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# EFFECT OF KNEE MUSCLE FATIGUE AND LACTIC ACID ACCUMULATION ON POSTURAL CONTROL

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## SUMMARY
This study investigated the effects of high intensity dynamic fatiguing exercise on blood lactate, maximal knee flexion and extension force and center of pressure (COP) sway during full body dynamic postural perturbations. Dynamic fatiguing exercise was performed using an ergometer until exhaustion. Pre and immediately after fatiguing exercise, maximal knee flexor and extensor force, blood lactate and a COP sway on a moving platform were measured. A significant interaction was observed between muscle force and COP sway, in which a reduction in muscle force was accompanied by increasing postural sway following fatiguing exercise. In conclusion muscle fatigue and lactic acid accumulation may lead to slowed propagation of efferent signals to maintain posture and as a result fatigued athletes are at greater risk for musculoskeletal injuries.

## INTRODUCTION
Muscle fatigue is an inevitable part of high intensity exercise and has been associated with reduced power output and work capacity of the skeletal muscle [1].

Fatigue-induced impairments in neuromuscular control may adversely alter joint proprioception [1, 2].and are believed to be a potential cause for the increased injury rates during the latter stages of athletic competition, Particularly during unexpected perturbations [3]. This study examined the effect of fatigue on the response of COP sway to fast, functionally relevant, full body perturbations. These perturbations are produced by a computer controlled platform that can be moved in three-dimensional space to produce sliding perturbations that resemble perturbations that might be encountered during certain sport activities.

## METHODS
Ten healthy subjects (age, mean ± SD, 26.5 ± 4.5 yr., body mass, 74.4 ± 7.3 kg, height, 1.8 ± 0.38 m) participated in the present study. Dynamic fatiguing exercise was performed on a bicycle ergometer (SRM, Germany) and consisted of light cycling followed by a ramping protocol starting at 100 W with the workload increased by 15 W.min$^{-1}$ until exhaustion. Three maximal voluntary contractions (MVC) of knee flexors and extensors were conducted before and immediately after dynamic fatiguing exercise. Moreover, blood lactate collected from fingertip was measured. Furthermore, anterior–posterior center of pressure (COP) sway from the subjects over the moveable force platform was recorded during rapid full body perturbations performed before and immediately after the high intensity exercise protocol from a motion capture system (Qualisys, Sweden).

## RESULTS AND DISCUSSION
MVC was reduced from the pre fatigue condition to the post fatigue condition by 33±13% for knee extensors (pre: 581.7±168.9 N; post: 441.7±143.3 N, $P < 0.001$) and by 24±18% for knee flexors (pre: 133.2±25.6 N; post: 109.2±25.1 N, $P < 0.001$). The concentration of blood lactate significantly increased immediately after exercise (pre: 1.66±0.39 mmol/l; post: 13.12±1.22 mmol/l, $P < 0.001$). Moreover, a fatigue-induced increase in the anterior–posterior COP sway during the perturbations (pre-fatigue: 98.14±32.9 mm; post fatigue: 106.86 ±38.85mm; $P < 0.05$) was observed. **(**Figure 1)

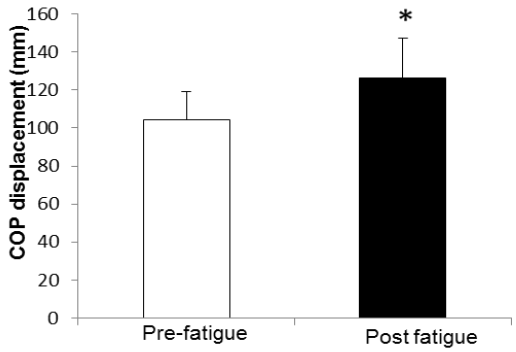


**Figure 1** Displacement of the center of pressure in the anterior-posterior direction during the pre and post fatigue conditions. $*P < 0.05$.

High-intensity, exercise-induced fatigue is associated with changes in motor output and alterations in proprioception which may occur at both the central and peripheral level. At peripheral level muscle fatigue is induced by reduced availability of metabolic substrates for muscle contraction, such as adenosine triphosphate, creatine phosphate, and glycogen, as well as increased metabolite accumulation, including lactic acid in the muscle, resulting in an inability to maintain a desired muscular force output [4]. This accumulation of metabolites and/or inflammatory substances (i.e., lactic acid or bradykinin) within the muscle during activity leading to fatigue have been shown to elicit increased muscle spindle static and/or dynamic sensitivity via reflex-mediated pathways from chemosensitive group III and IV afferents onto γ-motoneurons [5]. Altered somatosensory input due to muscle fatigue could result in deficits in neuromuscular and postural control around the joint thereby making skeletal structures more susceptible to injury [6].

## CONCLUSIONS

Muscle fatigue and lactic acid accumulation may lead to a reduction in propagation velocity of efferent signals to maintain posture. As a result muscle fatigue may increase the risk for musculoskeletal injury during rapid perturbations (as occurs in most sport activities). Therefore, exercise programs for patients and athletes should be carefully monitored for signs of fatigue to avoid deficits that might compromise dynamic joint stability.

## REFERENCES


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