Nonweight-bearing anterior knee laxity is related to anterior tibial translation during transition from nonweight bearing to weight bearing

By: Sandra J. Shultz, Yohei Shimokochi, Anh-Dung Nguyen, Jatin P. Ambegaonkar, Randy J. Schmitz, Bruce D. Beynnon and David H. Perrin

Shultz, S. J., Shimokochi, Y., Nguyen, A.-D., Ambegaonkar, J. P., Schmitz, R. J., Beynnon, B. D. and Perrin, D. H. (2006), Nonweight-bearing anterior knee laxity is related to anterior tibial translation during transition from nonweight bearing to weight bearing. J. Orthop. Res., 24: 516–523.

Abstract:

We examined the relationship between anterior knee laxity (AKL), evaluated while the knee was nonweight bearing, and anterior translation of the tibia relative to the femur (ATT), evaluated when the knee transitioned from nonweight-bearing to weight-bearing conditions in response to an applied compressive load at the foot. Twenty subjects with normal knees (10 M, 10 F; 25.2 ± 4.1 years, 169.8 ± 11.5 cm, 71.6 ± 16.9 kg) underwent measurements of AKL and ATT of the right knee on 2 days. AKL was measured at 133N with the KT-2000™. ATT was measured with the Vermont Knee Laxity Device and electromagnetic position sensors attached to the patella and the anteromedial aspect of the proximal tibia. Three trials for each measure were averaged and analyzed. Measurement consistency was high for both AKL (ICC = 0.97; SEM = 0.44 mm) and ATT (ICC = 0.88; SEM = 0.84 mm). Linear regression revealed that AKL predicted 35.5% of the variance in ATT ($p = 0.006$), with a prediction equation of $Y_{ATT} = 3.20 + 0.543(X_{AKL})$. Our findings suggest that increased AKL is associated with increased ATT as the knee transitions from nonweight-bearing to weight-bearing conditions. The potential for increased knee joint laxity to disrupt normal knee biomechanics during activities such as landing from a jump, or the foot strike phase of gait deserves further study.

Keywords: anterior cruciate ligament | kinematics | anterior–posterior tibial displacement

Article:

INTRODUCTION

Increased anterior knee joint laxity has been identified as a risk factor for anterior cruciate ligament (ACL) injury among females.[1] In a prospective study of nearly 1200 United States Military Academy cadets, generalized joint laxity (both males and females) and anterior knee laxity values that exceeded 1 standard deviation (SD) of the mean (in females) were found to be significant predictors of ACL injury risk.[1] Females with knee laxity values greater than 1 SD of the mean had a relative risk of suffering an ACL injury that was 2.7 higher compared to females with lower knee laxity values. These findings are supported by a retrospective study that identified a relationship between increased anterior knee laxity and ACL injury status,[2] as well as other studies that have reported an increased risk of lower extremity joint injury in lax and hyperlax individuals.[3–6] Although some studies have observed a link between generalized joint laxity with both lower extremity musculoskeletal injury and joint sprains in athletes,[3,6] others have only observed a link with knee and ankle joint sprains without concomitant differences in overall musculoskeletal injury rates.[4,5] Knee laxity, particularly varus-valgus laxity, has also been implicated as a risk factor for knee osteoarthritis, secondary to its effects on the load distribution and stresses placed on the joint.[7]

Although females typically have greater anterior knee joint laxity,[8–10] and are at increased risk of suffering ACL injury[11,12] and osteoarthritis[13,14] compared to males, the specific mechanism(s) by which increased knee joint laxity may modify risk is unknown. Potential mechanisms include reduced proprioceptive sensitivity to joint displacements and loads,[9,15] hormonal influences on ligament behavior and tensile strength,[16–18] and altered knee joint function during weight bearing.[15,19,20] Research indicates that anterior translation of the tibia relative to the femur occurs when transitioning from nonweight bearing to weight bearing with the knee near extension (e.g., 20–308 of flexion),[19,20] which is restrained by the ACL in the normal knee.[19] These findings, combined with evidence of abnormal increases in anterior tibial translation with sectioning of the ACL,[20] indicates the ACL is strained during weight bearing and plays a role in maintaining normal knee biomechanics during activities such as landing from a jump, or the foot strike phase of gait.[21] Hence, subjects that have increased laxity of the nonweight-bearing knee may also experience increased anterior displacement of the tibia relative to the femur as the joint transitions from nonweightbearing to weight-bearing conditions (e.g., at the point of initial ground contact), and this may be associated with altered alignment of the joint surfaces. This may be of particular concern for physically active females who, compared to males, demonstrate quadriceps dominant recruitment strategies[22–24] and greater anterior-directed shear forces[23,25] during deceleration; factors that would further accentuate anterior displacement of the tibia relative to the femur and increase ACL strain.

Anterior–posterior knee laxity is typically measured in the clinic with the subject nonweight bearing using the KT-2000™ or a similar arthrometer device. We are unaware of any literature that has examined whether subjects who have increased knee laxity as measured clinically during nonweight-bearing conditions also experience greater joint displacement upon acceptance of weight-bearing loads. This knowledge is critical to our understanding of joint mechanics upon foot strike, the point in time during most activities when ACL injury is thought to occur.[26] Hence, our purpose was to examine the relationship between anterior knee joint laxity and anterior translation of the tibia relative to the femur when transitioning from nonweight-bearing

to weight-bearing conditions using an applied compressive load to the foot to simulate the foot strike phase of gait. We hypothesized that when subjects transitioned from nonweight bearing to weight bearing via this compressive load, subjects with increased anterior knee laxity would experience greater increases in anterior tibial displacement relative to the femur.

MATERIALS AND METHODS

Twenty healthy subjects, (10 M, 10 F; 25.2 ± 4.1 years, 169.8 ± 11.5 cm, 71.6 ± 16.9 kg) who reported no injury or chronic pain in either lower extremity for the past 6 months, or any history of knee ligament injury or surgery, volunteered to participate. Our sample size of 20 participants was based on previous pilot data and adequate power for a two-predictor regression model. Prior to participating, all subjects were informed of the study and associated risks, and signed a consent form approved by the University's Institutional Review Board.

All testing was performed on the right knee in a laboratory setting, with identical procedures performed on two separate days to confirm measurement reliability. After obtaining the subject's height and weight, a single examiner (Y.S.) with previously established intratester reliability ($ICC_{2,k}$ ¼ 0.85; SEM ¼ 0.70 mm) measured anterior knee laxity (AKL) using the KT 2000™ Knee Arthrometer (MEDmetric® Corp; San Diego, CA). Knee laxity was defined as the amount of anterior tibial displacement at 133 N. As 133 N is the measure most commonly used in clinical practice and research, our desire was to compare this clinical measure with weight-bearing biomechanics. Participants were positioned supine per manufacturer's guidelines with the thigh supported just proximal to the popliteal fossa, the knees flexed to 25°, and the ankles placed in the manufacturer provided foot cradle. A Velcro strap placed around the subject's thighs minimized rotation of the lower extremity. The KT-2000™ was then attached to the leg in proper alignment with the joint line of the knee. With the participant relaxed, three anterior to posterior directed forces were applied to the anterior aspect of the tibia to identify a stable neutral point, followed by an anterior directed force just over 133 N to measure anterior tibial displacement in millimeters (mm). A bubble level fixed to the device ensured a direct anterior pull was achieved. Three trials were recorded.

Anterior tibial translation (ATT) was measured with the Vermont Knee Laxity Device (VKLD; University of Vermont, Burlington, VT). The subject was positioned in the VKLD with the knee unweighted, and a counterweight system was used to offset the gravity loads acting on the lower extremity, creating an initial zero shear load across the joint. The VKLD was then used to measure displacement of the tibia relative to the femur as the knee transitioned from nonweight-bearing to weight-bearing conditions (Fig. 1).[27] Subjects were positioned in the VKLD with the right foot strapped to the foot plate, the second metatarsal visually aligned with the anterior superior iliac spine (ASIS), and the anatomical flexion axes of the ankle and hip joints aligned with the mechanical axes of rotation of the VKLD counterweight system. Once properly positioned in the VKLD with the foot cradle in the locked position, the thigh and leg counterweights were applied to eliminate the posterior directed gravity forces acting on the thigh and shank thus creating the absolute zero shear load across the tibiofemoral joint. The thigh and shank counterweights, and their respective locations were selected by using the model of Zatsiorsky[28] to estimate segment masses and center of mass locations using the approach that has been previously reported.[27]

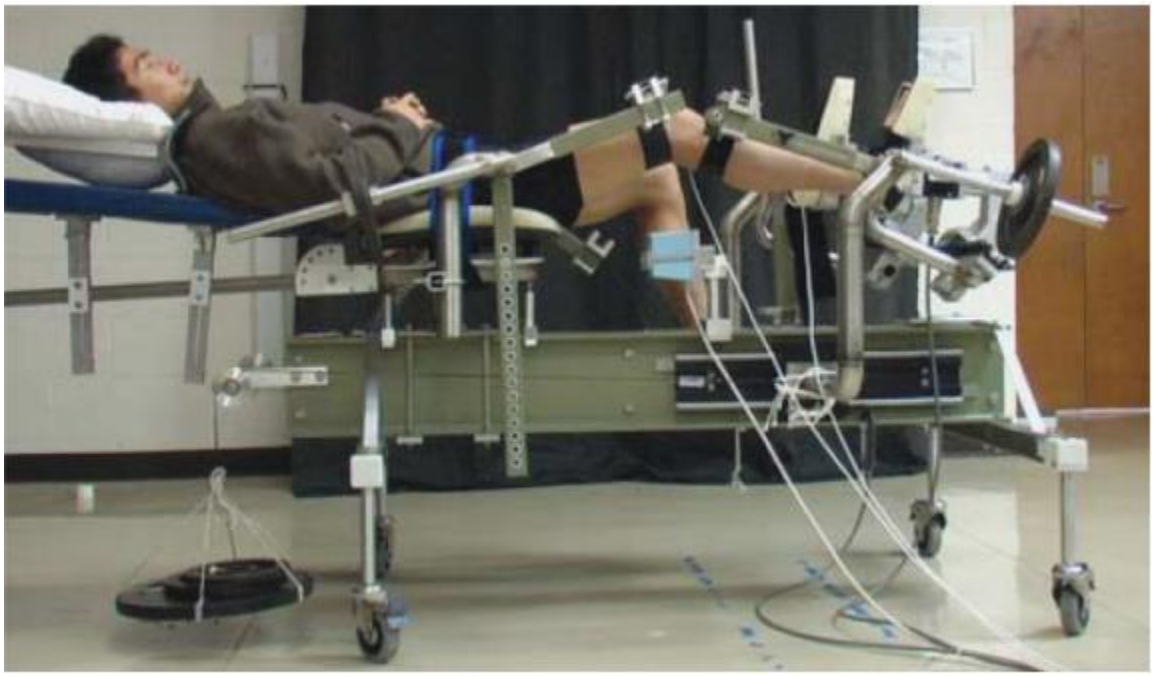

Figure 1. The Vermont knee laxity device.

Prior to data collection, electromagnetic position sensors (Mini Birds, Ascension Technologies, Colchester, VT) were securely strapped to the patella and the anteromedial aspect of the proximal tibia using the same approach previously reported.[27]The center of rotation of the knee and ankle joints were estimated using the centroid method that calculated the midpoint between the most medial and most lateral aspects of the knee joint lines and the medial and lateral malleoli, respectively. The hip joint center was also estimated using the centroid method, calculating the midpoint of a line defined anteriorly by a point placed medially from the ASIS greater trochanter midpoint at a distance equal to half the ASIS greater trochanter line distance, and defined posteriorly by a point placed posteriorly from the ASIS greater trochanter midpoint at a distance equal to the ASIS greater trochanter line distance. Pilot data supported the use of the digitization method to compute the hip joint center, and provided valid position data for knee flexion angle as verified by a standard, hand-held goniometer.

Following digitization of joint centers, the ankle and knee were flexed to 90°and 20°, respectively, and the subjects were instructed to relax their leg muscles. Three anterior to posterior forces were manually applied to the tibia just below the joint line to identify a reproducible neutral point. (Identifying a stable, reproducible neutral point with this procedure was confirmed with pilot testing and the collection of baseline data at the start of each trial.) Knee flexion angle was then confirmed (within ± 5°) at the start of each trial with both a hand-held goniometer and real-time knee flexion angle data from the position sensors. This approach allowed us to establish a resultant zero shear load across the tibiofemoral joint, and create the same initial conditions for the measurement of anterior tibial translation for all subjects. A six degree-of-freedom load transducer (Model MC3A, Advanced Medical Technology, Inc; Watertown, MA) located at the foot ensured that an initial zero compressive load was applied to the tibia. Once the zero shear and compressive loads were achieved, the foot cradle was

unlocked, and a compressive force equal to 40% of the subject's bodyweight was applied such that it acted through the ankle and hip axes. A compressive force equal to 40% of bodyweight was chosen to produce the loading condition experienced during double leg stance (assuming 50% of bodyweight applied to each leg, and 10% of bodyweight distributed below the knee). The tester did not indicate when the weight would be released and subjects were instructed to (1) not anticipate the release of the foot cradle, and (2) attempt to maintain the same knee position (20° knee flexion) upon joint loading. Following two to three practice trials, three cycles transitioning from nonweight bearing to weight bearing were completed for each subject.

Position data were collected at 100 Hz from the electromagnetic tracking system. The VKLD was developed to minimize the amount of metal that could potentially interfere with the signal from the sensors. Using commercially available software (Motion Monitor, Innovative Sports Training; Chicago, IL) we con- firmed there was no signal distortion, then low-pass filtered the raw position data at 10 Hz using a fourthorder zero lag Butterworth filter. A segmental reference system quantified the three-dimensional kinematics of the knee during the transition from nonweight bearing to weight bearing. For each segment the +Z axis was directed laterally, the +Y axis was directed superiorly, and the +X axis was directed anteriorly. Euler's equations were used to describe joint motion about the knee with a rotational sequence of Z Y' X".[29] ATT was calculated as the displacement of the tibia with respect to the femur in the A-P plane. Peak ATT was defined as the initial peak value when transitioning from nonweight bearing to weight bearing (Fig. 2). Knee flexion angles were obtained at the start of the trial and at peak ATT to quantify any change in knee flexion upon weight acceptance. We felt it was important to document the knee flexion change, as this could potentially change the relative orientation between the sensors in the A-P plane, and underestimate the measurement of ATT.

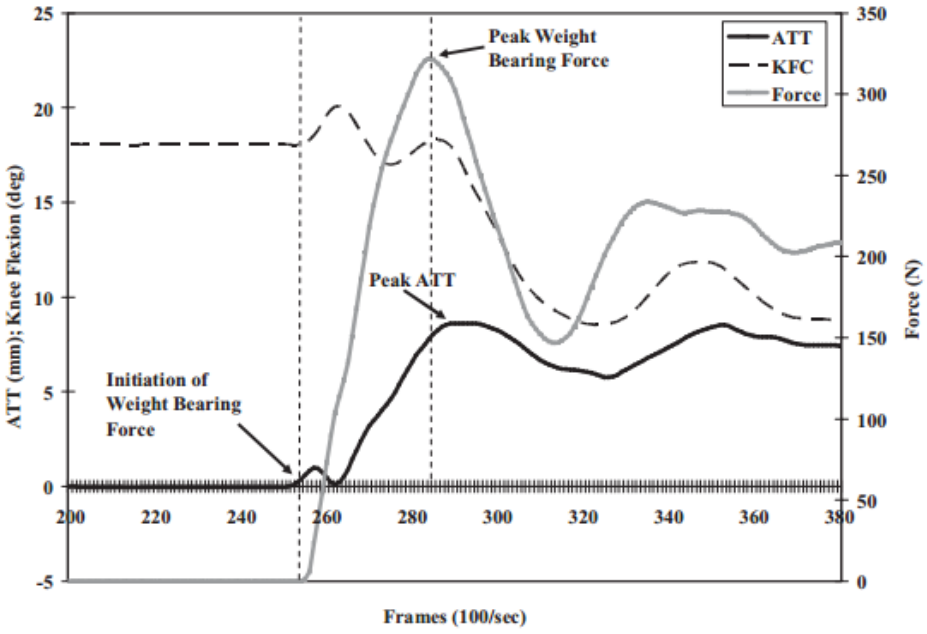

Figure 2. Sample graph showing anterior translation of the tibia relative to the femur (ATT) and knee flexion change (KFC) when transitioning from nonweight-bearing to weight-bearing conditions.

Three trials for both ATT measured by the VKLD and AKL measured by the KT-2000^{TM} were averaged and used for analysis. Repeated-measures ANOVA were used to calculate the intraclass correlation coefficients ($ICC_{2,k}$) and standard error of measurements (SEM) to confirm measurement consistency across repeat test days for AKL and ATT. Reliability coefficients were calculated to demonstrate the investigators could obtain consistent and precise values with both the KT-2000 and VKLD, and ensure that substantial measurement error did not confound our ability to identify a relationship between AKL and ATT. Linear regression was used to examine the relationship between AKL (predictor variable) and ATT (dependent variable) while also accounting for any change in knee flexion angle (suppressor variable). An experiment-wise type I error rate of 0.05 was used.

RESULTS

Table 1 reports the means ± standard deviations and reliability results for ATT and AKL. Day-today measurement consistency was high for both AKL (ICC = 0.97; SEM = 0.44 mm) and ATT (ICC = 0.88; SEM = 0.84 mm). Evaluation of knee flexion angle during the measurement of ATT confirmed that subjects began each trial at or near 208 of knee flexion (19.4 ± 2.1°), and flexed their knees an average of 7.1 ± 4.18 from the start of the trial to peak ATT. Linear regression revealed that AKL predicted 35.5% of the variance in ATT with a prediction equation of $Y_{ATT} = 3.20 + 0.543(X_{AKL})$ (Table 2). This equation indicates that the intercept (constant) of 3.20 mm is the value of ATT if AKL is zero, and the slope of the line is such that for every 1 mm increase in AKL, there is an approximate 0.5 mm increase in ATT. The change in knee flexion (KFC) explained an additional 10.1% of the variance in ATT, but the *F* change was not significant. Further, knee flexion had minimal impact on the relationship between AKL and ATT. This was evidenced by only small increases in the partial correlation between AKL and ATT ($r_{partial} = 0.596$ vs. 0.645) and the prediction coefficient for AKL (0.543 vs. 0.571) once the variance explained by knee flexion was accounted for.

Table 1. Means ± SD, Correlation Coefficients ($ICC_{2,k}$) and Standard Error of Measurement (SEM) for Anterior Knee Laxity (AKL) Measured with the KT2000^{TM} and Anterior Tibial Translation (ATT) Measured with the VKLD

Measure	Day 1	Day 2	$ICC_{2,k}$	SEM
AKL (mm)	7.5 ± 2.6	7.6 ± 2.3	0.97	0.44
ATT (mm)	7.3 ± 2.4	6.6 ± 2.3	0.88	0.84

Table 2. Model Summary for Linear Regression Analysis

					Change Statistics				
Model	*R*	R^2	Adjusted R^2	Std. Error of the Estimate	R^2 Change	*F* Change	*df*1	*df*2	Sig. *F* Change
1	0.596	0.355	0.319	1.970	0.355	9.903	1	18	0.006
2	0.676	0.456	0.393	1.865	0.102	3.177	1	17	0.093

1 Predictors: (Constant), AKL
2 Predictors: (Constant), AKL, FLC

DISCUSSION

Our primary finding was that increased anterior knee joint laxity, measured with the knee nonweight bearing, was able to explain approximately 36% of the increase in anterior displacement of the tibia relative to the femur as the knee transitioned from nonweight-bearing to weight-bearing conditions via an applied compressive load to the foot. Based on the suggested guidelines for interpreting small, medium, and large effect sizes, an R^2 value of 0.25 (R-value of 0.50) is considered a large effect, which suggests the relationship between AKL and ATT is relatively strong.[30] This is noteworthy, given the substantial differences in how the two measures were obtained. AKL was measured with the subject supine, with instructions to relax the thigh musculature. The ATT measurement, on the other hand, is a more dynamic measurement, requiring the subject to activate their muscles to maintain their knee at 20° of flexion upon weight acceptance.

Even with the moderately strong relationship noted, approximately 65% of the variance in ATT remains unexplained by AKL. Hence, other factors likely contributed to the variance in anterior tibial translation with an applied compressive load, including changes in knee flexion angle, joint geometry (e.g., the slope of the tibial plateau), joint congruency, and muscle coactivation levels. Although we carefully controlled knee flexion angle, there was some variability between subjects, and knee flexion typically increased somewhat once the weight bearing load was released (7.1 ± 4.1°; range 1.0–14.7°). We chose to account for this variable in the regression model to determine if this contributed to measurement error and suppressed the relationship between AKL and ATT. The regression equation revealed the change in knee flexion had little influence on the data and the ability of AKL to predict ATT within the flexion range measured. Further, post hoc analysis determined that a 10° increase in knee flexion would be required to underestimate the ATT measure by approximately 0.5 mm, which is within the measurement error of the VKLD (Table 1).

Relative muscle cocontraction likely had a greater influence on the unexplained variance in ATT. Given the dynamic nature of the task, and the instructions to maintain the knee at the 208 flexed position during load acceptance, some level of muscle activation was required to stiffen the joint upon loading, and may have modified the amount of ATT that was observed across subjects. Not accounting for this variable in our prediction model assumes that subjects used similar muscle activation strategies to stabilize the knee when transitioning from nonweightbearing to weight-bearing postures. Because we did not measure muscle activity, or the geometry of the thigh muscles, we are unable to examine the influence of neuromuscular parameters on intersegmental forces across the knee joint, and the extent to which these factors may modify the relationship between AKL and ATT. However, even with this factor unexplained, the regression equation indicates a relatively strong relationship between AKL and ATT, yielding an approximate 2:1 ratio between increases in AKL and ATT. Future work accounting for the combined influence of knee flexion, neuromuscular parameters, and joint geometry should further clarify the strength of the relationship between AKL and ATT.

Although increased joint laxity has been identified as a predictor of increased ACL injury risk in women,[1] the mechanism for this increased risk has received little attention. Our findings support that this relationship may in part be a biomechanical phenomenon, potentially leading to joint

instability or incongruity. When examining studies of ACL injury mechanisms, they typically describe an injury that occurs at or near foot strike with the knee near full extension.[26,31,32] As previously discussed, when compressive loads (simulating weight bearing) are applied to the knee joint near full extension (15–308 flexion), there is an anterior shift of the tibia relative to the femur,[19,20] which is restrained by the ACL.[21,33] This suggests that the ACL becomes strained during the transition from nonweight bearing to weight bearing, and may play an integral role in positioning the knee and resisting excessive joint motion during activities such as landing and foot strike during plant and pivot maneuvers. The current findings suggest that greater tibiofemoral joint displacement may occur upon foot contact in individuals with increased anterior knee laxity. Whether this modest increase in joint displacement in an otherwise healthy knee is sufficient to disrupt normal joint biomechanics and increase risk of ligament injury requires further study. However, limited studies examining neuromuscular strategies in females with increased knee laxity indicate this displacement may place greater demands on the neuromuscular system to actively stabilize the joint.[9,15]

Rozzi et al.[9] examined neuromuscular control in healthy male and female collegiate basketball and soccer athletes. Compared to males, females had greater knee laxity (mean difference of ~1.25 mm) and increased lateral hamstring activity when landing from a jump. They proposed that increased hamstring activity was an active strategy to stabilize the knee in the presence of reduced passive (i.e., capsuloligamentous) stability. In other work comparing neuromuscular control strategies in females with above-average and below-average knee laxity, females with increased knee laxity demonstrated approximately 20% higher activation levels of the bicep femoris both prior to (i.e., while standing on a single leg at 30° of knee flexion) and following a forward and rotational perturbation of the trunk and femur on the weight bearing tibia.[15] Medial and lateral gastrocnemius activation was also found to be approximately 10% higher prior to the perturbation. Studies examining both neuromuscular and biomechanical function are needed to clarify the link between anterior knee laxity, weight-bearing joint mechanics, and neuromuscular control strategies to determine whether these neuromuscular strategies serve to effectively stabilize the tibia during dynamic loading conditions.

The measurement of anterior knee joint laxity is commonly performed by clinicians, and requires minimal time and equipment. Hence, this method would be preferred over more sophisticated biomechanical analyses to identify those at risk for knee joint trauma during weight-bearing activity. Our objective for this study was to provide a sufficient level of experimental control and measurement precision to determine the link between a simple, clinical (nonweight bearing) measure of knee joint laxity and knee joint mechanics upon weight acceptance of a healthy limb in the anterior–posterior plane. One limitation of the study is that it simulated what occurs during a standing posture, with the subject in a supine position and the load applied in a single axial plane through the hip and knee axes of rotation. It may be that the muscular strategies that subjects used to maintain their knee joint in a fixed position upon weight acceptance in the current study were different compared to what occurs during upright weight bearing. Challenges that limit measurement precision during upright weight bearing include the ability to control the knee angle upon weight acceptance, trunk position relative to the knee, tibiofemoral shear loads prior to foot contact, and center of mass position over the foot. Hence, this model offers the best alternative to test our hypothesis by: (1) establishing a repeatable, initial zero-shear load reference position of the tibia relative to the femur through counterbalanced weighting, and (2)

applying an axial compression load that acts through the axes of rotation of the ankle and hip joints. This study was also limited to examination of the healthy knee in a relatively homogenous age group (range 18–31 years), and it is unknown whether similar relationships would be observed in an injured knee (e.g., ACL deficient or reconstructed) or other age populations (e.g., adolescent, older adults). Further, the sample size was not powered to examine sex differences, and future work is needed to determine if the prediction of ATT by AKL is similar for males and females.

Further research is needed to clarify the relationship between increased knee laxity (as measured during nonweight bearing), weightbearing knee joint neuromechanics, and increased risk of knee joint trauma. In addition to future directions previously identified, future work should explore the impact of joint laxity in frontal and transverse planes of motion. Although the current findings are limited to the anterior– posterior plane, there is evidence that healthy, young adult females who have increased anterior knee laxity also have increased valgus knee laxity compared to males.[7,34] Although largely studied as a risk factor for osteoarthritis rather than ACL injury risk,[35,36] increased valgus laxity has the potential to disrupt joint mechanics in the frontal plane, and together with anterior knee laxity impact transverse plane knee motion.[37] Additionally, examining the impact of cyclic increases in knee laxity across the menstrual cycle[38–40] would serve to further clarify the relationship between sex, knee joint laxity, and weight-bearing knee joint neuromechanics.

ACKNOWLEDGMENTS

Support was provided through an internal grant from the University of North Carolina at Greensboro. The authors thank Kathy Coughlin, research engineer in the Department of Orthopaedic Rehabilitation at the University of Vermont, for her technical assistance.

REFERENCES

1. Uhorchak JM, Scoville CR, Williams GN, et al. 2003. Risk factors associated with non-contact injury of the anterior cruciate ligament. Am J Sports Med 31:831–842.

2. Woodford-Rogers B, Cyphert L, Denegar CR. 1994. Risk factors for anterior cruciate ligament injury in high school and college athletes. J Athl Train 29: 343–346.

3. Acasuso-Diaz M, Collantes-Esteves E, SanchezGuijo P. 1993. Joint hyperlaxity and musculoligamentous lesions: study of a population of homogeneous age, sex and physical exertion. Br J Rheum 32:120–122.

4. Decoster LC, Bernier JN, Lindsay RH. 1999. Generalized joint hypermobility and its relationship to injury patterns among NCAA lacrosse players. J Athl Train 34:99–105.

5. Grana WA, Moretz JA. 1978. Ligamentous laxity in secondary school athletes. JAMA 240:1975–1976.

6. Nicholas JA. 1970. Injuries to knee ligaments: relationship to looseness and tightness in football players. JAMA 212:2236–2239.

7. Sharma L, Lou C, Felson DT, et al. 1999. Laxity in healthy and osteoarthritic knees. Arthrit Rheum 42:861–870.

8. Rosene JM, Fogarty TD. 1999. Anterior tibial translation in collegiate athletes with normal anterior cruciate ligament integrity. J Athl Train 34:93–98.

9. Rozzi SL, Lephart SM, Gear WS, et al. 1999. Knee joint laxity and neuromuscular characteristics of male and female soccer and basketball players. Am J Sports Med 27:312–319.

10. Shultz SJ, Kirk SE, Sander TC, et al. Sex differences in knee laxity change across the female menstrual cycle. J Sports Med Phys Fit (in press).

11. Arendt E, Dick R. 1995. Knee injury patterns among men and women in collegiate basketball and soccer. Am J Sports Med 23:694–701.

12. Arendt EA, Agel J, Dick R. 1999. Anterior cruciate ligament injury patterns among collegiate men and women. J Athl Train 34:86–92.

13. Felson DT, Naimark A, Anderson J, et al. 1987. The prevalence of knee osteoarthritis in the elderly. Arthritis Rheum 30:914–918.

14. Petersson IF, Jacobsson LTH. 2002. Osteoarthritis of the peripheral joints. Best Prac Res Clin Rhematol 16:741–760.

15. Shultz SJ, Carcia CR, Perrin DH. 2004. Knee joint laxity affects muscle activation patterns in the healthy knee. J Electromyogr Kinesiol 14:475–483.

16. Slauterbeck J, Clevenger C, Lundberg W, et al. 1999. Estrogen level alters the failure load of the rabbit anterior cruciate ligament. J Orthop Res 17: 405–408.

17. Slauterbeck JR, Hardy DM. 2001. Sex hormones and knee ligament injuries in female athletes. Am J Med Sci 322:196–199.

18. Wojtys EM, Huston L, Boynton MD, et al. 2002. The effect of menstrual cycle on anterior cruciate ligament in women as determined by hormone levels. Am J Sports Med 30:182–188.

19. Fleming BC, Renstrom PA, Beynnon BD, et al. 2001. The effect of weightbearing and external loading on anterior cruciate ligament strain. J Biomech 34:163–170.

20. Torzilli PA, Deng X, Warren RF. 1994. 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 22: 105–112.

21. Beynnon BD, Fleming BC, Labovitch R, et al. 2002. Chronic anterior cruciate ligament deficiency is associated with increased anterior translation of the tibia during the transition from non-weightbearing to weightbearing. J Orthop Res 20:332–337.

22. Huston LJ, Wojtys EM. 1996. Neuromuscular performance characteristics in elite female athletes. Am J Sports Med 24:427–436.

23. Malinzak RA, Colby SM, Kirkendall DT, et al. 2001. A comparison of knee joint motion patterns between men and women in selected athletic tasks. Clin Biomech 16:438–445.

24. Shultz SJ, Perrin DH, Adams JM. 2001. Neuromuscular response characteristics in men and women after knee perturbation in a single-leg weight-bearing stance. J Athl Train 36:37–43.

25. Chappell JD, Yu B, Kirkendall DT, et al. 2002. A comparison of knee kinetics between male and female recreational athletes in stop-jump tasks. Am J Sports Med 30:261–267.

26. Boden BP, Dean GS, Feagin JA, et al. 2000. Mechanisms of anterior cruciate ligament injury. Orthop 23:573–578.

27. Uh BS, Beynnon BD, Churchill DL, et al. 2001. A new device to measure knee laxity during weight bearing and non-weight bearing conditions. J Orthop Res 19:1185–1191.

28. Zatsiorsky VM, Seluyanov VN, Chugunova LG, editors. 1990. Contemporary problems of biomechanics. Boca Raton, FL: CRC Press.

29. Kadaba MP, Ramakrishnan HK, Wootten ME, et al. 1989. Repeatability of kinematic, kinetic, and electromyographic data in normal adult gait. J Orthop Res 7:849–846.

30. Cohen J. 1988. Statistical power analysis for behavioral sciences. 2nd ed. Hillsdale, NJ: Erlbaum.

31. Griffin LY, Agel J, Albohm MJ, et al. 2000. Noncontact anterior cruciate ligament injuries: risk factors and prevention strategies. J Am Acad Orthop Surg 8:141–150.

32. Olsen O, Myklebust G, Engebretsen L, et al. 2004. Injury mechanisms for anterior cruciate ligament injuries in team handball. Am J Sports Med 32: 1002–1012.

33. Hsieh H-H, Walker PS. 1976, Stabilizing mechanisms of the loaded and unloaded knee joint. J Bone Joint Surg 58-A:87–93.

34. Markolf KL, Graff-Radford A, Amstutz HC. 1978. In vivo knee stability: A quantitative assessment using an instrumented clinical testing apparatus. J Bone Joint Surg 60-A:664–674.

35. Sharma L, Hayes KW, Felson DT, et al. 1999. Does laxity alter the relationship between strength and physical function in knee osteoarthritis? Arthritis Rheum 42:25–32.

36. Sharma L. 2004. The role of proprioceptive deficits, ligamentous laxity, and malalignment in development and progression of knee osteoarthritis. J Rheumatol 31:87–92.

37. Markolf KL, Mensch JS, Amstutz HC. 1976. Stiffness and laxity of the knee: the contributions of the supporting structures. J Bone Joint Surg 58-A:583–595.

38. Deie M, Sakamaki Y, Sumen Y, et al. 2002. Anterior knee laxity in young women varies with their menstrual cycle. Int Orthop 26:154– 156.

39. Heitz NA. 1999. Hormonal changes throughout the menstrual cycle and increased anterior cruciate ligament laxity in females. J Athl Train 343:144– 149.

40. Shultz SJ, Sander TC, Kirk SE, et al. 2004. Relationship between sex hormones and anterior knee laxity across the menstrual cycle. Med Sci Sports Exer 36:1165–1174.