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Original Article

# The influence of fatigue in evertor muscles during lateral ankle sprain

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ABSTRACT

Ankle sprain in plantarflexion and inversion is one of the most common injuries occurring in daily activities and sports. Although acute symptoms may resolve quickly, many individuals have reported persistent pain and instability. Thus, understanding the factors that contribute to the occurrence of this type of injury is extremely important. Although sprains are multifactorial, a relationship can be established between sprain and fatigue. Therefore, the present study examined the latency and intensity of activation of the peroneus longus and brevis muscles under conditions of fatigue. Twenty-three women participated in the study, including 12 with functional instability of the ankle and 11 without a history of sprain. To induce fatigue, the volunteers maintained the force of eversion and plantarflexion at 70% of the maximum voluntary isometric contraction for as long as possible until a 10% decline in the rated force occurred. Ten simulations of ankle sprains were performed before and after fatigue at random for each side, with simultaneous recordings of the electromyographic signals, using a simulator platform for inversion sprain and plantarflexion. As a result, after fatigue, no change in latency was observed. However, a reduction in the intensity of contraction of the muscles analyzed in both groups was observed. Neuromuscular control was concluded to be compromised in situations of fatigue, while differences in muscle behavior were not observed between stable and unstable ankles.

## 1. Introduction

Ankle-foot complex lesions often occur in daily life activities [1], especially during athletic activity [1,2], and data from the literature indicate that lateral sprain accounts for the largest proportion of injuries of this joint [3]. Although acute symptoms may resolve quickly, many individuals have reported persistent pain and instability [4]. This persistent condition is referred to as chronic ankle instability and may originate from local mechanical or functional changes [5]. Functional ankle instability (FAI), which is the focus of this study, is a condition for which subjects report experiencing frequent episodes of ankle instability and sprain [6]. These symptoms are usually related to sensory motor alterations after an initial injury [7]. Studies indicate that people with FAI often present delayed response times of protective muscle activity in the region and reduced postural control and joint stability [8,9]. Thus, considering the high incidence of sprains and their repercussions, understanding the factors that contribute to the occurrence of this type of injury is extremely important.

The scientific literature suggests that intrinsic factors such as height, body mass, previous history of ankle injury, ligamentous laxity [10,5], sex (women are more frequently affected than men) and fatigue [11], or extrinsic factors such as the use of different shoes configurations or sole thicknesses [12–14] can be elements of risk for the development of sprains. In this work, we are interested in better understanding the specific role of fatigue in the occurrence of sprains.

Fatigue can be defined as “failure to maintain the required or expected force” [15] or failure to “continue working at a given exercise intensity” [16]. Despite these definitions, fatigue is a very complex phenomenon involving physiological processes that occur in structures from the motor cortex to the muscle contractile proteins, and the origin of fatigue can be divided into the following categories: central and peripheral [17]. Central fatigue is characterized by disturbances in neuromuscular transmission between the Central Nervous System (CNS) and the muscular membrane [18,19], reflecting a state in which actions and cognitions require increased effort or performance, which is interrupted without evidence of a reduction in peripheral motor factors

*Abbreviations:* FAI, functional ankle instability; CNS, central nervous system; SG, stability group; IG, instability group; PL, peroneus longus; PB, peroneus brevis; MVIC, maximum voluntary isometric contraction; RMS, root mean square

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[20]. Consequently, the recruited number of motor units and their firing rate decrease [17]. On the other hand, peripheral fatigue results from intrinsic muscle alterations due to neural, mechanical or energy events [21], which include changes in neuromuscular transmission and the propagation of the action potential, as well as decreased contractile strength of muscle fibers [17]. In this manner, from central and/or peripheral alterations, muscle fatigue can cause variations in proprioception [22] and motor control [23].

Given this background, studies indicate that fatigue can affect the control of the dynamic stabilizers of the ankle joint, such as the peroneus muscles, and their ability to generate evertor torque may be impaired or altered [24]. Notably, these are the main muscles that generate evertor strength to oppose the main mechanism of local injury, which is inversion associated with plantarflexion [2].

To investigate this assumption, studies have verified the effect of peroneus muscle fatigue on ankle sprain by analyzing variables from electromyographic signals, such as the amplitude and latency of a signal during sprain simulation. The amplitude of a signal represents the level of muscle activation, which is commonly analyzed by its Root Mean Square (RMS) value [25], and the latency of a signal represents the response time between a disturbance and muscle electrical activity at the beginning of muscle activation [26].

For instance, a study observed increased latency after fatigue by means of a concentric dynamic contraction protocol with elastic resistance to induce fatigue [27]. In another study, latency was unchanged after fatigue induced by concentric contractions performed on an isokinetic dynamometer at a speed of 60°/s [11]. In an experiment similar to the previous study, fatigue was induced through eccentric contractions at a speed of 120°/s, and unlike the results of other studies, reductions in the electromyographic signal amplitude and latency of the peroneus longus (PL) and peroneus brevis (PB) muscles were observed after fatigue [24]. Other authors found no change in latency but noted that the amplitude of the electromyographic signal of the peroneus muscles was reduced after concentrically and isometrically induced fatigue controlled by a force transducer [28].

Thus, considering the impacts of FAI and fatigue on global and local neuromuscular control, the evertor muscles, the PL and PB, are important stabilizers during lateral sprains. This study aims to analyze the contribution of these muscles to dynamic stabilization of the ankle during simulation of sprain in inversion and plantarflexion before and after the induction of local muscle fatigue. In addition, the study compared people without a history of sprains to people with FAI to verify how local dynamic stabilization is compromised under fatigue conditions in these two populations.

## 2. Method

### 2.1. Sample

A total of 23 women were selected and classified as physically active (performance of moderate activities for at least thirty minutes daily more than five times a week) and were subjected to the protocol using the International Physical Activity Questionnaire–version 6 [29]. Only women were selected because a previous study reported that men and women respond differently under fatigue conditions [11]. Additionally, the included women did not have a history of fracture or surgery of the lower limbs within the last six months or vestibular and/or neurological disturbances. The volunteers were divided into two groups in the following manner: 11 women without a complex ankle joint injury in the last 12 months were included in the Stability Group (SG) (mean and standard deviation: age 27.09 years ± 3.55, body mass 57.61 kg ± 6.94, height 1.61 m ± 0.05) [11], and 12 women with functional instability of the ankle classified by the Cumberland Ankle Instability Tool-CAIT version were included in the Instability Group (IG) (mean and standard deviation: age 27.00 years ± 3.67, body mass 62.51 kg ± 7.31, height 1.66 m ± 0.05) [30]. The SG and IG groups were similar in age, height and body mass according to the Mann–Whitney statistical test.

For standardization, the electromyographic activities of the muscles on the dominant side in the SG were analyzed, which was considered to be the side that an individual would use to kick a ball. For the IG, the limb with the ankle classified as functionally unstable was analyzed [31].

### 2.2. Experimental procedure

The procedures proposed in this study were approved by a Research Ethics Committee of the State University of São Paulo (opinion no. 900.407), and all volunteers provided consent after reading the Informed Consent Form. All work was carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki).

First, the participants were invited for a day to undergo assessments of anthropometric variables (body mass and height) and to familiarize themselves with the procedures adopted and the process of induction of fatigue [32].

Data collection was carried out after the familiarization session. Initially, the skin was prepared with local trichotomy, asepsis with 70% ethanol, removal of dead cells with sandpaper and local asepsis with alcohol [25]. Then, bipolar surface electrodes (MEDITRACE® 200, Ag/AgCl, 20 mm in diameter, circular) were placed on the PL and PB muscles (interelectrode distance of 20 mm) [33].

For placement of the electrodes on the PL, the participants were in a supine position with medial rotation of the knee. The electrodes were placed at 1/4th the distance along the line from the edge of the fibular head to the lateral malleolus. For placement of the electrodes on the PB, the participants remained in the same position. The electrodes were placed anterior to the PL tendon at 1/4th the distance along the line from the edge of the lateral malleolus to the head of the fibula [33].

Then, the volunteers positioned themselves in an orthostatic position on the platform, and their feet were fastened with straps across the midfoot region. The participants were instructed to distribute their weight evenly between the lower limbs, and then 10 sprain simulations were randomly performed. The volunteers wore blindfolds and earplugs during the test to minimize interference from external stimuli [1,34] (Fig. 1).

Subsequently, the procedure to induce fatigue was started (Fig. 2), which consisted of (1) positioning, (2) determination of the maximum

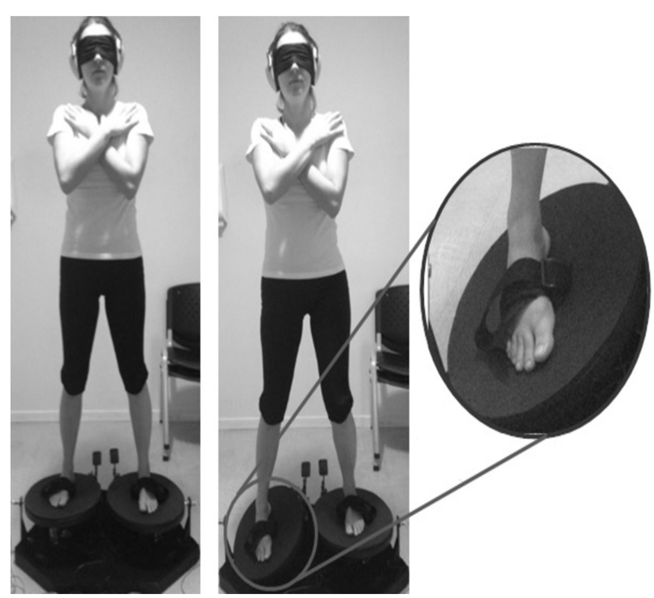

**Fig. 1.** Positioning during ankle sprain simulation.

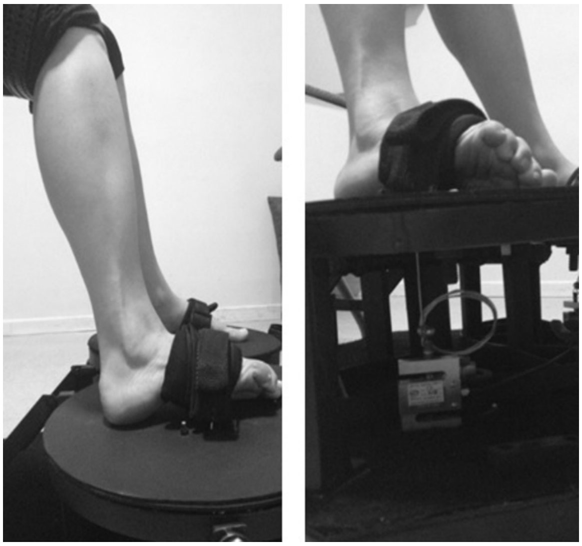

**Fig. 2.** Procedure for induction of fatigue on the peroneus longus and brevis muscles.

voluntary isometric contraction (MVIC) of the PL and PB, and (3) induction of fatigue. Each step is described below:

1) Positioning: the volunteer was seated with the joints of the hip and knee in 90° of flexion. Two straps were used to secure the lower limbs, with one next to the hip and another above the knee, to stabilize the proximal joints. The feet were fastened to the platform by the straps placed across the midfoot region.
2) Determination of the MVIC of the PL and PB: in this position, the participants were instructed to perform maximal eversion and plantarflexion for 5 s. This process was performed three times separated by a rest period of 45 s. The best value of the MVIC from the attempts was taken as a parameter for the induction of fatigue ;[35] in addition, this value of the MVIC was used later for electromyographic data normalization [36].
3) Induction of fatigue: the volunteers were instructed to perform eversion and plantarflexion and to maintain the strength at 70% of the MVIC [37]. Fatigue was defined as a decrease greater than 10% of the stipulated force [38] or voluntary withdrawal.

Throughout this process, the volunteers received visual feedback and verbal stimuli [32]. Then, another 10 simulations occurred randomly between the ankles.

To analyze the reliability and reproducibility of the measurements obtained, a test and retest procedure was conducted on 2 different days with 5 volunteers.

### *2.3. Materials*

A platform was built with two independent circular mobile plates (Fig. 3a) mounted on nonparallel axes (Fig. 3b), which allow the combined movement of inversion and plantar flexion, to simulate the ankle sprain. The maximum amplitude was 30° from the position parallel to the ground. The drive system included a pedal (Fig. 3c). Two inductive presence sensors (SENSE®) were placed in brackets attached to the base (Fig. 3d), which were arranged on either side of the platform plate and connected to the recording device such that the start of the fall of the plate could be determined during the simulation of the sprain. This design enabled later synchronization with the electromyographic signal to determine the latency of the muscles for analysis.

The platform also enabled the induction of fatigue. To this end, two load cells (Model MTS-1 50 kg, thickness 13 mm, height 64 mm and width 51 mm - BSL®) were placed at the base of the platform just below the lateral rim where the foot rests (Fig. 3e). A strap was used to attach the feet (Fig. 3f). One end of a steel cable was attached to the side edge of the strap, and a load cell was fastened to the other end of the cable (Fig. 3g) bilaterally. The output signal of the cells was connected to the recording device, which served as visual feedback for control of the maintenance of the force during the fatigue induction process.

The EMG 830 C (EMG SYSTEM®) data acquisition system, which consists of eight channels, was used to record the electromyographic signal. Each input channel has a configurable 100-fold amplifier, a Common Mode Rejection > 100 dB and $10^9$ Ohms of input impedance. The system has a Butterworth-type hardware filter with two poles that operate as a bandpass of 20 to 500 Hz. The amplified and filtered signals were digitized by an A/D converter with a 16-bit resolution. Two channels of the bioamplifiers received the raw electromyographic signal of the PL and PB, two received signals from the inductive presence sensors, and the other two received signals from the load cells. Data collection was managed by EMG lab software (EMG SYSTEM®), which allows recording with a sampling frequency of up to 2000 Hz, as used in this study.

### *2.4. Mathematical treatment and statistical analysis*

The MATLAB® program was used for data analysis. The mathematical package calculated the following variables: (1) LATENCY, i.e., the value of latency immediately after the beginning of the fall of the platform, which exceeded the average signal of approximately 50 ms by 3 standard deviations prior to the fall of the platform [39]; and (2) the RMS, which was calculated up to 200 ms after the fall of the platform to observe the amplitude of the electromyographic signal during the simulation of sprain.

For latency determination, the muscle signals analyzed passed through a bandpass filter of 30–300 Hz (6th order Butterworth), and then filtering was performed through a low-pass filter of 50 Hz (2nd order Butterworth), followed by rectification of the signal. The RMS value was normalized by the average interval of 4 s of the MVIC [36].

Furthermore, the mathematical package allowed characterization of fatigue through the Power Spectral Density function for calculation of the median frequency.

Nonparametric statistics were selected for the data analysis because the distribution of the data did not follow a normal distribution. Thus, comparative analysis of the variables was conducted (latency and RMS) before and after fatigue induction in each group through the Wilcoxon test and between the SG and IG through the Mann–Whitney test. A statistically significant P-value $< 0.05$ was adopted.

## 3. Results

Based on the proposed analyses, no differences were observed in the latency of the PL and PB muscles due to the sprain simulation before and after the induction of fatigue. However, these muscles showed a reduced intensity of activation as indicated by a decrease in RMS values under fatigue conditions compared to the initial sprain simulations when this muscle group was not fatigued. In addition, no differences were observed between the stability and instability groups in any of the comparisons performed (latency and RMS, before and after fatigue). These results are shown in the Figs. 4 and 5.

To determine that fatigue was achieved, the values of the median frequency of contraction for all individuals at the start time and at the end time of the fatiguing contraction were compared, and average reductions of 15.6% and 14.4% were noted for the PL and PB, respectively, reflecting statistically significant differences, $P < 0.05$.

Furthermore, to analyze the reliability and reproducibility of the measurements obtained, a test and retest procedure in five volunteers was performed. Spearman correlation coefficients from the test and retest procedures were verified (Latency: PL ρ between 0.700 and


**Fig. 3.** Simulator platform for ankle sprain and fatigue induction. Mobile plates (a); nonparallel axes (b); drive pedal (c); inductive presence sensor (d); load cell (e); strap to attach the feet (f); and steel cable (g).

0.947; PB ρ of 0.700 to 0.900 / RMS: PL ρ of 0.700 to 0.900; PB ρ = 0.900).

## 4. Discussion

Understanding how fatigue can influence the dynamic stability of the ankle joint is extremely relevant and of clinical interest considering that a better understanding may allow identification of new approaches to prevent the initial injury and reduce the risks of recurrent sprains. In this study, the initial hypothesis proposed was that after fatigue, a reduction in the intensity of muscular contraction and an increase in latency would be observed and that such changes would be more prominent in people with FAI. The hypothesis of reduced muscle activity of the PL and PB muscles after fatigue was confirmed. However, the latency of these muscles was not influenced by fatigue, and no differences in muscle behavior were observed between stable and functionally unstable ankles.

Previous studies have also analyzed the response of the peroneus muscles to fatigue during simulation of ankle sprain in relation to latency and/or activation level [11,24,27,28]. Rodrigues et al. analyzed the latency of the peroneus muscles using a fatigue induction protocol similar to that in the present study and did not observe latency changes during inversion sprain simulation with a maximum amplitude of 30° when evaluating participants without a history of injury to the ankle joint complex [28]. Jackson et al. found a reduction in the latency of the PL and PB after fatigue in healthy individuals, which was not observed by any other study. The protocol consisted of eccentric dynamic contractions of the peroneus muscles on an isokinetic dynamometer (at a speed of 120°/s), and the simulation was accomplished through abrupt inversion of 25–30°. With respect to the intensity of contraction of these muscles after fatigue, the result was similar to that found in this research, and these authors suggested that fatigue can impair an individual's ability to correct an unexpected inversion movement due to a reduction in evertor muscle activity [24]. In a similar experiment, fatigue was induced in healthy participants by means of concentric dynamic contractions of the ankle evertor with the use of an isokinetic dynamometer (at a speed of 60°/s). Latency was not influenced by fatigue during the simulation of inversion sprains at 20°. With respect to the level of muscle contraction, these authors observed an increase in muscle contraction in women and a decrease in contraction in men for the PL after the induction of fatigue and proposed that this finding may be due to differences inherent in the physiology and biomechanics of how each gender behaves under fatigue conditions [11]. In contrast to the results of the current research, one study found increased latency

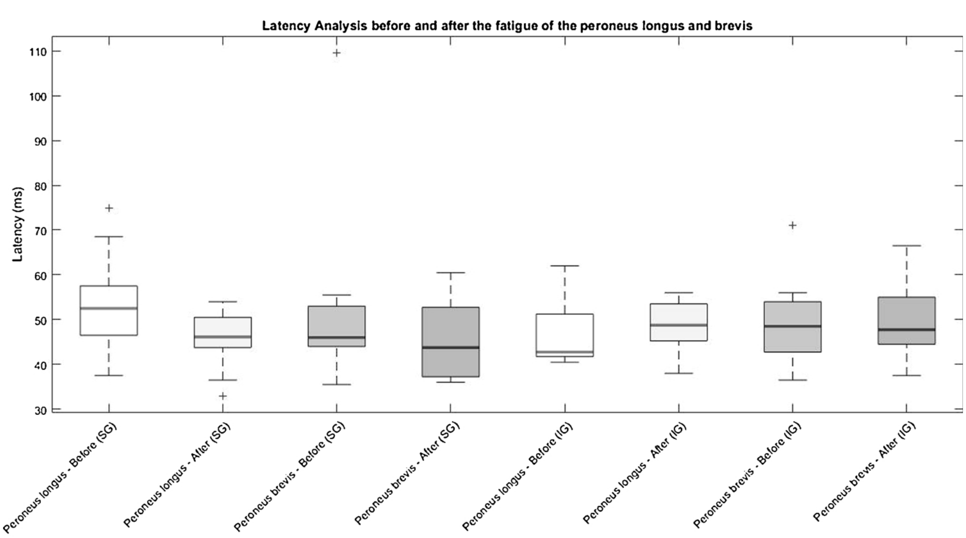


**Fig. 4.** llustration of the median and 1st and 3rd quartiles of the latency before and after fatigue of the peroneus longus and brevis muscles in the SG and the IG.

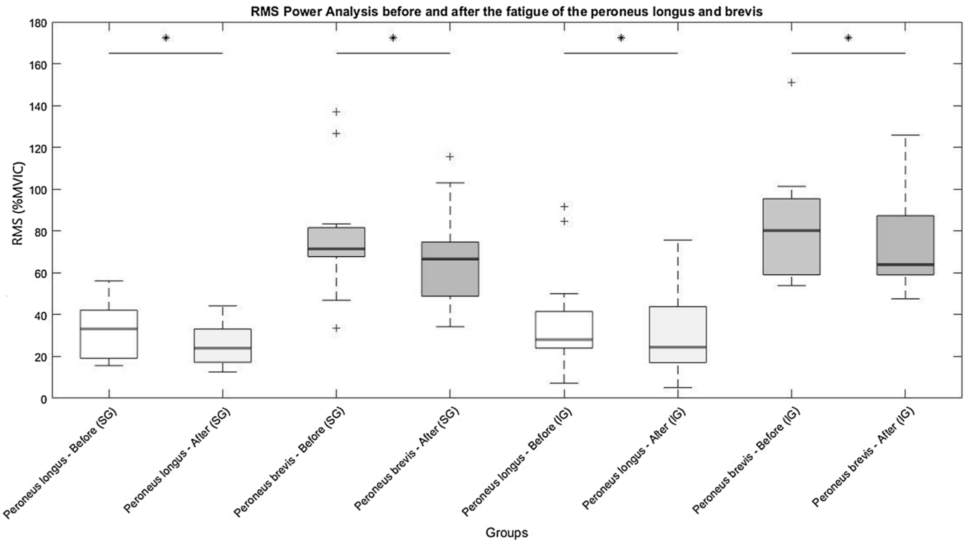


**Fig. 5.** Illustration of the median and 1st and 3rd quartiles of the RMS before and after fatigue of the peroneus longus and brevis muscles in the SG and the IG.

during simulation of inversion sprains at 20° after training with concentric dynamic contraction of the peroneus muscles using elastic resistance in healthy individuals [27].

Notably, comparisons with prior studies are difficult due to methodological differences between the experiments, as described above, which can also explain the discrepancy between the results.

Given the above information, the reason that fatigue does not influence the latency of the peroneus muscles is that the CNS is often able to compensate for the effects of neuromuscular fatigue [37] by recruiting new motor units or activating previously inactive muscles [38]. Additionally, some authors proposed that the increased sensitivity threshold of fatigued muscle spindles can be reconciled by an increase in the contribution from other sensory receptors involved in postural adjustment [39], which may help explain why the latency of the analyzed muscles was not affected by fatigue.

In addition, the latency during sudden inversion has been suggested to be too large to provide protection to the ankle joint complex. Substantial active torque is observed at 150 ms after sudden inversion [39], suggesting that latency may not be the central and main factor that guarantees or can maintain local stability under vulnerable conditions during fatigue since the total amplitude of inversion movement occurs from 60 to 110 ms [40,41]. Thus, an active response to abrupt inversion cannot occur in time to protect the capsule and lateral ligaments [39]. However, the tension generated by contraction of the musculature should be considered at the critical point and must be sufficiently large to resist the applied load [39,42]. Therefore, the movement required to oppose the injury event may reasonably be more related to the contraction intensity of these muscles than to their response time.

The intensity of muscle contraction influences the level of joint stiffness and the degree of local stability [43]. Thus, considering the importance of muscle activation to generate adequate movement and control unexpected inversion of the ankle-foot complex [39,42], the signal amplitude was analyzed using the RMS, which represents the amount of muscle activation. We found that fatigue affected the intensity of activation of the PL and PB muscles as indicated by a reduced RMS.

This reduction in the intensity of muscle contraction observed after fatigue may be related to changes in the neural input that activates the muscle as indicated by a decrease in the number of recruited motor units and a reduced frequency of triggering of motoneurons [17]. This pattern reduces a muscle's ability to generate force, indicating the possible influence of so-called central fatigue [44]. Peripheral fatigue can also explain this result because this form of fatigue involves a reduction in the propagation speed of the action potential through the neuromuscular junction and along the muscle fiber [45], in addition to excitation-contraction coupling failure, which may be related to the calcium levels available to participate in muscle contraction [46]. Thus, one can consider that fatigue changes the degree of activity of the PL and PB and consequently affects the level of local joint stability.

Another approach to identify possible risk factors for the development of sprains is to study people with and without chronic ankle instability [43,41,47]. Accordingly, the present study investigated the behavior of the PL and PB muscles between healthy and functionally unstable ankles under conditions of fatigue. From the comparison between stable ankles and functionally unstable ankles, no differences were found in any of the variables analyzed, both under resting conditions and upon fatigue, for both of the muscles investigated.

In the literature, no consensus has been established regarding the muscular behavior between stable and unstable ankles during simulation of lateral sprains. The results of some studies are consistent with the results obtained in this experiment [48,49], while other studies have reported different results [31,50]. This disparity between results can be justified because different criteria for inclusion and definitions of ankle instability were used [6], reflecting the variability of the samples among studies and thus complicating comparisons. Furthermore, none of the studies analyzed the peroneus muscles upon abrupt inversion and plantar flexion at 30°, and no previous comparison between stable and unstable ankles under fatigue conditions, as performed in this study, was identified.

Freeman was the first to substantiate functional instability, which proposed that capsular and ligament lesions of the ankle-foot joint complex may imply a lack of partial or permanent afferents [51]. This lack of sensory input would justify the occurrence of FAI due to a delay and reduction of the reflex response in the evertor muscles, thus affecting the ability of these structures to oppose an unexpected inversion movement [52]. However, in recent years, the literature has proposed that a lack of articular afference may not be the process by which individuals develop functional instability and that other factors such as central motor programming may be more relevant [53]. A study conducted by Caulfield et al. showed no change in the electromyographic activity of the peroneus muscles after initial contact with the ground in individuals with unstable ankles. However, this same research showed that before contact with the ground during the landing of a jump, these subjects exhibited reduced electromyographic activity of the PL muscle. Thus, the authors concluded that individuals with functional ankle instability showed changes in motor control, providing evidence of modifications in central programming [54], which is consistent with the hypothesis proposed by Riemann et al. that changes in central motor control may be more important than articular deafferentation and the reflex response of the peroneus muscles [53]. Therefore, the differences between stable and unstable ankles can be reasonably assumed to be more related to the capacity for muscle activation in a preparatory manner for an injurious event rather than the muscles' response time or level of activation during lateral sprain of the ankle.

Thus, given the results of the present study, FAI cannot be a consequence of a delay in the latency or the level of activation of the peroneus muscles upon sudden inversion and plantarflexion; that is, the possible proprioceptive changes found in people with FAI did not modify muscular behavior compared to healthy individuals, even in conditions of fatigue.

## 5. Conclusion

Neuromuscular control was found to be compromised under conditions of fatigue due to a reduction in the level of activity of the PL and PB muscles, which can consequently change the conditions of joint stability and predispose individuals to the occurrence of injuries. However, when people with stable ankles and with FAI were compared, differences in muscle activity or latency were not observed, both before and after fatigue.

## Brief summary

*Points that are already known*

- Fatigue can affect neuromuscular control.
- Peroneus muscles are responsible for protecting the ankle-foot complex from the main mechanism of local lesions, which is inversion associated with plantar flexion.
- People who have already suffered sprains can present with damage to mechanoceptors, which causes a change in neuromuscular control and results in persistent symptoms in chronic ankle instability.

*Points raised by the present study*

- Local fatigue affects neuromuscular control.
- Fatigue can alter the degree of activity of the peroneus longus and brevis muscles and consequently affects the level of local joint stability
- Joint stability is compromised under fatigue conditions, which may predispose individuals to the occurrence of injuries.

## Conflict of interest

The authors declare that they have no competing interest.

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