Associations Between Lower Extremity Muscle Mass and Multiplanar Knee Laxity and Stiffness: A Potential Explanation for Sex Differences in Frontal and Transverse Plane Knee Laxity

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Abstract:

Background: Compared with men, women have disproportionally greater frontal (varus-valgus) and transverse (internal-external) plane laxity and lower stiffness, despite having similar sagittal (anterior-posterior) plane laxity and stiffness. While the underlying cause is unclear, the amount of lower extremity lean mass (LELM) may be a contributing factor.

Hypothesis: Lower extremity lean mass would be a stronger predictor of frontal and transverse plane laxity and incremental stiffness than the sagittal plane. Associations between LELM and stiffness would be stronger at lower force increments.

Study Design: Descriptive laboratory study.

Methods: Sixty-three women and 30 men with no history of ligament injury were measured for knee laxity and incremental stiffness in the sagittal (−90- to 130-N posterior-to-anterior directed loads), frontal (±10-N·m varus-valgus torques), and transverse (±5-N·m internal-external rotation torques) planes and underwent dual-energy X-ray absorptiometry scans to measure LELM. Linear regressions examined the extent to which LELM predicted each laxity and stiffness value, while also accounting for a person's sex.

Results: Females (vs males) had greater laxity and less stiffness in the frontal and transverse planes but not the sagittal plane. Lower extremity lean mass was a poor predictor of sagittal laxity and stiffness (R^2 range = .021-.081; $P > .06$) but was a stronger predictor of frontal (R^2 range = .215-.567; $P < .01$) and transverse (R^2range = .224-.356; $P < .01$) plane laxity and stiffness. Associations were stronger for low (R^2 = .495-.504) versus high (R^2 = .215-.435) frontal plane stiffness but were similar for low (R^2 = .233-.293) versus high (R^2 = .224-.356) transverse plane stiffness. Once we accounted for a person's LELM, sex had little effect on laxity and stiffness (change in R^2 after removal = .01-.08; P = .027-.797).

Conclusion: Less LELM was associated with greater laxity and less stiffness in frontal and transverse planes, which may contribute to the disproportionally higher laxities and reduced stiffnesses observed in females in these planes.

Clinical Relevance: Frontal and transverse plane laxity and stiffness may be modifiable through strength training interventions that promote changes in muscle characteristics (eg, muscle cross-sectional area, stiffness) that may contribute to static knee joint stability, thus dynamic joint stability during sport activity.

Keywords: knee laxity | knee stiffness | muscle mass | ACL injury

Article:

Greater magnitudes of joint laxity are associated with a greater risk of anterior cruciate ligament (ACL) injury,[§] and women (who are at greater risk for ACL injury) have greater joint laxity than men.[3,15,28,34,37,42,45] However, joint laxity is not always uniform across anatomic planes,38 and even when similar on sagittal plane knee laxity measures, women still have 25% to 30% greater frontal (varus-valgus [VV] rotation) and transverse (internal-external [IER] rotation) plane knee laxity and decreased stiffness compared with men.[15,37,42] These greater frontal and transverse plane knee laxities in women during low, externally applied loads (eg, 5-10 N·m) are most pronounced upon initial joint loading,[35] and this may increase their potential for dynamic knee valgus (ie, greater hip adduction, hip internal rotation, and knee valgus) during the early phase of landing.[41] However, it is not yet clear why women have disproportionally higher frontal and transverse plane knee laxities.

In vivo laxity testing represents the combined resistance of the ligament, muscle, and capsule to a displacing load. Hence, musculotendinous structures that cross the joint may contribute to the passive resistance of joint displacements across anatomic planes. As men have greater muscle mass than women, and greater passive and dynamic resistance of the muscle-to-joint displacements are associated with greater cross-sectional area of the muscle,[4,33] sex differences in muscle mass may be magnified in anatomic planes where passive muscle resistance plays a greater role. This may be particularly true at lower force ranges where passive biomechanical properties of human muscles demonstrate a short range of stiffness (ie, the distortion but not breakage of cross bridges) when initially loaded.[31] However, while muscular protection of the knee and reduction in shear and rotational displacements have been examined during dynamic contractions,[19,23,29,48,49] we are not aware of any studies that have examined the passive role of muscles in contributing to in vivo knee laxity measures across multiple anatomic planes.

Recent studies examining associations between body mass and body mass index (BMI) with specific knee laxity measures support the need for such studies. Shultz et al[38] clustered 140 patients into groups based on their sagittal, frontal, and transverse plane knee laxity and examined the extent to which BMI (mass*height^{-2}), isometric thigh strength, and structural alignment (eg, hip anteversion, knee varus, navicular drop) predicted membership in a particular knee laxity group. Once they accounted for other structural factors, leaner and weaker patients (lower BMI and thigh strength) were more likely to be in laxity groups with higher magnitudes of frontal and transverse plane knee laxity but not necessarily sagittal plane laxity.[38] This is

consistent with other work noting strong negative correlations between body mass (total body weight [kg]) and transverse plane knee laxity[26] but not body mass or BMI with anterior knee laxity.[46] Because a reduction in either fat mass or muscle mass could lower body mass or BMI, it is difficult to parse out the contributions of muscle mass in the observed associations. However, as less thigh muscle strength (which is largely dependent on available lean muscle mass[2]) was also associated with greater frontal and transverse plane laxity,[38] the amount of lean mass may ultimately drive these associations between BMI[38] and body mass[26] with frontal and transverse plane knee laxity.

Understanding the contribution of muscle mass to knee joint laxity may have implications for our injury screening and prevention strategies. Should greater lean muscle mass be associated with less frontal and transverse plane knee laxity, this would suggest that knee joint laxity, typically considered a nonmodifiable anatomic risk factor, may be modifiable through strength training interventions that promote changes in muscle properties (eg, increased muscle mass, increased muscle stiffness), which enhance the ability of the muscle to passively (thus, dynamically) resist externally applied loads to the joint. Hence, we examined the extent to which lower extremity lean mass (LELM) contributed to sagittal, frontal, and transverse plane knee laxity and incremental stiffness, once controlling for a person's sex. Based on prior studies examining indices of body mass and knee joint laxity,[26,38,42,46] we expected that LELM would be a stronger predictor of greater laxity and decreased stiffness in the frontal and transverse planes as compared with the sagittal plane. We also expected that associations between LELM and stiffness would be stronger at lower force increments (initial loading).

MATERIALS AND METHODS

The study sample was obtained over a 3-year period (2009-2011) and consisted of physically active male (n = 30; mean ± standard deviation [SD] age, 20.4 ± 2.0 years; height, 179.4 ± 5.4 cm; weight, 75.7 ± 7.6 kg) and female (n = 63; mean ± SD age, 20.48 ± 2.4 years; height, 165.2 ± 7.6 cm; weight, 62.8 ± 9.2 kg) study participants who had been measured on their multiplanar knee joint laxity and also underwent dual-energy X-ray absorptiometry (DXA) to assess their body composition. All participants were physically active at least 30 minutes, 3 times a week, and were apparently healthy with no history of ligament, tendon, meniscus, or osteochondral injury to their dominant limb (defined as the stance limb when kicking a ball). All laxity and stiffness measures were obtained during a single test session, and DXA scans were performed within 7 days of the laxity test session. For female participants, testing was constrained to the first 7 days of the menstrual cycle (defined by the onset of menses) to minimize the risk of performing a DXA scan during pregnancy, control for cyclic changes in knee laxity, and obtain laxity values when they are typically at their nadir.39 All measurements were taken on the dominant limb. All participants read and signed a consent form approved by the University of North Carolina at Greensboro's institutional review board for the protection of human participants before inclusion.

Frontal (VV) and transverse plane (IER) laxity and stiffness measures were assessed with the Vermont Knee Laxity Device (VKLD), as described previously in detail.[35] Clusters of 3 optical light-emitting diode markers (Phase Space, San Leandro, California) were placed on the left foot, shank, thigh, and sacrum. Joint centers were determined via the Leardini et al[17] (hip) and

centroid[21] (knee and ankle) methods. With the participant supine, the knee was flexed to 20°, the thigh was securely fixed, and the foot and ankle (flexed 90°) were strapped to the foot cradle connected to a calibrated 6 degrees of freedom force transducer. With gravity and shear loads eliminated, VV laxity and stiffness were assessed by applying 0 to 10 N·m of valgus and varus torques to the distal tibia with a force transducer (Model SM-50, Interface, Scottsdale, Arizona). The IER laxity and stiffness were measured by applying 0 to 5 N·m of internal-external torques about the long axis of the tibia using a T-handle connected to a 6 degrees of freedom force transducer affixed to the foot cradle (MC3A, Advanced Medical Technology Inc, Watertown, Massachusetts). To ensure muscular relaxation during testing, participants were thoroughly familiarized to all laxity measures before the day of testing. On the day of testing, they were instructed to fully relax before each measurement trial, and muscle tension was visually and manually monitored during the test by the investigator. We also examined the real-time load-displacement response after each trial to examine for any changes in the curve suggestive of muscle guarding. If there was any evidence of muscle guarding, the trial was repeated. Kinematic (240 Hz) and load data (500 Hz) were simultaneously acquired during 3 continuous cycles for each set of torque rotations using an 8-camera optical system (Impulse, Phase Space) and Motion Monitor Software (Innovative Sports Training, Chicago, Illinois) and low pass filtered at 6 Hz (determined from residual analyses). Segmental coordinate systems were constructed with Euler equations describing 3-dimensional joint motions about the knee. VV_{LAX} and IER_{LAX} were calculated as the total VV and IER angular joint displacements at ±10- and ±5-N·m torques, respectively. Varus (VAR_K) and valgus (VAL_K) incremental stiffness were calculated as the change in torque divided by the change in angular displacement (N·m/deg) in 2-N·m increments (0-2, 2-4, 4-6, 6-8, and 8-10 N·m), while internal (IRK) and external (ER_K) rotation stiffness were calculated in 1-N·m increments (0-1, 1-2, 2-3, 3-4, and 4-5 N·m), thus producing 5 incremental stiffness values for each direction.[35] For the purpose of this study, the initial 40% (first 2 increments) and terminal 40% (last 2 increments) of the load-displacement response were averaged to obtain low ($VAR_{K\text{-}LO}$, $VAL_{K\text{-}LO}$, $IR_{K\text{-}LO}$, $ER_{K\text{-}LO}$) and high ($VAR_{K\text{-}HI}$, $VAL_{K\text{-}HI}$, $IR_{K\text{-}HI}$, $ER_{K\text{-}HI}$) incremental stiffness for each respective measure (Figure 1). Using similar methods, consistent VV and IER laxity (intraclass correlation coefficient [ICC] = .70-.96; standard error of the mean [SEM] = 0.9°-4.00°), low incremental VV and IER stiffness (ICC = .65-.84; SEM = 0.07-0.80 N·m/deg), and high VV and IER incremental stiffness (ICC = .03-.80; SEM = 0.12-0.51 N·m/deg) have been reported (note that low ICCs are limited to high incremental stiffness for internal rotation only).[35]

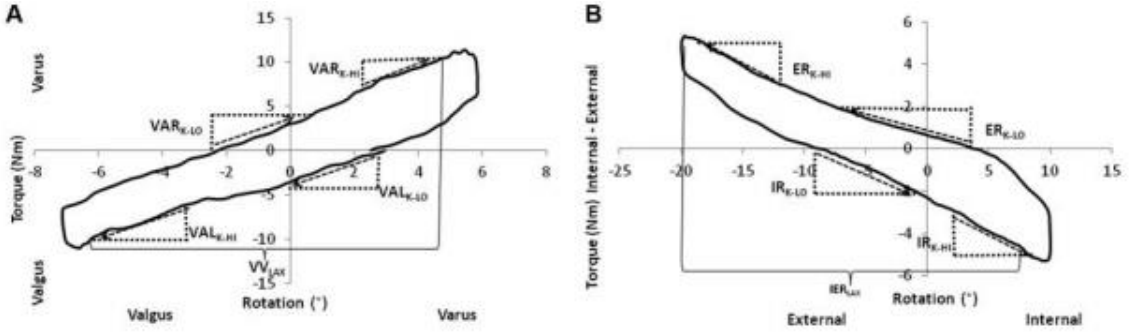

Figure 1. Representative (A) varus-valgus and (B) internal-external rotation load-displacement response and the laxity (±10-N·m VV_{LAX}, ±5-N·m IER_{LAX}) and initial ($VAR_{K\text{-}LO}$, $VAL_{K\text{-}LO}$, $IR_{K\text{-}LO}$, $ER_{K\text{-}LO}$) and terminal ($VAR_{K\text{-}HI}$, $VAL_{K\text{-}HI}$, $IR_{K\text{-}HI}$, $ER_{K\text{-}HI}$) stiffness measures acquired.

Anterior-posterior knee laxity (AP_{LAX}) and stiffness were measured using the KT-2000 Knee Arthrometer (MEDmetric Corp, San Diego, California), which has been shown to accurately track AP displacement of the tibia relative to the femur.[44] Although the VKLD is also capable of measuring AP laxity and stiffness and provides comparable measures to the KT-2000 arthrometer, we chose to use the KT-2000 arthrometer given its clinical accessibility (thus, greater transfer of our findings to clinical practice) and because values from this device are reported to more closely resemble tibiofemoral joint displacements obtained from planar radiographs, with less between-tester variation and less random measurement error.[44] With the participant positioned supine and the knee flexed to 25° ± 5° over a thigh bolster, and after applying 3 posterior-directed forces to provide a zero reference position, joint loads and displacements were collected simultaneously during 3 posterior-anterior loading cycles of the tibia relative to the femur from −90 N (posterior) to 130 N (anterior). To maximize measurement consistency, the thighs were stabilized with a Velcro® (Manchester, New Hampshire) strap to minimize lower extremity rotation, and a bubble level fixed to the device confirmed a direct posterior-anterior line of pull. Muscle relaxation was monitored in the same manner as frontal and transverse plane testing. Two experienced testers who were trained by the same investigator established strong measurement consistency prior to testing (ICC [SEM] = .96 [0.3 mm]; .93 [0.4 mm]). From the load-displacement data (low pass filtered at 10 Hz), AP_{LAX} was calculated as the total posterior-anterior displacement from −90 N to 130 N. Incremental anterior (ANT_K) and posterior (PST_K) stiffness were calculated as the change in force relative to the change in displacement (N/mm) in 5 increments for posterior stiffness (0-20, 20-40, 40-60, 60-80, and 80-90 N) and in 7 increments for anterior stiffness (0-20, 20-40, 40-60, 60-80, 80-100, 100-120, and 120-130 N). For the purpose of this study, the initial (first 3 increments for ANT_K and first 2 increments for PST_K) and terminal (last 3 increments for ANT_K and last 2 increments for PST_K) portions of the load-displacement response were calculated, representing the initial 40% to 43% ($ANT_{K\text{-}LO}$, $PST_{K\text{-}LO}$) and terminal 40% to 43% ($ANT_{K\text{-}HI}$, $PST_{K\text{-}HI}$) of the load-displacement curve (Figure 2).

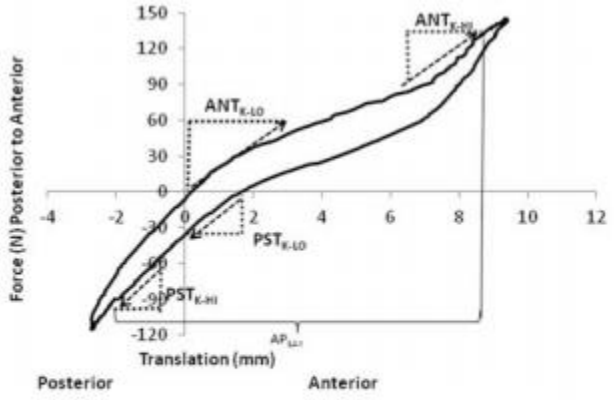

Figure 2. Representative anterior-posterior load-displacement response and the laxity (–90 N to +130 N) and initial ($ANT_{K\text{-}LO}$, $PST_{K\text{-}LO}$) and terminal ($ANT_{K\text{-}HI}$, $PST_{K\text{-}HI}$) stiffness measures acquired.

Participants underwent body composition testing via fan-beam DXA (Lunar Prodigy Advance, GE Healthcare, Madison, Wisconsin). The DXA measurements of LELM are reported to correlate well with those of computed tomography (R^2 = .86-.96)[18,47]and magnetic resonance

imaging scans (r = .93-.98).[6,9] While wearing lightweight athletic shorts and a T-shirt void of metal, participants' body height and mass were measured with a digital stadiometer and scale, respectively, and entered into the enCORE 2007 software (GE Healthcare). Participants were then centered on the midline of the DXA table while supine, and manual traction was applied to the distal tibias, arms, and head to ensure neutral spinal alignment and an equal bilateral position of the extremities. Participants were asked to remain completely still for the duration of the total body scan, which typically lasted 6 minutes. The region of interest (ROI) for LELM was defined superiorly from the inferior-lateral line through the neck of the femur to encapsulate the lateral hip and the entire thigh and shank of the left leg (Figure 3). From this ROI, the amount of bone, lean, and fat mass (kg) was calculated, and total LELM from the test leg was used for analysis. We chose to calculate total muscle mass rather than cross-sectional area because total muscle volume is a better estimate of actual muscle size.[2] The investigator established excellent test-retest reliability of LELM before data collection ($ICC_{2,1}$ [SEM] = .99 [0.21 kg]).

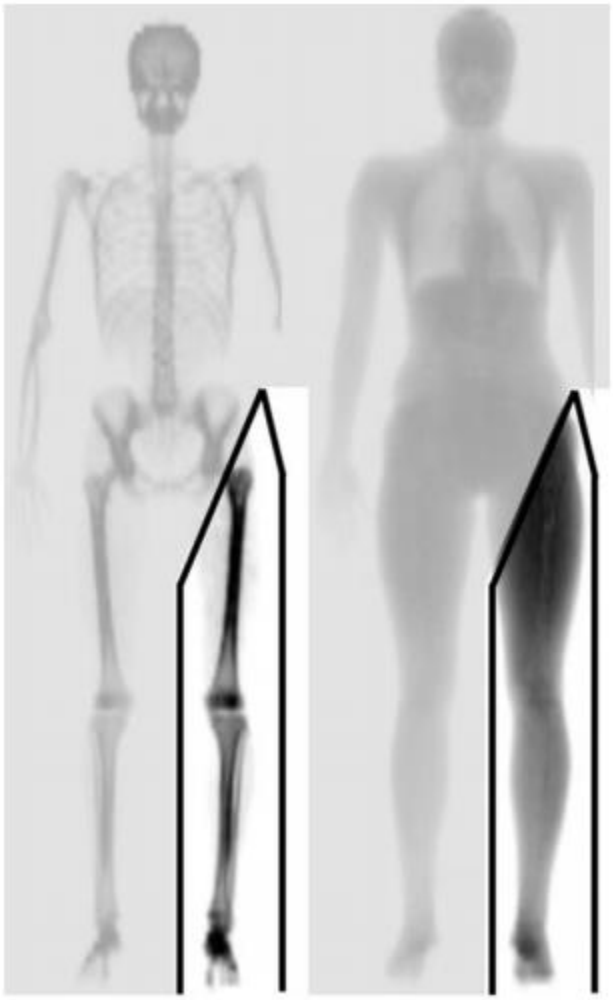

Figure 3. The defined region of interest for calculating the amount of lower extremity lean mass.

To analyze the data, linear regressions examined the extent to which LELM predicted each of the AP, VV, and IER laxity and stiffness variables, while also accounting for a person's sex.

Specifically, LELM, sex, and the interaction between LELM × sex were initially entered into the model. Then, sex and LELM × sex were removed in the second step. Our rationale for including sex initially was to account for other potential sex-dependent factors not included in the model and to ensure that LELM (which is substantially different for males and females) was not simply acting as a surrogate for these other sex-dependent factors. Including the LELM × sex interaction allowed us to determine if the relationship between LELM and each laxity and stiffness variable was dependent on the person's sex. Removing sex from the model in the second step allowed us to determine the extent to which the strength of the relationship between LELM with laxity and stiffness changed once sex was no longer accounted for. With a sample size of 93, and 3 predictors in the model, we had 88% to 99% power to detect a multiple R^2 of .15 to .25, which is considered a medium to large effect.[7]

RESULTS

Table 1 lists the means and SDs for the predictor variable and each of the dependent variables, stratified by sex (Note: Results for AP stiffness were limited to 59 females, as 4 females were too short for the standard arthrometer; $AP_{LAX}$was still obtained manually from reading the measurement dial of the KT-1000 Jr arthrometer [MEDmetric Corp]). Independent *t* tests confirmed that LELM was significantly different between males and females ($P < .001$), as were frontal ($P < .001$) and transverse ($P < .006$) plane laxity and stiffness. This confirmed our decision to initially include sex and LELM × sex in the model.

TABLE 1
Mean ± SD for LELM, Laxity, and Stiffness Stratified by Sex[a]

	Females (n = 63)		Males (n = 30)	
Variable	Mean ± SD	Range	Mean ± SD	Range
LELM, kg	7.3 ± 1.1[b]	5.2-10.4	10.8 ± 1.3	8.5-14.7
Sagittal plane				
AP_{LAX}, mm	9.0 ± 1.9	5.0-15.3	8.5 ± 2.0	5.1-12.4
$ANT_{K\text{-}LO}$, N/mm[c]	20.8 ± 6.4	11.5-39.4	18.7 ± 4.9	11.0-30.0
$ANT_{K\text{-}HI}$, N/mm[c]	38.2 ± 13.9	14.6-73.3	38.9 ± 11.4	20.2-57.5
$PST_{K\text{-}LO}$, N/mm[c]	24.9 ± 6.9	10.5-48.7	27.2 ± 7.9	15.4-44.0
$PST_{K\text{-}HI}$, N/mm[c]	39.8 ± 8.9	22.8-58.4	37.4 ± 11.3	19.5-58.8
Frontal plane				
VV_{LAX}, deg	11.3 ± 2.9[b]	3.9-16.6	6.7 ± 2.3	2.6-13.0
$VAR_{K\text{-}LO}$, N·m/deg	1.48 ± 0.55[b]	0.61-3.86	2.48 ± 0.81	1.08-4.23
$VAR_{K\text{-}HI}$, N·m/deg	1.73 ± 0.47[b]	1.07-3.05	2.55 ± 1.02	1.18-4.50
$VAL_{K\text{-}LO}$, N·m/deg	1.53 ± 0.69[b]	0.71-4.07	2.57 ± 0.91	1.14-4.30
$VAL_{K\text{-}HI}$, N·m/deg	1.51 ± 0.38[b]	0.90-3.25	2.10 ± 0.82	0.98-5.05
Transverse plane				
IER_{LAX}, deg	27.8 ± 7.6[b]	16.2-53.0	22.6 ± 4.8	14.7-35.0
$IR_{K\text{-}LO}$, N·m/deg	0.23 ± 0.08[b]	0.08-0.45	0.32 ± 0.09	0.15-0.50
$IR_{K\text{-}HI}$, N·m/deg	0.39 ± 0.11[b]	0.22-0.75	0.53 ± 0.10	0.29-0.71
$ER_{K\text{-}LO}$, N·m/deg	0.26 ± 0.10[b]	0.09-0.57	0.29 ± 0.07	0.14-0.41
$ER_{K\text{-}HI}$, N·m/deg	0.44 ± 0.13[b]	0.22-0.76	0.53 ± 0.10	0.30-0.76

[a]SD, standard deviation; LELM, lower extremity lean mass; AP, anterior-posterior; ANT, anterior; PST, posterior; VV, varus-valgus; VAR, varus; VAL, valgus; IER, internal-external; IR, internal; ER, external; LAX, laxity; K-LO, low stiffness; K-HI, high stiffness.
[b]Significantly different from males ($P < .05$).
[c]n = 59 for females.

The average LELM with men and women combined was 8.4 ± 2.0 kg. Descriptive data for each laxity and stiffness value and the regression summary statistics are presented in Table 2. In the sagittal plane, LELM was not a significant predictor of AP laxity or AP stiffness (*P* range =

.234-.840). The only exception was $ANT_{K\text{-}LO}$where LELM explained 6.7% of the variance once sex was removed (P = .015). In this case, for every 1-kg increase in LELM, there was a predicted 0.78 decrease in $ANT_{K\text{-}LO}$.

TABLE 2
Regression Summary of the Association Between LELM and Knee Laxity and Stiffness[a]

Variable	Mean ± SD	R^2 (P Value)	R^2 Change With Sex Removed (P Value)	Unstandardized Coefficients			
				Constant	LELM	Sex	Sex × LELM
Sagittal plane							
AP_{LAX}, mm	8.9 ± 1.9	.081 (.056)	—	7.783	.173	−6.118[b]	.461[c]
		.000 (.840)	−.080 (.024)	8.707	.02		
$ANT_{K\text{-}LO}$, N/mm	20.1 ± 6.0	.072 (.093)	—	27.929	−.963[c]	4.254	−.285
		.066 (.015)	−.006 (.765)	26.727	−.776[d]		
$ANT_{K\text{-}HI}$, N/mm	38.4 ± 13.3	.023 (.568)	—	35.122	−.412	31.499[c]	−2.973
		.001 (.833)	−.023 (.374)	39.662	−.148		
$PST_{K\text{-}LO}$, N/mm	25.7 ± 7.3	.044 (.278)	—	27.329	−.331	14.532	−1.024
		.003 (.617)	−.041 (.167)	23.998	.195		
$PST_{K\text{-}HI}$, N/mm	39.0 ± 9.7	.021 (.603)	—	46.922	−.967	−19.825	1.177
		.016 (.234)	−.005 (.797)	44.274	−.62		
Frontal plane							
VV_{LAX}, deg	9.8 ± 3.5	.567 (<.001)	—	22.24	−1.498[d]	−5.079	.536
		.559 (<.001)	−.008 (.461)	20.712	−1.289[d]		
$VAR_{K\text{-}LO}$, N·m/deg	1.80 ± 0.80	.504 (<.001)	—	−.012	.204[d]	−1.287	.145[c]
		.493 (<.001)	−.011 (.372)	−.53	.277[d]		
$VAR_{K\text{-}HI}$, N·m/deg	2.00 ± 0.80	.435 (<.001)	—	.666	.147[d]	−2.940[d]	.299[d]
		.387 (<.001)	−.048 (.027)	−.053	.243[d]		
$VAL_{K\text{-}LO}$, N·m/deg	1.87 ± 0.90	.495 (<.001)	—	−.663	.301[d]	−1.305	.118
		.486 (<.001)	−.009 (.454)	−.753	.311[d]		
$VAL_{K\text{-}HI}$, N·m/deg	1.70 ± 0.62	.215 (<.001)	—	1.172	.046	.045	.036
		.188 (<.001)	−.028 (.211)	.577	.133[d]		
Transverse plane							
IER_{LAX}, deg	26.1 ± 7.2	.255 (<.001)	—	49.459	−2.975[d]	−18.536[c]	2.209[b]
		.218 (<.001)	−.037 (.116)	40.093	−1.656[d]		
$IR_{K\text{-}LO}$, N·m/deg	0.28 ± 0.10	.233 (<.001)	—	−.058	.044[d]	.239[c]	−.031[b]
		.182 (<.001)	−.050 (.056)	.095	.021[d]		
$IR_{K\text{-}HI}$, N·m/deg	0.47 ± 0.14	.224 (<.001)	—	.05	.056[d]	.305[c]	−.039[b]
		.183 (<.001)	−.041 (.099)	.217	.029[d]		
$ER_{K\text{-}LO}$, N·m/deg	0.25 ± 0.08	.293 (<.001)	—	−.044	.037[d]	.234[b]	−.028[d]
		.249 (<.001)	−.048 (.066)	.074	.020[d]		
$ER_{K\text{-}HI}$, N·m/deg	0.44 ± 0.13	.356 (<.001)	—	.074	.044[d]	.419[d]	−.040[d]
		.316 (<.001)	−.037 (.068)	.142	.035[d]		

[a]LELM, lower extremity lean mass; SD, standard deviation; AP, anterior-posterior; ANT, anterior; PST, posterior; VV, varus-valgus; VAR, varus; VAL, valgus; IER, internal-external; IR, internal; ER, external; LAX, laxity; K-LO, low stiffness; K-HI, high stiffness.
[b]Significant at $P < .10$.
[c]Significant at $P < .20$.
[d]Significant at $P < .05$.

In the frontal plane, LELM explained 49% to 56% of the variance in VV_{LAX}, $VAR_{K\text{-}LO}$, and $VAL_{K\text{-}LO}$ (all $P < .001$). The strength of these relationships was relatively unchanged once sex was removed from the model (range in R^2 change = .008-.048). Lower extremity lean mass explained less of the variance in $VAR_{K\text{-}HI}$and $VAL_{K\text{-}HI}$, both in terms of the magnitude of the R^2 values (explaining 39% and 19% of the variance, respectively) and size of the coefficients (Table 2). Further, the relationship between LELM and $VAR_{K\text{-}HI}$ was dependent on a person's sex (ie, significant LELM × sex interaction). When interpreting this interaction, a 4-kg increase in LELM (representing a magnitude of change in LELM of 1 SD from the sample mean of 8.4 ± 2.0 kg) resulted in a 26.8% and 75.8% increase in $VAR_{K\text{-}HI}$ in women and men, respectively. Thus, LELM was a stronger predictor of greater $VAR_{K\text{-}HI}$ in men than it is in women.

In the transverse plane, LELM was a significant predictor of transverse plane knee laxity and stiffness; however, the strength of these relationships was generally less than that observed in the frontal plane (R^2 range = .224-.356; all $P < .001$). Further, the relationship between LELM and transverse plane knee laxity and stiffness tended to be more dependent on the person's sex, as the coefficient for LELM × sex reached significance for $ER_{K\text{-}LO}$ and $ER_{K\text{-}HI}$ ($P < .05$) and neared significance for IER_{LAX}, $IR_{K\text{-}LO}$, and $IR_{K\text{-}HI}$ (P range = .060-.085), and the strength of the coefficient for LELM tended to be smaller once sex and LELM × sex were removed from the model (Table 2). When interpreting the interaction between LELM and sex for $ER_{K\text{-}LO}$ and $ER_{K\text{-}HI}$, a 4-kg increase in LELM (representing a magnitude of change in LELM of 1 SD from a sample mean of 8.4 ± 2.0 kg) resulted in a 43.4% and 33.1% increase in $ER_{K\text{-}LO}$ and $ER_{K\text{-}HI}$ for women but only a 12.6% and 3.4% increase in $ER_{K\text{-}LO}$ and $ER_{K\text{-}HI}$ for men, respectively.

DISCUSSION

Based on prior research findings,[26,38,46] our expectation was that LELM would be a stronger predictor of frontal and transverse plane laxity and incremental stiffness than the sagittal plane and that associations between LELM and stiffness would be stronger at lower force increments. Our primary findings largely support our hypotheses in that LELM explained anywhere from 18% to 56% of the variance in VV and IER laxity and stiffness but less than 8% of the variance in AP laxity and stiffness. Further, associations between LELM and stiffness tended to be stronger at the lower force increments for frontal plane stiffness; however, this trend was not evident in the transverse plane where associations were similar between low and high stiffness increments.

To our knowledge, this is the first study to examine associations between LELM and sagittal, frontal, and transverse plane in vivo knee laxity measures. However, others have reported associations between BMI with VV_{LAX} and IER_{LAX} [38] and between body mass and IER_{LAX} and IER_K,[26] anthropometric variables that we found in our data to be low to moderately correlated with LELM (r = .67 for body mass; r= .36 for BMI). The lack of associations we observed between LELM and AP laxity and stiffness appears to be consistent with prior studies that reported no associations between BMI or body mass with anterior knee laxity.[38,46] The reason for this lack of association is not entirely clear. While studies examining changes in resting muscle tone before and after anesthesia report little to no change in AP_{LAX}in healthy knees,[14,25,36,43] similar studies have not been conducted for VV_{LAX} and IER_{LAX}. It may also be that the inherent nature of the measurement plays a role; whereas AP_{LAX} measures the linear arthrokinematic translation of the tibia relative to the femur, VV_{LAX} and IER_{LAX} measure the osteokinematic joint rotations in the frontal and transverse planes, respectively. While more work is needed to understand the collective passive and active contributions to each of these measures, current findings would suggest that the lower anterior laxity values previously observed in maturing males versus maturing females[1,40] are likely not caused by their emerging differences in muscle mass. However, it is also possible that these associations may be confounded by other sex-dependent factors (eg, lower extremity alignment, hormones) that are also emerging during this time, which have been reported to influence AP laxity and stiffness.[38,39] Further work is needed to address these collective contributions.

The stronger associations we observed between LELM with frontal and transverse plane knee laxity and stiffness suggest that LELM may play a greater role in resisting VV and IER rotational displacements and potentially explain the disproportionally higher VV and IER laxity and lower stiffness values as compared with AP laxity and stiffness observed in females versus males.[15,37,42] This is based on our findings that sex was typically a weak or nonsignificant predictor in the regression models when LELM was also accounted for, and removing sex (and its interaction with LELM) from the model typically had a negligible effect on the variance in frontal and transverse plane knee laxity explained by LELM (ie, R^2 change was less than 5% and only significant for $VAR_{K\text{-}HI}$). However, there were isolated cases where the influence of LELM was sex dependent, with LELM being a stronger predictor of $VAR_{K\text{-}HI}$ in men and $ER_{K\text{-}LO}$ and $ER_{K\text{-}HI}$ in women. While it is difficult to explain these findings based on the current data alone, these sex-specific associations may in part reflect the interplay between LELM and other known sex differences in anatomy (eg, joint geometry,[11] lower extremity alignment[28]) that may influence knee motion patterns,[24] thus the chronic stresses imposed on the ligaments during weightbearing activity.

While we found no comparative studies examining frontal plane laxity and stiffness, our findings of moderate associations between LELM and transverse plane knee laxity and stiffness appear to be consistent with the findings of Mouton et al,[26] who observed strong associations between body mass and sex with transverse plane knee laxity measured at 5-N·m torques (R^2 = .55) and knee stiffness when measured at low loads (2- to 5-N·m torques; R^2 = .38-.43) and high loads (5- to 10-N·m torques; R^2 = .22-.24). However, they tended to observe stronger associations based on body mass and sex than what we observed for LELM and sex, and sex appeared to be an equally important predictor in their models. Thus, we reanalyzed our data using body mass and sex (and the interaction of sex × body mass) to determine if overall body mass may be a stronger predictor than LELM. These secondary analyses revealed that the R^2values we obtained when predicting IER laxity and stiffness with body mass and sex (R^2 range = .19-.33) and with body mass once sex was removed (R^2 range = .18-.31) were similar in magnitude to what we observed for LELM and sex and LELM alone (Table 2). However, when examining VV laxity and stiffness, the variance explained by body mass and sex tended to be lower (R^2 range = .22-.46) and decreased considerably more when sex was removed from the model (R^2 = .12-.34) as compared with our original models with LELM (Table 2). Thus, other study characteristics (eg, difference in study participant demographics, measurement approach to IER laxity and stiffness) are more likely to explain these differences. One potential explanation is that they used the average laxity and stiffness values of the left and right limbs, which may have reduced the measurement error somewhat, thus strengthening the correlations among variables.

The current findings as well as those of Mouton et al[26] may have important clinical implications for ACL injury prevention strategies. Greater magnitudes of knee joint laxity have been consistently associated with a greater risk of ACL injury,[‖] and the greater magnitudes of frontal and transverse plane knee laxity in females have been associated with elements of dynamic knee valgus during the early phase of landing.[41] However, knee laxity to this point has been largely considered a nonmodifiable anatomic risk factor and has yet to receive attention in our ACL prevention strategies. As LELM appears to explain a substantial amount of variance in transverse and frontal plane knee laxity and stiffness, it may be possible to reduce laxity and increase stiffness in the frontal and transverse planes through strength training interventions that promote

changes in muscle characteristics (eg, muscle cross-sectional area, intrinsic muscle stiffness) that have the potential to contribute to static knee joint stability, thus dynamic joint stability during sport activity. Although neural adaptations are predominately responsible for strength changes in the early stages of strength training, increased cross-sectional area of skeletal muscle fibers (fiber hypertrophy) is generally regarded as the primary adaptation to long-term strength training (see review by Folland and Williams[8]). This can facilitate an increase in the number of cross bridges arranged in parallel, which has been associated with greater intrinsic (passive) stiffness properties of the muscle.[31] Such long-term strength training interventions may be of particular relevance to maturing females who, compared with males, develop more fat mass but not lean mass during this stage of development and who maintain higher magnitudes of knee laxity.[1,40] Yet, while a reduction in injury rates is typically observed in ACL prevention programs that include a traditional strengthening component,[10,12,22,30] the specific benefit of these strength training components on risk factor modification (of which knee laxity is only one of many risk factors proposed) has not yet been fully elucidated. Further research is needed to examine the extent to which changes in muscle characteristics in response to strength training interventions may influence transverse and frontal plane knee joint laxity and stiffness in a physically active female population.

This study was limited to associations between LELM and knee joint laxity in an effort to further discern the contributions of body mass and composition to interparticipant differences in multiplanar knee laxity.[26,38,46] However, as previously mentioned, it is acknowledged that other factors (eg, hormones, structural alignment, joint geometry) may interact with LELM to differentially load capsuloligamentous structures of the knee and influence the mechanical properties of the ligament that may also contribute to interparticipant differences in multiplanar knee laxity. Further, other intrinsic muscle properties that may not be solely related to LELM (eg, muscle stiffness, strength ratios, muscle architecture, etc) were not examined, and it is unknown if these characteristics may also contribute to static joint stability. Further research is needed to fully elucidate the combined contributions to multiplanar knee laxity and stiffness.

FOOTNOTES

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§ References 5, 13, 16, 20, 27, 32, 34, 45, 50.

‖ References 5, 13, 16, 20, 27, 32, 34, 45, 50.

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