Greater Muscle Co-Contraction Results in Increased Tibiofemoral Compressive Forces in Females Who Have Undergone Anterior Cruciate Ligament Reconstruction

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ABSTRACT: Individuals who have undergone ACL reconstruction (ACLR) have been shown to have a higher risk of developing knee osteoarthritis (OA). The elevated risk of knee OA may be associated with increased tibiofemoral compressive forces. The primary purpose of this study was to examine whether females with ACLR demonstrate greater tibiofemoral compressive forces, as well as greater muscle co-contraction and decreased knee flexion during a single-leg drop-land task when compared to healthy females. Ten females with ACLR and 10 healthy females (control group) participated. Each participant underwent two data collection sessions: (1) MRI assessment and (2) biomechanical analysis (EMG, kinematics, and kinetics) during a single-leg drop-land task. Joint kinematics, EMG, and MRI-measured muscle volumes and patella tendon orientation were used as input variables into a MRI-based EMG-driven knee model to quantify the peak tibiofemoral compressive forces during landing. Peak tibiofemoral compressive forces were significantly higher in the ACLR group when compared to the control group (97.3 ± 8.0 vs. 88.8 ± 9.8 N $\cdot$ kg^{-1}). The ACLR group also demonstrated significantly greater muscle co-contraction as well as less knee flexion than the control group. Our findings support the premise that individuals with ACLR demonstrate increased tibiofemoral compression as well as greater muscle co-contraction and decreased knee flexion during a drop-land task. Future studies are needed to examine whether correcting abnormal neuromuscular strategies and reducing tibiofemoral compressive forces following ACLR can slow the progression of joint degeneration in this population. © 2012 Orthopaedic Research Society. Published by Wiley Periodicals, Inc. J Orthop Res 30:2007–2014, 2012

Keywords: osteoarthritis; EMG-driven model; MRI; muscle volume; patella tendon orientation

Tears of the anterior cruciate ligament (ACL) are one of the most common sports related knee injuries. Female athletes sustain ACL injuries 2–8 times more often than male athletes.[1] Most ACL injuries require surgical intervention with approximately 175,000 ACL reconstruction (ACLR) surgeries performed annually.[2] Despite substantial improvements in surgical techniques and satisfactory outcomes in terms of restoring functional stability to the knee, individuals with ACLR have been shown to have a higher risk of developing early osteoarthritis (OA) at the knee.[3,4] The underlying mechanism(s) for the higher risk of knee OA in individuals following ACLR remain unclear. However, a potential explanation for the elevated risk of knee OA in this population may be associated with the abnormally high joint compressive force (defined as the force perpendicular to the joint/articular surface) resulting from altered neuromuscular strategies.

Several studies have reported that persons who have undergone ACLR exhibit increased hamstring activation and co-contraction of the quadriceps and hamstring muscles during various functional activities, such as walking, running, and landing from a jump.[5–7] While this muscle recruitment strategy in persons post-ACLR is hypothesized to increase joint stability and decrease the anterior shear loads at the knee by providing a posterior shear force from the hamstring muscles,[6,7] the strategy of increased muscle co-contraction has been shown to increase the tibiofemoral compressive force in a simulated ACL-deficient knee model.[8] As such, it is plausible that the increased muscle co-contraction utilized by individuals who have undergone ACLR also may contribute to increased compressive force of the tibiofemoral joint. Another common finding in this population is reduced knee flexion.[9,10] Deceased knee flexion may also result in greater tibiofemoral compressive forces as shock attenuation during weight acceptance may be impaired.[11]

While compressive forces are essential in regulating cellular metabolism and maintaining normal cartilage matrix,[12] elevated compressive forces may have a detrimental effect on articular cartilage. In vitro studies have shown that excessive and repetitive mechanical loading results in micro damage of the cartilage (e.g., surface fissures) and leads to osteoarthritic changes.[13] In addition, excessive compressive forces may be harmful to chondrocyte viability and may damage the extracellular collagen–proteoglycans matrix of the articular cartilage.[13–15] To gain a better understanding of a potential mechanism related to the elevated risk of knee OA development in persons who have undergone ACLR, a comprehensive assessment of knee joint loading in this population is warranted.

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The primary purpose of this study was to examine tibiofemoral compressive and shear forces as well as muscle co-contraction and knee flexion during a single-leg drop-land task between females who have undergone ACLR and healthy female controls. Tibiofemoral compressive and shear forces were quantified using a magnetic resonance imaging (MRI) based, subject-specific electromyography (EMG) driven model. We hypothesized that females post-ACLR would demonstrate similar peak anterior shear force but elevated peak tibiofemoral compressive forces during a drop-landing maneuver when compared to the females who have not undergone ACLR. We also hypothesized that females post-ACLR would demonstrate increased muscle co-contraction and decreased knee flexion during landing. Results from this study will contribute to a better understanding of the biomechanical factors that may contribute to the early onset of knee OA in the ACLR population.

METHODS

Subjects

Two groups of females between the ages of 18 and 35 participated. Ten females who have previously undergone ACLR constituted the experimental group while 10 healthy females without a history of ACLR served as the control group (Table 1). The ACLR group consisted of recreational female athletes who had primary unilateral ACL reconstruction (three right and seven left) using either an allograft or bone-patella-tendon-bone autograft and had returned to unrestricted sport activities for at least 6 months. Subjects in the ACLR group must have been at least 1 year post-surgery and no longer than 5 years post-surgery. The control group consisted of females who had no history of ACL injury and knee surgery, and were matched to those in the ACLR group based on age, limb dominance, and activity level (evaluated using Global Physical Activity Questionnaire).[16]

Subjects in either group were excluded from participation if they reported any of the following: (1) current lower extremity injury that resulted in any persistent pain and discomfort at the time of participation; (2) implanted biological devices (e.g., pacemakers, cochlear implants, clips) contraindicated for MRI measurements; (3) history of claustrophobia or severe anxiety; (4) pregnancy; and (5) any medical conditions that would impair the subject to perform the tasks described below. Furthermore, subjects in the ACLR group were also excluded from participation if they reported concomitant ligament injuries. Prior to participation, all procedures were explained to each subject and informed consent was obtained as approved by the Institutional Review Board of the University of Southern California Health Sciences Campus.

Procedures

Subjects participated in two data collection sessions: (1) MRI assessment and (2) biomechanical testing during a single-leg drop-land task. Data were obtained from the reconstructed limbs of the 10 ACLR subjects and the matched limbs of the control subjects. Data collected from these two testing sessions were used as input variables for the EMG-driven knee model. Briefly, the input variables included MRI-measured muscle volumes and patella tendon orientation as well as lower extremity joint kinematics and muscle activation (see below for details).

Sagittal and axial MR images of each subject's tested leg were obtained using a 3.0 T MRI system (GE Signa HDx 3.0 T). Axial images of the leg (ankle mortise to the iliac crest) were acquired using a spin-echo pulse sequence (TR: 2,600–3,700 ms, TE: 11.3 ms, slice thickness: 10 mm, matrix: 512×512). Sagittal-plane images of the knee were obtained using a spin-echo pulse sequence (TR: 1,100 ms, TE: 37 ms, slice thickness: 3 mm, matrix: 512×512). Sagittal-plane images were acquired at 0°, 15°, 30°, 45°, and 60° of knee flexion during static partial weight-bearing by having subjects push against a load of 111 N. This load was provided by a custom-made non-ferromagnetic MRI loading device.

For biomechanical testing, muscle EMG were recorded from the vastus lateralis (VL), vastus medialis (VM), rectus femoris (RF), semitendinosis (ST), biceps femoris long head (BFL), medial gastrocnemius (MG), and lateral gastrocnemius (LG) using pre-amplified bipolar surface electrodes at 1,500 Hz (MA300 EMG system, Motion Lab Systems, Baton Rouge, LA). The preamplifiers had a double-differential input design and a signal bandwidth ranging from 20 to 3,000 Hz. Prior to testing, EMG signals from each muscle were obtained while subjects performed a series of three maximum voluntary isometric contractions (MVIC). EMG data obtained from these tests were for normalization purposes.

Kinematic data were recorded at a rate of 250 Hz using an 8-camera motion analysis system (Vicon 612; Oxford Metrics, Oxford, UK). Ground reaction forces (GRF) were collected at a rate of 1,500 Hz using an AMTI force plate (AMTI, Watertown, MA). Reflective markers were attached to the following bony landmarks: distal 1st toe, 1st and 5th metatarsal heads, medial and lateral malleoli, medial and lateral femoral epicondyles, greater trochanters, anterior superior iliac spines, iliac crests, and the L5–S1 junction. Additional non-collinear tracking cluster markers were placed on the heels, lateral shanks, and lateral thighs.

For the single-leg drop-land task, subjects started from a single-leg standing position on a platform (height: 25 cm) in front of the force plate. Subjects were instructed to land with

Table 1. Subject Characteristics of the ACLR and Control Groups

	ACLR (n = 10)	Control (n = 10)	*p* Value
Age (year)	25.3 ± 2.4	24.9 ± 1.7	0.67
Body mass (kg)	60.3 ± 6.7	60.5 ± 5.5	0.97
Body height (cm)	164.2 ± 8.5	167.1 ± 4.5	0.36
Activity (METs · min/week)	4582.0 ± 3334.9	3644.0 ± 2219.5	0.47
Time after surgery (months)	36.2 ± 18.5		

the tested foot on the force plate and then jump upward as high as possible. For the purposes of the current study, we were interested in the deceleration phase of the drop-land task which was defined as the time from initial contact of the foot with the ground (i.e., when the vertical GRF exceeded 20 N) to maximum knee flexion. Three drop-land trials were collected for each subject.

Data Analysis

MRI Measurements

The cross sectional area of quadriceps, hamstrings, and gastrocnemius muscles was measured from each axial MR image. The muscle volume of each slice was computed by multiplying the cross sectional area of the muscle by the slice thickness of the image (10 mm). The sum of the measured muscle volumes from all slices (i.e., total muscle volume) was combined with the muscle pennation angle and fiber length as reported in previous in vitro studies[17,18] to calculate the physiological cross sectional area (PCSA) for each muscle. The PCSA was then multiplied by a specific tension value of 23 ($N \cdot cm^{-2}$) to approximate the maximum isometric muscle force for each muscle.[19,20]

The orientation of patellar tendon relative to the tibia was measured from the sagittal MR images at each of the five knee flexion angles using ImageJ software (National Institutes of Health, Bethesda, MD). The orientation of the paella tendon relative to the tibia was quantified by measuring the angle formed by the patellar tendon and medial tibia plateau. A linear regression line was fit to the five data points to estimate the patellar tendon orientation angle from 0° to 150° of knee flexion. Based on the estimated patella tendon orientation angle, the patella tendon orientation in the sagittal-plane was represented as a unit vector (relative to the tibia) using trigonometry functions.

Kinematic and EMG Variables

Visual3D software (C-motion, Germantown, MD) was used to compute the segmental kinematics of the tested lower-extremity. Raw trajectory data were filtered using a 4th order zero-lag Butterworth low-pass filter at 6 Hz. Segment mass and center of mass location were approximated from the data of Dempster.[21] Raw EMG signals were band-pass filtered (35–500 Hz), rectified, and smoothed with a 6-Hz low-pass filter. The smoothed EMG data were normalized to the highest EMG value recorded from either the MVIC or the drop-land task. The highest EMG value from either the MVIC or the drop-land task (including both the deceleration and acceleration phases) was used for normalization purposes. This ensured that all normalized EMG values during the drop-land task were below 100%. EMG data were processed using a custom MATLAB program (MathWorks, Natick, MA).

To quantify the level of co-contraction between the knee flexor and extensor muscles, a co-contraction index (CCI) was calculated for each subject.[22] Specifically, the CCI was quantified as the ratio of the averaged normalized flexor EMG (ST, BFL, MG, and LG) to the averaged normalized extensor EMG (VM, VL, and RF) multiplied by the averaged normalized EMG of all muscles during the deceleration phase of the drop-land task. During the deceleration phase of the drop-land task, the averaged normalized extensor EMG was always greater than the averaged normalized flexor EMG. Therefore, the EMG ratios used in the CCI calculation ranged between 0 and 1. Using this convention, a larger CCI value was indicative of greater muscle co-contraction.

Subject-Specific EMG-Driven Knee Model to Quantify Knee Loading

SIMM software (MusculoGraphics, Inc., Chicago, IL) was used to create a generic lower extremity musculoskeletal model.[19] The model included 10 musculotendon actuators: VL, VM, vastus intermedius (VI), RF, ST, semimembranosis (SM), BFL, biceps femoris short head (BFS), MG, and LG. The muscle anatomic parameters of the 10 muscles were based on the values reported by Friederich and Brand[17] and Wickiewicz et al.[18] Tendon slack lengths of the 10 muscles were based on the average values reported by Delp[19] and Lloyd and Buchanan.[23]

Normalized EMG and lower extremity kinematics (hip flexion/extension, hip adduction/abduction, hip internal/external rotation, knee flexion/extension, ankle plantar-/dorsiflexion) were used as input variables for the EMG-driven model. Kinematic data were used to determine individual muscle tendon lengths and contraction velocities for the Hill-type muscle model in SIMM. Normalized EMG data were used to represent the level of muscle activation. Muscle activation of the VI was estimated as the average of the VM and VL normalized EMG amplitudes. SM was assumed to have the same activation as ST, and BFS was assumed to have the same activation as the BFL.[24] A 40-ms electromechanical delay was used to adjust for the time difference between the onset of EMG signals and onset of force output.[24] In the SIMM model, the force magnitude (F^M) of each muscle was then calculated using Equation (1):

$$F^M = F^T = F^{Max}[f(l)f(v)\text{act} + f_p(l)]\cos(\theta) \quad (1)$$

where F^{Max} is the maximum isometric muscle force; l and v are instantaneous muscle fiber length and contraction velocity, respectively; $f(l)$ and $f(v)$ are the generic muscle length–tension and force–velocity relationships, respectively; act is muscle activation; $f_p(l)$ is the length–tension of the parallel elastic element; and θ is the pennation angle.[19,24] In SIMM, instantaneous muscle fiber length and contraction velocity were estimated based on the input muscle-tendon lengths and contraction velocities (determined from joint kinematics) through a series of iterative methods that satisfied the condition in which the instantaneous muscle force and tendon force were equal ($F^M = F^T$).[24]

The estimated maximum isometric muscle forces (derived from MRI-estimated PCSA) and patella tendon orientation unit vectors (as measured from MRI) were incorporated into the generic SIMM knee model for each subject. The magnitude and orientation of each muscle force vector was calculated from the model. The calculated muscle force vectors were combined with the anthropometry of the lower leg (foot and shank),[21] GRF, and the linear acceleration of the lower leg to calculate the tibiofemoral joint force during drop-landing (Equation 2)

$$\begin{aligned}\sum \text{Forces} &= \text{muscle forces} + \text{GRF} + \text{weight of the leg} \\ &\quad + \text{tibiofemoral joint force} \\ &= m \cdot a \end{aligned} \quad (2)$$

where m is the mass of the lower leg and a is the linear acceleration vector of the center of mass of the lower leg. The force and acceleration vectors were referenced to the three-dimensional reference frame located in the tibia (Fig. 1). The tibiofemoral compressive and shear forces were the components of the joint force vector along the longitudinal (defined by the line connecting the knee and ankle joint centers) and

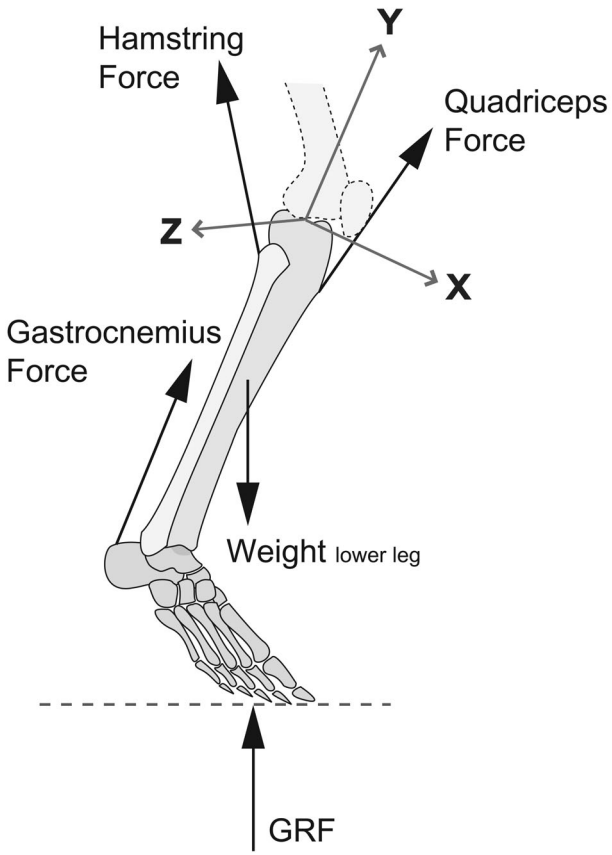

Figure 1. The free body diagram showing the tibia reference frame and the forces used to calculate the tibiofemoral shear and compressive forces.

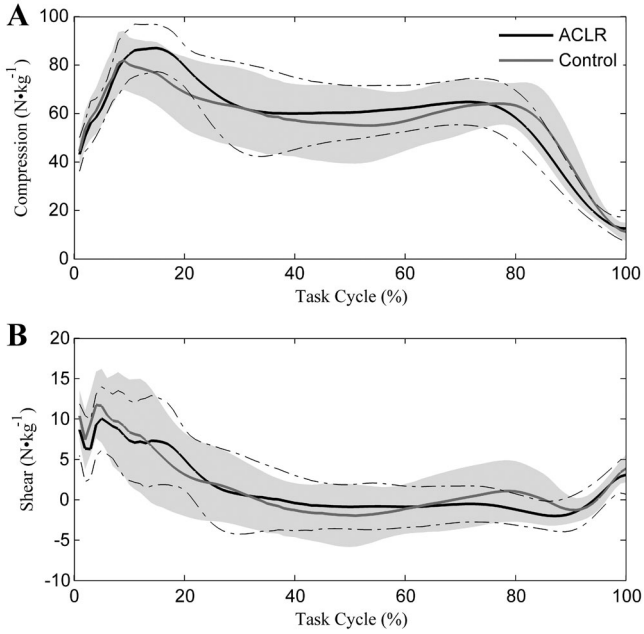

Figure 2. The ensemble average time-series curves of the tibiofemoral compressive (A) and shear (B) forces during the single-leg drop-land task. The mean ± SD for the ACLR group is depicted by the black solid and dashed lines. The mean ± SD for the control group is depicted by the grey line and shade. Positive values indicate an upward compressive force and anterior shear load resulting from the GRF and muscle forces. On average, the deceleration phase ended at 54% and 43% of the landing phase for the ACLR and control groups, respectively.

anterior–posterior axes, and were normalized to the body mass ($N \cdot kg^{-1}$) for each subject. The tibiofemoral joint forces calculated from Equation (2) represented the internal joint forces required to maintain the sagittal plane linear kinematics and dynamics of the lower leg given the muscle forces and GRF. For interpretation purposes, the signs of the internal joint forces calculated from Equation (2) were reversed to represent the total loading applied to the tibiofemoral joint via muscle forces and GRF.

Statistical Analysis

The primary dependent variables of interest included the peak tibiofemoral compressive and anterior tibial shear forces as well as the CCI value and peak knee flexion angle during the deceleration phase of the drop-land task. Secondary variables of interest included the GRF and muscle forces that contributed to the peak tibiofemoral compressive and shear forces at the time of peak compressive and anterior shear forces (i.e., the components of Equation 2). Secondary variables of interest also included the average normalized quadriceps, hamstrings, and gastrocnemius EMG. For each primary and secondary variable of interest, between-group differences were examined using independent t-tests with a significance level of $p \leq 0.05$ (SPSS software, Version 15.0, Chicago, IL).

RESULTS

The ensemble average time-series curves of the tibiofemoral compressive and shear forces for the two groups are shown in Figure 2. The ACLR group exhibited significantly greater peak tibiofemoral compressive forces than the control group (97.3 ± 8.0 vs. 88.8 ± 9.8 $N \cdot kg^{-1}$, $p = 0.025$; Table 2). Although the peak anterior shear force was smaller in the ACLR group compared to the control group, this difference did not reach statistical significance (12.2 ± 4.0 vs. 13.6 ± 4.0 $N \cdot kg^{-1}$, $p = 0.224$; Table 2).

The secondary analyses revealed that both the compressive and posterior shear forces generated by the hamstring muscles at the time of the peak tibiofemoral compressive and anterior tibial shear forces were significantly greater in the ACLR group than the control group (Table 3). No significant between-group differences were found for the other force components at the time of peak tibiofemoral compressive and anterior shear forces (Table 3).

On average, the ACLR group also demonstrated a significantly greater CCI value as well as a reduction in the peak knee flexion angle during the deceleration phase of drop-landing when compared to the control group (Table 2 and Fig. 3). The increased CCI in the ACLR group was the result of increased hamstring muscle activation ($33 \pm 12\%$ vs. $21 \pm 17\%$ of maximum, $p = 0.02$) as no significant differences in quadriceps ($36 \pm 5\%$ vs. $38 \pm 6\%$ of maximum, $p = 0.37$) and gastrocnemius activation ($28 \pm 7\%$ vs. $25 \pm 6\%$ of maximum, $p = 0.35$) was observed (Table 3).

Table 2. The Peak Tibiofemoral Compressive and Anterior Tibial Shear Forces, Co-Contraction Index, and Peak Knee Flexion during the Deceleration Phase of the Single-Leg Drop-Land Task

	ACLR (n = 10)	Control (n = 10)	*p* Value
Peak compression ($N \cdot kg^{-1}$)	97.3 ± 8.0	88.8 ± 9.8	0.025
Peak anterior shear ($N \cdot kg^{-1}$)	12.2 ± 4.0	13.6 ± 4.0	0.224
Co-contraction index	0.28 ± 0.10	0.18 ± 0.05	0.004
Peak knee flexion (°)	67.6 ± 8.8	79.1 ± 10.9	0.009

Table 3. The Compressive and Shear Components of the GRF and Estimated Muscle Forces That Contributed to the Peak Tibiofemoral Compressive and Shear Forces as Well as the Averaged EMG Values during the Deceleration Phase of the Landing

	ACLR (n = 10)	Control (n = 10)	*p* Value
Forces at peak compression ($N \cdot kg^{-1}$)			
GRF	28.5 ± 6.9	26.5 ± 4.3	0.46
Quadriceps	53.2 ± 9.1	47.3 ± 9.9	0.18
Hamstrings	3.2 ± 1.7	1.6 ± 1.5	0.04
Gastrocnemius	12.9 ± 3.3	14.0 ± 2.3	0.39
Forces at peak anterior shear ($N \cdot kg^{-1}$)			
GRF	−2.9 ± 1.3	−2.9 ± 1.4	0.94
Quadriceps	16.3 ± 5.7	16.5 ± 4.6	0.95
Hamstrings	−2.3 ± 1.3	−1.2 ± 0.7	0.03
Gastrocnemius	0.9 ± 0.3	1.1 ± 0.3	0.16
Average normalized EMG (% of max)			
Quadriceps	36.1 ± 5.0	38.4 ± 6.2	0.37
Hamstrings	32.7 ± 12.2	21.2 ± 6.7	0.02
Gastrocnemius	27.5 ± 7.0	25.3 ± 5.5	0.44

DISCUSSION

The primary purpose of this study was to test the hypothesis that females with ACLR would demonstrate increased tibiofemoral compressive forces, increased muscle co-contraction and decreased knee flexion during a single-leg drop-land task compared to uninjured healthy females. To our knowledge, this is the first study to estimate the compressive forces at the tibiofemoral joint in this population. Consistent with our hypothesis, females post-ACLR demonstrate greater peak tibiofemoral compressive forces when compared to healthy controls. Furthermore, the ACLR group also exhibited increased muscle co-contraction and decreased knee flexion during landing.

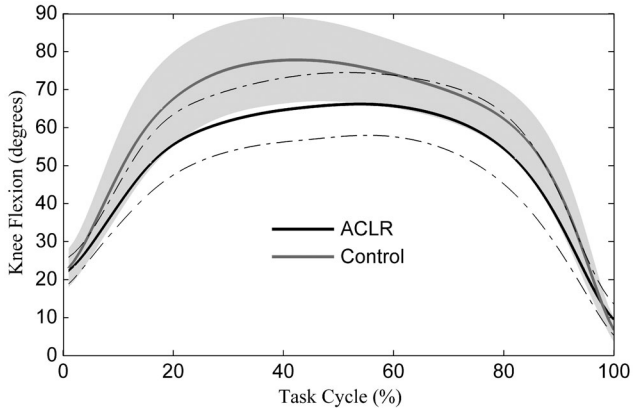

Figure 3. The ensemble average time-series curves for knee flexion during the single-leg drop-land task. The mean ± SD for the ACLR group is depicted by the black solid and dashed lines. The mean ± SD for the control group is depicted by the grey line and shade. On average, peak knee flexion occurred at 54% and 43% of the landing phase for the ACLR and control groups, respectively.

Excessive compressive forces have been shown to negatively impact articular cartilage health.[13–15] While the progression of joint degeneration appears to be a multi-factorial process, it stands to reason that the higher risk of knee OA in individuals post-ACLR may result, in part, from elevated tibiofemoral compressive forces. Although the observed increase in peak compressive force in the ACLR group was relatively modest (10%, approximately 510 N for a female with a body mass of 60 kg), intermittent high impact loading in the presence of bone, meniscus and/or articular cartilage damage could accelerate OA progression in this population. For example, higher impact forces combined with a reduction of tibiofemoral contact area owing to mensical damage or loss could result in substantial increases in cartilage stress. Longitudinal studies are needed to determine whether a causal

relationship exists between excessive joint loading and the increased risk of developing knee OA in individuals with ACLR. For example, studies examining whether individuals with greater tibiofemoral compressive forces are more likely to develop knee OA or examining whether decreasing tibiofemoral compression can reduce the progression of knee OA development in persons post-ACLR may provide insight into the mechanism(s) underlying the high risk of knee OA development in this population.

Most individuals who have undergone ACLR continue to participate in sports.[25] As such, the current study quantified tibiofemoral compressive forces during an athletic activity that is performed repeatedly during most sports (e.g., basketball, volleyball, etc.). Although it has been reported that the majority of the individuals who return to sport following ACLR do not return to their pre-injury levels,[25] the findings of the current study suggest that these individuals may be exposed to high impact forces nonetheless. Our results also would appear to support previous assertions that participation in sport following ACLR may be a risk factor with respect to knee OA development.[26]

The increased muscle co-contraction observed in our ACLR subjects is consistent with the findings of Ortiz et al.[6] and Vairo et al.[7] who also reported greater co-contraction in persons post-ACLR. The increased CCI in the ACLR group was mainly the result of increased hamstring muscle activation (Table 3). Increased hamstring muscle activation in persons post-ACLR has been hypothesized as a protective mechanism to decrease the anterior shear loads on the knee.[6,7] This premise was supported by our findings that the ACLR group on average had a similar peak anterior tibial shear force but a significantly greater posterior shear force generated from the hamstring muscles when compared to the control group (Tables 2 and 3).

Although the strategy of increased muscle co-contraction and hamstring activation may assist in stabilizing the knee, the increase in hamstring muscle force added little to tibiofemoral joint compression (Table 3). Nonetheless, an increase in hamstring muscle force would require a simultaneous increase in quadriceps force as any increase in knee flexion torque must be balanced by the knee extensors.[8] This was evident with our data in that the ACLR group had a greater compressive component of the quadriceps force at the peak tibiofemoral compressive force compared to the control group (Table 3). This difference, however, was not statistically significant.

The observed decrease in knee flexion during landing in subjects with ACLR also has been reported by Webster et al.[10] Given that knee flexion is a key factor for attenuating ground reaction forces,[11] impaired shock absorption during landing also may increase the compressive force of the tibiofemoral joint. Post-hoc analysis revealed that the ACLR group had a greater compressive component of the GRF at the time of the peak tibiofemoral compressive force than the control group (Table 3). However, this difference was not statistically significant.

Taken together, increased muscle co-contraction and decreased knee flexion may represent an altered neuromuscular strategy that influences tibiofemoral compressive forces in females who have undergone ACLR. However, the relationships among these biomechanical factors remain unclear given the limitations of the small sample size and analyses performed in the current study. Additional studies that incorporate a larger sample size, predictive statistical approaches, and/or interventions are required to advance our understanding of how altered neuromuscular strategies may contribute to increased tibiofemoral compression in the ACLR population.

The tibiofemoral compressive forces in the current study were quantified using a MRI-based EMG-driven knee model, as this approach allows for the estimation of muscle forces based on muscle activation levels. Traditionally, knee loading has been quantified using an inverse dynamics approach to estimate the resultant forces at the tibiofemoral joint.[27] However, this approach does not consider muscle forces and their contributions to joint loading.[28] While several advanced methods (i.e., inverse dynamics optimization and forward dynamics) have been proposed to estimate individual muscle forces,[29,30] a common limitation of these approaches is the inability of predicting antagonist muscle forces because of the lack of information regarding muscle co-contraction.[28,29] Given that increased muscle co-contraction has been observed in persons post-ACLR,[6,7] we feel that the use of an EMG-driven model was an appropriate method to quantify knee loading for the purpose of this study.

As with all modeling studies, there are several limitations that need to be acknowledged. While the muscle volumes were measured directly from MRI to create a subject-specific model, our PCSA calculations incorporated pennation angles and muscle fiber lengths reported from existing literature.[17,18] Based on previous in vitro cadaveric studies, the fiber lengths and pennation angles of the 10 muscles included in our knee model have been shown to be similar among cadaveric specimens.[17] Nonetheless, the use of cadaver-based pennation angles and fiber lengths may have limited the accuracy of calculating a subject-specific PCSA, and therefore the maximum isometric force of each muscle.

Another limitation of the current study was the generic nature and the lack of validation of the EMG-driven model to estimate muscle forces. Although it has been shown that the incorporation of subject-specific MRI-measured muscle volumes and moment arms significantly improves the accuracy of an EMG-driven model, this approach does not completely eliminate the errors in knee joint moment predictions.[31] In addition, generic values were used for muscle contractile or activation parameters (e.g., tendon slack length, electromechanical delay, etc.). To our knowledge, no

systematic changes in these modeling parameters have been observed in individuals with ACLR. For example, it has been shown that electromechanical delay of the quadriceps muscles is not altered in persons who have undergone ACLR.[32] Nonetheless, the use of generic values may have limited the accuracy of our muscle force and knee loading calculations. Most muscle contractile parameters are difficult to measure in vivo, and are often estimated through a mathematical optimization procedure.[24,33–35] Future modeling approaches that combine both direct measurements of muscle anatomic parameters and optimization procedures for muscle contractile parameters may further improve the accuracy and advance the application of an EMG-driven knee model.

Our model did not account for the influence of the patellofemoral joint when transferring the quadriceps forces to the patella tendon. While the values of this quadriceps force/patella tendon force ratio reported in the literature are somewhat inconsistent,[36,37] a common agreement among studies is that the transfer of quadriceps muscle force to the patella tendon decreases with knee flexion. Considering that the control subjects demonstrated greater knee flexion than the ACLR subjects during landing, one would expect that the patella tendon forces and therefore the knee compressive forces of the control subjects may have been overestimated to a greater degree when compared to the ACLR subjects. Thus, accounting for the influence of the patellofemorol joint force transfer mechanism would likely have resulted in greater group differences in tibiofemoral compression.

Furthermore, the sample sizes for both groups were relatively small and the information of concomitant meniscus injury was not obtained in the ACLR group. As such, the generalization of our findings to the entire ACLR population is limited. Given that many factors likely contribute to the development of knee OA in persons post-ACLR, our findings of elevated compressive forces in our small sample may only be representative of a subgroup of the entire ACLR population. In addition, the ACLR group consisted only of females with mixed graft types (allografts and bone-patella-tendon-bone autografts). Although increased muscle co-contraction has been observed in individuals with ACLR using various graft types,[5–7] it is possible that individuals with ACLR using different graft types may demonstrate different neuromuscular strategies. Future studies incorporating larger sample sizes and comparing different types of ACLR grafts are needed to better understand the loading profiles of the tibiofemoral joint in this population.

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