

Fatigue effect on muscle coactivation during inversion movement in females with chronic ankle instability

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HIGHLIGHTS

- Chronic ankle instability decreased muscle co-activation in all muscle pairs.
- Fatigue increased muscle co-activation in the FB- FL muscle pair.
- These results suggest that chronic ankle instability is an unprotected factor to the ankle.

ABBREVIATIONS

ANOVA	Analysis of variance
CAI	Chronic ankle instability
CAIT	Cumberland Ankle Instability Tool
EMG	Electromyography
FB	Fibularis brevis
FL	Fibularis longus
GL	Gastrocnemius lateralis
SENIAM	Surface Electromyography for the Non-Invasive Assessment of Muscles
TA	Tibialis anterior

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BACKGROUND: An ankle sprain is a relevant public health issue. Fatigue changes the neuromuscular response in people with chronic ankle instability (CAI).

AIM: To evaluate the muscle co-activation on people with chronic ankle instability using cross-correlation analysis.

METHOD: Twenty-four healthy women were selected and divided into stability and instability groups. Ankle sprain was simulated with a mechanical platform. Electrical muscle activity (fibularis brevis, FB; fibularis longus, FL; gastrocnemius lateralis, GL; and tibialis anterior, TA) and platform acceleration were recorded at 2KHz. Two sets of 8 right and eight left foot fall in random order were performed before and after the fatigue protocol. Fatigue protocol ended when the volunteer increased the test run time by 150% of the best round. Co-activation was calculated with cross-correlation. Agonist-agonist (FB-FL, FB-GL, and FL-GL) and agonist-antagonist (TA-GL, TA-FB, and TA-FL) pairs were evaluated. Statistical significance was $p < 0.05$.

RESULTS: Co-activation was lower for the instability group. Fatigue did not induce changes in 5 out of the 6 analyzed muscle pairs.

CONCLUSION: CAI is a factor of joint instability. Fatigue may not be relevant in altering joint stability. Therefore, interventions should be focused on enhancing joint stability.

KEYWORDS: Biomechanics | Motor control | Electromyography | Sprain | Injury | Cross-correlation

INTRODUCTION

Ankle sprains represent a significant public health concern. In the United State of America the incidence of ankle sprains is 2.1 per 1000 people per year¹. Furthermore, one-fifth of the Australian population experiences chronic musculoskeletal ankle disorders², while 61% of soccer players suffer from recurrent ankle sprains³.

Ankle sprains lead to ankle instability due to impaired muscular strength, a limited range of dorsiflexion motion, and delayed response of ankle muscles⁴. Persons with functional and mechanical ankle joint instability and history of two or more ankle sprains have chronic ankle instability (CAI)⁵.

Muscle co-activation is a variable extracted from the electromyographic signal that can be related to the ability to stabilize a joint. DeMers, Hicks, & Delp⁶ suggested that preparatory muscle co-activation could prevent injuries in individuals with chronic ankle instability (CAI). Lin, Chen & Lin⁷ proposed that the co-activation of tibialis anterior and fibularis longus decreases in individuals with CAI; Conversely, Suda, Amorim, & Sacco⁸ demonstrated similar co-activation patterns in ankle muscles of individuals both with and without ankle functional instability. Muscle co-activation is typically studied within agonist-antagonist muscle pairs^{9,10}; However, it is possible to classify muscle co-activation into three groups¹¹: 1) antagonist co-activation, involving simultaneous activation of agonist and antagonist muscles at the same joint; 2) synergistic co-activation, referring to muscles activating at the same joint to perform the same movement;

and 3) intramuscular co-activation, involving activation of different regions within the same muscle.

Fatigue alters the neuromuscular response in individuals with CAI. Fatigue impairs both physical and cognitive functions through interactions between performance and perceived fatigue¹². According to Gribble, Hertel, Denegar, & Buckley¹³, fatigue induced by lower limb isokinetic exercises impairs balance control in individuals with CAI, particularly when swaying in different directions. The performance on the Star Excursion Balance Test is impaired after fatigue in individuals with CAI¹³. However, fatigue resulting from strength training, proprioception training, or their combination does not significantly affect quiet standing postural control in individuals with CAI¹⁴. Therefore, does fatigue impact muscle co-activation in individuals with chronic ankle instability? To address this question, the purpose of this study is to assess the effects of fatigue on muscle co-activation in individuals with chronic ankle instability. The hypothesis posits that individuals with chronic ankle instability will exhibit reduced muscle co-activation during ankle inversion motion, and this reduction will be more pronounced under conditions of fatigue.

METHODS

This study is an observational study. The University Ethical Committee (n° 133.682) granted approval for this study. All participants were informed about the study's objectives, and they provided written consent to participate. The results of the CAIT test were utilized to categorize the participants into respective groups.

Subjects

The participants included twenty-four healthy women, who were divided into two groups: a stability group (n=12; 24.2±5.5 years old, 62.4±9.3 kg mass, 1.64±0.10 m tall) and an instability group (n=12; 21.3±2.8 years old, 60.8±11.1 kg mass, 1.62±0.10 m tall). The inclusion criteria for the instability group were as follows: pain resulting from ankle sprain, edema, or abnormalities during locomotion; experiencing episodes of ankle 'giving way' during daily activities or sports; sensations of joint instability; and a CAIT (Cumberland Ankle Instability Tool) score of <24 points⁵; 18-40 years old; and indoor football player for at least 3 years. The inclusion criteria for the experimental group were as follows: individuals aged between 18 and 40 years; indoor football players with a minimum of 3 years of experience. For the control group, inclusion criteria were: absence of pain resulting from ankle sprain, edema, or abnormalities during locomotion; no reported episodes of ankle 'giving way' during daily activities or sports; no sensations of joint instability; CAIT (Cumberland Ankle Instability Tool) score >24 points⁵; individuals aged between 18 and 40 years; indoor football players with a minimum of 3 years of experience. Exclusion criteria for both groups included: recent fractures or lower limb surgeries within the last six months; presence of any vestibular or nervous system conditions; musculoskeletal injuries and acute ankle sprains (<1 month) that would hinder the subject's ability to perform the test. The inclusion and exclusion criteria followed by our study were suggested by the study conducted by International Ankle Consortium⁵.

In Table 1, CAIT scores are presented. In the instability group, Student's t-test indicated a significant difference in CAIT scores between limbs (p=0.002). In the control group, CAIT scores were similar for both limbs (p=0.84).

Table 1. Mean and standard deviation of CAIT Score in both legs and both groups, as well as mean and standard deviation of the number of ankle sprains. In the Instability group, CAIT 1 pertains to the affected limb, while CAIT 2 pertains to the healthy limb. In the control group, CAIT 1 corresponds to the dominant limb, and CAIT 2 corresponds to the contralateral limb.

Group	CAIT 1	CAIT 2	n° of ankle sprains
Instability	18.5 ± 4.0	24.8±4.2	3.8±3.3
Control	28.1±1.6	27.9±2.6	0.6±0.7

Experimental setup

Participants stood on an inversion platform to undergo ankle inversion, constituting the inversion test. The participants were unaware of which foot would be subjected to inversion, and the order of falls was randomized. The individual responsible for operating the inversion platform was blinded to the groups. Surface electromyography (EMG) was used to measure the electrical activity of ankle muscles, while a triaxial accelerometer recorded platform motion. The inversion test was conducted both before and after the fatigue protocol.

Instruments

A mechanical platform (Figure 1) was employed to induce ankle inversion motion and simulate the motion associated with ankle sprain. The setup of the inversion platform was designed based on the works of Myers, Riemann, Hwang, Fu, and Lephart¹⁵ as well as Karlsson, Peterson, Andreasson, and Högfors¹⁶. It consisted of two rectangular boards measuring 320 mm x 220 mm, mounted on a foundation measuring 452 mm x 380 mm. These boards were linked by a hinge joint that facilitated a lateral inclination of 30° and

This is a safe angle that does not present any harm to the ankle joint^{15,16}. An attached pedal drive for each board was utilized to initiate this rotation.

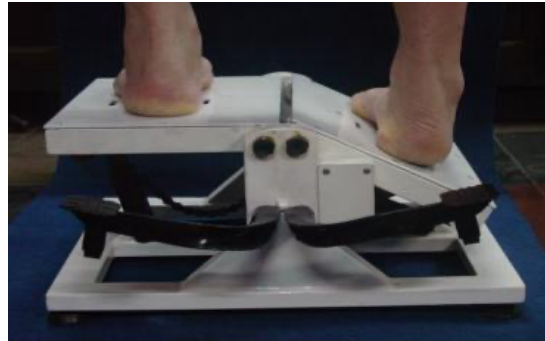


Figure 1. Inversion platform simulating the ankle sprain movement.

Surface electromyography and accelerometry data were collected using a 16-bit 8-channel signal acquisition system (EMG-800C, EMG System Brasil Ltda). Each channel was equipped with a common rejection module (>100 dB) and had an input impedance of 109 Ω . Acceleration and EMG signals were sampled at a rate of 2 kHz. For electromyography, disposable surface Ag/AgCl electrodes were positioned 20 mm apart on each muscle (fibularis brevis, FB; fibularis longus, FL; gastrocnemius lateralis, GL; and tibialis anterior, TA) as per the recommendations outlined by the Surface Electromyography for the Non-Invasive Assessment of Muscles (SENIAM) protocol¹⁷. We collected data only from the dominant limb (control group) and the limb affected by CAI (instability group); however, the electrodes were placed on both legs.

The data were processed using Matlab scripts (Matlab version R2015a). Raw EMG signals underwent initial filtering through a 20-500 Hz analog band-pass filter and were then pre-amplified by a factor of 100. The digitalized raw EMG signals were first demeaned, followed by filtering through a 200 Hz 4th order low-pass Butterworth filter, and a notch filter (60 Hz and harmonics) Butterworth filter. The rectified EMG signals were then normalized to 95% of the maximum signal amplitude. To determine the initiation and cessation of platform motion, the platform's acceleration data was utilized. The accelerometer was affixed to the inversion platform, and the acceleration signals were subjected to low-pass filtering (20 Hz 4th order Butterworth filter).

Procedures

Inversion test

Participants stood on the inversion platform and executed preliminary falls to acclimate to the equipment. To eliminate sensory feedback, their eyes and ears were covered. Subsequently, a total of 16 randomized falls were executed for each lower limb.

Fatigue protocol

Participants engaged in the Modified Southeast Missouri Agility Drill¹⁸ to induce muscular fatigue. This test entails a sequence of forward sprints, lateral shifts, and reverse running. The course, measuring 3.6 x 5.7 m in a rectangular shape, included the following sequence: a forward sprint, lateral shuffle to the right and then back, diagonal reverse running, followed by another forward sprint, lateral shuffle to the left and then back, and a final diagonal reverse run to the opposite side where the circuit originated. After completing the circuit, participants performed ten rapid countermovement jumps, as depicted in Figure 2.

Participants underwent this circuit three times to acquaint themselves with the routine and determine their preferred pace. Each round was timed. Participants aimed to complete as many rounds as possible until the round time reached 150% of their best round¹⁹. The time between rounds was 10 seconds, as determined by participants' subjective perception of exertion. Immediately after the fatigue protocol (within a maximum of 1 minute), participants repeated the inversion test. The second inversion test was completed within 5 minutes to mitigate any potential fatigue recovery effects. These intervals are suggested to prevent significant recovery of the effects of muscular fatigue²⁰.

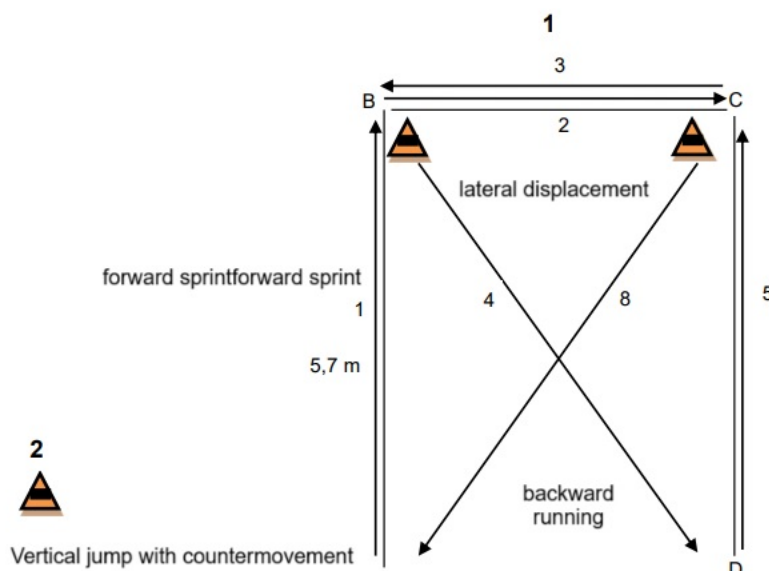


Figure 2. Illustration of the fatigue induction protocol was conducted in the following sequence: forward sprint (1), lateral displacement to the right outbound (2) and return (3), diagonal backward running (4) until reaching the contralateral side (D), repeating the same circuit but on the opposite side; and concludes with 10 countermovement jumps at station 2.

Variables

The epoch selected for the analysis of muscle co-activation ranged from 100 ms prior to the platform fall to 100 ms after. Muscle co-activation was quantified using cross-correlation²¹. The cross-correlation can be mathematically represented by Equation 1:

$$R_{xy}(\tau) = \frac{\frac{1}{N} \sum_{i=1}^N (x_i - \bar{x})(y_{i+\tau_{fs}} - \bar{y})}{\frac{1}{N} \sqrt{\sum_{i=1}^N (x_i - \bar{x})^2 \sum_{i=1}^N (y_i - \bar{y})^2}}$$

Where "x" and "y" represent the EMG time series. The muscle pairs were categorized into agonist-agonist and agonist-antagonist pairings²², and organized into synergistic co-activations (FB-FL, FB-GL, and FL-GL) as well as agonist co-activations (TA-GL, TA-FB, and TA-FL). The coefficient of determination, R², was assigned as the co-activation index, determined by evaluating the cross-correlation at zero lag for each pair.

Statistical analysis

Before the fatigue protocol, EMG activity was recorded for the muscles TA, FB, FL, and GL. In the instability group, the analyzed leg was the one affected by CAI. In the control group, the analyzed leg was the dominant one. This choice was made due to limitations in the channels of the acquisition system and the fact that after the fatigue protocol, changing leg cables would be challenging, potentially resulting in the loss of potential muscular fatigue effects.

We conducted a two-way analysis of variance (2-way ANOVA) to examine the impact of muscle fatigue (pre- and post-fatigue protocol) and groups (stability and instability) on the coactivation index. Subsequently, an analysis of variance (ANOVA) was conducted for each muscle pair. To discern variations among factor levels, the Tukey HSD post hoc test was utilized. The effect size was calculated using Cohen's d. A significance level of $p < 0.05$ was employed, and observed power as well as effect size was computed for all variables. Statistical analysis was executed using SigmaPlot software (version 11).

RESULTS

Table 2 presents the mean, standard deviation and confidence interval of co-activation index across groups and fatigue condition. There were no significant interaction effects in the TA-FC ($F_{1,361} = 3.8$, $p = 0.052$, power = 0.370), TA-FL ($F_{1,361} = 0.6$, $p = 0.443$, power = 0.050), TA-GL ($F_{1,361} = 1.1$, $p = 0.292$, power = 0.059), FC-FL ($F_{1,361} = 0.1$, $p = 0.106$, power = 0.050), FC-GL ($F_{1,361} = 0.4$, $p = 0.524$, power = 0.050) and FL-GL ($F_{1,361} = 1.3$, $p = 0.259$, power = 0.074).

The two-way ANOVA revealed that fatigue impacted the FB-FL ($F_{1,361} = 6.2$, $p = 0.013$, power = 0.621, $d = -3.53$, $r = -0.87$) pair and did not affect the TA-FB ($F_{1,361} = 2.1$, $p = 0.145$, power = 0.172, $d = 2.03$, $r = 0.71$), TA-FL ($F_{1,361} = 0.3$, $p = 0.568$, power = 0.050, $d = -0.80$, $r = -0.37$), TA-GL ($F_{1,361} = 1.3$, $p = 0.246$, power = 0.0851, $d = 1.62$, $r = 0.63$), FB-GL ($F_{1,361} = 3.7$, $p = 0.055$, power = 0.358, $d = -2.74$, $r = -0.81$), and FL-GL ($F_{1,361} = 0.8$, $p = 0.352$, power = 0.050, $d = -1.33$, $r = -0.55$) pairs. The Tukey's HSD post hoc test demonstrated that fatigue led to an increase in the cross-correlation index within the FB-FL pair ($p = 0.013$) (Figure 3).

The group affected the TA-FB ($F_{1,361} = 33.2$, $p < 0.001$, power = 1.000, $d = -8.16$, $r = -0.97$), TA-FL ($F_{1,361} = 27.2$, $p < 0.001$, power = 1.000, $d = -7.40$, $r = -0.96$), TA-GL ($F_{1,361} = 16.3$, $p < 0.001$, power = 0.985, $d = -5.67$, $r = -0.94$), FB-FL ($F_{1,361} = 8.6$, $p = 0.003$, power = 0.803, $d = -4.19$, $r = -0.90$), FB-GL ($F_{1,361} = 8.15$, $p = 0.005$, power = 0.772, $d = -4.01$, $r = -0.89$), and FL-GL ($F_{1,361} = 8.4$, $p = 0.004$, power = 0.787, $d = -4.10$, $r = -0.90$) pairs. Post hoc Tukey HSD test showed TA-FB ($p < 0.001$), TA-FL ($p < 0.001$), TA-GL ($p < 0.001$), FB-FL ($p = 0.003$), FC-GL ($p = 0.004$) and FL-GL ($p = 0.004$) pairs were higher in the stability group (Figure 4).

Table 2. Cross-correlation index (mean±standard deviation) and interval confidence in the muscle pairs of interactions, groups and fatigue condition (* $p < 0.05$).

Pair of Muscles	Cross-correlation index							
	Control		Instability		Group		Fatigue condition	
	Pre	Post	Pre	Post	Control	Instability	Pre	Post
TA-FB	0.04 ± 0.05 [0.03 - 0.05]	0.03 ± 0.03 [0.02 - 0.03]	0.01 ± 0.02 [0.01 - 0.02]	0.01 ± 0.01 [0.01 - 0.02]	0.03 ± 0.04 [0.03 - 0.04] *	0.01 ± 0.02 [0.01 - 0.02]	0.02 ± 0.04 [0.02 - 0.03]	0.02 ± 0.02 [0.02 - 0.02]
TA-FL	0.03 ± 0.03 [0.02 - 0.04]	0.03 ± 0.03 [0.02 - 0.03]	0.01 ± 0.01 [0.01 - 0.02]	0.02 ± 0.02 [0.01 - 0.02]	0.03 ± 0.03 [0.02 - 0.03] *	0.02 ± 0.02 [0.01 - 0.02]	0.02 ± 0.02 [0.02 - 0.02]	0.02 ± 0.02 [0.02 - 0.03]
TA-GL	0.03 ± 0.04 [0.02 - 0.04]	0.02 ± 0.02 [0.02 - 0.03]	0.02 ± 0.02 [0.01 - 0.02]	0.02 ± 0.02 [0.01 - 0.02]	0.03 ± 0.03 [0.02 - 0.03] *	0.02 ± 0.02 [0.01 - 0.02]	0.02 ± 0.03 [0.02 - 0.03]	0.02 ± 0.02 [0.02 - 0.02]
FB-FL	0.75 ± 0.14 [0.72 - 0.78]	0.79 ± 0.14 [0.76 - 0.82]	0.69 ± 0.17 [0.66 - 0.73]	0.74 ± 0.18 [0.71 - 0.78]	0.77 ± 0.14 [0.75 - 0.79] *	0.72 ± 0.18 [0.69 - 0.74]	0.72 ± 0.16 [0.69 - 0.74]	0.76 ± 0.17 [0.74 - 0.79] *
FB-GL	0.75 ± 0.16 [0.71 - 0.78]	0.79 ± 0.17 [0.76 - 0.83]	0.71 ± 0.17 [0.67 - 0.74]	0.73 ± 0.18 [0.70 - 0.76]	0.77 ± 0.17 [0.74 - 0.80] *	0.72 ± 0.17 [0.69 - 0.74]	0.72 ± 0.17 [0.70 - 0.75]	0.76 ± 0.17 [0.73 - 0.78]
FL-GL	0.85 ± 0.11 [0.83 - 0.88]	0.85 ± 0.12 [0.82 - 0.88]	0.79 ± 0.17 [0.76 - 0.83]	0.82 ± 0.14 [0.80 - 0.85]	0.85 ± 0.12 [0.83 - 0.87] *	0.81 ± 0.16 [0.79 - 0.83]	0.82 ± 0.15 [0.80 - 0.84]	0.84 ± 0.13 [0.82 - 0.85]

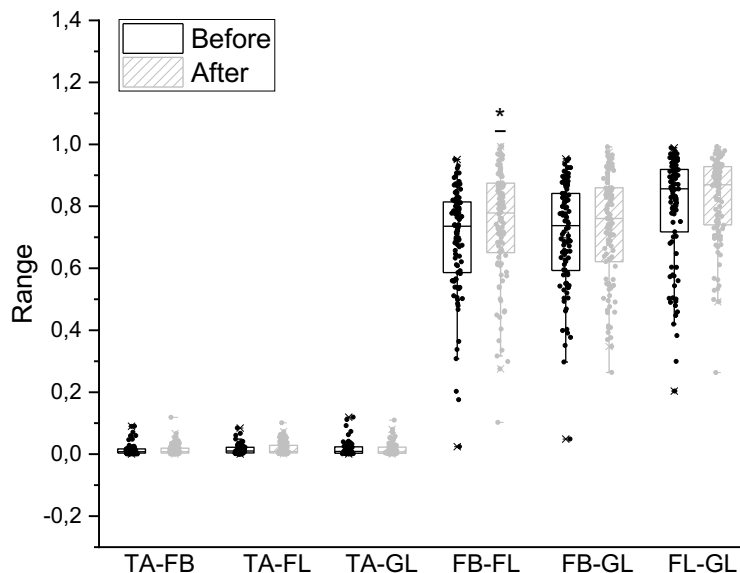


Figure 3. Cross-correlation index (mean±standard deviation) in the pair of muscles before and after fatigue protocol (* $p < 0.05$).

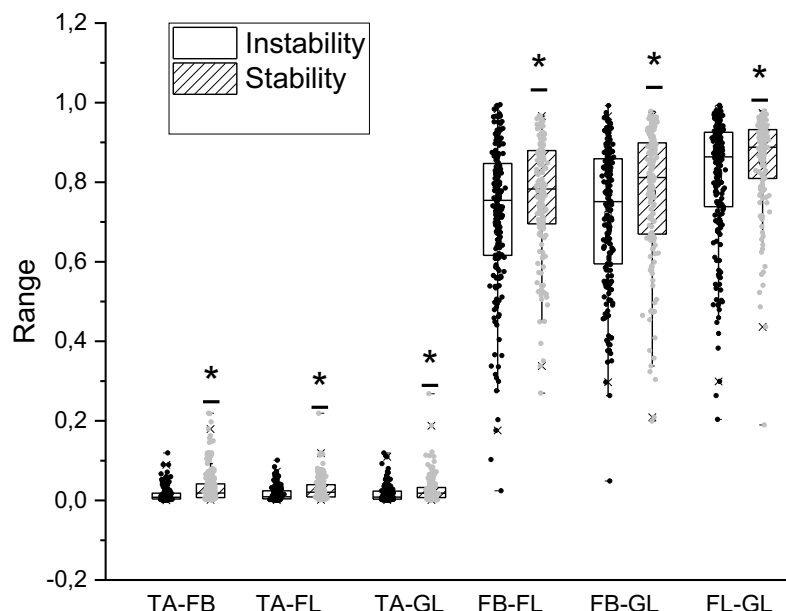


Figure 4. Cross-correlation index (mean±standard deviation) in the pair of muscles into groups (* $p < 0.05$).

DISCUSSION

We assessed the effect of muscle coactivation fatigue in individuals with CAI. Muscle coactivation during ankle inversion simulation was investigated. Coactivation enhances stiffness and stability to protect the joint^{23,24}. However, muscle coactivation can also increase in unstable situations to balance the center of mass.

Individuals with CAI exhibited lower antagonistic and synergistic coactivations. This finding is significant as it suggests a reduced ability to generate joint stiffness, potentially leaving the ankle joint less protected in the primary mechanism of ankle sprain, which is inversion. The literature presents conflicting data on muscle coactivation of antagonist muscle pairs in individuals with CAI. Lin, Chen & Lin⁷ demonstrated decreased TA-FL coactivation and similar TA-GL coactivation between individuals with and without CAI during running or jumping. Souza²⁵ observed lower levels of coactivation in anterior tibial and fibular muscle pairs. Suda, Amorim & Sacco⁸ found no differences in muscle coactivation in individuals with functional ankle instability. A decrease in antagonistic coactivation in anterior tibial-soleus and anterior tibial-short fibular pairs can be observed during short and medium latency in individuals with CAI²⁵. In our findings, CAI reduces the protective capacity through muscle coactivation, which is expected in individuals with some form of injury^{6,26}.

Fatigue affected synergistic coactivation in the FB-FL muscle pair. The effects of fatigue on muscle coactivation are conflicting in the literature. Coactivation impairs force generation capacity in individuals with CAI²⁷. However, fatigue increased antagonist muscle coactivation at the knee and ankle²⁸, enhancing coactivation in hamstring muscles during knee extensions on an isokinetic dynamometer²⁹. Balance training, proprioceptive training, and strength training are effective in treating and improving certain aspects in individuals with chronic ankle instability³⁰.

Our findings allow us to comprehend that the ankle joint in individuals with CAI requires greater stability. Consequently, it is imperative to implement an intervention that can mitigate the effects of CAI on ankle coactivation. Strength training appears to enhance coactivation levels in older adults³¹, but there is limited information on strength training approaches to enhance muscle coactivation in individuals with chronic ankle instability. Balance, strength, and proprioceptive training improve certain balance-related aspects in individuals with CAI³⁰. Alternatively, in terms of intervention strategies, a meta-analysis supported the use of a brace to prevent recurrent ankle sprains in individuals with or without CAI³².

The various methods employed for calculating muscle coactivation with EMG complicate their comparison, and a standardized method for assessing muscle coactivation is lacking³³. Few studies employ cross-correlation to estimate muscle coactivation. Milner³³ utilized cross-correlation to verify the absence of cross-talk. De Luca & Mambrito³⁴ employed cross-correlation to assess the firing rate of

motor units in pairs of antagonist muscles and suggested that when a rapid compensatory force is required and there is task uncertainty, muscle coactivation arises.

CONCLUSION

CAI reduced muscle coactivation in all pairs, confirming this hypothesis and implying a decreased protective factor at the ankle. Fatigue increased muscle coactivation only in the FB-FL pair. This pair emphasizes the eversion movement, counteracting the primary mechanism of ankle sprain, namely inversion. However, the antagonist muscle pairs remained unchanged, indicating that fatigue did not render them more vulnerable.

What do these findings suggest for clinical professionals dealing with patients with CAI? There is a need for the use of accessories or the implementation of training programs that can enhance ankle stability, and fatigue will not worsen stability, regardless of whether the patient has CAI or not.

Our data pertains to muscle coactivation in the temporal domain, and it would be insightful to explore the behavior of muscle coactivation in the frequency domain. This type of analysis sheds light on how the nervous system governs movement and the implications that CAI might have on the communication between the nervous system and muscle coactivation. This understanding holds potential relevance for clinical practitioners, as it could offer valuable insights into designing interventions and training programs that optimize ankle stability and mitigate the impact of fatigue, both for individuals with CAI and those without.

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