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Neuroplasticity Associated With Anterior Cruciate Ligament Reconstruction

STUDY DESIGN: Controlled laboratory study.

BACKGROUND: Anterior cruciate ligament (ACL) injury may result in neuroplastic changes due to lost mechanoreceptors of the ACL and compensations in neuromuscular control. These alterations are not completely understood. Assessing brain function after ACL injury and anterior cruciate ligament reconstruction (ACLR) with functional magnetic resonance imaging provides a means to address this gap in knowledge.

OBJECTIVE: To compare differences in brain activation during knee flexion/extension in persons who have undergone ACLR and in matched controls.

METHODS: Fifteen participants who had undergone left ACLR (38.13 ± 27.16 months postsurgery) and 15 healthy controls matched on age, sex, height, mass, extremity dominance, education level, sport participation, and physical activity level participated. Functional magnetic resonance imaging data were obtained during a unilateral knee motor task consisting of repeated cycles of knee flexion and extension.

RESULTS: Participants who had undergone ACLR had increased activation in the contralateral motor cortex, lingual gyrus, and ipsilateral secondary somatosensory area and diminished activation in the ipsilateral motor cortex and cerebellum when compared to healthy matched controls.

CONCLUSION: Brain activation for knee flexion/extension motion may be altered following ACLR. The ACLR brain activation profile may indicate a shift toward a visual-motor strategy as opposed to a sensory-motor strategy to engage in knee movement.

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KEY WORDS: *brain, knee, motor control, neuromuscular control, neuroscience, rehabilitation, sports injury*

Individuals who experience a primary anterior cruciate ligament (ACL) injury are at substantial increased risk of experiencing a second ACL injury, despite surgical reconstruction and rehabilitation.[64] Athletes who attempt to return to activity are at an exceptionally high risk of reinjury (30 to 40 times greater relative to those without injury history).[92] The mechanisms for the heightened injury risk may extend beyond the physiological and biomechanical changes of the reconstructed knee joint and may involve a systematic neurological response to the injury.[34,42,69,90] Neuroplasticity following ACL injury is likely due to a combination of altered sensory feedback from the injury, as well as behavioral motor control compensations. The lost ligament mechanoreceptors and associated physiologic cascade of inflammation and joint effusion may alter input to the central nervous system (CNS).[39,62,77,95] Simultaneously, experience-dependent factors, including behavioral changes due to injury-associated pain, instability, compensatory movement patterns, and physical rehabilitation, also can cause unique and interacting neuroplastic changes, along with the afferent disruption.[44,60,61,68,91]

The altered afferent input into the CNS due to the lost mechanoreceptors of the native ACL[77,95] decreases innervation to the primary sensory cortex.[87] This may lead to absent somatosensory-evoked potentials in those with ACL injury and anterior cruciate ligament reconstruction (ACLR).[70,88] The efferent output also is altered, with disrupted gamma-motor neuron feedback loops,[41] delayed long latency reflexes,[51] and altered spinal and cortical excitability.[29,67] Depressed cortical excitability after ACLR increases the stimulus required at the motor cortex to generate quadriceps contractions.[43,44,67] The increased need for input to the motor cortex may contribute to the increased frontal and parietal cortex activation for knee joint position and force control after ACLR.[5,6] As the frontal and parietal brain regions have a high degree of connectivity to the motor cortex and provide sensory and cognitive contributions for motor planning, increased activation of these regions may be able to provide a compensatory mechanism to increase stimulus to the motor cortex.[5,6,28,83,93] This combined disrupted input and limitation of the efferent signal contributes to arthrogenic muscle inhibition and motor control

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changes, leading to sensory-motor nervous system compensations.[11-13,17,27,57,86]

Previous investigators have hypothesized that following ACL injury, the CNS may increase reliance on alternative sensory sources, such as visual-feedback and spatial awareness.[1,17,20,25,59] One previous investigation used neuroimaging to quantify brain activation differences between persons with ACL deficiency who did not return to previous levels of physical activity and a healthy control group.[35] Those with ACL deficiency had increased activation in the posterior inferior temporal gyrus (visual processing), presupplementary motor area (motor planning), and secondary somatosensory area (pain and sensory processing).[35] While this initial work supports the conceptual framework of neuroplastic changes after ACL injury and the possibility of altered sensory-visual-motor brain activation, it is unknown how these changes may present after reconstruction, rehabilitation, and return to activity. As biomechanical deficits remain years after completion of rehabilitation and return to activity,[57,74] understanding how the brain is generating knee motion may help us to understand why motor deficits persist.

The purpose of this study was to compare brain activation during knee flexion/extension between persons who have undergone ACLR and a matched control group. Based on previous literature, it was hypothesized that those with ACLR would have brain activation differences related to motor planning (premotor and motor cortex) and sensory function (cross-modal sensory-visual regions and secondary somatosensory area) relative to the matched controls.

METHODS

Participants

Subjects were recruited from the local University and orthopaedic clinics. Prospective participants were directed to an online survey to determine whether they met the following inclusion criteria: magnetic resonance imaging [MRI] compliance, minimum score of 5 on the Tegner[9] scale, and participating in running and cutting/change-of-direction activity on the Marx[53] scale at least once a week. Subjects also were screened to include only left-side ACLRs. This was done to allow aggregation of the brain activation data without concern for unique unilateral brain changes that might have been missed if the cohort had mixed left and right ACLRs. Individuals with a history of other lower extremity injuries were excluded. In addition, control participants had no history of lower extremity injury.

All ACLR participants were 6 months to 5 years postsurgery, cleared for full return to activity by their physician, and engaged in regular physical activity. Participants in the control group were matched to those in the ACLR group by age, sex, height, mass, leg and arm dominance, education level, and physical activity history and current level, including specific sport participation by level and years of participation.

Individuals with ACLR (n = 131) were screened, and 15 fit the inclusion criteria and agreed to participate in the study. Potential control participants (n = 371) were screened, and 15 fit the inclusion criteria and provided a viable match to a participant with ACLR. The MRI restrictions (limited metal implants, no metal dental work) and the strict matching criteria greatly reduced participant inclusion. Participant demographics are described in **TABLE 1**. Participants with ACLR engaged in a standardized rehabilitation program, with 13 of the 15 coming from the same local physical therapy network. All participants with ACLR reported engaging in extensive unilateral strengthening and range-of-motion exercises, progressing to agility and plyometric training before return to activity. All participants completed the International Knee Documentation Committee Subjective Knee Evaluation Form (IKDC)[30] to assess subjective knee function, as well as a short rehabilitation survey.

This study was approved by The Ohio State University Institutional Review Board, and informed consent was obtained prior to study enrollment. An a priori power analysis was completed using our preliminary data (fmripower.org),[56] allowing for estimation of power with the same scanning parameters and experimental protocol as the current investigation. Using the sensorimotor cortex as a primary region of interest, we had 80% power with a .05 type I error rate and 13 participants per group.

Data Collection

Functional magnetic resonance imaging (fMRI) data were collected on a 3.0-T MAGNETOM (Siemens AG, Munich, Germany) scanner using a 12-channel array, receiver-only head coil. The fMRI session consisted of 90 whole-brain, gradient-echo, echo-planar scans, acquired every 3.0 seconds with an anterior/posterior phase encoding direction (slice thickness, 2.5 mm; 55 transversal slices). This equated to 10 whole-brain data sets per knee movement block, or 40 whole-brain activation maps for knee movement, contrasted with 50 whole-brain maps for rest. After the fMRI scanning, an anatomical 3-D, high-resolution, T1-weighted image (repetition time, 2000 milliseconds; echo time, 4.58 milliseconds; field of view, 256-mm matrix; slice thickness, 1 mm; 176 slices) was completed for registering the activation data and for brain region identification and normalization to compare the ACLR and matched participants.

Each participant was positioned supine in the scanner, with the legs placed on a custom cushion that limited knee flexion to 45°. Each participant then performed cyclic, non–weight-bearing knee extension/flexion from 45° of flexion to terminal extension of the involved or matched control knee, in this case all on the left side. The movement was triggered by a 2-second visual prompt and paced with an auditory metronome that provided a cue to flex or extend the knee at 1.2 Hz or 36 knee extension/flexion cycles per 30-second stimulus (**FIGURE 1**).[36,37] The knee movement session included 4

TABLE 1 Participant Demographics, IKDC Instrument, and Head Motion Parameters During the fMRI Task*

	ACLR	Control	*P* Value†
Sex, n			...
Male	7	7	
Female	8	8	
Age, y	21.7 ± 2.7	23.2 ± 3.5	.13
Height, m	1.7 ± 0.1	1.7 ± 0.1	.49
Weight, kg	70.4 ± 15.8	69.7 ± 14.3	.92
Tegner activity level scale	7.2 ± 1.3	6.8 ± 1.5	.27
Marx activity rating scale	12.5 ± 3.9	11.2 ± 2.9	.31
Limb dominance			...
Right	13	13	
Left	2	2	
Time from surgery, mo	38.1 ± 27.2	...	...
Graft type, n		...	...
Hamstring graft	13		
Patellar tendon graft	2		
IKDC score	88 ± 8.1	98 ± 2.1	<.001
Absolute head motion, mm	0.57 ± 0.35	0.52 ± 0.23	.63
Relative head motion, mm	0.15 ± 0.08	0.14 ± 0.05	.62

Abbreviations: ACLR, anterior cruciate ligament reconstruction; fMRI, functional magnetic resonance imaging; IKDC, International Knee Documentation Committee Subjective Knee Evaluation Form.
**Values are mean ± SD unless otherwise indicated.*
†Reported from t tests comparing group means.

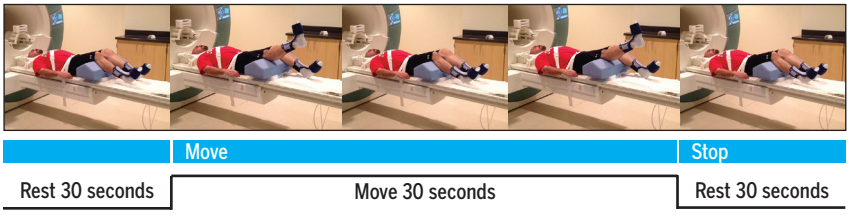

FIGURE 1. Experimental setup. The stimuli for the flexion/extension movements were cued with a visual prompt and paced with a metronome.

blocks of 30-second knee movements interspersed with 5 blocks of a 30-second rest. This movement paradigm was selected because it is a validated technique to assess brain activation for knee movement,[36,37] with limited head motion artifact and participant discomfort.[36,37]

Knee flexion/extension is a critical component of daily physical function, and fMRI is limited by any accessory head motion; therefore, completing a more dynamic weight-bearing lower extremity task is exceedingly difficult and has high risk to generate excessive activation artifact. Head movement artifact was limited with padding and straps to at most 1 mm absolute and 0.30 mm relative displacement across all participants. There was no between-group difference in head motion ($P>.05$). An ankle-toe splint was used to restrict ankle and toe movement, and the participant was monitored for accessory motions. The thigh, pelvis, and torso were secured to the table with straps, and the head was surrounded by molded padding and sandbags to limit head translation. Practice of the movement was completed in a full-mock scanner session prior to completing the actual fMRI session to ensure the participant could complete the movement smoothly with minimal head motion.

Data Analysis

The fMRI technique used in this study quantified the blood oxygen level–dependent signal via the hemodynamic response (blood flow) to various stimuli or tasks.[21] Functional MRI collection and analysis has been validated against actual neural recordings.[23,46] The reliability of fMRI quantification of the neural activation associated with knee movement has been determined to be high.[55,58] The fMRI image analyses and statistical analyses were performed using the Oxford Centre for Functional MRI of the Brain Software Library.[82] Image analysis began with standard prestatistic processing applied to individual data, which included nonbrain removal, spatial smoothing at 6 mm, and standard motion correction and realignment parameters (3 rotations and 3 translations) as covariates to limit confounding effects of head movement.[32] High-pass temporal filtering at 90 Hz and time-series statistical analyses were carried out using a linear model with local autocorrelation correction. Functional images were coregistered with the respective high-resolution T1 image and the standard Montreal Neurological Institute template 152 using linear image registration. This registration process allowed data from each participant to be spatially aligned on a standardized brain template for comparison.

To our knowledge, the present study is the first to perform a whole-brain analysis to examine knee motor control after ACLR. The only previous study to use neuroimaging in a similar population examined low-functioning, ACL-deficient patients.[11] It is likely that the brain activation pattern differences in the present sample might not exactly match those of the previous work, considering the highly selective matching, higher physical activ-

ity level, and the reconstruction status of our sample and differences in analysis method (Oxford Centre for Functional MRI of the Brain Software Library versus Statistical Parametric Mapping by the Wellcome Trust Centre for Neuroimaging) and scanning parameters (3-T Siemens scanner versus 1.5-T Philips scanner).

The subject-level analysis of knee movement relative to rest was completed using a z score greater than 4.6 and a (corrected) cluster significance threshold of $P = .001$. The cluster correction for multiple comparisons uses a variant of the Gaussian random field theory to decrease type I error in statistical parametric mapping of imaging data by evaluating the activation not only at each voxel, but also at the surrounding voxel cluster (as it is unlikely that the voxel tested and surrounding voxels are active above the threshold due to chance).[71]

The paired contrast between the participants with ACLR and matched controls was performed with group z statistic images set at a threshold of z scores of greater than 3.5 and a corrected cluster significance level of $P = .01$. The higher threshold and lower P value for both the participant- and group-level analyses were selected to mitigate interparticipant variability, decrease probability of motion artifact in the data, as well as further decrease the probability of type I error and multiple-comparison error beyond traditional measures.[71]

RESULTS

The ACLR group had a significantly lower IKDC score (88 ± 8.1) compared to the control group (98 ± 2.1; $P<.001$). The brain area activation is reported as contralateral (indicating activation on the opposite side of motion, or the right side, as the movement was always completed with the left knee) or ipsilateral (being the same side of motion, or the left side). The results are presented as z score (activation level relative to the contrast of ACLR versus control participants) and percent signal change for each group from baseline to knee movement in **TABLE 2**. The ACLR group demonstrated increased activation of the contralateral primary motor cortex, ipsilateral lingual gyrus, and secondary somatosensory cortex, and diminished activation of the ipsilateral motor cortex and vermis of the cerebellum area, compared to the matched control group (**FIGURE 2**, **TABLE 2**).

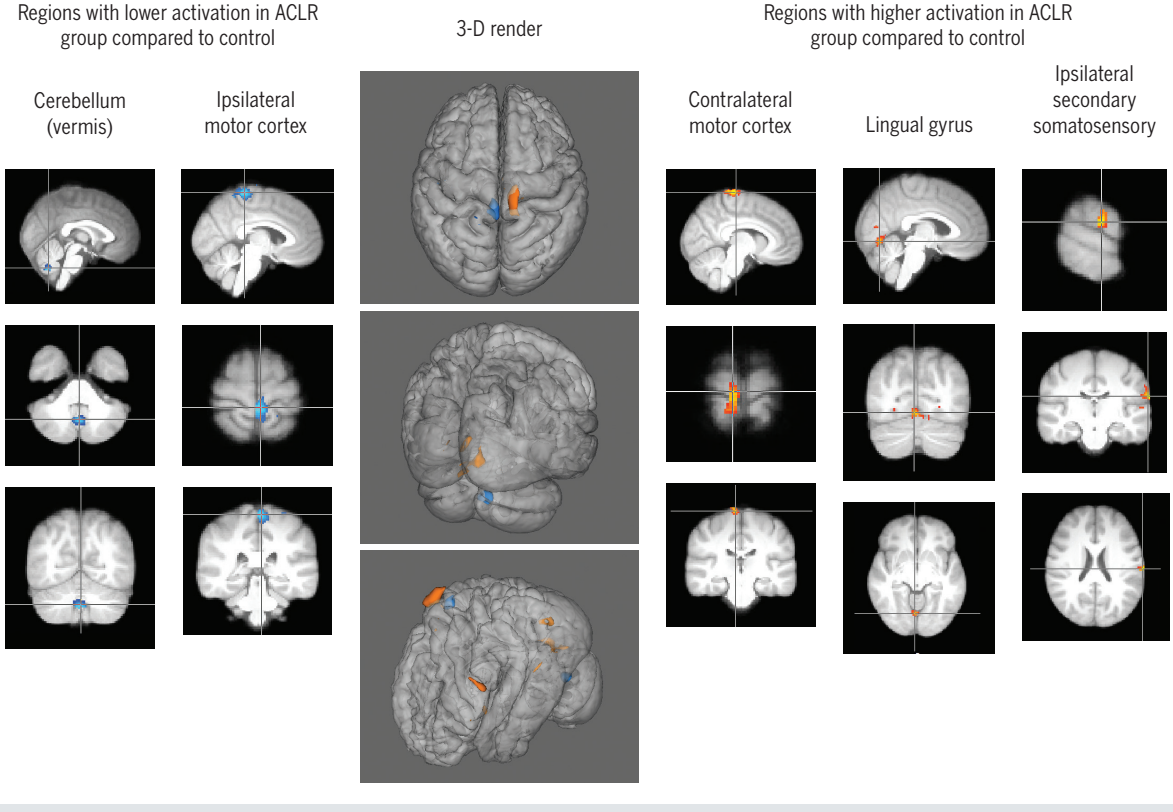

FIGURE 2. Cross-sections of each area that demonstrated higher or lower activation in the ACLR cohort relative to controls. Blue indicates lower activation in the ACLR cohort and orange indicates higher activation. The center 3-D rendering is a top-down view (top), posterior view (middle), and side view (bottom) of a partially transparent 3-D rendering of the brain activations on a standard brain template. The regions are labeled contralateral if the peak voxel of the cluster is in the hemisphere contralateral to the left leg movement (right hemisphere), and ipsilateral if the peak voxel of the cluster is in the hemisphere ipsilateral to the left leg movement (left hemisphere). Abbreviation: ACLR, anterior cruciate ligament reconstruction.

DISCUSSION

The purpose of this study was to quantify the brain activation changes associated with ACLR during a knee flexion/extension task. Utilizing neuroimaging, the results of this investigation suggest that after ACLR, specific brain regions responsible for sensory, motor, and sensory-visual-spatial processing may have altered activation. The primary motor cortex exhibited greater activation during involved-limb knee extension/flexion in the ACLR group, which may be due to the increased need for cortical drive to engage the quadriceps after injury and reconstruction.[4,45] The increased primary motor cortex activation corroborates previous research indicating that motor cortex excitability is diminished after ACLR.[45,67]

The finding of depressed motor cortex excitability suggests that greater motor cortex activation is required to achieve motor drive and/or that motor cortex input from the rest of the brain in the form of structural or functional connectivity must increase to achieve motor drive.[47,89] Following ACL injury and subsequent reconstruction, which disrupts sensory input, and the development of altered motor control strategies to compensate for the associated biomechanical insufficiencies (strength, range of motion), the processing demands on the motor cortex may increase to maintain even simple motor control integrity.[10,14] Of note, we utilized the peak voxel of an activation cluster to determine the brain region; therefore, the motor cortex cluster may include the supplementary motor area anteriorly and the primary sensory cortex posteriorly at the edges of the activation threshold. However, these appeared to not be distinct activation increases relative to the primary motor area and may implicate increased premotor as well as motor cortex activation in the ACLR group.

We also observed that the secondary somatosensory area was activated to a greater degree in the ACLR participants, similar to the findings of Kapreli et al[35] in ACL-deficient individuals. This area is responsible for somatosensory processing, with the anterior region integrating sensory stimuli and the posterior region addressing painful stimuli.[19,85] Our participants were beyond the acute stage of injury and did not report any discomfort during the fMRI, but did have a significantly decreased IKDC score, indicating a level of subjective knee dysfunction. The increased activation in the secondary somatosensory area may represent a functional cortical sensory processing reorganization secondary to the knee trauma and/or treatment increasing nociceptive-related processing during any related involved-knee movement. Interestingly, the secondary sensory area activation was on the ipsilateral side of movement, which may indicate a bilateral neuroplastic effect of the injury to induce adaptations in sensory processing.

The contralateral side of the brain controls the ipsilateral leg, but the secondary somatosensory area functions bilaterally, with little lateralization in activation due to unilateral stimuli.[85] Thus, the increased ipsilateral secondary somatosensory activation after injury, reconstruction, and rehabilitation may represent functional reorganization of sensory processing in both hemispheres. Adapted sensory processing also has been demonstrated with increased parietal lobe activation to reproduce joint positions in those with ACLR.[6] The disruption to sensory processing after ACLR is further supported by the absent or depressed somatosensory-evoked potentials after injury that are not restored with reconstruction.[88] It is possible that, due to the pain and lost input to the primary sensory cortex associated with the injury, sensory processing for movement becomes altered with increased activation of higher-level sensory integration areas.

The ACLR group also exhibited in-

TABLE 2 Brain Region Activation Data for Areas With Statistically Significant Higher or Lower Activation in the ACLR Cohort Relative to Controls During Involved-Knee Movement*

	Higher Activation			Lower Activation	
Measure/Group	Contralateral Motor Cortex	Lingual Gyrus	Ipsilateral Secondary Somatosensory	Cerebellum (Vermis)	Ipsilateral Motor Cortex
Mean region signal change, %					
ACLR	2.18	0.67	0.71	−0.26	0.47
Control	1.59	0.28	0.29	0.10	0.91
Peak signal change, %					
ACLR	4.35	1.88	1.57	−0.07	1.93
Control	3.02	1.11	0.83	0.38	2.54
Signal change SD, %					
ACLR	0.70	0.39	0.22	0.11	0.51
Control	0.55	0.29	0.20	0.13	0.62
Mean *z* value	5.15	4.58	5.60	5.77	5.18
MNI coordinates (peak voxel)					
x	8	4	−68	2	−2
y	−30	−72	−20	−62	−34
z	76	−10	22	−42	68

Abbreviations: ACLR, anterior cruciate ligament reconstruction; MNI, Montreal Neurological Institute.
**All voxels are cluster corrected (P<.01).*

creased activation of the lingual gyrus, a brain region involved in the cross-modal processing of congruent visual and sensory feedback[31,49,75] for limb positioning,[3,15] sensory-visual spatial navigation,[18,50] attention,[52] memory,[7] and movement perception.[15,18,80] Recent data suggest that the higher-order visual cortex may have a motor-oriented organization due to the connections with the sensorimotor cortex that use feedback from visual regions to control motor output.[8,22,81] Activation and connectivity of the lingual gyrus region also have been shown to adapt with altered sensory input and motor demands.[40,48,49,54,75] Therefore, the increased lingual gyrus activation after ACLR may be due to the adapted sensory feedback of the lost ACL mechanoreceptors and the continued motor demands (all individuals were returned to activity).[6,48,88,95] In addition to the altered afferent input influencing sensory-visual brain activation, the targeted rehabilitation to increase quadriceps activation immediately after surgery[24] may increase conscious awareness of the injured joint, creating a visual-motor link during recovery.[17,59] Thus, the participants with higher lingual gyrus activation may be engaging in a visually biased strategy to engage knee movement.[4,49,76,81]

The decreased ipsilateral motor cortex activation during knee motion may be the result of the extensive unilateral rehabilitation that is provided after surgical reconstruction. Upon enrolling in the study, participants completed a short survey regarding their rehabilitation, and all indicated extensive unilateral exercises throughout rehabilitation. Motor control of the lower extremity tends to engage bilaterally, whereby the ipsilateral motor cortex activates to inhibit contralateral or bimanual contraction during unilateral movement (as completed in this study).[83] A recent study indicated that only 45 minutes of balance training can change the structural morphology and functional activity of the motor cortex, thus the months of focused rehabilitation likely contributed to the activation differences.[84] Thus, it is possible that the ipsilateral motor areas became more efficient and required less neural activation to execute unilateral movement due to the therapy targeting the involved knee.[16] Alternately, cortical motor control may become less bilateral due to inhibition and compensations after injury to increase reliance on the contralateral knee.[2,57] While unilateral rehabilitation is advised to address the significant asymmetries in strength and function after ACLR, the bilateral neurological effects are well documented, ranging from gamma-motor neuron dysfunction[42] to cortical excitability.[45,67]

The depressed ipsilateral motor cortex activation as observed in our study adds to the contralateral adaptations after this unilateral trauma. Additionally, the decreased cerebellum activation may be a consequence of increased contralateral motor cortex activation increasing cortical descending control after injury.[72,94] These decreases in brain activation may influence the depressed postural control[17,59,65] and altered neuromuscular control of the contralateral limb after unilateral ACLR.[64,65,92] This finding provides further evidence that the "healthy" knee may not serve as a sufficient comparison to gauge functional status, as brain activation changes that influence bilateral lower extremity function may have occurred.

When compared to the only other neuroimaging study after ACL injury,[35] the present study found fewer regions that had increased activation in the control participants relative to those with ACLR. Specifically, only the ipsilateral motor cortex and cerebellum were found to activate more in the controls. However, Kapreli et al[35] noted that several regions, including the cerebellum, basal ganglia, cingulate motor area, parietal cortex, thalamus, and both sensorimotor cortices, had increased activation in their control group relative to their ACL-deficient group. This is likely due to the similarity in our ACLR cohort and matched controls. Kapreli et al[35] enrolled ACL-deficient, less active, and poorer functioning individuals and matched them with healthy controls, which could have resulted in a greater difference in brain activation compared to the more active ACLR cohort.[2] In an attempt to further isolate the effects of ACL injury and reconstruction, we matched participants by sex, limb dominance, activity level, motor skill specialization, mass, height, sport participation (by sport, level, and years of participation), education level, and age, and completed a paired analysis with a decreased alpha value. Kapreli et al[35] only matched on sex, limb dominance, activity level, and motor skill specialization.

Clinical Implications

As traditional rehabilitation encourages a focus of attention on the knee with increased visual and cognitive knee position control during movement training, it is likely that the brain activation differences are in part due to the rehabilitation process. Previous reports of altered neuromuscular control following ACL injury, as that reported in the present study, suggest that there are acute injury effects as well as chronic long-term neuroplastic changes associated with rehabilitation and motor adaptations.[2,43,57,79] To influence the sensory-visual-related brain activation, clinicians may consider incorporating varied visual conditions via blindfold, external targets, stroboscopic glasses, or dual tasking during rehabilitation. Forcing the focus of attention to the external environment with instruction or feedback, as opposed to the typical internal feedback of focusing on knee position or the quadriceps, in rehabilitation[33] may assist in inducing an adaptive neuroplastic response and improve patient function. Alternatively, a direct approach to reducing visual feedback (blindfold, stroboscopic glasses, virtual reality) during rehabilitation may be beneficial to encourage increased utilization of proprioceptive sensory input, as opposed to increasing the reliance on a visual-spatial neural strategy.[25] To influence motor cortex activation, clinicians may consider motor-learning approach-

es, such as various forms of augmented feedback, to facilitate advancement in expertise during movement training.[63,73,78] Previous investigators have reported that using biofeedback and skill training can directly influence motor cortex excitability and activation.[38,66,73]

Limitations

It is possible that the brain activation differences observed in the current study were not due to the injury but were prospective in nature and present in those who went on to experience ACL injury. It is unlikely that this would account for all of the activation differences observed, as activation of the inferior temporal region or lingual gyrus has only been documented in ACL-injured individuals during a knee motor task[26,35] and not reported in the study of healthy individuals engaging in the same task.[36,37] The relative activation of this area indicates that the injury or rehabilitation process could have induced at least some of the neurological differences, and it is unlikely that they are entirely predisposing phenomena.

Neuroimaging is prone to variability and spurious findings. To limit potential error, we included additional controls in the analysis to decrease this variability and utilized conservative corrections and thresholding.[71] Additionally, each participant's activation pattern was contrasted with that of a control participant matched on many of the factors that generate this variability, including age, sex, height, mass, activity level history, current activity level, education level, hand and leg dominance, and previous and current sport participation. While our sample size was relatively small, we completed an a priori power analysis to ensure sufficient participants based on previous neuroimaging reports and the noise associated with our scanner, specifically via pilot data analysis. Also, while fMRI is a powerful modality for assessing brain function, it is unable to quantify the neural activation of complex, multijoint, or dynamic lower extremity tasks due to any task-correlated head motion generating excessive data artifact. The simple motor task utilized in our study required extensive measures to mitigate head movement, including participant task practice, a mock scanner session, head foam, bracing, strapping, and other restraints to ensure head stabilization during movement.

Future Directions

A longitudinal design with control for the rehabilitation and surgical intervention will allow determination of within-participant neuroplasticity due to injury, surgery, and rehabilitation. The next steps in quantifying musculoskeletal injury–induced neuroplasticity will require more advanced motor control tasks, such as force or position matching and multijoint movements, to improve the clinical applicability of these results. The integration of transcranial magnetic stimulation and/or electroencephalography with fMRI also presents an opportunity to quantify brain function with superior spatial and temporal resolution to further capture aspects of motor control that may be playing a role in the ACL injury risk profile. As more investigators begin to explore the neurological changes associated with motor control after musculoskeletal injury, novel rehabilitation approaches that maximize both nervous system and musculoskeletal system adaptations will be developed.

CONCLUSION

The current study found brain activation differences between individuals with a history of ACLR and matched healthy controls during a knee flexion/extension task. After ACL injury, reconstruction, rehabilitation, and return to activity, knee motion requires increased activation of motor, visual, and secondary sensory areas in the brain. These brain activation differences indicate a possible neuroplastic effect of musculoskeletal trauma that is not normalized after treatment or return to activity. Clinicians may consider embracing the principles of neuroplasticity in musculoskeletal rehabilitation, including motor-learning and visual-motor compensations, to address the brain activation differences after injury. ◉

KEY POINTS

FINDINGS: Anterior cruciate ligament injury, surgery, and rehabilitation may cause specific brain activation changes related to sensory-visual-motor control.

IMPLICATIONS: Following ACLR and return to activity, an altered sensory-visual knee neural control strategy remains that may have the capability to be targeted with novel rehabilitation strategies.

CAUTION: The current study design is unable to determine when the neuroplastic changes occurred after injury or whether any of the brain activation differences were present prior to injury.

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