

# THE EFFECT OF JOINT POSITION AND FATIGUE ON AGONIST/ANTAGONIST CO-ACTIVATION DURING ISOMETRIC KNEE EXTENSION

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## INTRODUCTION

Agonist/antagonist co-activation has been proposed to be a potential injury prevention mechanism for the anterior cruciate ligament (ACL). However, varying results have been reported throughout the literature, with respect to agonist/antagonist co-activation across various knee positions. As anterior tibial translation is proposed to be greatest at more extended knee positions and if agonist/antagonist co-activation is a mechanism for reducing ACL strain, it may be speculated that co-activation would be greatest near full extension. Furthermore, the level of co-activation may also be limited subsequent to fatigue inducement of the antagonist muscle group. Therefore, the purpose of this study was to investigate the effect of joint position and knee flexor fatigue on agonist/antagonist co-activation during isometric knee extension.

## METHODS

Eight healthy, recreationally active individuals (5 men, 3 women) with no history of lower limb injury volunteered for participation in this study. The subjects visited the laboratory 6 times, with at least 48 hours between sessions. Each testing session occurred at a pre-determined, and randomly ordered knee position (10, 30, 50, 70, 90 deg knee flexion). Electromyography (EMG) was collected from 7 muscles (vastus medialis (VM), vastus lateralis (VL), rectus femoris (RF), medial hamstring

(MH), lateral hamstring (LH), medial gastrocnemius (MG), and lateral gastrocnemius (LG)) during each visit. Subjects sat in the Biodex accessory chair, the lower leg was secured to the resistance adaptor and the knee was then placed in the pre-determined testing position. The subjects first performed 5 manually resisted, plantar-flexion maximal voluntary contractions (MVC), each lasting approximately 5 s with 2 minutes between each contraction. Subjects then performed 5 isometric knee extension MVC's (5 s, 2 min rest). Subjects subsequently performed 5 isometric knee flexion MVC's in a similar manner. The fatigue protocol consisted of sub-maximal and maximal knee flexor contractions and began with the subjects performing a 5 s sub-maximal (50% MVC) contraction, which was followed by a 5 s rest period. Following 5 sub-maximal contractions, the subjects were instructed to perform a 5 s knee flexor MVC. The subjects were considered fatigued when their MVC torque decreased by 50%, or when they were no longer able to maintain 50% MVC for the entire 5 s duration. Immediately following the fatigue protocol, the subjects were asked to perform 5 isometric knee extension MVC's, with 2 min separating each contraction.

## RESULTS AND DISCUSSION

The results of this investigation revealed no significant difference between pre-fatigue and post-fatigue co-activation values for the

MH and LH; however, the MG and LG co-activation values were found to be significantly greater post-fatigue ( $p < 0.05$ ) (Figure 1). An effect of knee angle on co-activation was found to be significant for the MG and LG ( $p < 0.05$ ), as co-activation was greatest at 70 deg knee flexion. However, no effect was revealed for the MH and LH (Figure 2).

With no change in MH and LH co-activation with fatigue, it is suggested that the neural drive to the hamstrings is maintained after the fatigue protocol and the functional ability of the hamstrings as antagonists is not effected by muscular exhaustion. As it has been shown that the gastrocnemius muscle acts as an antagonist to the ACL (Fleming et al., 2001), the results of this study suggest that the function of the gastrocnemius is more apparent with fatigue of the hamstring muscle group.

The lack of a significant change in hamstring co-activation throughout the knee range of motion indirectly suggests that hamstring co-activation is not initiated by strain on the ACL. As research has suggested that ACL strain is greatest with quadriceps femoris muscle contraction at extended positions (Grood et al, 1984), these results indirectly demonstrate that the ACL-hamstring reflex is not the only moderator of hamstring co-activation. It may be that a common drive to the agonist and antagonist muscle groups is responsible for antagonist co-activation (Psek & Cafarelli, 1993).

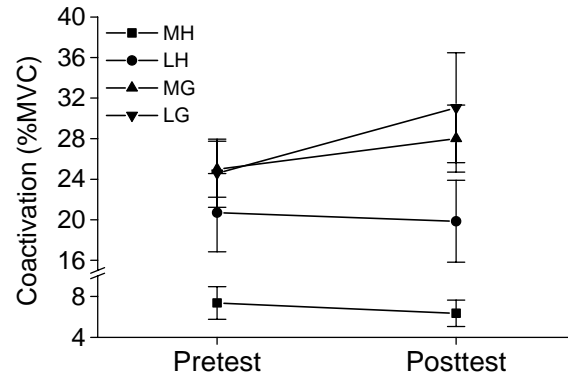


Figure 1. Co-activation levels as a function of pre- and post-fatigue tests, for the MH, LH, MG, and LG.

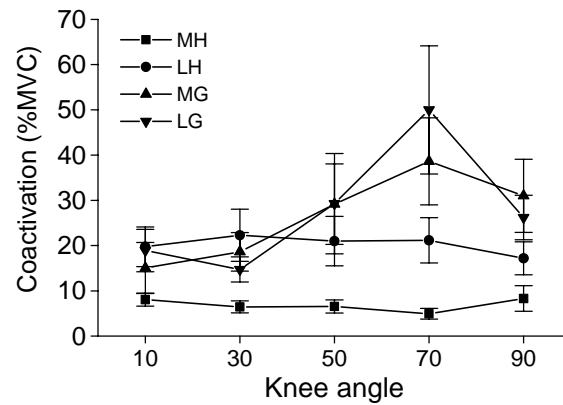


Figure 2. Co-activation levels as a function of knee flexion angle, for the MH, LH, MG, LG.

## REFERENCES

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