The Role of Axial Compressive and Quadriceps Forces in Noncontact Anterior Cruciate Ligament Injury : A Cadaveric Study

Simon J. Wall, David M. Rose, E. Grant Sutter, Stephen M. Belkoff and Barry P. Boden

Am J Sports Med 2012 40: 568 originally published online December 14, 2011
DOI: 10.1177/0363546511430204

The Role of Axial Compressive and Quadriceps Forces in Noncontact Anterior Cruciate Ligament Injury

A Cadaveric Study

Simon J. Wall,* FRCS, David M. Rose,* MRCS, E. Grant Sutter,* MS, Stephen M. Belkoff,* PhD, MPH, and Barry P. Boden,†‡ MD
Investigation performed at the Johns Hopkins International Center for Orthopaedic Advancement, Baltimore, Maryland and The Orthopaedic Center, Rockville, Maryland

Background: Compressive and quadriceps forces have been associated with noncontact anterior cruciate ligament (ACL) injury. The purpose of this study was to quantify the relative importance of each load component during noncontact ACL injury.

Hypothesis: We hypothesized that the introduction of a quadriceps force lowers the axial compressive force threshold to produce ACL injury.

Study Design: Controlled laboratory study.

Methods: Six pairs of fresh-frozen cadaveric knees, flexed to 15°, were loaded with axial compression (group A) or compression with a quadriceps force (group B) until failure. All specimens underwent axial compressive loading under displacement control with a time to peak load of 50 msec. The initial displacement of the MTS actuator was 8 mm and was increased in 2-mm increments with successive tests until catastrophic damage of the joint occurred. Failure was determined by a combination of clinical specimen examination and force-displacement data analysis and by dissection and direct visualization after failure was recognized. Differences in failure load between groups were examined using a paired *t* test (significance, $P \leq .05$).

Results: In group A, there were 2 isolated ACL injuries, 2 ACL ruptures combined with a tibial plateau fracture, and 2 isolated tibial plateau fractures. In group B, there were 5 isolated ACL ruptures and 1 tibial plateau fracture. There was a significant difference in the average failure load between groups A and B: 10 832 N (95% confidence interval [CI], 9743-11 604 N) and 6119 N (95% CI, 4335-7903 N), respectively.

Conclusion: Isolated compressive forces displayed an ability to produce an ACL injury in this cadaveric model, but the addition of a quadriceps load significantly reduced the compressive force required for ACL injury.

Clinical Relevance: Compressive and quadriceps forces contribute to noncontact ACL injury and should be taken into account when developing ACL injury prevention programs and rehabilitation after ACL reconstruction.

Keywords: anterior cruciate ligament rupture; knee injury; tibiofemoral compression; quadriceps

Of all anterior cruciate ligament (ACL) injuries, 70% have been reported to be noncontact injuries (NC-ACLIs).[2] The exact mechanism of NC-ACLI is not known. Axial compressive forces are capable of creating ACL injuries.[1,4,7,12] A recent video analysis of NC-ACLI identified a vulnerable landing position, which is hypothesized to result in rapid excessive axial loading of the knee.[4] This vulnerable position consists of a flat-footed landing with an extended knee and the hip in flexion. The flat-footed landing position reduces the ability of the calf muscle to contract and absorb energy, thus resulting in a greater impulse to the knee than would be the case with a flexed knee. Tibial plateau angle, in relation to the femoral condyles, may also play a role in ACL injury, especially when the lower extremity is in a vulnerable landing position.[1,7,12]

Another explanation of NC-ACLI is that it is the result of a forceful, eccentric quadriceps contraction. During an unplanned or disrupted maneuver, the anterior vector of a forceful quadriceps contraction on a relatively extended

‡Address correspondence to Barry P. Boden, MD, c/o Elaine P. Henze, BJ, ELS, Medical Editor and Director, Editorial Services, Department of Orthopaedic Surgery, Johns Hopkins Bayview Medical Center, 4940 Eastern Avenue, #A665, Baltimore, MD 21224-2780 (e-mail: ehenze1@jhmi.edu).

*Johns Hopkins International Center for Orthopaedic Advancement, Baltimore, Maryland.

†The Orthopaedic Center, Rockville, Maryland.

The authors declared that they have no conflicts of interest in the authorship and publication of this contribution.

The American Journal of Sports Medicine, Vol. 40, No. 3
DOI: 10.1177/0363546511430204

knee has been postulated to result in an anterior force on the tibia, straining and potentially tearing the ACL.[7] However, the 1 study to date that showed rupture of the ACL with isolated quadriceps forces produced failure in only 6 of 11 cadaveric specimens.[7]

To our knowledge, no studies have attempted to elucidate the role of a forceful quadriceps contraction in the axially loaded knee or to determine the relative contribution of each toward the injury mechanism. The goal of the current study was to compare the axial compressive load required to damage the ACL with and without the presence of a simulated forceful quadriceps contraction. We hypothesized that the introduction of a quadriceps force lowers the axial compressive force threshold required to produce ACL injury.

MATERIALS AND METHODS

Specimen Preparation

Six pairs of fresh-frozen cadaveric knees (donor age range, 47-59 years) were acquired from the Maryland State Anatomy Board and underwent dual-energy x-ray absorptiometry scanning of the proximal femur to rule out osteoporosis. After thawing at room temperature, each knee was examined clinically by an orthopaedic surgeon for ligamentous integrity by means of an anterior drawer test; all were deemed stable. The specimens were then sectioned through the tibia and fibula 15 cm distal to the joint line and through the femur 20 cm proximal to the joint line. All skin and muscle tissue was removed from the specimens with careful dissection, leaving intact all the static constraints of the knee and the extensor mechanism. The distal 8 cm of each sectioned tibia and the proximal 8 cm of each sectioned femur were cleaned thoroughly. The tibia and fibula were then potted in 3-inch- and 2-inch-diameter polyvinyl chloride pots, respectively, with dental acrylic.

Each polyvinyl chloride pot was inserted into a corresponding aluminum pot on the custom-designed and -built testing rig (Figure 1). The femoral pot was attached directly to an MTS actuator (Model 858, MTS Corp, Eden Prairie, Minnesota) and fixed at an angle of 15° with respect to the actuator. The tibial pot was fixed parallel to the actuator in a cradle that allowed it to rotate freely about its long axis and was mounted on an X-Y table on the MTS table. The knees were therefore positioned in approximately 15° of flexion. We chose this knee flexion angle to re-create the "vulnerable knee position" documented in a previous video analysis study.[4]

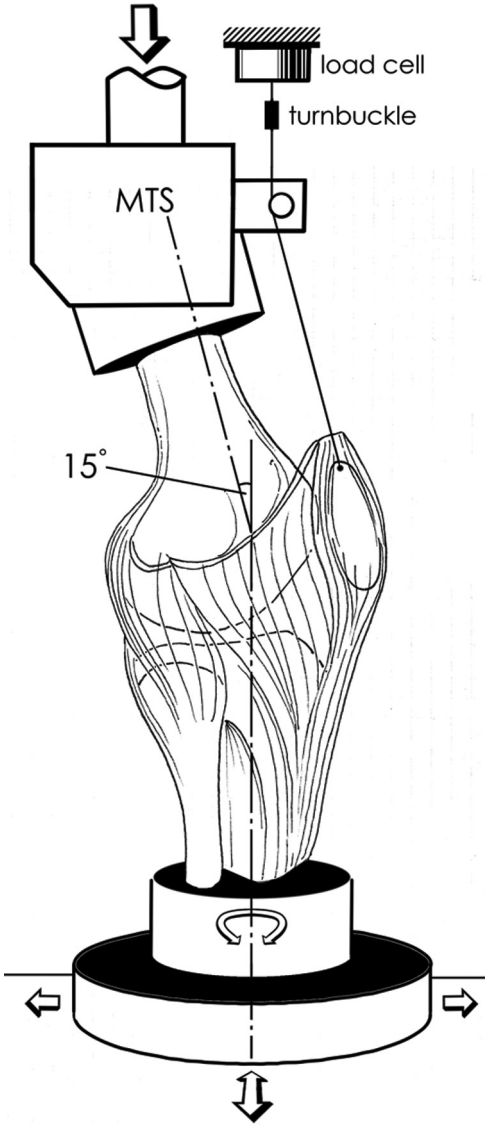

Figure 1. Each knee specimen was potted in polyvinyl chloride pipes and placed in 15° of flexion in a custom-designed fixture on a materials testing machine. A 1500-N simulated quadriceps load was generated by pretensioning a cable attached to the patella, passed over a pulley and connected to a load cell on the crosshead (not shown). The tibia/fibula were potted in polymethylmethacrylate but allowed to rotate about the tibia's long axis and to translate in the horizontal plane. For specimens tested in compression only, the quadriceps cable was not installed through the patella.

Specimen Testing

One of each pair of knees, alternating left and right, was randomly assigned to undergo an isolated axial compressive force (group A), and the contralateral knee was assigned a combined/concurrent simulated isometric quadriceps contraction and axial compressive loading (group B).

In group B, the simulated quadriceps contraction was achieved by looping a length of one-eighth-inch steel cable through the patella via 2 longitudinal drill holes. The cable was linked to a beam on the MTS machine, independent from the actuator, and a load cell was connected that measured the tensile force within the extensor mechanism. A preload of approximately 1500 N of tensile force was then applied statically to the patella immediately before axial loading by means of a turnbuckle linking the extensor mechanism cable to the load cell. We chose 1500 N as the quadriceps force because it approached the reported

TABLE 1
Demographics and Measurements of Groups A and B[a]

Specimen No.	Age, y	T Score	Side	Group	Displacement, mm	Axial Load, N	Quadriceps Load, N	Failure
1	50	0.4	R	B	8	9701	2237	No injury
					10	7171	288	No injury
					12	4962	2573	No injury
					14	4119	3088	ACL tear
		−0.6	L	A	8	8356	NA	No injury
					10	9672	NA	Tibial plateau fracture
2	59	−0.8	L	B	8	3642	3058	Cement settling
						7007	2804	No injury
					10	8382	2818	ACL tear
		−0.4	R	A	8	8175	NA	No injury
					10	10 416	NA	No injury
					12	9724	NA	No injury
					14	9634	NA	ACL tear, plateau fracture
3	50	0.9	R	B	8	3853	2652	No injury
					10	5157	2491	No injury
					12	6402	2622	ACL tear
		1	L	A	8	11 514	NA	No injury
					10	12 689	NA	ACL tear, fracture
4	54	−0.2	L	B	8	7538	2370	Tear of posterior-lateral bundle of ACL
		−0.6	R	A	8	11 154	NA	Lateral tibial plateau fracture
5	51	0.7	L	B	8	8517	2210	No injury
					10	2820	1947	Lateral plateau fracture
		1	R	A	8	7386	NA	No injury
					10	10 376	NA	No injury
					12	11 329	NA	Tibial spine avulsion
6	47	0.7	R	B	8	5316	2631	No injury
					10	3749	3169	No injury
					12	7453	2676	Tear of posterior-lateral bundle of ACL
		0	L	A	8	6580	NA	No injury
					10	9505	NA	No injury
					12	10 716	NA	No injury
					14	10 514	NA	Tear of posterior-lateral bundle of ACL

[a]R, right; L, left; ACL, anterior cruciate ligament; NA, not applicable.

voluntary contraction force (3000 N) of the quadriceps in 15° of extension[18] during testing, as determined during pilot work, but did not approach the overload capacity of the cell. Tensioning of the extensor mechanism was performed immediately before the axial load application to reduce the effect of viscoelastic creep on the extensor mechanism. The ultimate "quadriceps force" delivered to the knee was consistently greater than 1500 N because of the independent relationship of the quadriceps force anchor point to the MTS actuator. As the MTS actuator was advanced, the tension in the extensor mechanism increased, simulating a rapid and forceful quadriceps contraction.

All specimens underwent axial compressive loading by displacing the MTS linear actuator under displacement control over a period of 50 msec to simulate the time to peak of the ground-reaction force during a typical jump landing.[16] The initial displacement of the MTS actuator was 8 mm. If the force-displacement curve suggested damage, that is, an increased displacement with a sudden decrease in load, then the specimen was removed and subjected to an anterior drawer test and inspected for fractures under fluoroscopy. If no failure occurred, the displacement was increased by 2-mm increments with each subsequent test until catastrophic damage of the joint occurred. Once the specimen failed, specific injuries were confirmed by dissection and direct visualization and were photographed.

Statistical Analysis

The difference in axial compressive load needed to cause injury to the ACL between groups was analyzed for significance ($P < .05$) using a paired t test (Stata 10, Stata Corp, College Station, Texas).

RESULTS

There was a significant difference in failure force between groups A and B (Table 1). For group A specimens (isolated axial force), the mean failure load was 10 832 N (95% confidence interval [CI], 9743-11 604 N), and failure occurred

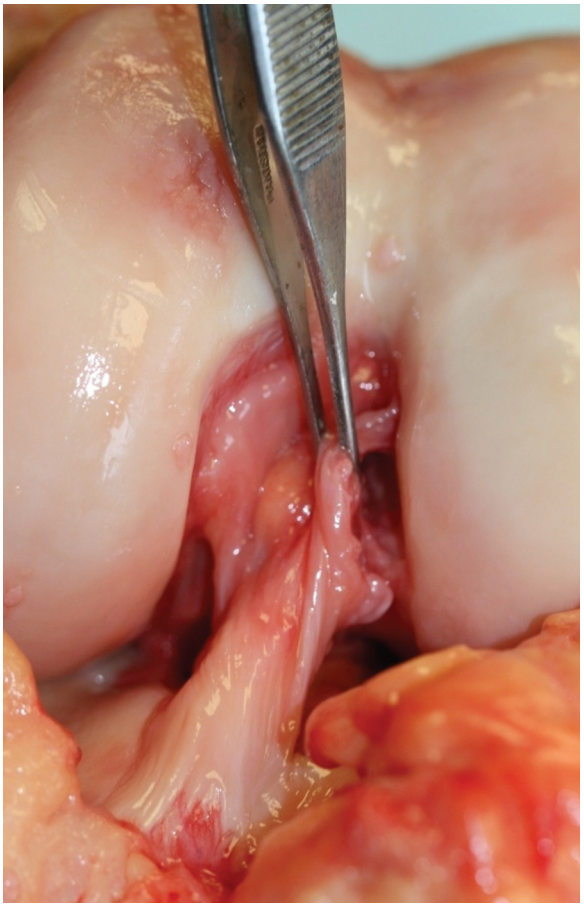

Figure 2. After the specimen failed, the knee was exposed, and direct visualization indicated a midsubstance anterior cruciate ligament rupture.

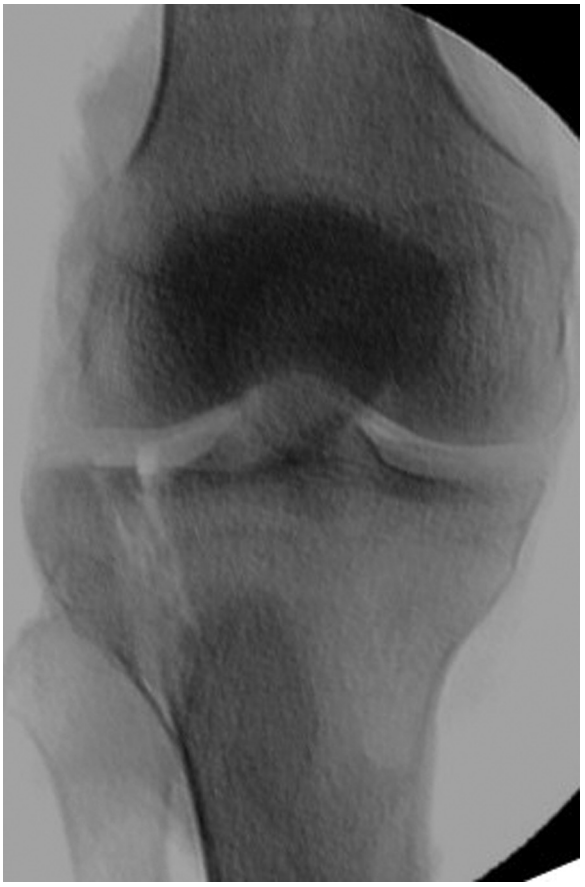

Figure 3. A fluoroscopy image taken after the axial loading–only test shows a tibial plateau fracture.

as a result of ACL rupture (1 knee) (Figure 2), combined tibial plateau fracture and ACL rupture (2 knees), isolated tibial plateau fracture (2 knees) (Figure 3), and ACL tibial spine avulsion (1 knee).

For group B specimens (axial and quadriceps force), the mean axial load at failure was 6119 N (95% CI, 4335-7903 N). The mean quadriceps load at failure was 2587 N (95% CI, 2175-2999 N) (Figure 4). Of the 6 specimens, 5 failed by ACL rupture and 1 by tibial plateau fracture.

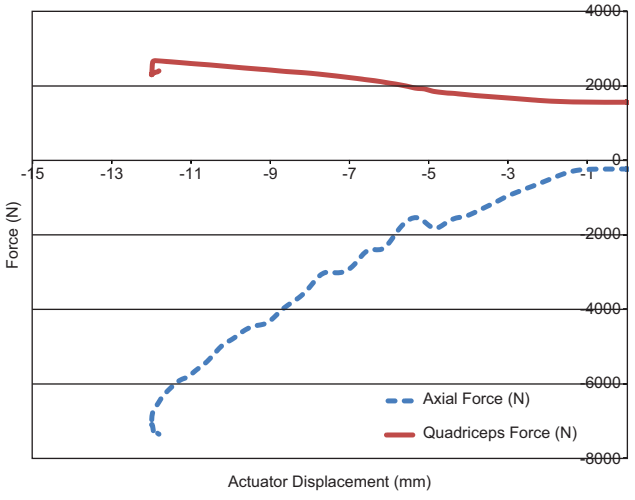

Figure 4. Typical force versus actuator (axial) displacement readout for an axial displacement (12 mm) with quadriceps tendon force test. The quadriceps tendon preload was approximately 1500 N and rose to 3000 N during testing. Peak axial joint reaction force was approximately 7000 N.

DISCUSSION

Although the results of our model support the reports that an ACL injury can be created by an axial compressive load in a knee that is close to full extension,[12,13] they also suggest that ACL injury under compressive load alone may occur in conjunction with a fracture. We had only 2 isolated ACL injuries in group A (1 via tibial spine avulsion); the remaining 2 ACL injuries were in conjunction with a tibial plateau fracture. We concur with Meyer et al[12,13] that excessive joint compressive forces can result in complete

ACL rupture in human cadaveric knees. Our compressive forces (9743-11 604 N) were higher than those found in the study by Meyer et al (2900-7800 N),[12] which may be the result of the 5-kg weight that they attached to the femur via a pulley to apply a slight posterior preload. This posterior load transmitted shear force across the knee joint and may have functioned in a manner similar to the shear component of the quadriceps load in our study by lowering the compressive axial force needed to injure the ACL. Compressive force differences between the studies may also be attributed to specimen quality. Our specimens all had normal bone mineral density, whereas the bone mineral density of the specimens in the study by Meyer et al[12] was not measured. Our compressive forces were also higher than those found in porcine knees flexed to 70° (1812-2659 N).[19] It is difficult to reconcile cross-species differences. Maximum peak vertical ground-reaction forces experienced by 1-legged landings after jumping maneuvers have been estimated to range from 2 to 18 times the body weight.[11] The compressive loads reported for group A would have to be at the high end of this physiological spectrum to produce injury. It is difficult to determine how much lower the compressive forces to produce ACL injury would have been if hip flexion and a higher posterior tibial slope were taken into account.

Several authors have shown that an axial weightbearing compressive force combined with the posterior slope of the tibial plateau can produce an anterior tibial force in knees with intact or deficient ACLs.[6,17] In a videotape analysis of ACL-injured and control athletes, Boden et al[4] showed that injured athletes first contacted the ground with the hindfoot or entirely flat-footed compared with initial contact of the forefoot in controls. There was minimal change in the average ankle position of the injured athletes (4.1°; range, 10.70° to –6.6°) compared with that of controls (43.8°; range, 22.9° to –20.9°) over the first 5 frames (30-Hz frames/s).[4] These findings indicate that the calf muscles are not functioning properly to absorb the ground-reaction forces. Additionally, the injured athletes attained the flat-foot position substantially sooner than the controls.[4] The shorter time span reduces the ability of the calf muscles to contract and absorb forces, resulting in increased impulsive forces at the knee.[3] On the basis of these findings, the authors of this study, as well as others, have concluded that axial compressive forces are a critical component of NC-ACLI.[4,8,19]

Our results confirmed the hypothesis that the introduction of a quadriceps force lowers the axial force threshold necessary to generate NC-ACLI. That 5 of 6 specimens in group B failed with an isolated ACL injury suggests that the contribution of the quadriceps force results in a more clinically relevant injury pattern than the axial compressive load alone (group A). The quadriceps has been reported to be an antagonist of the ACL and may contribute to NC-ACLI during a forceful eccentric contraction at the point of initial ground contact.[5,7,9,15] Previous reports have postulated that the anterior vector of the quadriceps (as applied to the tibia through the patellar tendon) is the primary contributing force to ACL injury.[5,7,10] Because the angle of NC-ACLI has been reported to occur between 10° and 30° of knee flexion, the compressive component of the quadriceps reaction force is larger than the anterior shear component applied through the patellar tendon. In one biomechanical study, the quadriceps compressive force was estimated to be 2 to 3 times greater than the anterior shear force at knee flexion angles of 15°.[14]

As the knee is flexed, the anterior shear forces decrease while the compressive forces stay relatively constant and equivalent to the total patellar tendon force.[14] Because the anterior shear force is a small component of the quadriceps contraction, it is likely insufficient to cause ACL injury. Rupture of the ACL with isolated quadriceps force has been produced in a previous cadaveric study, but the quadriceps force used was high (4500 N). van Eijden et al[18] reported that the maximum voluntary quadriceps contraction at 15° of knee flexion is close to 3000 N. It is difficult to envision a scenario in which the quadriceps would be contracted to such a magnitude in isolation of other muscles to generate the shear component of 2160 N reportedly needed to damage the ACL.

The quadriceps may contribute to NC-ACLI by more than just the compressive and anterior forces on the tibia. It is possible that instead of contracting eccentrically, the quadriceps contraction has a temporary concentric/isometric component that limits and/or prevents knee flexion.[4] In the dangerous landing position, hip flexion causes the center of mass to be posterior to the foot.[4] Therefore, to prevent a backward fall, the quadriceps may need to contract concentrically/isometrically and allow the athlete to center the trunk over the leg.

One limitation of our study is that several of the cadaveric specimens, especially in group A, failed by fracture or ACL injury combined with a fracture instead of isolated ACL injury. The likely explanation is that a pure axial compressive force without a concomitant quadriceps contraction does not perfectly replicate the injury situation. The specimens in this study, although in the normal range, had bone mineral density values on the lower end of those expected for the young active population at risk and therefore may have fractured instead of sustaining isolated ACL injuries. It is also likely that the position of the leg and/or application of forces did not exactly reproduce the NC-ACLI mechanism.

Our current study did not include hip flexion, which may have enhanced the risk of ACL injury versus fracture. Hip flexion effectively increases the posterior tibial slope, making it easier for the lateral femoral condyle to slide (pivot shift) posteriorly on the tibial plateau and rupture the ACL with a compressive force.[1] Doing so would have theoretically lowered the compressive forces to failure and increased the chances of an ACL injury over a tibial plateau fracture.

The study is also limited because it did not account for the full dynamic situation at the knee, such as the effect of hamstring and/or gastrocnemius contraction or valgus forces, especially in female athletes. Because of the complexity of setting up a cadaveric study with multiple muscle forces and varying abduction moments, we decided to focus on the axial and quadriceps components of the injury. Our quadriceps contraction was simulated with a 1500-N preload and then isometric contraction during the test

while we recorded the resulting quadriceps load. It is unlikely that this scenario exactly replicates the in vivo situation; however, lacking definitive data on the quadriceps load magnitude and rate, we assumed the previously mentioned load and documented the outcome. It is unknown what effect the repetitive nature of the tests had on our specimens. It is possible that microdamage accumulation may have occurred in soft tissue structures, for example, the ACL, and cartilage fissures or subchondral bone that would otherwise not have been the case if a single-load application had been conducted. We chose to conduct displacement controlled tests of increasing magnitude because conducting load-controlled compression tests at such high loading rates would have placed our testing equipment at undue risk of damage. It is also unknown what effect removing the specimen from the fixture to conduct drawer tests had on subsequent loading, although every effort was made to return the specimen to its previous position. Alternatively, the current data may provide conservative force estimates for injury to the ACL.

In summary, our results have shown that an axial compressive force to the knee can produce disruption of the ACL complex, but it often occurs in conjunction with a fracture. Addition of a quadriceps force significantly lowers the axial compressive force threshold needed to disrupt the ACL. Thus, forces sustained while landing from a jump are sufficient to cause injury to the ACL, but in the presence of quadriceps contraction, the injury threshold is significantly reduced.

REFERENCES

1. Boden BP, Breit I, Sheehan FT. Tibiofemoral alignment: contributing factors to noncontact anterior cruciate ligament injury. *J Bone Joint Surg Am*. 2009;91(10):2381-2389.
2. Boden BP, Dean GS, Feagin JA Jr, Garrett WE Jr. Mechanisms of anterior cruciate ligament injury. *Orthopedics*. 2000;23(6):573-578.
3. Boden BP, Sheehan FT, Torg JS, Hewett TE. Noncontact anterior cruciate ligament injuries: mechanisms and risk factors. *J Am Acad Orthop Surg*. 2010;18(9):520-527.
4. Boden BP, Torg JS, Knowles SB, Hewett TE. Video analysis of anterior cruciate ligament injury: abnormalities in hip and ankle kinematics. *Am J Sports Med*. 2009;37(2):252-259.
5. Chappell JD, Creighton RA, Giuliani C, Yu B, Garrett WE. Kinematics and electromyography of landing preparation in vertical stop-jump: risks for noncontact anterior cruciate ligament injury. *Am J Sports Med*. 2007;35(2):235-241.
6. Dejour H, Bonnin M. Tibial translation after anterior cruciate ligament rupture: two radiological tests compared. *J Bone Joint Surg Br*. 1994;76(5):745-749.
7. DeMorat G, Weinhold P, Blackburn T, Chudik S, Garrett W. Aggressive quadriceps loading can induce noncontact anterior cruciate ligament injury. *Am J Sports Med*. 2004;32(2):477-483.
8. Hsu V, Stearne D, Torg J. Elastic instability, columnar buckling, and non-contact anterior cruciate ligament ruptures: a preliminary report. *Temple Univ J Orthop Surg Sports Med*. 2006;1(1):21-23.
9. Li G, Rudy TW, Allen C, Sakane M, Woo SLY. Effect of combined axial compressive and anterior tibial loads on in situ forces in the anterior cruciate ligament: a porcine study. *J Orthop Res*. 1998;16(1):122-127.
10. Markolf KL, Burchfield DM, Shapiro MM, et al. Combined knee loading states that generate high anterior cruciate ligament forces. *J Orthop Res*. 1995;13(6):930-935.
11. McNitt-Gray JL. Kinetics of the lower extremities during drop landings from three heights. *J Biomech*. 1993;26(9):1037-1046.
12. Meyer EG, Baumer TG, Slade JM, Smith WE, Haut RC. Tibiofemoral contact pressures and osteochondral microtrauma during anterior cruciate ligament rupture due to excessive compressive loading and internal torque of the human knee. *Am J Sports Med*. 2008;36(10):1966-1977.
13. Meyer EG, Haut RC. Excessive compression of the human tibio-femoral joint causes ACL rupture. *J Biomech*. 2005;38(11):2311-2316.
14. Nisell R. Mechanics of the knee: a study of joint and muscle load with clinical applications. *Acta Orthop Scand Suppl*. 1985;56(216):1-42.
15. Pandy MG, Shelburne KB. Dependence of cruciate-ligament loading on muscle forces and external load. *J Biomech*. 1997;30(10):1015-1024.
16. Richards DP, Ajemian SV, Wiley JP, Zernicke RF. Knee joint dynamics predict patellar tendinitis in elite volleyball players. *Am J Sports Med*. 1996;24(5):676-683.
17. Torzilli PA, Deng X, Warren RF. The effect of joint-compressive load and quadriceps muscle force on knee motion in the intact and anterior cruciate ligament-sectioned knee. *Am J Sports Med*. 1994;22(1):105-112.
18. van Eijden TM, Weijs WA, Kouwenhoven E, Verburg J. Forces acting on the patella during maximal voluntary contraction of the quadriceps femoris muscle at different knee flexion/extension angles. *Acta Anat (Basel)*. 1987;129(4):310-314.
19. Yeow CH, Cheong CH, Ng KS, Lee PVS, Goh JCH. Anterior cruciate ligament failure and cartilage damage during knee joint compression: a preliminary study based on the porcine model. *Am J Sports Med*. 2008;36(5):934-942.