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Eccentric loading of triceps surae modulates stretch shortening cycle behaviour- A possible therapeutic mechanism

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Abstract

Context: Eccentric exercises are increasingly being used to treat lower limb musculoskeletal conditions such as Achilles tendinopathy. Despite widespread clinical application and documented efficacy, mechanisms underpinning clinical benefit remain unclear. Positive adaptations in motor performance are one potential mechanism. **Objective:** To investigate how an eccentric loading intervention influences measures of stretch-shortening cycle (SSC) behaviour during a hopping task. **Design:** Within subjects repeated measures observational study. **Setting:** University motion analysis laboratory. **Participants:** Healthy adults. **Interventions:** A single intervention of 5 sets of 10 eccentric plantarflexion contractions at 6 RM using a commercial seated calf raise machine. **Main outcome measures:** Lower limb stiffness, sagittal plane ankle kinematics, and temporal muscle activity of the agonist (soleus) and antagonist (tibialis anterior) muscles, measured during sub-maximal hopping on a custom-built sledge-jump system. **Results:** Eccentric loading altered ankle kinematics during sub-maximal hopping; peak ankle angle shifted to a less dorsiflexed position by 2.9° and ankle angle pre-contact shifted by 4.4° ($p < 0.001$). Lower limb stiffness increased from 5.9 to 6.8 Nm^{-1} ($p < 0.001$), whilst surface EMG measures of soleus occurred 14 to 44% earlier ($p < 0.001$) following the loading intervention. **Conclusions:** These findings suggest that eccentric loading alters SSC behaviour in a manner reflective of improved motor performance. Decreased ankle excursion, increased lower limb stiffness and alterations in motor control may represent a positive adaptive response to eccentric loading. These findings support the theory that mechanisms underpinning eccentric loading for tendinopathy may in part be due to improved ‘buffering’ of the tendon by the neuromuscular system.

Keywords: Achilles tendon, Eccentric exercise, Lower limb stiffness, Motor control, Rehabilitation

Main Text

Introduction

Eccentric contractions are defined as muscle activity that occurs when the force applied to the muscle exceeds the momentary force produced by the muscle itself¹ and are used to decelerate, brake or absorb energy². The intentional elicitation of eccentric contractions under load is referred to as eccentric loading and has for many years been used in both training and rehabilitative settings³. One common application of eccentric loading is in the management of Achilles tendinopathy (AT). First reported on in the early 1990's⁴ and made popular by Alfredson, et al.⁵, eccentric loading involves the patient performing progressive, loaded, eccentric contractions of the ankle plantarflexors. Whilst efficacy for eccentric loading for AT has been demonstrated⁶, the mechanisms underpinning its effect are not fully understood⁷. Proposed mechanisms include structural tendon adaptation, tendon length changes, neurovascular ingrowth, neurochemical alterations, fluid movement, and neuromuscular changes (see⁸ for a recent review).

The stretch-shortening cycle (SSC) describes the pre-activation, lengthening and shortening of a musculotendinous unit during ground contact⁹ and is a common feature of terrestrial locomotion¹⁰. It exists to simplify the motor control of locomotion and enhance its efficacy through the utilisation of muscle pre-activation, storage of elastic energy, and utilisation of the spinal stretch reflex¹¹. SSC behaviour can be regulated according to changes in task¹² and environment¹³, and can be modulated in response to training¹⁴ or pathologic conditions such as AT¹⁵. Recently, the role of the SSC in the development and management of AT has received increased attention^{15,16}, based on the premise that whilst AT has a multifactorial pathogenesis¹⁷, deficits in motor performance are considered a major pathogenic component¹⁸. In terms of regulating SSC behaviour, the most important phase is the eccentric phase¹¹; whilst it is well-known that eccentric loading improves motor performance¹⁹, we have limited understanding on how SSC behaviour can be modulated by eccentric loading of the plantarflexors²⁰.

The purpose of this study was to investigate how an eccentric loading intervention of the plantarflexors affects SSC behaviour. To do this, we measured several correlates of SSC-behaviour (lower limb stiffness, ankle kinematics and agonist/antagonist muscle activity) during a controlled SSC task (sub-maximal hopping on a sledge jump system) before and 7 days after an eccentric loading protocol. We hypothesised that following the intervention lower limb stiffness would increase driven by alterations of muscle activity. Furthermore, we hypothesised that ankle kinematics would demonstrate a shift towards a more plantarflexed position. A clearer understanding of how eccentric loading effects SSC behaviour may assist in explaining the mechanisms underpinning its efficacy and inform their clinical applications in conditions such as AT. For instance, it can be used to inform clinical decision making about when and why eccentric loading can be incorporated into patient management, providing the potential to improve clinical outcomes in this challenging population.

Methods

This study drew upon a previously described protocol investigating the effect of eccentric fatigue on SSC behaviour²¹.

Design

This was a within subjects repeated measures observational study. The independent variable was eccentric loading status (Baseline vs. Eccentric) and the dependent variables were temporal measures of lower limb stiffness (k), ankle kinematics (angle 80 ms prior to foot contact, ankle angle at contact, peak ankle angle, and stretch amplitude) and surface electromyographic (sEMG) activity (onset, peak, and offset timings for soleus and tibialis anterior).

Participants

We recruited 11 healthy volunteers (5 males and 6 females; mean age 23.2 ± 6.7 years) and excluded those with a history of AT, lower limb surgery in the preceding 12 months, a co-existing

lower quadrant musculoskeletal disorder, or a significant visual or motor impairment. All participants provided written informed consent, with procedures being approved by the local university human research ethics committee.

Procedures

Sub-Maximal Hopping Task

Participants performed continuous sub-maximal hopping on a custom-built sledge jump system. Such systems have demonstrated validity and reliability²² and the details of the protocol employed in this study have been described in detail elsewhere¹⁵. The advantage of hopping on such a system as this is that the SSC can be isolated to the ankle and the confounding effects of fatigue can be eliminated due to the low load nature of the task. Following a warm-up, participants hopped continuously at a sub-maximal level (one that could be sustained ‘indefinitely’) for 15 seconds. Following a 45 second rest period, this was repeated, for a total of 5 trials (see fig 1). Five trials were chosen as pilot testing had established that exclusion of the first trial, as well as the initial hop for any given trial results in stable measures within-trial, between multiple trials, and across multiple testing occasions. With this protocol we were able to analyse the full series of trials and given that participants hopped at 1.3 Hz, provided approximately 20 hops per trial.

Kinematics

Sagittal plane ankle kinematics were recorded using a 14-camera Vicon MX motion analysis system (Vicon, Oxford Metrics, Oxford, UK) operating at 250 Hz. Retro-reflective markers were fixed to participants’ skin according to a customised marker set and model for the lower quadrant which established anatomically-relevant ankle, knee and hip joint axes of rotation and joint centres²³. Ankle angles at specific time-points relative to initial ground contact (80 ms pre-contact and contact) were captured along with peak dorsiflexion angle. Ankle dorsiflexion stretch amplitude was calculated as the difference between the ankle angle at contact, and peak dorsiflexion angle.

Electromyographic Measures

Soleus and tibialis anterior muscle activities were recorded, using an AMT-8 (Bortec Biomedical Ltd) surface electromyography (sEMG) system. Bipolar differential surface electrodes (Ag / AgCl) were placed on the belly of each muscle with the reference electrode on the medial malleolus. Skin impedance (< 15kOhms) was achieved by skin preparation and signals were pre-amplified, analogue filtered (10 – 500Hz band pass) and digitised at sampling rate of 1000Hz. All data was synchronised on dedicated hardware running a customised program (Labview, National Instruments, Austin, Texas, 2011).

Intervention- Eccentric Loading Protocol

The employed eccentric loading protocol has previously been comprehensively described elsewhere²¹, which was applied on a single occasion using the dominant limb. Using a commercial seated calf raise machine participants warmed-up (3 x 10 repetitions at 4/10 RPE) and completed a 6 RM test upon which the eccentric loading dose was based. The 6 RM was always achieved within 3 attempts (group mean 6 RM 27.4 kg ± 6.6). Following a 10 minute break, participants performed a single eccentric loading protocol consisting of 5 sets of 10 eccentric plantarflexion repetitions at their 6 RM, interspersed by 60 second rest periods.

Protocol

Participants attended for baseline testing and the eccentric loading intervention on day 1. Participants returned for post-intervention testing 7 days later with testing performed at a consistent time of day on both occasions. This model was chosen to best isolate neuromuscular changes rather than those that may occur at days 1-3 with delayed onset muscle soreness (DOMS) following a novel exercise or tendon changes that may occur with multiple training occasions²⁴. All participants were instructed to continue with their normal daily activities over the intervening week, and specifically instructed not to undertake any change in physical activities. Likewise,

participants were asked if any experienced DOMS was still evident at day 7 testing and none reported so.

Statistical Analyses

Kinematic and sEMG data were processed in accordance with international standards²³. For each trial, the following ankle kinematic measures were calculated; ankle angle 80 ms prior to ground contact, ankle angle at ground contact, peak ankle angle and ankle stretch amplitude and with the exclusion of the first trial, mean values of the remaining 4 trials were analysed. Lower limb stiffness was calculated using the following method²⁵:

$$K_n = \frac{M \times \pi (T_f + T_c)}{T_c^2 \left(\frac{T_f + T_c}{\pi} \frac{T_c}{4} \right)}$$

K_n = lower limb stiffness (Nm⁻¹), M = Mass (kg), T_f = Flight time (s) and T_c = Contact time (s)

Temporal measures of muscle activity for soleus and tibialis anterior were calculated relative to ground contact; onset, peak and offset, using the protocol of Allison²⁶ and described by Debenham, et al.¹⁵.

Statistical analysis was conducted using SPSS version 20 (SPSS, Chicago, IL, USA). Descriptive statistics were used to establish mean values for all variables in each group (Baseline vs. Eccentric). A linear mixed model was used for all statistical comparisons between groups. Age, gender height and body mass were input as covariates and adjusted for within the model. A fixed main effects model was fitted, with a type III sum of squares used to assess statistical significance. For each dependent variable parameter estimates were utilised, and main effects were compared as pairwise comparisons using a Bonferoni correction for repeated measures. The residuals were tested for normality as required by the linear mixed model with a set significance level of $p < 0.05$. Based on a previous study employing these methods¹⁵ a minimum sample size of 10 participants was determined sufficient to test the null hypothesis with a type I error of five percent, with power set at 80%.

Results

Descriptive statistics are presented in Table 1. Following the eccentric loading protocol, lower limb stiffness significantly increased by 0.9 Nm^{-1} ($p < 0.001$; 95% CI 0.7 to 1.1) (Fig 2b). Ankle angle 80ms pre-contact significantly decreased by 4.4° ($p < 0.001$; 95% CI 3.1 to 5.8°) although ankle angle at contact did not change significantly. There was a shift in peak ankle angle towards a less dorsiflexed position (Fig 2a); this angle significantly decreased by 2.9° ($p < 0.001$; 95% CI 1.5 to 4.3°) and ankle stretch amplitude significantly increased by 1.0° ($p = 0.001$; 95% CI 0.3 to 1.7°).

sEMG measures of soleus occurred 44.7%, 17.4% and 14.6% earlier for onset, peak and offset value respectively (soleus onset: $p < 0.001$; 95% CI 28 to 40; soleus peak: $p < 0.001$; 95% CI 36 to 49; soleus offset: $p < 0.001$; 95% CI 14 to 27 (Fig 3)). Tibialis anterior onset was delayed by 13.4% ($p = 0.03$; 95% CI 1 to 23); tibialis anterior peak was not significantly different ($p = 1.00$; 95% CI -13 to 8) and tibialis anterior offset was earlier ($p < 0.001$; 95% CI 28 to 43)) (Fig 3).

Discussion

Whilst the efficacy of eccentric loading for AT has been demonstrated⁶ and theories regarding its efficacy have been explored²⁷, this is the first study to determine in which way SSC behaviour is modulated by eccentric loading. Our findings demonstrate that following eccentric loading, peak ankle angle shifts to less dorsiflexion (6.5 to 3.6° dorsiflexion) and lower limb stiffness increases (5.9 to 6.8 Nm^{-1}), whilst agonist/antagonist muscle activity moves to a more agonist-dominant pattern (e.g. soleus onset 34 ms earlier vs. tibialis onset 6 ms later) (see Figs 2 and 3).

Consistent with our hypothesis, we found that peak ankle angle shifted towards a position of relative plantarflexion. Whilst modest, these findings likely represent a clinically meaningful change when compared with the 7.7° and 3.2° changes in peak torque angle that have been previously reported at the hamstrings²⁸ and plantarflexors²⁹ respectively following eccentric

loading. Previous research has found that eccentric loading leads to increases in muscle strength and power²⁷ and an improved ability to produce force in the descending limb of the muscle fascicle length-tension curve³⁰; our findings are consistent with these phenomena. Furthermore, our findings may reflect an increase in force producing capacity of the muscle similar to those seen by Masood, et al.³¹ as increases in strength imply the plantarflexors have an increased capacity to resist excursion into dorsiflexion. Improved muscle performance also protects the tendon by conferring increased stiffness upon the tendon, thereby improving its capacity to resist strain³². As such, these increases ensure tendon strain is not excessive, as is believed to be pathogenetic with AT³³.

Also consistent with our hypothesis, we observed a 15% increase in lower limb stiffness following the eccentric loading intervention from 5.9 to 6.8 Nm⁻¹ which was achieved by decreasing contact time and increasing flight time. Our findings are consistent with Elmer, et al.²⁰ who in their study of healthy volunteers observed a 10% increase in lower limb stiffness following an eccentric loading task. Increases in stiffness are generally associated with improvements in both performance in healthy individuals, and clinical improvements in patients with a pathologic condition³⁴. Our findings therefore likely reflect a true positive change in spring behaviour associated with the aforementioned alterations in muscular function following the intervention. Furthermore, given the 1 week timeframe involved, it is most likely that changes are neural rather structural in origin. Whilst we did not measure sEMG amplitude, the alterations in timing imply an integration of motor activity, which in turn results in increased stiffness³⁵. Whilst it may be speculated that the increase in stiffness may be in part due to the impact that eccentric loading has on the connective tissues, the observed changes in SSC behaviour following a single loading event are unlikely to have occurred due to changes in tendon material or mechanical properties³⁶.

Consistent with our hypothesis, following the eccentric loading intervention, temporal muscle events occurred earlier in 4 out of our 6 measures and this is the first time temporal changes

in sEMG have been demonstrated following an eccentric loading intervention. These findings most likely reflect training-induced changes in neural activation as described by Markovic, et al.³⁵. Such changes include increased neural drive to the agonist muscle, as we observed with the hastening of soleus activity at all-time points. Likewise, our observed changes toward a more agonist dominant pattern of SSC performance may reflect changes in activation strategies (i.e. improved intermuscular co-ordination)³⁵. The only other study exploring plantarflexor sEMG following eccentric loading is that of Masood, et al.³¹. Their findings are consistent with our own; they observed an increase in plantarflexor sEMG amplitude following an eccentric loading intervention. Given that Cadore, et al.³⁷ observed no increase in sEMG amplitude following eccentric loading, it may be that eccentric loading induces changes in muscle timing rather than amplitude. This theory is supported by Masood, et al.³¹ who suggest that eccentric loading-induced changes in amplitude ultimately normalises muscle activity (i.e. return dysfunctional motor performance to ‘normal’).

Limitations to our study must be acknowledged. Our study was conducted on healthy volunteers; whilst this limits the generalisability of our findings to the target clinical population (AT), our findings form the basis upon which comparisons can be made with equivalent studies on the clinical population³¹. Although conventional sEMG is widely used to measure the electrical activity of skeletal muscle during activity, the information it provides may not reflect the activity of the whole muscle³⁸. Given that intramuscular variations in muscle activity exist, findings such as ours may not reflect the true change in muscle behaviour³⁹. Whilst errors in kinematic measures may also occur due to soft tissue artefact of skin-attached markers, high within-subject reliability implies our methods accommodate this. Likewise, whilst the one week might be expected to result in natural variation in measures, pilot testing demonstrated high reliability (e.g. ICC of 0.77 for ankle stretch amplitude). Finally, whilst our study is designed to inform mechanisms underpinning a clinical intervention, the employed loading protocol is not reflective of standard clinical practice⁵.

This design however was purposeful, reflecting our interest in isolating our observations to changes in motor behaviour in response to loading rather than inducing chronic changes in tendon structure. Finally, our study did not measure the activity profile of gastrocnemius. Whilst this data would add value it was chosen to measure soleus in isolation due to its primary role in low level SSC-activities such as sub-maximal hopping and running⁴⁰.

The eccentric phase of the SSC appears to be its critical phase¹¹. Likewise, it is known that eccentric loading improves eccentric, but not concentric muscle performance³⁷. Our study has demonstrated that eccentric loading induces what appear to be positive changes in SSC behaviour. Varied opinions exist regarding the mechanisms that underpin eccentric loading for AT⁷ with theories directed towards resolution of pathologic tendon structure and function⁴¹ or improvements in motor performance⁴². Our findings provide support to the theory that changes in motor performance underpin such benefits. We also suggest that improvements in tendon structure that may occur in response to eccentric loading are secondary to improvements in motor performance. Our findings of reduced excursion into dorsiflexion and increased stiffness may represent the ‘buffering’ capacity of the muscle to protect the tendon as proposed by Lindstedt, et al.¹. Furthermore, AT is associated with excessive loading into dorsiflexion¹⁵. Regardless of whether this kinematic phenomenon is a cause or consequence of tendinopathy it represents a maladaptive biomechanical state; excessive dorsiflexion during the SSC is sub-optimal for the muscles length-tension properties and under such circumstances excessive load is placed upon the passive structures. Our findings of reduced dorsiflexion indicate that this phenomenon reduces following eccentric loading and may play an important role in protecting the tendon from excessive tensile loading. Admittedly, given these methods it is difficult to ascertain whether these findings are unique to eccentric loading, or may be observed with combined eccentric-concentric loading. The theory that clinical benefits are conferred by improvements in motor performance (i.e. strength) is gaining traction, with Beyer, et al.⁴³ observing superior outcomes with resistance

loading (combined heavy eccentric and concentric contractions) rather than by eccentric loading. Replication of this study with a combined group is warranted; the remaining mechanism of efficacy being related to the changes in peak torque angle that occur following eccentric, but not concentric loading²⁹.

Finally, whilst our study has no direct link to the pathogenesis of AT, it is relevant to speculate that our findings support those of McCrory, et al.⁴⁴ who stated that reduced plantarflexor performance acts as a factor to increase the prevalence of AT. Combining our findings with those of Masood, et al.⁴⁵ observing improvements in symptoms and motor performance following eccentric loading in patients with AT, we suggest that impaired plantarflexor performance may be a significant pathogenic component of AT.

Conclusions

In summary, we have shown that in healthy adults, eccentric loading results in positive changes in ankle kinematics, lower limb stiffness, and agonist/antagonist muscle activity. These findings may reflect an increase in force producing capacity which leads to decreased loading in end range dorsiflexion, and possibly protecting the tendon from excessive load and providing an opportunity to heal.

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Conflict of Interest

The authors of this study have no professional relationships with companies or manufacturers who will benefit from the results of the present study.

The results of the present study do not constitute endorsement by ACSM

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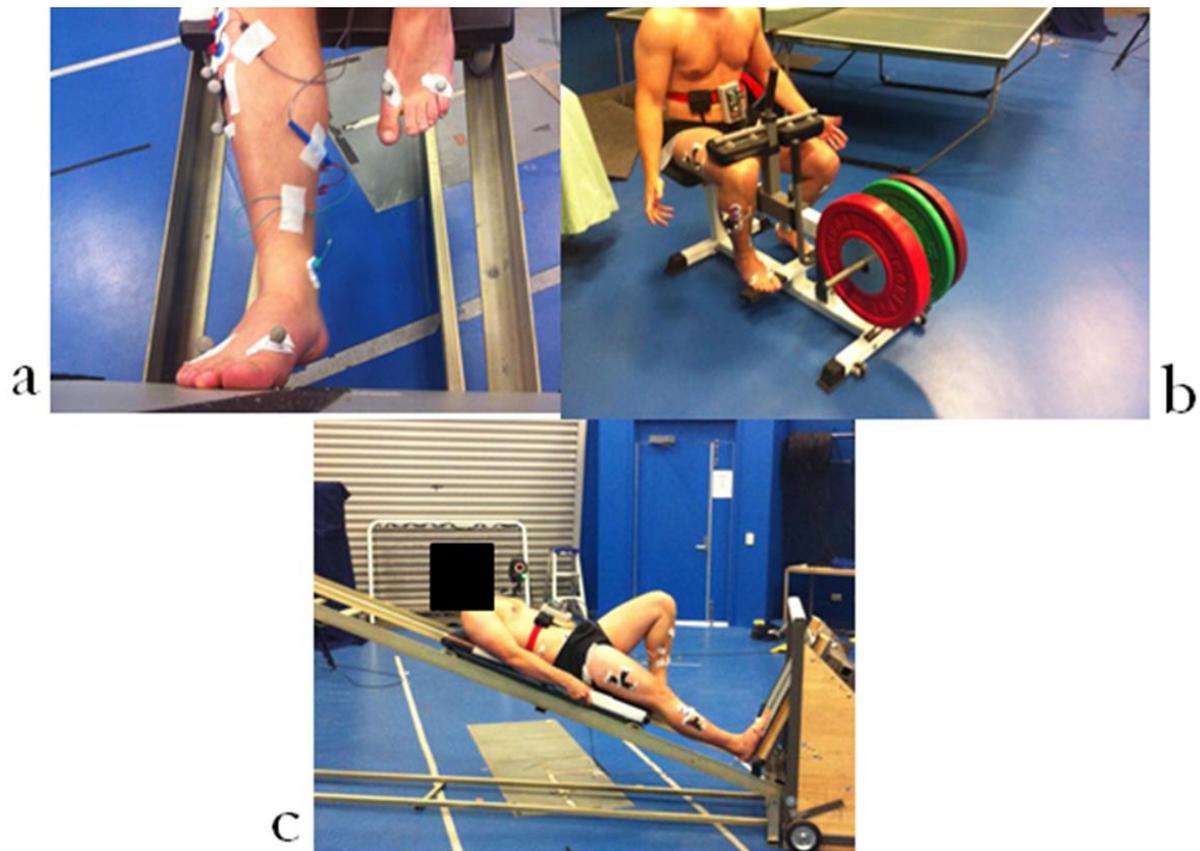


Figure 1) Experimental set-up **a)** participant hopping on sledge-jump system, viewed from above; sEMG and 3D motion-analysis marker set in situ; **b)** participant performing a loaded single-leg calf raise; **c)** participant hopping sledge jump system- hip and knee remain in a neutral position (used by permission²⁰).

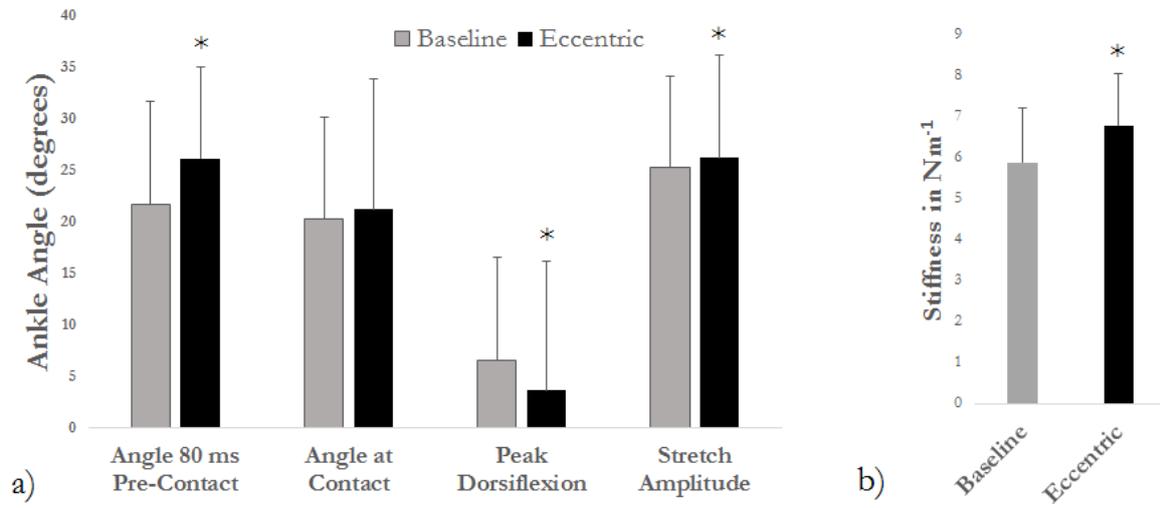


Figure 2- a) Ankle kinematics and **b)** lower limb stiffness at baseline and following the eccentric loading protocol.

* denotes significant difference ($p < 0.05$)

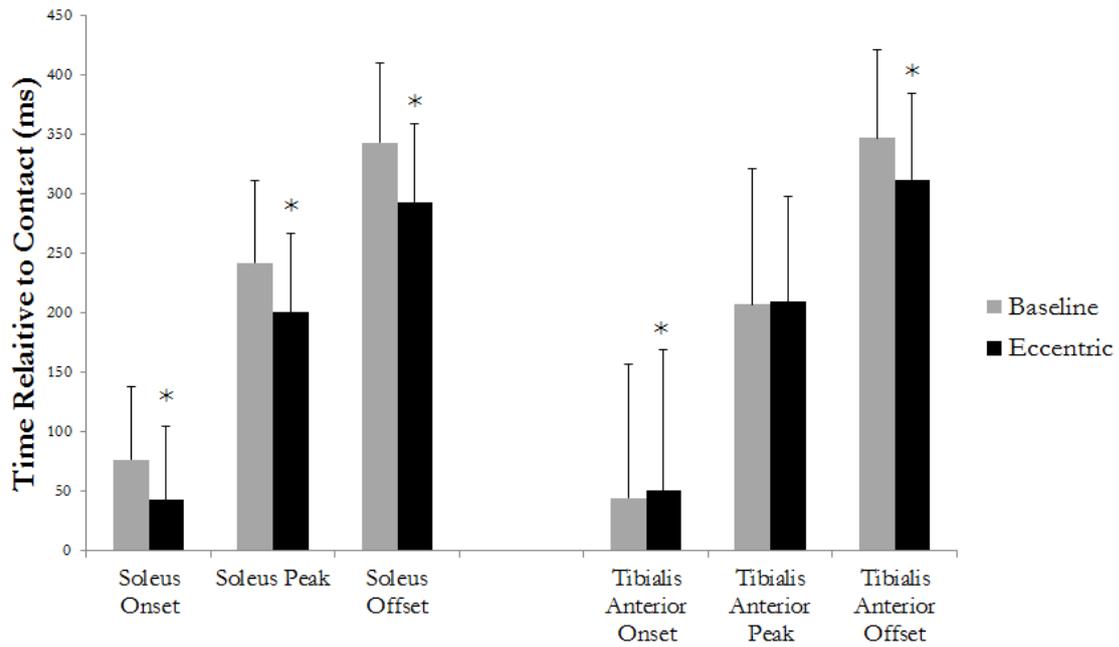


Fig 3- Soleus and tibialis anterior muscle onset timings at baseline and following the eccentric loading protocol.

* denotes significant difference ($p < 0.05$)

Table 1 – Descriptive Statistics (Mean \pm SD)

	Baseline	Post-Loading (7 days)
Hopping frequency (Hz)	1.3 (.16)	1.3 (.24)
Flight time (ms)	341 (73)	371 (101)*
Contact time (ms)	455 (60)	416 (61)*
Stiffness (Nm⁻¹)	5.9 (1.34)	6.8 (1.26)*
Ankle angle 80ms pre-contact (°DF)	-27.1 (10.1)	-26.1 (8.9)*
Ankle angle at contact (°DF)	-20.2 (10.0)	-21.2 (12.7)
Peak ankle angle (°DF)	6.5 (10.0)	3.6 (12.5)*
Ankle stretch amplitude (°)	25.2 (8.9)	26.2 (10.0)*
Soleus onset (ms)	76 (62)	42 (62)*
Soleus peak (ms)	242 (69)	200 (67)*
Soleus offset (ms)	343 (67)	293 (66)*
Tibialis anterior onset (ms)	44 (113)	50 (119)
Tibialis anterior peak (ms)	207 (114)	209 (89)
Tibialis anterior offset (ms)	347 (74)	312 (73)*

DF dorsiflexion

* significant difference (p<.05)