Difference in Response Latency of the Peroneus Longus Between the Dominant and Nondominant Legs

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Context: The latency of the peroneus longus in response to an inversion perturbation is a key component in the prevention of lateral ankle sprains. In addition, the dominant ankle is sprained more frequently than the nondominant ankle, but the cause of this has not been examined. Objective: To investigate the combination of these 2 research-supported statements, the purpose of this study was to use an inversion perturbation that replicates the mechanism of a lateral ankle sprain to determine whether there is a difference in the latency of the peroneus longus between the dominant and nondominant legs. Design: Repeated-measures single-group design. Setting: University laboratory. Participants: 15 physically active healthy volunteers with no previous history of an ankle sprain or lower extremity surgery or fracture. Interventions: Outer sole with fulcrum was used to cause 25° of inversion at the subtalar joint on landing from a 27-cm step-down task. Participants performed 10 trials on both the dominant and nondominant leg. Main Outcome Measures: 2 latency measures of the peroneus longus of both the dominant and nondominant leg, calculated as the amount of time from the moment of touchdown of the fulcrum until muscle activity exceeded 5 and 10 SD above baseline muscle activity. Results: The latency of the peroneus longus of the nondominant leg was significantly shorter when using both 5 SD ($F_{1,14} = 9.34, P = .009, d = .895$) and 10 SD ($F_{1,14} = 18.56, P = .001, d = .920$) above baseline muscle activity. Conclusions: This difference in latency may be a result of the different demands placed on the dominant and nondominant legs during activity and may predispose the dominant ankle to a greater number of ankle sprains than the nondominant ankle.

Keywords: reflex, ankle sprain, landing

The lateral ankle sprain is one of the most common injuries in athletics, as initially identified by Garrick,¹ and the peroneus longus provides the primary dynamic defense against it.²,³ When the subtalar joint is forced into inversion, the mechanoreceptors in the lateral ankle ligaments are activated,⁴ which increases the sensitivity of the muscle spindles found in the peroneus longus. This increased sensitivity in conjunction with the inversion motion causes a reflexive contraction of the peroneus longus.⁵ This reflex response of the peroneus longus helps control

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the inversion moment and protects against a lateral ankle sprain. However, this reflexive activation of the peroneus longus does not occur until after the inversion moment has already begun because of the time lag in the monosynaptic pathway between the muscle spindles and the alpha motoneurons. This delay between the inversion perturbation and onset of the response of the peroneus longus has been called the peroneal latency or closed-loop reflex response. Up to this point the preferred method of examining the amount of time it takes the peroneus longus to respond when the ankle is forced into inversion or a combination of inversion and plantar flexion has been the use of a tilt platform. However, the validity of this model has been questioned, indicating the need for a new methodology to replicate the mechanism of a lateral ankle sprain.

It has been shown that athletes place different demands on the left and right limbs; for example, soccer players use the right leg to kick a soccer ball 80% of the time, and most athletes place a greater demand on the dominant limb. Research has demonstrated that among elite soccer players, the knee flexors of the dominant leg were significantly weaker than those of the nondominant leg, and that among Australian football players, the quadriceps of the dominant leg is injured more frequently than the quadriceps of the nondominant leg. These differences between the dominant and nondominant limb suggest that athletes place different demands on and even rely on 1 limb more than the other, which may lead to injury. Little research has examined the effect of limb dominance on the rate of ankle sprains, but it is logical to conclude that the different demands placed on the limbs may predispose 1 limb to more injuries than the other. Nonetheless, previous research has been inconsistent in its findings on the injury rate of the dominant ankle versus the nondominant ankle. Yeung et al monitored 380 athletes over the course of a competitive soccer season and found that the dominant ankle was sprained 2.4 times more often than the nondominant ankle. Furthermore, Ekstrand and Gillquist investigated ankle sprains among soccer players and found that 36 of the 39 ankle sprains were of the dominant ankle. Beynnon et al and Surve et al, however, failed to find such a difference between the 2 ankles. It should be noted, however, that of the 20 sprains reported in the Beynnon et al study, 14 were of the dominant ankle, although this finding was not significant. To our knowledge, there has not been any research conducted investigating why there is a potential difference in the number of ankle sprains between the dominant and nondominant ankle. If the cause of the possibly greater number of ankle sprains of the dominant ankle can be identified, exercise protocols can be initiated to help prevent or reduce the number of ankle sprains of the dominant ankle.

Previous research examining the latency of the peroneus longus has concluded that this muscle cannot react quickly enough to protect the ankle complex from injury when it is unexpectedly forced into inversion. However, recent research has found that the peroneus longus may be able to react quickly enough to protect the ankle against a lateral ankle sprain. Because prior research has demonstrated that the ankle of the dominant limb may be sprained more frequently than the ankle of the nondominant limb, and the peroneus longus is a key component in the prevention of lateral ankle sprains, a potential explanation for why some researchers found that the dominant ankle is sprained more frequently may lie in the behavior of the peroneus longus. Specifically, the different demands placed on the dominant and nondominant limbs could cause a difference in the latency of the peroneus longus between the 2 limbs. Therefore, the primary purpose of this
study was to determine whether there was a difference in the latency of the peroneus longus between the dominant and nondominant leg using a dynamic approach to a forced, laboratory-induced ankle inversion. Previous research has investigated the latency of the peroneus longus using a tilt platform during quiet stance. However, the validity of this model has been called into question, and ankle sprains rarely occur while an individual stands with equal weight distribution on both limbs. Because many ankle sprains occur when landing from a jump onto the foot of another player, an outer sole with fulcrum was developed, similar to one used in a previous study to force the ankle into inversion on landing from a step-down task. We hypothesized that the latency of the peroneus longus of the nondominant limb would be significantly shorter than the latency of the dominant limb, thus shedding light on contributors to the possible inequality between dominant- and nondominant-ankle-sprain rates.

Methods

Participants

Fifteen healthy participants (age 21.07 ± 1.07 y, height 1.69 ± 0.09 m, mass 63.45 ± 11.97 kg) volunteered for testing. All were free from any previous history of an ankle sprain, lower extremity fracture, or lower extremity surgery. All male participants had to wear a US men’s shoe size 10 to 11, and all female participants had to wear a US women’s shoe size 8 to 9, to qualify for inclusion in this study. Furthermore, all participants were physically active, participating in at least 30 minutes of physical activity 4 d/wk. Any individual who had previously suffered an ankle sprain, lower extremity fracture, or surgery or did not meet the shoe-size requirement was excluded from the study. Each participant signed an informed-consent document approved by our institutional review board.

Instrumentation

Eight detachable outer soles (4 with fulcrum and 4 flat) made of orthoplast were developed for this project. Left and right outer soles were developed for the average US men’s shoe size (10–11) and the average US women’s shoe size (8–9). To produce 25° of inversion on landing, a 6-mm-thick and 30-mm-high fulcrum was placed 20 mm from the medial border of the outer sole and ran the length of the outer sole (Figure 1). A similar mechanism has been used previously to force the ankle into inversion, but not to measure the latency of the ankle musculature. The outer sole was attached to the participant’s athletic shoe using Velcro straps. All participants were required to wear low-top, flat-soled athletic shoes for testing. Although it is documented that lateral ankle sprains are often the result of inversion and plantar flexion, the current study attempted to isolate only the influence of the inversion moment on the latency of the peroneus longus so that a clearer understanding of this component could be achieved.

Muscle activity was recorded with a multichannel electromyography (EMG) amplifier–processor unit (MyoClinical, Noraxon USA Inc, Scottsdale, AZ) using pairs of wet-gelled bipolar Ag–AgCl disc surface electrodes (Blue Sensor SE, Ambu Inc, Denmark) interfaced with a notebook computer. The raw EMG signal was amplified with an input impedance of 10 MΩ, with the gain set at 1000× and
a common-mode rejection ratio >115 dB. Surface EMG electrodes were placed over the most prominent part of the muscle belly of the peroneus longus with a 2-cm interelectrode distance. Electrode-placement sites were shaved, abraded, and cleaned according to standard electromyographic procedures. The electrode placement was similar to that used previously,\textsuperscript{13,16} and proper placement was verified by manual muscle testing.

A metal landing surface (Figure 2) was developed, and the signal from the landing surface was synchronized with the EMG processor. Metal was also attached

\textbf{Figure 1} — Bottom of outer sole with fulcrum.

\textbf{Figure 2} — Participant waiting to perform the step-down task onto the metal landing area.
to the fulcrum and to the lateral border of the outer sole (Figure 1). When the fulcrum made contact with the landing area, a spike was produced in 1 of the EMG channels, indicating ground contact and closely coinciding with the beginning of the inversion moment. When the lateral border of the outer sole made contact with the landing area, a second spike was produced in a different EMG channel, indicating that the participant had completed the task and the subtalar joint was in $25^\circ$ of inversion. The synchronization between the EMG signal and the landing area can be viewed in Figure 3.

**Procedures**

The participants were randomly assigned to order of testing (dominant or non-dominant leg first). To be consistent with previous research, the dominant leg was defined as the leg the participants would use to kick a soccer ball.$^5,18,25,26$ The

![Figure 3](image-url)  

**Figure 3** — Filtered and rectified EMG signal of the peroneus longus (channel 1) and filtered and rectified signal from the landing area (channels 13 and 14). Latency was calculated as the time from contact of the fulcrum with the landing area (channel 13) until muscle activity exceeded baseline muscle activity by both 5 and 10 SD.
participants stood on a 27-cm-high box on the nontesting leg and moved the foot of the testing leg behind them by flexing the knee and extending the hip; this position prevented them from seeing which outer sole was affixed to the sole of the shoe. Next, either the outer sole with fulcrum or flat outer sole was secured to the participants’ shoe with Velcro, in random order. The purpose of the flat outer sole was to prevent anticipation of the inversion perturbation. The outer soles were of a similar mass to prevent the participants from anticipating which was attached. After the outer sole was secured, participants were instructed to swing their leg through and allow the foot to hang down in front of them in a natural position (Figure 2). After confirming that there was no preactivity in the peroneus longus, the participants were instructed (verbally given the signal “go”) to step down off the box onto the testing leg. When instructed to step down, the participants leaned forward until they lost their balance and were forced to step down onto the testing foot (Figure 4). They were not allowed to use flexion of the contralateral knee to lower themselves down. Participants were instructed to land flat-footed to help keep the initiation of the inversion moment as consistent as possible, and all trials were visually inspected to ensure that this occurred. In addition, the metal only covered the back third of the fulcrum, so any trials that resulted in a toe landing were not recorded and subsequently discarded. After landing, the outer sole was removed and placed behind the participant. The same procedure was followed until 10 trials had been performed with the outer sole and fulcrum, and then the other leg was tested.

Figure 4 — Lateral border of the outer sole contacting the landing area, indicating that the participant has completed the 25° of inversion.
The latency of each of the 10 trials for the outer sole with fulcrum was averaged for the peroneus longus of the dominant and nondominant legs separately.

Recent research\(^5\) has suggested that habituation may occur when the ankle is repeatedly forced into inversion, so practice trials should be given to reduce the effects of this habituation. In the current study, no practice trials were allowed. The main reason for not allowing them was to preserve the unexpected nature of the inversion moment that causes a lateral ankle sprain. This study attempted to closely replicate this mechanism, and practice trials would have removed this element. In addition, the flat outer sole was randomly interchanged with the outer sole with fulcrum to help prevent anticipation and habituation. In addition, the data were examined and there were no signs of habituation for latency across trials.

**Data Reduction**

Both the EMG signal and the signal from the landing surface were band-pass filtered (sixth-order Butterworth, with cutoff frequencies of 8 and 535 Hz) and full-wave rectified. The dependent variable was latency, in milliseconds, of the peroneus longus. Latency was defined as the time from contact of the fulcrum with the landing area, which closely coincided with the initiation of the inversion moment, to the time of muscle activity exceeding 5 SD from baseline. Baseline activity was defined as the muscle activity that was recorded 200 milliseconds before landing. Latency values were also calculated using muscle activity exceeding 10 SD from the baseline. This latency variable is a measure of the amount of time it takes the main evertor of the foot–ankle complex to become active after the initiation of the forced inversion. Previous research has used 2 SD\(^{11,15,17}\) and 10 SD\(^{5,13,16}\) above baseline muscle activity to determine the onset of muscle activation. In the current study, 2 SD was deemed not a large enough value to distinguish between muscle activity in the peroneus longus in preparation for landing and muscle activity in the peroneus longus in response to the inversion perturbation. In an attempt to be consistent with some of the previous research examining latency of the peroneus longus,\(^{5,13,16}\) this study reported the latency of the peroneus longus using 5 SD and 10 SD above baseline muscle activity.

Because of the dynamic nature of the task, all data were visually inspected immediately after collection for any excessive signs of preactivity before landing and to confirm that activation of the peroneus longus occurred after touchdown of the fulcrum on the landing area. All data were exported into Microsoft Office Excel 2007 (Microsoft Corp, Redmund, WA) for calculation of the reflex latencies. The data were visually inspected again, and 14 trials were discarded because of spikes in muscle activity during the 200 milliseconds before landing.

**Data Analysis**

Two 1-way analyses of variance with repeated measures were conducted (1 each for 5-SD and 10-SD thresholds) to determine whether there was a statistically significant difference between the latencies of the peroneus longus of the dominant leg and the nondominant leg. An alpha level of .05 was established a priori as the criterion for statistical significance. All statistical analyses were conducted with the Statistical Package for Social Sciences version 14.0 (SPSS) for Windows.
Results

The results revealed a significant difference between the dominant and nondominant legs in terms of the latency of the peroneus longus when using 5 SD above baseline as the threshold: $F_{1,14}= 9.34, P = .009, d = .895$. There was also a significant difference between the 2 legs when using 10 SD as the threshold: $F_{1,14}= 18.56, P = .001, d = .920$. The means and standard deviations are presented in Table 1.

Discussion

The results revealed that the peroneus longus of the nondominant limb had a significantly shorter latency than that of the dominant limb. The effect size was also large$^{32}$ for both the 5-SD threshold ($d = .895$) and the 10-SD threshold ($d = .920$), indicating the large influence of limb dominance on the latency of the peroneus longus. This increase in latency of the peroneus longus of the dominant limb may help explain why some researchers have found that the dominant ankle is injured at a significantly higher rate than the nondominant ankle.$^{25,26}$

Because the current study found a significantly longer latency of the peroneus longus of the dominant limb, the next step was determining what factors may lead to this difference. It has been established that different demands are placed on the dominant and nondominant legs$^{7,21}$ and that the dominant leg may be placed in positions that would lead to a lateral ankle sprain more frequently than the nondominant leg.$^{26}$ These different demands may cause an alteration in the gamma-motoneuron system. Gamma motoneurons innervate the intrafusal fibers of the muscle spindle and can adjust the sensitivity of these intrafusal fibers,$^{6}$ thus making the muscle spindles of the peroneus longus more or less sensitive to an unexpected stretch of the muscle caused by an inversion perturbation. Because of the different demands placed on the dominant and nondominant limbs,$^{7,21}$ an alteration in the gamma-motoneuron system$^{33}$ of the dominant limb may occur, which would disturb the gamma-motoneuron loop, leading to a decrease in the recruitment of alpha motor units$^{29}$ and causing an increase in the response latency. If the gamma motoneurons of the peroneus longus of the dominant ankle reduce the sensitivity of the muscle spindles to an unexpected stretch, increased response latency would be the result.

Table 1  Latency (ms) of the Peroneus Longus for Dominant and Nondominant Legs

<table>
<thead>
<tr>
<th>Criterion for onset of muscle activity</th>
<th>Dominant leg (mean ± SD)</th>
<th>Nondominant leg (mean ± SD)</th>
<th>Effect size (Cohen’s $d$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>5 SD above baseline</td>
<td>44.60 ± 12.75*</td>
<td>34.40 ± 6.91*</td>
<td>.895</td>
</tr>
<tr>
<td>10 SD above baseline</td>
<td>65.63 ± 13.81*</td>
<td>51.91 ± 12.58*</td>
<td>.920</td>
</tr>
</tbody>
</table>

Baseline = muscle activity 200 ms before touchdown of fulcrum.

* Significant at $P < .01$. 

The exact cause of this disruption of the peroneus longus of the dominant limb is unknown, but the fact that different demands are placed on the dominant limb compared with the nondominant limb\textsuperscript{7,21} could help explain these differences. Future research should focus on determining the exact cause of this discrepancy.

Previous research using a tilt platform to force the ankle into inversion has used different criteria to determine the onset of the peroneus longus when calculating latency. The most common criterion has been 2 SD above baseline muscle activity\textsuperscript{10,15,17} or 10 SD\textsuperscript{5,13,16} above baseline muscle activity, taken 100–200 milliseconds before the release of the trapdoor. The current study determined the latency of the peroneus longus using both 5 and 10 SD above baseline muscle activity 200 milliseconds before touchdown of the fulcrum. Although a direct comparison cannot be made with tilt-platform research because of differing methodologies and threshold criteria, it is insightful to examine how the latencies found in the current study compare with previous work on peroneal latency. Table 2 presents a comparison between the results of the current study using 10 SD above baseline muscle activity and previous work that used 10 SD above baseline muscle activity to determine the onset of the peroneus longus. Of the 3 previous tilt-platform studies using 10 SD above baseline activity,\textsuperscript{5,13,16} 2 found values very similar to those of the current study,\textsuperscript{5,13} and 1 study\textsuperscript{16} found a much greater latency of the peroneus longus than the current study. The similarity in latency values between the current study and 2 recent studies\textsuperscript{5,13} using different methodologies but the same criteria to determine the onset of the peroneus longus may support the use of 10 SD above baseline muscle activity to determine the onset of the peroneus longus, but further work needs to be done to determine which level is most appropriate.

This was an initial study investigating the difference in the latency of the peroneus longus between the dominant and nondominant legs. To our knowledge, it is the first study to specifically examine this difference. One prior study did find that the latency of the peroneus longus of the right leg was greater than the latency of the peroneus longus of the left leg, but limb dominance was not defined.\textsuperscript{16} Because the peroneus longus provides the primary active defense against

Table 2  Comparison of Previously Determined Response Latencies of the Peroneus Longus (Tilt-Platform Research) Using Similar Onset Criteria as in the Current Study

<table>
<thead>
<tr>
<th>Study</th>
<th>Criterion for onset of muscle activity (response latency)</th>
<th>Mean peroneus longus latency</th>
<th>Difference in latency from current study</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jackson et al\textsuperscript{5}</td>
<td>10 SD above baseline</td>
<td>57.3 ms</td>
<td>−1.47 ms</td>
</tr>
<tr>
<td>Kernozek et al\textsuperscript{13}</td>
<td>10 SD above baseline</td>
<td>50.3 ms</td>
<td>−8.47 ms</td>
</tr>
<tr>
<td>Lynch et al\textsuperscript{17}</td>
<td>10 SD above baseline</td>
<td>76.6 ms</td>
<td>+17.83 ms</td>
</tr>
</tbody>
</table>

The comparisons are between previous research using 10 SD above baseline muscle activity to determine the onset of the peroneus longus and the values found in the current study (the dominant and nondominant latency values of the current study were averaged for the comparison) using the same onset criteria.
a lateral ankle sprain, the quicker this muscle is activated after the ankle is forced into inversion, the greater the chance it has at preventing or attenuating the rate at which a lateral ankle sprain occurs. The current study found that the latency of the peroneus longus of the dominant limb was more than 10 milliseconds greater than the latency of the peroneus longus of the nondominant limb. This finding aids in the investigation of why the ankle of the dominant limb may be sprained more frequently than the ankle of the nondominant limb. Previous research has found that muscle-strength imbalances between the 2 lower extremities are associated with an increase in lower extremity injuries. However, research is needed to examine potential strength imbalances of the muscles of the foot–ankle complex between the dominant and nondominant limbs, as well as any possible proprioceptive differences. Future research should also examine whether the dominant foot–ankle is placed in situations more likely to cause a lateral ankle sprain (landing on the foot of another player) than the nondominant ankle, which has previously been suggested as the cause for the higher number of ankle sprains of the dominant ankle but has not been investigated. A disruption in the gamma-motoneuron system of the peroneus longus between the dominant and nondominant legs should also be investigated to determine whether this is the source of the difference in latency between the dominant and nondominant legs.

There are limitations to the current study. Because we used a landing task, there was potential preactivation in the peroneus longus in preparation for landing. This was controlled for by ensuring that the peroneus longus was silent before beginning the task and by visually inspecting all data. Another limitation is the way the participants landed on the fulcrum. They were instructed to land flat-footed, and metal was only placed along the back third of the fulcrum, so any trials in which the participants landed on the toe would be discarded. Nonetheless, there may still have been some trials when the participants did not land perfectly flat-footed, and this would affect the stretch reflex of the peroneus longus. Finally, no practice trials were allowed in the current study to preserve the unexpected nature of a lateral ankle sprain. Recent work has suggested that practice trials be given when performing repeated inversion of the ankle to help prevent habituation. However, that research found habituation in terms of muscle-activation level, not latency. Therefore, the habituation found in previous work to repeated inversion perturbations does not directly apply to this study because of different methodologies and different EMG measurements. In addition, no habituation was noted in the current study in terms of latency. Future work should specifically examine whether habituation occurs using the fulcrum methodology.

Conclusions

The results of this study indicate that the latency of the peroneus longus of the dominant limb is significantly longer than the latency of the peroneus longus of the nondominant limb. Because the peroneus longus provides dynamic defense against a lateral ankle sprain, this finding suggests that the increase in latency may be a contributor to the greater number of dominant-ankle sprains noted in previous literature.
References