



ISB 2013
BRAZIL

XXIV CONGRESS OF THE INTERNATIONAL
SOCIETY OF BIOMECHANICS

XV BRAZILIAN CONGRESS
OF BIOMECHANICS

COACTIVATION WITHIN THE TRICEPS SURAE MUSCLE GROUP IS ALTERED IN ACHILLES TENDINOPATHY

¹Nicole Grigg, ¹Claudio Pizzolato and ¹Justin Kavanagh

¹Centre for Musculoskeletal Research, Griffith Health Institute, Griffith University, Gold Coast, Australia
n.grigg@griffith.edu.au www.griffith.edu.au/health/musculoskeletal-research

INTRODUCTION

Tendinopathy is a chronic musculoskeletal disorder for which the mechanisms underlying pathogenesis are not fully understood. While an animal model of Achilles tendinopathy has implicated central neural mechanisms in the pathogenesis of the disorder [1], neuromuscular function in humans with Achilles tendinopathy has received relatively little attention. To date, studies have shown that individuals with Achilles tendinopathy exhibit higher electromyography (EMG) signal amplitudes of the medial gastrocnemius (MG) and soleus (SOL) muscles during eccentric rehabilitative exercises [2] and increased SOL first volitional wave magnitude, representative of greater descending neural drive [3]. While these findings support the notion that impaired neuromuscular function coexists with Achilles tendinopathy, studies to date have not investigated coactivation within the triceps surae muscle group. It is particularly important to investigate the relative activation levels of the muscles within the triceps surae group given that, intratendinous shear due to the production of different amounts of force from each muscle may be involved in the pathogenesis of Achilles tendinopathy [4, 5]. Furthermore, eccentric exercise (EE) is a popular conservative treatment for Achilles tendinopathy, although, it is not always successful with some studies reporting patient satisfaction rates of approximately 60% [6, 7]. Understanding coactivation in Achilles tendinopathy may facilitate the development of more targeted exercise based treatments designed to address specific neuromuscular changes, similar to what has been achieved with targeted training of the vastus medialis in patellofemoral pain syndrome.

The aim of the current research was to determine the EMG amplitudes of the MG, lateral gastrocnemius (LG) and SOL muscles during EE and to subsequently, calculate the coactivation ratios for the MG and LG relative to the SOL.

METHODS

Eleven males with unilateral mid-portion Achilles tendinopathy (mean age 48.2 ± 3.8 years, height 181.6 ± 2.0 cm and mass 97.3 ± 6.9 kg) and nine male controls without tendinopathy (mean age 49.0 ± 4.5 years, height 180.6 ± 2.2 cm and mass 92.6 ± 5.6 kg) participated in the research. Lower limb kinematics and EMG were recorded while participants performed an EE protocol used for the treatment of Achilles tendinopathy [6]. The Achilles tendinopathy

group performed EE using the limb with symptomatic tendinopathy, while for the control group allocation of the left or right limb to EE was counterbalanced. The protocol involved the participant standing with the forefoot of the test limb on the edge of a step in a position of maximal plantarflexion. Eccentric loading occurred as the heel was lowered below the level of the step to a position of maximal dorsiflexion. The contralateral limb was used to return the body to the start position. Six sets of 15 eccentric loading repetitions were completed. The orientations of the lower limb segments were recorded throughout the EE protocol by an eleven camera motion analysis system sampling at 200 Hz using the PlugInGait (SCAR) model within Vicon Nexus. Surface EMG was recorded from the MG, LG and SOL using a bipolar electrode configuration, an inter-electrode distance of 20 mm and with the electrodes aligned parallel to the muscle fibers. For the MG and LG, electrodes were placed distal to the knee and medial and lateral to the midline, respectively. Soleus electrodes were positioned on the posteriolateral aspect of the lower leg below the LG. Prior to electrode application the skin was shaved, abraded and cleaned with alcohol. EMG signals were sampled at 1000 Hz using a wireless EMG system (Zero Wire) and synchronized with kinematic data using Vicon Nexus.

The maximum and minimum sagittal ankle joint angles, ankle joint range of motion (ROM) and average angular velocity of the ankle joint were determined for each exercise repetition. EMG signals were band-pass filtered using a 4th order zero-lag Butterworth filter with cut off frequencies of 20 and 300 Hz. EMG amplitude for each exercise repetition was calculated as the root mean square (RMS). To determine the level of coactivation for each repetition the RMS of the MG and LG was normalized to the RMS of the SOL. As such, coactivation ratios of one were representative of equivalent EMG amplitudes, and ratios greater than one were typical of higher MG or LG amplitude relative to the SOL.

The effect of limb (tendinopathy or control) on each of the dependent variables was investigated using a general linear modeling approach, wherein all 90 exercise repetitions were included in the analysis. Means and standard errors produced by the general linear models are presented in text.

RESULTS AND DISCUSSION

Compared to the control limb the tendinopathy limb was characterized by significantly less dorsiflexion and significantly more plantarflexion during EE (Table 1). Despite these differences in peak ankle joint angles the ankle joint ROM was not significantly different between control and tendinopathy limbs. The tendinopathy limb performed EE at a significantly slower velocity compared to the control limb (Table 1).

Table 1: Kinematic characteristics of eccentric exercise.

	Control	Tendinopathy
Peak dorsiflexion (°)	22.1 (1.3)	12.9 (1.1)*
Peak plantarflexion (°)	-10.1 (1.2)	-17.4 (1.0)*
Ankle ROM (°)	32.2 (0.7)	30.3 (0.6)
Average angular velocity (°/s)	37.5 (1.5)	29.9 (1.3)*

*Significantly different from control limb ($p < .05$)

For all muscles EMG amplitude was significantly higher in the tendinopathy limb compared to the control limb (Figure 1). The MG/SOL coactivation ratio was not significantly different between tendinopathy and control limbs, although the ratio of LG to SOL activation was significantly higher for the tendinopathy limb (Figure 1).

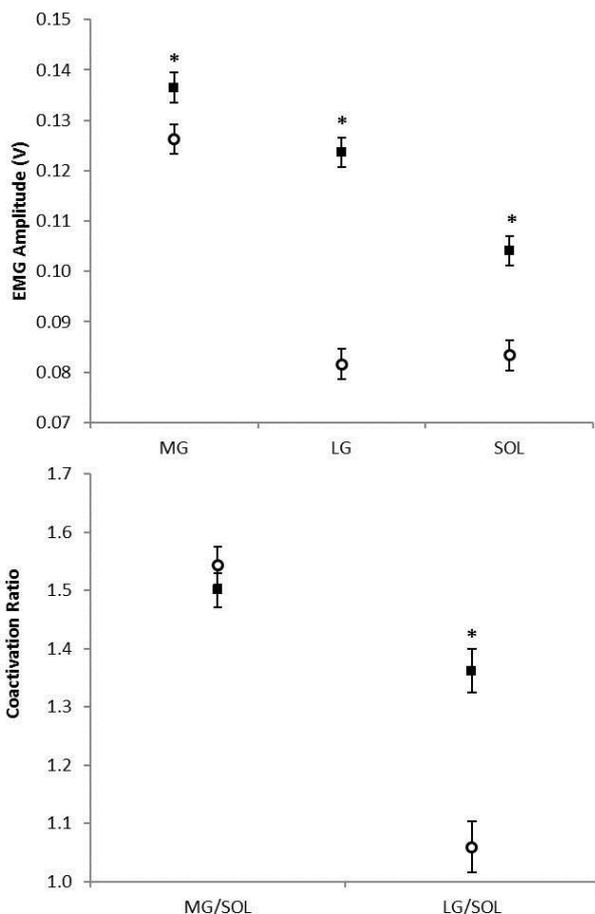


Figure 1: Mean EMG amplitudes (top) and EMG coactivation ratios (bottom) for control (○) and tendinopathy (■) limbs during eccentric exercise. Error bars represent 95% confidence intervals. * Significantly different from control limb ($p < .05$)

The performance of eccentric exercise in a relatively plantarflexed position and at a slower velocity by individuals with symptomatic Achilles tendinopathy is in

line with previous studies which, reported reduced peak dorsiflexion during EE in the presence of Achilles tendon pain [9] and, that individuals with Achilles tendinopathy exhibited a slower dorsiflexion velocity during running [8]. While the rationale for these changes is unclear, there is the potential that the reduction in peak dorsiflexion angle and angular velocity were strategies to reduce Achilles tendon loading and avoid pain. Alternatively, changes in muscle architecture, which have been shown to coincide with Achilles tendinopathy [9], may have been associated with ROM shift and slower velocity.

The observed higher EMG amplitudes for the MG, LG and SOL in symptomatic tendinopathy are consistent with a previous study which utilized the same EE protocol and also found higher EMG amplitudes (normalized to maximum voluntary contraction) for the MG and SOL in symptomatic Achilles tendinopathy when compared to a control condition [2]. These observations align with the finding of increased neural drive in Achilles tendinopathy [3] and have been suggested to be compensatory mechanisms for concomitant changes in muscle architecture, increased tendon compliance and loss of strength with Achilles tendinopathy [2, 3].

This is the first study to show that Achilles tendinopathy is characterized by a significantly higher LG to SOL coactivation ratio compared to healthy individuals. This indicates that, relative to the SOL, the activation of the LG was disproportionately high in tendinopathy. This finding provides preliminary support for the hypothesis that varying levels of force from the muscles within the triceps surae group, resulting in shearing between tendinous components, may be associated with Achilles tendinopathy [4, 5]. Understanding changes in coactivation ratios with tendinopathy provides the opportunity to develop targeted exercise based treatments to address these changes.

CONCLUSIONS

Individuals with Achilles tendinopathy exhibit functional kinematic adaptations and higher activation levels during the EE commonly used in tendinopathy rehabilitation. Importantly, this is the first study to investigate coactivation in tendinopathy and has demonstrated that Achilles tendinopathy is associated with disproportionately high LG activation. These findings of altered function and neuromuscular activation with Achilles tendinopathy may be utilized to develop more targeted exercise based treatments.

REFERENCES

1. Andersson G, et al. *Br J Sports Med.* **45:** 399-406, 2010.
2. Reid D, et al. *Phys Ther Sport.* **13:**150-155, 2012.
3. Wang H, et al. *J Orthop Sports Phys Ther.* **41:**785-794, 2011.
4. Arndt A, et al. *Foot Ankle Int.* **20:** 444-449, 1999.
5. Bojsen-Moller J, et al., *J Appl Physiol.* **97:** 1908-1914, 2004.
6. Sayana M, et al., *J Sci Med Sport.* **10:** 52-58, 2007.
7. Rompe J et al., *Am J Sports Med.* **35:** 374-383, 2007.
8. Alfredson H, et al. *Am J Sports Med.* **26:** 360-366, 1998.
9. Henriksen M, et al. *Br J Sports Med.* **45:** 393-398.
10. Ryan M, et al. *Foot Ankle Int.* **30:** 190-1195, 2009.
11. Padhiar N, et al. *Disabil Rehabil.* **30:**1640-1645, 2008.

