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Symposium: Fatigue mechanisms limiting exercise performance

Chair: Michael McKenna & Mark Hargreaves

Neuromuscular fatigue: interactions between central and peripheral factors

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Over the past years evidence from us and others has accumulated indicating that the development of peripheral locomotor muscle fatigue is confined to a certain limit which varies between humans. Central motor drive to these muscles – and therefore exercise performance – during human athletic activities appears to be regulated to avoid the development of peripheral locomotor muscle fatigue beyond an "individual critical threshold". The existence of a degree of peripheral fatigue that is never exceeded during high intensity endurance exercise prompted us to propose the role of peripheral locomotor muscle fatigue as a carefully regulated variable. In various experiments we have challenged this postulate from different perspectives and our results further verify its validity. Based on the knowledge gained from previous observations we outlined a model linking central motor drive with the metabolic milieu within the working locomotor muscles. Our model suggests that, during high intensity endurance exercise, somatosensory feedback from the working muscles imposes inhibitory influences on the magnitude of central motor drive with the purpose to regulate and restrict the level of exercise-induced peripheral locomotor muscle fatigue. As we also qualified, this proposed feedback mechanism is likely only one of several potential contributors to the modulation of central motor drive and that its relative contribution will change with varying conditions such as, for example, severe hypoxemia. In more recent work, we experimentally tested this model *via* pharmacologically modifying somatosensory pathways originating in the working limbs during whole body exercise. After initial difficulties with the lumbar epidural application of a local anesthetic and the associated loss of locomotor muscle strength we switched to an intrathecally applied opioid analgesic. These experiments were the first ever to selectively block lower limb afferent feedback during cycling exercise without affecting maximal locomotor muscle force output. In the absence of neural feedback from the working limbs, central motor drive was about 8% higher during the opioid trial and end-exercise peripheral locomotor muscle fatigue exceeded, for the first time, the critical threshold by nearly 50%. The outcome of these studies further confirms our hypothesis claiming that afferent feedback inhibits central motor drive and restricts the development of peripheral fatigue to an individual critical threshold.

Fatigue during intermittent exercise: novel insights and real-world applications

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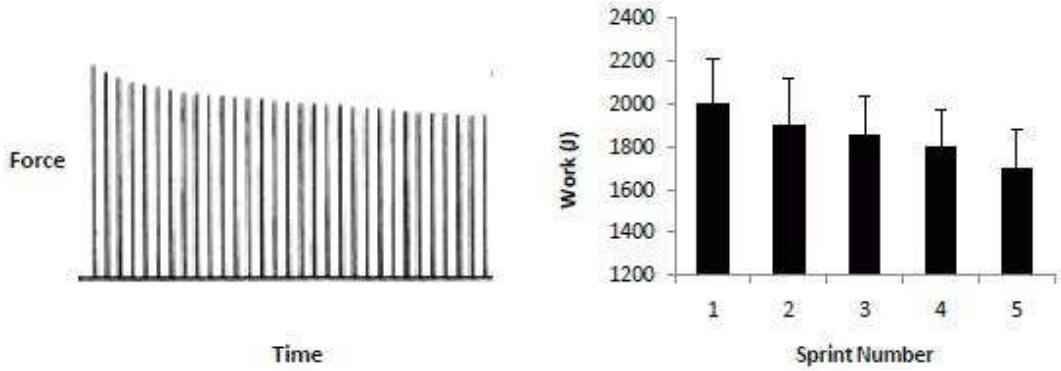
There is a reversible decline in the force production of muscles when they are used at near their maximum capacity. This has been classically demonstrated by stimulating repeated short tetani in an isolated fibre (e.g., left-hand panel of figure; Lannergren & Westerblad, 1991). Such experiments have provided valuable insights regarding potential determinants of fatigue (Allen, Lamb, & Westerblad, 2008). Nonetheless, the application of such findings to dynamic exercise has been questioned. However, a similar pattern for the decline in muscle performance can also be observed when athletes are asked to repeat short-duration sprints (< 10 s), interspersed with brief recoveries (< 30 s) (*e.g.* right-hand panel of figure; Bishop, Edge, Davis, & Goodman, 2004). An additional advantage of this approach is that it is possible to investigate the potential influence of neural/brain factors on the fatigue process.



We are interested in how fatigue manifests during intermittent sprint exercise, and the potential underpinning muscular and neural mechanisms. Such information is important as a better understanding of the factors contributing to fatigue is arguably the first step in order to design interventions (*i.e.* training programs, ergogenic aids) that could eventually improve intermittent-sprint ability.

At the muscle level, limitations in energy supply, which include phosphocreatine hydrolysis and the degree of reliance on anaerobic glycolysis and oxidative metabolism, and the intramuscular accumulation of metabolic by-products, such as hydrogen ions, emerge as key factors responsible for fatigue. Although not as extensively studied, the use of surface electromyography techniques have revealed that failure to fully activate the contracting musculature and/or changes in inter-muscle recruitment strategies (*i.e.* neural factors) are also associated with fatigue outcomes. Via the use of deception, it has recently been demonstrated that prior knowledge of the end-point of exercise (*i.e.* sprint number) is also able to influence the mechanical output profile (*i.e.* fatigue) during intermittent sprint exercise.

Allen, D.G., Lamb, G.D., & Westerblad, H. (2008). Skeletal muscle fatigue: cellular mechanisms. *Physiological Reviews* **88(1),** 287-332.

Bishop, D., Edge, J., Davis, C., & Goodman, C. (2004). Induced metabolic alkalosis affects muscle metabolism and repeated-sprint ability. *Medicine & Science in Sports & Exercise,* **36(5),** 807-813.

Lannergren, J., & Westerblad, H. (1991). Force decline due to fatigue and intracellular acidification in isolated fibres from mouse skeletal muscle. *Journal of Physiology,* **434,** 307-322.