly greater than all of the other trials. Overall, there was a linear increase in $\dot{V}O_{2start}$ as the recovery interval was decreased from 30 to 5 s ($r = 0.99$, $P < 0.01$). There was no significant difference between the $\dot{V}O_{2peak}$ recorded in any of the trials (Table 1). The $D_{\dot{V}O_2}$ was significantly correlated with the W' remaining in the CWR ($r = 0.79$, $P < 0.01$; Fig. 5).

Changes in CP. There was a group mean increase in CP of 18 W ± 20 W and a group mean reduction in the W' of 0.6 ± 0.6 kJ during the study. The change in CP was statistically significant ($P < 0.05$), whereas the change in W' was not. There was an inverse correlation between the change in CP and the change in W' ($r = 0.89$, $P < 0.01$). The group mean peak $\dot{V}O_2$ measured during the 3-min all-out test increased by 260 ± 223 mL·min^{-1} ($P < 0.01$). When the sessions were arranged in order of execution, no significant differences were found in the peak $\dot{V}O_2$ between most cases. However, a solitary significant difference of approximately 2% was noted between the fourth and the sixth sessions (3416 ± 916 vs 3488 ± 969 mL·min^{-1}, $P < 0.05$).

DISCUSSION

Our goal was to test the predictive ability of the W'_{BAL} model in a variety of conditions and to examine the relationship between $\dot{V}O_2$ and W'. We report three novel results in this investigation. First, we expected to find a predictable W'_{ACT}, regardless of the way the intermittent work or recovery durations were prescribed. This was not the case. Rather, a larger than expected W'_{ACT} was observed as work interval duration was reduced (trials 60–30, 40–30, and 20–30; Fig. 2). Second, reducing recovery duration from 30 to 20 s also resulted in an underprediction of the W'_{ACT}, although this difference was small in absolute terms (<2 kJ). Third, there was a positive correlation between the $D_{\dot{V}O_2}$ and the W' available for CWR exercise. The findings of the study have important implications for both training prescription and performance management during competition.

These observations can be interpreted in multiple ways with respect to the W'_{BAL} model. One possibility is that intermittent exercise reduces the $\tau_{W'}$, speeding the recovery of the W' during intermittent exercise (Fig. 2). Priming exercise has previously been reported to increase the CP (18), and it is known that the CP and the $\tau_{W'}$ are correlated (22). Consideration of equation 3 indicates that a larger D_{CP} would result in a faster $\tau_{W'}$, irrespective of whether that larger D_{CP} was the result of a lower recovery power or a higher CP. It is therefore possible that prior intermittent exercise increased

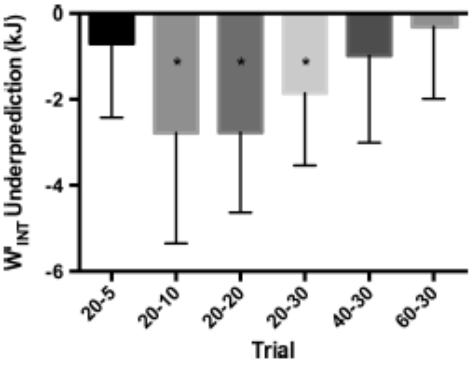

FIGURE 3—Group mean ± SD underprediction of W'_{ACT} using equations 1 and 2. Starred bars indicate trials where underpredictions reached statistical significance.

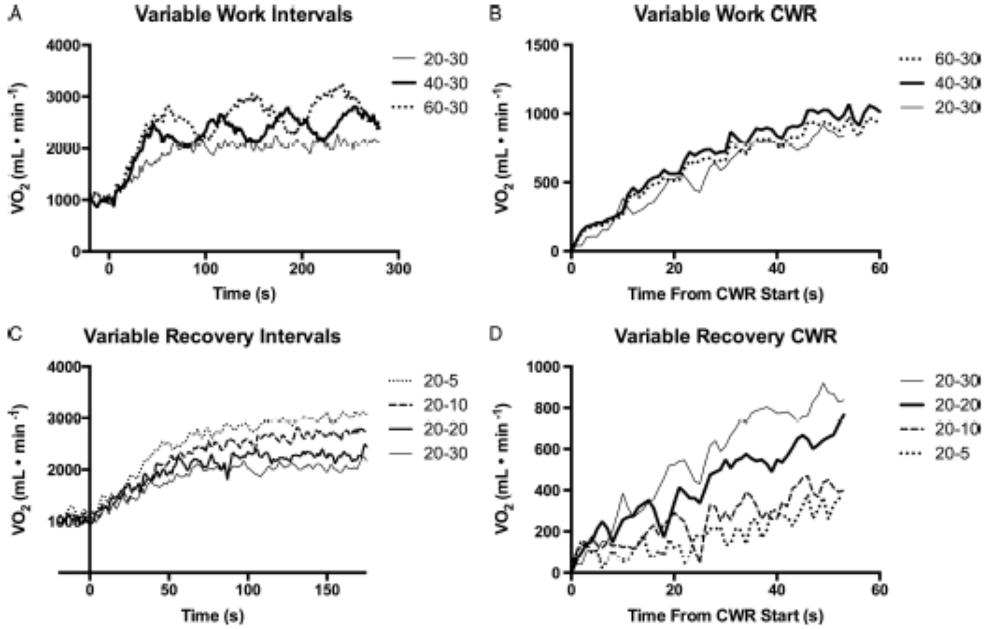

FIGURE 4—A–D. Patterns of $\dot{V}O_2$ expressed as group mean data. Left panels (A and C) indicate $\dot{V}O_2$ recorded during intermittent exercise. Right panels (B and D) reflect $\dot{V}O_2$ recorded during the subsequent CWR bout, measured using $\dot{V}O_{2\,start} = 0$. Note that as work durations shorten (A), $\dot{V}O_2$ begins to resemble a slow exponential increase, which begins to rise more quickly as recovery intervals are shortened (C). CWR patterns seem similar in the variable work duration traces (B) but differ markedly in the variable recovery duration trials (D).

the CP and the D_{CP} and hence permitted a more complete recovery of the W' before CWR exercise began.

It has been reported that exercise above CP (where W' would be used) is associated with disproportionally increased perfusion of Type II muscle fibers (10). Recent work also indicates that the CP can be increased in hyperoxia (26). These results suggest that fiber-specific improvements in O_2 delivery may result in enhanced exercise tolerance. It is possible that intermittent exercise positively affects the CP through a mechanism similar to postexercise hyperemia (23). That is, as exercise moves from, for example, 300 W down to 20 W during a short recovery interval, muscle perfusion may remain higher on average than might be the case with a longer recovery interval. The net result would be muscle that remains better oxygenated (supporting PCr resynthesis) and well "flushed" (removing accumulating, fatigue-related metabolites) due to a higher net blood flow. This might be expected to result in a faster recovery of the W'.

Previous reports indicating that heavy-intensity priming exercise results in an increase in apparent W' during subsequent exercise (8,15) suggest that our results could also be explained by an increased W'. In the present study, the mean power output for the intermittent portion of the majority of the trials fell within the heavy exercise domain. The preceding intermittent exercise may therefore have functioned to prime the muscle (i.e., raise the W') before the CWR bout (8,15). The apparent priming effect on the W' seemed to increase as the ratio of work to rest decreased from 2 (trial

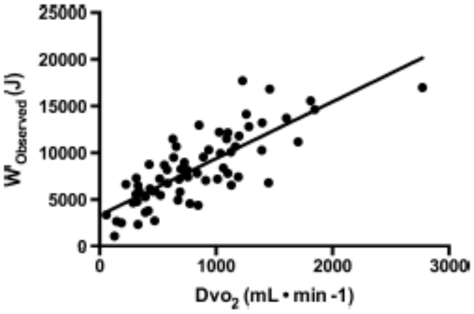

FIGURE 5—A significant correlation was noted between the D_{VCE} and the W' observed during CWR ($r^2 = 0.63$, $P < 0.01$). Note that there may be some overleveraging of the regression line due to a single outlier.

60–30) to 0.67 (trial 20–30) (Fig. 3). This may suggest that intermittent exercise protocols resulting in a lower mean power output within the heavy domain might provide an effective priming stimulus.

An explanation invoking priming becomes troublesome in the context of the variable recovery duration data, however. These data indicate that the estimated W' available for CWR exercise increased in the face of a constant work interval duration as the recovery duration was reduced from 30 to 10 s (trials 20–30 and 20–10, work–recovery ratio varying from 0.67 to 2) (Table 1). The observed W' during the CWR portion of the trial did not approach the predicted value until the recovery duration was reduced to 5 s and the work–recovery ratio became 4 (Fig. 3). However, it is apparent that the W' does not simply represent an "energy store" but is also related to the depletion of substrates or accumulation of metabolites to some critical limiting values (11,12,14,16,21). It is possible that relatively longer work intervals may result in a greater accumulation of metabolites implicated in fatigue (i.e., [P_i], [H^+], and [Ca^{2+}]) and/or a greater depletion of substrates (i.e., [PCr], [ATP], and [glycogen]), thereby reducing the apparent W' remaining. This reasoning may be supported by a comparison between trial 60–30 and trial 20–10 (Table 1). Despite having an equal work–recovery ratio and an identical mean P during the intermittent portion of the trial, the 20–10 protocol resulted in a considerably larger W'_{ACT} as compared with the 60–30 condition. Thus, the intermittent protocol used here may represent a fundamentally different priming stimulus compared with CWR exercise followed by a long recovery. Interestingly, both of the existing studies indicating an increase in W' with priming exposed the subjects to both active recovery (20 W cycling) and several minutes of passive rest before the subsequent work bout (8,15). There have been reports that [PCr] recovery may exhibit an overshoot to a level greater than resting baseline in the period after exercise cessation (17,19). An increased W' may be, at least in part, a consequence of that overshoot.

The linear relationship between $D_{\dot{V}O_2}$ and the W' available for CWR exercise represents an extension of previous results correlating the amplitude of the $\dot{V}O_{2SC}$ and the absolute size of the W' (27) and the modeled discharge of the W' and the $\dot{V}O_{2SC}$ (22). A previous study also reported significant correlations between indices of anaerobic exercise performance and the amplitude of the $\dot{V}O_{2SC}$ (2). The present results suggest that the lower the $\dot{V}O_{2\text{start}}$, the more capacity there is for subsequent fatiguing work (Fig. 5). This increased "muscle reserve" may reflect effects on fiber recruitment and/or metabolite concentrations. It may therefore be conceptually helpful to consider these factors in the context of a multicompartment model of the W' previously proposed (22). In such a scenario, the CP and the W' remain constant. However, separate "compartments" (notionally similar to Type I and Type II fiber populations) are assumed to make discrete individual contributions to the macroscopic W' and possess differing time constants of W' reconstitution, owing to differences in fiber-specific aerobic and anaerobic capacity. The faster-recovering compartment might tend to contribute more to the overall work capacity during intermittent exercise and therefore potentially lead to an extended $T_{\lim}$ during subsequent CWR.

Overall model performance. The group mean $\tau_{W'}$ in the 60–30 trial (403 s; Fig. 2) is in good agreement with that derived for the W'_{BAL} model previously (377 s) using the same work and recovery durations (22). Considering the mean across all conditions, the model as applied to the present data tended to underpredict time to exhaustion by approximately 27 s and underpredict the W' remaining for CWR by approximately 1.6 kJ (Table 1). Thus, while the model remains reasonably robust over a wide variety of conditions, it may be refined to account more specifically for work and recovery durations during intermittent exercise.

Limitations. During the 2-wk intervention period, CP improved in nine subjects and decreased in two. The group mean CP increased by approximately 9%, in keeping with other studies that have described the efficacy of high-intensity interval training on various physiological parameters (5–7). The changes in W' represented almost a mirror image of the CP, increasing in three subjects and decreasing in the remainder, with the difference not achieving statistical significance, consistent with Vanhatalo et al. (25). Moreover, the peak $\dot{V}O_2$ measured in the 3-min all-out test increased by 8% during the experimental testing. It is possible that the training effect observed on CP may complicate the interpretation of the W' recovery data. However, the randomized order of the sessions would be expected to obviate an order effect. We note that the subjects who showed the smallest increase in CP (in particular the subject who improved by only 1 W) showed $\tau_{W'}$ and W' underprediction profiles closest to the group mean values depicted in Figures 1 and 2. Moreover, when placed in order of execution, no significant differences were noted between the peak $\dot{V}O_2$ values recorded during the intermittent protocols, except the fourth and the sixth experimental sessions, which showed a difference of approximately 2%. Collectively, this suggests that the randomization was successful in equally distributing any effect of the (likely unavoidable) improvements in fitness during the study, such that our results chiefly reflect differences in work and recovery duration between the intermittent exercise protocols used.

Practical implications. Whether through an increase in absolute W' or an increase in CP, there seems to be a clear advantage to subsequent exercise performance in limiting work duration during intermittent severe intensity cycling exercise. There may also exist an optimum recovery duration, but more work will be required to fully elucidate this. On the basis of the present data, it would seem that limiting the work interval to 20 s or less and maintaining a recovery interval of between 20 and 10 s would be most advantageous. These results are useful to coaches and the athletes they counsel. As cycling races are often decided by rapid accelerations in the final kilometers, the athlete who best preserves their W' until the last possible moment has a distinct advantage. These results are also important with respect to training prescription. For example, it is now common coaching practice to use "microinterval" protocols (e.g., 15–15 s or 30–30 s [3,4]) interchangeably with more traditional interval work because these microinterval protocols seem less taxing to the athlete. The present results lend credence to these reports. However, our results also suggest that there may be more complex physiology at work than is assumed by many sports practitioners who may think of work and rest intervals purely in terms of accumulated work. Therefore, it may be advisable that athletes continue to be counseled to train in ways most applicable to the way they intend to race. Finally, although the group mean $\tau_{W'}$ in the present study closely corresponds with previous reports (11,22), we have found subjects who seem to recover their W' considerably faster. For example, subject 9 ($CP = 366$ W, $\tau_{W'} = 104$ s in the 60–30 condition) had a calculated $\tau_{W'}$ more than 200 s faster than the apparent asymptote of equation 3. It may therefore be advisable to develop a "personalized" predictive function for the estimation of $\tau_{W'}$ for well-trained athletes.

In conclusion, these results indicate that reductions in work interval duration during intermittent exercise result in a greater-than-expected improvement in subsequent severe-domain CWR performance. These results also indicate that, in the setting of sufficiently short work duration, reductions in recovery duration can also yield subsequent CWR performance in excess of model predictions. Finally, there is a positive relationship between $D_{\dot{V}O_2}$ and the amount of W' available for subsequent CWR exercise, such that optimizing intermittent exercise to minimize fatigue during subsequent exercise may be linked to minimizing $\dot{V}O_2$. The mechanisms responsible for these phenomena remain unclear but may relate to possible priming effects of intermittent exercise on W' and/or CP with consequent effects on the rate of W' reconstitution.

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The authors declare no conflict of interest.

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Chapter 6: Validation of a Novel Intermittent W′ Model for Cycling Using Field Data

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6.0 Abstract

Recently, an adaptation to the critical power (CP) model has been published, which permits the calculation of the balance of W′ remaining (W'_{BAL}) at any time during intermittent exercise. As the model is now in use in both amateur and elite sport, the purpose of this investigation was to assess the validity of the W'_{BAL} model in the field. Data were collected from the bicycle power meters of eight trained triathletes. W'_{BAL} was calculated and compared between files where subjects reported becoming prematurely exhausted during training or competition and files where the athletes successfully completed a difficult assigned task or race without becoming exhausted. Calculated W'_{BAL} was significantly different between the two conditions ($p < 0.0001$). The mean W'_{BAL} at exhaustion was 0.5 ± 1.3 kJ (95% CI = 0 – 0.9 kJ), whereas the minimum W'_{BAL} in the non-exhausted condition was 3.6 ± 2.0 kJ (95% CI = 2.1 – 4.0 kJ). Receiver-operator characteristic (ROC) curve analysis indicated that the W'_{BAL} model is useful for identifying the point at which athletes are in danger of becoming exhausted (area under ROC curve = 0.914 (SE: 0.05, 95% CI: 0.82 – 1.0, $p < 0.0001$). The W'_{BAL} model may therefore represent a useful new development in assessing athlete fatigue state during training and racing.

6.1 Introduction

Athletes in a variety of sports face the challenge of pacing. For example, cyclists are often observed drafting in an attempt to conserve energy for a final sprint and marathon runners tend to surge between periods of slower running in an effort to “crack” competitors. The amount of energy available to an athlete as well as their ability to accurately gauge their state of fatigue will necessarily help to determine the ultimate performance outcome. It would be to an athlete’s advantage to be able to quantitatively evaluate energy availability and fatigue state when formulating an optimal pacing strategy.

There are several mathematical constructs available to help understand the state of an athlete’s energy reserves. (176) In recent years, there has been a renewed interest in the critical power (CP) model:

$$P = \frac{W'}{Tlim} + CP \qquad \textbf{Eq. 6.0}$$

Where P equals the power output at any time t, T_{lim} is time until exhaustion, and W' represents the work capacity available above the CP.

The CP is best understood as a threshold phenomenon, defining the boundary between the heavy and severe exercise intensity domains (43, 112, 127, 130, 185, 225). It represents the highest power output that can be sustained whilst maintaining a physiological steady state, and appears to occur at a power output close to the maximal lactate steady state (MLSS) (130, 188). Exercise above the CP results in an inexorable rise in $\dot{V}O_2$ (in the face of a constant external power output), such that the maximum ($\dot{V}O_{2MAX}$) is attained prior to exhaustion (43, 127, 128, 130).

The work capacity above CP, the parameter W′, is fixed; that is, the W′ remains constant regardless of the rate of its discharge. The construct appears robust, as the depletion and reconstitution of the W′ can be calculated with some precision under a variety of circumstances (60, 84, 168, 206). Given that, during training and competition, athletes often surge above the CP, and then take periods of relative recovery, the W′ may perhaps be best viewed as a battery that is alternatively depleted (by working above CP), and recharged (during exercise below CP).

Morton et al. (168) published the first adaptation of the CP model to intermittent exercise, which is of particular relevance given the above mentioned race scenarios. However, this model is based on certain physiological assumptions, which may be challenged (65, 84, 206). In particular, the Morton formulation relied on a linear recovery of the W′, whereas recent data imply a curvilinear recovery (84, 206). In light of these new data, Skiba et al. (206) developed a novel continuous model of the W′ balance remaining at any time during intermittent exercise (W'_{BAL}).

Although the W'_{BAL} model is now being used in elite sport, (178) there have been no published studies on the validity of the model in the field, aside from a single case study in the original publication. (206) Given the present ubiquity of on-bicycle power measurement devices in cycling and triathlon, there now exists the realistic possibility of assessing the efficacy of the model using real training and competition data. The purpose of the present investigation was therefore to evaluate the validity of the W'_{BAL} model in the field. If the W'_{BAL} model accurately reflects the state of charge or discharge of the W′ during intermittent exercise, it should be possible to predict athlete exhaustion, which theoretically coincides with the W'_{BAL} model reading zero.

6.2 Methods

Data files were retrospectively obtained from six well-trained male triathletes and two well-trained female triathletes (mean ± SD: age: 35 ± 3.8 years, height: 1.72 ± 0.1 m, mass: 72.9 ± 15.1 kg , CP: 258 ± 25 W, W′: 16.6 ± 3.1 kJ; Table 6.0) who had been training with commercially available on-bike power meters. The validity, reliability and accuracy of these devices have been previously reported (31, 97). Files were selected based upon the athlete's reported inability to complete an assigned training task that involved maintaining a supra-CP work rate, or inability to keep pace with rivals or execute a desired strategy during competition due to the sudden onset of fatigue that forced a reduction in power below the CP. For the purposes of this analysis, such athletes were classified as having achieved 'volitional exhaustion'. For comparison, files were also obtained from the same athletes training or racing sessions that the athletes reported were difficult, but did not result in exhaustion or force a reduction in power output below the CP.

Data were only included if the subject possessed a CP and W′ established from field-testing consisting of at least three or four best-power-for-time trials. The mean power held for the entirety of the test duration was used in the calculation of the CP and W′, and the standard error (SE) for both the CP and W′ were calculated (Table 6.0). Due to differences in preferred testing conditions between different athletes and their coaches and the retrospective nature of the study, the actual length of the predictive trials could not be standardized among participants. However, all predictive trials were between approximately 2 and 20 min duration, as has been previously recommended. (37) In all cases these predictive trials began from a moving start with the athlete pedalling at power output less than 30 W. All power meters were appropriately zeroed as per the manufacturer's instructions before data collection began, and samples were collected at least every 1.2 s.

Data analysis

Data files were analysed using the continuous equation previously reported by Skiba et al. (206)

$$W'_{BAL} = W' - \int_0^t W'_{EXP} \cdot e^{\frac{-(t-u)}{\tau_{W'}}} \cdot du \qquad \textbf{Eq. 6.1}$$

Where W'_{EXP} is representative of the amount of the starting W′ that is presently expended, while (t-u) is equal to the time in seconds where the athlete is recovering below CP. The $\tau_{W'}$ is the time constant of the reconstitution of the W′.

The $\tau_{W'}$ was calculated using the regression equation previously reported by Skiba et al. (206).

$$\tau_{W'} = 546 \cdot e^{(-0.01 \cdot Dcp)} + 316 \qquad \textbf{Eq. 6.2}$$

Where D_{CP} is equal to the difference between the recovery power and the athlete's CP. Recovery power was calculated as the mean of all data points in the file recorded below CP.

The subject's predicted W'_{BAL} at the time of volitional exhaustion was calculated. If the subject became exhausted multiple times within the same file, these values were also recorded. The mean across all recorded values for W'_{BAL} at exhaustion, and the SD, SE and 95% confidence intervals (CI) were calculated for the entire population of subjects (SPSS, Armonk, NY).

A slightly different procedure was carried out utilizing files where athletes reported expending considerable effort without frank exhaustion. In general, the minimum predicted W'_{BAL} in the file was recorded. However, if a data file contained multiple instances where the athlete substantially depleted the W′ (i.e. arbitrarily defined as a

W'_{BAL} driven to less than 50% of baseline W'), this was also recorded as an additional data point corresponding to ‘non-exhaustion’.

An unpaired t-test with Welch’s correction was used to determine if a significant difference existed between model predictions of the W'_{BAL} in the non-exhausted and exhausted data sets. In order to calculate a diagnostic threshold that defines exhaustion on the basis of the $W'_{BAL,}$ receiver-operator characteristic (ROC) curve analysis was used (GraphPad Prism 6, Graphpad Software, San Diego, CA) (179, 247). The ROC methodology was developed as a means for differentiating between signal and noise in the analysis of radar data (179). It was first used in medical decision making in the late 1950’s, and is now in wide use as a means of assessing the diagnostic accuracy and usefulness of a particular test (for review, see (247)). In the context of this investigation, we compared the W'_{BAL} calculated at the time of volitional exhaustion across all files with the lowest recorded W'_{BAL} from files where the athletes did not become exhausted. The area under the ROC curve was calculated to determine the diagnostic accuracy of the W'_{BAL} model, where a value of 1.0 is indicative of a perfect test and a value of 0.5 indicates that there is no distributional difference between the data sets (i.e. the test in question is no more accurate than flipping a coin to determine a positive or negative result).

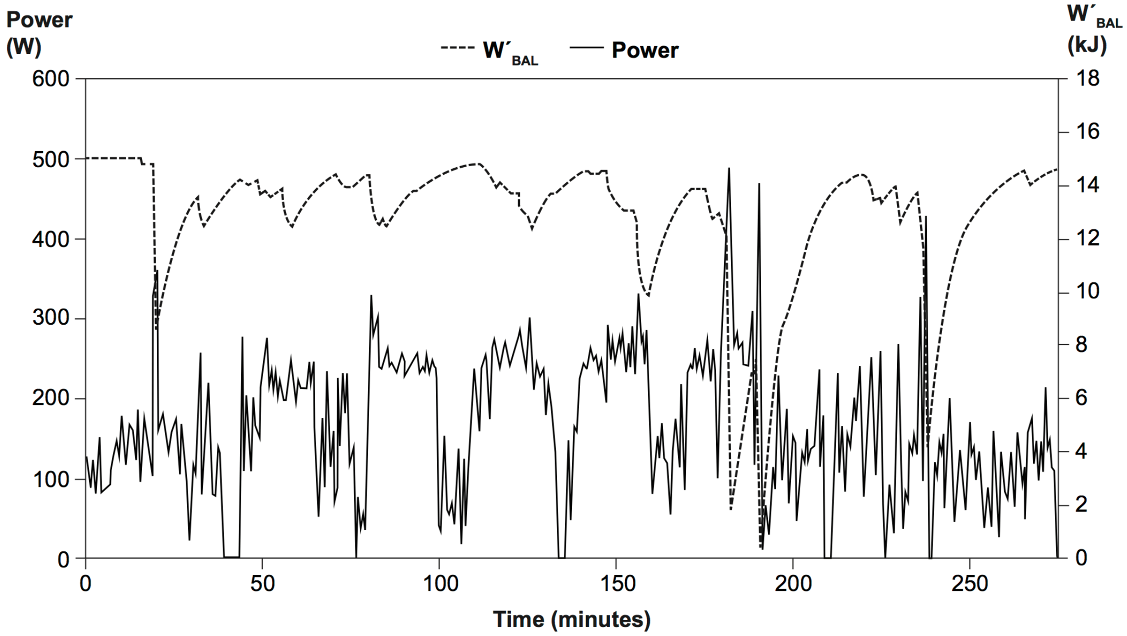
6.3 Results

The CP and W′ values calculated from field-testing were robust in most cases (Table 6.0). The mean SE was ~ 0.6% for the CP estimates and ~ 7.4% for the W′ estimates. In six of the eight subjects, the SE for the W′ was less than 10%.

Table 6.0: *Subject characteristics, CP, W' and associated standard error calculations. Reprinted from (207), with permission.*

Subject (M/F)	**Age (y)**	**Height (M)**	**Mass (Kg)**	**CP (W)**	**SE CP (W)**	**SE CP (%)**	**W' (kJ)**	**SE W' (kJ)**	**SE W' (%)**
1(M)	31	1.73	63	269	1	0.30%	14.7	0.7	4.80%
2(M)	39	1.7	70	288	6	2.10%	20.1	3.9	19.40%
3(F)	30	1.6	66	205	0	0	17.5	0.233	1.30%
4(M)	34	1.93	104	227	2	0.80%	20.2	1.1	5.40%
5(M)	41	1.73	67	268	2	0.70%	17.1	1.05	6.10%
6(M)	34	1.8	84	260	0	0	17.8	0.251	1.40%
7(F)	34	1.62	55	257	1	0.40%	10.7	0.7	6.50%
8(M)	37	1.7	74	292	3	1%	15	2.1	14%
Mean	35	1.7	72.9	258.3	1.9	0.60%	16.6	1.3	7.40%
SD	3.8	0.1	15.1	29.4	2	0.70%	3.1	1.2	6.20%

A total of 22 data files containing instances of athlete exhaustion were examined. Three of these files included multiple instances of athlete exhaustion, which required a reduction in power below the CP for some period of time. This resulted in the identification of 26 candidate data points corresponding with athlete exhaustion. Mean W'_{BAL} at exhaustion was calculated as 0.5 ± 1.3 kJ (95% CI = 0 – 0.9 kJ). The SE was calculated as 1.0 kJ (95% CI = 0.7 – 1.3 kJ). A representative model output for an athlete who reached exhaustion is shown in Figure 6.0A.



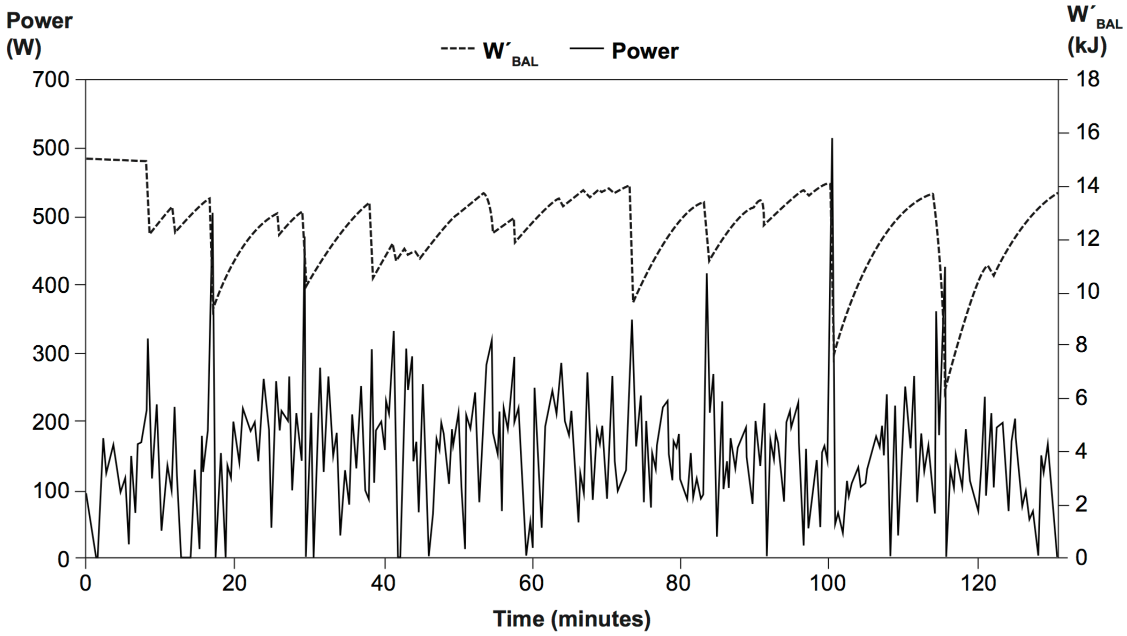


Fig. 6.0A and 6.0B: *Graphs indicate typical appearance of traces for exhausted (A) and non-exhausted (B) conditions in a representative athlete (CP = 292 w, W' = 15 kJ). The solid trace is indicative of power output, while the dashed trace is indicative of the calculated W'*$_{BAL}$*. In panel A, the subject experienced extreme fatigue at approximately 180 and 190 min and was forced to reduce power output below CP to facilitate recovery. In panel B, the subject was able to deplete the W'*$_{BAL}$ *as low as approximately 6 kJ without significant problems. Periods of zero power output correspond to downhill segments of the racecourse. Reprinted from (207), with permission.*

A total of 23 data files containing instances where athletes expended W′ but did not become exhausted were collected. Two files were identified where athletes depleted the W′$_{\mathrm{BAL}}$ below 50% of baseline twice without becoming exhausted. This resulted in the identification of 25 data points that corresponded to substantial W′$_{\mathrm{BAL}}$ depletion without concomitant exhaustion. Mean W'_{BAL} in these instances was calculated as 3.6 ± 2.0 kJ (95% CI = 2.1 – 4.0 kJ). The SE was calculated as 1.6 kJ (95% CI = 1.3 – 2.2 kJ). A representative model output for a non-exhausted athlete is shown in Figure 6.0B.

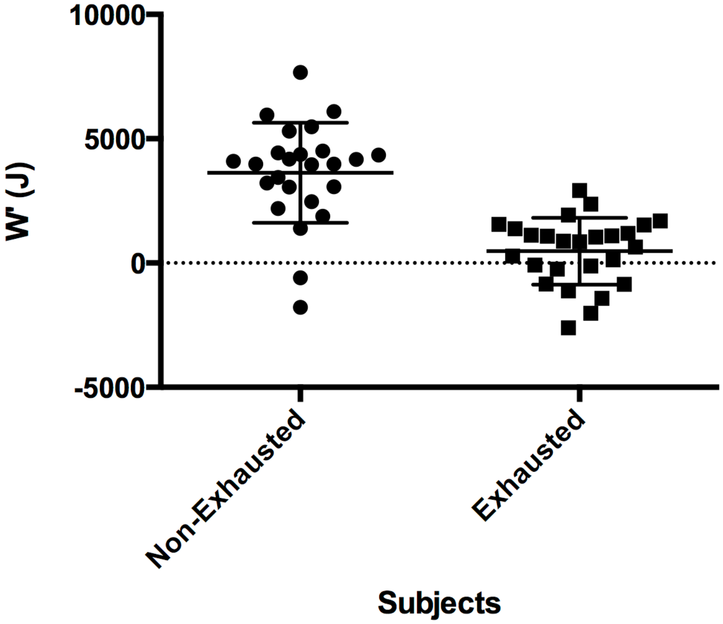


Fig. 6.1: *Distribution of calculated W'_{BAL} in the non-exhausted and exhausted states, respectively with error bars indicating 95% CI. Reprinted from (207), with permission.*

An unpaired t-test indicated a significant difference in W'_{BAL} between the exhausted and non-exhausted states ($p < 0.0001$). The area under the ROC curve was calculated to be 0.914 (SE: 0.05, 95% CI: 0.82 – 1.0, $p < 0.0001$), indicating that the W'_{BAL} model represented an excellent diagnostic test in the population studied (Figures 6.1 and 6.2).

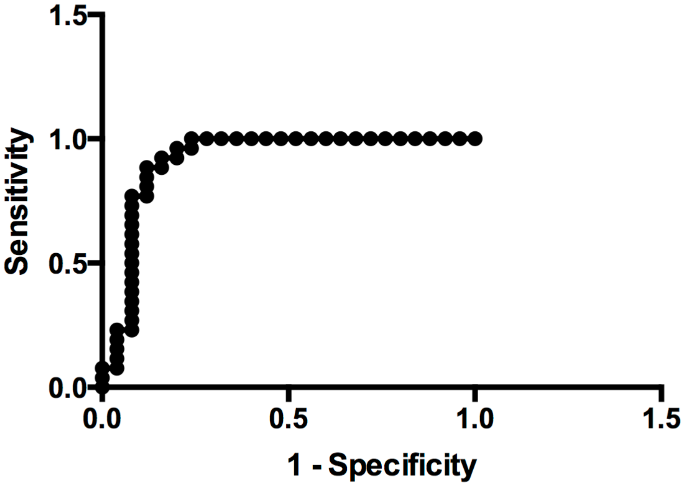


Fig. 6.2: *ROC curve demonstrating quality of model as discriminator between the exhausted and non-exhausted states. The calculated area under the curve is equal to 0.91, and is indicative of an excellent diagnostic test. Reprinted from (207), with permission.*

6.4 Discussion

The W′ parameter of the CP model has been previously calculated using several different work rate-forcing functions: constant power, varied power, "all-out" sprint, ramp incremental, self-paced and intermittent exercise (59, 88, 223, 224). This is the first investigation to apply the W'_{BAL} model variant to highly stochastic, field-derived power meter data. Although this group of subjects did not become exhausted at precisely the point at which the model predicted a $W'_{BAL} = 0$, the mean W'_{BAL} at exhaustion 0.5 ± 1.3 kJ is well within the SE of the W′ in this study and others (37, 121, 124). This suggests that W'_{BAL} model is at least as accurate as the CP model used to calculate the W′, and may provide a useful means of identifying when a subject is nearing exhaustion.

The results of the ROC curve analysis indicate that the W'_{BAL} model represents a robust method of differentiating between exhaustion and non-exhaustion in this population (Figures 6.1 and 6.2). ROC models are typically used in evaluating diagnostic medical testing, where the risks associated with a test being falsely classified as positive or negative have substantial consequences for health. On the basis of the present data, it is possible to achieve 95% sensitivity with 24% 'false positives' if the 'threshold' for W′ depletion (which predicts athlete exhaustion) is set at 2.5 kJ. This is comparable to the typical criteria used for judging statistical tests in research ($p < 0.05$ for type I error, $p < 0.2$ for type II error). If the threshold is set at $W'_{BAL} = 1.5$ kJ, 80% of athletes will be appropriately classified as exhausted and 88% appropriately classified as non-exhausted. Given anecdotal reports from a number of athletes (who were not part of the present study) indicating feelings of extreme fatigue at $W'_{BAL} < 1.5$ kJ, it is both statistically and practically defensible to discourage athletes from proceeding below a W'_{BAL} of 1.5 kJ (typically less than 10% of the W′) if they wish to avoid premature exhaustion. Importantly, the present ROC methodology can be applied iteratively as more data are collected in order to refine the estimate of the W'_{BAL} threshold that is associated with exhaustion.

There are several factors worthy of analysis as we consider potential improvements to the W'_{BAL} model. We must first consider the fundamental characteristics of the CP model, as the performance of the W'_{BAL} model will necessarily be affected by the reliability of the CP and W′ estimates used. The SE for the W′ is approximately 1 kJ in the very best cases, (121, 124) but can be as high as ~2 kJ (37). Examination of Table 5.0 indicates that, in most cases in the present study, the SE for the CP and W′ was generally 2 W or less and 1.1 kJ or less, respectively, indicating mathematically robust models. An equally important factor, however, is the test-retest reliability of the parameters comprising the model; this is typically higher for the CP than the W′ (130, 224). The test-retest reliability for the W′ has been reported to be as low as approximately 7% (0.8 kJ). (211)

Another important factor which may influence the accuracy of the W'_{BAL} is the actual time course of the W′ recovery. The original regression equation reported by Skiba et al. (206) showed considerable inter-individual variability with respect to the $\tau_{W'}$. We recently tested a subject in our laboratory with a calculated $\tau_{W'}$ more than 200 s faster than the asymptote of Eq. 5.1 (208). It may therefore be advisable to calculate a personalized predictive function for the τ_W such that the model may be specifically tuned to each athlete and therefore improve the quality of the W'_{BAL} predictions. This will require a prospective study, rather than the retrospective protocol described here.

It is perhaps surprising that using a mean recovery power for the calculation of $\tau_{W'}$ results in as robust a model as it does. This may be explained in part by recent observations applying the W'_{BAL} model to intermittent exercise in a laboratory setting.(208) In that investigation, subjects depleted approximately 50% of the W′ using intermittent exercise, then immediately switched to constant work rate exercise (CWR) to deplete the remainder of the W′. We observed that the W′ available for CWR was directly proportional to the difference in $\dot{V}O_2$ between the end of the intermittent exercise bout and $\dot{V}O_{2MAX}$. Since mechanical power output is one factor driving $\dot{V}O_2$, using a mean D_{CP} for the calculation of a single $\tau_{W'}$ may suffice simply because it provides a reasonable approximation of the mean oxidative metabolic rate of the muscle over time.

The final source of error in the calculation of the W'_{BAL} may be the athletes themselves, i.e. that they were not actually exhausted at the time of exercise termination. Due to the retrospective nature of the study, the athletes did not set out on the training or racing task with the goal of reaching exhaustion prematurely. During laboratory based CP testing, exhaustion is typically defined as a fall in cadence of greater than 5 rpm despite vigorous encouragement. In the field, such guidelines do not apply and athletes abandon the task when they ‘feel’ they cannot go on. Nevertheless, competitive athletes are typically highly motivated and are unlikely to abandon a race or assigned training task unless under severe duress.

6.5 Practical applications and conclusions

In summary, the present study indicates that the W'_{BAL} model represents a robust method of assessing exhaustion (gauged by complete depletion of the W′) in a population of well-trained triathletes. The demonstrable utility of the W'_{BAL} model as applied to field data suggests the possibility of programming a cycling computer or GPS device to monitor W'_{BAL} during training and racing, such that athletes can consider adjusting their pacing strategy accordingly. Application of the model in a larger, more diverse population of athletes is warranted to substantiate the present findings. Further research will be required to ascertain whether modifications to the model (in particular, individual tuning of the $\tau_{W'}$) might further enhance the predictive power of the model.

Chapter 7: Intramuscular Determinants of the Ability to Recover Work Capacity Above Critical Power

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7.0 Abstract

The critical power (CP) model includes two parameters: the CP and the W'. Whist the CP appears to be a measure of aerobic metabolism, the physiological basis of the work capacity above CP (the W') remains less well understood. **PURPOSE:** The primary purpose of this investigation was to analyse the relationship between the recovery of the W' and the recovery of intramuscular substrates and metabolites using ^{31}P and ^{1}H magnetic resonance spectroscopy. **METHODS:** Ten healthy people (four females and six males) were tested to determine CP and W' for single leg extensor exercise. They subsequently exercised in the bore of a 1.5T MRI scanner at a supra-CP work rate predicted to result in exhaustion in 3 min. Following exhaustion, subjects rested in place for 1, 2, 5, or 7 minutes, and then attempted to repeat the effort. The difference in W' between the two bouts was used to derive the time course of W' recovery, which was then compared to the recovery of creatine phosphate [PCr], pH, carnosine content, and to the behaviour of a novel derivation of the W'_{BAL} model. **RESULTS:** The recovery kinetics of the W' closely correlated with the prediction of the novel model ($r = 0.97$, $p < 0.05$). [PCr] recovered considerably faster ($t\frac{1}{2} = 38\ s$) than W' ($t\frac{1}{2} =\ 232\ s$). However, the W' available for the second exercise bout was directly correlated with the difference between [PCr] at the beginning of the work bout and [PCr] at exhaustion ($r = 0.99$). Muscle carnosine content was curvilinearly related to the rate of W' recovery, with higher carnosine content correlated with faster recovery. **CONCLUSION:** The kinetics of W' recovery in single leg extensor exercise is comparable to that observed in whole body exercise, suggesting a conserved mechanism. The extent to which the recovery of the W' can be directly attributed to the recovery of [PCr] is unclear. The relationship of the W' to muscle carnosine content suggests novel future avenues of investigation.

7.1 Introduction

Muscular fatigue is multifactorial, with a number of proposed and interrelated mechanisms based upon the type, intensity and duration of exercise (3). However, despite the multitude of factors involved, the fatigue process for durations between 2 and ~30 min can be modelled using relatively simple mathematics. One particularly useful construct is the critical power (CP) model, as it is able to predict time to exhaustion over a wide range of power outputs and time scales (119, 130), in both synergistic muscle group and whole-body exercise (for review, see (130)).

The CP model (164) describes the hyperbolic relationship between power output and time to exhaustion using two parameters: the CP and the W'.

$$P = \frac{W'}{Tlim} + CP \qquad \textbf{Eq. 7.0}$$

In this model, P is equal to power output and T_{lim} is equal to time-to-exhaustion at that power output. The CP is principally a parameter of oxidative metabolism, representing the highest power output for which it is possible to maintain a physiological steady state (112, 130, 134, 185, 186). The W' represents the finite energy store available to the subject should they exceed CP (130, 185, 239).

The CP model assumes that the W′ does not vary with rate of discharge. Moreover, as discussed in Chapter 2, the depletion and reconstitution of the W′ can be calculated under a variety of circumstances (60, 84, 168, 206). These observations suggest a highly conserved and organized physiological process, and suggest the possibility of identifying particular metabolic correlates of the W′. Indeed, a number of recent experiments suggest that depletion of the W′ is related to the accumulation of metabolites and / or depletion of substrates to limiting values (62, 63, 84, 88, 134, 185). However, the precise physiological determinants of the W′ remain unclear.

One possible means of elucidating the relative importance of the different facets of muscle metabolism to the overall W′ is by viewing them in the context of post-exercise recovery. Ferguson et al. (84) reported that the W′ recovered considerably more quickly than plasma lactate, but more slowly than pulmonary $\dot{V}O_2$ following whole body exercise. The extent to which these observations directly relate to the recovery of the exercising muscle mass is difficult to know, since pulmonary $\dot{V}O_2$ does not always correlate with the recovery of muscle $\dot{V}O_2$ (142). It may therefore be instructive to directly interrogate intramuscular metabolic disturbance during exhaustive exercise and subsequent recovery through the use of ^{31}P magnetic resonance spectroscopy (^{31}P-MRS). ^{31}P-MRS offers the opportunity to simultaneously observe intramuscular high-energy phosphate and pH, both of which have been implicated as determinants of the W′ (130).

Any proposed role for a pH-dependent mechanism in the depletion and recovery of the W′ requires careful consideration of buffering capacity. Pre-exercise alkalosis has not been found to alter the CP or W′ (227). However, there has been substantial interest in the possible pH buffering effects of carnosine, a β-alanine / histidine dipeptide. Intramuscular carnosine may be increased through the ingestion of β-alanine, and recent studies have documented a positive correlation between increased carnosine and exercise performance (15, 111, 220). Muscle carnosine is easily measured by ^{1}H- MRS, and may yield information pertinent to the present investigation.

Given that both intramuscular [PCr] and the W′ become substantially depleted at the point of exercise intolerance (62, 63, 84), and both the recovery of [PCr] and the recovery of the W′ exhibit curvilinear kinetics (84, 206), our primary hypothesis was that recovery of the W′ would be significantly correlated with the recovery of intramuscular [PCr]. We also hypothesized that the recovery of the W′ would be significantly correlated with the recovery of pH and muscle carnosine.

7.2 Mathematical framework

Both Ferguson et al. (84) and Skiba et al. (206, 208) (Chapters 4 and 5) reported curvilinear recovery of the W', the former following constant work rate (CWR) exercise and the latter during intermittent exercise (Eq. 7.1). Skiba et al. (206) also demonstrated a dependence of the time constant of W' recovery ($\tau_{W'}$) on the difference between the recovery power output and the CP (D_{CP}) (Eq. 3).

$$W'_{BAL} = W' - \int_0^t W'_{exp} \cdot e^{\frac{-(t-u)}{\tau_{W'}}} \cdot du \qquad \textbf{Eq. 7.1}$$

where W'_{BAL} represents the balance of W' remaining, W' equals the subject's known W′ as calculated from the 2-parameter CP model, W'_{exp} is equal to the expended W′, and $(t\text{-}u)$ is equal to the time in seconds between segments of the exercise session that resulted in a depletion of W′

$$\tau_{W'} = 546 \cdot e^{(-0.01 \cdot Dcp)} + 316 \qquad \textbf{Eq. 7.2}$$

There are several well-characterised analogous mathematical systems available to conceptualize W'. For instance, W' can be thought of as analogous to a tank of water, which may be filled by a tap (metabolism) and emptied by a drain of variable size (physical work) (176). Whilst this example emphasizes ‘depletion’, the analogy can be equally applied to an ‘accumulation’ hypothesis, in which the vessel is filled by some metabolite that induces fatigue upon reaching a particular level. However, such a system implies a linear progression of the ‘refill’ of the W' ‘tank’, which may not be strictly correct in light of recent results (84, 206).

An alternative model may be developed using basic principles of chemical kinetics. Such a model suggests that we consider the muscle to be a tank within which the W' is a chemical reactant. There are several important properties of such a kinetics-based model that makes it attractive in light of previous observations. Formal derivation from first principles effectively recovers the equation empirically derived by Skiba et al. (206) (Appendix 1).

$$W' = W'_o - W'_{\exp} e^{-D_{CP}t/W'_o} \qquad \textbf{Eq. 7.3}$$

Both Eq. 7.1 and 7.3 dictate a curvilinear recovery of the W', whilst Eq. 7.2 and 7.3 dictate slowing of recovery as recovery power approaches CP (i.e. as D_{CP} approaches zero). In contrast to Eq. 7.2, however, Eq. 7.3 is easily scaled to the power output of the exercise modality, as the $\tau_{W'}$ is calculated as the starting W' (W'_o) divided by D_{CP} (Appendix 1). Critically, this means that the W' recovery model does not require fitting to the data but is instead calculated from the known D_{CP} and independently estimated W'_o . We confirmed the accuracy of this alternative formulation through retrospective analysis of the data reported by Skiba et al. (206). The derived $\tau_{W'}$ values for the seven subjects in the aforementioned study from our laboratory were correlated with those calculated using the new model formulation ($r = 0.84$, $p = <0.001$; Figure 7.0) (Eq. 7.3, Appendix 1). Thus, Eq. 7.3 was utilized for the analysis of the data in the present study.

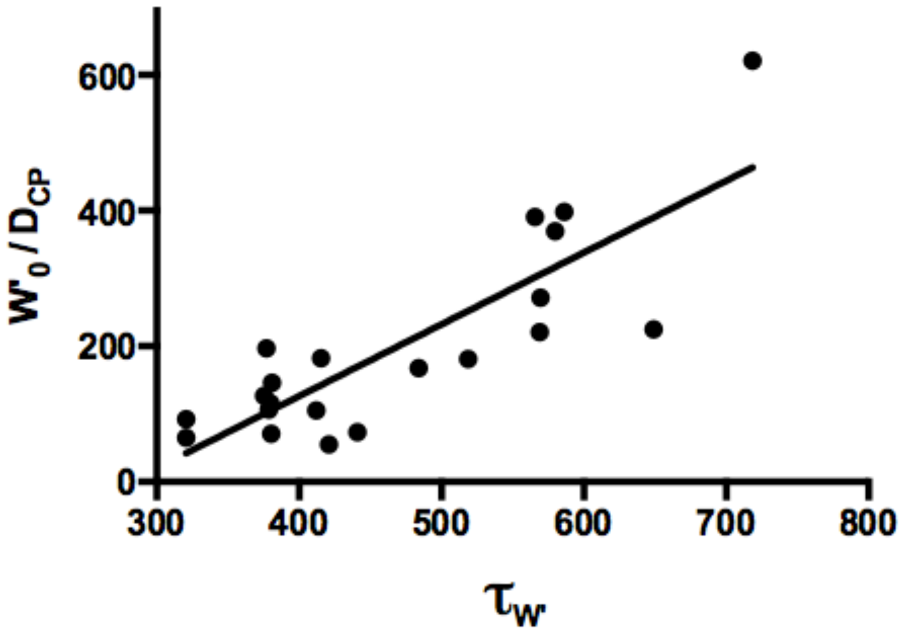


Figure 7.0: *Comparison of* $\tau_{W'}$ *as calculated by regression Eq. 7.2 to that proposed in Eq. 7.3. The two are well-correlated (r = 0.84, p = <0.001).*

7.3 Methods

Ten healthy people (four females and six males, mean ± SD: age 22 ± 7 yr, height 1.71 ± 0.1 m, body mass 71.8 ± 15.4 kg) volunteered to participate in this study. The subjects were all recreational athletes, but were not highly trained. Three subjects had a history of strength / power training, whilst the remainder participated in endurance sports such as swimming, running and cycling. The study was approved by the University of Exeter Research Ethics Committee. After the experimental procedures, associated risks, and potential benefits of the study protocol had been explained to the subjects, they were required to give their written informed consent in order to participate. Subjects were instructed to arrive at the laboratory in a rested and fully hydrated state, and at least 3 h postprandial. They were also asked to avoid strenuous exercise in the 24 h preceding each testing session. Subjects were asked to refrain from caffeine and alcohol for 3 h before each test. All tests were performed at the same time of day (± 2 h) at sea level in an air-conditioned laboratory or MRI suite at 20ºC. At least 48 hours separated each test. All subjects completed all experimental trials, with the exception of one subject, who was unavailable for ^{1}H spectroscopy.

Phase 1 Testing

During the first phase, subjects reported to the physiology laboratory. Using an ergometer previously described (44, 134), subjects completed at least three and not more than five separate knee extension protocols to exhaustion. During each trial, the subject was required to maintain a rhythm of 40 extensions per minute in time with an audible electronic metronome. Each trial featured a different mass and time to exhaustion was recorded. All trials resulted in exhaustion (defined as inability to complete full range of motion, maintain time with the metronome, or decision to stop work) between 90 and 600 s. Subjects were given strong vocal encouragement throughout the task. Work was calculated by the Newtonian equation $m \times g \times h$; where m = mass, g = 9.81 m/s^2, and h = the displacement of the mass lifted by the subject. CP and W′ were calculated by plotting joules expended against time limit for each task and plotting a linear regression through the points where W′ = y-intercept and CP = slope of the line.

Phase 2 testing

During the second phase of the protocol, the subjects performed four separate exercise protocols within the bore of a 1.5 T superconducting magnet. In each case, the subject performed a conditioning bout (B_C) of single-leg knee extension exercise at the power output expected to result in fatigue in 180s (WR_{180}) until exhaustion, followed by a passive recovery interval of either 1, 2, 5, or 7 minutes (RI_1, RI_2, RI_5 or RI_7) with the leg resting fully extended on the scanner bed. (Due to the unusual exercise modality, this power output was increased from that applied in the other studies to ensure the subjects remained motivated and worked to exhaustion). After the RI elapsed, the subject undertook the experimental bout (B_E) of single-leg knee extension exercise at WR_{180} until exhaustion. As previous work has noted that a conditioning bout of exhaustive exercise does not alter the CP (84), it was assumed that any change in work capacity between B_C and B_E must be due to a change in the W′. Thus, work done in B_E was divided by work done in B_C to determine recovery of the W′ after each experimental visit.

Equipment and ^{31}P-MRS measurements

^{31}P-MRS was performed in the University of Exeter Magnetic Resonance Research Centre (Exeter, UK) with a 1.5-T superconducting MR scanner (Intera, Philips). Participants were positioned within the scanner, head first in a prone position with a 6 cm

^{31}P transmit/receive surface coil placed within the scanner bed and positioned such that the subjects' right *rectus femoris* muscle was centred directly over it. Survey images were initially acquired to determine that the muscle was positioned correctly relative to the coil. Several preacquisition steps were then carried out to optimize the signal from the muscle under investigation. An automatic shimming protocol was undertaken within a volume that defined the quadriceps muscle to optimize the homogeneity of the local magnetic field, thereby leading to maximum signal collection. Tuning and matching of the coil were subsequently performed to maximize energy transfer between the coil and the muscle.

To ensure that scanning took place at the same point of muscle contraction, thereby ensuring the muscle was at a consistent distance from the coil at the time of data sampling, the subject was audibly cued at the same rate as during the CP and W′ determination trials. The subject was also visually cued via a display consisting of two vertical bars, one that moved at a constant rate with a frequency of 0.67 Hz and one that monitored foot movement via a sensor within the pulley to which they were connected. The subject endeavoured to match the movements of these two bars. The work done by the subject was recorded in the same fashion as during the W′ and CP determination trials.

Before exercise, during exercise, and during recovery, data were acquired every 12 s, with a spectral width of 1500 Hz. The subsequent spectra were quantified via peak fitting, with the assumption of prior knowledge, using the jMRUI (version 2) software package and the AMARES fitting algorithm (221). Spectra were fitted with the assumption that P_i, PCr, α-ATP (2 peaks, amplitude ratio 1:1), γ-ATP (2 peaks, amplitude ratio 1:1), β-ATP (3 peaks, amplitude ratio 1:2:1), and phosphodiester peaks were present. In all cases, relative amplitudes were corrected for partial saturation due to the repetition time relative to the longitudinal relaxation time (T1). Intracellular pH was calculated using the chemical shift of the P_i spectral peak relative to the PCr peak (217).

Equipment and ^{1}H spectroscopy measurements

^{1}H spectroscopy was undertaken with a 4 element, wrap around coil. A voxel was selected in the right *rectus femoris* at approximately mid-thigh (at the same location as ^{31}P-MRS was undertaken) of dimensions 20x30x50 mm, at which location point-resolved spectroscopy (PRESS) was undertaken. 96 measures were averaged, with a repetition time (TR) = 2000 ms, echo time (TE) = 31 ms, 1024 data points and spectral bandwidth = 1200 Hz. Water and carnosine peak areas were calculated within jMRUI (ver 4) software. Carnosine values were expressed as peak size relative to the water peak having taken into account respective T1 and T2 (transverse relaxation) times.

Statistics

Recovery of the W′ was analysed in each subject and for the group as a whole using both linear regression and comparison to the recovery kinetics predicted by Eq. 7.3. A paired T-test was utilized to compare predictions of W′ recovery as calculated by Eq. 7.3 with the fraction of W′ recovery actually observed for each time point.

[PCr], [P_i], and pH at exhaustion were compared between B_C and B_E among all experimental conditions utilizing repeated-measures ANOVA. Recovery of [PCr] as a fraction of resting [PCr] during the recovery intervals were plotted against time, and fit exponentially utilising eq. 3.1 in the General Methods. The recovery kinetics was compared with those of the W' via linear regression. $D_{[PCr]}$ was defined as the difference between [PCr] at the end of the recovery following B_C and the [PCr] at the point the subject became exhausted during B_E. $D_{[PCr]}$ was compared to the theoretical recovery of the W′ predicted by Eq. 7.3 utilizing linear regression.

Resting muscle carnosine content was compared to pH at exhaustion in both B_C and B_E by linear regression. In addition, carnosine content was linearly regressed against change in pH between B_C and B_E, as well as the minimum pH in both B_C and B_E.

In all cases, analyses were carried out using GraphPad Prism (GraphPad, San Diego, CA, USA). Significance was accepted at the 0.05 level and data are reported as mean ± SD.

7.4 Results

The individual subjects' CP and W' data are presented in Table 7.0. All models were highly linear (r^2 =0.99-1.0; mean CP = 8.1 ± 2.79 W, S.E.E = 0.01 – 0.28 W; mean W′ = 1.14 ± 0.93 kJ, S.E.E = 0.005 – 0.207 kJ). Representative subject data is reported in Figure 7.1.

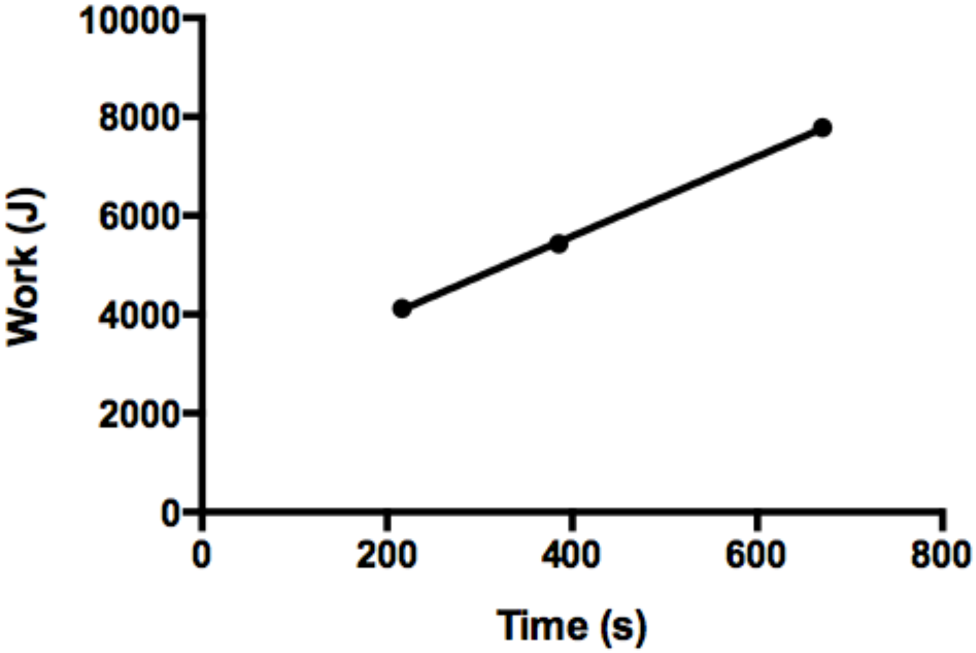


Figure 7.1: *Representative subject data used in the calculation of CP and W'. (CP = 8.1 ± 0.13 W; W' = 2.35 ± 0.61 kJ).*

Table 7.0: *Individual subject data for CP, W', and the $T_{1/2}$ of recovery for W' and [PCr]. Note that [PCr] recovers almost 6-fold faster than the W'.*

Subject	**CP (W)**	**W' (kJ)**	**W' $T_{1/2}$ (s)**	**[PCr] $T_{1/2}$ (s)**
1	13.3	3.15	201	47
2	6.05	0.61	184	30
3	6.03	0.83	364	51
4	8.07	2.35	134	26
5	11.5	0.65	409	32
6	5.15	0.78	270	42
7	7.66	0.47	173	26
8	10.7	0.46	229	29
9	5.53	1.58	426	77
10	7.14	0.56	172	34
Mean ± SD	8.1 ± 2.79	1.14 ± 0.93	232 ± 108	39 ± 16

Recovery of the W′ after B_C as evidenced by work capacity in B_E was highly variable among subjects ($t_{\frac{1}{2}}$ = 135 s – 426 s). The $t_{\frac{1}{2}}$ of the group mean W′ recovery relationship ($W'_{t\frac{1}{2}}$) was 232 ± 108 s. The group mean recovery time course was best represented by a linear function with respect to time ($r = 0.99$, $p < 0.01$) (Figure 7.2). Because the shortest recovery period was 60 s, it was not possible to properly characterize the early kinetics of the recovery. However, fully 57% of the W′ recovered by 60 s, reaching 96% recovery by 420 s. The W′ recovery data closely correlated with model predictions (Eq.4, $r = 0.97$, $p < 0.05$), and the t-test did not indicate any significant difference between the model predictions and the observed W′ recovery.

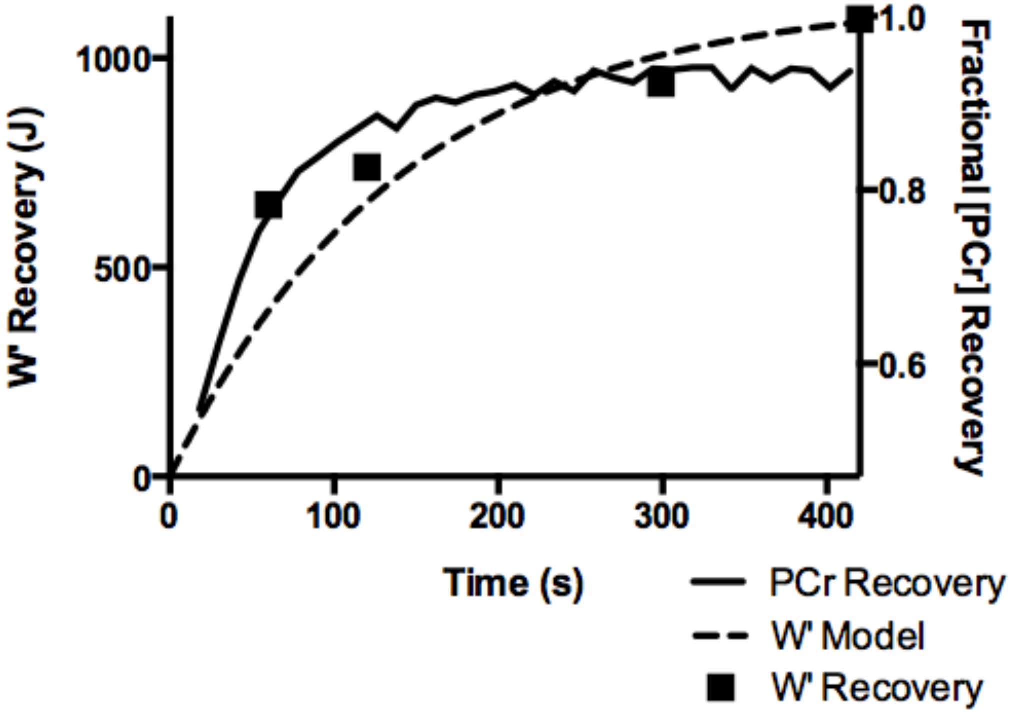


Figure 7.2: *Group mean recovery of [PCr], W' and modelled W'. Note that the W' model was not fitted to these data, but rather was determined directly from* D_{CP} *(known) and the subject's* W'_o *(independently estimated). T-tests did not indicate a significant difference between W' recovery and the W' model at any time point. Error bars are omitted for clarity.*

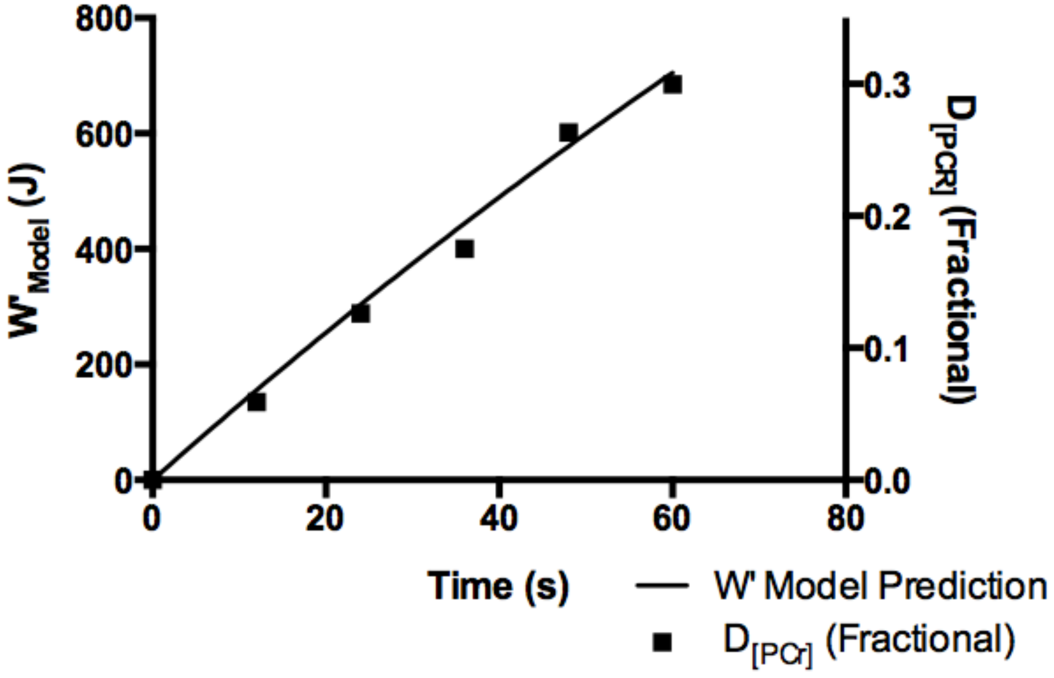


Figure 7.3: *Recovery of mean* $D_{[PCr]}$ *(fractional) and modelled W' for a representative subject during the first 60 s of recovery. Note the high degree of correlation (r =0.99, p <0.0001).*

[PCr], [P_i] and pH at exhaustion during B_C were not significantly different from those measured during B_E in any experimental condition ($p > 0.05$). [PCr] recovery after B_C was well fit by a single exponential (r^2=0.99), with a $t_{\frac{1}{2}} = 39$ s ($\tau = 57$ s); however, there was no correlation between the $\tau_{[PCr]}$ and the interpolated $\tau_{W'}$ values ($r = 0.38$, $p > 0.05$). In contrast, the recovery of $D_{[PCR]}$ was closely correlated with model predictions for W′ recovery ($r = 0.99$, $p < 0.01$) (Figures 7.3 and 7.4a+b) whereas the correlation between $D_{[PCR]}$ and actual recovery of the W′ approached but did not reach statistical significance ($r = 0.93$, $p = 0.06$).

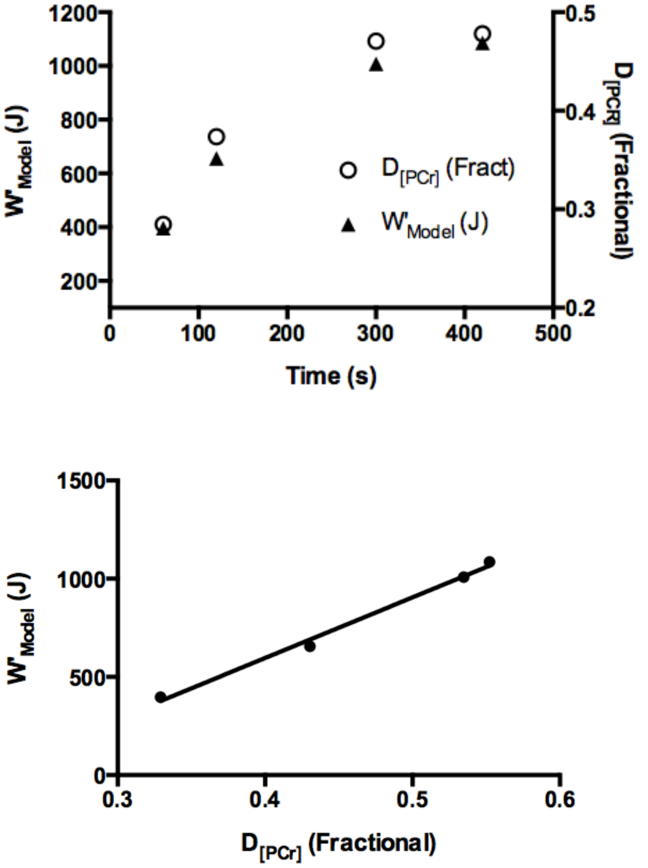


Figure 7.4a and 7.4b: *Relationship between group mean model-predicted W′ recovery (triangles) and the difference between [PCr] (circles) at the beginning and end of* B_E*. The two quantities are highly correlated (*$r = 0.99$*,* $p < 0.01$*).*

There was no correlation between the magnitude of the W′ and pH at exhaustion in either B_C or B_E, nor any relationship between the recovery of pH between the first and second bouts of exercise and the recovery of the W′. B_C exhibited a slightly lower end exercise pH than B_E ($p < 0.05$). No apparent relationship between pH, change in pH at exhaustion or minimum pH and carnosine concentration was found. However, nonlinear regression revealed an inverse curvilinear relationship between carnosine concentration and the W′ $t_{1/2}$ ($r^2 = 0.55$; Figure 7.5). There appeared to be a single outlier, which lowered the apparent strength of this relationship. Exclusion of this subject raised the r^2 to 0.80.

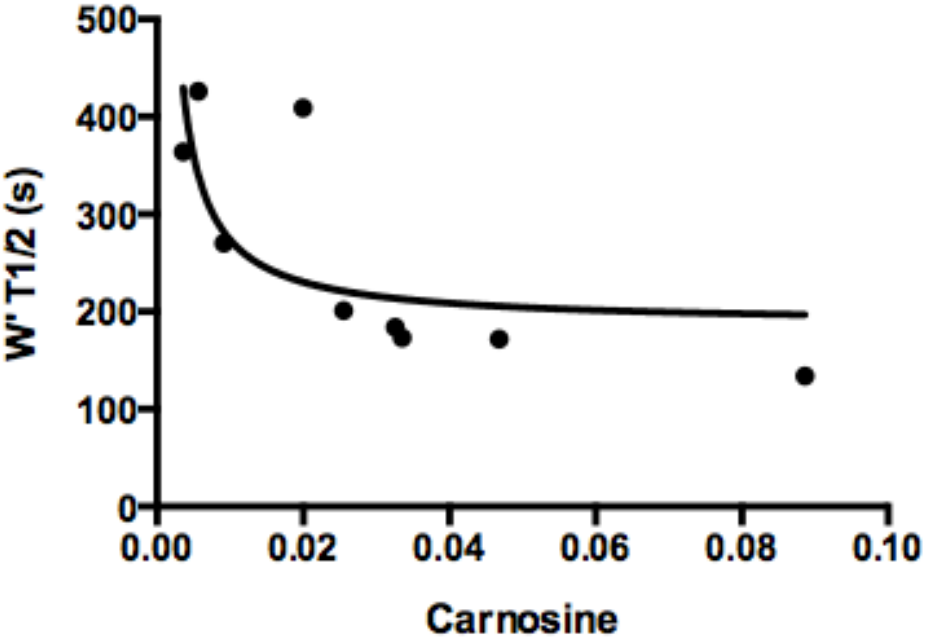


Figure 7.5: *Relationship between* $W'_{t\frac{1}{2}}$ *and carnosine concentration. Note that the curve is likely overleveraged by a single outlier.*

7.5 Discussion

We report four novel findings in this study. First, recovery of the W′ appears to be more linear in isolated leg extension exercise than it does in whole body exercise (84, 206, 208) (Figure 7.2). However, the time course of the recovery maintains good agreement with expected kinetics. Second, the recovery of [PCr] appeared faster than the W′ (Figure 7.2). Third, we found that recovery of $D_{[PCr]}$ was directly correlated with the theoretical W′ recovery predicted by Eq. 4 (Figure 7.3 and 7.4a+b). Finally, there appears to be a curvilinear relationship between the rate of recovery of the W′ and muscle carnosine content (Figure 7.5).

In contrast to the observations made for large muscle mass exercise (84), the recovery of the W′ for small muscle mass knee extensor exercise appears to be linear over the observed time points (Figure 7.2). This observation is perhaps due, in part, to the unusual exercise modality (e.g. some subjects stopping exercise due to discomfort rather than true exhaustion). A linear recovery would be unusual for a biological process, and this finding should be interpreted with caution. However, we noted that the subjects most experienced in this exercise modality and who have participated in several studies in our laboratory showed the most linear recovery kinetics, and none of the subjects exhibited clearly curvilinear kinetics over the time points studied.

We found strong correlations between our kinetic model of the W′ and observed recovery of the W′ when applied to the group data (Figure 7.2), and to the individual subject data sets, with the majority (6 of 10) achieving statistical significance. Moreover, paired t-tests did not indicate a significant difference between model predictions and observed W′

recovery. This is an important finding, because the novel derivation of the W'_{BAL} model presented here is not fitted to the data per se. Rather, the parameters are either a direct result of the experimental design (D_{CP}) or are estimated independently (W_o'). The agreement between the data and the model supports this model enhancement. The group average $W'_{t\frac{1}{2}}$ of 232 s corresponds to an interpolated $\tau_{W'}$ of 334 s, and is compatible with results reported in whole body exercise by Ferguson et al. ($W'_{t\frac{1}{2}}$ = 234 s, interpolated $\tau_{W'}$ of 334 s) (84), and Skiba et al. ($\tau_{W'}$ = 377 s for recovery at 20 W) (206).

It is important to note that even a strictly linear recovery would not preclude the pronounced curvilinear kinetics previously reported in whole-body exercise. We can be relatively certain that the present results relate predominantly to *rectus femoris*, owing to our use of a small (6 cm) receiving coil. Mathematically, if other isolated muscles demonstrate a similarly linear recovery pattern, and each is responsible for some portion of the total W′ observed in whole-body exercise, the sum of these recovery functions will produce a curvilinear relationship (Figure 7.6; Appendix 2). Notably, James and Green (116) have developed an alternative power-duration construct for cycling exercise that similarly relies on the sum of the power production of individual motor units.

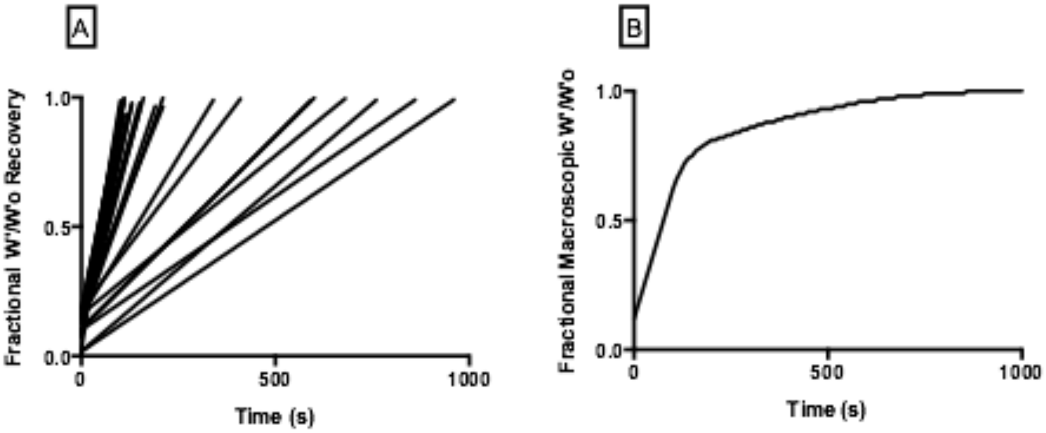


Figure 7.6: *'Microscopic' W′ recoveries that are linear in nature (Panel A) will sum to form a curvilinear function, with the precise shape of the curve determined by the recoveries of the individual parts (Panel B). The linear components could be representative of individual motor units or muscle bellies.*

Strong relationships have been noted between depletion of the W′ and the attainment of $\dot{V}O_2$max in both intermittent (60, 208) and continuous high-intensity cycling exercise (173, 185, 244). Moreover, it has been observed that the depletion of the W′ in single-leg extension exercise elicits consistently low measures of [PCr] both in the present and other studies (62, 63, 134). We were therefore surprised that there was not a more robust relationship between the $\tau_{[PCr]}$ and the interpolated $\tau_{W'}$ values. This is likely due to the fact that the first observed W′ recovery time point is not recorded until 60s, by which time close to 70% of the [PCr] recovery had occurred. At that point, relatively large changes in $\tau_{[PCr]}$ would yield relatively small absolute changes in the shape of the final part of the curve. We do note excellent correlation ($r = 0.99$, $p < 0.01$) between the recovery of the $D_{[PCr]}$ and the expected recovery of the W′ in our model (Figure 7.3 and 7.4a+b). This strengthens previous work from our laboratory indicating that depletion of the W′ is directly correlated with D_{VO2} (208). Collectively, these data suggest that the both the depletion *and* recovery of the W′ is directly related to the 'oxidative reserve' of the muscle, i.e. the D_{VO2} or the $D_{[PCr]}$, representing the difference between the present oxidative metabolic rate and the maximum possible.

A limitation of the present study is that calibrated ^{31}P-MRS was not used, and thus we do not have *stoichiometric* data. We have observed stronger kinetic relationships in whole-body exercise using absolute rather than relative units for $\dot{V}O_2$ and W′. It is possible that a similar situation would apply here. However, there are reasons to question the extent to which [PCr] can define the power – duration relationship. For example, it has been reported that subjects may continue to exercise for some time at zero or near-zero [PCr] before complete depletion of the W′ and subsequent exhaustion (c.f. (225) Figure 2). It has also been reported that subjects are able to maintain $\dot{V}O_{2MAX}$ for some time before full depletion of the W′ and exhaustion occurs (34, 190). Our laboratory recently reported that, upon exhaustion at a supra-CP work rate, it is possible to reduce the work rate slightly such that the subject is able to continue to exercise above CP briefly without recovery of [PCr] or pH (63). Coats et al. (66) have reported similar results for whole-body exercise. Consistent with this, the present study suggests the possibility that achieving $\dot{V}O_{2MAX}$ / a lower limiting value of [PCr] may be a *necessary* but not *sufficient* condition for complete depletion of the W′ and concomitant exhaustion.

We found an inverse curvilinear relationship between muscle carnosine content and the $W'_{t\frac{1}{2}}$ (Figure 7.5). However, we were unable to find any correlation between carnosine and minimum pH, pH at exhaustion, the recovery of pH between the first and second bouts of exercise, or change in pH at exhaustion between the first and second work bouts. This is perhaps not surprising given that carnosine may account for < 15% of the buffering capacity in human muscle (15, 111)). Moreover, the role of pH in fatigue has recently been questioned, particularly in studies involving isolated muscle preparations (for review, see (3)). After fatiguing contractions, force is often recovered considerably faster than pH is (17, 54, 198). Similar conclusions may be drawn from in-vivo feline experiments, showing that pH may be lowered as low as 6.3 with less than 10% reduction in tetanic force and no reduction in shortening velocity (2). Indeed, previous work from our laboratory demonstrates a closer relationship between the W′ and a ‘critical’ or limiting minimum for [PCr] than with pH (134). As carnosine appears to be a pleiotropic molecule in the context of skeletal muscle, it is possible that its primary mechanism of action with respect to fatigue is through some process unrelated to pH. For example, carnosine is known to be an important calcium sensitizer (76, 77, 148), having been demonstrated to potentiate force response in both type I and type II muscle fibres (77). As previously noted, carnosine is β-alanine / histidine dipeptide, and there is some literature suggesting that β-alanine supplementation can improve exercise performance (111, 220), perhaps mediated by an increase in muscle carnosine (15, 111). These data therefore suggest new avenues of investigation involving the role of muscle carnosine in shaping the power – duration relationship.

7.6 Conclusion

In conclusion, single-leg extensor exercise demonstrates a recovery pattern of the W′ that bears kinetic similarity to that observed in whole-body exercise, and which exhibits correlation with a model of the W′ derived from kinetic first principles. The model is closely correlated with $D_{[PCR]}$. However, the extent to which the recovery of the W′ can be firmly ascribed to [PCr] recovery remains uncertain. From a practical perspective, the present study reinforces the validity of the recently-developed model for tracking changes in W′ during intermittent (206, 208) and variable power (207) exercise. Moreover, this study presents a mathematical framework that permits the extension of the W'_{BAL} model to almost any muscle group or exercise modality. Importantly, this same framework customises the model to individual subjects on the basis of their respective power-duration curves (i.e. the CP and W′) and the experimental conditions (i.e. the D_{CP}).

7.7 Appendix 1

It is possible to derive the equation presented by Skiba et al. (206) from first principles.

Here the *W'* is conceptualized in the framework of chemical kinetics. During periods of exertion above critical power (*CP*), *W'* is depleted at a rate directly proportional to the difference between the power output and *CP*.

$$\frac{dW'}{dt} = -(P - CP)$$ **Eq. 7.4**

This first-order linear differential equation can be solved for a segment of time from *u* to *t* in which *P* exceeds *CP*, such that the amount of *W'* remaining, W'(t), is calculated as follows:

$$W'(t) = W'(u) - (P - CP)(t - u)$$ **Eq. 7.5**

During bouts of recovery in which *P* is less than *CP*, the rate of change of *W'* depends on the amount of *W'* remaining (i.e., recovery slows as *W'* approaches the initial *W', W_o'*) and the power output relative to CP.

$$\frac{dW'}{dt} = \left(1 - \frac{W'}{W'_o}\right)(CP - P)$$ **Eq. 7.6**

The first-order differential equation is solved using standard methods as follows.

$$D_{CP} = CP - P$$
$$\int \frac{dW'}{\left(1 - \frac{W'}{W'_o}\right)} = \int D_{CP}\, dt$$ **Eq. 7.7**

The integral is solved using the substitution rule. Note also that *P* is considered constant with respect to time, such that D_{CP} is constant.

$$\ln\left(1 - \frac{W'(t)}{W'_o}\right) = \frac{D_{CP}}{-W'_o} t + const$$ **Eq. 7.8**

For any time = *u* that follows the expenditure of *W'*, *W'(t) = W'(u)*, which by definition is less than W_o'. We substitute these values into the equation, and solve algebraically for *W'(t)* to obtain the final solution:

$$W'(t) = W'_o - \left(W'_o - W'(u)\right) e^{-\frac{D_{CP}}{W'_o}(t-u)}$$ **Eq. 7.9**

It is possible to analyse the special case of a single segment of time in which the athlete exercises above *CP*, such that the initial value for $W'(t) = W_o'$. The recovery after such a bout can be modelled using the following equation:

$$W'(t) = W'_o - W'_{\exp} e^{-D_{CP} t / W_o}$$ **Eq. 7.9.1**

where W'_{exp} is the *W'* expended during the prior segment in which $P > CP$.

To calculate the time course of *W'* for an entire power file, we compute *W'* depletion for each segment of the power time course in which P > CP and W' recovery when P < CP.

7.8 Appendix 2

Model of W' recharge kinetics:

Here the notion that a number of linearly recovering entities (e.g., different synergistic muscles or individual muscle fibres or groups of fibres) sum to form the apparent nonlinear macroscopic recovery of a larger system (a muscle or muscle group) was tested.

For the purposes of the simulation, it was assumed that the macroscopic W' recharge rate was solely a function of the difference between CP and power output, and that 0 <= W' <= W_o', where W_o' represents the fully charged W' at rest. The macroscopic W' was the arithmetic sum of multiple "microscopic" W', one for each component of the system (Figure 7.3A). The microscopic W' recharge rate for a given component was a function of the amount of the total energy, (CP-P)*t, that it drew. This was defined as the fractional recharge, f_i. Different components of the muscle (fibres) or synergistic muscle group (individual muscles) in question may have different W_o' and f_i values, with the properties of the distributions of these values determining the macroscopic W' properties (Figure 7.3B).

$$\frac{W'(t)}{W'_o} = \left(1 - \frac{W'_{\exp}}{W'_o}\right) + \frac{(CP-P)t}{W'_o}, W' \geq 0 \qquad \textbf{Eq. 7.9.2}$$

$$\frac{W'_i(t)}{W'_{oi}} = \left(1 - \frac{W'_{\exp i}}{W'_{oi}}\right) + \frac{f_i(CP-P)t}{W'_{oi}}, W'_i \geq 0, 0 \leq f_i \leq 1, \sum_{i=1}^{n} k_i = 1 \qquad \textbf{Eq. 7.9.3}$$

Chapter 8: General Discussion and Conclusion

Since A.V. Hill's seminal work (105)on the curvilinear relationship between velocity and time in human athletic records, there has been considerable advancement with respect to the mathematics of human performance. Despite this, there has been relatively little work in the area of the mathematics of intermittent CP models (60, 168). The present work developed and tested a novel tool to aid in the understanding of intermittent exercise, and provided important insights into the physiological mechanisms underlying the CP model.

8.0 Research questions addressed

The present work addressed several novel questions.

1) Study 1 (Chapter 4)
   a. Is it is possible to calculate the balance of W′ remaining during intermittent exercise by integrating the amount of W′ expended, which recovers exponentially when the power output falls below CP?
   b. Is the rate of recovery of the W′ during intermittent exercise curvilinearly related to the difference between recovery power and CP?
   c. Is the depletion of the W′ during intermittent exercise correlated with the rise in $\dot{V}O_2$ noted during intermittent exercise in the severe domain?
2) Study 2 (Chapter 5)
   a. Is the W'_{BAL} model robust to variations in work or recovery duration?

   b. Is the amount of W′ remaining after a period of intermittent exercise correlated with the difference between the $\dot{V}O_2$ at that time and $\dot{V}O_{2MAX}$?

3) Study 3 (Chapter 6)
   a. Is the W'_{BAL} model able to accurately predict complete depletion of the W′ and concomitant exhaustion during stochastic exercise?

4) Study 4 (Chapter 7)
   a. Is the W'_{BAL} model transferrable to small muscle mass exercise?
   b. Does the recovery of the W′ correlate with the recovery of intramuscular [PCr], pH or [P_i] as assessed by ^{31}P-MRS?

8.1 Summary of the main findings

Study 1 (Chapter 4) detailed the development of the novel W'_{BAL} model, which describes the discharge and reconstitution of the W′ during intermittent exercise (206). It produced two important findings. Firstly, the data indicated a temporal correlation between the discharge of the W′ and the progressive loss of efficiency noted during intermittent exercise above CP, which has important mechanistic implications. Secondly, it showed a highly predictable change in the time constant of reconstitution of the W′ as a function of the difference between recovery power output and the CP. Together, these findings imply a very particular mathematical framework to aid in the search for and understanding of the underlying physiology of the W′. The findings are also compatible with the notion of a multi-compartment model of the W′, notionally equivalent to the type I and type II fibre pools.

Study 2 (Chapter 5) advanced both the mechanics and the physiological bases of the W'_{BAL} model presented in Study I (208). By varying work or recovery duration, it was observed that the time constant of W′ recovery decreased as work interval was shortened. However, it was also observed that the time constant of W′ recovery could be shortened further by reducing recovery duration in the setting of a sufficiently short work duration. Finally, it was noted that the W′ available for constant work rate exercise immediately following a period of intermittent exercise was linearly correlated with the difference between $\dot{V}O_2$ at the start of CWR and $\dot{V}O_{2MAX}$ (r = 0.79, p < 0.01). Collectively, these data imply the relevance of both the accumulation and depletion hypotheses of the W′. Despite the variability in $\tau_{W'}$, the W'_{BAL} model was accurate to within -1.6 ± 1.1 kJ when averaged across all conditions.

Study 3 (Chapter 6) demonstrated the practical application of the W'_{BAL} model to the performance of a population of well-trained triathletes (207). Using ROC analysis, it was found that the "threshold" for exhaustion using the W'_{BAL} model is set to 1.5 kJ, 80% of athletes will be appropriately classified as exhausted and 88% appropriately classified as non-exhausted. Given anecdotal reports from a number of athletes (who were not part of study III) indicating feelings of extreme fatigue at $W'_{BAL} < 1.5$ kJ, it is both statistically and practically defensible to discourage athletes from proceeding below a W'_{BAL} of 1.5 kJ (typically less than 10% of the W′) if they wish to avoid premature exhaustion during training or competition. Importantly, this chapter demonstrates the utility of the model outside the range duty cycle durations and intensities studied in Chapters 4 and 5.

Study 4 (Chapter 7) represented an attempt to use a modified W'_{BAL} model derived from first principles, and in so doing make the model transferrable from large muscle mass to small muscle mass exercise. This information was then used to identify particular intramuscular determinants of the ability to recover the W′. The W'_{BAL} model correlated closely with the difference between the minimum [PCr] at the end of one work bout and the [PCr] at the start of the next (the $D_{[PCr]}$). However, the measured W′ appeared to recover considerably faster than the model prediction over the first minute. Moreover, the observed W′ recovery appeared to be highly linear during small muscle mass exercise. Finally, it was possible to develop a mathematical construct demonstrating that multiple linear recoveries would likely sum to a macroscopic curvilinear pattern, indicating the observations of curvilinear recovery in whole body exercise and linear recovery during small muscle mass exercise are not mutually exclusive.

8.2 Balancing mathematics and physiology: Limitations of the present work and questions arising

Einstein famously said, “As far as the laws of mathematics refer to reality, they are not certain, and as far as they are certain, they do not refer to reality (78).” This work represents an attempt to mathematically codify a complex and inherently noisy biological system. This is a risky enterprise and it is advisable to resist the temptation to over-interpret modelled parameters.

To what extent is the W′ knowable?

The chief limitation of the present work involves the error inherent in measuring the W′, and the implications for correctly calculating the W'_{BAL}. As noted in Chapter 6 (207), the SE for the W′ may be as low as 0.6 to 1 kJ in the best cases (121, 124, 173), but can be as high as ~2 kJ (37). Depending upon the magnitude of the W′, this might represent 10-15%. Part of the issue is that the W'_{BAL} model is inherently deterministic, rather than probabilistic. In other words, the model assumes that the W′ is a knowable quantity and that it is possible to run it down to zero. With this in mind, the ROC analysis in Chapter 6 (207) should remind us that we are always viewing the W′ through a blurry lens, and that it may not be possible to know *exactly* where we are at any given time.

Although athletes, coaching and management staff want accurate and precise measurements of ability, it is important for practitioners to avoid "over-promising and under-delivering". The CP and W'_{BAL} models represent tools to help athletes, coaches and physiologists. These models should not be misinterpreted as any sort of "final word" on ability or exercise tolerance.

Application to stochastic data

Another limitation involves the application of the model to highly stochastic or field data. Although it is possible to obtain excellent results assuming a constant recovery power calculated as the mean of all power values less than CP (e.g. Chapters 4-6)(206-208), Chapter 4 mentions the more mathematically strict possibility of attempting calculation of a new D_{CP} every second. However, that which is mathematically justifiable may not be strictly physiologically appropriate. Biological systems may exhibit varying degrees of inertia that may not be evident without invasive measurement. Indeed, as noted in Chapter 5 (208), work and recovery duration may independently effect recovery kinetics. It is not known how long it may take for any hypothesized change in recovery time constant to be made manifest. As the physiological determinants of the W′ remain uncertain, it would be wise to remain cautious of modifying the equations or approach for the sake of mathematical parsimony.

$\dot{V}O_{2MAX}$ and $\dot{V}O_2$ modelling vs. W′ mathematics and modelling

The present work, among others, may lead us to the conclusion that the W′ may be best defined by the ‘size’ of the severe domain; that is, the ‘space’ between the CP and $\dot{V}O_{2MAX}$ (43, 130, 173, 225, 228). From the perspective of whole body energetics, this makes for a tidy paradigm. While extending these findings, the data presented in Study 4 (Chapter 7) also suggest that there may exist some interesting nuances. The recovery of the W′ appears to correlate closely with $D_{[PCr]}$. However, whilst the model tracked $D_{[PCr]}$ well, the W′ appeared to have a considerably faster early recovery period in single leg extension exercise. This would be consistent with a multi-compartment model of the W′ proposed in Chapter 4.

Fatigue is always multifactorial, and it is unlikely that depletion of the W′ can be ascribed to a single physiological disruption. As noted in Chapter 2, exhaustion of the W′ is associated with demonstrable central fatigue (45), an apparently ‘limiting’ [PCr], pH, and

[P$_i$] (134, 225) as well as the attainment of $\dot{V}O_{2MAX}$ (43, 130, 173, 225, 228). Indeed, all subjects in Chapters 4 and 5 achieved $\dot{V}O_{2MAX}$ at exhaustion. However, the results reported by Chidnok et al. (63) and Coats at al (66) may prove instructive as we search for physiological underpinnings of the W'_{BAL} model. In both of these studies, subjects who reached exhaustion during CWR exercise in the severe domain were able to continue exercising for a (short) period of time if the work rate was reduced to a lower level within the severe domain. This suggests that $\dot{V}O_{2MAX}$ may be a *necessary* but not *sufficient* condition for exhaustion during exercise in the severe domain. However, the observation that exhaustion is at least *coincident* with $\dot{V}O_{2MAX}$ makes for an interesting mathematical test of the CP model.

Let us return to the notion that the W′ may be represented by a volume of liquid in a tank. Since exercise to exhaustion in the severe domain often terminates soon after the attainment of $\dot{V}O_{2MAX}$, one may be tempted to conceptually ‘fill the tank’ with a quantity of PCr or oxygen, i.e. propose a causative relationship.

$\dot{V}O_2$ kinetics have been described by (129):

$$\dot{V}O_2\,(t) = \dot{V}O_{2\text{baseline}} + A_p\left(1 - e^{-\left(\frac{t-TD_p}{\tau_p}\right)}\right) + A_s\left(1 - e^{-\left(\frac{t-TD_s}{\tau_s}\right)}\right) \qquad \textbf{Eq. 8.0}$$

where $\dot{V}O_2$ (t) represents the absolute $\dot{V}O_2$ at a given time t; $\dot{V}O_{2\text{baseline}}$ represents the mean $\dot{V}O_2$ in the baseline period; A_p, TD_p, and τ_p represent the amplitude, time delay, and time constant respectively describing the phase II kinetics. A_s, TD_s, and τ_s represent the amplitude of, time delay before the onset of, and time constant describing the kinetics of the $\dot{V}O_{2sc}$. ([PCr] kinetics may also be described using exponential equations (132, 194), though it is important to remember that these kinetics may be described by alternative models (10)). In addition, although the $\dot{V}O_{2sc}$ is often fit as an exponential, it is not universally recognised to be an exponential process at this time (129, 242). As Whipp et al. have referred to TD_s and τ_s as a “parameters of convenience” (242), they are used here for the purposes of mathematical parsimony, recognising that a slightly different integration would be required should a definitive model of the $\dot{V}O_{2sc}$ be demonstrated in the future.

It may be posited that there should be a relationship between the total volume of oxygen consumed and the W′, e.g., that the area under the CP curve is proportional to a corresponding area under the $\dot{V}O_2$ curve, and any difference (D) between them should be constant. While power output does not appear in the $\dot{V}O_2$ equation noted above, $\dot{V}O_2$ is clearly dependent upon work rate. Assuming that A_p is in some way related to power output (in the simplest case, work rate (P) multiplied by some constant K) and integrating the A_p term[1], the result is:

$$D = ((\tau_p \cdot e^{\left((TD_p - t)TD_s\right)} + t) \cdot \boldsymbol{P} \cdot K) - (t^2 \cdot \frac{CP}{2}) \qquad \textbf{Eq. 8.1}$$

---

[1] A_s is not addressed for the sake of mathematical simplicity, however, it would be treated in exactly the same way as the A_p term, assuming an exponential process.

The important observation with respect to these mathematics is that *P* remains in the equation. Thus, given the $\dot{V}O_2$ kinetic model above, the *total* oxygen consumed remains dependent upon work rate. Therefore, the W′ can have no *fixed* oxygen *(or PCr*) cost. The area under the power-duration curve (the W′) is *not* directly proportional to the area under the $\dot{V}O_2$ curve given the constraints the $\dot{V}O_2$ model (Eq. 8.0) imposes. In this way, *the longer it takes to deplete the W′, the more oxygen (or PCr) will be consumed, irrespective of the fact that the W′ is invariant*. This suggests that the mathematics of the linkage between the W′ and $\dot{V}O_2$ cannot be solely explained by $\dot{V}O_2$ or [PCr] kinetics via a simple tank analogy. Rather, $\dot{V}O_2$ kinetics may represent an overarching paradigm through which we can broadly understand the physiology of human performance (e.g. Figure 2.1). In other words, although subjects achieve $\dot{V}O_{2MAX}$ at approximately the same time as they fully deplete the W′, we must be careful to avoid making overly simplistic mechanistic inferences.

One interpretation of the data presented in Chapter 7 is that the W'_{BAL} model tracks $D_{[PCr]}$ quite closely. As [PCr] resynthesis is an index of aerobic function, it may be that the W'_{BAL} model primarily works because it (in a general sense) reflects the ‘aerobic contribution’ to the W′. With this in mind, perhaps the hypothesized multi-compartment model presented in Chapter 4 (206) could address different physiological mechanisms entirely, not simply different (or *only*) fibre populations or motor units. That is, the model would attempt to account for the physiological heterogeneity of the exercising muscle mass as discussed in Chapter 2) In such a formulation, there may exist additional components that exhibit considerably faster kinetics. One possibility may relate to the carnosine data reported in Study 4 (Chapter 7), where higher carnosine concentrations correlated with faster recovery of the W′. Carnosine functions as a Ca_i sensitizer in type I and type II myocytes, and as an enhancer of Ca-dependent Ca_i release at the SR in type I myocytes. There may, therefore, be a link between calcium transport at the SR and the W′. Though speculative, this would be compatible with previously hypothesized (4, 71, 72) (and recently observed (75)) mechanisms by which the presence of a high level of P_i in fatiguing muscle fibres causes calcium phosphate (CaPi) precipitation in the SR, potentially reducing both the amount of calcium available to initiate contraction and the driving force for calcium out of the SR. In-vitro data suggest that CaP_i precipitate solubilises quickly ($t_{1/2} = 10$ s), though it is unclear how or how much this process might change in-vivo (75). This mechanism may be worthy of investigation as one potential candidate phenomenon related to the relatively rapid early phase of W′ recovery.

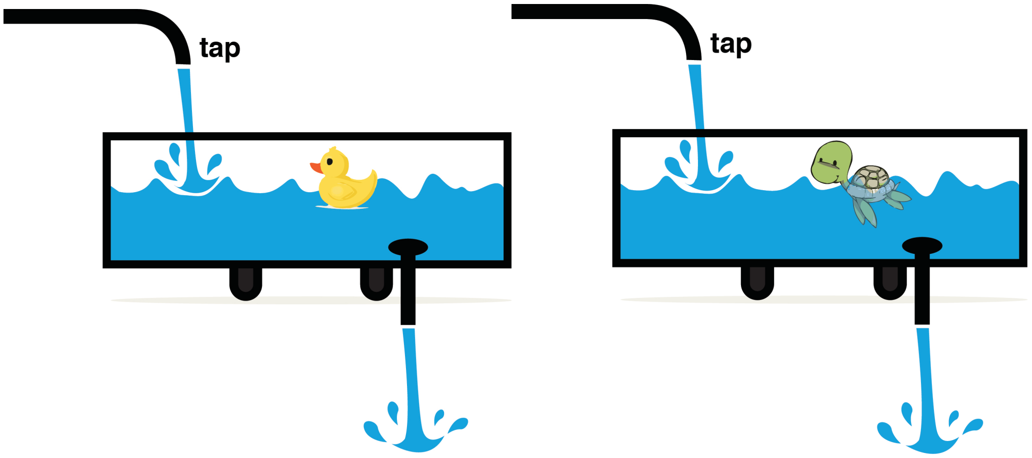


Figure 8.0: *A "multi-tub" model of the W', where the height of the bath toy is representative of the level of some different necessary substrate. When the toy reaches the bottom, exhaustion ensues. It is also possible to view the model in terms of accumulation,*

whereby the level of some metabolite rises until the bath toy reaches the top and exhaustion ensues.

Implications of muscle physiology for W′ mathematics

The present iteration of the W'_{BAL} model as practically applied is solved over the whole of a given exercise session, utilizing a single time constant. This method of solution has an interesting physiological implication that may not be intuitively apparent.

Let us imagine a subject performing intermittent exercise to exhaustion. When our subject is below CP, the subject is reconstituting W′. When the subject is above CP, they are depleting W′. 'Depleting' is defined to mean that the number of joules of W′ available for exercise above CP is decreasing; that is, the W′ is always trending lower from one moment to the next when the subject is above CP. However, as discussed in *General Methods* (Chapter 3), careful consideration of the calculation demonstrates that the W′ is not falling quite as quickly as might be expected. Because a *single* time constant is used to calculate the *integral for the whole exercise session*, there is a tiny amount of "recovery" implied even when the subject is above CP. It is simply "drowned out" because W′ depletion is often happening orders of magnitude more quickly. In other words, depletion of the W′ as modelled by the W'_{BAL} equation during intermittent exercise is not strictly *linear*, but rather very slightly *curvilinear,* even though this may not be apparent to the naked eye. There is a mathematically implicit *microscopic* reconstitution happening during *macroscopic* depletion.

It is reasonable to question whether this model behaviour is physiologically appropriate. From a purely reductionist viewpoint, if one imagines that every motor unit has its own CP and W′, it could be that this (albeit tiny) amount of energy is representative of motor units that have become exhausted, are no longer providing meaningful power, but are being 'recharged' because they are still consuming O_2 and resolving whatever physiological insults they have suffered. There exists the possibility that they could be reactivated later. This schema is not wholly speculative, as there is precedent for intra-exercise recovery of metabolites. For example, in 2004 Krustrup et al. (146) demonstrated that during cycling at 80% of $\dot{V}O_{2MAX}$, analysis of quadriceps biopsy specimens indicated a drop in [PCr] and ATP concentrations in essentially all fibres after 3 and 6 minutes of exercise. However, by 20 minutes there exist populations of both type I and type II fibres that have completely recovered their [PCr] and ATP concentrations, and are most likely not producing force. Further work will be necessary to determine whether these seemingly quiescent fibres can be "re-recruited" or "recycled", and how such behaviour may relate to intermittent exercise performance and the W'_{BAL} model.

Alterative mathematical strategies to calculate W'_{BAL}

One way of resolving the mathematical difficulties noted above is through a recasting of the W'_{BAL} model. It is possible to solve the model using the mathematics presented in Chapter 7, Appendix 2, using a "segmental approach". That is, segments that are above or below CP are analysed independently. When above CP, W′ is depleted in a strictly additive way (i.e. if 200 J are expended the first second and 200 J are expended the next, a total of exactly 400 J have been expended). When below CP, the W′ recovers exponentially. Depletion of the W′ becomes a completely linear enterprise, whilst recovery remains curvilinear.

Chapter 7, Appendix 2 presented the ordinary differential equation (ODE):

$$\frac{dW'}{dt} = \left(1 - \frac{W'}{W_o}\right)(CP - P),\ CP > P$$

Eq. 8.2

This resulted in the final analogous form of the W′$_{BAL}$ model:

$$W'(t) = W_o - \left(W_o - W'(u)\right)e^{-D_{CP}/W_o (t-u)}$$

Eq. 8.3

If one attempts to use this equation to analyse whole-body intermittent exercise data, a $\tau_{W'}$ faster than that which can be interpolated from the $t_{1/2}$ reported by Ferguson et al. (84) or those reported in Chapters 4 or 5 for 20 W recovery (206, 208) is recovered. For example, utilizing the group average data of Ferguson et al. (CP = 213 and W′ = 21.6 kJ, respectively), it is possible to compare model predictions of W′ remaining to the amount of W′ remaining actually measured by Ferguson et al. (84) (Table 8.0).

Table 8.0: *Comparison between predicted and measured W′ utilizing the data reported by Ferguson et al. (84) and equation 8.3.*

Time (s)	**W′ Predicted (kJ)**	**W′ Actual (kJ)**
120	14.1	7.8
360	21	14.1
900	21.6	18.5

As reported in Chapter 4, the time course of recovery as reported by Ferguson et al. (84) would yield an apparent $\tau_{W'}$ of 336 s. However, the above model predictions imply a three-fold faster recovery, with $\tau_{W'}$ equal to 112 s. Despite this, this methodology *also* predicts a more rapid fatigue during intermittent exercise in some cases. For example, it is possible to compare the W'_{BAL} model to equation 8.3 (the $W'_{BAL\text{-}ODE}$ model) for a subject from the study presented Chapter 4 (Figure 8.1). The subject performed work intervals in the severe domain for 60 s interspersed with 30s recovery at 20 W. The $W'_{BAL\text{-}ODE}$ model predicts exhaustion approximately 300 s sooner than the W'_{BAL} model.

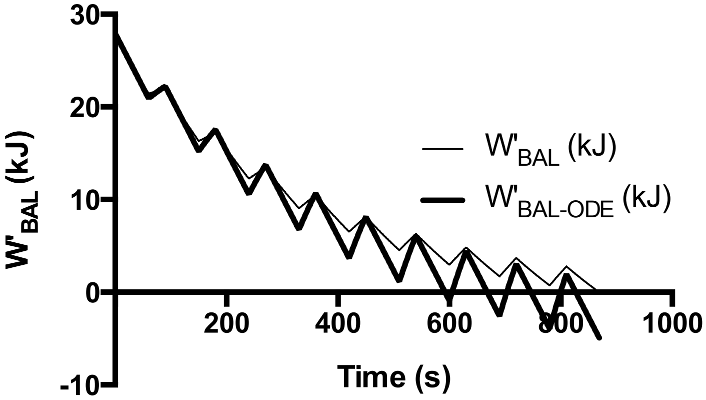


Figure 8.1: *Comparison of W'*$_{BAL}$ *model as tested in Chapters 4 and 5 to the ODE form presented in equation 8.3, which adds a constant K (W'*$_{BAL\text{-}ODE}$*). Subject performed a series of square wave intervals, with a 60s work interval at 328W, and a 30s recovery interval at 20W, until exhaustion. Note that both models predict a similar W'*$_{BAL}$ *at the end of each recovery interval, but that the W'*$_{BAL\text{-}ODE}$ *model predicts a W'*$_{BAL}$ *of 0 approximately 300 s early.*

Thus, solving the problem of a slightly curvilinear depletion of the W′ reveals a potentially larger problem. However, there exists a possible solution. In Chapter 5, speculation was offered that the under-prediction evident in the W′$_{BAL}$ model could be the result of a transient increase in CP during intermittent exercise, i.e. an unexpected increase in D_{CP}. Accepting that premise, it is possible to express the hypothesis mathematically using a constant *K* that is sensitive to the characteristics of the recovery (i.e. duration or other factors).

$$\frac{dW'}{dt} = \left(1 - \frac{W'}{W_o}\right) K\left(CP - P\right),\ CP > P \qquad \textbf{Eq. 8.4}$$

This has the effect of providing the final form:

$$W'(t) = W_o - \left(W_o - W'(u)\right)e^{-{KD_{CP}}/{W_o}(t-u)} \qquad \textbf{Eq. 8.5}$$

This new solution form has a most interesting effect. Note the location of *K* in the exponent of the final equation. It suggests that *recovery itself should exhibit an "efficiency"* that could be accounted for by adjusting the apparent D_{CP}. Caution is required when introducing new model components to improve data fit. Irrespective of mathematical expedience, experimentation will be required in order to discover if there exists an *observable physiological justification* for making such a modification in the future. This said, by applying equation 8.5 to some of the data available from Chapter 4, some interesting behaviour may be observed.

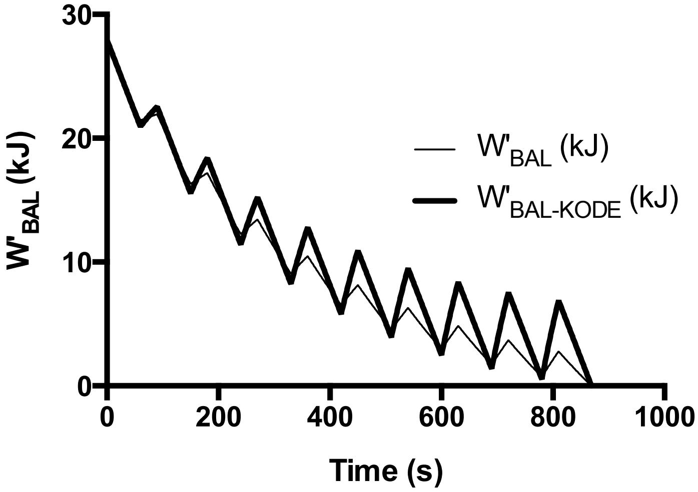


Figure 8.2: *Comparison of W'_{BAL} model as tested in Chapters 4 and 5 to the ODE form*

presented in equation 8.5, which adds a constant K ($W'_{BAL-KODE}$). Subject performed a series of square wave intervals, with a 60s work interval at 328W, and a 30s recovery interval at 20W, until exhaustion. Note that both models predict a similiar W'_{BAL} at the end of each work interval.

Figure 8.2 depicts a comparison of the W'_{BAL} model to equation 8.5 (the $W'_{BAL-KODE}$ model) for the same subject from the study presented Chapter 4. The subject performed work intervals in the severe domain for 60 s interspersed with 30s recovery at 20 W. In order for the $W'_{BAL-ODE}$ model to predict exhaustion at the same time as the W'_{BAL} model, the proposed constant *K* would need to be set to 1.28. That is, the solution implies a 28% functional increase in CP during the intermittent exercise protocol. Notably, this is precisely the group average increase in CP reported by Soares-Caldeira et al. (213) for intermittent exercise utilizing a recovery duration of 30 s (e.g. the same recovery duration as the subject modelled above). This is also compatible with earlier observations by Turner et al., Essén, and Astrand et al. (11, 12, 64, 81, 219) that suggest intermittent exercise may represent a lower 'functional' intensity. This observation encourages additional study and comparison of the $W'_{BAL-KODE,}$ $W'_{BAL-ODE}$ and W'_{BAL} equation forms.

Tank analogies vs. alternative paradigms

As discussed in the Introduction (Chapter 2), a simple tank model predicts a linear, rather than curvilinear recovery of the W′. The present work and others demonstrate that recovery is very likely curvilinear in nature during whole body exercise (84, 206-208), though possibly more linear in small muscle mass exercise (Chapter 7). It has been demonstrated mathematically that these behaviours are not *necessarily* mutually exclusive. The re-casting of the model presented in Chapter 7, Appendix 1 dictates that $\tau_{W'}$ is dependent upon the state of the W′ at any given instant. In other words, the lower the W′ falls, the faster the recovery, which slows as the recovery proceeds. Yet, however appealing or tidy the mathematics appear, it is necessary to carefully consider the physiology implied by these models.

Recall that the re-casting of the equation in Chapter 7, Appendix 1 is derived from the mathematics of chemical kinetics. That is, the W′ is considered to be analogous to a chemical reactant in a vessel. By its nature, such a model assumes both free diffusion and equal distribution of reactants; that is, the probability of reactant interaction is homogenous throughout the volume. As discussed previously both in this chapter and in Chapter 2, the exercising limb, and even the constituent muscles and motor units, are spatially and physiologically heterogeneous structures (125, 150, 151, 153, 181, 196, 197, 200). For example, there is a lack of free diffusion (due to membranes and tissue planes), as well as unequal distribution (i.e. discrete organelles). There exist differences in perfusion of different fibre types (9, 204), and uneven [PCr] depletion amongst fibre types (28, 136).

It is mathematically *possible* to account for spatially heterogeneous reactions. However, any such treatment necessarily involves partial differential equations with complex solution forms (Clarke DC, *private communication*). Irrespective of the quantitative difficulties involved, any such framework would remain unacceptably speculative, since so little is known about the precise nature and localization of the determinants of the W′. However, some elements of the above noted heterogeneity may be more easily considered within a broader anatomical context utilizing modalities such as multichannel NIRS (139), MRI (79) or magnetic resonance spectroscopy (55, 140, 193). Given recent findings that link exercise above CP with dramatic changes in muscle perfusion in a rat model (68), it may also be possible to re-cast the problem in terms of the dynamics of perfusion.

Linear recovery during small muscle mass exercise

Chapter 7 raises the possibility (and problem) of linear recovery of the W′ during small muscle mass exercise, at least over the time points studied in the present work. Strict linearity is unexpected for a biological system. Moreover, the positive y-intercepts noted for recovery graphs plotted for each of the subjects in Chapter 7 imply that either the subjects instantaneously recovered some portion of the W′, or that all of them ceased exercise before completely depleting the W′. Neither of these explanations is very plausible. Rather, the most straightforward explanation is that the relationship between W′ recovery and time is indeed curvilinear, but that much of the curve exists before the first time point (60 s). Thus, it will be necessary to replicate the experiment in Chapter 7, with recovery time points at 10-15 s intervals during the first minute in order to better characterise the early kinetics.

Metabolite accumulation vs. substrate depletion and implications for regulation of the W′

The results of Study II (Chapter 5) indicate that a physiologically correct model may need to address both depletion of substrates and accumulation of metabolites. Although carbohydrate stores were not directly addressed in this work, it was noted in Chapter 2 that there exists some evidence that glycogen stores may play some part in defining the W′ (162). (However, forthcoming unpublished work from our laboratory may provide some challenge to this point). Early biopsy work by Essén (80) demonstrated that intermittent exercise exhibits a relative glycogen sparing effect in comparison to maximal work rate exercise of similar intensity (267 vs. 273 W, respectively). This dovetails with other work indicating that the overall metabolic response to intermittent exercise is more similar to CWR exercise at a lower intensity than it is to CWR exercise at an intensity matching the intermittent work rate (i.e the work rate of the 'on' interval) (12, 82).

In particular, it is worthwhile to carefully consider the results of Study II in light of Essén et al. (82), who reported that the metabolic response to intermittent exercise was similar to that of CWR exercise of the same *mean* power output and oxygen uptake. Study II in the present work demonstrated that intermittent exercise yielding the *same* mean power output (the 60-30 and 20-10 conditions) resulted in substantially *different* times to exhaustion when the subject switched to CWR exercise (208). This indicates that the situation is likely more complex than a simple consideration of mean power output. It is not unreasonable to hypothesize that, despite an equal mean power output, the two different intermittent exercise conditions resulted in a significantly different metabolic milieu. Study II also showed a direct relationship between D_{VO2} and the W′ available for CWR exercise (208). Thus, much would seem to depend upon the precise makeup of the intermittent exercise session and the way it interrogates skeletal muscle metabolism.

Mean power output cannot be used as an absolute surrogate for the metabolic state of a subject under all conditions.

Of note, there have been more recent developments with respect to the ‘accumulation hypothesis’. In the time since the experiments reported in this thesis were concluded, a study was published which directly addresses the point of circulating metabolites. Johnson et al. (124) demonstrated that prior severe intensity arm cranking exercise reduces the W′ during subsequent leg cycling exercise, without altering the CP. One possible explanation for this is that circulating metabolites have remote effects upon exercising muscle. Indeed, Pollack et al. (182) recently demonstrated that infusion of metabolites at concentrations typically found in resting muscle (pH 7.4 + 300 nMol ATP + 1 mMol lactate) had no discernible effect on perception of muscular fatigue. Similar results were reported for infusion of the individual metabolites. However, as the delivery of the combination of H^+, ATP and lactate were increased, there were dose-dependent increase in feelings of fatigue, and eventually, sensations of pain (182). As noted previously, studies using acetaminophen (154) or opioid analgesics (8) seem to improve exercise performance to some extent. A complete discussion of these data in light of neurological control is beyond the scope of this thesis. However, studying the effects of such interventions on the CP and W′ could yield additional insight into the control mechanisms governing the power-duration relationship.

8.3 Balancing mathematics and physiology: future studies

Exercise intensity

The present work has examined the behaviour of the W'_{BAL} model during intermittent whole body exercise whilst varying recovery power (Chapter 4, (206)), as well as work and recovery durations (Chapter 5, (208)). The next logical experiment involves systematically varying the intensity of the work interval (within the severe domain) during intermittent exercise. Such an experiment is likely to be more complicated than it appears, as it would necessarily involve the determination of the upper limit of the severe domain. This process is not entirely straightforward and involves several potential sources of error (112). However, given the robust behaviour of the W'_{BAL} model during stochastic exercise where subjects often reached 200% or more of CP for brief periods (Chapter 6, (207)), it is possible that $\tau_{W'}$ is relatively insensitive to intensity.

The effects of accumulation on recovery

As noted above, Johnson et al. (124) demonstrated that prior severe intensity arm cranking exercise reduces the W′ during subsequent leg cycling exercise. One explanation for this observation is that concentrations of circulating metabolites may play a part in determining the W′. It would be interesting to look for an independent effect of circulating metabolites on the W'_{BAL} model (i.e. a slowing or speeding of $\tau_{W'}$). This could be accomplished through direct intravenous manipulation of factors implicated in fatigue (i.e. (182)), or through the use of prior exercise with unrelated muscle groups (124). Either methodology would entail certain drawbacks. For example, an intravenous infusion may lack some key metabolite or fatigue mediator. Alternatively, prior exercise with an unrelated muscle group may have associated centrally-mediated effects (e.g.

subject motivation) that could complicate interpretation.

Intensity and time domains

It is important to remember that the CP model is a mathematical representation of a rather tiny slice of a power-duration curve (comprising the region from approximately 2 minutes to 30 minutes) that stretches from just a few seconds in the case of very short events, to 24 hours or more in the case of ultra-endurance cycling events. The model only applies to power outputs above the CP (i.e. power output within the severe domain); it tells us nothing about fatigue below the CP. The mathematics and physiology of locomotor performance in the more extreme ranges (both in terms of intensity and duration) of human endurance may be fertile ground for new discovery. For example, it may be possible to develop a novel construct (one which may include the W'_{BAL} model) to address the relationship between power output and time to exhaustion in a way that spans exercise domains. This would be extremely useful in ultra-endurance events, where an athlete may need to exercise care to avoid fatigue due to W' depletion on a steady climb, intermittently raise power output well into the extreme domain in order to cover attacks, and also guard against fatigue due to other mechanisms during long periods spent in the moderate or heavy domains. One way the problem could be approached might be as a segmental model that attempts to address different physiological systems, notionally similar to that developed by Busso et al. (51), or the multi-tank model proposed earlier. This said, parameterisation of such a model would likely to be extremely difficult, with each new term requiring many additional test points, perhaps so many as to be impractical, depending upon the precise model structure.

Implications for quantifying training stress and longitudinal performance modelling

The two-parameter CP model predicts what an athlete may be capable of in terms of power output at any time *t*, whilst the W'_{BAL} permits the calculation of how much this absolute ability is available at any given time during an exercise session. However, it is well known to any sports fan that an athlete's performance may change on a day-to-day basis. There exists another category of performance model that allows us to relate training stimulus to athlete response. As was briefly mentioned in Chapter I, the most widely known of these constructs is the Banister impulse response (IR) model (18, 20, 86, 169), which quantitatively relates performance ability at a specific time to the cumulative effects of prior training loads (216). The original paper modelled the training and performance of a competitive swimmer (21). Since then, the IR model has been applied to diverse sports such as running (169, 245), swimming (102, 103, 172), cycling (46, 47, 49, 50), triathlon (22, 159), weightlifting (52, 53) and the hammer throw (48).

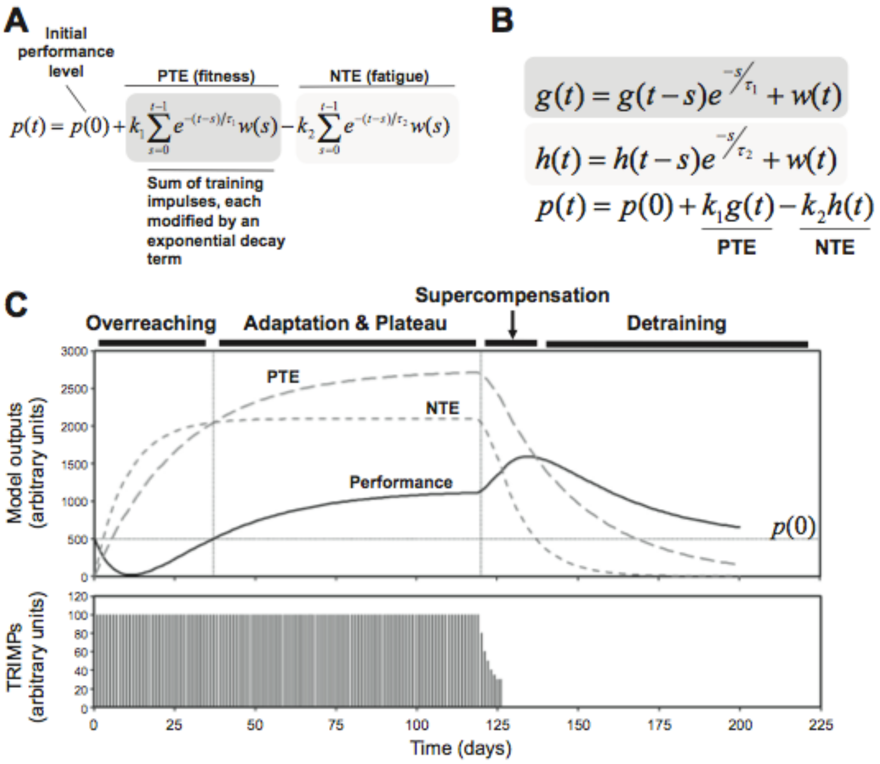
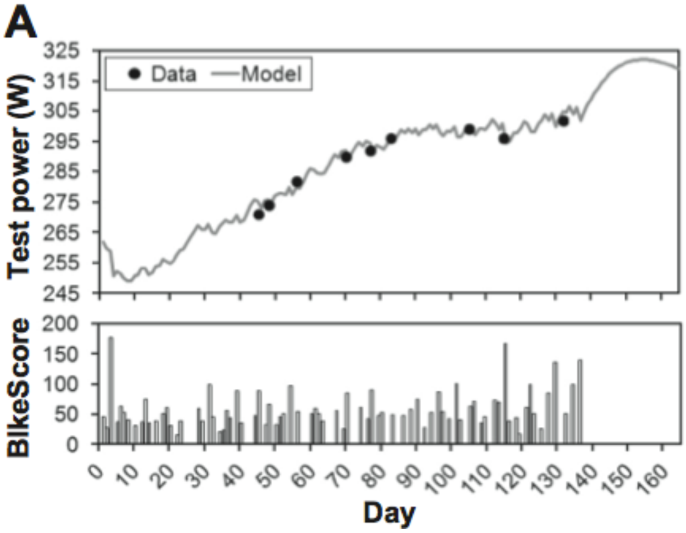


Figure 8.3: *Definition and description of the impulse-response (IR) model. The IR model predicts performance based on the simple premise that it is the sum of base-level performance and positive training effects (PTEs) minus negative training effects (NTEs). Panel A: summation equation form of the IR model. Panel B: recursion equation form of the IR model. This form is most useful for spreadsheet-based calculations. Panel C: the IR model recapitulates the known qualitative features of the training response. In the bottom graph, simulated daily training load was plotted as a function of time. The athlete performed workouts of 100 training impulses (TRIMPs) per day for 120 days. The following 7 days featured a taper in which daily TRIMPs were progressively reduced to 30. Training was ceased thereafter. PTE, NTE, and performance were calculated from the simulated TRIMPs and used the following parameter values: p(0) = 500,* k_1 *= 1,* k_2 *=*

2, $\tau_1 = 27$*, and* $\tau_2 = 10$*. Arbitrary units (AU) were used for p(0),* τ_1*, and* k_2*, whereas* τ_1 *and* τ_2 *were expressed in units of days. Figure taken from (65) with permission.*

The model is comprised of a two-component system in which training is posited to cause both positive and negative effects, respectively attributed to "fitness" or positive training effect (PTE) and "fatigue", or negative training effect (NTE) (Figure 8.3). The equations for each of these two components were of the same form as the equation they had first proposed, and performance was calculated as the difference between the two. Then, further assumptions were specified to describe how performance changed with time. In response to a given training load, the NTE initially outweigh the PTE such that the subsequent performance capacity is decreased. However, the NTE dissipates faster in time than the PTE, such that the PTE eventually outweigh the NTE and performance capacity increases (Figure 8.3). Equation variables are then altered until the resultant performance curve matches the athlete's actual observed performances (Figure 8.4). Based on this relatively straightforward process, the IR model can capture much of the variance in performance data collected over time ($r^2 > 0.90$ in some cases) (46, 169, 245). The present author has used these models to prescribe the training of several athletes, resulting in 4 world-championship titles in duathlon and triathlon, and one world record in triathlon at the Half-Ironman distance (Figure 8.5).



Best-fit parameter values:

Parameter	Value	Units
$p(0)$	262	W
k_1	0.18	W/AU
k_2	0.23	W/AU
τ_1	36	days
τ_2	21	days

Figure 8.4: *Practical implementation of the IR model. Training and performance data were used to fit the IR model for an individual athlete. Here, BikeScore (analogous to TRIMPS or TSS) was the metric used to estimate daily training loads (bottom). Performance data were determined from periodic time trials. The predicted performance [p(t)] was estimated by fitting the five IR model parameters (right) using nonlinear regression implementation). The* r^2 *for the model fit was 0.98. Figure taken from (65) with permission.*

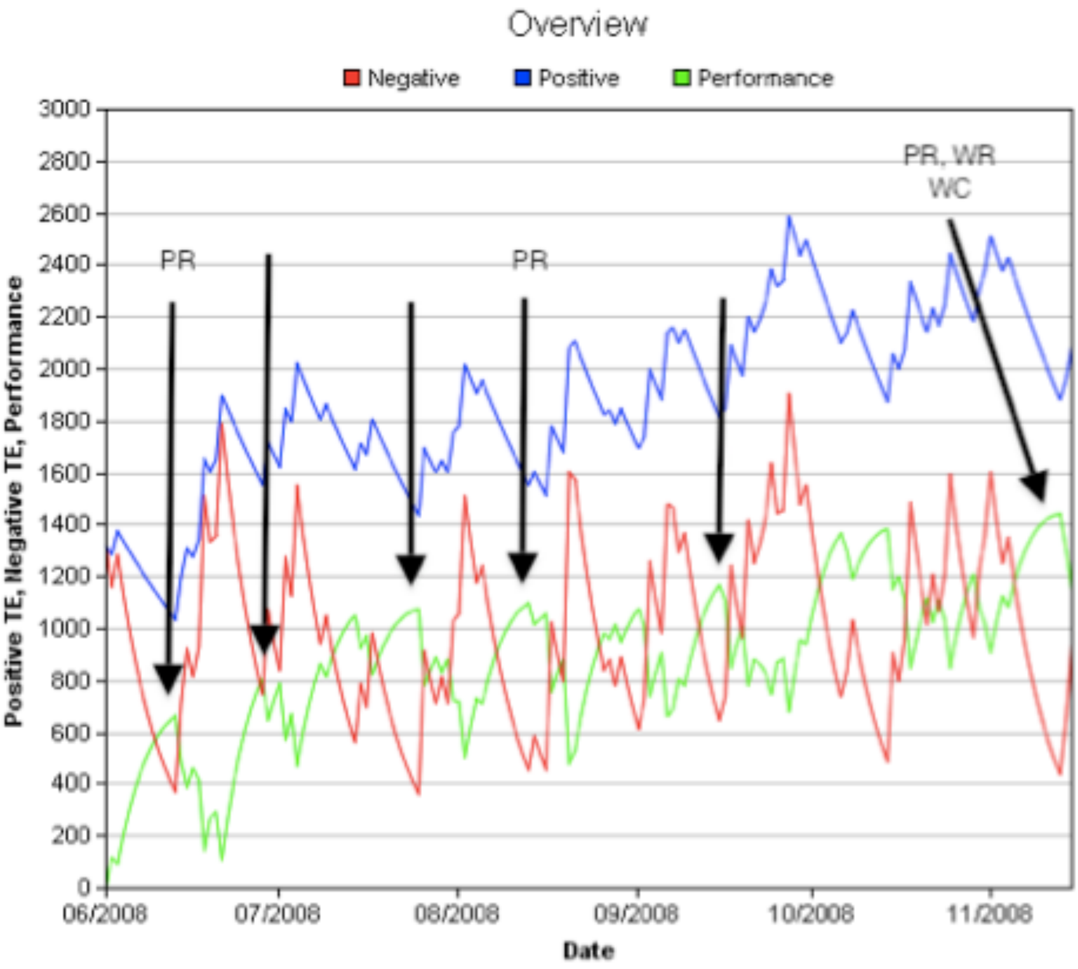


Figure 8.5: *Performance analysis for an elite female triathlete, cycling leg. Red line = NTE or "fatigue"; blue line = PTE or "fitness", green line = performance status. Arrows = podium placing in international competition. PR = personal record, WR = world record, WC = world championship. Training was developed to result in peaks in the performance curve on race days.*

The IR model is founded upon the input of some numerical measure of training load on a day-to-day basis, which may be expressed most simply as the product of intensity and duration. Quantifying duration is simple, but quantifying intensity is more challenging because intensity is a function of work rate and the resultant metabolic stress. This relationship between work rate and adaptive stimulus is nonlinear in nature, and has traditionally been illustrated by the exponential increase of blood lactate as a function of work rate (69, 83). As such, it is a challenge to quantify and compare workouts of differing volumes and intensities in terms of their abilities to induce physiological adaptations.

A number of metrics exist for estimating training load, including session rating of perceived exertion (87), ordinal categorization (171, 195), summated heart rate zone score and excess post-exercise oxygen consumption (39, 123, 216). The best-known system of training quantification, however, is Eric Banister's Training Impulse (TRIMP). Predicated upon heart-rate reserve as a measure of intensity, TRIMP accounts for the observation that higher workloads are more metabolically taxing (exponentially so) than workloads performed for the same duration at lower intensity (19).

$$TRIMP = t \cdot k \cdot FHRR \qquad \textbf{Eq. 8.4}$$

$$FHRR = \frac{HR_{avg} - HR_{rest}}{HR_{max} - HR_{rest}} \qquad \textbf{Eq. 8.5}$$

where *t* = duration of the exercise bout in minutes, *HR* = heart rate (beats per min), *FHRR* = fraction of the heart rate reserve and $k = 0.64e^{1.92 \cdot \mathrm{FHRR}}$ or $0.86e^{1.67 \cdot \mathrm{FHRR}}$ for males or females, respectively.

The reliance of TRIMPS on heart rate is problematic, as heart rate is sensitive to changes in temperature, hydration, and cardiac drift, among other factors. This could lead to assignment of an erroneously high training stress if the subject executed a workout under a substantial thermal stress. It may therefore be helpful to attempt to assign training stress based upon actual mechanical power output. Indeed, a training stress score (TSS) was developed for cycling (5) based upon data output from bicycle power meters. This score is predicated upon a transformed average power of a workout that accounts for the variability of the workout's intensity arising from changes in power output due to hills, wind, drafting, etc. The theoretical physiological cost of the workout is curvilinearly related to intensity using a function based upon lactate accumulation, such that large power outputs induce disproportionately higher physiological stress than lower power outputs. In essence, it represents a TRIMPS based upon work rate, rather than a physiological response (e.g. HR) that may not be wholly related to work rate. A more detailed discussion of these metrics has been reported elsewhere (65).

Because of the close correlation between the relative discharge of the W'_{BAL} model and the rise in $\dot{V}O_2$ reported in Studies 1 and 2 (206, 208), it may be possible to leverage the W'_{BAL} model as a means to objectively and differentially weight exercise. In other words, power output or velocity at any time *t* would be weighted in inverse proportion to the calculated W'_{BAL} at time *t*, i.e. the lower the W'_{BAL}, the higher the physiological stress incurred. This may represent a uniquely customizable way of assigning training stress: it would be dependent upon the subject's personal CP, W′, and recovery characteristics as defined by their W'_{BAL} model. These nascent mathematics may represent fertile ground for future study. It is possible that an improved system of training quantification based upon the W'_{BAL} model presented in this thesis would result in better performance predictions once entered into the IR model.

Direct practical applications of the W'_{BAL} model

Study I (Chapter 4, (206)) raised the possibility that the W'_{BAL} model might be useful in analysing the data of cyclists during competition. This was confirmed by Study III (Chapter 6, (207)). Indeed, both the present author and others have used the W'_{BAL} model to help prepare athletes for competition and analyse subsequent performance in world championship and Olympic competition. This includes the Canadian national squad (178), the Australian Institute of Sport (Martin, D. *private communication*), and British Triathlon (Williams, T. *private communication*). The model has been incorporated into several software systems, including the open-source *Golden Cheetah* cycling analytics package. With time, it may be possible to "crowdsource" large data sets and leverage them to make improvements to the model.

Perhaps more interestingly, there exists the possibility that the model could be used to give real time feedback to an athlete in training and competition. It is not difficult to imagine a graphical interface on a smartphone or GPS unit that resembled a battery, informing an athlete as to the relative state of charge or discharge of the W′. Such technology could fundamentally alter the way athletes approach training and competition, and it will be very interesting to observe how it is disseminated.

Although the application to cycling would seem most natural given the focus of the present work, the Introduction points out that the CP model has been successfully applied to a number of sports, including running (115), swimming (230) and rowing (137). This raises the possibility that the alternative formulation using CS and D′ could be applied to any number of sports, such as soccer, field hockey or lacrosse. The National Basketball Association (NBA) has invested heavily in the *SportsVue* and *Catapult* motion analysis systems. These have been placed in every professional basketball arena in the United States, and provide real-time position and speed telemetry to coaching and management staff on the sidelines. The W'_{BAL} model could provide important information to help determine optimal player substitution strategies or tactical decision-making.

8.4 Conclusions

Irrespective of the difficulties in ascribing discrete *physiology* to the mathematics presented in this work, it is important to remember that the original purpose of the CP - W′ paradigm (and indeed this thesis) was to develop a robust mathematical model of human *performance* (164). In this regard, the W'_{BAL} model developed in Chapter 4 (206) appears to perform admirably. Although some variability was noted with respect to work and recovery duration, Chapter 5 demonstrates that the W'_{BAL} model was accurate to within 1.6 kJ when averaged across all conditions (208). Moreover, Chapter 6 supports similar conclusions when the model is applied to highly stochastic data (207), also indicating that it meets criteria as a diagnostic test of volitional exhaustion. Importantly, it also demonstrated that the W'_{BAL} model is useful outside the duty cycle durations and intensities studied in Chapters 4 and 5. Chapter 7, in concert with Chapters 3 and 4, yield both additional support and new insight into our understanding of the interaction between muscle metabolism, the pulmonary $\dot{V}O_2$ signal, and the power-duration relationship as it relates to intermittent exercise. Whether or not it can be meaningfully improved in the mathematical sense, the W'_{BAL} model represents an important addition to the scientific armamentarium, which may be brought to bear in the struggle to understand the physiology that defines human performance.

Oh me! Oh life! of the questions of these recurring,
Of the endless trains of the faithless, of cities fill'd with the foolish,
Of myself forever reproaching myself, (for who more foolish than I, and who more faithless?)
Of eyes that vainly crave the light, of the objects mean, of the struggle ever renew'd,
Of the poor results of all, of the plodding and sordid crowds I see around me,
Of the empty and useless years of the rest, with the rest me intertwined,
The question, O me! so sad, recurring—What good amid these, O me, O life?

Answer.
That you are here—that life exists and identity,
That the powerful play goes on, and you may contribute a verse.

-Walt Whitman (1819-1892)

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