

Investigating the effects of age and osteoarthritic bone damage on the size of knee joint ligaments: a cadaveric study

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Background: This study investigated the impact of age and osteoarthritis on knee ligament dimensions including the patellar, cruciate, and collateral ligaments.

Methods: Measurements were taken using digital calipers in 18 dissected cadaveric knee joints, including knee joint ligament lengths, thicknesses, and widths, as well as tibial condylar width and intercondylar notch width. Percent degeneration of the articular cartilage due to osteoarthritis was assessed with surface area measurements.

Results: When controlling for tibial condylar width and age in a linear regression, the cartilage degeneration due to osteoarthritis is significantly negatively correlated with the intercondylar notch width, lengths of the anterior cruciate and medial collateral ligaments, and the width of the anterior cruciate ligament. Results of the linear regression also indicate a significant positive correlation between tibial condylar width and the anterior cruciate ligament width and the lengths of the patellar ligament, both cruciate ligaments, and both collateral ligaments. Surprisingly, age positively correlated with the lengths of the anterior cruciate and medial collateral ligaments, and the width and thickness of the anterior cruciate ligament, suggesting possible compensatory hypertrophy in response to joint instability.

Conclusion: The findings emphasize that osteoarthritis affects knee ligaments' structure, potentially leading to ligament atrophy and decreased joint stability. Clinically, these results highlight the need for comprehensive assessment and treatment strategies that address all joint tissues. Further research with larger and more diverse samples and advanced imaging techniques is recommended to deepen understanding and improve management of knee osteoarthritis and ligament pathologies.

Background

Cartilage is a connective tissue found throughout the human body including the spine, airways, synovial joints, growth plates, and the nose.¹ In humans, there are three types of cartilage: hyaline, fibrous, and elastic cartilage, which play important roles in bearing loads in joints, forming structures like the nose and external ears, joint lubrication, and the development and growth of long bones.¹ There are diseases, like osteoarthritis (OA) that affect cartilage.² Among the adult population,

OA is a highly prevalent, painful, and progressive musculoskeletal condition that most commonly affects the hand, hip, and knee joints.^{2,3,4} In Canada, 3.9 million people over the age of 20 years old have diagnosed OA,⁵ with the knee joint being the most commonly affected joint.² OA is commonly characterized by progressive cartilage degeneration in a joint accompanied by changes in the underlying bone and with a decrease in joint space.^{2,4,6}

OA results in a variety of disturbances in an individu-

al's life, including pain, stiffness, swelling, and changes in their gait, which can put them at an increased fall risk.^{2,3,7} Historically, it was thought that OA only affected the bone surfaces in joints, because the cartilage degeneration due to OA was readily visible upon examination of dissected arthritic joints.³ Due to the obvious degenerative changes in bone surfaces affected by OA, the severity of OA is commonly graded using the International Cartilage Repair Society grading system, which bases the severity of OA on the depth of cartilage degeneration.³ Research has shown that the severity of OA is positively correlated with age.^{3,7}

In addition to the well-known association of cartilage changes with arthritis, there are other tissue changes within joints that are associated with OA that are not commonly used to gauge the severity of the disease.³ Further research into the disease has revealed that OA affects more than the bone surfaces in joints; OA is now classified as a whole-joint disease because it affects almost all of the tissues within joints.^{4,6,8,9} For example, histological examinations of anterior cruciate ligaments (ACL) in knee joints and MRI analyses of collateral ligaments in patients with OA have shown that OA is associated with degenerative changes in the ligaments of the knee and knee joints.⁴ This study also showed that bony insertion sites for collateral ligaments and adjacent bones had pathological changes associated with OA.⁴

There has been research into the effects of Body Mass Index (BMI), knee joint size, and sex on the size of the ACL in the knee joint.¹⁰ It was found that an increased BMI was associated with a larger ACL.¹⁰ Similarly, relationships around knee joint size, measured using bicondylar width and intercondylar notch, were confirmed and showed increased ACL size with increased knee joint size.¹⁰ The study found that BMI and bicondylar width together could predict ACL volume and mean cross-sectional area.¹⁰ Furthermore, after adjusting for BMI and bicondylar width, women had significantly smaller average ACL volume, cross-sectional area, and length when compared to men.¹⁰ Further research into how to normalize ligament size to joint size, because it has been shown that larger joint sizes are associated with larger ACL sizes,¹⁰ found that femoral epicondylar width measured on MRIs and radiographs could be used to predict the ACL length.¹¹

Despite this association of age, OA, and ligament degeneration in the knee joint, there is limited research on how age and OA affect the size of ligaments in the knee joint. Regarding ligament mechanics, research has shown that OA impairs the integrity of tissues in the knee joint such as the ACL and posterior cruciate ligament (PCL) as well as the medial and lateral collateral ligaments (MCL and LCL, respectively).¹² Additionally, there have been investigations into how factors like

sex and body size affect ACL size,¹⁰ but there is limited literature on whether OA and age affect the length and width of the cruciate and collateral ligaments in the knee joint. This study aims to fill in this knowledge gap by investigating how age and percentage of bone surfaces affected by arthritis in the knee joint affect the size of cruciate and collateral ligaments in knee joints.

Materials & Methods

Cadaver Specimens

Research Ethics Board (REB) approval was obtained for the study through Dalhousie University (REB file #: 2023-6581). This study utilized a total of 18 dissected knee joints obtained from ten human cadavers provided by the Human Body Donation Program at Dalhousie University. The cadavers are embalmed with a solution consisting of 4% formaldehyde, 65% ethanol 190 proof, 5% phenol, 21% propylene glycol, 1% sodium acetate, and 4% Dettol. The embalming process is completed by injecting 30-50 litres of the chemical solution through the femoral artery. Fourteen of these dissections were already available in the Dalhousie University gross anatomy lab, and four additional knee joints were dissected specifically for this study during the summer of 2023. There was a total of eight left and ten right knee joints. The mean age of the cadavers at death was 84.94 years (range 72-102 years). The time from embalment to dissection ranged from 4 months to 29 months (mean 12.2 months). Specimens with partial or total knee replacements were excluded from the study.

Measurements

The specimens were carefully dissected following standard anatomical procedures to expose the necessary ligaments, the overall knee joint, and the patellar and femoral condylar surfaces. Each measurement (Table 1) was taken by the same researcher a total of three times on different days using digital calipers. The three measurements were recorded in millimeters (mm) and averaged for comparative analysis. The following measurements were taken:

For measurement of the LCL (Figure 1) and MCL the knee joint was placed in full extension.¹⁷ The length of the LCL was measured as the linear distance from the most proximal portion of its femoral attachment to the most distal portion of its fibular attachment (LCL-L).^{17,18} The length of the MCL was measured as the linear distance from the most proximal portion of its femoral attachment to the most distal portion of its fibular attachment (MCL-L).¹⁷ The LCL-T and MCL-T were the largest medial-lateral measurements of the LCL^{17,18} and MCL¹⁷, respectively.

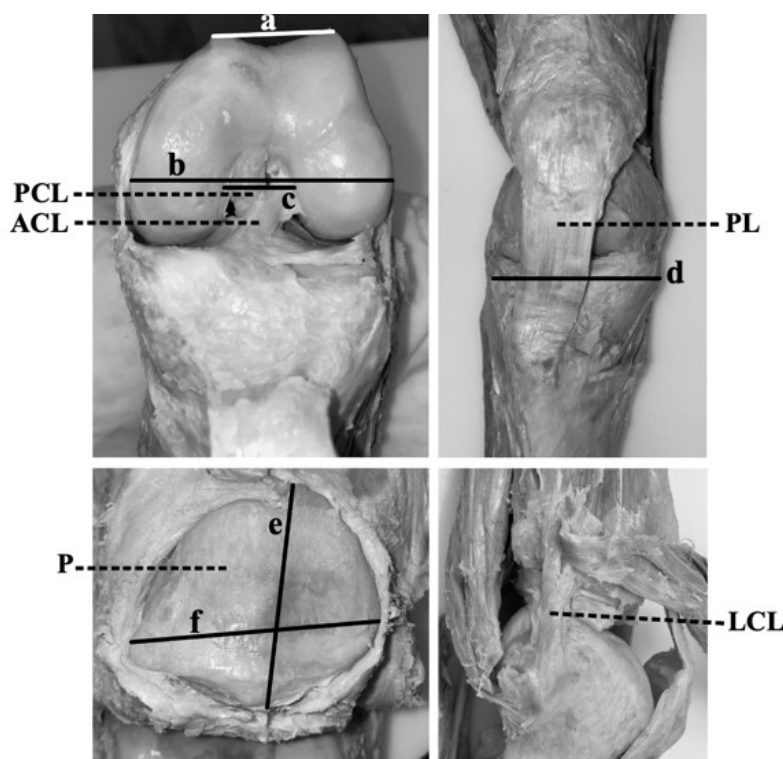
For measurements of the ACL (Figure 1) the knee joint was flexed to 90° and for measurements of the PCL (Figure 1) the knee joint was fully extended. ACL-L¹⁴ and PCL-L were measured as the largest linear dis-

Table 1 | Measurements Obtained from Specimens.

Measurement	Abbreviation	Measurement (cont.)	Abbreviation (cont.)
Femoral epicondylar width	FCW	Lateral collateral ligament length	LCL-L
Medial-lateral tibial condylar width	TCW	Lateral collateral ligament thickness	LCL-T
Intercondylar notch width	INW	Medial collateral ligament length	MCL-L
Trochlear groove width	TGW	Medial collateral ligament thickness	MCL-T
Patellar width	PW	Anterior cruciate ligament length	ACL-L
Patellar height	PH	Anterior cruciate ligament width	ACL-W
Patellar ligament length	PLL	Anterior cruciate ligament thickness	ACL-T
Patellar ligament thickness	PLT	Posterior cruciate ligament length	PCL-L
Percent surface area of patellar surface of femur affected by osteoarthritis	%FC	Posterior cruciate ligament width	PCL-W
Percent surface area of articular surface of patella affected by osteoarthritis	%P	Posterior cruciate ligament thickness	PCL-T
Total percent surface area (femoral condyles + patella) affected by osteoarthritis	TotOA		

tance between the femoral and tibial attachments of each ligament. The ACL-W and PCL-W were the largest medial-lateral linear distance of the ACL¹⁹ and PCL. Similarly, the ACL-T and PCL-T were the largest anterior-posterior linear distance of the ACL and PCL. To calculate the %FC, %P, and TotOA the knee joints were placed in maximal flexion and a photo was taken

perpendicular to the surface of interest (Figure 2). The femoral condyle and patella photos were uploaded to SketchAndCalc,²⁰ and a scale was created using the TGW and PW measurements, respectively. The total surface area and total surface area affected by arthritis were traced using an Apple iPad Pro (Figure 2). Areas showing any signs of OA regardless of severity were in-

**Figure 1 | Figure labelling structures and showing the various measurements taken from each knee joint prosection.**

a = trochlear groove width (TGW), b = femoral epicondylar width (FCW), c = intercondylar notch width (INW), d = medial-lateral tibial width (TCW), e = patellar height (PH), f = patellar width (PW), PCL = posterior cruciate ligament, ACL = anterior cruciate ligament, P = patella, PL = patellar ligament, LCL = lateral collateral ligament

cluded in the affected areas. The total mm² were converted to percentage of surface area affected by OA.

Statistical Analysis

Statistical analyses of the measurements obtained during data collection were made using IBM SPSS v. 20 software.²¹ Alongside descriptive statistics, the relationships between parameters not including ligament sizes were evaluated using 2-tailed Pearson correlation analysis. Because ligament sizes depend on the size of the knee joint and age,¹⁰ standard linear regression analyses were performed to adjust for the effects of covariates when analyzing the relationship between ligament sizes and arthritic surface areas of the knee joints. Any values with $p < 0.05$ were considered statistically significant in this study.

Results

In total, 18 specimens (8 left and 10 right knee joints) with a mean age of 84.94 years (range 72–102 years) were included in this study. The mean, standard deviation, and minimum to maximum range of the measurements taken from each knee joint prosection are demonstrated in Table 2. The mean lengths of the patellar, lateral collateral, medial collateral, anterior cruciate, and posterior cruciate ligaments were 78.49 mm, 63.48 mm, 89.99 mm, 35.89 mm, and 36.13 mm, respectively (Table 2). The mean thickness values of the patellar, lateral collateral, medial collateral, anterior cruciate, and posterior cruciate ligaments were 3.51 mm, 1.87 mm, 1.87 mm, 3.95 mm, and 4.05 mm, respectively (Table 2). The mean width of the ACL was 8.85 mm, and the PCL average width was 11.43 mm (Table 2). The widest TCW was 80.53 mm on average, with an average INW of 20.09 mm (Table 2). The mean percentage of surface

areas of the articular surface of the patella and the patella surface of femur affected by arthritis were 64.40% and 39.10%, respectively, giving an average combined arthritic surface area of 45.82% (Table 2).

A Pearson correlation analysis was used to determine the relationship between tibial condylar width and ligament size parameters. The results of the Pearson correlation analysis show a statistically significant positive relationship of TCW with LCL length, MCL length, ACL length, PCL length, and PCL width (Table 3). ACL length was most positively correlated with TCW ($r = 0.803$, $p < 0.001$). There was no significant correlation between ligament thickness values, PLL, or ACL-W (Table 3).

There was no significant correlation between age and femoral condylar or dorsal patellar surface area affected by OA (Table 4). Contrarily, the femoral condylar and dorsal patellar surface areas affected by OA were statistically positively correlated with each other ($r = 0.755$, $p < 0.001$; Table 4).

The following results of a multivariate linear regression analysis of total surface area affected by OA, tibial condylar width, and age with knee joint parameters are shown in Table 5. The derived prediction model for each parameter included the TotOA, TCW, and age. Of the dependent variables analyzed, the INW, MCL length, ACL length, and ACL width had statistically significant negative correlations with TotOA while controlling for TCW and age (Figure 3). The model predicting intercondylar notch width explained 18.7% of the variability in INW. The models predicting MCL length, ACL length, and ACL width explained 64.5%, 77.6%, and 25.7% of the variability in these outcomes, respectively. Using this linear regression model, for every one percent increase

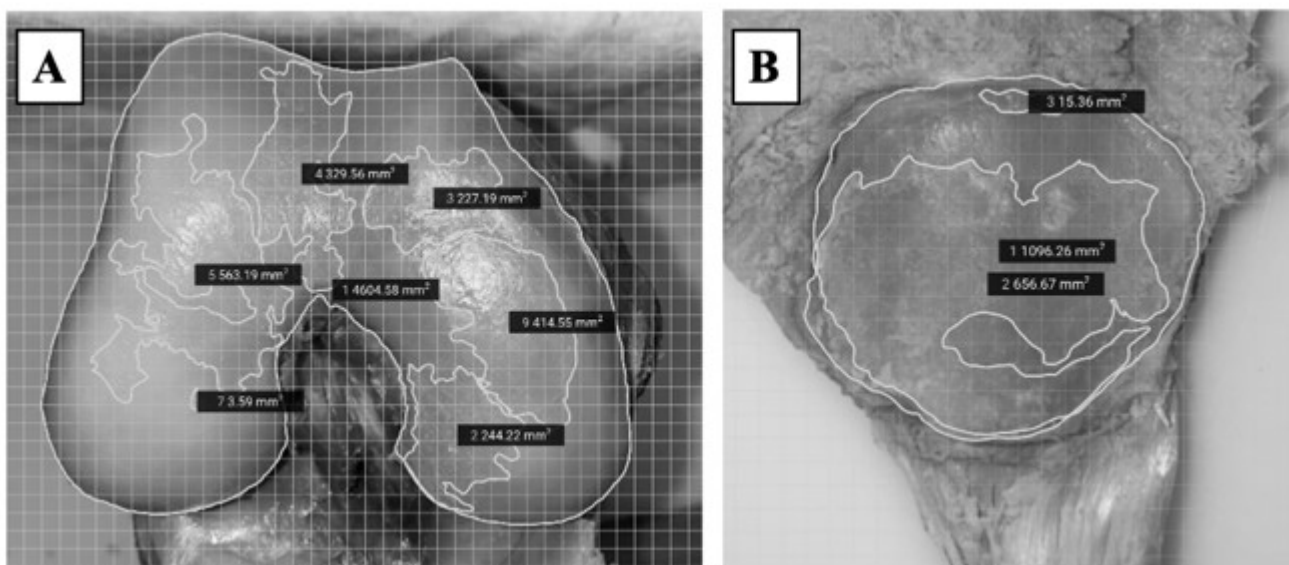


Figure 2 | Photos used for the determination of percent surface area of femoral condyles affected by osteoarthritis, %FC, (A) percent surface area of dorsal surface of patella affected by osteoarthritis, %P, (B) with image analysis using SketchAndCalc area calculator software.²⁰

Table 2 | Anatomical Measurements of the Specimens.

	Left limbs (n=8)		Right limbs (n=10)		All limbs (n=18) ^a	
	Mean (SD)	Range	Mean (SD)	Range	Mean (SD)	Range
Age	86.37 (8.193)	75-102	83.80 (9.07)	72-102	84.94 (8.54)	72-102
FCW	85.59 (6.89)	75.70-95.43	84.58 (6.92)	75.82-94.79	85.03 (6.72)	75.70-95.43
TCW	80.90 (6.69)	71.05-89.62	80.22 (6.69)	72.37-93.07	80.53 (6.50)	71.05-93.07
INW	19.97 (2.91)	16.58-23.93	20.20 (2.53)	16.15-23.86	20.09 (2.63)	16.15-23.93
TGW	38.04 (3.88)	31.69-42.80	39.28 (4.12)	31.37-44.84	38.73 (3.95)	31.37-44.84
PW	46.36 (6.57)	36.82-56.99	47.29 (7.26)	39.46-59.10	46.87 (6.78)	36.82-59.10
PH	44.41 (4.17)	38.80-49.77	44.69 (5.12)	38.25-51.91	44.57 (4.59)	38.25-51.91
PLL	78.45 (10.72)	60.68-92.80	78.52 (12.36)	60.64-100.93	78.49 (11.32)	60.64-100.93
PLT	3.59 (0.42)	3.06-4.28	3.44 (1.16)	2.19-6.36	3.51 (0.89)	2.19-6.36
%FC	38.69 (19.30)	3.60-66.95	39.43 (21.07)	9.36-71.93	39.10 (19.71)	3.60-71.93
%P	70.82 (18.36)	29.30-90.41	59.26 (23.59)	8.61-86.63	64.40 (21.64)	8.61-90.41
TotOA	47.03 (18.49)	10.50-73.53	44.86 (20.59)	9.18-76.55	45.82 (19.15)	9.18-76.55
LCL-L	63.51 (4.99)	57.22-71.51	63.44 (5.77)	55.49-71.28	63.48 (5.28)	55.49-71.51
LCL-T	1.90 (0.43)	1.34-2.54	1.84 (0.38)	1.41-2.51	1.87 (0.39)	1.34-2.54
MCL-L	89.22 (9.22)	72.00-97.34	90.60 (10.74)	69.49-102.39	89.99 (9.82)	69.49-102.39
MCL-T	1.65 (0.31)	1.32-2.04	2.06 (0.45)	1.28-2.69	1.87 (0.44)	1.28-2.69
ACL-L	37.42 (4.75)	27.56-41.09	34.65 (7.63)	16.23-44.86	35.89 (6.49)	16.23-44.86
ACL-W	9.51 (1.49)	6.97-11.49	8.31 (2.58)	3.11-11.97	8.85 (2.19)	3.11-11.97
ACL-T	4.24 (0.96)	2.34-5.85	3.72 (1.40)	1.36-5.69	3.95 (1.22)	1.36-5.85
PCL-L	37.07 (4.25)	30.19-42.20	35.38 (4.30)	30.19-43.28	36.13 (4.24)	30.19-43.28
PCL-W	11.50 (1.73)	8.38-13.90	11.37 (1.28)	9.83-13.86	11.43 (1.45)	8.38-13.90
PCL-T	3.85 (1.08)	3.02-6.40	4.21 (1.12)	2.74-6.10	4.05 (1.08)	2.74-6.40

Age is measured in years and surface areas are in percentages (%FC, %P, TotOA). All other units are in mm.
SD (standard deviation of the mean).

See Table 1 for abbreviation meanings.

^a Right and left knee joints from 8 cadavers were dissected for a total of 16 specimens. A single right knee joint from an additional 2 cadavers were dissected, bringing the total sample size to 18 specimens.

Table 3 | Correlations of the tibial condylar width and ligament size parameters.

Variables	Pearson Correlation with TCW	2-tailed Significance (p)
PLL	0.400	0.100
PLT	0.430	0.075
LCL-L	0.701**	0.001
LCL-T	-0.128	0.612
MCL-L	0.700**	0.001
MCL-T	0.158	0.530
ACL-L	0.803**	<0.001
ACL-W	0.121	0.633
ACL-T	-0.170	0.499
PCL-L	0.543*	0.020
PCL-W	0.474*	0.047
PCL-T	0.226	0.367

2-tailed Pearson correlation analysis of tibial condylar width against ligament size parameters. Total sample size = 18. * p < 0.05, ** p < 0.01.
See Table 1 for abbreviation meanings

in the total percent surface area affected by osteoarthritis, the INW decreases by 0.700 mm (p = 0.039), the MCL length decreases by 0.572 mm (p = 0.014), the ACL length decreases by 0.575 mm (p = 0.003), and the ACL width decreases by 0.740 mm (p = 0.025; Table 5). The scatterplots in Figure 3 provide a visual for this negative correlation.

In addition, the width of the ACL and the lengths of the patellar ligament, LCL, MCL, ACL, and PCL were all significantly positively correlated with tibial condylar width. The models predicting patellar ligament length, LCL length, MCL length, ACL length, ACL width, and PCL length explained 20.9%, 39.8%, 64.5%, 77.6%, 25.7% and 19.4% of the variability in these outcomes, respectively. For every one millimeter increase in the tibial condylar width, the PLL increases by 0.860 mm (p = 0.020), the

LCL length increases by 0.781 mm (p = 0.016), the MCL length increases by 1.229 mm (p < 0.001), the ACL length increases by 1.262 mm (p < 0.001), the ACL width increases by 0.812 mm (p = 0.023), and the PCL length increases by 0.774 mm (p = 0.035; Table 5).

Finally, MCL length, ACL length, ACL width, and ACL thickness were all significantly positively correlated with age. The models predicting MCL length, ACL length, ACL width, and ACL thickness explained 64.5%, 77.6%, 25.7%, and 17.9% of the variability in these outcomes, respectively. For every one-year increase in age, the MCL length increases by 0.501 mm (p = 0.015), the ACL length increases by 0.319 mm (p = 0.043), the ACL width increases by 0.663 mm (p = 0.024), and the ACL thickness increases by 0.642 mm (p = 0.035; Table 5).

Table 4 | Correlations of age and percentage of surfaces affected by arthritis.

		Age	%FC	%P
Age	Pearson Correlation	1	0.146	0.119
	Significance (2-tailed)		0.564	0.639
%FC	Pearson Correlation	0.146	1	0.755**
	Significance (2-tailed)	0.564		<0.001
%P	Pearson Correlation	0.119	0.755**	1
	Significance (2-tailed)	0.639	<0.001	

Pearson correlation analysis of age, percentage of femoral condylar surface affected by osteoarthritis, and percentage of patellar surface affected by osteoarthritis.
Total sample size = 18.
** Correlation is significant at the 0.01 level (2-tailed).
See Table 1 for abbreviation meanings.

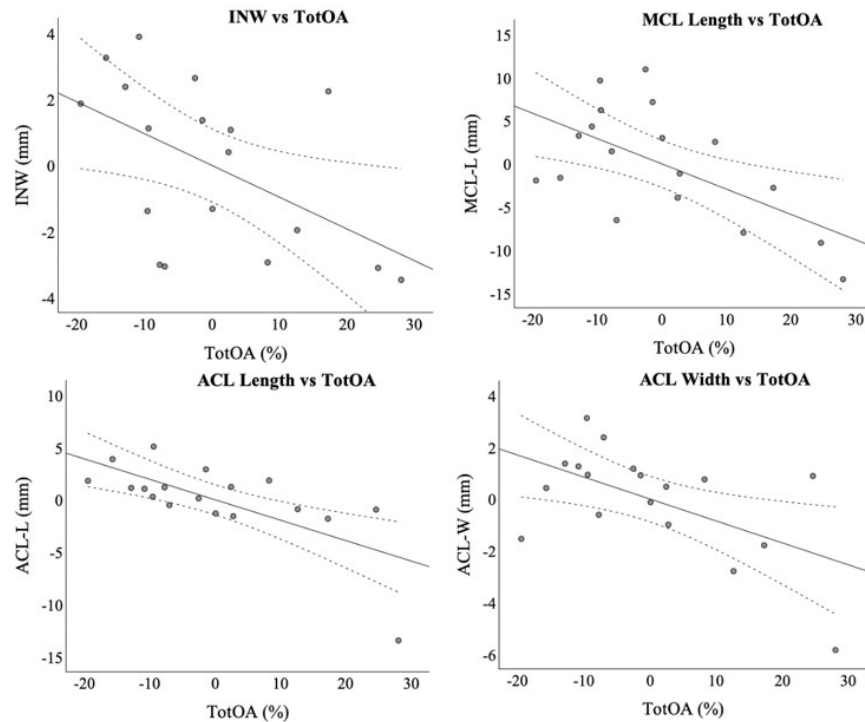


Figure 3 | Linear regression analysis demonstrating the correlation of intercondylar notch width (INW), MCL length (MCL-L), ACL length (ACL-L), and ACL width (ACL-W) with total percentage of surface area affected by osteoarthritis (TotOA).

The regression line (solid line) and corresponding 95% confidence interval (dashed lines) are shown for each regression analysis.

Table 4 | Multivariate linear regression analysis of total surface area affected by osteoarthritis, tibial condylar width, and age with knee joint parameters.

	R ² ^a	F ^b	ANOVA p	TotOA β ^c	p	TCW β	p	Age β	p
PLL	0.209	2.499	0.102	-0.439	0.171	0.860*	0.020	0.521	0.074
PLT	0.025	1.144	0.366	0.053	0.877	0.434	0.253	0.087	0.775
INW	0.187	2.302	0.122	-0.700*	0.039	0.405	0.243	-0.009	0.974
LCL-L	0.398	4.754	0.017	-0.044	0.872	0.781*	0.016	0.139	0.565
LCL-T	0.014	1.083	0.388	-0.549	0.128	0.223	0.552	0.084	0.785
MCL-L	0.645	11.309	<0.01	-0.572*	0.014	1.229**	<0.001	0.501*	0.015
MCL-T	-0.137	0.317	0.813	-0.007	0.986	0.247	0.539	0.217	0.514
ACL-L	0.776	20.607	<0.001	-0.575**	0.003	1.262**	<0.001	0.319*	0.043
ACL-L	0.257	2.962	0.068	-0.740*	0.025	0.812*	0.023	0.663*	0.024
ACL-T	0.179	2.234	0.129	-0.572	0.086	0.414	0.235	0.642*	0.035
PCL-L	0.194	2.361	0.115	-0.265	0.402	0.774*	0.035	0.195	0.485
PCL-W	0.177	2.219	0.131	-0.358	0.267	0.666	0.066	-0.040	0.886
PCL-T	-0.073	0.614	0.617	-0.246	0.499	0.491	0.219	0.310	0.340

Multivariate regression analysis demonstrating the relationship of TotOA, TCW, and age with knee joint parameters.

* Correlation is significant at the 0.05 level. ** Correlation is significant at the 0.01 level.

^a R² = Adjusted R Square of model

^b F = F-Statistic

^c β = Standardized Coefficients Beta (95% confidence interval)

Total sample size = 18.

See Table 1 for abbreviation meanings.

Discussion

Summary and interpretation of findings

This study aimed to investigate the impact of age and OA on the size of knee ligaments, specifically the patellar, cruciate, and collateral ligaments. Our findings indicate several key points:

- 1. Correlation of tibial condylar width with ligament sizes:** As shown in Table 3, TCW is significantly positively correlated with the width of the PCL and the lengths of the LCL, MCL, ACL, and PCL. The positive correlation between TCW and ligament dimensions (Table 3) aligns with previous studies that have shown larger knee joints have larger ligaments.¹⁰ In previous studies, FCW was used to approximate knee joint size; however, Pearson correlations conducted for this study imply that TCW is an equivalent approximation of knee joint size compared to FCW. This relationship is crucial for understanding how anatomical variations in knee joint size can influence ligament mechanics and potentially affect joint stability and susceptibility to injury. Furthermore, when controlling for age and TotOA, the TCW, which is used as an indication of knee joint size, is significantly positively correlated with the width of the ACL and the lengths of the patellar ligament, LCL, MCL, ACL, and PCL (Table 5). This finding supports that larger knee joints, as indicated by TCW, tend to have longer ligaments and a wider ACL, highlighting the importance of joint size in ligament anatomy and possibly joint stability and function.
- 2. Correlation of arthritic surfaces and age:** Pearson correlation analysis shows that age does not significantly correlate with the TotOA (Table 4). Contrarily, the percentage of OA affecting the two measured surface areas, the femoral condyles and dorsal surface of patella, are significantly positively correlated with each other ($r = 0.755$, $p < 0.001$, Table 4). This finding enables the combination of both surface areas, the TotOA, to be used for subsequent linear regression analysis.
- 3. Impact of osteoarthritis on ligament sizes:** When controlling for age and knee joint size (TCW), there is a negative correlation between the TotOA and the lengths of the MCL and ACL, as well as the width of the ACL (Table 5, Figure 3). It indicates that as OA progresses, the ligaments within the knee joint, specifically the MCL and ACL, undergo degenerative changes leading to a reduction in their size. This could be due to chronic inflammation⁷ and mechanical stress associated with OA,⁶ which may result in ligament atrophy or degradation over time.^{8,9} These changes could contribute to the overall decline in joint function and stability seen in patients with advanced OA. This finding underscores the degenerative impact of OA on knee ligament structure, sug-

gesting that OA progression may lead to ligament atrophy or structural compromise.

- 4. Age-related changes in ligament size:** Age is positively correlated with the lengths of the MCL and ACL, as well as the width and thickness of the ACL (Table 5). The finding that ligament dimensions increase with age is somewhat unexpected, given that aging is typically associated with tissue degeneration.²² One possible explanation is that as individuals age, compensatory mechanisms such as ligament hypertrophy may occur in response to chronic joint instability or degeneration. This hypertrophy could be an adaptive response to maintain joint stability despite the degenerative changes associated with aging and OA.^{6,22}

Clinical Implications

The study's findings reinforce the understanding that OA is not merely a disease affecting the cartilage but a whole-joint disease impacting various joint tissues, including ligaments.^{4,8,9} This comprehensive view has several clinical implications. Firstly, clinicians should assess not only the cartilage but also other joint structures such as ligaments, bone surfaces, and surrounding tissues in patients with OA. This may require updating imaging and diagnostic approaches to provide a more comprehensive view of joint health. Secondly, treatment plans should address the multifactorial nature of OA. For instance, physical therapy regimens could focus on strengthening ligaments and muscles surrounding the joint, and pharmacological treatments might need to be complemented with interventions targeting different joint tissues.

Additionally, the study demonstrates that both age and OA severity influence the size and morphology of knee ligaments,²² which has further implications. Clinicians should consider the patient's age when evaluating knee ligament health, as age is positively correlated with changes in ligament size. Standard reference values for ligament dimensions might need to be age-adjusted to improve diagnostic accuracy. Rehabilitation programs could be tailored based on the patient's age and OA severity, optimizing recovery and functionality, with specific exercises aimed at preserving ligament integrity and joint stability for older patients or those with advanced OA.

The finding that OA severity negatively correlates with ligament size emphasizes the need for early and targeted interventions. Early detection of OA and prompt intervention can potentially slow down ligament degeneration, preserving joint function. Regular monitoring of patients with OA can help in adjusting treatment plans to mitigate the impact on ligaments. Clinicians should adopt a holistic approach in managing OA, considering the interconnectedness of joint tissues and the cumula-

tive impact on joint health. Strategies aimed at reducing OA progression could have a protective effect on ligaments, contributing to overall joint stability and mobility.

Limitations and Future Directions

There are several limitations to this study that should be acknowledged. The sample size was relatively small, and all specimens were from elderly individuals, which may limit the generalizability of the findings to younger populations. Additionally, the study relied on cadaver specimens that were embalmed, which may not fully capture the dynamic properties of ligaments in living individuals. Future research should aim to include a larger and more diverse sample size, including younger individuals and patients with varying degrees of OA severity. Longitudinal studies tracking ligament changes over time in living patients could provide more comprehensive insights into the progression of OA and age-related changes in ligament dimensions. Advanced imaging techniques, such as Magnetic Resonance Imaging (MRI), could be utilized to non-invasively assess ligament properties in vivo.

The study acknowledges the variability in OA severity among the specimens, ranging from light cartilage wear to more extensive bony involvement. Due to technological limitations at the research location, the study used percentages of affected surface areas as a proxy for OA severity. This approach allowed for a quantitative assessment of OA severity; however, it is recognized that the inability to measure the depth of erosion objectively is a limitation of the study. Future research could benefit from more advanced imaging techniques or histological analysis to quantify both the surface area and depth of cartilage erosion, providing a more comprehensive assessment of OA severity and its relationship to ligament changes.

The use of prediction models in this study, while providing valuable insights, has several limitations that warrant discussion. Firstly, the linear regression model's prediction may not accurately reflect the realistic progression of OA in the knee joint. This prediction could lead to implausible outcomes, such as a 14 mm decrease in INW for a 20% increase in OA-affected area, which is not physiologically feasible given the mean INW of approximately 20 mm. Similarly, the model's suggestion that the MCL length increases by 0.5 mm for each year of age is biologically improbable, as it would imply a 5 mm increase over a decade in adulthood when skeletal growth has ceased. These unrealistic predictions highlight the limitations of applying linear models to complex biological systems, especially with a small sample size. Additionally, the models may not account for other factors influencing ligament size, such as individual variability, lifestyle, and genetic predisposition. Future research should aim to develop more sophisticated models that incorporate non-linear relationships

and a broader range of variables, using larger and more diverse sample sizes to improve the accuracy and applicability of predictions in knee joint OA progression and its effects on ligamentous structures.

Conclusion

This study contributes to the growing body of knowledge on the anatomical and pathological changes associated with OA and aging in the knee joint. The findings highlight the complex interplay between joint size, OA severity, and ligament dimensions, offering valuable insights for clinical practice and future research. Understanding these relationships is essential for improving the diagnosis, treatment, and management of knee OA and associated ligament pathologies.

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