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James R. Debenham

University of Notre Dame Australia, james.debenham@nd.edu.au

M Travers

William Gibson

University of Notre Dame Australia, william.gibson@nd.edu.au

A Campbell

G Allison

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# Eccentric Fatigue Modulates Stretch-shortening Cycle Effectiveness – A Possible Role in Lower Limb Overuse Injuries

## Authors

J. Debenham<sup>1</sup>, M. Travers<sup>2</sup>, W. Gibson<sup>1</sup>, A. Campbell<sup>2</sup>, G. Allison<sup>2</sup>

## Affiliations

<sup>1</sup> School of Physiotherapy, University of Notre Dame Australia, Fremantle, Australia

<sup>2</sup> School of Physiotherapy and Exercise Science, Curtin University, Perth, Australia

## Key word

- fatigue
- stretch shortening cycle
- eccentric contraction
- stiffness

## Abstract

The role of fatigue in injury development is an important consideration for clinicians. In particular, the role of eccentric fatigue in stretch-shortening cycle (SSC) activities may be linked to lower limb overuse conditions. The purpose of this study was to explore the influence of ankle plantarflexor eccentric fatigue on SSC effectiveness during a hopping task in healthy volunteers. 11 healthy volunteers (23.2±6.7 years) performed a sub-maximal hopping task on a custom-built sledge system. 3D motion capture and surface EMG were utilised to measure lower limb stiffness, temporal kinematic measures and muscle timing measures at baseline and immediately

following an eccentric fatigue protocol. A linear mixed model was used to test whether measures differed between conditions. Compared to baseline, eccentric fatigue induced increased stiffness during the hopping task (+15.3%;  $P<0.001$ ). Furthermore, ankle stretch amplitude decreased (-9.1%;  $P<0.001$ ), whilst all other ankle kinematic measures remained unchanged. These changes were accompanied by a temporal shift in onset of activity in soleus and tibialis anterior muscles (-4.6 to -8.5%;  $p<0.001$ ). These findings indicate that eccentric fatigue alters SSC effectiveness in healthy volunteers. These findings may be applied to inform pathogenetic models of overuse injury development.

## Introduction

Modulation of stretch-shortening cycle (SSC) effectiveness in response to loading history implies it has a role in athletic performance, injury prevention and rehabilitation [10]. For example, SSC effectiveness improves with training [26], and deteriorates in the presence of overuse injuries. Whilst SSC dysfunction has been demonstrated in several overuse conditions [20, 35, 38], due to its intimate relationship with the SSC, the condition that has received the most attention is Achilles tendinopathy (AT) [14, 33, 41]. An important physiological phenomenon that links SSC effectiveness and injury is fatigue. Further understanding the relationship between fatigue and SSC effectiveness may assist in the rehabilitation and prevention of overuse injuries such as AT.

The SSC is a phenomenon associated with human locomotion, describing the muscle function in which a pre-activated musculotendinous unit lengthens (eccentric phase) then immediately shortens (concentric phase) [42]. It simplifies

and optimises the neural efficiency of terrestrial locomotion and is dependent upon co-ordinated storage/return of elastic energy and muscle activation under the control of neural strategies. The most meaningful measure of SSC effectiveness is stiffness of the lower limb [8]. Stiffness increases or decreases, regulated in accordance with task and environmental changes by the modulation of muscle activity via feedforward and feedback neural activity [16, 32]. Furthermore, stiffness is modified in response to acute and chronic loading histories [39, 40], as well as in accordance with pathologic conditions such as AT [14], where as a rule, increased stiffness is positive, and decreased stiffness is negative to the individual's performance.

The pathogenesis of lower limb overuse injuries such as AT are complex and multifactorial [30]. However, one important feature appears to be SSC dysfunction [14, 33, 41] where impairments in the spring mass system [7] result in abnormal loading of musculoskeletal tissues [10]. Whilst SSC dysfunction is likely mediated by multiple factors, fatigue appears to be a significant com-

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## Correspondence

**James Debenham, MSc**  
School of Physiotherapy  
University of Notre Dame  
Australia  
19 Mouat Street  
6959 Fremantle  
Australia  
Tel.: +61/8/9433 0996  
Fax: +61/8/9433 0544  
[james.debenham@nd.edu.au](mailto:james.debenham@nd.edu.au)

ponent of this process [11, 19]. For instance, exhaustive running decreases lower limb stiffness [15], mediated by alterations in the neural control of the SSC. It has been suggested that this fatigue leads to increased passive tissue loading and an increased risk of injury [37].

Whilst optimal SSC efficiency requires all components to act in unison, the eccentric phase is the primary determinant of effective SSC effectiveness [12], with approximately two-thirds of extensor muscle activity occurring during the braking phase [37]. As such, a close exploration of fatigue, with a focus on the eccentric phase is warranted, which to our knowledge has yet to be done. In line with the importance of the eccentric phase of the SSC, preliminary data exists suggesting that impairments in eccentric muscle performance are an important feature of overuse injuries [1, 11, 34]. However, research to date has only explored the effect of fatigue on SSC modulation using SSC fatigue models. However, SSC fatigue models such as exhaustive running induce significant systemic fatigue [19], which makes divorcing the role of systemic fatigue from the neural response to fatigue not possible. Given the potential importance of eccentric fatigue in the modulation of SSC effectiveness and its potential to contribute to a number of lower limb overuse pathologies, this paper explores the modulatory effect of eccentric fatigue on temporal measures of SSC effectiveness.

SSC effectiveness can be measured in several ways: at a tissue level, at a kinematic/muscle activation level and at a systems level [9]. In our exploration, we measured the temporal changes in lower limb stiffness, and corresponding changes in associated ankle kinematic and agonist/antagonist muscle activity during a sub-maximal single-limb hopping task on a sledge-jump system. Comparisons between baseline (BAS) values and those taken immediately after an ankle plantarflexor eccentric-only fatiguing protocol (FAT) were performed.

## Materials and Methods



### Participants

This was an observational study, employing a within-subjects, repeated measures design. We recruited healthy university students in Perth, Western Australia (see **Table 1** for participant characteristics). Participants were excluded if they had a history of AT, lower limb surgery in the preceding 12 months, co-existing lower quadrant musculoskeletal disorder, or a significant visual or motor impairment. Participants received a full explanation of the procedures before providing written consent. Our study meets the ethical standards of this journal as described by Harriss and Atkinson [21].

### Measurements

#### Sub-maximal hopping task

SSC effectiveness was investigated using a submaximal single-limb hopping model on a custom-built sledge-jump system (**Fig. 1a, c**). Such systems are employed to optimise reliability

by reducing the degrees of freedom in the task and eliminate task fatigue as a confounding factor [18]. Furthermore, given our interest in focusing the task to the ankle, this model unloads participants, enabling this process. Following familiarisation, participants were instructed to hop on the sledge using their dominant limb, at a submaximal effort level, consciously maintaining a neutral hip and knee. Pilot testing demonstrated that this was achieved; mean joint excursion angles for the ankle, knee and hip were  $28.6^\circ (\pm 10.2)$ ,  $9.8^\circ (\pm 4.8)$  and  $6.3^\circ (\pm 2.3)$  respectively, with high reliability (e.g., ICC of 0.87 of ankle stretch amplitude 0.77 for within, between and across trial measures). Participants hopped continuously for 15 s, repeated 5 times, with 45 s rest between trials. This task was performed before and immediately after (within 5 min) the fatigue intervention.

### Kinematic measures

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, according to an established cluster-based method [6]. This established set-up enabled determination of anatomically-relevant ankle, knee and hip joint axes of rotation and joint centres [6].

### Electromyographic measures

Soleus and tibialis anterior muscle activity 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 ( $< 15 \text{ k}\Omega$ ) was achieved by skin preparation and signals were pre-amplified, analogue-filtered (10–500 Hz band pass) and digitised using an 18-bit A-D card utilising a sampling rate of 1 000 Hz. All data was temporally synchronised and recorded on dedicated hardware running a customised Labview program (National Instruments, Austin, Texas, 2011).

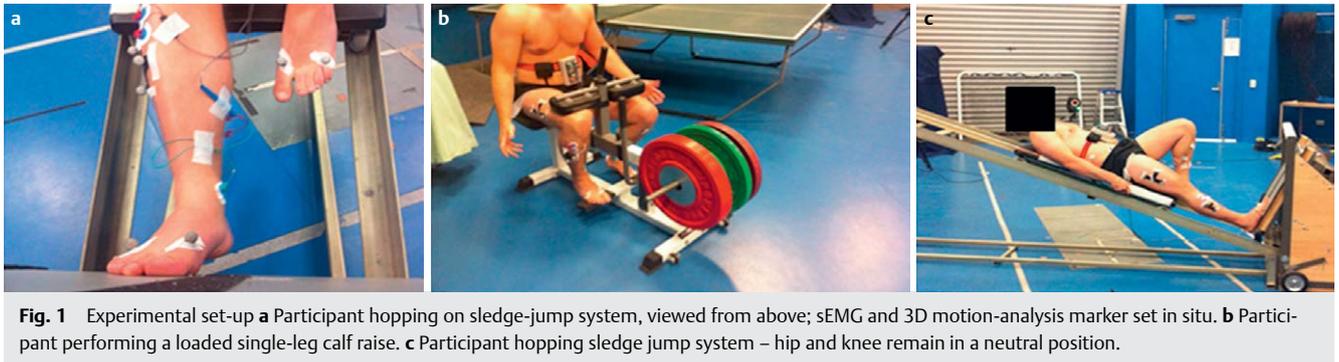
### Interventions

#### Fatiguing protocol

Positioned in a commercial seated calf-raise machine (**Fig. 1c**) and using only their dominant limb, participants completed a warm-up of 3 sets of 10 repetitions using a 10-kg weight before a '6 Repetition Maximum Test' (6 RM; the maximum weight a participant can lift 6 times) was conducted. Participants performed 6 isotonic repetitions, beginning at 12.5 kg; on successful completion, weight was increased by 2.5 kg. All sets were separated by 60 s rest and continued until the participant could not successfully complete the task. Successful completion of the task was judged by whether or not the participant could complete the 6 repetitions through full range without employing compensatory strategies such as hip flexion or trunk movements that might facilitate hip flexor contribution. The final successful weight, which for each participant was achieved within 3 trials, was considered their 6RM (group mean  $27.4 \text{ kg} \pm 6.6$ ). Participants performed 5 sets of 10 eccentric contractions at their 6RM [5]. An assistant raised the weight so that the participant only had to perform the eccentric component. The aim of the protocol was to induce standardised eccentric fatigue in the plantarflexors. If a participant fatigued early or late, sets were added or removed at the discretion of the chief investigator. All partici-

**Table 1** Participant characteristics.

n = (male:female)	11 (3:8)
age	23.2 ( $\pm 6.7$ ) years
height	170.1 ( $\pm 8.2$ ) cm
mass	70.7 ( $\pm 13.3$ ) kg



**Fig. 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.

pants completed 5 or 6 sets. Neither during this protocol, nor on re-testing, did participants experience pain or discomfort beyond the transient discomfort associated with muscle fatigue.

### Data analysis

Kinematic and sEMG data were processed using Vicon Nexus motion analysis software (Vicon, Oxford Metrics, Oxford, UK). Kinematic data were inspected for broken trajectories that can occur as a result of marker occlusion. All breaks <20 frames in length were filled using standard procedures (i.e., cubic spline interpolation). Data was filtered using a fourth-order Butterworth filter operating at a frequency cut-off of 20Hz for the marker trajectories and 50Hz for the ground contact data as determined by residual analysis [44]. Lower limb anatomical and joint coordinates were calculated in accordance with the standards outlined by the previously described International Standards of Biomechanics [6,45]. Data was exported from Nexus for further analysis using a customised LabVIEW program (National Instruments, Austin, Texas, 2011). For each trial, the following ankle kinematic measures were calculated; ankle angle 80ms prior to ground contact, ankle angle at ground contact, peak ankle angle and ankle stretch amplitude. Lower limb stiffness was calculated using the following validated method [13]:

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

$K_n$ : Lower limb stiffness ( $\text{Nm}^{-1}$ )

M: Mass (kg)

$T_f$ : Flight time (s)

$T_c$ : Contact time (s)

Temporal measures of muscle activity for the soleus and tibialis anterior were calculated relative to ground contact: onset, peak and offset. The EMG signal was full-wave rectified and onsets detected using an integrated protocol [2]. Trial linear envelopes (LE) were created using a fourth-order, zero-lag Butterworth low-pass filter (10Hz) and temporally synchronised to ( $T=0$ ) foot contact.

### Statistics

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 (BASE vs. FAT). 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 significance level of  $p < 0.05$ .

### Results



Following eccentric fatigue, lower limb stiffness increased +15.3% from 5.9 ( $\pm 1.3$ ) to 6.8 ( $\pm 1.7$ )  $\text{Nm}^{-1}$  ( $p < 0.001$ ; 95% CI 0.7 to 1.1) (► Fig. 2b). Our primary kinematic variable, stretch amplitude (► Fig. 2a) decreased -9.1% from 25.2 ( $\pm 8.9$ ) to 23.4 ( $\pm 8.5$ )° ( $p < 0.001$ ; 95% CI -1.1 to -2.4). There was no statistically significant difference in any other kinematic measure between BAS and FAT.

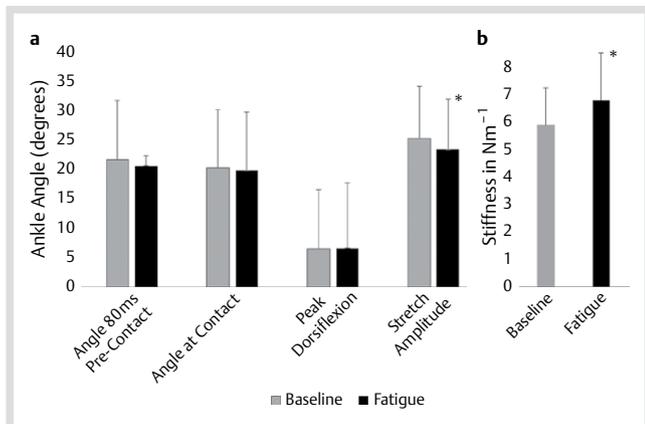
For all temporal sEMG measures (► Fig. 3), relative timings occurred between 4.6–8.5% earlier following the fatigue intervention (soleus onset: 76 ( $\pm 62$ ) to 61 ( $\pm 51$ ) ms ( $p < 0.001$ ; 95% CI 9 to 21); soleus peak: 242 ( $\pm 69$ ) to 222 ( $\pm 62$ ) ms ( $p < 0.001$ ; 95% CI 14 to 27); soleus offset: 343 ( $\pm 67$ ) to 322 ( $\pm 58$ ) ms ( $p < 0.001$ ; 95% CI 14 to 27); tibialis anterior onset 44 ( $\pm 113$ ) to 32 ( $\pm 102$ ) ms ( $p = 0.03$ ; 95% CI 1 to 23); tibialis anterior peak 207 ( $\pm 114$ ) to 193 ( $\pm 111$ ) ms ( $p = 0.01$ ; 95% CI 3 to 25); tibialis anterior offset 347 ( $\pm 74$ ) to 331 ( $\pm 68$ ) ms ( $p < 0.001$ ; 95% CI 8 to 23).

### Discussion

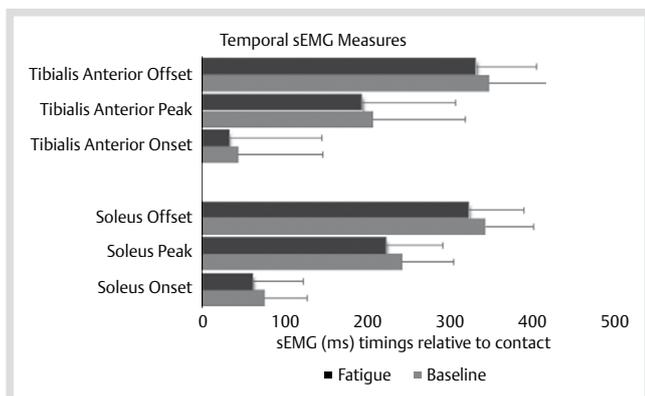


The impact of eccentric fatigue on SSC effectiveness is potentially an important pathogenetic component for lower limb overuse conditions such as AT and to our knowledge, this is the first study that has explored how eccentric fatigue modulates SSC effectiveness. We found that eccentric fatigue results in increased lower limb stiffness and a corresponding decrease in stretch amplitude, accompanied by a hastening of muscle activity. Lower limb stiffness increased by 15.3% ( $0.9 \text{ Nm}^{-1}$ ), stretch amplitude decreased by 9.1% ( $1.8^\circ$ ) (► Fig. 2) and all temporal measures of muscle activity for the tibialis anterior and soleus hastened by 4.6–8.5% (12–21 ms) (► Fig. 2).

Whilst the findings on stiffness are inconsistent with the existing literature [4,22,28], findings on muscle activity are consistent [28]. A number of possible explanations for these findings exist. Whilst the majority of other studies investigating the effect of fatigue on lower limb stiffness have observed decreases in stiffness [25,28,37], systemic fatigue was induced by the methodology employed. Our experimental model attempted to mitigate the influence of systemic fatigue and isolate changes to



**Fig. 2** Mean ( $\pm$ SD) for **a** kinematic and **b** lower limb stiffness values at baseline and fatigue during the hopping task; \* denotes significant difference ( $P < 0.05$ ).



**Fig. 3** Mean ( $\pm$ SD) sEMG values at baseline and fatigued, for tibialis anterior and soleus during the hopping task. All measures are significantly different ( $P < 0.05$ ).

the neural control of the SSC following fatigue. Fatigue appears to generate feed-forward changes in agonist and antagonist muscle activity, which drives the increase in stiffness. According to Oliver, De Ste Croix, Lloyd, et al. [37], increased stiffness may represent a potentiated state within the neuromuscular system, where in response to fatigue, neural control is modulated in a positive (i.e., protective) manner. The ability to maintain short ground contact time appears to be a key determinant to maintaining SSC performance under fatigue [22] and participants may have increased stiffness in order to achieve this.

Fatigue is a pathogenetic feature of lower limb overuse conditions (e.g., AT [36]) and may be perceived at a systems level as a threatening stimulus. Using our experimental model, it has been suggested that in the presence of an overuse injury, a threat is perceived and individuals attempt to limit exposure to that threat by increasing stiffness [14]. The findings of the current study support this theory, suggesting that fatigue may be perceived at a 'control-level' as a threatening stimulus in the same way that pain is [24].

We observed a decrease in ankle stretch amplitude, but no other changes in ankle kinematic measures. The only other study to explore ankle kinematics during an SSC following fatigue was performed by Kuitunen, Avela, Kyrolainen, et al. [27]. They measured lower limb kinematics during sub-maximal hopping on a sledge system following a hopping fatigue protocol. Once fatigued, they observed increased ankle stretch amplitude in the

region of 15% relative to baseline, compared to increases at the knee and hip of 50 and 300% respectively. These findings indicate that in the presence of fatigue, individuals absorb load at the hip and knee as opposed to the ankle, which our findings also support. In our model, participants were unable to yield at the hip or knee; nevertheless, relative attempts were made to limit yielding at the ankle, as appeared to occur in the study by Kuitunen, Avela, Kyrolainen, et al. [27] showing in fact a slight reduction in stretch amplitude at the ankle. Whilst the fatiguing nature of the 2 studies differed, in combination these findings support the theory that in the presence of a threatening stimulus, a local stiffening strategy is employed to limit such exposure [24].

Limitations of our study require acknowledgement. Our fatiguing protocol isolated the plantarflexors in an eccentric manner, meaning that fatigue of other muscles (antagonist, synergists) in a manner that would induce SSC and systemic fatigue limit the generalisability of our findings. However, this model was specifically chosen in order to isolate the eccentric role of the plantarflexors in the absence of systemic fatigue. This is important given that eccentric plantarflexor activity is the primary modulator of SSC effectiveness [12,23]. Similarly, this protocol would inevitably cause exercise-induced muscle damage, which will have contributed to mechanisms responsible for the changes observed. This study is unable to distinguish between this and other-related phenomena (e.g., motor drive [43]), and future studies should attempt to divorce such mechanisms from one another. We employed a submaximal single-limb hopping task on a sledge jump system, which has been used previously to investigate a number of neural and mechanical properties of the apparatus of the SSC [18,29]. Whilst this limits external validity, it does facilitate the intimate exploration of the mechanics and control of the SSC. Finally, we employed the method described by Dalleau, Belli, Viale, et al. [13] to measure lower limb stiffness. However, participants were instructed to hop with a static hip and knee, which was mostly successful (pilot testing demonstrated mean hip and knee excursion of  $6.3^\circ (\pm 2.3)$  and  $9.0^\circ (\pm 4.4)$ ). Given mean ankle excursion was around  $25^\circ$ , we believe this method can be considered to provide a surrogate measure of ankle stiffness.

Our findings have a number of potential clinical implications, surrounding firstly the pathogenesis, and secondly the management of lower limb overuse conditions (e.g., AT). These are based on the theory that deficits in muscle performance underpin the fatigue damage observed in such conditions [36], and fatigue resilience is a critical requirement in the rehabilitation of such conditions [31]. In the context of endurance sport (e.g., running) if reduced muscle performance is considered a risk factor for the development of 'AT', when combined with excessive activity (i.e., running), the musculotendinous unit eventually loses its resilience to repeated impact. Given that muscles act as 'buffers to passive tissues, (joint, tendon and bone) (add buffer ref), fatigue of the muscle will result in increased load being placed on the passive tissues, leading to tissue breakdown once the individual load tolerance of that tissue has been exceeded. In the same light, given the association between such pathologic conditions and associated motor dysfunction [14], a clinical paradigm exists where 'normalising' such motor performance deficits is an integral component of rehabilitation [31]. Our study provides affirming data, albeit in experimental terms, that a muscle's capacity to tolerate fatigue is a critical component of the pathogenesis and rehabilitation of lower limb overuse condi-

tions. We observed increased lower limb stiffness and reduced stretch amplitude, which is in contrast to many previous studies [15, 17, 22]. This most likely reflects a novel phenomenon that in the absence of metabolic fatigue, it is observed that motor control strategies exist where the lower limb responds to fatigue by increasing stiffness. Given that fatigue loading reduces tendon stiffness [3], motor performance may be the most important mediator of ankle mechanics in the presence of fatigue (i.e., fatigue resilience). If tendon fatigue was the dominant feature we would have expected an increase in stretch amplitude and a decrease in lower limb stiffness indicative of tendon yielding. During conditioning and rehabilitation, consideration should be given to ensuring an adequate motor performance profile of the plantarflexors to protect against fatigue damage and the predisposition to developing an overuse injury.

To our knowledge, fatigue as a perceived threatening stimulus in the same light as pain has yet to be considered. Our findings allow us to make the suggestion that this may be the case. In the presence of experimental and clinical pain, changes at multiple levels within the neuromusculoskeletal system produce a redistribution of activity within and between muscles, utilising a most commonly observed stiffening strategy, which is believed to be an attempt to protect the painful region [24]. Pain is a perceptual experience elicited in response to actual or threat of tissue damage. If fatigue is contextualised as a threatening stimulus, which is appropriate given that it elevates the risk of injury [46], and it is acknowledged that to experience pain in the presence of fatigue would be inappropriate from an evolutionary perspective, it would make sense for the neuromusculoskeletal system to elicit a protective motor response to such a threat, as has been demonstrated in this study.

In summary, we have shown that eccentric fatigue of the ankle plantarflexors alters SSC effectiveness. Specifically, lower limb stiffness increases mediated by an increase in muscle activity, whilst ankle stretch amplitude decreases. These changes occur in a manner that appears to be independent of systemic responses and that recognises that fatigue may pose a potential injury threat to the musculoskeletal system.

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