Variation in ACL and MCL Strain Before Initial Contact Is Dependent on Injury Risk Level During Simulated Landings

Nathaniel A. Bates,*†‡§ PhD, Nathan D. Schilaty,†‡§ PhD, DC, Aaron J. Krych,†‡ MD, and Timothy E. Hewett,†‡§‖ PhD

Investigation performed at the Mayo Clinic, Rochester, Minnesota, USA

Background: The existent literature has well explored knee ligament kinetics and strain at and after initial contact (IC) during landing tasks. However, little is known about knee ligament biomechanics in flight before IC.

Purpose: To quantify and compare change in anterior cruciate ligament (ACL) and medial collateral ligament (MCL) strain before IC relative to after IC.

Study Design: Descriptive laboratory study.

Methods: A total of 40 cadaveric specimens were analyzed after being subjected to simulated landings in a mechanical impact simulator. External joint loads of varying magnitudes were applied to mimic relative injury risk load levels from an in vivo cohort and were coupled with an impulse force to represent initial ground contact. Implanted strain gauges continually recorded ligament strain. Kruskal-Wallis tests evaluated the significance of risk level and pre- and post-IC factors, while Wilcoxon each-pair tests evaluated differences within both factors.

Results: Strain responses during simulated landing tasks for the ACL ($P \geq .545$) and MCL ($P \geq .489$) were consistent after IC regardless of the level of relative injury risk simulated in each trial. Before IC, the level of injury risk kinetics applied to a specimen differentiated strain response in the ACL ($P < .001$) and MCL ($P < .001$), as higher risk profiles produced greater changes in ligament strain. Mean baseline strain was 4.0% in the ACL and 1.0% in the MCL. Mean change in strain from the ACL ranged from 0.1% to 3.9% pre-IC and from 2.9% to 5.7% post-IC, while the MCL ranged from 0.0% to 3.0% pre-IC and from 0.9% to 1.3% post-IC.

Conclusion: Within each ligament, post-IC strain response lacked statistical differences among simulated risk profiles, while pre-IC response was dependent on the risk profile simulated. Individually, neither pre- nor poststrain changes were enough to induce ACL failure, but when combined over the course of a full landing task, they could lead to rupture.

Clinical Relevance: Prevention and rehabilitation techniques should aim to limit the presence of increased risk biomechanics in flight before landing, as impulse delivery at IC is inevitable.

Keywords: ACL; MCL; knee injury; landing; sports medicine; initial contact; joint biomechanics

Based on video analysis, anterior cruciate ligament (ACL) injures are generally believed to occur within 0 to 67 ms of initial ground contact when an athlete is landing or cutting.[31,32] However, as direct monitoring of a live ACL during a rupture event is not currently feasible, these data have never been explicitly collected. What is known is that athletes with poor neuromuscular control predispose their knees to large ligamentous loads by landing on their lower extremity with increased knee valgus angle and moment as well as decreased flexion at initial contact (IC).[23] While the timing of loads and ligament strains from mechanical impact simulator data corroborates the timing estimated from video analyses (N. A. Bates, PhD, et al, unpublished data, 2019),[30,51] there remains speculation in the orthopaedic community that ACL rupture occurs as a product of exaggerated quadriceps contraction before IC.[14,49] Relative to a maximum voluntary contraction, electromyography of musculature around the knee has estimated preactivation before landing around 20% for the biceps femoris, 30% for the rectus femoris and tibialis anterior, and 85% for the vastus lateralis and gastrocnemius.[35] However, these preactivations can change according to the conditions that an athlete encounters during a dynamic task and subsequently correspond to differences in joint stiffness, kinematics, and dynamics in flight and continued throughout the performance of a dynamic task.[36] Furthermore, poor

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neuromuscular control has been associated with ACL injuries, and preactivation of this musculature occurs in flight before IC.[16,26,42] Accordingly, joint dynamics leading up to IC are likely critical to changes in ACL strain as well as the maintenance of ACL integrity throughout a landing task.

Those athletes who exhibit poor neuromuscular control are referred to as "high injury risk" in the literature.[3,23,38] Within a population of 205 prospectively screened athletes, 8 of 9 athletes who went on to ACL injury were originally identified to present with both knee abduction moments and angles in the top third of the population cohort examined.[23] Randomized controlled trials with targeted neuromuscular training regimens have demonstrated that these protocols can be effective in the reduction of high-risk biomechanics, knee joint dynamics, and ACL injury incidence across a cohort population.[19,20,22,44,45,47,50] Specifically, this preventive training has proven most efficacious in high-risk athletes[20,21] and is echoed by the ability of changes in muscle preactivation to alter knee joint dynamics.[36] Accordingly, if high-risk athletes are predisposed to injury and exhibit kinetic and kinematics disparities from their lower-risk counterparts at IC, then it is also likely that these high-risk athletes predispose their ACL to greater strain and their knee to greater joint loading while in flight before IC with the ground when landing from a jump. As such, ligament strain behavior pre- and post-IC is likely important to an ACL injury event. However, given the limitations of in vivo investigation, ligament behavior relative to IC has not previously been examined. The effects of varied kinematics post-IC have been examined in a robotic simulation model, where ligament strains between male and female models were found to be largely comparable.[5,6]

Furthermore, interventional training is important to the integrity of additional soft tissue structures in the knee, as medial collateral ligament (MCL) injury is concomitantly reported in up to one-third of noncontact ACL ruptures.[33,41,53] These structures are similarly susceptible to increased strain from frontal plane torque loading,[4] although the ACL is more responsive to higher-risk motion profiles throughout landing.[7,8] MCL integrity is important, as surgically unrepaired concomitant MCL injuries lead to increased strain and increased probability of secondary injury on ACL reconstruction grafts.[11,48] Although it is known that the MCL does not significantly absorb additional knee load immediately after ACL failure,[43] as with the ACL, MCL response to the sudden and strong delivery (or impulse) of forces to the leg at IC remains unexplored.

Development of the mechanical impact simulator now allows for the investigation of intra-articular mechanics in cadaveric specimens during jump landing simulations, which reliably replicates the clinical presentation of ACL injuries.[9,10] From this simulator, it is known that the absolute magnitude of ACL strain at 33 ms and 67 ms after impact is higher than ACL strain at IC (N. A. Bates, PhD, et al, unpublished data, 2019). Likewise, it is known that peak ACL strain increases during landing simulations that emulate very-high-injury-risk kinetics as opposed to simulations that emulate baseline- or moderate-risk kinetics.[7] However, how the distribution of changes in ACL strain is influenced before and after impulse delivery at IC remains unreported.

The objective of this study was to quantify change in ACL (ΔACL) and change in MCL (ΔMCL) strain pre-IC (from neutral limb alignment through IC) and post-IC (from IC to peak strain) and then to subsequently compare differences between the pre-IC and post-IC strain changes. The hypothesis tested was that ΔACL and ΔMCL strain pre-IC would be greater in high-risk simulated landings than lower-risk simulations but that risk profiles would not exhibit differences in ΔACL or ΔMCL strain post-IC. The second hypothesis tested was that ΔACL strain pre-IC would be greatest in the failure trial.

METHODS

A total of 46 cadaveric lower extremity specimens were acquired from an anatomic donations program (Anatomy Gifts Registry) to complete mechanical impact simulations. Six specimens were excluded owing to structural weakness during setup (n = 2), substandard bone stiffness (n = 1), and equipment failure/inconsistency in testing execution (n = 3). Therefore, 40 specimens were available for analysis (20 male, 20 female; age = 41.5 ± 8.3 years [mean ± SD]; mass = 85.8 ± 25.2 kg; height = 173.4 ± 10.4 cm). Explicit details of specimen preparation, setup, and testing protocol for the mechanical impact simulator have been published in the literature.[9,10] Briefly, each specimen was resected of soft tissue superior to the top of the patella, inverted, and then potted into custom fixtures mounted atop a 6-axis load cell (Omega160 IP65/IP68; ATI Industrial Automation Inc) such that the long axis of the femur was aligned with the vertical axis of the load cell (Figure 1A). During tissue resection, the quadriceps and hamstrings tendons were left

*Address correspondence to Nathanial A. Bates, PhD, Mayo Clinic, 200 First Street SW, Rochester, MN 55902, USA (email: batesna@gmail.com).
†Department of Orthopedic Surgery, Mayo Clinic, Rochester, Minnesota, USA.
‡Department of Biomedical Engineering and Physiology, Mayo Clinic, Rochester, Minnesota, USA.
§Sports Medicine Center, Mayo Clinic, Rochester, Minnesota, USA.
‖Department of Physical Medicine and Rehabilitation, Mayo Clinic, Rochester, Minnesota, USA.

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Ethical approval was not sought for the present study.

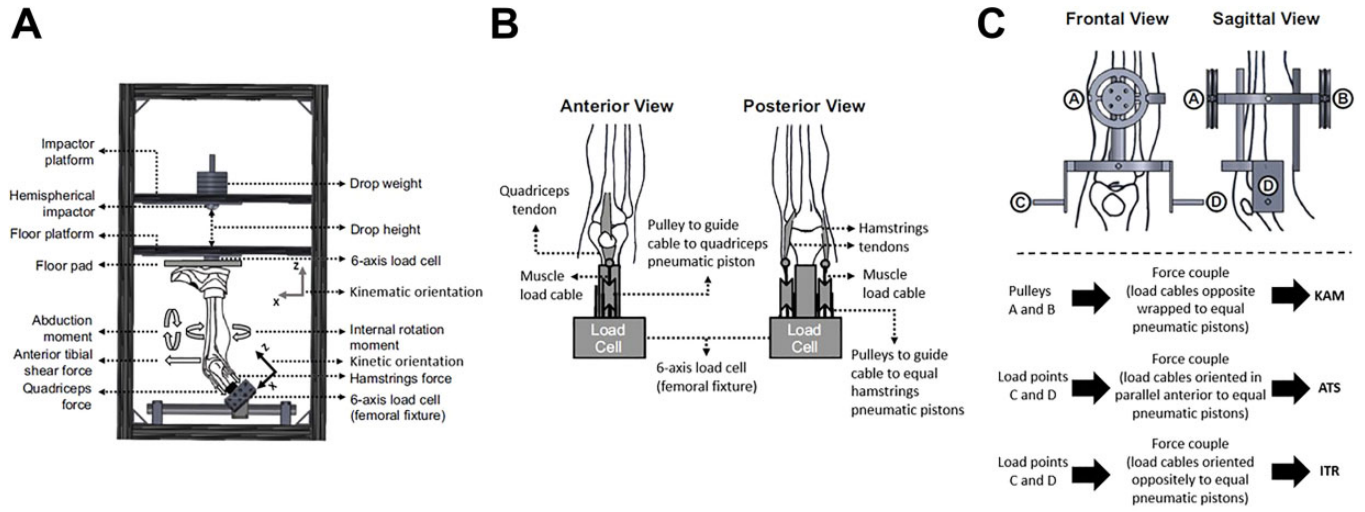

Figure 1. (A) Meta-view of custom-designed mechanical impact simulator for creation of ACL ruptures.[9] (B) Cable pulley system used to deliver pneumatically actuated loads to the quadriceps and hamstrings tendons. (C) External fixation frame attached to the tibia and used to deliver pneumatically actuated KAM, ATS, and ITR loads to each specimen. ACL, anterior cruciate ligament; ATS, anterior tibial shear; ITR, internal tibial rotation; KAM, knee abduction moment. This figure has been reproduced from Bates et al[8] and Kiapour et al.[29]

TABLE 1
Externally Applied Loads for the Mechanical Impact Simulator Based on the In Vivo Cohort[a]

	KAM		ATS		ITR	
Risk	In Vivo Cohort, Percentile	Load, N·m	In Vivo Cohort, Percentile	Load, N	In Vivo Cohort, Percentile	Load, N·m
Baseline	2	2.4	0	40	0	1.0
Low	—	—	—	—	33	9.7
Moderate	68	27.0	—	—	67	18.6
High	99	53.6	90	98	100	53.7
Very high	200	114.6	—	—	—	—

[a]ATS, anterior tibial shear; ITR, internal tibial rotation; KAM, knee abduction moment.

intact. With cable clamps and carbon fiber rope (Ø = $^{7}/_{64}$ inch, Amsteel-Blue; Samson), these tendons were affixed to pneumatic pistons (SMC Corp) that applied simulated quadriceps and hamstrings muscle forces to the specimen throughout each simulation in a 1:1 ratio at a magnitude of 450 N (Figure 1B).[9,10,37] These muscle force actuators were activated at the start of each trial and remained constant throughout.

The tibia was oriented vertically and secured at a 25° flexion angle with respect to the femur, to represent the typical IC position that young athletes present when landing from a 31-cm drop.[2] Tibial position was maintained by resting an 18.1-kg ground platform on the sole of the foot of each specimen. A uniaxial load cell (1720ACK-10kN; Interface Inc) was housed within the ground platform and aligned with the heel of the specimen to measure vertical ground-reaction force. A custom-designed fixture was then clamped to the tibia and affixed to several pneumatic cylinders via carbon fiber rope. The rotary and linear attachments on this tibial clamp allowed the actuation of the pneumatic cylinders to apply simulated knee abduction torque (ie, knee abduction moment [KAM]), anterior tibial shear (ATS), and internal tibial torque (ITR) at the knee joint (Figure 1C).

Pneumatic loads were applied approximately 1 second before IC. These loads were applied by separate actuators that were simultaneously engaged to preset magnitudes, as indicated in Table 1. The magnitude of these loads was previously determined from in vivo kinetics calculated with 3-dimensional motion analysis from a cohort of 44 healthy athletes (age = 23.3 ± 4.1 years; mass = 72.6 ± 13.9 kg; height = 172 ± 10 cm) who completed drop vertical jump tasks off a 31-cm box.[9] For these values, the 0th percentile of the population was established as the baseline risk for simulation. The remainder of the population values was separated into relative injury risk classifications based on KAM and ITR, as indicated in Table 1. Population percentiles were selected, as previous literature has shown that relative injury risk classification is expected to divide athletic populations roughly into thirds based on functional outcomes and KAM loading.[3,39] During simulations, the magnitude of each load (KAM, ATS, and ITR) was

individually randomized, which resulted in a stratification of 26 simulated landing conditions for each specimen with coupled external loads. For the purposes of this study, risk profiles were designated when KAM and ITR loading conditions exhibited matching percentiles of loading (ie, baseline risk = 0th percentile for KAM and ITR; moderate risk = 67th percentile for KAM and ITR; high risk = 100th percentile for KAM and ITR). For the very-high-risk profile, KAM was set to the 200th percentile of the population, as it remains the single kinetic factor with the most significant association to ACL strain and injury risk.[4,8,23] The mechanical influence of each of these varied loading conditions on ACL strain during simulated landings has previously been quantified.[8]

During testing, a 34.0-kg weight sled was suspended with electromagnets 31 cm above the ground platform.[9] An electronic trigger was used to release this weight sled to gravity along vertically oriented linear bearings to impact the ground platform in line with the long axis of the tibia of the specimen. Simulations were performed until soft or hard tissue damage was demonstrated on the intra-articular knee structures of the specimen. For any specimen that survived the full randomized protocol intact, external loads were reset to the maximum percentile tested, and the impact weight sled was adjusted to 0.5× bodyweight. For each consecutive impact, the external loads were increased by 20% increments until soft or hard tissue damage was induced.

Before testing, 2 portals were cut into the anterior aspect of the knee medial and lateral to the patellar tendon. Through these portals, a custom barbed differential variance resistance transducer (DVRT; LORD MicroStrain) strain gauge was implanted onto the distal third of the anteromedial bundle of the ACL, as described in the literature.[9,10,17,18,29,34,40] A second DVRT was implanted into the midsubstance of the MCL across the tibiofemoral joint line.[9,10,40] These DVRTs were used to determine ligament strain. Strain was defined as change in length of the DVRT divided by total length of the DVRT at the neutral position of each ligament. The neutral position for the ACL was determined by manually articulating the tibia along the anterior/posterior axis until a displacement plateau was identified.[9,10] Such a plateau would represent the location where the ligament ceased to mechanically respond to change in position and thus indicate the zero strain length of the structure. For the MCL, a similar protocol was followed to determine neutral position. However, instead of anterior/posterior articulation of the tibia, an abduction/adduction articulation of the tibia about the knee joint was applied. Change in strain pre-IC was determined by subtracting ligament strain at the initialization of each trial from ligament strain at IC. Likewise, change in strain post-IC was determined by subtracting ligament strain at IC from the maximum ligament strain value recorded after IC occurred. IC was determined by the point where the vertical ground-reaction force first continually exceeded 25 N.

Throughout simulations, 6-axis joint loading at the knee, ACL strain, MCL strain, and vertical ground-reaction forces were continuously collected at 10 kHz. All data were synchronized with an electronic trigger. Collected data were filtered through a 12-Hz, low-pass, fourth-order Butterworth filter[9,34] and processed with custom code written in LabVIEW (Version 2016; National Instruments) and MATLAB (Version 2015b; The MathWorks Inc).

Statistical analysis was performed in JMP Pro (v 14; SAS Institute Inc). Strains from each ligament were analyzed separately. Statistical significance was evaluated with a Kruskal-Wallis test with factors of relativity to IC (pre-IC, post-IC) and risk profile (baseline risk, moderate risk, high risk, very high risk, prefailure, and failure). Wilcoxon each-pair post hoc tests were used to evaluate differences within factors where significance was identified. Significance was set a priori at $\alpha < 0.05$ and Bonferroni corrected in cases of multiple comparisons. Assessments were performed separately in a cohort of 19 specimens that survived the full testing protocol, including the very-high-risk profile simulations (15 male, 4 female; age = 39.1 ± 8.9 years; mass = 93.6 ± 22.52 kg; height = 175.9 ± 8.6 cm), and in a cohort of 34 specimens that survived moderate-risk profile simulations (20 male, 14 female; age = 41.5 ± 8.4 years; mass = 86.9 ± 23.3 kg; height = 174.3 ± 10.4 cm). This division of cohorts was employed to determine whether specimens that exhibited lower failure thresholds altered the mechanical observations made in the cohort that completed the full simulation protocol. Data collected from the failure trial were analyzed only pre-IC because strain data are uninterpretable at the point of ACL failure,[10,29,43] but no failures were documented before IC. A matched-pairs analysis between pre- and post-IC strains was performed to verify results, as we have multiple data points being collected within subject and within trial.

RESULTS

Very-High-Risk Group

Relativity to IC ($P < .001$) (Figure 2) and risk profile ($P < .001$) were significant factors to ΔACL strain during simulated landings. Risk profile was significant to ΔACL strain pre-IC ($P < .001$) but was not significant post-IC ($P = .919$). Pre-IC, ΔACL strain was greater in the prefailure and failure profiles than it was in the moderate- or baseline-risk profiles ($P \leq .001$). Likewise, pre-IC ΔACL strain was greater in the failure profile than the high-risk profile ($P = .008$) and greater in the very-high-risk profile than in the baseline- or moderate-risk profile ($P \leq .005$). Finally, pre-IC ΔACL strain was greater in the high-risk profile than the baseline-risk profile ($P = .035$). The magnitude of ΔACL strain was greater post-IC than pre-IC for the baseline-risk ($P < .001$), moderate-risk ($P < .001$), and high-risk ($P = .008$) profiles, but there was no difference for the very-high-risk ($P = .804$) and prefailure ($P = .327$) profiles. Matched-pairs analysis did not change significance for any profile. The mean ± SD baseline ACL strain before any load was applied was 4.0% ± 5.2%.

Relativity to IC ($P = .004$) (Figure 3) and risk profiles ($P < .001$) were significant factors to ΔMCL strain during simulated landings. Risk profile was a significant factor to ΔMCL strain pre-IC ($P < .001$) but was not significant

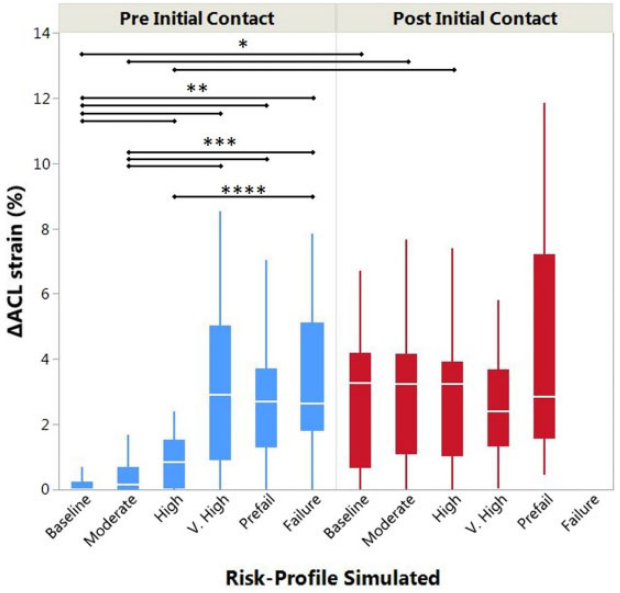

Figure 2. Bar plot of ΔACL strain vs risk profile simulated for the 19 specimens that completed high-risk simulations. Data displayed as medians with interquartile range bars. Data are grouped pre-IC and post-IC for each risk profile (baseline risk, low risk, moderate risk, high risk) as well as the prefailure and failure simulations. Strain from the failure trial is not reportable post-IC. *Significant difference between pre-IC and post-IC. **Significant difference from baseline risk. ***Significant difference from moderate risk. ****Significant difference from high risk. ACL, anterior cruciate ligament; IC, initial contact.

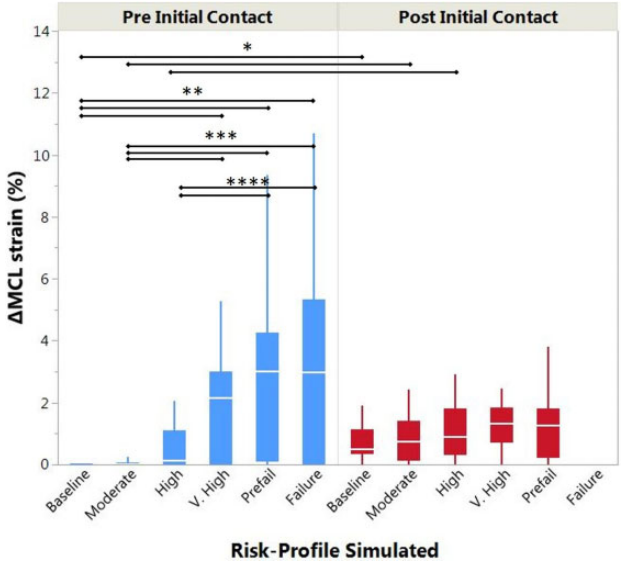

Figure 3. Bar plot of ΔMCL strain vs risk profile simulated for the 19 specimens that completed high-risk simulations. Data displayed as medians with interquartile range bars. Data are grouped pre-IC and post-IC for each risk profile simulated (baseline risk, low risk, moderate risk, high risk) as well as the prefailure and failure simulations. Strain from the failure trial is not reportable post-IC. *Significant difference between pre-IC and post-IC. **Significant difference from baseline risk. ***Significant difference from moderate risk. ****Significant difference from high risk. IC, initial contact; MCL, medial collateral ligament.

post-IC ($P = .795$). Pre-IC, the very-high-risk and prefailure profiles exhibited greater ΔMCL strain than the baseline-, moderate-, or high-risk profile ($P \leq .026$). Pre-IC, the failure profile exhibited greater ΔMCL strain than the baseline- and moderate-risk profiles ($P \leq .009$). The magnitude of ΔMCL strain was greater post-IC than pre-IC for the baseline-risk ($P < .001$), moderate-risk ($P < .001$), and high-risk ($P = .021$) profiles but not for the very-high-risk ($P = .140$) or prefailure ($P = .096$) profile. Matched-pairs analysis indicated that ΔMCL strain was also greater pre-IC than post-IC for the very-high-risk ($P = .028$) and prefailure ($P = .018$) profiles. The mean ± SD baseline MCL strain before any load was applied was 0.8% ± 2.6%.

Moderate-Risk Group

Relativity to IC ($P < .001$) (Figure 4) and risk profile ($P < .001$) were significant factors to ΔACL strain during simulated landings. Risk profile was significant to ΔACL strain pre-IC ($P < .001$) but was not significant post-IC ($P = .545$). Pre-IC, ΔACL strain was greater in the prefailure and failure profiles than it was in the moderate- or baseline-risk profile ($P < .001$). The magnitude of ΔACL strain was greater post-IC than pre-IC for the baseline-risk ($P < .001$) and moderate-risk ($P < .001$) profiles, but there was no difference for the prefailure profile ($P = .094$). Matched-pairs analysis indicated that prefailure profiles also had greater ΔACL strain pre-IC than post-IC ($P = .043$). The mean ± SD baseline ACL strain before any load was applied was 4.0% ± 5.1%.

Relativity to IC ($P < .001$) (Figure 5) and risk profile ($P < .001$) were significant factors to ΔMCL strain during simulated landings. Risk profile was significant to ΔMCL strain pre-IC ($P < .001$) but was not significant post-IC ($P = .489$). Pre-IC, ΔACL strain was greater in the prefailure and failure profiles than it was in the moderate- or baseline-risk profile ($P < .001$). The magnitude of ΔACL strain was greater post-IC than pre-IC for the baseline-risk ($P < .001$) and moderate-risk ($P < .001$) profiles, but there was no difference for the prefailure profiles ($P = .809$). Matched-pairs analysis did not change significance for any profile. The mean ± SD baseline MCL strain before any load was applied was 1.0% ± 2.5%.

DISCUSSION

The cadaveric simulation data supported the hypothesis that ΔACL and ΔMCL strain pre-IC would be greater in high-risk simulated landings than lower-risk simulations but that risk profiles would not exhibit differences in ΔACL or ΔMCL strain post-IC. After IC, median ΔACL strain was between 2.4% and 3.3% for the very-high-risk simulation group and 3.0% to 3.3% for the moderate-risk simulation group (Figures 2 and 4). Regardless of which risk profile was simulated,

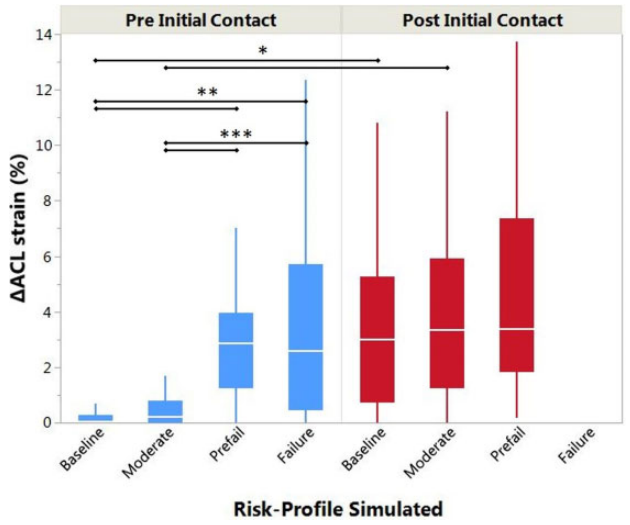

Figure 4. Bar plot of ΔACL strain vs risk profile simulated for the 33 specimens that completed moderate-risk simulations. Data displayed as medians with interquartile range bars. Data are grouped pre-IC and post-IC for each risk profile simulated (baseline risk, low risk, moderate risk, high risk) as well as the prefailure and failure simulations. Strain from the failure trial is not reportable post-IC. *Significant difference between pre-IC and post-IC. **Significant difference from baseline risk. ***Significant difference from moderate risk. ACL, anterior cruciate ligament; IC, initial contact.

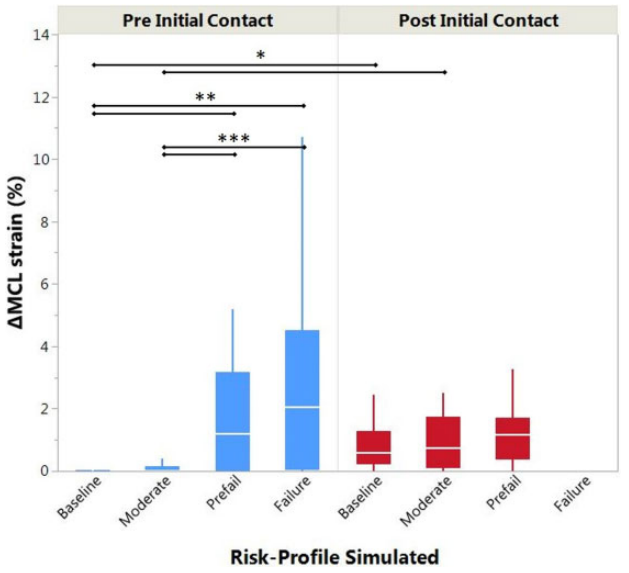

Figure 5. Bar plot of ΔMCL strain vs risk profile simulated for the 33 specimens that completed moderate-risk simulations. Data displayed as medians with interquartile range bars. Data are grouped pre-IC and post-IC for each risk profile simulated (baseline risk, low risk, moderate risk, high risk) as well as the prefailure and failure simulations. Strain from the failure trial is not reportable post-IC. *Significant difference between pre-IC and post-IC. **Significant difference from baseline risk. ***Significant difference from moderate risk. IC, initial contact; MCL, medial collateral ligament.

the median strain response of the ACL to impulse delivery at IC was within a 1% strain differential. Before IC, the median ΔACL strain was highly dependent on risk profile and ranged from 0.0% to 2.9% in the very-high-risk simulation group and from 0.0% to 2.8% in the moderate-risk simulation group. Therefore, the external kinetics (KAM, ATS, ITR) applied to the knee joint had a greater influence on whether injury events were induced, as they presented variable ΔACL strains in opposition to the consistent strain induced from impulse delivery. Patterns of mechanical behavior for the MCL were similar but with smaller magnitudes of change. Before IC, median ΔMCL strain ranged from 0.0% to 3.0% in the very-high-risk simulation group and from 0.0% to 2.1% in the moderate-risk simulation group (Figures 3 and 5). After IC, median ΔMCL strain ranged from 0.5% to 1.3% in the very-high-risk simulation group and from 0.6% to 1.2% in the moderate-risk simulation group.

This ligamentous strain response indicates that poor neuromuscular activation at the time of landing is more deleterious than impact delivery itself. When impact occurs on a specimen in a baseline-risk simulation, the additive effect of native ACL strain plus ΔACL strain from impact does not approach reported thresholds of ligament rupture.[8,13,34,52] However, in a high-risk simulation, the additive effect of native ACL strain plus ΔACL strain from greater external kinetics plus ΔACL strain from impact does approach the failure threshold. Thus, in the absence of poor neuromuscular control, the distribution of impulse force generated across the knee upon landing from a jump does not represent a tangible threat of damage to the ACL.

Neither external kinetic loads nor impulse delivery delivered sufficient force to the ACL or MCL to generate rupture individually. Mean failure strain has been reported between 15.0% and 17.9% for the ACL[9,10,13,52] and 17.1% for the MCL.[28] Baseline strains in the present study were 4.0% for the ACL and 0.8% for the MCL. Summation of these baseline strains with the largest ΔACL and ΔMCL strains before IC or the highest ΔACL and ΔMCL strains after IC equates to absolute strain of 7.9%, 2.4%, 8.7%, and 3.9%, respectively. Even when the large standard deviations associated with DVRT data collected from soft tissue structures are accounted for,[5,8,10,12] these values are well below the expected failure range of both ligaments. However, when pre-IC strain changes, post-IC strain changes, and baseline ligament strains are all summated, the peak absolute strains attain the previously reported failure values of 15.3% ± 8.7% for the ACL and 5.1% ± 5.6% for the MCL.[10] Such loads put the ACL at high risk for injury, while the MCL maintained a significant safety factor in most specimens. Accordingly, these mechanical conditions at the knee during landing aid in the justification of why less than one-third of patients with ACL injury present concomitant MCL injury[33,41,53] despite both ligaments being loaded from knee abduction.[4,8]

The second hypothesis tested was that ΔACL strain pre-IC would be greatest in the failure trial. This hypothesis was partially supported. Indeed, the failure trial produced the largest raw magnitude of pre-IC ΔACL strain.

However, this value was significantly different from some, but not all, of the other risk profiles. For the very-high-risk and moderate-risk testing groups, the prefailure profile lacked statistical difference from the failure profile group. Furthermore, the profiles tested within the very-high-risk group also lacked statistical significance from the failure profile. These observations likely indicate that while ACL strain exhibits a direct correlation with increases in injury risk level,[7,8] there may also exist a cutoff threshold at which the risk level simulated places most specimens at risk of rupture. Consequently, this behavior supports the need for injury-risk screening in athletes, as the cutoff for increased ACL injury risk in vivo has been established at 25.25 N·m of KAM upon landing from a 31-cm drop.[23,38]

In vivo randomized controlled trials have shown that the effects of compliant, targeted neuromuscular control interventions on the reduction of modifiable biomechanical variables at IC and during the stance phase of jump landing is greatest for athletes classified as high risk for injury.[20,21,45,46] Therefore, targeting frontal plane control of the knee and hip joints during neuromuscular interventions remains critical to injury prevention tactics. However, the influence of neuromuscular training on joint mechanics during the flight phase of jump landing, before IC, remains uninvestigated. These interventions have also been shown to reduce vertical ground-reaction forces upon landing,[15,24,27] which leads to less impulse force on the knee and lower ΔACL strain. Literature has demonstrated that reduction of vertical ground-reaction force during landing is a positive biomechanical response in that a prospective cohort of athletes demonstrated that those with higher ground-reaction forces were more likely to progress to ACL injury.[23] Therefore, effectiveness of compliant neuromuscular training in the reduction of ACL injuries across a population of athletes with high-risk landing tendencies is justified by the present study, as targeted training has been proven to diminish the magnitude of both sources of ACL strain generation presently identified (external kinetics from neuromuscular activation and impulse ground-reaction forces from IC).

One limitation of the present investigation is that during in vivo landings, muscle activations and kinetic loading would be dynamic in conjunction with impulse delivery. Not only would the loading magnitude be dynamic, but the activations would also be time synchronized relative to ground contact based on electromyography data collected in vivo from participants during the performance of drop landing tasks. In the mechanical impact simulator, kinetic loads are dynamically applied 1 second before IC but stabilize at a constant value before impact, while muscle activations are constantly applied at a preset magnitude immediately before the start of a simulation. In addition, the current model was limited to quadriceps and hamstrings activations. In an ideal case, our simulator would be able to apply an in vivo–informed load to all of the muscle tendon units that originate or insert around the tibiofemoral joint. Such a precise simulation of muscle activations would potentially preclude the need for external force applications, as the surrounding musculature would prepare the joint environment in the same physiologic manner as during an in vivo drop landing. Furthermore, no stratification of applied quadriceps and hamstrings muscle forces was examined in the current construct, which prevented correlation analyses between enacted muscle forces and intra-articular joint dynamics. While not an exact replication of in vivo landing conditions, these load applications are a realistic improvement over previous simulators that applied loading with static weights.[9,34] Despite this limitation, the mechanical impact simulator was still able to replicate the clinical presentation of ACL and concomitant injuries, which lends validity to the reported outcomes.[10] Furthermore, the external kinetic loads applied to each specimen in the mechanical impact simulator represent the peak values calculated from motion capture analyses performed on the in vivo cohort described in the Methods section. Therefore, although external loading was applied before impact delivery, the simulation model ensured that the worst-case scenario was achieved for each risk profile simulated. A future iteration of mechanical impactor simulations would be encouraged to investigate electromyography timing of muscle activation during landing in vivo and then coordinate the pneumatic actuators to dynamically apply forces respective to their real-time activations. However, such an adaption would be nontrivial in development and expenditure. Furthermore, it should be noted that ACL injures are predicted to occur within 50 ms of IC by video analysis[25,31,32] and within 55 ms of IC by the mechanical impact simulator (N. A. Bates, PhD, et al, unpublished data, 2019). Investigation has shown that motor time in a hamstrings muscle in males is >82 ms, premotor time is >134 ms, and reaction time is >213 ms, with even greater response times in females.[1] Accordingly, unless these muscle contractions are activated in flight before IC, it is unknown if dynamic application of muscle loads at IC would influence ACL strain before injury onset.

CONCLUSION

ACL and MCL strain responses during simulated landing tasks were consistent after IC regardless of the level of injury risk kinetics applied to a specimen. Before IC, the level of injury risk kinetics applied to a specimen differentiated ACL and MCL response, as higher risk profiles produced greater changes in ligament strain. Individually, neither pre-IC nor post-IC strain changes were enough to induce ACL failure, but when combined over the course of a full landing task, they may lead to rupture. Reduction in external loads applied to the joint before IC subsequently reduced ACL strain and probability of ligament failure when impulse was delivered. Future investigation should work to quantify the effects of neuromuscular training on lower extremity biomechanics while athletes are in flight before landing from a jump.

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