Published in final edited form as:

Exerc Sport Sci Rev. 2015 April ; 43(2): 65–66. doi:10.1249/JES.0000000000000045.

The Critical Power Framework Provides Novel Insights into Fatigue Mechanisms

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Skeletal muscle fatigue constitutes a keystone concept in physiology and medicine at the very core of human endeavor, quality of life and our very survival, as also noted in the article by Grassi and colleagues in this issue of the *Journal* (3). The elite athlete coordinates superior cardiovascular, metabolic and biomechanical capabilities to stave off fatigue and achieve superb feats of athletic performance. At the other end of the spectrum enhanced fatigability is pathognomonic of pathologies such as heart failure, diabetes, pulmonary and vascular occlusive diseases. Exercise tolerance and the capacity to delay fatigue are improved by exercise training and/or enhanced O₂ delivery.

Resolving the mechanistic bases of fatigue is absolutely central to advancing our understanding of physiological and metabolic control processes and fulfilling the National Institutes of Health mandate to relieve the burden of human suffering. However, this endeavor is complicated by the multivariate definitions of fatigue, its task/intensity/condition specificity and simultaneous participation of multiple intramuscular and central mechanisms. Bergstrom and colleagues (2) pioneering work utilized muscle biopsies to demonstrate that, during prolonged heavy constant-load exercise, fatigue occurred concomitantly with muscle [glycogen] depletion. Exercise and dietary strategies that lowered or raised pre-exercise muscle [glycogen] caused commensurate alterations in time-to-fatigue. Since then the plethora of intra- and extra-muscular mediators proposed includes: increased inorganic phosphate, hydrogen ions, reactive O₂ species, temperature, decreased calcium regulation, [creatine phosphate], sodium/potassium pump function, and blood [glucose] as well as altered central motor drive, sympathetic nervous system function and endocrine control.

Grassi, Rossiter, and Zoladz (3) define fatigue as “a reduction in muscle force or power for a given muscle activation” and exploit the commanding role of O₂ delivery (and O₂ consumption, $\dot{V}\dot{O}_2$) on exercise tolerance (1,5). Notably they emplace their consideration of fatigue within the Critical Power (critical velocity) concept (Figure). This is a crucial perspective because above critical power skeletal muscle metabolic homeostasis cannot be maintained and time-to-exhaustion is predictable from W' (anaerobic work capacity) and the power sustained relative to critical power. Specifically, for all exercise above critical power pulmonary and muscle $\dot{V}\dot{O}_2$ increase to $\dot{V}\dot{O}_{2\max}$ (via $\dot{V}\dot{O}_2$ slow component, decreased muscle efficiency). Muscle [creatine phosphate] decreases and [adenosine diphosphate_{free}], [inosine and adenosine monophosphate], [potassium], [H⁺] and blood/muscle [lactate] all increase inexorably to fatigue/exhaustion (4,6), driving the free energy change of ATP

hydrolysis downwards. Under these circumstances, fatigue/exhaustion does not necessarily occur concomitantly with the achievement of $\dot{V}\text{O}_2\text{max}$ but rather depletion of W' . Grassi and colleagues (3) draw on an eclectic range of animal and human models to illuminate the compelling relationship between the $\dot{V}\text{O}_2$ profile, muscle energetics and the fatigue process(es) above critical power.

For many years the $\dot{V}\text{O}_2$ slow component was tacitly ignored as it simply did not fit with muscle energetics models (4). Grassi et al.'s linking $\dot{V}\text{O}_2$ inextricably with fatigue is a bold and timely move that promises to yield novel insights into the mechanistic bases for fatigue.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure.

Schematic of the Power-time relationship as established from four independent exhausting exercise bouts at constant power or velocity (solid dots). Critical power (CP, or Velocity) is the asymptote for power (actually a finite metabolic rate, $\dot{V}\dot{O}_2$). For exercise >critical power (severe domain) $\dot{V}\dot{O}_2$ and [lactate] increase inexorably, there is no intramuscular stability of [creatine phosphate] or $[H^+]$ (4) and fatigue occurs when W' (hatched areas) is expended. Note: W' is the same for each exercise bout but its rate of expenditure decreases with decreasing power towards critical power. Time-to-fatigue (t) is predicted from critical power and W' parameters as $t = W'/(P-CP)$. Below critical power (but >lactate threshold, heavy domain) efficiency decreases and lactate becomes elevated but these (and the intramuscular [creatine phosphate], $[H^+]$) are stabilized and prolonged exercise sustainable.