Motor Cortex Activation and Landing Neuromuscular Control after Anterior Cruciate Ligament Reconstruction


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INTRODUCTION

Anterior cruciate ligament (ACL) injury and reconstruction may induce brain motor control adaptations that influence neuromuscular function and potentially, future injury risk. Traditional neuromuscular control assessment and therapy may not adequately address this neuroplasticity. Functional magnetic resonance imaging (fMRI) provides a detailed method to assess brain changes to bridge this gap in knowledge. The purpose of this work is to determine the relationship between biomechanically measured neuromuscular control during drop landing and brain activation during a knee motor control task in those with ACL reconstruction and matched controls.

METHODS

Participants were matched on height, mass, extremity dominance, education level, history and current physical activity level. Eight left anterior cruciate ligament reconstructed (ACLR) (25.5±1.37 years, 1.70±0.13 m, 75.6±19.2 kg, Tegner activity level 6.0 ± 1.5, 23±18 months post-surgery) and 8 matched healthy controls (23.6±3.14 years, 1.75±0.05 m, 73.5±12.24 kg, Tegner activity level 6.0 ± 1.5) participated. The brain fMRI was collected during a unilateral knee motor task consisting of repeated cycles of extension and flexion while lying supine in the MRI scanner (figure 1). The brain activation pattern for knee movement was completed on a subject-specific level on the contrast between knee movement and rest with a general linear model fixed-effects analysis a priori threshold at p <0.01 corrected (figure 2) with the Oxford Centre software package FSL. The motor cortex was selected as an a priori region of interest and the voxel cluster with the peak activation was extracted for analysis.

A drop vertical jump task was completed to assess functional neuromuscular control. Participants were fitted with a full body point cluster marker set to track 3D kinematics via a Vicon passive optical motion capture system. Participants then completed three drop vertical jumps consisting of leaning forward from a 30.5 cm plyometric box until falling, landing and then immediately jumping up to a target for maximum height. Data were processed using Visual 3D software to assess peak knee flexion during the initial landing from the box.

A Pearson correlation was completed on the relationship between motor cortex activation during knee extension-flexion and peak knee flexion angle during landing.
RESULTS AND DISCUSSION

Motor cortex brain activation in participants with ACL reconstruction was significantly correlated with peak knee flexion during landing (r = -0.717, p = 0.034). There was no correlation for the control group (r = 0.275, p = 0.509) or the combined group (r = -0.211, p = 0.433). There was no difference in peak knee flexion or motor cortex activation between groups.

Table 1: Mean and standard deviations of peak motor cortex activation (percent signal change) and peak knee flexion (degrees) for each group

<table>
<thead>
<tr>
<th>Group</th>
<th>Motor Cortex Activation</th>
<th>Peak Knee Flexion</th>
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<tbody>
<tr>
<td>ACL</td>
<td>1.243 ± 0.245</td>
<td>82.0 ± 14.0</td>
</tr>
<tr>
<td>Control</td>
<td>1.6 ± 0.677</td>
<td>76.15 ± 7.37</td>
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The ACL reconstructed cohort had a strong negative correlation between motor cortex activation and knee flexion during landing. This suggests that, as demand on the motor cortex increases to complete knee movement, the relative amount of knee flexion during landing decreased. Knee flexion was selected as it is a common metric of neuromuscular control during drop landing, with more knee flexion indicating increased ability to control the landing and decreased sagittal plane moments [1]. In contrast, a landing with decreased knee flexion is associated with increased injury risk, poorer neuromuscular control and increased sagittal plane moments [1]. The association of motor cortex activation indicates there is likely a relationship between how the brain generates knee motor control and the neuromuscular control displayed during drop landing after ACL reconstruction.

The lack of a relationship in the control group between motor cortex activation and knee flexion may indicate that the fMRI task does not offer sufficient physical demand for controls. While the relative activation level of the motor cortex between cohorts was similar, previous work has indicated greater motor planning and sensory area activation in those with ACL injury, possibility indicating the task is neurologically more challenging in this cohort [2]. It may be that, in a healthy population, a more challenging motor control task is required to be related to the more functional drop landing.

Additionally, this being a retrospective analysis, it is unknown if this relationship was present prior to the injury and may have played a role in the initial loss of neuromuscular control associated with the ACL injury mechanism or developed due to the injury. Also, the intensive unilateral rehabilitative therapy after injury may induce a greater cortical-functional relationship after ACL injury. The great deal of training to restore muscular function post injury may increase the level of cortical vs. sub-cortical or spinal cord contribution to knee neuromuscular control [3].

CONCLUSIONS

ACL reconstruction may induce specific neuroplastic adaptations in motor control within the brain that translate into neuromuscular control during landing.

REFERENCES


ACKNOWLEDGEMENTS

The authors gratefully acknowledge support for this study from the National Athletic Trainers’ Association and National Strength and Conditioning Association doctoral student grants and The Ohio State University College of Medicine pilot award.