

THE EFFECT OF PERIPHERAL SOMATOSENSORY STIMULATION ON ANKLE
FUNCTION IN INDIVIDUALS WITH CHRONIC ANKLE INSTABILITY

A Thesis
By
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Abstract

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Peripheral somatosensory stimulation (PSS) is a clinical intervention capable of improving neural and muscle function by increasing neural excitability in injured patients. PSS has been effective in improving function in neurologically-impaired populations, however, its effect in patients with chronic ankle instability (CAI) is unknown. The purpose of this study was to conduct a preliminary investigation of a single-session intervention of PSS to the ankle on neural excitability and dynamic balance in patients with CAI. Ten participants with CAI were recruited (6M/4F, 22.1±3.6yrs, 174.7±10.1cm, 83.2±14.8kg). Participants reported for 3 total sessions: a control session where they were measured for neural excitability and dynamic postural stability indices (DPSI) before & after 60 min of quiet sitting; an intervention session where they were measured before and after 60-minutes of PSS (100Hz, 1000µsec, 1 ms, suprasensory threshold) to the common peroneal nerve, and a 24-hr follow-up. Neural excitability was measured through reflexive excitability from the Hoffman-reflex (H-reflex) as well as through measures of transcranial magnetic stimulation

(TMS). DPSI were extracted from an in-ground force plate while electromyography (EMG) data was extracted from the tibialis anterior (TA), peroneus longus (PL) and soleus (SOL) muscles during a hop-to-stabilization task. No significant differences were observed for neural excitability nor dynamic balance measures following treatment. However, medium-large effect sizes (η^2) for some neural excitability variables prompted us to further investigate the data. Encouraging effect sizes were seen across these measures from Pre-Intervention to Post-Intervention time points, however DPSI measures revealed small effect sizes indicating minimal changes. Preliminary results utilizing sensory stimulation from PSS revealed no statistical significance but did present encouraging effect sizes that suggest the intervention could possibly be effective in increasing neural excitability. The preliminary nature of this study implies that sufficient power may not have been achieved; therefore, further research should test a larger sample size to better monitor changes pre- and post-intervention and continue to investigate the ideal duration and dosage of stimulation.

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I would like to thank my thesis committee, Dr. Alan Needle, Dr. Jennifer Howard, and Dr. Jared Skinner for the support and guidance they have provided me during this project. You all have been so helpful over these past two years, and I have enjoyed getting to know and work with all of you. I would like to give an additional thank you to Dr. Needle for pushing me to step out of my comfort zone and offering his advice and support every step of the way. A final thank you is needed for my peers in the Biomechanics, Neuromuscular and Electrophysiology Labs for the mental and emotional support throughout this sometimes very stressful process. Without all of the help, I truly could not have accomplished any of this.

Dedication

I would like to dedicate this project to my Mom and Dad. Thank you both for always supporting me and providing me with all the opportunities to grow and become the person I am today. Thank you for pushing me to always try new, sometimes scary, things and teaching me to approach every challenge head on. Thank you for always being there throughout this project, whether it be distracting me from my stressors with funny texts, or the late-night dinner phone calls to decompress after a long day in the lab. I couldn't have done this without you guys.

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Introduction

Ankle sprains are the most common injury affecting physically active individuals with 60% of individuals reporting having an ankle sprain across their lifespan (Hiller et al., 2012; Holland et al., 2019). Following an initial ankle sprain, approximately 40-70% of individuals experience re-injury or recurring symptoms (Wikstrom et al., 2019). Chronic ankle instability (CAI) is the most common ailment resulting from recurring ankle sprains, which is defined as the pattern of repeated episodes of rolling or giving way at the ankle joint following the initial sprain (Hertel & Corbett, 2019). CAI also leads to decreased health-related quality of life and limited functionality while performing activities of daily living (ADLs) resulting in decreased physical activity (Houston et al., 2015; Hubbard-Turner & Turner, 2015). Ankle sprains are portrayed as minor injuries due to their commonality in physically active populations, but the likelihood of re-injury and the severity of the potential long-term effects of ligamentous injuries are much more serious. The need for rehabilitation strategies to be evaluated and progressed is vital, in order for individuals with CAI to regain ankle functionality to decrease the likelihood of re-injury and improve their quality of life.

Understanding the etiology of ankle sprains is essential for comprehending why current rehabilitation strategies have failed to diminish the prevalence and impact of these injuries. The ankle sprain is the baseline injury for all individuals with CAI, so if the sprain can be more effectively treated before becoming a recurring injury, development of CAI can be reduced. CAI can induce mechanical or neural constraints on the ankle. Potential mechanical changes include increased ankle laxity, inducing a feeling of looseness or instability, decreased stiffness, and the inability to stress-shield the joint (Needle et al., 2014). Potential neurological changes originate from sensory aberrations including pain and acute swelling, or

deafferentation which decreases peripheral sensation at the ankle joint (Needle et al., 2017). These neuromechanical changes can therefore lead to abnormal joint contact forces and changes in motor planning, leaving the joint in a more vulnerable position prior to its contact with the ground, and contributing to degenerative joint tissue damage and early-onset of osteoarthritis (Wikstrom et al., 2019).

The current rehabilitation methods for ankle sprains focus primarily on impairment-based rehabilitation, focusing on patient-specific deficits. Epidemiologic data suggests that these current rehabilitation methods have not resulted in a decreased recurrence of ankle injuries (Herzog et al., 2019). This may be due to the majority of rehabilitation treatments focusing on the impairment without addressing the possible underlying cause of the injury: the central nervous system (CNS) changes (Needle et al., 2017; O'Driscoll & Delahunt, 2011). Recent evidence suggests that maladaptive central nervous system plasticity secondary to sensorimotor changes at the ankle joint leads to altered motor planning, which places individuals at increased risk for subsequent injury (Hass et al., 2010). Maladaptive neuroplasticity decreases cortical excitability and function, however research indicates that improving cortical excitability via neuromodulatory therapy, for example, can increase muscle activation and functional outcomes (Bruce et al., 2020). Therefore, neuromodulatory-based interventions should aim to correct maladaptive neuroplasticity caused by the initial injury in order to rehabilitate the initial ankle injury and restore functionality.

Peripheral somatosensory stimulation (PSS) is a clinically accessible, non-invasive treatment that is administered via bursts of electrical stimulation through electrodes placed on the skin over peripheral nerve(s) (Conforto et al., 2010). Unlike transcutaneous electrical stimulation (TENS), which applies a sensory stimulus over skin to gate pain stimuli yielding a localized effect, PSS stimulates the nerve and yields a radiating effect along the

sensorimotor distribution of that nerve and is hypothesized to increase cortical excitability and induce plasticity; thereby, improving motor function from the previously maladaptive plastic state (Chipchase et al., 2011). In both healthy individuals and patients with stroke, generating greater somatosensory feedback through PSS has increased strength and range-of-motion of the limb receiving the stimulation resulting in improved patient-reported outcome measures (Celnik et al., 2007; Stockley et al., 2020). Given decreases in cortical excitability observed in individuals with CAI, combined with the peripheral sensory aberrations (e.g., deafferentation) described in this population, PSS has the potential to improve not just motor function, but patient outcomes in this population (Kim et al., 2019; Needle, Swanik, et al., 2013).

Previous research utilizing PSS for rehabilitation has predominantly examined the efficacy of treatments in populations of individuals having suffered from a subacute stroke, with minimal testing done in healthy populations as comparative measures (Celnik et al., 2007; Conforto et al., 2002, 2010; Ghaziani et al., 2018; Khaslavskaja et al., 2002; Khaslavskaja & Sinkjaer, 2005; Klaiput & Kitisomprayoonkul, 2009). There are mixed conclusions from research performed in these stroke populations due to the varying stimulation intensity and delivery parameters. All studies noted increased functional outcomes in their population, but some studies concluded that suprasensory stimulation was superior to subsensory in single-session treatments. In contrast, others found that subsensory stimulation led to greater retention of motor function outcome measures (Stockley et al., 2020). There is currently no research observing the use of PSS in populations with musculoskeletal injury, however, results from a study examining the effects of somatosensory stimulation of the common peroneal nerve on cortical excitability in healthy

populations demonstrated the efficacy of suprasensory stimulation in the lower extremity in increasing cortical excitability (Khaslavskaia et al., 2002; Khaslavskaia & Sinkjaer, 2005).

Maladaptive plasticity in individuals with CAI generates changes in motor patterns that predispose these individuals to re-injury. PSS may be a new, effective treatment for this population due to its efficacy in increasing neuronal excitability. Increases in neuronal excitability could correct the maladaptive neuroplasticity experienced in this population and in turn, decrease the likelihood of re-injury. In order to assess the effects of this treatment on the musculoskeletal injury, we wished to pursue the following the specific aim:

Specific Aim: To evaluate the feasibility & efficacy of a PSS intervention on modifying neural excitability and dynamic balance following a single-session treatment in adults with CAI.

Hypothesis: We hypothesize that PSS will increase neural excitability and improve dynamic balance beyond the individual's baseline.

If successful, the implementation of PSS in clinical rehabilitation of ankle sprains could improve ankle function, decrease the chance of re-injury and improve the patient's quality of life.

Measures and Methods

Study Design

This study implemented a crossover design. The independent variables are treatment (stimulation vs. control) and time (before and after treatment). Dependent variables are $H_{\max}:M_{\max}$ ratio (H:M ratio) from Hoffmann reflex test, motor threshold (MT), mid-point intensity (I_{50}), MEP_{\max} , MEP size, dynamic postural stability indices (DPSI) and EMG amplitude from the hop to stabilization test.

Participants

Twelve participants aged 18-35 with CAI were recruited, with ten completing the study. CAI was determined by a participant having a history of ankle sprains with the first being longer than one year ago while also testing with a score of greater than 10 on the Identification of Functional Ankle Instability (IdFAI) questionnaire. Subjects were recruited from Appalachian State University community through emails, classroom announcements and word-of-mouth recruitment. Participants were required to have a history of ankle sprains, with the first sprain being more than one year ago, and continue to feel symptoms of instability (i.e., rolling or giving way). Subjects were excluded from the study if they had experienced a sprain or lower extremity injury within the last three months prior to study enrollment, if they were currently enrolled in an ankle rehabilitation program, or if they had a history of a lower extremity fracture and/or required surgical reparation of their lower extremity following an injury. Additionally, participants were excluded if they had a pacemaker, a history of seizures, a family history of epilepsy or seizures, suffered from recurrent fainting episodes, were taking medications associated with risk of seizure or have

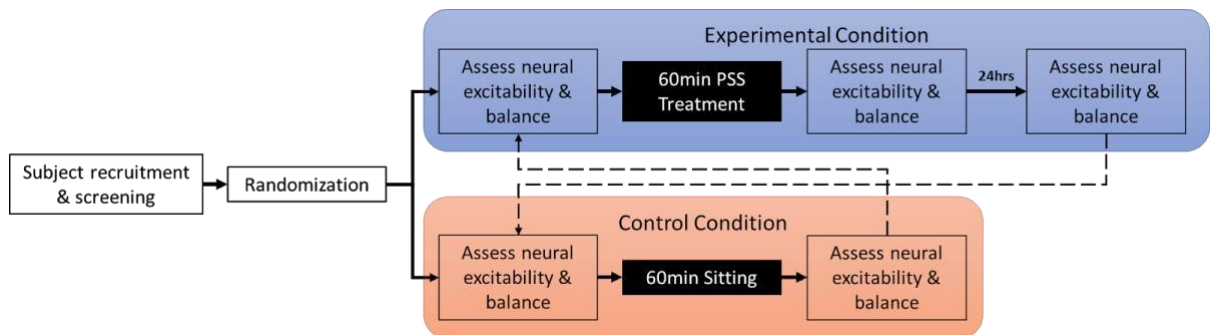
had a concussion in the last six months (Rossi et al., 2021). Females were also excluded if they were currently pregnant or planning to become pregnant over the course of the study.

Testing Sessions

Testing session consisted of assessment of neural excitability and balance, which were collected immediately before and after the intervention and 24 hours following the testing sessions. The intervention session consisted of 60 minutes of PSS treatment, while the control session consisted of 60 minutes of sitting. Participants were required to wait one-week between experimental and control conditions in order to allow for adequate washout. Participants were scheduled to complete all three sessions within this one-week span. Conditional testing sessions lasted 2.5-3 hours each, while the 24-hour follow up lasted 30-45 minutes, for a total time commitment of 5.5-7 hours over the three-day span.

Figure 1

Testing Session Schedule



Measures

Assessment of Reflexive and Neural Excitability

Prior to assessing neural excitability, electromyography (EMG) sensors were placed on the muscles of the lower leg (tibialis anterior, TA; peroneus longus, PL; and soleus, SOL). Locations for the sensors were determined by instructing the participant to dorsiflex, evert and plantarflex their ankle, for the TA, PL and SOL, respectively. Once the muscle was identified through palpation, the area was shaved (if necessary), cleaned with isopropyl alcohol, and abraded. Surface EMG Electrodes (Motion Lab Systems, Inc., Baton Rouge, LA) connected to an EMG amplifier (B&L Electronics, Santa Ana, CA) were then placed over the locations, secured with tape and covered by a wrap to minimize displacement of the sensors.

While prone on the table, participants' reflexive excitability was measured using the Hoffmann reflex (H-reflex) which was identified by locating the sciatic nerve prior to its bifurcation in the popliteal fossa (Hoffman et al., 2003). The exact location was identified using short (1ms) electrical pulses using an electrical stimulator with a bar electrode (DS7R, Digitimer LTD, Hertfordshire, UK) and observing for a maximal muscle response in the directions of the plantarflexion and eversion. Additionally, the location of the common peroneal nerve was marked for PSS treatment. Once the sciatic nerve was identified, the electrode was placed along the nerve and brief electrical pulses (square wave, 1 ms duration) were applied every 10 seconds, increasing by 1-2mA each pulse until a maximal motor response was observed from TA, PL and SOL. The peak-to-peak amplitudes of both H-waves (50-100ms after the pulse) and M-waves (10-50ms after the pulse) were extracted, and the ratio of the maximal H-wave (H_{max}) to the maximal M-wave (M_{max}) was extracted for analysis.

Following H-reflex assessment, participants were seated in a chair with an elastic cap on their head and were familiarized with TMS procedures. A double-conical magnetic coil connected to a 2T magnetic stimulator (200-2, Magstim LTD, Wales, UK) was placed at the vertex of the skull (indicated by the participant). Pulses were delivered through the coil starting at 10% stimulation output and progressing gradually until the motor threshold (MT) was surpassed and there was a small motor response in the lower extremity. That intensity was recorded, and pulses of that intensity were applied every 5s while moving the coil to locate the lower extremity hotspot, identified as the location on the skull that led to the largest motor response in the lower extremity muscles of the tested limb. The coil was then positioned at the hotspot as a range of 40-50 intensities, randomly distributed, ranging below the motor response threshold to above a maximal response, and applied every 5-7s. The peak-to-peak amplitudes of the motor evoked potentials (MEP) were obtained and plotted against the stimulus intensity, forming a stimulus-response curve (Devanne et al., 1997). The participant's RMT was determined from this curve which was utilized in the next phase of TMS. In this step, ten pulses were administered at 90, 110 and 130% of the RMT as participants contracted their PL muscle in order to amplify the readings (Needle, Palmer, et al., 2013).

Peak-to-peak amplitudes of H and M waves were measured and the maximum values of each were used to form the $H_{max}:M_{max}$ ratio for data analysis (Hoffman et al., 2003). The stimulus-response curve was fitted to a modified Boltzmann equation (Equation 1) using a Levenberg-Marquardt algorithm to determine the maximum response (MEP_{max}), the slope parameter (m), and the intensity at 50% of the curve (I_{50}) (Devanne et al., 1997). The peak-to-peak amplitudes of the facilitated MEP's were averaged at each intensity and extracted for analysis (Bruce et al., 2020; Stirling et al., 2018).

Equation 1:

$$y = MEP_{min} + \frac{(MEP_{max} - MEP_{min})}{1 + e^{m(I_{50} - x)}}$$

Assessment of Dynamic Balance

Dynamic balance was assessed using a hop-to-stabilization test (Rosen et al., 2013; Wikstrom et al., 2005). Participants performed 3 maximal, countermovement jumps on a Vertec (Sports Imports, Columbus, OH) to determine their average maximal jump height. Participants were then re-instrumented with EMG sensors (Bagnoli-4, Delsys Inc., Boston, MA) on previously located TA, PL and SOL muscles. Participants stood 70cm from an inground force plate (AMTI, Watertown, MA) and jumped forward to a height of 50% of their average maximal vertical jump height. Participants were instructed to land on the force plate on a single leg and stabilize as quickly as possible and maintain this balanced position for 15s. Participants were required to complete this test five times successfully without the “touch down” of the untested lower extremity, the touching of the Vertec or the ground with a hand or without stabilizing balance by anchoring untested lower extremity against the tested lower extremity. Force plate and EMG data was simultaneously recorded at 1000 Hz in custom LabVIEW software (National Instruments, Austin, TX).

EMG data from the hop-to-stabilization was used to create a complete linear envelope by bandpass filtering, rectifying, and lowpass filtering. Data was then normalized to ensemble peak activity of each muscle, and an extraction 250ms prior to and following initial ground contact with the force plate was used to obtain average EMG values used to determine Pre- and Post- phases (Rosen et al., 2013). Dynamic postural indices were

calculated via ground reaction forces measured from the force plate during the hop-to-stabilization jump task (Equations 2-5) (Wikstrom et al., 2005). Indices were divided into individual components of medial-lateral stability indices (MLSI), anterior-posterior stability indices (APSI), and vertical stability indices (VSI).

Equation 2:

$$DPSI = \sqrt{\frac{\Sigma(0 - x)^2 + \Sigma(0 - y)^2 + \Sigma(\text{body weight} - z)^2}{\text{number of data points}}}$$

Equation 3:

$$APSI = \sqrt{\frac{\Sigma(0 - y)^2}{\text{number of data points}}}$$

Equation 4:

$$MLSI = \sqrt{\frac{\Sigma(0 - x)^2}{\text{number of data points}}}$$

Equation 5:

$$VSI = \sqrt{\frac{\Sigma(\text{body weight} - z)^2}{\text{number of data points}}}$$

PSS Procedures

PSS was implemented on the experimental condition via a clinical electric stimulator (Vectra NEO, Chattanooga Rehab, Dallas, TX). Participants were seated in a chair while their common peroneal nerve was located and positioning of 1-in. round electrodes were determined. Somatosensory stimulation was delivered via a train of five pulses (pulse width: 1ms; pulse frequency 10 Hz, intensity range: suprasensory) (Conforto et al., 2002) for one hour during the experimental treatment session. A visual representation of these stimulation

parameters can be found in Figure 2. The threshold was continually monitored throughout the session in order to ensure continued sensation of paresthesia in the lower limb. In order to keep the duration of treatment constant, the control treatment session featured participants being seated without stimulation for an hour. To control arousal and attention during both treatment sessions, participants watched a nature documentary for the duration of their session.

Figure 2

PSS Stimulation Wave Schematic



Note. Example of one train of pulses delivered during the PSS intervention session. Each train is delivered ten times per second.

Data Analysis

Data was assessed with one-way repeated measures analyses of variance (ANOVA) with the factor of time (5 levels; Pre-Intervention, Post-Intervention, 24Hr-Post-Intervention,

Pre-Control, Post-Control) being assessed. For muscle activation during landing, factorial repeated-measures ANOVA was used to investigate a muscle (3 levels) and phase (2 levels) as additional factors. Fisher's least significant decrease (LSD) was used post hoc to determine locations of significant differences. Partial eta squared was used as a measure of ANOVA effect size with 0.01, 0.06, and 0.14 considered small, medium, and large, respectively. Effect sizes between specific time points (e.g., pre/post-intervention or pre/post control) were explored with Cohen's d, with 0.20, 0.50, and 0.80 considered small, medium, and large. An a priori level of significance was set at 0.05.

Results

Demographics

Twelve participants with CAI were recruited to participate in the study with ten ultimately completing the study (Table 1). Two male participants opted not to complete their initial testing session due to discomfort with transcranial magnetic stimulation (Figure 3).

Table 1

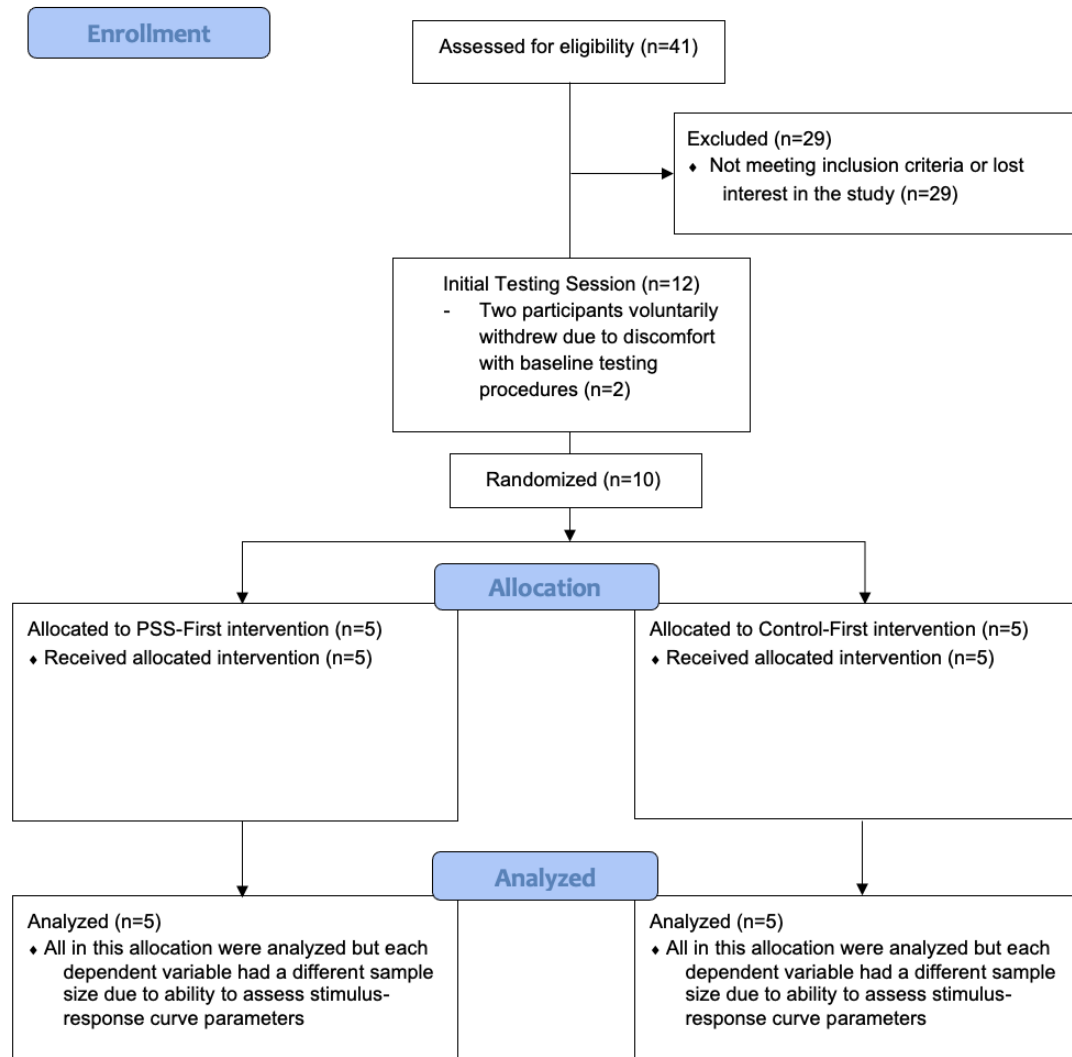
Participant Demographics

n	10
Sex (m/f)	6/4
Age (y)	22.1±3.6
Height (cm)	174.7±10.1
Mass (kg)	83.2±14.8
IFDAI	18.3±3.6

Note. Age, height, mass and IDFAI means ± standard deviation

Figure 3

CONSORT Flow Diagram



Note. CONSORT Flow Diagram of Subject Recruitment, Enrollment, Allocation and Analysis

Reflexive Excitability

H-Reflex

For reflexive excitability, ANOVA results revealed no significant time-by-muscle interaction effect ($n=8$) ($F_{[8,56]} = 0.586, p = 0.785, \eta^2 = 0.077$). However, there was a significant main effect of muscle ($F_{[2,14]} = 26.419, p < 0.001, \eta^2 = 0.791$). Fisher's LSD post hoc comparisons revealed the SOL had significantly higher H-reflexes than both the TA ($p=0.001$) and the PL ($p<0.001$). No significant differences were seen between TA and PL $H_{max}:M_{max}$ ratio. There was also no significant main effect of time ($F_{[1,7]} = 0.021, p = 0.889, \eta^2 = 0.003$). Effect sizes showed a medium increase in reflexive excitability of the TA from Pre-Int to Post-Int ($d=0.631$) (Table 2) (Figure 4).

Table 2

Hmax:Mmax Ratios

	Pre-Con	Post-Con	Pre-Con to Post- Con Effect Size (d)	Pre-Int	Post-Int	24Hr Post	Pre-Int to Post-Int Effect Size (d)	Pre-Int to 24Hr Post- Int Effect Size (d)
TA	0.137 (0.037)	0.153 (0.069)	0.274	0.130 (0.074)	0.195 (0.126)	0.152 (0.071)	0.631	0.312
PL	0.151 (0.106)	0.149 (0.090)	0.017	0.146 (0.083)	0.163 (0.075)	0.147 (0.106)	0.222	0.013
SOL^a	0.452 (0.215)	0.492 (0.203)	0.192	0.426 (0.202)	0.462 (0.174)	0.416 (0.154)	0.191	0.056

Note. Mean (Standard Deviation) and Effect Sizes (Cohen's d) $H_{max}:M_{max}$ Ratios across muscle - time. ^a Significant difference from the TA and PL

Cortical Excitability

MT

For MT, ANOVA results revealed no significant main effects of time for each of the muscles (Table 3): TA (n=8) ($F_{[4,28]} = 0.877, p = 0.490, \eta^2 = 0.111$), PL (n=5) ($F_{[4,16]} = 0.979, p = 0.446, \eta^2 = 0.197$), and SOL (n=6) ($F_{[4,20]} = 2.603, p = 0.067, \eta^2 = 0.342$). Effect sizes showed a large decrease in threshold intensity between Pre-Int to Post-Int ($d=1.067$) and Pre-Int to 24 Hr Post-Int ($d=1.031$) time points in the SOL (Figure 7). Medium-sized decreases were revealed Pre-Con to Post-Con in both TA ($d=0.697$) and SOL ($d=0.573$) (Figures 5 and 7).

Table 3

Motor Threshold Data

	Pre-Con	Post-Con	Pre-Con to Post- Con Effect Size (d)	Pre-Int	Post-Int	24Hr Post	Pre-Int to Post-Int Effect Size (d)	Pre-Int to 24Hr Post-Int Effect Size (d)
TA	46.495 (17.204)	36.414 (11.056)	0.697	39.289 (7.501)	43.632 (21.215)	37.488 (15.161)	0.273	0.151
PL	41.457 (16.438)	37.710 (2.901)	0.317	33.388 (13.460)	34.469 (13.192)	31.855 (12.686)	0.081	0.117
SOL	39.543 (7.498)	42.985 (3.982)	0.573	46.217 (14.724)	31.673 (12.433)	30.234 (16.237)	1.067	1.031

Note. Means (Standard Deviations) and Effect Sizes (Cohen's d) of MT across muscle and time.

I₅₀

For *I₅₀*, ANOVA results revealed no significant main effects of time for each of the TA (n=5) ($F_{[4,16]} = 1.455, p = 0.262, \eta^2 = 0.267$), PL (n=5) ($F_{[4,16]} = 1.062, p = 0.407, \eta^2 = 0.210$) and SOL (n=5) ($F_{[4,16]} = 0.566, p = 0.691, \eta^2 = 0.124$) (Table 5). Effect sizes showed a large decrease in *I₅₀* in the SOL from Pre-Int to Post-Int (d=0.920) (Figure 10). Medium-to-large sized decreases in threshold intensity were seen Pre-Int to Post-Int in both TA (d=0.668) and PL (d=0.644) (Figures 8 and 9).

Table 4

I₅₀ Data

	Pre-Con	Post-Con	Pre-Con to Post-Con Effect Size (d)	Pre-Int	Post-Int	24 HR Post	Pre-Int to Post-Int Effect Size (d)	Pre-Int to 24Hr Post-Int Effect Size (d)
TA	53.558 (6.490)	53.254 (5.144)	0.052	51.428 (8.981)	46.892 (3.388)	50.972 (9.623)	0.668	0.049
PL	53.367 (3.800)	52.491 (5.673)	0.182	51.326 (6.120)	48.454 (1.525)	49.531 (4.363)	0.644	0.338
SOL	59.106 (14.285)	55.750 (6.425)	0.303	56.628 (5.516)	52.597 (2.825)	54.163 (11.089)	0.920	0.282

Note. Means (Standard Deviations) and Effect Sizes (Cohen's d) of *I₅₀*

across muscle and time.

MEP_{max}

For *MEP_{max}*, ANOVA results revealed no significant main effects of time for the TA (n=5)

($F_{[4,16]} = 0.519, p = 0.723, \eta^2 = 0.115$), PL (n=4) ($F_{[4,12]} = 2.940, p = 0.066, \eta^2 = 0.495$), and the SOL (n=5) ($F_{[4,16]} = 0.434, p = 0.782, \eta^2 = 0.098$). Cohen's d calculation revealed a large effect size from Pre-Con to Post-Con (d=1.028) for the PL. Effect sizes revealed a medium increase in excitability from Pre-Int to Post-Int (d=0.505) and Pre-Int to 24 Hr Post-Int (d=0.733) for the TA (Figure 11) and Pre-Int to 24 Hr Post-Int (d=0.578) for the PL (Figure 12).

Table 5

MEPmax Data

	Pre-Con	Post-Con	Pre-Con to Post-Con Effect Size (d)	Pre-Int	Post-Int	24 Hour Post	Pre-Int to Post-Int Effect Size (d)	Pre-Int to 24Hr Post-Int Effect Size (d)
TA	0.334 (0.227)	0.322 (0.225)	0.005	0.246 (0.098)	0.369 (0.331)	0.347 (0.170)	0.505	0.733
PL	0.109 (0.066)	0.059 (0.018)	1.028	0.094 (0.062)	0.080 (0.046)	0.123 (0.034)	0.264	0.578
SOL	0.143 (0.161)	0.103 (0.145)	0.263	0.202 (0.374)	0.153 (0.217)	0.088 (0.083)	0.160	0.419

Note. Means (Standard Deviations) and Effect Sizes (Cohen's d) of Maximal M-Wave Amplitude across muscle and time.

MEP sizes

For MEP size, ANOVA results revealed a significant muscle-by-Intensity (n=9) ($F_{[2,16]} = 5.765, p = 0.013, \eta^2 = 0.419$) interaction effect. Fisher's LSD post hoc comparisons revealed significant differences between PL and SOL at both 90% (p=0.030)

and 110% ($p=0.006$) intensities and between TA and SOL at 110% ($p=0.018$). ANOVA results also revealed significant muscle ($F_{[2,16]} = 4.685, p = 0.025, \eta^2 = 0.369$) and intensity ($F_{[1,8]} = 18.347, p = 0.003, \eta^2 = 0.696$) main effects.

ANOVA results revealed non-significant time-by-muscle ($F_{[8,64]} = 1.394, p = 0.216, \eta^2 = 0.148$), time-by-Intensity ($F_{[4,32]} = 0.591, p = 0.671, \eta^2 = 0.069$) and time-by-muscle-by-intensity ($F_{[8,64]} = 1.418, p = 0.206, \eta^2 = 0.151$) interaction effects.

ANOVA results also revealed a no significant main effect of time ($F_{[4,32]} = 0.488, p = 0.744, \eta^2 = 0.058$) (Table 6).

Cohen's d calculations revealed effect sizes of MEP amplitudes at different intensities. Effect sizes revealed a medium decrease in response from Pre-Int to 24 Hr Post-Int ($d=0.580$) at the SOL at 90% intensity. At 110% intensity, a medium decrease was also revealed at the SOL from Pre-Int to 24 Hr Post-Int ($d=0.643$).

Table 6*MEP Intensities 90/110 Data*

		Pre- Con	Post- Con	Pre-Con to Post- Con Effect Size (d)	Pre-Int	Post-Int	24Hr Post	Pre-Int to Post- Int Effect Size (d)	Pre-Int to 24Hr Post- Int Effect Size (d)
90	TA	0.036 (0.026)	0.037 (0.035)	0.039	0.035 (0.023)	0.046 (0.051)	0.025 (0.026)	0.298	0.369
	PL^a	0.084 (0.080)	0.056 (0.042)	0.444	0.051 (0.045)	0.141 (0.127)	0.053 (0.039)	0.124	0.045
	SOL	0.016 (0.011)	0.013 (0.010)	0.245	0.065 (0.114)	0.036 (0.044)	0.017 (0.029)	0.336	0.580
110	TA^a	0.112 (0.056)	0.142 (0.080)	0.432	0.092 (0.062)	0.119 (0.058)	0.112 (0.082)	0.441	0.264
	PL^a	0.147 (0.08)	0.164 (0.150)	0.140	0.148 (0.154)	0.141 (0.127)	0.166 (0.140)	0.049	0.120
	SOL	0.044 (0.033)	0.042 (0.033)	0.070	0.104 (0.152)	0.084 (0.119)	0.034 (0.029)	0.094	0.510

Note. Means (Standard Deviations) and Effect Sizes (Cohen's d) of MEP 90/110 across intensity, muscle, and time.

^a Significantly different from the SOL

Performance Measures

Postural Stability Indices

For DPSI, ANOVA results revealed no significant main effect of time (n=10) ($F_{[4,36]} = 2.606, p = 0.052, \eta^2 = 0.225$) (Table 7).

ANOVA results of the individual components of DPSI revealed a non-significant time-by-direction interaction effect ($F_{[8,72]} = 1.314, p = 0.250, \eta^2 = 0.127$). ANOVA results did reveal significant main effects of time ($F_{[4,36]} = 3.174, p = 0.025, \eta^2 = 0.261$)

and direction ($F_{[2,18]} = 683.491, p = 0.000, \eta^2 = 0.987$) for PSI components. Fisher's LSD post hoc comparisons revealed significant differences between the Post-Con and Pre-Int time points ($p=0.045$) and the Post-Int and 24Hr Post-Int time points ($p=0.003$) when comparing VSI to the MLSI & APSI directions.

Table 7

Postural Stability Indices

	Pre-Con	Post-Con	Pre-Con to Post-Con Effect Size (d)	Pre-Int	Post-Int	24Hr Post	Pre-Int to Post-Int Effect Size (d)	Pre-Int to 24Hr Post-Int Effect Size (d)
DPSI	0.478 (0.050)	0.472 (0.053)	0.116	0.492 (0.046)	0.491 (0.057)	0.469 (0.048)	0.019	0.489
MLSI	0.031 (0.004)	0.031 (0.005)	0.000	0.037 (0.010)	0.033 (0.008)	0.030 (0.005)	0.442	0.885
APSI	0.131 (0.015)	0.137 (0.012)	0.442	0.137 (0.012)	0.136 (0.013)	0.132 (0.019)	0.080	0.315
VSI	0.459 (0.049)	0.453 (0.053)	0.118	0.470 ^a (0.046)	0.469 (0.058)	0.449 ^a (0.049)	0.019	0.442

Table 7. Means (Standard Deviations) and Effect Sizes (Cohen's d) of postural stability indices across time groups.

^a Significantly different from MLSI and APSI

Muscle Activation

For muscle activation, ANOVA results revealed non-significant interaction effects of time-by-muscle-by-phase ($n=10$) ($F_{[8,72]} = 0.282, p = 0.970, \eta^2 = 0.030$), time-by-muscle ($F_{[8,72]} = 0.868, p = 0.547, \eta^2 = 0.088$), time-by-phase ($F_{[4,36]} = 0.148, p = 0.963, \eta^2 =$

0.016), or muscle-by-phase ($F_{[2,18]} = 1.243, p = 0.312, \eta^2 = 0.121$). Additionally, there was no significant main effect of time ($F_{[4,36]} = 1.138, p = 0.354, \eta^2 = 0.112$). Significant main effects of muscle ($F_{[2,18]} = 7.530, p = 0.004, \eta^2 = 0.456$) and phase ($F_{[1,9]} = 27.192, p = 0.001, \eta^2 = 0.751$) were observed with Post-Landing phases revealing higher activation than Pre-Landing and PL and SOL muscles having greater activation than the TA at both phases. Effect sizes revealed a medium decrease in muscle activation in the SOL from Pre-Int to Post-Int ($d=0.563$) in the Post-Landing phase.

Table 8

EMG – Balance

Muscle	Phase	Pre-Con	Post-Con	Pre-Con to Post-Con Effect Size (d)	Pre-Int	Post-Int	24HR Post	Pre-Int to Post-Int Effect Size (d)	Pre-Int to 24HR Post-Int Effect Size (d)
TA	Pre	0.261 (0.071)	0.290 (0.080)	0.383	0.296 (0.107)	0.258 (0.117)	0.293 (0.123)	0.339	0.026
	Post	0.450 (0.170)	0.424 (0.144)	0.165	0.470 (0.122)	0.445 (0.140)	0.445 (0.124)	0.190	0.203
PL	Pre	0.423 (0.108)	0.452 (0.132)	0.240	0.424 (0.159)	0.406 (0.147)	0.419 (0.138)	0.118	0.034
	Post	0.563 (0.155)	0.574 (0.179)	0.066	0.535 (0.165)	0.523 (0.183)	0.545 (0.111)	0.069	0.071
SOL	Pre	0.467 (0.156)	0.478 (0.091)	0.086	0.408 (0.092)	0.385 (0.090)	0.443 (0.149)	0.253	0.283
	Post	0.574 (0.114)	0.575 (0.144)	0.008	0.525 (0.086)	0.474 (0.095)	0.542 (0.131)	0.563	0.153

Note. Means (Standard Deviations) and Effect Sizes (Cohen’s d) for EMG activity during dynamic balance task across muscle, phase, and time groups.

Discussion

Introduction

The purpose of this study was to evaluate the feasibility & efficacy of a PSS-based intervention on modifying neural excitability and dynamic balance following a single-session treatment in adults with CAI. We hypothesized that PSS would increase neural excitability and improve dynamic balance beyond the individual's baseline. While we failed to see any statistically significant differences across time for neural excitability measures, there were encouraging effect sizes that support our hypotheses that a PSS intervention may be effective in increasing excitability in a larger subset. Statistical significance was likely not achieved due to sample size, where we were unable to fit a number of individual data points to curve parameters due to inconsistencies that would have affected the goodness of the fit ($R^2 \geq 0.7$) of the curve. However, it is worth noting that all subjects that completed initial testing were able to withstand the intervention with no adverse events reported, supporting the feasibility of utilizing PSS in this population. The presence of promising effect sizes that revealed increased H:M ratios in the TA, decreased MT in the SOL and TA and decreased I₅₀ measurements also in the SOL and TA from Pre-Int to Post-Int and Pre-Int to 24 Hr Post-Int suggest that single-session utilization of PSS may have some effect on increasing neural excitability that warrant further investigation.

Neural Excitability

There were no statistically significant changes in measures of reflexive excitability over the course of the intervention, but there was a sizable effect size increase in H:M ratio in

the TA from the Pre-Int time point to the Post-Int time point. Increases in the H:M ratio support the idea that a sensory stimulation intervention could improve reflexive excitability as these findings are consistent with previous research utilizing PSS that found increased sensory input is effective at increasing motor activation (Khaslavskaia et al., 2002; Khaslavskaia & Sinkjaer, 2005). The increased H:M ratio suggests that there was a greater quantity of motor neurons being recruited and an increase in alpha motor neuron pool excitability in the spinal cord in response to the electrical stimulus being applied to the nerves. This can potentially improve muscle recruitment in the stabilizing muscles (TA, PL and SOL), which causes the increase in the ratio (Palmieri et al., 2004). These excitability increases were identified as occurring in the motor cortex due to increased synaptic efficacy following the stimulation of the afferent fibers of the common peroneal nerve (Khaslavskaia et al., 2002). This finding is important for people with CAI because the H-reflex can determine excitability changes in this population which is generally observed to have decreased excitability following injury.

There were no statistically significant differences seen in measures of cortical excitability; however, we did observe medium-to-large effect sizes from Pre-Int to Post-Int and Pre-Int to 24Hr Post-Int time points. The data lost due to the inability to fit a stimulus-response curve to the Boltzmann equation (Equation 1) with adequate goodness of fit potentially contributed to the lack of statistically significant differences in this data, but the effect sizes showed encouraging trends. Decreased I_{50} measurements were observed in all three muscles while decreases in MT were observed in just the SOL at the Pre-Int to Post-Int time points. MEP_{max} increases were also observed in the TA from Pre-Int to Post-Int while decreases were observed in I_{50} and MT measures. An unexpected change we also found was medium decreases of MT measures in the TA and SOL from Pre-Con to Post-Con time

points, possibly indicating that excitability can increase as participants became more familiar and comfortable with the testing procedures.

The decreases in the effect sizes of MT in the SOL and I₅₀ measures in all muscles from the Pre-Int to Post-Int time points indicate a leftward shift in the stimulus-response curve which can be attributed to a decreased recruitment threshold for motor neurons that could lead to quicker and greater activation of the respective muscles following the PSS intervention (Devanne et al., 1997). In a study by Khaslavskaia et al., (2002) they found MEP_{max} changes following 60 minutes of sensory stimulation (split over 2 sessions in the previous study) of the common peroneal nerve consistent with the trends we saw in our data. In their study, MEP_{max} increased significantly by a greater than 50% growth following the sensory stimulation of the afferent fibers of the nerve, suggesting greater neuron recruitment following the stimulation (Khaslavskaia et al., 2002). These findings were discovered in healthy populations, which can explain the differences in their findings versus ours, but even in a different population the findings are promising for individuals with CAI. The quicker and higher-total muscle activation supports the theory that PSS could help decrease re-injury rates in people with CAI as previous research indicates that diminished neural excitability of surrounding stabilizing muscles is one of the earliest onset deficiencies of musculoskeletal injury (Kim et al., 2019; Lepley & Lepley, 2021; Norte et al., 2021; Pietrosimone & Gribble, 2012). If further research utilizing PSS continues to see greater muscle activation following the stimulation, its incorporation into a clinical rehabilitation program could be beneficial in reducing re-injury rates.

Not all of our cortical excitability measures exhibited meaningful changes nor encouraging effect sizes, as neither MEP 90/110 measures nor MEP_{max} variables revealed any significant differences over time. These variables indicate the lack of change in cortical

motor neuron firing before and after receiving the intervention which we would have expected to increase significantly following PSS treatment. There were also inconsistencies between muscles as some measures such as MT showed large effect sizes for the SOL but small sizes for the TA and PL. Other measures such as MEP_{max} revealed inconsistencies between time points where the PL had a large effect from Pre-Con to Post-Con but neither the TA nor SOL have even a small effect between those time points. These differences, though inconsistent, may have been due to the differing innervations of the muscles, as the TA and PL are both innervated by the common peroneal nerve, but the SOL is innervated by the tibial nerve. This difference could have led to the inconsistent MT changes since we targeted only the common peroneal with the PSS intervention. Our small sample size also may have been a contributing factor to these variables not revealing significant differences as a post-hoc power analysis revealed that for underpowered variables, we would have required a range of 8-16 participants to have results at appropriate power.

Performance Measures

Postural stability and muscle activation measures revealed no significant effects of time-by-muscle for either measure but did reveal significant main effects of time and direction for postural stability and significant main effects of time and phase for muscle activation. Effect sizes revealed decreases in all three components of DPSI from the Pre-Int to 24Hr Post-Int time points, with the largest sized decreases being seen for MLSI direction. Though the decrease in these measures is potentially encouraging for the intervention's efficacy, as the decreased muscle activation indicates improved balance, there is a potential factor of time of testing session present that could also have been a cause of the improved stability. Due to the randomized crossover design of the study, half of the participants

received the control before the intervention which would have resulted in them performing the hop-to-stabilization task twice before the intervention measures were recorded. Another possible factor influencing improvements was that both Post-Control and Post-Intervention measures were prefaced by 5 hops in the Pre-Control and Pre-Intervention time points, which could have also factored into the learning effect. Our outcomes of non-significant balance improvements are not surprising though, as previous studies have discovered a link between muscle activation and improved balance (Rosen et al., 2013) and we saw no improvements in muscle activation across time. It is theorized that increases in cortical excitability could have the potential to improve muscle activation which in turn would increase stability. Although our intervention revealed encouraging trends of increased excitability, one intervention of PSS may not have been enough stimulation to evoke the changes needed to see balance improvements.

Previous investigations have focused on PSS efficacy in regaining function and strength in the upper extremity of populations post-subacute stroke and have noted that strength and perceived function improved (Conforto et al., 2002) following stimulation. Conforto's study, along with research by the Klaiput research team (Klaiput & Kitisomprayoonkul, 2009), did see functional increases via grip and pinch strength following single-session utilization of PSS, but these studies administered 2 hours of stimulation to their sub-acute stroke populations compared to our hour-long intervention to the lower extremity, similarly to the design by Khaslavskaja (2002). Given the CAI population utilized in our study, we would want to see functional improvements in balance and/or strength following the intervention which previous research has shown is a realistic expectation. Bruce et al., (2020) found that improving neural excitability in this population can result in improvements in balance and strength over several weeks of receiving neural stimulation,

though a different intervention than PSS. Before incorporating PSS into a clinical intervention program, we would need to see similar functional improvements both immediately following treatment as well as over several weeks like the observations in the Bruce et al., (2020) study.

Conclusion

This is the first study to examine the feasibility and efficacy of PSS in a population with musculoskeletal injuries, determining its effects on neural excitability and dynamic postural stability. Despite non-significant results likely due to an underpowered sample, the effect sizes from our results warrant further investigation into whether or not PSS can improve neural excitability and postural stability in patients with CAI. It was potentially efficacious for improving reflexive excitability to the lower leg muscles and cortical excitability from the common peroneal nerve to the brain, but there was limited impact on balance. Overall, this study led us to believe that the implementation of PSS in clinical rehabilitation of ankle sprains is feasible, but the efficacy of the intervention in this population is still unknown and requires further research with a greater sample size in order to obtain statistically significant results.

The concepts investigated in this study have potentially significant implications for a clinical practitioner, though there are several steps before they are ready to be used in a clinical setting. Our PSS intervention was administered by a clinically-accessible stimulator making its future clinical utilization an inexpensive option. However, before use as a therapeutic treatment, future randomized controlled trials with a larger sample size need to first be performed. Additionally, further research testing should be conducted to determine the proper dosage of stimulation, duration of treatment and number of interventions needed to maximize long-term outcomes of the stimulation.

Review of Literature

Introduction

Ankle sprains are the most common injury among physically active individuals, affecting approximately 60% of this population (Hiller et al., 2012). These ankle sprains have a negative effect on the physical activity level of the injured individuals, decreasing their quality of life as a result (Houston et al., 2015). Studies have shown that individuals with CAI have increased difficulty performing ADL's, are overall less physically active, and reported diminished personal well-being (Houston et al., 2015; Hubbard-Turner & Turner, 2015). Research also suggests that nearly two-thirds of all ankle injuries result in re-injury and present a 40% chance of development of CAI (Hertel & Corbett, 2019; Wright et al., 2017). CAI is best expressed as repeated sensations of "giving way" or rolling of the ankle, which are both often associated with recurrent injury (Hertel & Corbett, 2019). The recurrent nature of ankle sprains can be attributed to neuroplasticity changes in the brain, particularly decreased cortical excitability (Needle et al., 2017), which contributes to the lingering effects of the initial injury. Therefore, the importance of evolving rehabilitation strategies is essential to combat the persistency of ankle instability due to its prevalence in the general population. The utilization of an alternative rehabilitation strategy such as PSS, which can address the neurological changes and neural deficiencies that result from CAI, may bring light to the injury and open the door to additional treatments that decrease the re-injury rate and increase the functional restoration of the ankle joint.

Etiology of Chronic Ankle Instability

The ever-developing theories about the cause of recurrent ankle injuries have focused on a combination of mechanical and neurological damages rather than the dated theory of primarily mechanical damage being the cause (Hertel & Corbett, 2019). Some mechanical changes are present in ankle injuries, specifically individuals with CAI, likely due to the decreased collagen fiber content in the ankle joint, which leads to decreased stiffness and increased laxity (Wikstrom et al., 2019). However, research has led us to progress from the theory that only mechanical changes play a role in the recurrent spraining of the ankle in individuals with CAI to the current understanding that CNS changes occur as well. A research study conducted by Hass et al., (2010) noted changes in motor planning due to increased lateral translation of the center of pressure in patients with CAI, indicating modified motor planning that places these patients at risk of re-injury (Hass et al., 2010). Recent research has begun to investigate CNS changes by utilizing non-invasive techniques better to understand their role in re-injury and development of CAI.

We can understand the CNS by measuring and tracking changes using non-invasive brain functionality measures like functional magnetic resonance imaging (fMRI), electroencephalography (EEG), or transcranial magnetic stimulation (TMS) (Kapreli et al., 2009; Needle et al., 2017). fMRI is a technique used to visualize hemodynamic changes occurring across the entire brain by quantifying the hemodynamic response function from blood oxygen level-dependent reactions (Friston et al., 1995). EEG is used for understanding the timing and type of processing occurring during sensorimotor stimuli and functional tasks due to the excellent temporal, but limited spatial, resolution it provides compared to other neuroimaging techniques (Needle et al., 2017). TMS is used as a tool to investigate alterations of the cortices and the functional integrity and excitability of descending motor

pathways to control muscle following joint injury (Needle et al., 2017). Reflexive excitability, the number of motor neurons capable of responding to an excitatory stimulus within a given motor neuron pool, is another change that can be tracked using brain functionality measures (Klykken et al., 2011). This functionality can be tested by determining the Hoffmann reflex (H-reflex), which measures alpha motor neurons' reflexive excitability within a targeted motor pool (Klykken et al., 2011). The H-reflex can be used to determine changes in muscle excitability and response to a peripheral stimulus which can be used to determine or monitor changes following injury. The utilization of these interventions in populations of individuals with musculoskeletal injuries has led to new insights regarding the cause of the injury recurrence, typically among patients with CAI (Kim et al., 2019).

The CNS has the ability to adapt according to stimuli it receives from its various afferent pathways and when this adaptation occurs in the brain and causes the brain to make changes to its cortical properties, it is referred to as neuroplasticity (Hebb, 1949; Kapreli et al., 2009). Neuroplasticity occurs following ligamentous injury when there are alterations to sensory feedback from the joint, due to the increased laxity and deafferentation caused from the injury (Needle et al., 2017). These alterations contribute to decreased cortical excitability and increased demand on the movement planning areas during motor tasks (Needle et al., 2017). Neuroplastic changes that alter proprioception and motor planning include decreased sensation and somatosensory activation, both of which play a role in increasing the risk of reinjury. A study by Needle et al., (2014) compared somatosensory activation and changes to ankle loading in healthy populations versus those with CAI and found that people with CAI are less able to couple the sensory activation with the amount of ligamentous laxity throughout the load (Needle et al., 2014). These findings indicate that following injury, the ankle joint has increased difficulty interpreting load on the joint, leading to the joint reaching

harmful joint angles that it would have prevented in the uninjured state. Further studies included in the review by Kim et al., (2019) discovered that neurological changes following recurrent ligamentous injuries lead to diminished reflexive excitability of surrounding stabilizing muscles, changes in cortical excitability in the descending corticospinal pathways, peripheral deafferentation of sensory receptors, and impaired postural control suffered from the sprain(s) (Hass et al., 2010; Kim et al., 2019; O’Driscoll & Delahunt, 2011; Pietrosimone & Gribble, 2012). The diminished excitability caused by recurrent injury results in fewer available motor neurons in response to the stimulus, leading to delayed activation of the protective muscles surrounding the joint. The decreased excitability is not limited to the segmental level, though it is notable within the motor cortex. The combination of peripheral deafferentation and the subsequent decreased reflexive excitability results in a greater demand on the CNS and causes the motor cortices to remap in such a way that it places individuals at risk for further injury (Needle et al., 2017). This ultimately leads to the change in motor planning as described by Hass et al., (2010) as patients with CAI had increased lateral translation of the center of pressure during gait initiation, indicating modified motor planning that places these patients at risk of re-injury (Hass et al., 2010).

This research provides evidence that supports the hypothesis that decreased motor excitability is present in people with CAI. Thus, decreased motor excitability needs to be considered for interventions because it leads to an increase in reoccurrence to injury and continued ankle motor and neural dysfunction. The muscle inhibition caused by these excitability changes can be persistent and can negatively affect the motor cortex by placing further demand on the motor planning and visual areas of the brain rather than the somatosensory system via sensory feedback from the ankle (Kim et al., 2019; Needle et al., 2017). With the increased visual processing and motor planning, the brain activates and

initiates motor drive differently than in the pre-injured state resulting in different movement patterns than before injury and ultimately leading to recurrent injury (Needle et al., 2017).

Treatment Methods for Ankle Instability

The current treatment strategies for rehabilitation of ligamentous injuries are ineffective in preventing the re-injury of a chronically unstable ankle, evidenced by rates of re-injury up to 47% following an initial ankle sprain (Herzog et al., 2019). The current standard of care for ankle sprains is impairment-based rehabilitation, which is treatment that focuses on physical deficiencies. This impairment-based rehabilitation includes balance training and functional strength training. However, research has revealed that although these interventions result in increased patient self-reported function, they ultimately have not positively affected the chance of re-injury because they are addressing only the neuromuscular aspects of the injury, not restoring CNS function caused by the injury (McKeon et al., 2008; Wright et al., 2017). There is inconclusive evidence regarding patient adherence to ligamentous injury rehabilitation programs, but a recent review conducted by Walker et al., (2020) found inconsistent correlations between adherence to these programs and patient strength and balance measures (Walker et al., 2020). These current clinical rehabilitation methods may be effective in restoring self-reported function, but without positively affecting the recurrent injury issue, there needs to be an advancement of the interventions to better restore CNS function caused by the injury.

Neuromodulatory therapies, which utilize technology to modify nerve activity by delivering treatment directly to the targeted area, are an emerging part of current clinical rehabilitation. These clinical therapies enhance learning, reduce pain and improve function by increasing corticomotor excitability in a more targeted manner than typical clinical

interventions (Schabrun et al., 2013). Some commonly used neuromodulatory techniques – like Kinesio-taping, focal joint cooling and joint mobilization – are used to enhance learning, reduce pain and improve patient function but have resulted in minimal clinical effects, requiring more advanced clinical options (Harkey et al., 2014). Another popular technique that is used in most clinical settings is neuromuscular electrical stimulation (NMES), which utilizes electrical stimulation to increase corticomotor excitability, but its effects in patients with CAI have not been studied.

One recent investigation by Bruce et al., (2020) used a cortically-mediated intervention aimed at restoring neural excitability. They found that in individuals with CAI, increases in excitability can be manipulated via tDCS, a neuromodulatory intervention applying a small electrical current between areas of the cortex, in conjunction with an eccentric training program (Bruce et al., 2020). Cortical excitability and muscle activation were monitored and compared during dynamic balance tasks over the 4-week training program, with improvements in both, as well as hopping tasks, resulting from the intervention. Another finding from this study was the success of neuromodulatory therapy in increasing retention. The experimental group who received tDCS along with their training program retained their balance increases weeks after the training concluded. At the same time, the group that did not receive the tDCS showed no signs of retention (Bruce et al., 2020). This study suggests that other forms of neuromodulatory therapy targeting improved cortical excitability and muscular activation may be effective in increasing functionality in the CAI population.

Peripheral Somatosensory Stimulation

PSS is a therapeutic approach administered via electrical stimulation over a peripheral nerve in an effort to increase motor function by inducing cortical plasticity (Conforto et al., 2010; Klaiput & Kitisomprayoonkul, 2009; Stockley et al., 2020). It is a widely available method of treatment due to its ability to be delivered via various electrical stimulation units. Somatosensory stimulation via peripheral nerve stimulation has been proven to enhance activation of the primary sensorimotor cortex while also increasing cortical excitability in contralateral cortices (Celnik et al., 2007). The modern utilization of sensory stimulation has primarily been in stroke populations in an effort to promote increased motor function in the paretic limb in the upper extremity. Consistently successful measures have been discovered in these stroke populations in particular, with many studies finding immediate and prolonged increases in hand strength in populations suffering from acute, sub-acute or chronic stroke (Celnik et al., 2007; Conforto et al., 2002, 2007, 2010; Ghaziani et al., 2018; Kattenstroth et al., 2018; Klaiput & Kitisomprayoonkul, 2009; Peurala et al., 2002) (Table 9). The efficacy of PSS for functional retention has been noted, as well, in these populations through post-treatment testing indicating that functional measures were maintained up to 3 months following the completion of repetitive peripheral sensory (Conforto et al., 2010).

Table 9*PSS Stimulation Parameters from Previous Research*

	Celnik et al., (2007)	Conforto et al., (2010)	Conforto et al., (2007)	Conforto et al., (2002)	Ghaziani et al., (2018)	Kattenstroth et al., (2018)	Klaiput & Kitisomprayoonkul, (2009)	Peurala et al., (2002)
Total dose delivered (hrs; (duration x frequency))	2	24	2	2	125	7.5	2	14.7
Duration of each session (hrs)	2	2	2	2	1	0.75	2	2 x 0.33 day
Frequency of Therapy	One session	3x per week for 1 month (12 sessions)	One session	One session	Daily throughout hospital stay (max 4 weeks)	5x per week for 2 weeks	One session	3-week inpatient period (average: 21.6 +/- 6 sessions)
Intensity (Hz)	Each train = 5 pulses, 1ms duration delivered at 10Hz	Each train = 5 pulses, 1ms duration delivered at 10Hz	1ms duration delivered at 10Hz	Each train = 5 pulses, 1ms duration delivered at 10Hz	Suprasensory: Continuous deliver of 10Hz, 250microsec; Subsensory: 10Hz delivered over 3s every 2.5 mins, 250microsec	Each train delivered at 20Hz burst for 1.4s with 5s inter-train intervals	Each train = 5 pulses, 1ms duration delivered at 10Hz for 500ms	Monophasic constant current twin pulses at 50Hz
Format of RSS	2 electrode bars to stimulate ulnar and median nerves	2 electrode bars to stimulate median nerve	Surface electrodes to stimulate median nerve	Surface electrodes to stimulate median nerve	Surface electrodes on wrist, elbow, and shoulder	Glove with built in electrodes contacting each fingertip	Carbon rubberized electrodes overlaying ulnar and median nerve sites	Glove or shock electrode (wrist)
Length of Follow Up	1h and 24hr Post-test	1 month and 2-3 months	Immediately after RSS and 1 month later	Immediately after/24h after RSS	Post intervention and 6 months	Mean 2.9 +/- 1.4 days	Immediately after RSS only	Immediate after 3-week period only

However, there has been minimal research conducted in the lower extremities. The limited existing research has been performed on healthy populations and has not yet ventured into the area of musculoskeletal injury. That research utilized repetitive electrical nerve stimulation over the common peroneal nerve both at rest and following 30-minute exercise bouts (Khaslavskaia & Sinkjaer, 2005). The researchers found that even in populations unaffected by injury, increased sensory input may be effective in promoting motor learning and muscle strength and function by increasing cortical excitability and subsequently, increasing motor activation (Khaslavskaia et al., 2002; Khaslavskaia & Sinkjaer, 2005). Current research leads us to believe there may be a place for PSS in the rehabilitation space of ligamentous injuries. The primary benefit of PSS methods is the increased neural excitability it causes, which data suggests is a promising intervention for improving motor function in individuals with ligamentous joint injury (Bruce et al., 2020; Needle et al., 2017). In addition to the increased excitability of the stimulated peripheral nerve, research indicates that PSS over the common peroneal nerve (CPN) is effective in increasing motor evoked potential of the agonist muscle (tibialis anterior) without also increasing the motor potential of the antagonist muscle (soleus) which further increases the neural excitability of the joint (Khaslavskaia et al., 2002). This may further increase the efficacy of PSS in the CAI population as previous research has indicated that one of the earliest onset deficiencies seen in ankle instability is the decreased reflexive excitability of surrounding stabilizer muscles (Pietrosimone & Gribble, 2012).

PSS is a non-invasive treatment, and the safety of the stimulation has been reviewed and there have been no serious adverse effects across multiple studies in the paretic population (Stockley et al., 2020). In addition, some studies have also paired PSS with TMS

with no reported adverse effects among their testing populations (Celnik et al., 2007; Conforto et al., 2010).

Summary

There is a need for additional research regarding alternative treatment options for individuals with CAI due to the prevalence of the injury. The current rehabilitation methods have proven to be inadequate likely due to the lack of focus on the cortical neuroplasticity that occurs as a result of the injury and re-injury (Needle et al., 2017). Following the initial sprain and re-injury, decreased cortical excitability and neural activation make it difficult for individuals with CAI to return to their pre-injured states, resulting in adverse gait patterns and continued increase of re-injury (Needle et al., 2017). Impairment-based rehabilitation treatments remain ineffective for limiting re-injury, requiring a shift in focus to rehabilitation alternatives addressing the neurological effects of CAI. PSS has generated positive preliminary results in both upper extremity tests in paretic populations and lower extremity tests in healthy populations, resulting in increased cortical excitability and muscular activation in both parties (Celnik et al., 2007; Conforto et al., 2010; Khaslavskaja et al., 2002). Although somatosensory stimulation has yet to be tested in populations with musculoskeletal injuries, we believe PSS may improve ankle function, neural activation, and cortical excitability in individuals with CAI.

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Appendix A

Raw Data Figures

Figure 4

Hmax:Mmax Ratios for Muscles

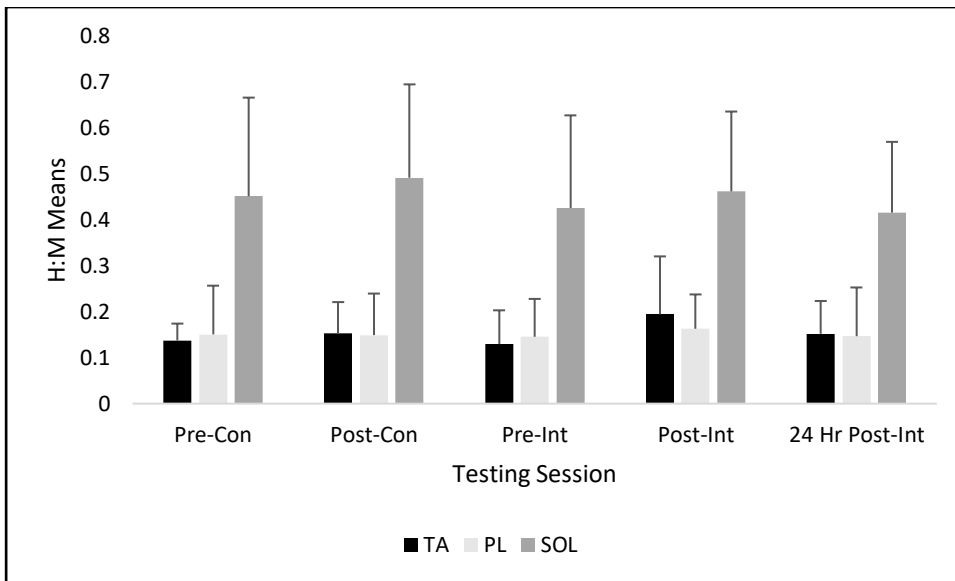


Figure 5

Motor Threshold for Tibialis Anterior over Time

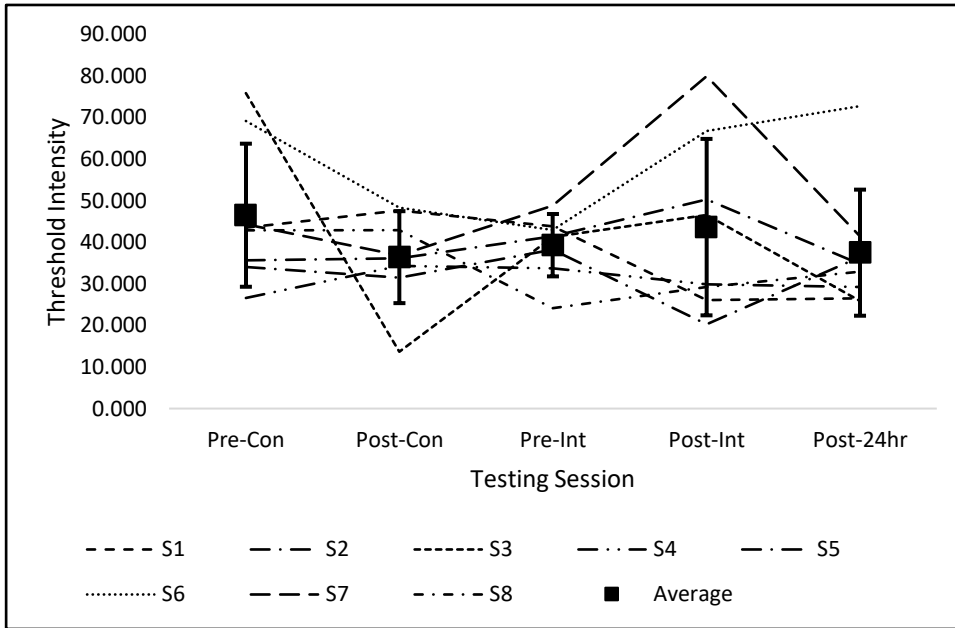


Figure 6

Motor Threshold for Peroneus Longus over Time

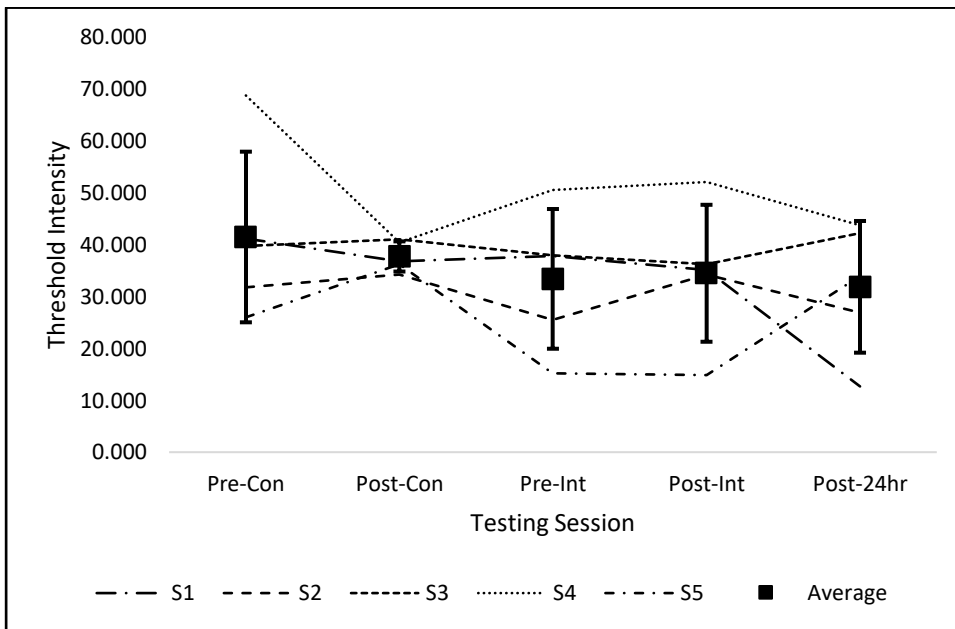


Figure 7

Motor Threshold for Soleus over Time

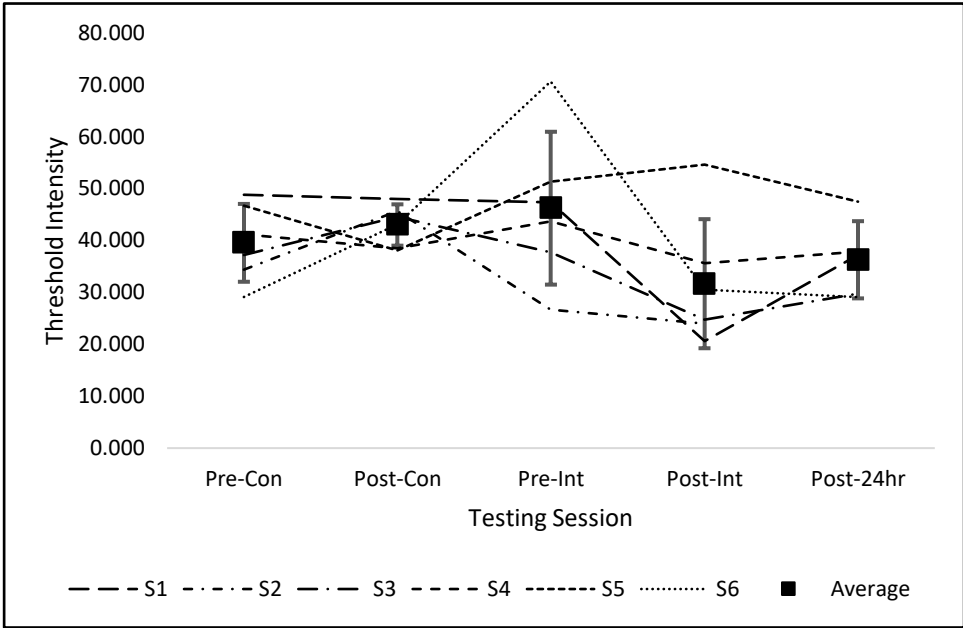


Figure 8

Midpoint Intensity of Tibialis Anterior over Time

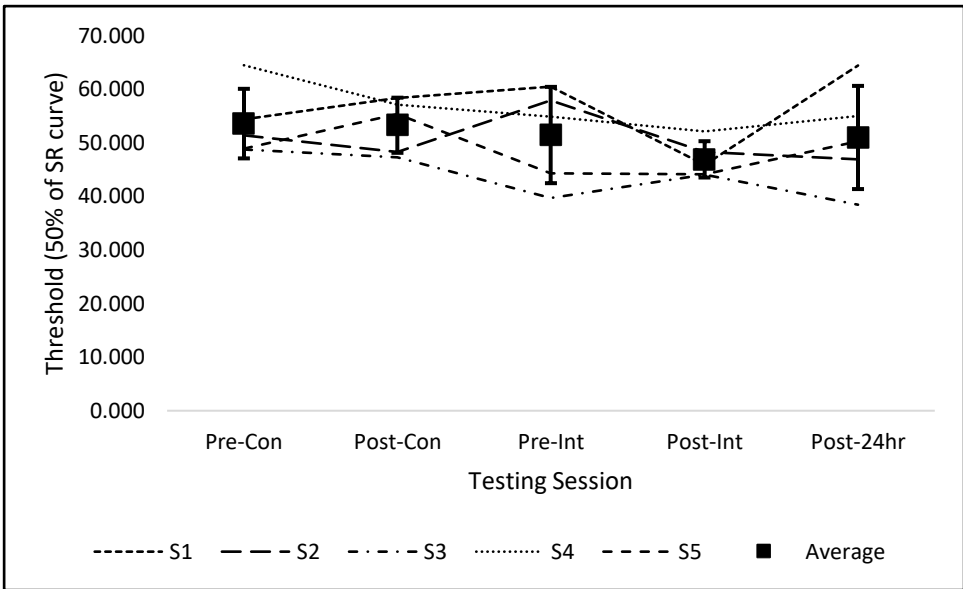


Figure 9

Midpoint Intensity of Peroneus Longus over Time

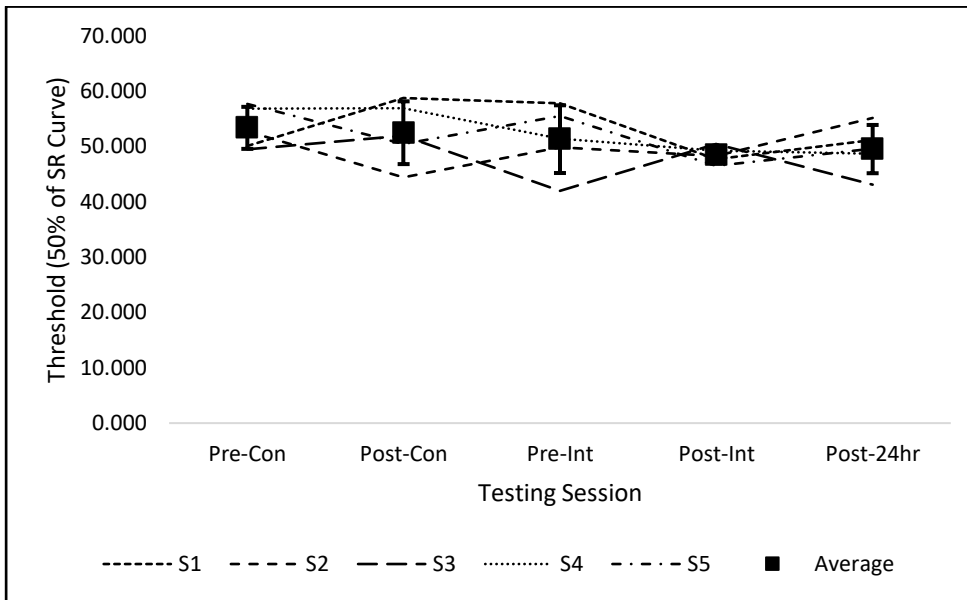


Figure 10

Midpoint Intensity of Soleus over Time

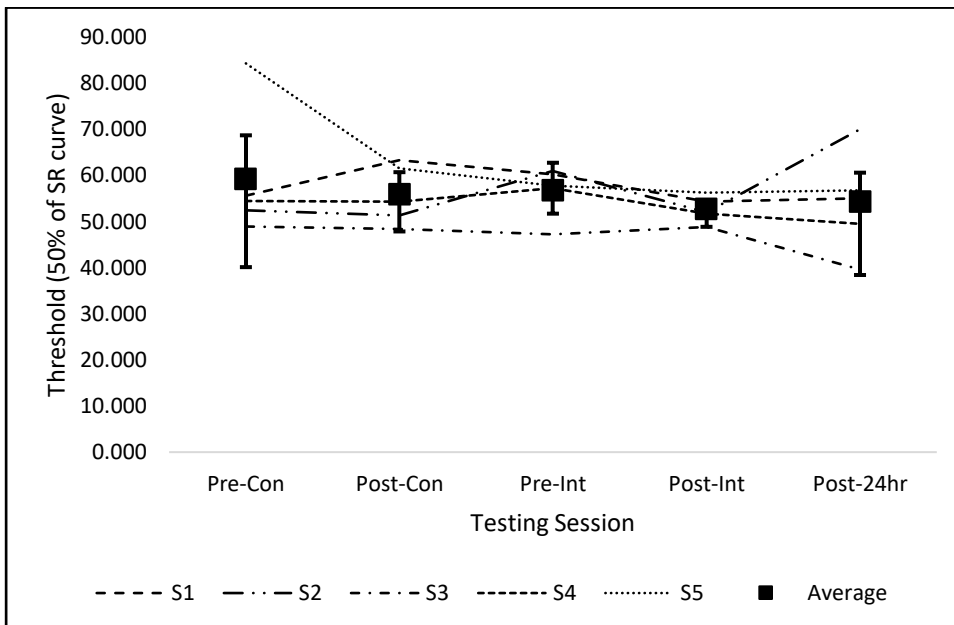


Figure 11

MEPmax Values for Tibialis Anterior over Time

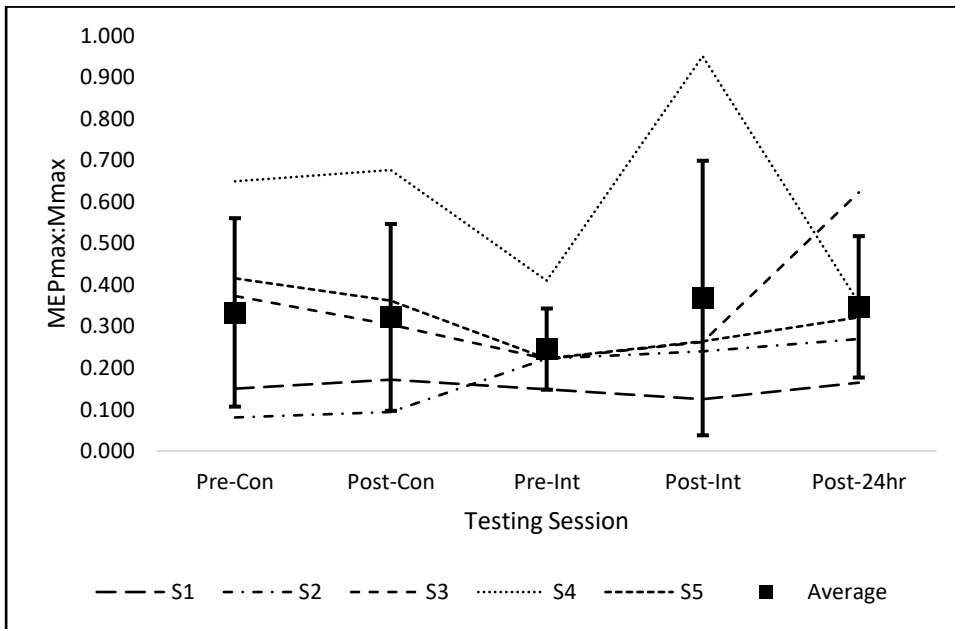


Figure 12

MEPmax Values for Peroneus Longus over Time

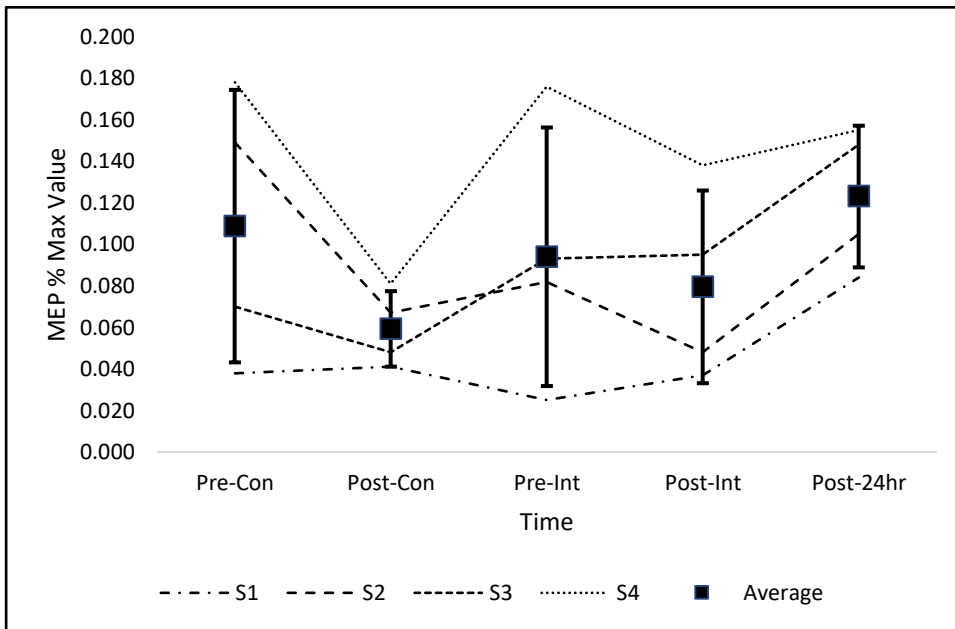
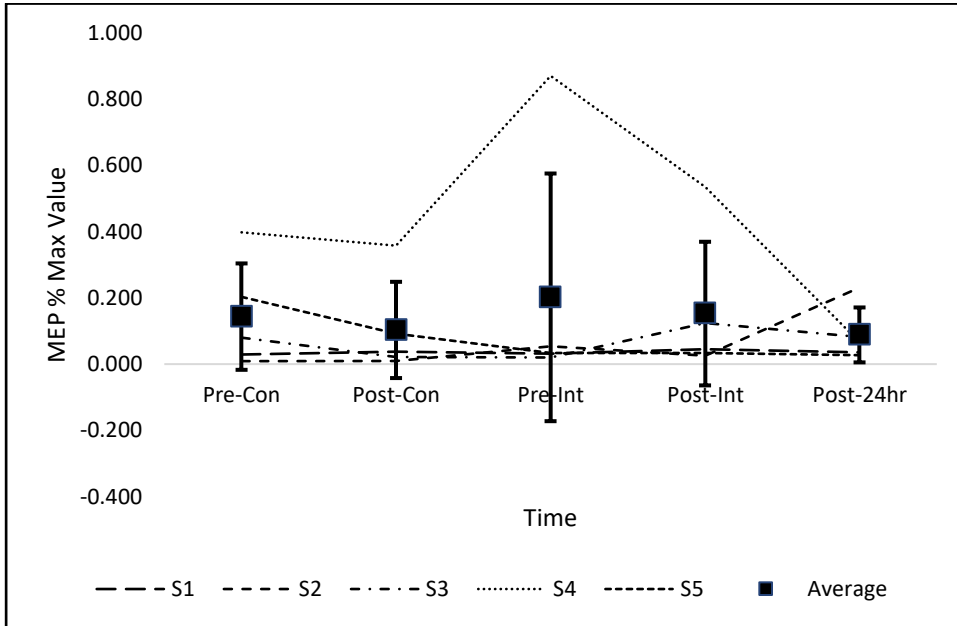


Figure 13

MEPmax Values for Soleus over Time



Vita

Jacob Barton is originally from Swansboro, North Carolina, and is the son of Thomas and Beth Barton. He graduated from Swansboro High School in June 2014. He attended the University of North Carolina at Chapel Hill from 2014 to 2018 and graduated with a Bachelor of Arts in Sport and Exercise Science. Jacob then worked as a Physical Therapy Technician at Axis Physical Therapy in Jacksonville, NC, from August 2018 to April 2020. He then attended Appalachian State University from August 2020 to August 2022 where he earned a Master of Science in Exercise Science. While at Appalachian State University he worked as a research graduate assistant in the Injury Neuromechanics Lab. Jacob is pursuing a career in Human Performance and Injury Prevention research in professional sports.