

Anterior Knee Stiffness Changes in Laxity “Responders” Versus “Nonresponders” Across the Menstrual Cycle

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Context: Although changes in anterior knee laxity (AKL) across the menstrual cycle have been reported, the effects of cyclic knee laxity changes on the underlying characteristics of the load-displacement (stiffness) curve generated during anterior loading of the tibia relative to the femur are relatively unknown.

Objective: To describe the anterior load-displacement curve during anterior loading of the tibia relative to the femur using incremental stiffnesses and to compare underlying stiffness measures between days of the cycle when AKL is at its minimum and maximum.

Design: Descriptive laboratory study.

Setting: University laboratory.

Patients or Other Participants: Fifty-seven recreationally active women.

Main Outcome Measure(s): Anterior knee laxity and 6 incremental stiffness measures (N/mm) were obtained with an instrumented knee arthrometer on days 1–6 of menses and days 0–8 postovulation during 2 consecutive menstrual cycles. Participants were then classified in tertiles based on the maximum change (difference between maximum and minimum) in AKL, and incremental stiffness was compared on days of

minimum versus maximum laxity between the lowest (<1.24 mm cyclic laxity change = laxity “nonresponders” [n = 19]) and highest (>1.75 mm cyclic laxity change = laxity “responders” [n = 19]) tertiles.

Results: All participants displayed decreasing stiffness initially (0–20 N > 20–40 N and 40–60 N), followed by incrementally increasing stiffness (40–60 N < 60–80 N < 80–100 N < 100–130 N) ($P \leq .05$). Responders demonstrated decreased stiffness between the days of minimum and maximum AKL at the 10–130-N increment versus the 0–20-N and 20–40-N increments ($P \leq .05$); nonresponders had no change in stiffness.

Conclusions: Participants who experienced larger magnitudes of cyclic changes in AKL also experienced decreases in terminal (100–130 N) stiffness during anterior knee joint loading. Decreases in incremental stiffness at higher anterior directed loads may adversely affect passive restraint systems, resulting in altered arthrokinematics during functional activity.

Key Words: anterior cruciate ligament, arthrometry, biomechanics

Key Points

- The load-displacement curve generated during anterior loading of the tibia relative to the femur demonstrated 4 changes in stiffness during arthrometer testing (0–130 N).
- Larger magnitudes of cyclic change in anterior knee laxity resulted in decreased stiffness only at the higher loading ranges (100–130 N).
- To distinguish knee laxity responders from nonresponders, use of terminal stiffness (100–130 N) may be most appropriate.

Joint stiffness ($\Delta\text{load}/\Delta\text{displacement}$)¹ is the inverse of joint compliance and characterizes deformation of the soft tissue structures connecting one bone to another in response to an applied load or torque.² Ligamentous stiffness has been implicated in the ability of the passive restraint system to maintain optimal arthrokinematic function.¹ Quantifying the characteristics of the load-displacement curve (stiffness) is thought to be important with respect to the clinical functioning of the knee joint,³ but examining stiffness only as the inverse of overall displacement occurring during predetermined joint loading magnitude (as is done in clinical laxity testing) may conceal important physiologic information about the nature of the loading response. That is, observing smaller components of the entire loading process may result in a more complete

understanding of the tissue response to loading.² Additionally, alterations in knee stiffness have been suggested as factors in anterior cruciate ligament (ACL) injury risk.⁴

With regard to anterior stiffness of the knee joint, a breakpoint or subjective approach to measuring the anterior knee load-displacement response is typically defined by lower to higher stiffness during increasing loads.^{5,6} This response is most commonly assessed using an instrumented knee arthrometer that measures the anteriorly applied load and anterior displacement of the tibia with respect to the femur. Although originally defined as a compliance index (the inverse of stiffness), anterior translation of the tibia relative to the femur during anterior knee loading from 67 to 89 N has been used as a diagnostic criterion for ACL rupture.⁷ Others have qualitatively and quantitatively

suggested that 3 discreet stiffness phases are observed during anterior knee arthrometry testing,^{8,9} with a steep initial phase due to overcoming the weight of the leg, a less steep middle phase in which tibial displacement occurs with relatively lesser resistance, and a steep later phase in which the soft tissue restraint to anterior translation increases.⁹ This steep later phase is thought to reflect full engagement of the ACL in restraint.⁹ Most investigators^{1,7-9} of anterior knee stiffness have focused on ACL tear diagnostics, but some authors^{10,11} have suggested that laxity (and thus stiffness) is a factor in the biomechanics of deceleration maneuvers associated with noncontact ACL injury mechanisms. Assessing stiffness at more systematic and discrete intervals than the 2 or 3 typically reported during loading may offer a more detailed assessment of passive restraint mechanics.⁴ This would give us a better description of load-displacement behavior across the entire loading cycle, which may allow differentiation of individuals with passive restraint mechanics thought to be at greater risk of injury. Moreover, assessing stiffness at fixed, discrete intervals may provide more direct comparisons of the load-displacement response across time.

Females are reported to experience changes in knee laxity across the menstrual cycle, largely thought to be due to cyclic changes in their hormone concentrations. However, in part because of substantial interparticipant differences in the magnitude and timing of sex hormone changes across the menstrual cycle, not all women experience substantial changes in knee laxity.^{12,13} Given the critical role of the ACL in guiding tibiofemoral joint motion, a better understanding of knee joint behavior in those individuals who experience large increases in their cyclic knee laxity (“responders”) may allow greater insight into potential mechanisms of ACL loading and injury in females, who are known to be at greater risk for ACL trauma. Few previous authors^{4,14,15} have studied cyclic changes in anterior knee stiffness. Investigating stiffness rather than laxity values occurring during predetermined joint loading magnitude may better reflect the tissue tensile properties at various loading magnitudes.⁴ Measures of stiffness are suggested to be associated with the clinical “end feel” of tissue testing,⁹ which may lend insight into tissue restraint mechanics during periods of increasing loading. Perhaps most importantly, stiffness measures at greater magnitudes of joint loading may lend insight into tissue behavior associated with the ACL injury mechanism, possibly aiding in identifying those who may be more predisposed to ACL injury. Reports on stiffness changes across the menstrual cycle are mixed, with one group¹⁴ noting no change in stiffness (yet significant changes in laxity), whereas another⁴ demonstrated increasing midrange (of the loading range) stiffness from ovulation to the luteal phase of the menstrual cycle (as well as significant changes in laxity). Because stiffness changes have only been compared at discrete testing days in the luteal phase⁴ or reported for days delimited by specific sex hormone concentrations,¹⁴ maximum changes in knee laxity (and, thus, potentially stiffness) may not have been captured. Testing at these limited time points may explain the mixed findings, as the literature suggests wide variability in the timing (ie, day of the cycle) and magnitude of cyclic changes in laxity among females.^{4,12,13} We are not aware of any authors who have directly compared the magnitude of change in tissue

stiffness properties simultaneously with the magnitude of change in laxity measures at more than 3 measurement points across the menstrual cycle in those individuals who display larger (responders) versus smaller (nonresponders) changes in anterior knee joint laxity. If ACL injury does not occur uniformly across the menstrual cycle,¹⁶ addressing such comparisons may shed further light on the potential consequences of these interindividual differences in laxity responsiveness across the menstrual cycle.

Thus, the purposes of our study were first to describe the anterior load-displacement curve generated during anterior loading of the tibia relative to the femur using incremental stiffness measures and then to compare underlying stiffness values between days of the cycle when anterior knee laxity (AKL) is at its minimum and maximum in females who experience small versus large magnitudes of AKL change throughout their menstrual cycles. We hypothesized that those individuals who experience a greater magnitude of change in AKL (responders) across the menstrual cycle would demonstrate larger, increment-specific decreases in stiffness compared with those who experience the least amount of change (nonresponders).

METHODS

These data represent secondary analyses from a larger study in which the primary aim was to determine the magnitude and timing of cyclic variations in the knee.¹³ Participants were 57 recreationally active females who had been active between 2.5 and 10 hours per week for the past 3 months; had a body mass index <30 kg/m²; were nonsmokers; had no history of injury involving the osteochondral surface, ligament, tendon, capsule, or menisci; had no connective tissue disorders; and consumed no alcohol 24 hours before any test session.¹⁷ Participants had normal menstrual cycles lasting 26 to 32 days that varied no more than ± 1 day between months; had not used oral contraceptives or other hormone-stimulating medications for the past 6 months; and had no history of pregnancy or plans to become pregnant. Before enrollment, participants signed a consent form approved by the University of North Carolina at Greensboro Institutional Review Board, which also approved the study. Participant numbers are reduced from those of the larger study¹³ of laxity (N = 75) because of instrumentation issues with the real-time collection of continuous load-displacement data from the arthrometer.

Participants were tested during 2 menstrual cycles. During the first 6 days after the onset of menses (by self-report) and the first 8 days after evidence of ovulation (for a total of 14 days of measurement for each cycle), AKL was measured each morning (7:00 to 9:00 AM), before any physical activity, to capture individual cyclic variations.¹¹ To estimate the day of ovulation, participants were provided ovulation kits (One Step Ovulation Predictor; CVS Corporation, Woonsocket, RI [sensitivity = 20 mIU/mL LH, accuracy = 99%]) to begin using on day 8 of the menstrual cycle. Two investigators were trained in the laxity measures and established excellent intratester reliability and precision (intraclass correlation coefficient [ICC] [2,k] \pm standard error of measurement [SEM] for tester 1 = 0.96 \pm 0.3 mm, tester 2 = 0.97 \pm 0.4 mm).¹¹ To optimize measurement consistency, 1 examiner performed

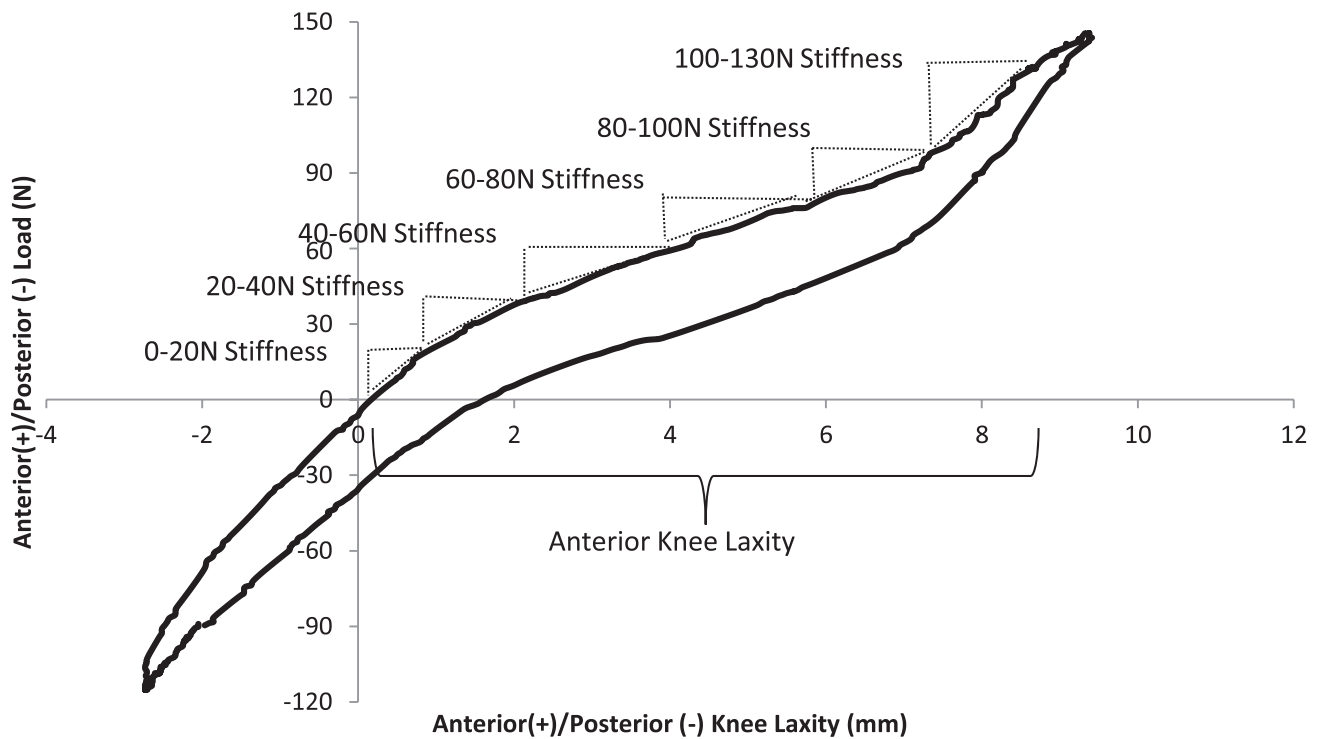


Figure 1. One participant's anterior knee load-displacement curve.

all measurements. Procedures for obtaining knee laxity data using the KT-2000 Knee Arthrometer (MEDmetric Corporation, San Diego, CA) have been previously reported.¹¹ With the knee flexed to $25^{\circ} \pm 5^{\circ}$, 3 posteriorly directed forces were applied to the tibia to establish a zero reference point, followed by a posteriorly directed force to 90 N to an anteriorly directed force of 133 N (Figure 1). Surface electromyographic electrodes monitored any measurable muscle activity or guarding. Three trials of real-time load (N) and displacement (mm) data were collected to a personal computer via analog-to-digital conversion, low-pass Butterworth filtered at 10 Hz, and saved for later calculation of incremental stiffness values. For the purposes of this investigation, data from participants' second cycle of testing were analyzed to ensure there was no effect on stiffness measures from habituation to the arthrometry measurements.

Although laxity typically increases during the early luteal phase,^{14,18} the interindividual timing of these changes is quite variable,^{12,13} and larger cyclic changes can occur in other phases of the cycle.⁴ Because of this, the maximum magnitude of cyclic change in AKL for each participant was determined by identifying the cycle days when AKL was at its minimum and maximum values. Participants were then stratified into thirds based on the absolute magnitudes of their cyclic laxity change scores across the menstrual cycle (<1.24 mm, nonresponders; 1.24 – 1.75 mm, moderate cyclic laxity change; and >1.75 mm, responders). This resulted in 19 participants (age = 21.4 ± 2.6 years, height = 1.64 ± 0.06 m, mass = 64.4 ± 9.7 kg) being classified as nonresponders (mean laxity change = 1.0 ± 0.2 mm, range = 0.68 – 1.23 mm) and 19 participants (age = 22.5 ± 3.5 years, height = 1.66 ± 0.06 m, mass = 60.2 ± 7.5 kg) being classified as

responders (mean laxity change = 2.1 ± 0.3 mm, range = 1.76 – 2.86 mm).

Similar to previous authors² who looked at incremental stiffness values of transverse- and frontal-plane knee motions, we compared incremental stiffness across the loading range on the days of the menstrual cycle for which minimum and maximum laxity values were recorded. The arthrometer load-displacement data were exported to a spreadsheet for calculation of stiffness values. To obtain incremental stiffness values, we plotted load-displacement curves for each trial and then divided the values into load increments of 0–20, 20–40, 40–60, 60–80, 80–100, and 100–130 N. For each increment, *stiffness* was calculated as the change in load divided by the change in displacement ($\text{N}\cdot\text{mm}^{-1}$) and averaged across 3 trials. A graphic example of these calculations is found in Figure 1. The change in cyclic stiffness for each of the 6 increments was computed by subtracting the incremental stiffness value on the day of maximum laxity from the incremental stiffness value on the day of minimum laxity.

We established between-days consistency and precision of incremental stiffness measures using the ICC (2,3) and SEM in 38 males (who do not experience cyclic laxity changes) tested approximately 2 weeks apart. Independent *t* tests confirmed significant differences in the magnitude of cyclic knee laxity changes between responders and nonresponders. A repeated-measures analysis of variance first compared the 6 incremental stiffnesses of the load-displacement curve on the day of minimum laxity across all 57 participants to characterize the incremental stiffness pattern. A 2 (group) \times 6 (increment) repeated-measures analysis of variance then examined the magnitude of change in incremental stiffness from days of minimum to maximum laxity between low (nonresponder) and high (responder) cyclic laxity change groups. Post hoc compar-

Table 1. Between-Days Incremental Stiffness Measurement Consistency (Intraclass Correlation Coefficient [ICC] [2,3]) and Precision (Standard Error of Measurement [SEM]) in Males Assessed 2 Weeks Apart

Direction	Stiffness Increment, N	ICC (2,3)	SEM (N·mm ⁻¹)
Anterior	0–20	0.87	4.2
	20–40	0.93	2.6
	40–60	0.85	2.9
	60–80	0.95	2.5
	80–100	0.94	3.2
	100–130	0.89	5.3
Posterior	0–20	0.90	3.2
	20–40	0.90	3.4
	40–60	0.89	3.3
	60–80	0.87	4.4

isons consisted of main-effects testing (with Bonferroni correction) for main effects and interactions. The alpha level was set at $< .05$.

RESULTS

Test-retest consistency of incremental stiffness measures taken 2 weeks apart in males was strong, with the ICCs ranging from 0.85 to 0.95 (Table 1).

Responder and nonresponder descriptive information for age, height, mass, minimum and maximum AKL, and maximal cyclic laxity change is presented in Table 2. In support of grouping participants into responder and nonresponder categories based upon the magnitude of laxity change, an independent *t* test revealed that responders demonstrated greater cyclic change in AKL than did nonresponders ($P \leq .001$).

The incremental stiffness from 0 to 130 N of anterior load on the minimum laxity day for 1 participant is shown in Figure 1. The incremental stiffness analysis across all 57 participants is consistent with this pattern, showing an initial period of decreasing stiffness (0–20 N $>$ 20–40 N and 40–60 N), followed by incrementally increasing stiffness (40–60 N $<$ 60–80 N $<$ 80–100 N $<$ 100–130 N) ($P \leq .05$), which demonstrated 4 significant changes in stiffness (Figure 2).

When examining changes in incremental stiffness between days of minimum and maximum laxity, we found no main effect for laxity response group ($P = .300$) or increment ($P = .144$) but did note a significant increment-by-response group interaction ($F_{5,180} = 2.92$, $P = .015$). Follow-up post hoc testing revealed that although nonresponders had no change in incremental stiffness between the days of minimum and maximum AKL, responders decreased their stiffness at the 100–130-N increment when compared with the 0–20-N and 20–40-N increments. Thus, the only difference observed in the change in the stiffness measures between responders and nonresponders was in the 100–130-N increment (Figure 3). No main effects of cyclic stiffness change for laxity response group ($P = .300$) or increment ($P = .144$) were seen.

DISCUSSION

Our primary findings were that stiffness changes in anterior knee joint loading occurred in smaller load increments than previously reported and that terminal stiffness (100–130 N) increments were most affected by

Table 2. Participants' Descriptive Data

Characteristic	Anterior Knee Laxity Across Menstrual Cycle	
	Nonresponders ^a	Responders ^b
Age, y	21.4 ± 2.6	22.5 ± 3.5
Height, m	1.64 ± 0.06	1.66 ± 0.06
Mass, kg	64.4 ± 9.7	60.2 ± 7.5
Anterior knee laxity, mm		
Minimum	5.2 ± 1.1	6.4 ± 2.8
Maximum	6.2 ± 1.1	8.5 ± 3.1 ^c
Maximal cyclic change	1.0 ± 0.2	2.1 ± 0.3 ^c

^a Less than 1.24 mm cyclic laxity change.

^b More than 1.75 mm cyclic laxity change.

^c Responders greater than nonresponders ($P \leq .05$).

cyclic changes in AKL across the menstrual cycle. In the following discussion, we will first consider the merit of the current characterization of incremental stiffness in comparison with previous methods and then consider the clinical consequences of incremental stiffness changes in laxity responders versus nonresponders across the menstrual cycle.

Characterizing Knee Stiffness

We described anterior knee stiffness as the incremental stiffness of anterior knee loading across multiple fixed loading intervals. Previously, anterior knee stiffness had been measured as a singular, fixed-interval stiffness^{7,19} at 3 intervals based upon either quantitative estimation of slope change⁴ or qualitative curve breakpoint estimation.⁸ Our use of the 6-increment stiffness approach avoids the nondefined intervals of previous measures that require greater data-reduction efforts to discern⁸ while allowing more exact determination of stiffness with a usable number of increments not available with instantaneous stiffness measures (originally introduced to identify 2 breakpoints in the curve⁹). We believe the current approach allowed a comprehensive description of load-displacement response data while attempting to balance the occasionally at-odds concepts of ease of data handling and data resolution. It may also provide a means for more consistent data comparisons between repeated measurements (eg, days of the cycle).

Our findings support previous work reporting increasing stiffness properties as anterior knee loading is progressed.^{8,9} The anterior loading curve was characterized by an initial increment (0–20 N) of moderate stiffness followed by a decreased stiffness increment (20–40 N), followed by continually increasing stiffness up to the 100–130-N increment. The larger initial stiffness has been attributed to soft tissue compression when the arthrometer matches the weight of the lower extremity.^{8,9} Although purely conjecture, another explanation for this greater initial stiffness may be short-range stiffness (ie, the increased stiffness associated with the initial elongation of contracting muscle²⁰) from some level of resting muscle tone in the conscious participant. This idea is supported by work¹⁷ demonstrating greater AKL in the unconscious versus conscious patient. Whereas previous authors^{4,8} have reported singular definitions of terminal stiffness using a 2- or 3-part loading model, our study demonstrates that stiffness continues to increase significantly from the 60–80 to 80–100 to 100–130-N increment. This suggests greater

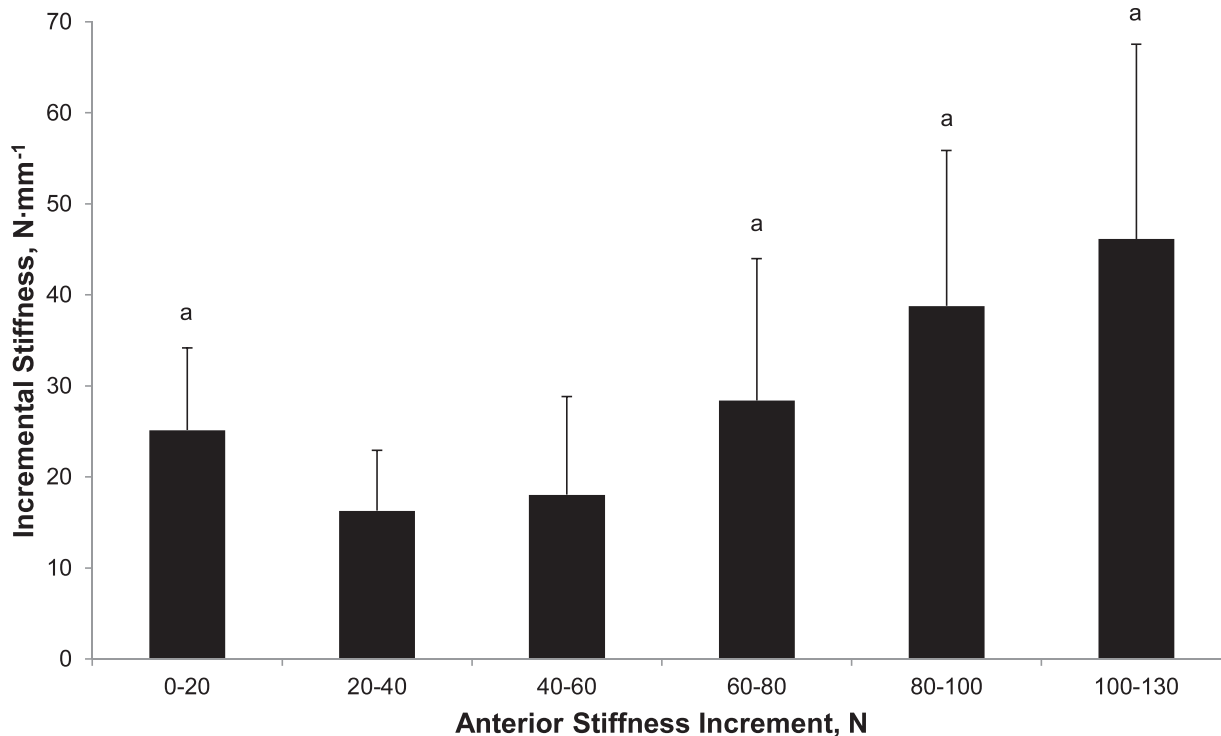


Figure 2. Incremental stiffness values across the loading cycle for all 57 participants on day of minimum anterior knee laxity. ^a Indicates significant difference ($P \leq .05$) in incremental stiffness across increments with 0–20 N > 20–40 N and 40–60 N < 60–80 N < 80–100 N < 100–130 N.

resistance to deformation as load increases and more complex tissue behavior than reported to date during the later phases of anterior loading.

Greater Cyclic Changes in Knee Stiffness in Responders

In our investigation, laxity responders (ie, individuals who experienced larger increases in AKL across the menstrual cycle) displayed an associated decrease in stiffness in the higher incremental loading range (100–130 N). Traditionally, this increment has been referred to as the *terminal stiffness of the joint*, which is thought to be the point at which the ACL is fully engaged in restraining anterior tibial motion.⁹

We are the first to report significant alterations in terminal (100–130 N) stiffness across the menstrual cycle. Romani et al¹⁵ found no change in 89–134-N stiffness among 3 days of the cycle representing menses, ovulation, and luteal phases, whereas Park et al⁴ demonstrated that individually calculated stiffness changes in load-displacement data resulted in midrange stiffness (approximately 30–70 N) that increased significantly (approximately 20%) from ovulation to the luteal phase, with no change in the follicular phase. Conversely, Deie et al¹⁴ noted no change in stiffness (calculated at the 134-N tangent) across 3 points in the menstrual cycle, even though laxity increased at 89 and 134 N. Although all authors confirmed the phase of the menstrual cycle by verifying sex hormone concentrations consistent with that phase of the cycle, all were limited by the fact that only a single day within each phase was measured. Given the interparticipant variability in the magnitudes and patterns of cyclic changes previously

demonstrated in females,¹³ it may be that previous authors did not find significant changes in stiffness simply because they did not adequately capture days of the cycle when laxity changes (and thus stiffness) across the menstrual cycle were maximized. Further, it is possible that only those with substantially large changes in knee laxity experience changes in stiffness, given that we observed changes in stiffness values only in those with an appreciable change in AKL (ie, responders; >1.75 mm). This suggestion is supported by the findings of Deie et al,¹⁴ who demonstrated a significant laxity change of approximately 0.6 mm without associated changes in stiffness. Observable changes in stiffness appear to depend upon the magnitude of the cyclic laxity changes, and not all individuals demonstrate this magnitude of change.

It is also possible that the previous authors did not have enough “resolution” in their stiffness measures to fully identify cyclic stiffness changes. The singular stiffness measures reported by Romani et al¹⁵ and Deie et al¹⁴ may have used ranges of anterior knee load that were too wide (89–134 N) or too small (tangent to 134 N), respectively, to discretely quantify anterior knee joint stiffness throughout the loading range. Taken together, the use of anterior knee testing on multiple days of the cycle, determination of responders and nonresponders, and incremental approach to calculating stiffness may have optimized our ability to observe the cyclic impact of decreased terminal (100–130 N) stiffness on the day of maximal AKL.

Because of decreased terminal (100–130 N) stiffness at the time of maximal cyclic joint laxity in responders, the ACL may become somewhat less effective in passively restraining anterior tibial translation if cyclic changes in knee laxity are of sufficient magnitude. This less effective

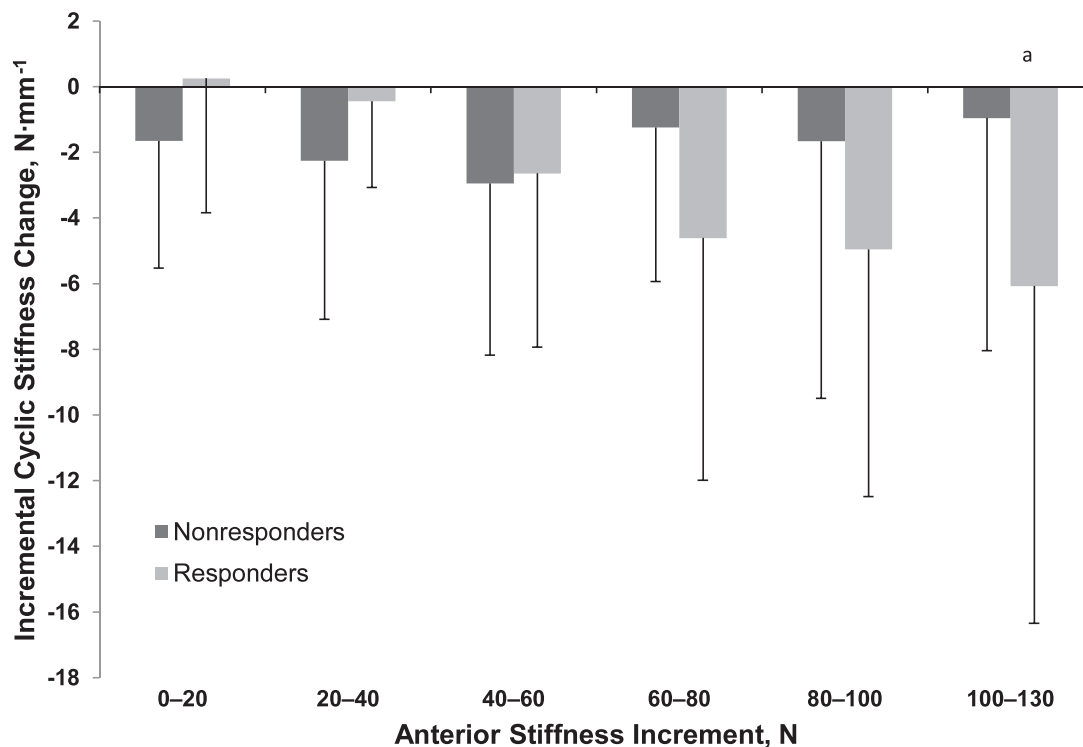


Figure 3. Anterior incremental cyclic stiffness change by group. ^a Indicates responders 100–130 N < 0–20 and 20–40; responders 100–130 N < nonresponders 100–130 N ($P = .015$).

restraint in responders may have clinical manifestations. Physically induced changes via partial transection in ACLs in animal models suggest that observed decreases in stiffness are accompanied by decreases in ultimate tensile force.²¹ Although decreased ACL cross-sectional area was likely ultimately responsible for decreases in ultimate tensile force, decreased tissue stiffness properties may also indicate an ACL that fails under lesser loads. Reliance on other components to maintain anterior stability may be increased, which in turn may predispose other knee structures to injury.²² Lastly, the current data suggest that decreased stiffness in the terminal (100–130 N) loading region, when large cyclic increases in knee laxity are observed, may result in greater anterior tibial translation during periods of greater knee loading. This in turn may affect knee arthrokinematics and produce a more anteriorly positioned tibia relative to the femur, which may affect knee arthrokinematics adversely.²³

During weight-acceptance activity, greater baseline and cyclic knee laxity¹⁰ and increasing axial load¹¹ are all related to greater anterior tibial translation. Furthermore, increases in general joint laxity and AKL (that likely correspond with decreased stiffness) are related to greater frontal-plane and transverse-plane knee laxities,²⁴ which have been associated with adverse hip and knee biomechanics during landing.²⁵ Collectively, these findings suggest that decreased stiffness may affect multiplanar and multijoint lower extremity biomechanics during physical activity.

We had already recognized large interparticipant variations in knee laxity changes across the cycle in our sample.¹³ The reasons for these large differences in responsiveness (responder or nonresponder status) are not yet well understood. Although interparticipant differences

in sex hormone changes across the menstrual cycle in part explain these interparticipant differences in responsiveness,²⁶ the variable delay in these alterations in response to hormone changes¹² and the fact that some women experience their largest changes in laxity in phases of the cycle when hormones are at their nadirs⁴ suggest that other factors (eg, cyclic tension,²⁷ such as that associated with exercise and genetic variants²⁸) may combine or interact with sex hormone concentration changes to influence collagen structure and metabolism and laxity responsiveness.¹³ Work is ongoing to understand these complex underlying physiologic processes in order to develop reliable algorithms to identify laxity-responsive females and their susceptibility to ACL injury risk. Better understanding and identification of responders may ultimately help us to better prescribe prevention programs for those at risk of ACL injury.

The resultant 1.1-mm greater change in cyclic laxity in responders may have potential implications for functional knee joint biomechanics. In a study²⁹ of the early phase of simulated weight acceptance, anterior tibial translation increased approximately 0.5 mm for every 1-mm increase in AKL. Furthermore, 1-mm greater AKL plus a general joint laxity score of 0.9 more predicted 21% greater knee work absorption, 9% greater knee joint stiffness, and 6% lower predicted ankle stiffness during drop-jump maneuvers.¹¹ Finally, female cadets with absolute laxity values that were 1 SD or more (1.9 mm) above the laxity mean at 134 N had a 2.7 times greater relative risk of noncontact ACL injury.³⁰ Collectively, these findings indicate that individuals classified as responders (2.1-mm mean cyclic laxity change) may have different deceleration mechanics and a greater risk of ACL injury compared with those

individuals experiencing lesser magnitudes of cyclic joint laxity.

This investigation was limited by several factors. Although we took great care to ensure that each participant was as relaxed as possible without any associated muscle guarding, resting muscle tone and subtle voluntary contraction are possible complicating factors during joint arthrometry testing. Additionally, the increasing variance in stiffness change scores as loading increased (Figure 3) may have affected our ability to identify further differences between responders and nonresponders. The increased variance here could also be attributed to increased discomfort by the participant and subsequent muscle guarding. Even though we took great care to identify participant apprehension or guarding by monitoring muscle activity via surface electromyography, subtle changes in muscle tone are not observable with this method. Moreover, the behavior of the passive restraints at 130 N may not fully represent ACL loads during injury given that failure of the ACL in female cadavers has been reported to occur at 1266 ± 527 N.³¹ As such, the observed stiffness we measured may not represent near-injurious loading situations.

CONCLUSIONS

Using an incremental analysis, we generated a load-displacement curve during anterior loading of the tibia relative to the femur that is more complex in behavior than previously described, with 4 changes in stiffness during arthrometer testing. Future modeling studies involving the anterior passive restraint system may be better optimized using a model with more than the traditionally defined 2 breakpoints. With respect to assessment of cyclic changes, larger magnitudes of cyclic change in AKL resulted in decreased stiffness at only the higher loading ranges (100–130 N). This result suggests that in order to distinguish responders from nonresponders in knee stiffness, common use of terminal (100–130 N) stiffness may be appropriate. Decreases in terminal stiffness may be detrimental to passive joint restraint and subsequent biomechanics observed during functional activity. Future researchers should build upon these findings by understanding why some women experience larger magnitudes of terminal stiffness and laxity changes compared with others and how changes in terminal stiffness may affect movement mechanics that are thought to be high risk with regard to noncontact ACL injury.

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