*SWACSM Abstract*

# Altered Cortical Activation Patterns in the Motor Cortex Post ACL Reconstruction Compared to Healthy Control

RYAN KIM, LANA KAYALI, & ABI RAE STINE

Sports Medicine Lab; HHSC; Loyola Marymount; Westchester, CA

*Category: Undergraduate*

*Advisor / Mentor: An, Yong Woo (yongwoo.an@lmu.edu)*

ABSTRACT

Following anterior cruciate ligament reconstruction (ACLR), patients often experience persistent knee dysfunction, such as knee strength deficit, possibly correlated with altered cortical activation in the brain. However, it remains unclear if ACLR patients exhibit different cortical activation patterns in the primary motor cortex (M1) during strength tasks, when compared to healthy control. **PURPOSE**: To examine electrocortical activation patterns in the M1 during submaximal isometric contraction of the quadriceps between an ACLR patient and a healthy control. **METHODS**: Both ACLR (female, 21yrs,180.34cm, 77.11kg) and control (female, 39yrs, 160.02cm, 61.23kg) performed isometric extension of the reconstructed or matched knees at 10% of their maximal voluntary isometric contraction (MVIC). Electrocortical activation was measured using a 64-channel mobile electroencephalograph (EEG) in response to 30 trials of unanticipated Transcranial Magnetic Stimulation (TMS) over the hotspot, a specific M1 location producing the largest amount of quadriceps activation. TMS were delivered at 120% (AMT 120) and 140% (AMT 140) of the active motor threshold (AMT), the minimum TMS intensity to induce measurable muscle twitch. Averaged event-related potential (ERP) was used to compare presence of positive (P40, P80, P200) and negative (N20, N60, N100) peaks from the onset of TMS between subjects. **RESULTS**: For AMT 120 the ACLR had ERP peaks at N20, P40, and P200 whereas the control had peaks at P80 and N100. For AMT 140 the ACLR had peaks at N20, P40, N60, and P80 whereas the control had peaks at N20, P80 and N100. **CONCLUSION**: Our preliminary data demonstrates that early ERP peaks in ACLR compared to the control may indicate altered neural processing at the M1, with greater differences at AMT120. Such neuroadaptation over the M1 in the ACLR may result in improper regulation of muscle contraction, leading to permanent strength deficit. Future studies are warranted to determine how the altered brain's function affects the knee function in ACLR patients to enhance rehabilitation programs after ACLR.