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ORIGINAL ARTICLE

# Decoupling of laxity and cortical activation in functionally unstable ankles during joint loading

**Alan R. Needle · C. Buz Swanik · Michael Schubert · Kirsten Reinecke · William B. Farquhar · Jill S. Higginson · Thomas W. Kaminski · Jochen Baumeister**

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**Abstract**
*Purpose* Recent studies have highlighted central nervous system alterations following ligamentous injury that may contribute to joint instability. However, research has not observed cortical responses to joint loading or sensory changes in the context of joint laxity following injury.
*Methods* Forty-two subjects were stratified into healthy (CON), unstable (UNS), and coper (COP) groups using ankle injury and instability history. Event-related desynchronization (%) from electroencephalography quantified somatosensory cortex activity as the ankle was loaded using an arthrometer.
*Results* Cortical activation increased as the ankle was loaded ($F = 63.05$, $p < 0.001$), but did not differ between groups ($F = 1.387$, $p = 0.268$), despite greater laxity in UNS ($F = 3.58$, $p = 0.038$).
*Conclusions* Increased somatosensory cortex activity was observed with joint loading; however, though UNS demonstrated a degree of mechanical instability, no differences in magnitude of cortical activation were observed. Continued research should explore how the relationship between cortical activation and joint stiffness is affected following ligamentous injury.

**Keywords** Ankle arthrometry · Event-related desynchronization · Functional ankle instability · Neuromechanical decoupling · Mechanical instability

**Abbreviations**

| | |
|---|---|
| ACL | Anterior cruciate ligament |
| fMRI | Functional magnetic resonance imaging |
| EEG | Electroencephalography |
| TMS | Transcranial magnetic stimulation |
| ERD | Event-related desynchronization |
| CAIT | Cumberland ankle instability tool |
| CON | Healthy control group |
| COP | Coper group |
| UNS | Unstable group |
| BASE | Baseline |
| LOAD-1 | Early loading (1st 1,000 ms) |
| LOAD-2 | Late loading (2nd 1,000 ms) |

## Introduction

The most common sequelae following ligamentous injury such as an ankle sprain is functional ankle instability—recurrent sensations of rolling or "giving-way" at the joint (Anandacoomarasamy and Barnsley 2005; Freeman et al.

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A. R. Needle (✉)
Department of Health and Exercise Science, Appalachian State University, ASU Box 32071, 111 Rivers Street, Boone, NC 28604, USA
e-mail: needlear@appstate.edu

C. B. Swanik · W. B. Farquhar · T. W. Kaminski
Department of Kinesiology and Applied Physiology, University of Delaware, Newark, DE, USA

M. Schubert · K. Reinecke · J. Baumeister
Institute of Sports Medicine, Department of Exercise and Health, University of Paderborn, Paderborn, Germany

J. S. Higginson
Department of Mechanical Engineering, University of Delaware, Newark, DE, USA

J. Baumeister
Department of Neuroscience, Norwegian University of Science and Technology (NTNU), Trondheim, Norway

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1965; Hertel 2002). While the etiology of this pathology remains unknown, proposed theories have suggested increased joint excursion secondary to ligamentous laxity may contribute to this problem; however, laxity does not consistently correlate with function (Freeman 1965; Markolf et al. 1984; Needle et al. 2014). Alternately, diminished sensation from ligamentous mechanoreceptors and subsequently decreased proprioception has been offered as an explanation for loss of functional stability; but this relationship also remains ambiguous (Freeman et al. 1965; Hertel 2002; Needle et al. 2014). Recent reports have suggested changes in the central nervous system and cortex exist among subsets of patients with functionally unstable ankles and knees (Kapreli et al. 2009; Baumeister et al. 2011; Heroux and Tremblay 2006; Pietrosimone et al. 2012), yet no studies have directly investigated the relationship between joint loading and cortical activation.

Ligamentous injury was originally tied to increased joint laxity (mechanical instability) that was believed to lead to sensations of functional instability (sensations of giving-way). However, not all patients with a history ankle sprain or functional instability display residual laxity (Hertel 2002). It was, therefore, hypothesized that while the ligament may return to its resting length, damaged mechanoreceptors may contribute to alterations in nervous system activity and subsequent instability (Freeman 1965; Hertel 2002; Hubbard 2008). To date these changes remain unclear; very limited research has directly quantified nervous system activity at its multiple levels (peripheral, segmental, cortical) (Munn et al. 2010; Needle et al. 2014). Recent research utilizing direct recordings of peripheral afferent activity has observed decreased muscle spindle afferent activity during joint loading in anterior translation among functionally unstable ankles (Needle et al. 2013b). However, this sensory signal represents only the initial part of the complex sensorimotor system as afferent traffic will ascend to the somatosensory cortex where integration and formation of a motor response will occur. Understanding how the somatosensory cortex detects and interprets joint loading could contribute to our understanding of how greater than half of ankle sprains develop into functionally instability, while other successfully "cope" and experience no residual problems (Wikstrom and Brown 2014).

Limited investigations have directly measured cortical function among previously injured joints, with studies into the knee model far more prevalent than those investigating the ankle. Original research found diminished somatosensory evoked potentials during arthroscopy, in ACL-deficient subjects, leading authors to conclude that injury to the ACL could alter central nervous system function (Valeriani et al. 1999). These findings were supported in studies using functional magnetic resonance imaging (fMRI) (Kapreli et al. 2009) and electroencephalography (EEG) (Baumeister et al. 2008, 2011). While evidence indicates that ligamentous injury contributes to neuroplasticity, no studies have directly measured the magnitude of the somatosensory response to joint loading or how the amplitude of cortical activation relates to laxity among groups of unstable joints. Event-related desynchronization (ERD) in the upper alpha (α-2) frequency has been associated with increased activation in the somatosensory and motor cortices (Pineda 2005; Martinez-Jauand et al. 2012). However, no data is available to determine whether ligamentous loading could cause desynchronization in this frequency and how the cortical response to loading would differ following injury.

As inconsistencies exist related to proprioceptive deficits following joint injury, there is ambiguity as to what the most effective treatment programs may be to maximize individual patient outcomes. Utilizing direct measures of central nervous system activity during joint loading, as well as including a group of copers for comparison to unstable ankles could reveal individualized changes that would dramatically increase our understanding of this pathology. The advent of ankle arthrometry has provided clinicians and researchers a method to apply a standard load to the capsuloligamentous structures of the joint (Kovaleski et al. 2002). Using this technique, unstable ankles were observed to have decreased peripheral afferent activity in response to joint loading (Needle et al. 2013b). Therefore, we hypothesized that decreased activation in the somatosensory cortex may be observed during a similar load. The aim of this study was, therefore, to utilize EEG to measure the quantity of somatosensory cortex activation during gradual joint loading and to investigate its relationship to laxity. Furthermore, we aimed to compare these variables across subsets of healthy, functionally unstable, and coper ankles.

## Materials and methods

### Experimental design

A case control design was employed in this study. Dependent variables include event-related desynchronization (ERD) in the upper alpha (α-2, 10–12 Hz) frequency band at the CP3 and CP4 electrodes. Independent variables include group, time of loading, and side.

### Participants

Forty-two participants were recruited for this study, with 31 subjects providing data usable for analysis (Table 1). Subjects were stratified into three groups using a history of ankle injury questionnaire and the 30-point Cumberland

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**Table 1** Subject characteristics for electroencephalography (EEG) analysis

| | CON | COP | UNS |
|---|---|---|---|
| Subjects recruited | 17 | 9 | 16 |
| Usable EEG recordings | 14 | 6 | 11 |
| Age (years) | 21.35 ± 2.98 | 21.86 ± 4.06 | 20.83 ± 3.51 |
| Height (cm) | 168.5 ± 9.3 | 171.6 ± 12.7 | 166.9 ± 11.1 |
| Mass (kg) | 62.49 ± 11.6 | 69.17 ± 13.6 | 63.30 ± 13.54 |
| Gender (Male/Female) | 6M/8F | 3M/3F | 3M/8F |

Ankle Instability Tool (CAIT) (Hiller et al. 2006). The CAIT quantifies sensations of rolling and giving-way that patients experience at the ankle, with higher scores indicating greater perceived ankle stability. Healthy ankles (CON) had no history of ankle sprains and a CAIT score ≥28. Copers (COP) had a history of one or more ankle sprains, and a CAIT score ≥28. Functionally unstable ankles (UNS) had a history of one or more ankle sprains and a CAIT score ≤24 (Hiller et al. 2006). All injured subjects had a history of injury on only one ankle so that the contralateral ankle could be used for comparison. Participants were free from lower extremity injury and concussion for at least 12 months, and had no history of fracture and surgery.

## Instrumentation

Joint loading and laxity were assessed using an instrumented ankle arthrometer (Blue Bay Research, Milton, FL). The arthrometer consists of a footplate with heel and dorsal foot clamps affixed to a load handle. A 3-degree kinematic linkage system connected a tibial pad and foot plate to provide values of anterior and posterior joint displacement (Kovaleski et al. 2002). Analog signals of force and ankle displacement were transmitted to a laptop and recorded using custom LabVIEW software (National Instruments, Austin, TX).

Cortical activation was measured using 22 Ag/AgCl electrodes (F7, F3, FZ, F4, F8, FC3, FCZ, FC4, C3, CZ, C4, CP3, CPZ, CP4, T5, P3, PZ, P4, T6, O1, OZ, O2) embedded in an elastic cap (QuickCap™, Compumedics Neuroscan, Chalotte, NC) in accordance with the international 10:20 system. EEG was recorded and stored using Scan 4.5 Software with a NuAmps amplifier system (11 subjects were collected using a SynAmps amplifier system; Compumedics Neuroscan, Charlotte, NC). A ground electrode was placed at the mid-forehead, while linked mastoid processes served as an average reference [(A1 + A2)/2] (Nunez and Srinivasan 2006). Digital triggers from the ankle arthrometer were sent to Scan software so that force data were synchronized with cortical activity.

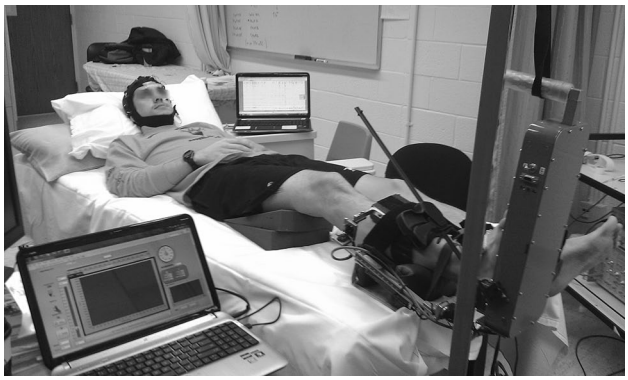

**Fig. 1** Subject positioning for EEG data collection. The ankle arthrometer was suspended to support the weight of the ankle between testing blocks. A curtain blocked the subject's view of their ankle throughout testing

## Procedures

Participants reported for a single testing session. After providing institution-approved informed consent (UDIRB #308402-3), the QuickCap™ was fitted to the participant and an impedance test (<5 kΩ) ensured a sufficient signal-to-noise ratio. Participants then lay supine on a padded treatment table, with the knee flexed ~30°, as the ankle arthrometer was affixed to the foot and suspended to support weight of the participant's ankle (Fig. 1). After familiarization with the EEG recordings and the procedure, five testing blocks were performed on each ankle as continuous cortical activity was recorded in Scan software at 1,000 Hz/32 bit and amplified (Baumeister et al. 2012).

Each testing block consisted of 10 anterior–posterior translations from a force of −30 to 130 N with 5 s of rest between each trial (Fig. 2). Integrated visual feedback was used to ensure a consistent rate of loading and unloading of the ankle joint (50 N/s) and an appropriate rest period between each test block. Between each block, 30 s of baseline eyes open and eyes closed EEG data was collected. Participants were asked to keep their eyes open throughout testing blocks while blinking comfortably, and impedance and artifacts from participant motion was monitored at all times. The same investigator (ARN) performed all translations at the ankle joint, and the side tested first was determined randomly.

## Data reduction

Total laxity was calculated as the peak displacement minus the minimum displacement. In addition, laxity was calculated for the first 1,000 ms of the load, as well as the second 1,000 ms of the load as the range of excursion over that time period. The average across all trials was used for analysis.

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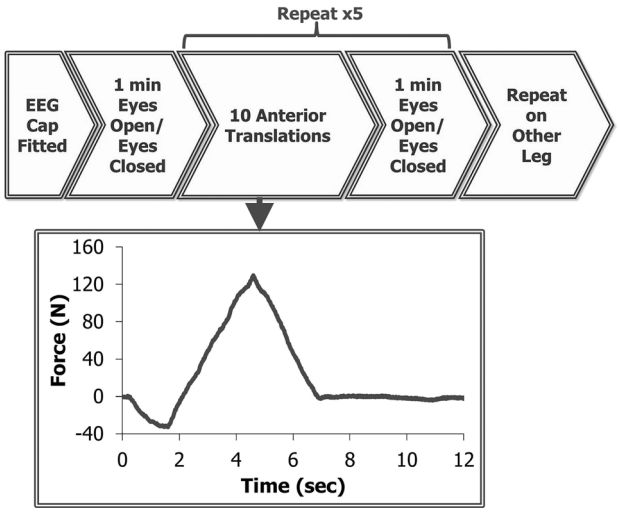


**Fig. 2** Order of testing for data collections. Anterior translations were applied from −30 to 130 N at a rate of 50 N/s. Five seconds were allowed between each translation within a block

For analysis, the physiological signals were band-pass filtered (0.5–30 Hz) and visually inspected for artifacts. Electrodes above and below the eye (VEOU, VEOL) were used to complete a linear derivation for removal of ocular artifacts from blinking. Continuous data from the EEG were synchronized using triggers from the arthrometer to determine the start and end of each translation. The data were then cut into 4-s epochs: 2,000 ms prior to start of the translation to 2,000 ms following the start of the translation. Based on pilot analysis, ERD in the upper alpha frequency band was calculated from artifact-free segments for the CP3 and CP4 electrodes, corresponding to the left and right somatosensory cortices, respectively. Higher desynchronization within this frequency band is indicative of greater cortical activation relative to a baseline period, and inhibition of the corticothalamic pacemaker cells (Pineda 2005; Pfurtscheller and Lopes da Silva 1999). Event-related desynchronization was calculated at baseline (BASE, −2,000 to −1,000 ms prior to loading), early loading (LOAD-1, 0–1,000 ms from the start of loading), and late loading (LOAD-2, 1,000–2,000 ms from the start of loading).

### Data analysis

Total laxity was compared with a 2-way analysis of variance (ANOVA) with one within-subject factor (Side, 2 levels) and one between-subjects factor (Group, 3 levels). Laxity was also assessed across times using a 3-way ANOVA with two within-subject factors (Side, 2 levels; Time, 2 levels) and one between-subjects factor (Group, 3 levels). Cortical activation was assessed with a 3-way ANOVA with two within-subject factors (Side, 2 levels; Time, 3 levels) and one between-subjects factor (Group, 3 levels). Post hoc analysis was performed using Tukey's post hoc and pairwise comparisons. An alpha level was set a priori at 0.05.

Relationships between laxity and cortical activity were assessed with Pearson product–moment correlation coefficients for the overall main effect as well as correlations within each group. Correlation coefficients for groups were converted to z-scores and the differences between z-scores were used to determine differences between groups (Fisher 1921).

## Results

### Joint laxity

A significant main effect of group was observed for total ankle laxity ($F_{(2,36)} = 4.87$, $p = 0.013$), although there was no side by group interaction effect ($F_{(2,36)} = 0.052$, $p = 0.956$) (Fig. 3). Tukey's post hoc tests found UNS had significantly more laxity than the CON group ($p = 0.010$, $d = 0.95$), while COP was not significantly different from either group ($p > 0.05$). No group by time interaction effect ($F_{(2,36)} = 0.204$, $p = 0.816$) was observed for laxity, but significant main effects were observed for time ($F_{(1,36)} = 11.81$, $p = 0.002$), as well as group ($F_{(2,36)} = 3.58$, $p = 0.038$). Pairwise comparisons found significantly greater joint excursion in LOAD-2 than LOAD-1 ($p = 0.002$, $d = 0.35$). Tukey's post hoc indicated UNS ankles had significantly greater laxity than CON ($p = 0.029$, $d = 0.73$), while the COP group was not significantly different from either group ($p > 0.05$).

### Cortical activation

No interaction effect was observed for group, time, and side ($F_{(4,50)} = 0.919$, $p = 0.333$); as well as no main effects for side ($F_{(1,25)} = 0.722$, $p = 0.403$) or group ($F_{(2,25)} = 1.387$, $p = 0.268$). A significant main effect of time was observed ($F_{(2,50)} = 63.05$, $p < 0.001$). Pairwise comparisons revealed BASE had significantly less cortical activation than either LOAD condition ($p < 0.001$, $d = 1.4$), and LOAD-2 had significantly higher activation than LOAD-1 ($p = 0.002$, $d = 0.44$) indicating an increase in activation as load was applied (Fig. 4).

### Laxity–cortex correlations

As no effect of side was detected for any variable, correlations were only calculated for the involved side's data. Pearson correlation coefficients are displayed in Table 2. Main effects were observed where higher total laxity and early laxity (0–1,000 ms) correlated with higher LOAD-1

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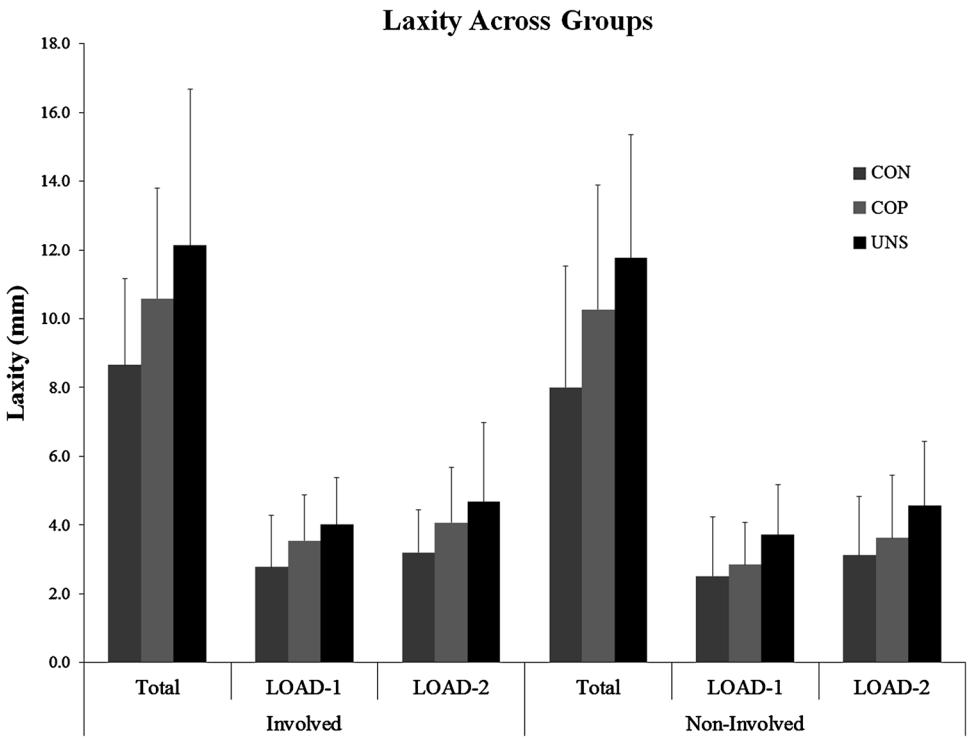


**Fig. 3** Laxity (mm) across groups for total displacement and laxity during 1st and 2nd second of loading. *Error bars* 1SD. A significant difference was observed between CON and UNS groups

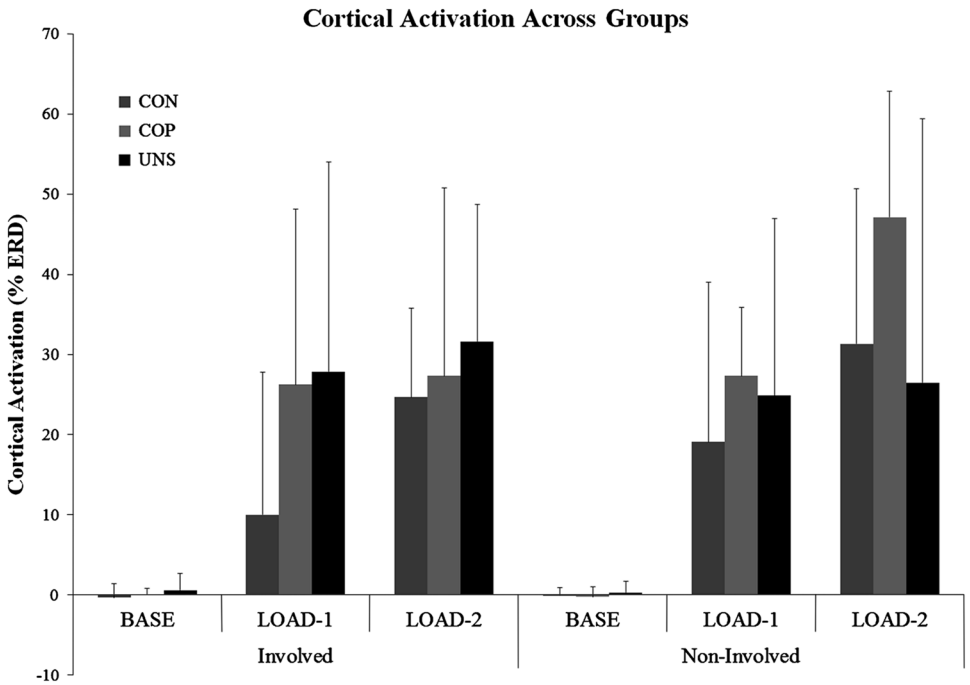


**Fig. 4** Cortical activation (% ERD) across groups and load conditions. No effects of group or side were observed; however, both load conditions were higher than baseline; and the 2nd second of loading (LOAD-2) had greater cortical activation than the 1st second of loading (LOAD-1). *Error bars* 1SD

EEG activity (total: $r = 0.383$; early: $r = 0.548$; Fig. 5). Higher early laxity also positively correlated with LOAD-2 EEG activity ($r = 0.468$). Increased late laxity (1,000–2,000 ms) correlated with both LOAD-1 ($r = 0.652$) and LOAD-2 ($r = 0.729$) EEG for the CON group only, although these were not significantly different from either group.

## Discussion

The primary findings of this study were that cortical activation increased as the joint was gradually loaded; but, despite differences in mechanical laxity, no changes in the quantity of somatosensory activation existed between healthy, coper, and unstable ankles. Furthermore, correlations were

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**Table 2** Correlation coefficients between ERD in the contralateral somatosensory cortex and joint laxity within each group and overall

| | LOAD-1 | | | | LOAD-2 | | | |
|---|---|---|---|---|---|---|---|---|
| | CON | COP | UNS | ALL | CON | COP | UNS | ALL |
| Laxity | | | | | | | | |
| Total | 0.515 | −0.574 | 0.480 | 0.383[a] | 0.499 | −0.519 | 0.537 | 0.234 |
| 0–1,000 ms | 0.394 | 0.464 | 0.579 | 0.548[a] | 0.525 | 0.539 | 0.371 | 0.468[a] |
| 1,000–2,000 ms | 0.652[a] | −0.371 | 0.363 | 0.316 | 0.729[a] | −0.274 | 0.499 | 0.273 |

[a] Significant correlation ($p < 0.05$)

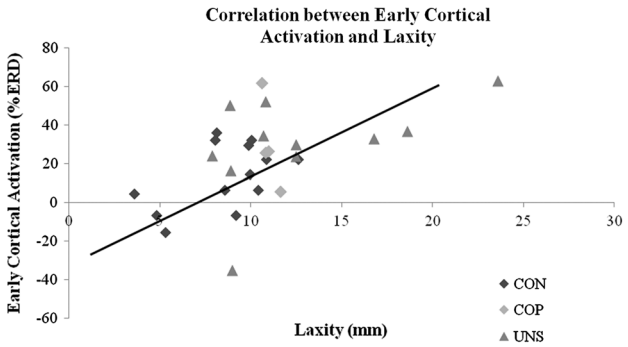


**Fig. 5** Relationship between cortical activation (% ERD, *vertical axis*) and laxity (mm, *horizontal axis*) during first second of loading (LOAD-1)

observed between the degree of joint laxity and amount of cortical activation, with these correlations appearing stronger among healthy controls. This potentially implies a degree of dissociation or decoupling, where differences in ankle laxity were not reflected as altered somatosensory activation.

## Joint laxity

Our results indicate a degree of mechanical laxity is present in unstable ankles, while copers were not significantly different from either group. While ligamentous laxity may have existed as an initial theory to explain recurrent symptoms following injury, research has been unable to consistently establish the link between mechanical deficits and functional instability (Freeman et al. 1965; Hertel 2002). Multiple researchers have suggested that while excessive laxity may contribute towards complaints of instability, it does not explain these symptoms across all patients (Hiller et al. 2011; Wikstrom and Brown 2014). All patients in this study reported history of a unilateral sprain and functional instability; however, no differences in laxity between involved and uninvolved sides were observed. This increased laxity may therefore either exist innately and was present prior to injury, or, alternately, represent a change in the neural regulation of passive joint stiffness, perhaps through altered regulation of muscle tone, even bilaterally (Needle et al. 2013a, 2014). Future prospective studies may provide additional insight into these changes.

## Cortical activation

Although laxity differences could be present in patients with functional ankle instability, of greater importance may be how the somatosensory cortex perceives that laxity. In order to prepare-for and react-to injurious loads the cortex must be able to consistently detect and predict the joint's position, movement, and load occurring at the joint, and initiate appropriate preparatory and reactive muscular responses (Freeman and Wyke 1967; Clark et al. 1985; Jahnke and Struppler 1989; Johansson 1991; Needle et al. 2014). This study was the first to quantify changes in the somatosensory cortex in response to the magnitude of joint loading. While activation increased with load across all groups, no differences in cortical activation were detected across groups or sides.

As a joint is loaded, the surrounding mechanoreceptors from the ligament as well as cutaneous and musculotendinous units would elicit action potentials along afferent axons towards the spinal cord (Johansson et al. 1991; Clark et al. 1985; Jahnke and Struppler 1989). The sensory signal would then continue ascending in the central nervous system along several sensory tracts, passing through thalamus and terminating in the primary somatosensory cortex (Hall 2011). At rest, the thalamocortical pacemaker cells emanate a rhythm of approximately 10 Hz (mu rhythm); however, as sensory information ascends, the activity of these pacemaker cells is suppressed leading to desynchronization in the upper alpha frequency (Pineda 2005; Steriade 2005). Increased event-related desynchronization in this frequency therefore indicates greater somatosensory cortex activation in reference to a period of quiet baseline. While this desynchronization has been observed during motor tasks or motor imagery, as well as in response to sights and sounds, research has not previously determined if joint loading would cause desynchronization (Steriade 2005; Pineda 2005; Basar 2012). Our findings not only support an increase in desynchronization over the somatosensory

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cortex as forces are applied to the ankle, but also a greater ERD amplitude as force increased. As this desynchronization appears to be useful for quantifying the amplitude of activity reaching the somatosensory cortex during ankle loading, it may provide further insight into whether cortical sensation of joint load might be affected by various conditions or treatment techniques.

Among subjects in the current study, differences did not exist in the amplitude of cortical activation over the somatosensory cortex between uninjured joints, those with functional instability, and copers, despite group differences in joint laxity. Previous research has suggested that peripheral sensation of load may be affected among unstable ankles, but our data suggests that those deficits do not exist in the somatosensory cortex (Needle et al. 2013b). This could imply that synapses at the spinal cord, proprioceptive input from other mechanoreceptors (such as cutaneous receptors on the plantar and dorsal foot), or perhaps increased anticipation in the sensory cortex might be able to compensate and overcome peripheral sensory deficits (Martinez-Jauand et al. 2012). Were additional proprioceptive input or anticipation able to mask peripheral deficits, this may explain why investigators have had difficulty establishing differences in proprioceptive measures in these subsets. To overcome this, testing in a randomized and/or more functional setting may be optimal to detect group differences. However, an important feature of the patient subset in this study is the presence of mechanical laxity in the unstable group. As no differences were detected in desynchronization, but a difference in mechanical laxity was observed among unstable ankles, it could imply that a smaller degree of cortical activation is elicited for each millimeter of joint displacement. This could support the presence of neuromechanical decoupling—a dissociation occurring between the nervous system's perception of joint laxity—occurring following injury (Needle et al. 2014).

Interpretation of this data is complicated by the fact that no effect of side was observed, despite all injured joints reporting a history of unilateral injury. This remains consistent with the lack of side-to-side differences in laxity seen in our subset, and may represent bilateral adaptations following injury. This is consistent with multiple studies that have identified alterations to both limbs after unilateral injury, suggesting that changes in cortical centers may be responsible for this change (Evans et al. 2004; Pietrosimone et al. 2012). However, there were no differences in somatosensory cortex activity suggesting that another factor may be contributing to only unilateral complaints of instability.

Limited investigations have studied cortical changes among subsets of unstable joints. The use of EEG has not been utilized in ankle instability research; however, some studies have looked at cortical changes following ACL injury. Similar to Valeriani et al. (1999), our findings support the sensation of ligamentous stimulation in the cortex, although the methodology in this study allowed us to observe the response through a stimulus (ligamentous loading), rather than in response to a brief electrical pulse (Pfurtscheller and Lopes da Silva 1999). Our findings differ from previous EEG studies in ACL-injured subjects where differences were observed between experimental groups (Baumeister et al. 2008, 2011). However, these studies utilized proprioceptive tasks that required a motor response and frontal cortex processing of joint position or force replication. As a result, increased somatosensory cortex activity was observed, but also changes in the frontal–parietal network, indicating altered working memory (Baumeister et al. 2011). The present study utilized an isolated sensory stimulus and quantified the somatosensory activation; however, it remains plausible that cortical changes in functional ankle instability may affect sensorimotor integration and working memory that would occur in response to loading. Future research should study the effect of ligamentous loading on the frontal-parietal network among these populations in order to better identify deficiencies following injury.

One other important characteristic of this study that may impact our observations is the manner in which load was applied. Fifty anterior translations were applied in a controlled manner to ensure at least 25 artifact-free trials would be available for analysis. However, it is also possible that repeated loading may have served to enhance sensory function at the joint, eliminating deficits among unstable ankles. Prior research has supported the use of joint mobilization for enhancing sensorimotor function at the joint (Hoch and McKeon 2011). Although the mechanism for this improvement is unclear, it could be secondary to greater mechanoreceptor stimulation facilitating fusimotor drive and subsequently muscle spindle sensitivity (Johansson et al. 1991). Alternately, it is possible that repeated joint loading could increase cognitive awareness at the joint. While we attempted to control for joint mobilization effects by standardizing the rate of loading and including periods of rest between trials, the repetitive loading may have improved sensorimotor function affecting our results. Continued research might investigate whether the continuous trials were able to improve cortical function among these subsets of injured ankles.

### Relationship between Laxity & Cortical Activation

Higher total laxity was associated with a greater magnitude of somatosensory activation across all subjects. This may lend support to the hypothesis that ankles with greater laxity have greater somatosensation as a possible protective mechanism. For instance, if a patient had higher joint

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laxity, there may be greater anticipation in the somatosensory cortex in the early portion of loading to prepare an earlier reaction (Pineda 2005; Martinez-Jauand et al. 2012). Because of the rapid rate that injuries occur, this anticipatory activity may be crucial for appropriately preparing and reacting to a perturbation (Konradsen 2002; Henderson and Dittrich 1998). Similarly, early displacement correlated with cortical activation in both the 1st and 2nd second of loading. While prior research has emphasized the early portion of joint stiffness for prevention of injury, it has been suggested that less displacement (higher stiffness) is most important for improving sensation at the joint (Johansson 1991). However, these data suggest that subjects with greater early joint displacement with respect to loading have a greater quantity of somatosensory cortex activation throughout the load. This may again serve a protective role, where subjects that lack the early resistance to loading for protecting the joint may rely on higher cortical activation to try and prevent injury.

While no significant differences were observed between group correlations, only healthy ankles showed a correlation between later laxity and both early and late cortical activation. Higher activation was observed before the displacement occurs, suggesting healthy controls may be better at anticipating their joint displacement throughout the load, and modulating their sensory activation accordingly (Martinez-Jauand et al. 2012). Similarly, it also provides evidence that only in healthy ankles is late cortical activation modulated to the individuals' laxity. This dissociation in unstable ankles may indicate an impaired ability to predict joint position and provide appropriate stabilization, ultimately leading to errors in coordination (Needle et al. 2014).

## Limitations

There are several factors that may limit interpretation of the present study. With regards to study design, our investigation used a combination of functionally unstable ankles without including mechanical instability as an independent variable. This would result in groups of functionally unstable ankles, mechanically unstable ankles, and those ankles with a combination (Delahunt et al. 2010). As functional instability (sensations of giving-way) would be considered the primary complaint of a patient following an ankle sprain, we opted to only quantify functional instability in group stratification. Future studies may further stratify these patients into sub-groups.

In addition, central nervous system activation was measured in a precise, controlled manner with simultaneous measurement of joint laxity. We attempted to use time intervals in our calculation that would eliminate subject anticipation, but it is possible that the position of the patient and the controlled manner may have made it difficult to observe significant differences between groups. In addition, use of the instrumented ankle arthrometer, while validated for loading of the ankle ligaments, would also provide cutaneous stimulation to the plantar and dorsal foot and heel. Forces were standardized across all subjects; however, these factors may not be typical of an individual experiencing an ankle sprain during activity. Future investigations should explore cortical activation throughout functional activities such as jumping and cutting and throughout injury simulations.

One final limitation that should be considered is related to the number and type of loading applied. Event-related desynchronization measures typically use a large number of trials ranging from 40 to 100 (Pfurtscheller et al. 2006; Del Percio et al. 2010; Yamanaka and Yamamoto 2010). We had selected 50 trials for this investigation in order to minimize patient discomfort, limit adaptation effects, and ensure consistent loading by the investigator. However, trials were lost on each individual due to the presence of signal artifacts secondary to movement and several subjects were therefore lost in analysis, despite attempting to limit this loss by investigating only the first 2 s of the 3 s load. It is possible by increasing the number of trials, a clearer change may have been observed with load and across groups. Future studies utilizing this methodology should be encouraged to collect additional trials. Also, investigations into joint laxity typically utilize both anterior displacement and inversion–eversion rotation measures (Kovaleski et al. 2002). Only anterior displacement was used in this study due to the large amount of time required to obtain an adequate number of trials on both ankles, and to be consistent with previous research exploring these sensory deficits in response to joint loading. (Needle et al. 2013b). Future studies may consider including inversion rotation to assess the effect of various loading directions on somatosensory cortex activity.

## Conclusions

Our data suggest that somatosensory cortex activation increases as the joint is loaded with an arthrometer. However, among a subset of mechanically lax ankles with functional instability, no differences in cortical activation exist when comparing to both healthy ankles and copers. While it is possible that unstable ankles do not undergo additional cortical activation for the greater joint excursion they experience, healthy controls appear to better couple the magnitude of sensory activation with their ligamentous laxity throughout the load. The results of this study imply that modulating sensory activation to ligamentous laxity may be

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important in preventing ankle injury, and potentially rehabilitating patients following injury. Future studies should address how cortical activation is affected in subsets of unstable ankles without mechanical laxity, as well as how cortical activation and processing change relative to joint loading and which specific rehabilitation protocols (i.e. balance, perturbation, or plyometric training) may improve these factors.

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