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# Possible contributions of group III/IV muscle afferent feedback to exercise performance

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***Abstract*** Exercise performance cannot be maintained indefinitely, i.e., it deteriorates progressively. Traditionally, deterioration of exercise performance has been attributed to failure of peripheral or central functions of muscle activity. However, muscle rigor (i.e., complete failure of muscle contractile function) never occurs and the muscle force exerted never decreases to zero even with sustained maximal muscle contraction. Furthermore, an increase in central motor output to skeletal limb muscles, and consequently, enhancement of exercise performance, is often observed at the end of a time trial race at which impairment of muscle contractile function is greater. These indicate that only failure of peripheral or central function of muscle activity determines exercise performance. However, recent studies have elucidated that group III/IV muscle afferent inputs to the central nervous system have an important role in the regulation or limitation of exercise performance. This article reviewed two viewpoints regarding contributions of group III/IV muscle afferent feedback to regulation of exercise performance.
***Keywords*** : muscle fatigue, central nervous system, interplay

## Introduction

Performance during prolonged or intermittent exercise differs depending on the exercise and interval duration, and exercise intensity. Exercise-induced changes in performance have been attributed to muscle fatigue. Many investigators have defined muscle fatigue as "any exercise-induced reduction in the capacity to generate force and power output"[1]. Although the cause of muscle fatigue remains controversial, many investigators have assumed that impairment at the neuromuscular junction or at sites distal (i.e., peripheral fatigue)[2] and proximal to the neuromuscular junction (i.e., central fatigue)[3] is responsible for muscle fatigue[4]. If this assumption is true, the maximal voluntary force or power output must continue to decrease throughout the sustained maximal muscle contraction and must be less than that required during sustained submaximal muscle contraction immediately after contraction-induced exhaustion, as the physiological mechanisms at the sites described above are progressively impaired to complete failure. However, these phenomena have not been shown to occur. For example, maximal voluntary force converges with a given constant force ("critical power") through a 5-min all-out muscle contraction[5]. Furthermore, the maximal voluntary power output during sprint cycling lasting a few seconds reaches approximately 70% of the resting maximal voluntary power output immediately after exhaustion is induced by sustained cycling exercise with approximately 20% of the resting maximal voluntary power output[6]. These observations suggest that only impairments of the peripheral organs and central motor pathways do not induce deterioration in performance during maximal or submaximal exercise[7].

Recently, some investigators have argued that afferent signals from group III and/or IV receptors in active muscles restrict central motor output (CMO), and thus limit exercise performance[8,9]. They concluded that this restriction of CMO was a protective mechanism to prevent catastrophic failure in the homeostasis of active muscles[9]. Group III/IV muscle afferents originating in active limb muscles are stimulated by intramuscular metabolic by-products[10] and augment afferent input to the central nervous system (CNS). Evidence that their input can reduce muscle voluntary activation (i.e., CMO), leading to reduction in maximal voluntary contraction (MVC), has been demonstrated for an elbow flexor muscle that was rendered ischemic with a sphygmomanometer cuff after a sustained MVC[11]. Furthermore, the muscle recruitment pattern, which is estimated by the amplitude and frequency spectrum of surface electromyography (SEMG), is dependent on the intramuscular metabolic milieu of the active limb muscles during repeated cycling sprints (RCS)[12]. Hence, afferent signals from group III/IV receptors in active limb muscles appear to be one of the determinants

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that regulate exercise performance.

However, there are two viewpoints on how group III/IV muscle afferent feedback regulates CMO during exercise. Accordingly, this article reviews these viewpoints and discusses the role of group III/IV muscle afferent feedback in regulating exercise performance.

**Viewpoint 1: Muscle afferent feedback from active limb muscles reflexively regulates exercise performance**

In cardiovascular and ventilatory response, the heart rate, blood pressure, and ventilation during exercise evoked by electrical muscle stimulation are similar to those of exercise performed voluntarily[13]. In contrast, the cardiovascular response to exercise is considerably reduced, compared to a control condition, when subjects voluntarily perform exercise with no afferent feedback from the active limb muscles, which is achieved through pharmacological spinal block[14]. These observations suggest that cardiovascular and ventilatory responses to exercise are reflexively regulated by muscle afferents.

As is the case with these responses, group III/IV muscle afferent feedback has been thought to reflexively regulate exercise performance. Evidence of group III/IV muscle afferents reflexively regulating CMO and exercise performance was shown in studies on humans in which $^{31}$P magnetic resonance spectroscopy and muscle afferent block were used[5,15]. In the magnetic resonance spectroscopy study, when the muscle force throughout 5 min of all-out maximal isometric contractions (60 MVCs) converged to 54% MVC, muscle metabolic milieu associated with peripheral fatigue at the end of the all-out contractions was similar to that at task failure of submaximal isometric contraction with 54% MVC[5]. This similar level of muscle metabolic milieu between different contractile regimes (i.e., maximal vs. submaximal contractions) is consistent with the notion that projections of group III/IV muscle afferents to the CNS limit the CMO for peripheral fatigue to prevent exceeding the critical threshold ("tolerance limit")[3]. This highlights the reflexive characteristics of group III/IV muscle afferent feedback to the CNS. Furthermore, in the muscle afferent block study, a pharmacological block (injection of fentanyl) significantly increased the CMO, as estimated by SEMG, during a 5-km cycling time trial in which subjects were free to choose power output compared to the placebo condition, and the indices of peripheral fatigue (i.e., potentiated twitch, maximal rate of force development, and maximal rate of relaxation) exceeded those measured in the placebo condition (critical threshold)[15]. The fact that muscle afferents block released CMO and augmented peripheral fatigue at the end of the time trial also supports the view that group III/IV muscle afferent feedback reflexively regulates CMO and exercise performance.

Recently, peripheral fatigue induced by constant-load, single-leg knee-extension exercise to the point of exhaustion has been shown to curtail the time to exhaustion of the consecutively exercising contralateral leg compared to that of the contralateral leg without pre-fatigue[16]. Cycling exercise tolerance was reduced after an arm-cranking exercise compared with that without prior exercise despite less peripheral fatigue that did not reach a critical threshold[17]. These results mean that group III/IV muscle afferent feedback also restricts CMO to active limb muscles whose peripheral fatigue does not reach a critical threshold when accumulation of group III/IV muscle afferent inputs associated with peripheral fatigue from various skeletal muscles reaches the critical threshold.

However, other researchers have demonstrated evidence against this viewpoint. In endurance exercise, power output and/or CMO have been shown to increase at the end of exercise despite a very high concentration of muscle metabolites, leading to peripheral fatigue[15,18]. As group III/IV muscle afferent inputs to the CNS gradually increase throughout endurance exercise, CMO should be reflexively limited by the increase in group III/IV muscle afferent feedback and hence should decrease proportionally. Therefore, the increase in CMO at the end of exercise, referred to as the "end spurt", is contrary to the viewpoint that group III/IV muscle afferent signals reflexively regulate CMO and exercise performance.

**Viewpoint 2: Group III/IV muscle afferent feedback from active limb muscles regulates exercise performance in accord with endogenous reference signals**

Group III/IV muscle afferent feedback can limit CMO and exercise performance, but the limitation is unlikely to be due to a stereotyped function such as reflex inhibition. Some researchers have argued that training history, muscle substrate reserve, muscle metabolic rate, prior experience, and knowledge regarding exercise duration and distance (i.e., endogenous reference signals) alter the interpretation of group III/IV muscle afferent feedback, and that endogenous reference signals also preprogram power output and pacing strategies before the initiation of exercise[19,20]. Based on this argument, the effects of group III/IV muscle afferent feedback on exercise performance would vary according to endogenous reference signals in that situation. This role of endogenous reference signals is exemplified in the work of Ansley et al.[21], who reported that subjects provided with incorrect information on the duration (30 s) of a cycling sprint exerted lower power output at 33-36 s of the sprint, compared to those provided with correct information (36 s) when a 36-s cycling sprint was actually performed. If group III/IV muscle afferent feedback limited exercise performance at 33-36 s of the sprint, subjects provided with incorrect information must exert higher power output at 0-30 s, compared with those provided with correct information, because the greater group III/IV muscle afferent signals associated with peripheral fatigue by the higher power output would

result in lower power output at the end of the sprint. However, this higher power output was not observed. Therefore, the lower power output at 33-36 s of the sprint is not due to greater group III/IV muscle afferent feedback. Because subjects provided with the incorrect information subconsciously preprogrammed pacing strategy to optimize performance during a 30-s cycling sprint based on the incorrect information (i.e., endogenous reference signals) and predicted group III/IV muscle afferent feedback produced by the 30-s sprint, group III/IV muscle afferent inputs to the CNS at 33-36 s of the cycling sprint, which the subjects could not predict, would be interpreted to limit CMO and performance only in condition with the incorrect information.

Reduced intramuscular pH by sodium bicarbonate ingestion, which in turn decreases group III/IV muscle performance during repeated 10-s cycling sprints performance during repeated 10-s cycling sprints[22]. The data reported here appear to support viewpoint 2. In this study, the number of sprints was not announced. As a result, subjects could not preprogram pacing strategy to reach the limit of maintenance of homeostasis at the end of RCS. In this case, information on the critical level of intramuscular pH appeared to be less important. Therefore, metabolic perturbations in active skeletal muscles did not reflexively limit exercise performance during RCS, and endogenous referent signals (i.e., knowledge of a number of sprints) modified the role of group III/IV muscle afferent feedback.

A study on humans that involved manipulation of resistive load applied in RCS showed further evidence that regulation of exercise performance by group III/IV muscle afferent feedback is modified by endogenous reference signals[23]. The peak power output and CMO at the last two cycling sprints during RCS (10×10-s cycling sprints) was lower in the RCS with a light load than that with a heavy load despite similar muscle metabolic milieu between the light- and the heavy-load conditions[23]. These results indicate that group III/IV muscle afferent feedback associated with peripheral fatigue does not determine exercise performance only during RCS. When RCS was performed in the light-load condition, a cycling sprint at a pedaling rate close to the limit (>180 rpm) was repeated. Hence, RCS in the light-load condition would put subjects at risk of severe damage to muscles and joints. As a result, the CNS prevents catastrophic damage to muscles and joints by limiting CMO in a feedforward manner. Indeed, it is also possible that augmented afferent feedback from group III receptors resulting from a high pedaling rate reflexively limited exercise performance and CMO during RCS in a light-load condition because group III muscle afferents are sensitive to mechanical stimuli[10]. However, exercise performance could have been compromised in the first sprint during RCS in a light-load condition if the afferent signals from group III receptors resulting from a high pedaling rate in active limb muscles reflexively limited CMO. In this viewpoint, the "end spurt," which is an increase in CMO at the end of exercise despite severe metabolic milieu, can be explained by the effects of knowledge of exercise duration and number of sprints on the interpretation of group III/IV muscle afferent feedback.

## Perspective for further research

Recent studies have claimed that exercise tolerance is determined by psychobiological factors such as motivation rather than group III/IV muscle afferent feedback[24,25]. Increased sense of fatigue (i.e. mental fatigue) induced by prolonged psychological tasks (incongruent Stroop task or response inhibition) increases sense of effort to absolute work intensity and shortens time to exhaustion in subsequent submaximal contraction or cycling[26,27]. This suggests that the sense of effort during exercise also is one of the important determinants independent of group III/IV muscle afferent feedback. However, no positive evidence that only psychological factors such as motivation determine exercise performance has been shown. Furthermore, it has been argued that group III/IV muscle afferent feedback influences sense of effort[28,29]. Therefore, further studies are required to elucidate the contributions of psychological factors to exercise performance independent of metabolic milieu in active limb muscles.

Finally, the cognitive demand required for maximal contraction has been argued to differ from that required for submaximal contraction[30]. Additionally, Matsuura et al.[31] have suggested that changes in cognitive process by heat exposure alter power profiles, but not peak and mean power output, during each cycling sprint in RCS. Therefore, we also must carefully analyze muscle force and power during exercise and take into account the contractile regimen applied to elucidate the interplay between psychological factors and group III/IV muscle afferent feedback.

## Conflict of Interests

The author declares that there is no conflict of interests regarding the publication of this article.

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### References

1) Vollestad NK. 1997. Measurement of human muscle fatigue. *J Neurosci Methods* 74: 219-227.
2) Allen DG, Lamb GD and Westerblad H. 2008. Skeletal muscle fatigue: cellular mechanisms. *Physiol Rev* 88: 287-332.
3) Gandevia SC. 2001. Spinal and supraspinal factors in human muscle fatigue. *Physiol Rev* 81: 1725-1789.

4) Bigland-Ritchie B, Jones DA, Hosking GP and Edwards RH. 1978. Central and peripheral fatigue in sustained maximum voluntary contractions of human quadriceps muscle. *Clin Sci Mol Med* 54: 609-614.
5) Burnley M, Vanhatalo A, Fulford J and Jones AM. 2010. Similar metabolic perturbations during all-out and constant force exhaustive exercise in humans: a $^{31}$P magnetic resonance spectroscopy study. *Exp Physiol* 95: 798-807.
6) Marcora SM and Staiano W. 2010. The limit to exercise tolerance in humans: mind over muscle? *Eur J Appl Physiol* 109: 763-770.
7) Noakes TD and St Clair Gibson A. 2004. Logical limitations to the "catastrophe" models of fatigue during exercise in humans. *Br J Sports Med* 38: 648-649.
8) Amann M. 2012. Significance of Group III and IV muscle afferents for the endurance exercising human. *Clin Exp Pharmacol Physiol* 39: 831-835.
9) Noakes TD, St Clair Gibson A and Lambert EV. 2005. From catastrophe to complexity: a novel model of integrative central neural regulation of effort and fatigue during exercise in humans: summary and conclusions. *Br J Sports Med* 39: 120-124.
10) Adreani CM, Hill JM and Kaufman MP. 1997. Responses of group III and IV muscle afferents to dynamic exercise. *J Appl Physiol* 82: 1811-1817.
11) Gandevia SC, Allen GM, Butler JE and Taylor JL. 1996. Supraspinal factors in human muscle fatigue: evidence for suboptimal output from the motor cortex. *J Physiol* 490: 529-536.
12) Matsuura R, Ogata H, Yunoki T, Arimitsu T and Yano T. 2006. Effect of blood lactate concentration and the level of oxygen uptake immediately before a cycling sprint on neuromuscular activation during repeated cycling sprints. *J Physiol Anthropol* 25: 267-273.
13) Krogh A and Lindhard J. 1917. A comparison between voluntary and electrically induced muscular work in man. *J Physiol* 51: 182-201.
14) Fernandes A, Galbo H, Kjaer M, Mitchell JH, Secher NH and Thomas SN. 1990. Cardiovascular and ventilatory responses to dynamic exercise during epidural anaesthesia in man. *J Physiol* 420: 281-293.
15) Amann M, Proctor LT, Sebranek JJ, Pegelow DF and Dempsey JA. 2009. Opioid-mediated muscle afferents inhibit central motor drive and limit peripheral muscle fatigue development in humans. *J Physiol* 587: 271-283.
16) Amann M, Venturelli M, Ives SJ, McDaniel J, Layec G, Rossman MJ and Richardson RS. 2013. Peripheral fatigue limits endurance exercise via a sensory feedback-mediated reduction in spinal motoneuronal output. *J Appl Physiol* 115: 355-364.
17) Johnson MA, Sharpe GR, Williams NC and Hannah R. 2015. Locomotor muscle fatigue is not critically regulated after prior upper body exercise. *J Appl Physiol* 119: 840-850.
18) Amann M, Proctor LT, Sebranek JJ, Eldridge MW, Pegelow DF and Dempsey JA. 2008. Somatosensory feedback from the limbs exerts inhibitory influences on central neural drive during whole body endurance exercise. *J Appl Physiol* 105: 1714-1724.
19) Lambert EV, St Clair Gibson A and Noakes TD. 2005. Complex systems model of fatigue: integrative homoeostatic control of peripheral physiological systems during exercise in humans. *Br J Sports Med* 39: 52-62.
20) Ulmer HV. 1996. Concept of an extracellular regulation of muscular metabolic rate during heavy exercise in humans by psychophysiological feedback. *Experientia* 52: 416-420.
21) Ansley L, Robson PJ, St Clair Gibson A and Noakes TD. 2004. Anticipatory pacing strategies during supramaximal exercise lasting longer than 30 s. *Med Sci Sports Exerc* 36: 309-314.
22) Matsuura R, Arimitsu T, Kimura T, Yunoki T and Yano T. 2007. Effect of oral administration of sodium bicarbonate on surface EMG activity during repeated cycling sprints. *Eur J Appl Physiol* 101: 409-417.
23) Matsuura R, Arimitsu T, Yunoki T and Yano T. 2011. Effects of resistive load on performance and surface EMG activity during repeated cycling sprints on a non-isokinetic cycle ergometer. *Br J Sports Med* 45: 820-824.
24) Marcora S. 2008. Is peripheral locomotor muscle fatigue during endurance exercise a variable carefully regulated by a negative feedback system? *J Physiol* 586: 2027-2028.
25) Marcora S. 2010. Counterpoint: Afferent feedback from fatigued locomotor muscles is not an important determinant of endurance exercise performance. *J Appl Physiol* 108: 454-456.
26) Pageaux B, Marcora SM and Lepers R. 2013. Prolonged mental exertion does not alter neuromuscular function of the knee extensors. *Med Sci Sports Exerc* 45: 2254-2264.
27) Pageaux B, Lepers R, Dietz KC and Marcora SM. 2014. Response inhibition impairs subsequent self-paced endurance performance. *Eur J Appl Physiol* 114: 1095-1105.
28) Craig AD. 2002. How do you feel? Interoception: the sense of the physiological condition of the body. *Nat Rev Neurosci* 3: 655-666.
29) Craig AD. 2009. How do you feel—now? The anterior insula and human awareness. *Nat Rev Neurosci* 10: 59-70.
30) Halperin I, Chapman DW and Behm DG. 2015. Non-local muscle fatigue: effects and possible mechanisms. *Eur J Appl Physiol* 115: 2031-2048.
31) Matsuura R, Arimitsu Y, Yunoki T, Kimura T, Yamanaka R and Yano T. 2015. Effects of heat exposure in the absence of hyperthermia on power output during repeated cycling sprints. *Biol Sport* 32: 15-20.