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The mechanistic basis of the power–time relationship: potential role of the group III/IV muscle afferents

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The power–time relationship for high-intensity exercise is well known to be hyperbolic and generalizable to multiple exercise modalities in humans and other species. The critical power (CP) is mathematically defined as the asymptote of this hyperbola, while the curvature constant (W′) represents a fixed amount of work that can be performed above CP before reaching exhaustion. Importantly, CP is the highest intensity in which a steady state can be obtained for small muscle mass exercise, as assessed by intramuscular metabolic perturbation, and during whole-body exercise, assessed by oxygen uptake. However, experimental evidence demonstrating that CP represents a threshold for steady state (≤ CP) or non-steady state (> CP) intramuscular metabolic perturbation during whole-body exercise, a necessity for the validation of the CP concept, has remained elusive.

In a recent article published in *The Journal of Physiology*, Vanhatalo and colleagues aimed to clarify the mechanistic bases of the power–time parameters (i.e. CP and W′) during whole-body exercise in relation to muscle metabolism and fibre type distribution (Vanhatalo *et al.* 2016). To this end, they performed two experimental protocols with multiple muscle biopsies prior to, and following, high-intensity cycling tests of varying duration. Notably, the authors present the first evidence demonstrating that CP demarcates intensities which result in steady-state (≤ CP) and non-steady state (> CP) intramuscular metabolic responses for whole-body exercise (i.e. phosphocreatine, creatine, pH, lactate, and glycogen). Moreover, Vanhatalo *et al.* (2016) documented that a greater CP was associated with a higher type I muscle fibre proportion and a lower type IIx proportion. These findings build upon previous work to further validate the CP concept and extend our understanding of the mechanisms determining the power–time relationship. The authors also determined that the size of the W′ is not proportional to any specific muscle fibre type population, further supporting the growing evidence that W′ is determined by the integration of a multitude of physiological mechanisms. Indeed, by compiling evidence from this study and several other recent publications, questions arise regarding the potential mechanistic role of the group III/IV muscle afferents in determining W′; however, little work has been performed with this focus.

Are the group III/IV muscle afferents an important determinant of W′?

The central projection of group III/IV muscle afferent feedback appears to constrain motoneuronal output and therefore limit the neural activation of the working muscle, probably to prevent peripheral fatigue from surpassing an individual and task-specific 'critical fatigue threshold' (for review see Amann, 2011). Interestingly, using blood flow occlusion during handgrip exercise, it has been demonstrated that the magnitude of peripheral fatigue development during exercise was directly associated with the magnitude of W′ (Broxterman *et al.* 2015). Moreover, it was recently demonstrated that attenuation of feedback from these group III/IV muscle afferents resulted in greater intramuscular metabolic perturbation during whole-body exercise and that a tight relationship exists between the level of peripheral fatigue developed and intramuscular metabolic perturbation (Blain *et al.* 2016). Collectively, these findings suggest that group III/IV muscle afferent feedback serves to constrain motoneuronal output to the working muscle, which limits the level of peripheral fatigue development, intramuscular metabolic perturbation, and the amount of work performed, and therefore W′. Further correlative evidence supporting this potential link is the observation that W′ is reduced in certain specific populations, such as chronic obstructive pulmonary disease or chronic heart failure, recognized to exhibit overactive group III/IV muscle afferents.

Are the 'critical power' and the 'critical peripheral fatigue threshold' concepts related?

In a similar fashion to the 3 min all-out cycling test performed by Vanhatalo *et al.* (2016), the repeated sprint model results in a biphasic profile with a gradual decrease in power output between the early sprints (∼W′), and a plateau-like phase characterized by a nearly constant power output until the last sprint (∼CP) (Hureau *et al.* 2014). Importantly, in this scenario, the level of pre-existing quadriceps fatigue and associated intramuscular metabolic perturbation, which lead to a stimulation of the group III/IV muscle afferents, alters the first phase (∼W′) but not the plateau-like phase during the repeated sprint protocol (∼CP) (Hureau *et al.* 2014). These data support the concept of a relationship between the firing of group III/IV muscle afferents and W′. Moreover, reaching the plateau-like phase during repeated sprints is associated with an individual and task-specific nadir of neuromuscular function (i.e. unchanged fatigue with subsequent sprints) (Hureau *et al.* 2016), corresponding to the attainment of a 'critical peripheral fatigue threshold' (for review see Amann, 2011). Interestingly, it appears that CP and the critical peripheral fatigue threshold are reached concomitantly during *all-out* exercise (Hureau *et al.* 2016). Peripheral fatigue is progressively developed during exercise intensities above CP until a critical threshold is attained resulting in exhaustion (constant load trial) or a reduction of exercise intensity ≤CP (self-paced trial). In the study by Vanhatalo *et al.* (2016), and previous investigations using small muscle mass exercise, a constant degree of intramuscular metabolic perturbation was attained at exhaustion for various intensities above CP, which occurred concomitantly with the complete utilization of W′. In light of the recent findings from Blain *et al.* (2016), this is further evidence that the levels of peripheral fatigue and intramuscular metabolic perturbation, and therefore W′, are constrained (i.e. 'critical peripheral fatigue threshold') during exercise performed above CP.

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The study performed by Vanhatalo *et al.* (2016) provides important mechanistic insight into the CP concept by demonstrating that CP demarcates intensities that result in steady-state (≤ CP) and non-steady-state (> CP) intramuscular metabolic perturbations for whole-body exercise. The study also demonstrated that CP, but not W′, is related to muscle fibre type proportions. Rather, W′ is likely to be associated with multifactorial determinants (e.g. intramuscular energy store depletion and metabolite accumulation) and not limited to a single physiological process. Here, we suggest, as one piece of this puzzle, that the amount of W′ may be associated with the feedback from the group III/IV muscle afferents, which prevents an abnormal homeostatic challenge and excessive development of peripheral fatigue during exercise (Blain *et al.* 2016). Consistent with the proposed multifactorial aspect of W′, the firing of the group III/IV muscle afferents is driven by several factors, such as the intramuscular metabolic perturbation or muscle fibre type distribution. Therefore, future studies should focus on the integrated mechanisms determining W′, which will have important implications for understanding exercise tolerance in health and disease.

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Additional information

Competing interests

None declared.

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