Differences in Cortical Activation During Anterior Tibial Translation Between Females with High and Low Laxity

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INTRODUCTION: Anterior Cruciate Ligament (ACL) injuries total more than 200,000 incidences annually, with around 70% attributed to noncontact mechanisms during sports. Greater anterior knee laxity (AKL) is a strongly established prospective risk factor for ACL injuries among females. Substantial research has concentrated on the mechanical aspects of greater AKL, whereas relatively little is known about how AKL may affect somatosensory function and the associated brain processing. Therefore, the purpose of this study is to investigate differences in somatosensory cortical activity between individuals with high and low AKL. We hypothesized that females with greater AKL would exhibit less somatosensory cortical activation during passive knee joint loading compared to females with lower AKL.

METHODS: All participants provided written informed consent approved by the institutional review board for performing research involving human participants before participating. Twenty-eight right-handed and right-footed, recreationally active females $(21.7 \pm 3.6 \text{ yrs}, 1.7 \pm 0.06 \text{ m}, 66.7 \pm 10.6 \text{ kg})$ were recruited from the university community and surrounding areas. This was a 2-day study. On day 1, participants were initially screened to form high (>7mm; N = 14) and low (<5mm; N = 14) laxity groups using a KT2000, filled out activity inventories, and were familiarized with a joint loading procedure using a novel knee device. The joint loading consisted of six applications (trials) of an anteriorly directed force of 130N across three joint loading phases (LP): LP1 (0-65N), LP2 (65-130N), LP3 (130N, held for 1 sec) followed by a 10-sec rest phase. On day 2, five blocks of the passive anterior knee joint loading were performed while simultaneously collecting electroencephalography (EEG) data from a 32-channel EEG cap. Each block began in an unloaded condition in which 3 minutes of resting data was collected. Power spectral density (PSD) of the C4 electrode was computed as the average power in the alpha frequency band (8-13 Hz). Cortical activation was operationalized as the inverse of alpha PSD in each load phase after subtracting alpha PSD at rest such that greater decreases in PSD during loading were interpreted as greater cortical activity. Change in alpha PSD within each load phase was then averaged across four trials (trials 2-5) and four blocks (blocks 2-5). T-tests analyzed demographic differences between laxity groups. EEG analyses included a repeated measures ANOVA, which analyzed the somatosensory cortical activation between groups (2 levels (high/low laxity) and across load phase (3 levels (LP 1, LP 2, LP3).

RESULTS SECTION: There were no significant demographic differences other than anterior laxity (Table 1). The ANOVA demonstrated that PSD (alpha power) did not differ between high and low laxity groups during the three joint loading phases. However, a main effect trend (F (2,52) = 2.781, p = .07) indicated a potential reduction in alpha power cortical activation from LP1 to LP3 across all participants.

DISCUSSION: These findings suggest that despite apparent mechanical disparities at the knee, sensory information propagating from the knee joint to the somatosensory cortex during passive joint loading designed to strain the ACL does not differ between females with high and low AKL. Although our original hypotheses were not corroborated in this study, the emerging trend of decreased alpha power from the EEG electrode over the somatosensory cortex from LP1 (initial loading) to LP3 (static loading) prompts further exploration. This trend could potentially yield valuable insights into a putative relationship between knee laxity and information processing in the somatosensory cortex. It is essential to acknowledge that the study is limited to a single electrode (C4) situated over the somatosensory cortex. Therefore, we neglected to measure the activity in the premotor cortices, which are concurrently active during passive joint motion. Additionally, due to the unique nature of joint loading with a novel knee device and instances of leg paresthesia, only 20 trials of EEG data were averaged per participant, which is notably smaller than typical EEG models' 50 to 100 trials.

SIGNIFICANCE/CLINICAL RELEVANCE: The current study represents a preliminary step taken to understand the neural role of the ACL during joint loading with a long-term vision of attempting to develop brain-based interventions in an effort to reduce ACL injuries.

Laxity Group				
N = 28	High	Low	p-value	Effect size (Cohen's d)
Age(year)	21.6±3.2	21.8±3.6	0.45	0.05
Mass(kg)	67.3±12.6	66.0 ± 8.5	0.38	-0.12
Height(cm)	165.8 ± 6.1	165.0±8.5	0.36	-0.14
$BMI(kg/m^2)$	24.3±3.7	24.3±2.9	0.48	022
Marx	8.4 ± 3.8	9.5±3.7	0.21	0.30
Tegner	5.5±1.6	5.1±1.6	0.28	-0.23
Beighton	1.6±1.3	3.1±1.3	0.29	0.21
KT_AKL(mm)	7.8±0.9	4.2±0.7	<.001	-4.51

Table 1. Participants' Demographics, Physical Activity Rating Scale, and Knee Laxity

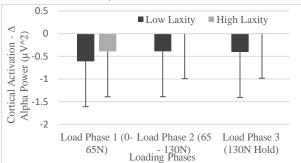


Figure 1. Somatosensory cortical activation (alpha power) at C4 electrode between groups per load phase

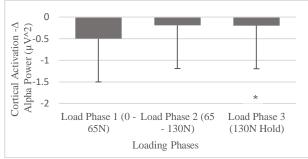


Figure 2. Somatosensory cortical activation (alpha power) per load phase

Trend (p = 0.7). for load phase with Load Phase 3 > Load Phase 1