Journal of Athletic Training 2023;58(2):136–142
doi: 10.4085/1062-6050-0520.21

www.natajournals.org

Mechanical and Sensorimotor Outcomes Associated With Talar Cartilage Deformation After Static Loading in Those With Chronic Ankle Instability

Kyeongtak Song, PhD, ATC*†; Brian Pietrosimone, PhD, ATC†; J. Troy Blackburn, PhD, ATC†; Darin A. Padua, PhD, ATC†; Joshua N. Tennant, MD‡; Erik A. Wikstrom, PhD, ATC†

*Department of Physical Education, Yonsei University, Seoul, Republic of Korea; †MOTION Science Institute, Department of Exercise and Sport Science and ‡Department of Orthopaedics, University of North Carolina at Chapel Hill

Context: Those with chronic ankle instability (CAI) demonstrate deleterious changes in talar cartilage composition, resulting in alterations of talar cartilage loading behavior. Common impairments associated with CAI may play a role in cartilage behavior in response to mechanical loading.

Objective: To identify mechanical and sensorimotor outcomes that are linked with the magnitude of talar cartilage deformation after a static loading protocol in patients with and those without CAI.

Design: Cross-sectional study.

Setting: Laboratory setting.

Patients or Other Participants: Thirty individuals with CAI and 30 healthy individuals.

Main Outcome Measures(s): After a 60-minute off-loading period, ultrasonographic images of the talar cartilage were acquired immediately before and after a 2-minute static loading protocol (single-legged stance). Talar cartilage images were obtained and manually segmented to enable calculation of medial, lateral, and overall average talar thickness. The percentage change, relative to the average baseline thickness, was used for further analysis. Mechanical (ankle joint laxity) and sensorimotor (static balance and Star Excursion Balance Test) outcomes were captured. Partial correlations were computed to determine associations between cartilage deformation magnitude and the mechanical and sensorimotor outcomes after accounting for body weight.

Results: In the CAI group, greater inversion laxity was associated with greater overall ($r = -0.42$, $P = .03$) and medial ($r = -0.48$, $P = .01$) talar cartilage deformation after a 2-minute static loading protocol. Similarly, poorer medial-lateral static balance was linked with greater overall ($r = 0.47$, $P = .01$) and lateral ($r = 0.50$, $P = .01$) talar cartilage deformation. In the control group, shorter posterolateral Star Excursion Balance Test reach distance was associated with greater lateral cartilage deformation ($r = 0.42$, $P = .03$). No other significant associations were observed.

Conclusions: In those with CAI, inversion laxity and poor static postural control were moderately associated with greater talar cartilage deformation after a 2-minute static loading protocol. These results suggest that targeting mechanical instability and poor balance in those with CAI via intervention strategies may improve how the talar cartilage responds to static loading conditions.

Key Words: ankle laxity, balance, Star Excursion Balance Test, ultrasonography

Key Points

- Greater inversion laxity and poor static postural control were associated with greater talar cartilage deformation after a 2-minute static loading protocol in those with chronic ankle instability.
- Mechanical stability and postural control should be targeted during intervention strategies to restore or improve cartilage loading behavior in those with chronic ankle instability.

Lateral ankle sprains are the most common injury linked with physical activity and athletic participation, accounting for approximately 60% of all injuries during interscholastic and intercollegiate sports.[1] Further, up to 75% of individuals who sprain their ankle develop chronic ankle instability (CAI).[2] Most importantly, CAI is a major contributing factor in the development of posttraumatic ankle osteoarthritis (PTOA),[3] for which there are no effective conservative treatments once the disease becomes severe. Thus, it is critical to identify modifiable factors associated with ankle PTOA progression to develop early interventions that can slow or prevent PTOA in those with CAI.

A hallmark feature of ankle PTOA is a decline in articular cartilage health, which manifests initially as deleterious compositional changes (eg, reduced proteoglycan density, collagen disorganization) in the talar or subtalar cartilage (or both).[4] Compositional changes are theorized to decrease the ability of the cartilage to appropriately respond to mechanical loading (ie, altered cartilage loading behavior)[5] but are difficult and expensive to quantify via specialized magnetic resonance imaging (MRI) sequences. A recent investigation[6] using ultrasonog-

raphy, a more clinically scalable tool, demonstrated altered talar cartilage behavior (ie, greater deformation) in those with CAI relative to an uninjured control group during static and dynamic loading conditions. In addition, the static loading condition resulted in more talar cartilage deformation than the dynamic loading condition.[6,7] Thus, it is important to determine if CAI-related outcomes are linked with increased cartilage deformation after static loading.

Elucidating factors related to altered cartilage behavior in those with CAI could provide clinicians with therapeutic targets, which if addressed, could slow ankle PTOA progression. Authors[8] of current CAI paradigms theorized that structural adaptations (eg, ligament laxity) and sensorimotor dysfunction (eg, static and dynamic postural control) facilitate altered biomechanics and subsequently ankle joint loading patterns, which eventually lead to altered cartilage composition. Therefore, common impairments associated with CAI may play a role in cartilage behavior in response to mechanical loading. Hence, the aim of our investigation was to identify possible associations between ankle joint laxity, static and dynamic postural control, and talar cartilage deformation after a standardized static (ie, standing) loading protocol. We hypothesized that greater ankle joint laxity and poorer postural control (ie, worse static balance, less normalized Star Excursion Balance Test [SEBT] reach distance) would be linked with greater talar cartilage deformation after a 2-minute single-limb stance in those with CAI but not in the control group.

METHODS

Participants

A total of 30 individuals with CAI and 30 healthy individuals between 18 and 35 years of age participated in the study. We defined *CAI* based on International Ankle Consortium guidelines[9]: a history of $\geq$1 ankle sprains, $\geq$2 giving-way episodes within the past 6 months, and a score of $\geq$11 on the Identification of Functional Ankle Instability instrument (IdFAI). If a participant reported bilateral CAI, the limb with worse IdFAI scores was used for testing. Healthy individuals were included if they had no history of ankle sprains or giving-way episodes and scored $<$11 on the IdFAI. For the healthy control group, the *dominant limb*, defined as the limb used for kicking a ball, was tested. We excluded anyone with a history of (1) lower extremity surgery or fracture, (2) balance or vision problems, or (3) acute ($<$12 weeks) or chronic musculoskeletal or head injuries. Self-reported function, as measured by the Foot and Ankle Ability Measure (FAAM), was assessed but not applied as an inclusion criterion.

Procedures

We used a cross-sectional design as part of a larger study.[6] This study focused on the possible mechanisms driving talar cartilage deformation after a static loading protocol in those with CAI. Participants reported for 2 testing sessions separated by 1 week. During the first session, they completed ankle joint laxity and standing postural control assessments. They then returned for an ultrasonographic (US) assessment of talar cartilage thickness before and after a standardized 2-minute static loading protocol (ie, standing) during the second session. All participants provided written informed consent before testing, and the study was approved by the University of North Carolina at Chapel Hill Institutional Review Board.

Ankle Joint Laxity Assessment

An instrumented ankle arthrometer (Blue Bay Research, Inc) was used to quantify the anteroposterior (AP) load-displacement and inversion-eversion rotational laxity characteristics of the involved ankle joint complex.[10] The test-retest reliability of ligament laxity quantification using this type of arthrometer was good to excellent.[10] For AP displacement, the ankle was loaded with 125 N of anterior and posterior force after starting in a neutral position. For rotational laxity, the ankle was loaded to 4 Nm of torque in each direction. Three trials in each direction were performed and the results averaged for further analysis.

Postural Control Assessment

A force plate (AccuSway Balance Platform; Advanced Mechanical Technology, Inc) was used to conduct the single-limb static balance assessments. Before testing, each participant was allowed 3 practice trials. Three 10-second trials were then conducted while participants stood on the involved limb with their hands on their hips and eyes open.[11] Force-plate data were collected at 50 Hz and filtered using a fourth-order, zero-lag, low-pass Butterworth filter with a cutoff frequency of 5 Hz.[11] Time-to-boundary (TTB) minima means (in seconds) in the AP and mediolateral (ML) directions were calculated based on a rectangle foot model using a custom code for MATLAB (version R2019a; The MathWorks, Inc).[11] Smaller values indicated less time to respond to a postural perturbation before exceeding the limits of stability, resulting in worse postural control. This protocol generated reliable and precise data and has been shown to discriminate between those with and those without CAI.[11]

Dynamic postural control was assessed using the SEBT. Participants completed 3 trials in the anterior, posteromedial, and posterolateral SEBT directions as previously reported.[12] Each person performed 4 practice trials in each direction before data collection. Reach distances were normalized to the participant's leg length (ie, anterior-superior iliac spine to ipsilateral medial malleolus). Normalized SEBT reach distances were a reliable measure (intraclass correlation coefficient [ICC]) of dynamic balance (ICC [2,1] = 0.85–0.96)[12] and discriminated between those with and those without CAI.[13] The extracted outcomes for both static and dynamic postural control were averaged across the 3 trials and used for the analysis.

Talar Cartilage Assessment

Talar cartilage thickness was assessed by calculating the cross-sectional area of a 2-dimensional image. Before the preloading assessment, participants sat in the long-sit position for 60 minutes to unload the ankle cartilage and allow fluid rebound.[6] The US images of the talar cartilage were acquired using a tablet-based ultrasound unit (model Lumify L12-4; Koninklijke Philips N.V.) with a 12-MHz linear probe. Participants were positioned with their back against a wall, their knee positioned at 90° of flexion, and

Table 1. Participant Demographics, Injury History Characteristics, and Self-Reported Function

Variable	Group	
	Chronic Ankle Instability (n = 30)	Control (n = 30)
Sex, No. males, females	11, 19	7, 23
	Mean ± SD	
Age, y	20.5 ± 2.2	19.8 ± 4.4
Height, cm	171.5 ± 6.7	170.9 ± 8.6
Weight, kg[a]	75.7 ± 16.2	66.0 ± 13.1
Identification of Functional Ankle Instability score[a]	22.2 ± 5.4	0.3 ± 0.8
Foot and Ankle Ability Measure score, %		
Activities of Daily Living subscale[a]	87.4 ± 11.0	99.9 ± 0.3
Sport subscale[a]	76.7 ± 17.3	100 ± 0
National Aeronautics and Space Administration Physical Activity Status Scale score	6.0 ± 2.1	6.4 ± 2.2
Ankle sprains, No.	4.4 ± 4.5	NA
Giving-way episodes within past 6 mo, No.	9.6 ± 11.5	NA

Abbreviation: NA, not applicable.

[a] Indicates difference between groups ($P < .05$).

their ankles in a foot-flat position (approximately 50° of plantar flexion).[6,14] The probe was placed transversely in line with the medial and lateral malleoli and rotated in the sagittal plane to maximize reflection off the articular cartilage surface. The distance between the wall and the posterior calcaneus was measured using a tape measure that was secured to the treatment table and recorded to ensure consistent positioning across all time points.[6] A transparency grid was placed over the US screen to confirm probe placement consistency over time.[6] Three images of talar cartilage thickness were obtained at the preloading assessment.

In the next step, participants stepped off the table with their uninvolved limb, shifted their weight to the involved limb, and stood on the involved limb for 2 minutes with the knee at approximately 20° of flexion.[6,7] They were instructed to avoid touching the ground with their uninvolved limb but were allowed to use their hands to briefly touch an adjacent wall to maintain balance as needed. At the end of the loading protocol, participants sat back on the table, and 3 additional talar-cartilage US images were obtained using identical methods. Posttest images were acquired within 3 minutes of completing the loading protocol.

Ultrasound Image Analysis

To reduce bias, all US images were blinded such that the assessor was unaware of the timing (preloading or postloading). The images were manually segmented using ImageJ software (version 1.52; National Institutes of Health) to identify the overall, medial, and lateral cross-sectional area of the talar cartilage.[6,14] The cartilage was divided into medial and lateral portions of the talar dome by bisecting the overall volume. Each cross-sectional area was then normalized to the length of the cartilage-bone interface imaged to estimate the average thickness (measured in mm).[6,14] This technique showed great intrarater test-retest reliability for the US talar cartilage measurement (ICC = 0.95–0.99).[14] Deformation was reported as the percentage change in average thickness from preloading to postloading using the following formula: $\%\Delta = [(\text{Mean}_{\text{post}} - \text{Mean}_{\text{pre}})/(\text{Mean}_{\text{pre}})] \times 100$, where $\%\Delta$ = the percentage change, $\text{Mean}_{\text{post}}$ = postloading mean, and Mean_{pre} = preloading mean.[6,14] A higher negative score indicated greater cartilage deformation.

Statistical Analysis

We calculated descriptive statistics for demographics and all dependent variables. Partial correlations were conducted to evaluate the relationships between (1) ankle joint laxity and talar cartilage deformation, (2) TTB outcomes during single-limb stance and talar cartilage deformation, and (3) normalized SEBT reach distances and talar cartilage deformation while accounting for body weight within each group. The correlation coefficient (r) was interpreted as *weak* ($r = 0.00$–0.40), *moderate* ($r = 0.41$–0.69), or *strong* ($r = 0.70$–1.00).[15] A positive correlation indicated that worse static postural control and less SEBT reach distance were associated with greater talar cartilage deformation, whereas a negative correlation indicated that increased laxity was associated with greater talar cartilage deformation. As an exploratory analysis, we performed separate stepwise linear regressions to determine the amount of variance in overall talar cartilage deformation explained by laxity as well as the static and dynamic postural control variables. Body weight was entered into the regression models first, followed by the dependent variables of interest. All analyses were computed using SPSS (version 21.0; IBM Corp), and an α level of .05 was used to determine statistical significance.

RESULTS

Means and SDs for group demographics and injury characteristics are found in Table 1. Descriptive statistics for all dependent variables in each group are presented in Table 2. All correlations, coefficients of determination (R^2 values), and P values in the CAI and control groups are provided in Tables 3 and 4, respectively.

In the CAI group, greater inversion laxity was moderately correlated with greater overall ($r = –0.42$, $P = .03$; Figure A) and medial ($r = –0.48$, $P = .01$) talar deformation after accounting for body weight. A shorter ML TTB minima mean was moderately correlated with greater overall ($r =$

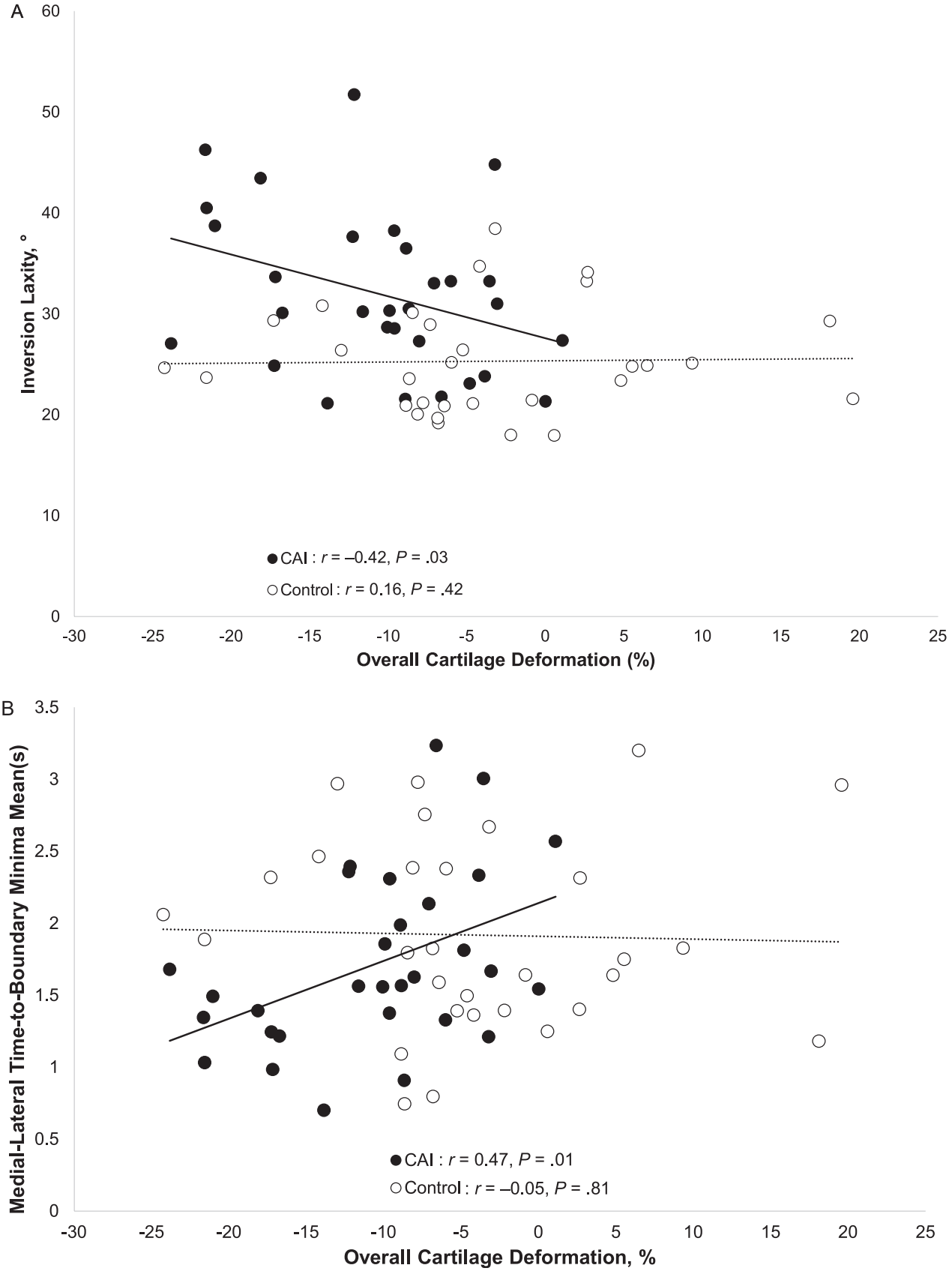

Figure. Scatterplots between (A) inversion laxity and overall cartilage deformation and (B) time-to-boundary mean in the mediolateral direction and overall cartilage deformation. A greater negative % indicates greater cartilage deformation. Abbreviation: CAI, chronic ankle instability.

Table 2. Cartilage Deformation, Ankle Joint Laxity, and Postural Control

Variable	Group, Mean ± SD	
	Chronic Ankle Instability (n = 30)	Control (n = 30)
Cartilage deformation, % change		
Overall[a]	−10.9 ± 7.0	−6.8 ± 6.0
Medial[a]	−13.0 ± 8.2	−5.8 ± 8.9
Lateral	−8.9 ± 7.7	−6.9 ± 8.9
Ankle joint laxity		
Anterior, mm	11.0 ± 2.7	10.9 ± 2.9
Posterior, mm	8.5 ± 2.1	8.1 ± 2.0
Inversion, °[a]	32.1 ± 8.1	25.3 ± 5.3
Eversion, °	22.4 ± 5.0	24.8 ± 5.1
Static postural control, time-to-boundary mean minima, s		
Medial-lateral	1.7 ± 0.6	1.9 ± 0.7
Anterior-posterior	4.8 ± 1.4	5.3 ± 1.5
Dynamic postural control, % of leg length		
Anterior[a]	65.0 ± 6.5	69.0 ± 5.2
Posteromedial	78.5 ± 7.9	80.2 ± 6.4
Posterolateral[a]	74.3 ± 9.5	79.2 ± 6.5

[a] Indicates difference between groups ($P < .05$).

0.47, $P = .01$; Figure B) and lateral ($r = 0.50$, $P = .01$) talar deformation after accounting for body weight. No significant correlations were identified between normalized SEBT reach distances and talar cartilage deformation. The stepwise regression analysis also showed that inversion laxity and ML TTB minima mean were significant predictors of overall talar cartilage deformation while accounting for body weight ($\Delta R^2 = 0.37$, $P < .01$).

In the control group, a shorter posterolateral SEBT reach distance was moderately correlated with greater lateral cartilage deformation after accounting for body weight ($r = 0.42$, $P = .03$). No other significant correlations were found in the control group ($P > .05$). The stepwise regression analysis also revealed no significant predictors for overall talar cartilage deformation while accounting for body weight ($P > .09$).

DISCUSSION

Our primary finding was that a greater magnitude of cartilage deformation after a static loading protocol was associated with greater inversion laxity and poorer static—but not dynamic—postural control in those with CAI. However, these relationships were not seen in the healthy control group. Thus, these results support our a priori hypothesis. Talar cartilage composition (ie, type II collagen, proteoglycans) was disrupted in those with CAI, which influenced greater cartilage deformation.[4,16] Our results provided evidence that mechanical alterations and static postural control impairments, which were common alterations in those with CAI, may begin to explain why cartilage deformation was greater in the CAI group relative to an uninjured control group after a static mechanical load. These variables may represent potential targets for treatment protocols to mitigate the progression toward ankle PTOA in those with CAI.

Mechanical joint instability occurs due to damage of the lateral ligaments during ankle sprains, which leads to residual ankle joint laxity.[17] This increased joint laxity is thought to alter ankle joint kinematics and eventually result in cartilage degeneration.[18,19] Lee et al[20] observed that ankles with anterior talofibular ligament damage displayed greater talar cartilage T2 MRI relaxation times (ie, altered composition) compared with an uninjured group. Similarly, ankles with unilateral CAI demonstrated greater and anteromedially translated peak contact strain versus contralateral healthy ankles during standing.[18] Previous researchers[21] also noted that individuals with CAI had an anterior talar positional fault, relative to a control group, which may have placed the talus in a position that alters joint contact strain. Given that cartilage composition influenced cartilage behavior and disrupted lateral ligament integrity altered both tibiotalar kinematics and talar composition,[7,18,19] it is not surprising that more talar cartilage deformation occurred when inversion laxity was greater.

Earlier authors[22] showed that poorer static postural control was associated with higher T1ρ MRI relaxation times (ie, altered cartilage composition) in those with CAI. Our results were consistent, indicating that poorer ML static postural control was correlated with greater talar cartilage deformation after static loading. Postural control, a proxy for neuromuscular control, was commonly impaired in those with CAI.[23] Although the exact mechanism driving this association remains unknown, impaired neuromuscular control may lead to altered joint

Table 3. Correlations Between Ankle Joint Laxity and Balance and Cartilage Deformation After Accounting for Body Weight in Those With Chronic Ankle Instability

Variable	Deformation								
	Overall			Medial			Lateral		
	r	R^2	*P*	*r*	R^2	*P*	*r*	R^2	*P*
Ankle joint laxity									
Anterior	0.17	0.03	.39	0.08	0.01	.68	0.25	0.06	.21
Posterior	−0.14	0.02	.48	0.01	<0.01	.99	−0.22	0.05	.27
Inversion	−0.42	0.18	.03	−0.48	0.23	.01	−0.19	0.04	.34
Eversion	−0.22	0.05	.26	0.08	0.01	.71	−0.26	0.07	.19
Static balance, time-to-boundary minima mean									
Medial-lateral	0.47	0.22	.01	0.33	0.11	.09	0.50	0.25	.01
Anterior-posterior	0.18	0.03	.37	0.21	0.04	.29	0.13	0.02	.52
Star Excursion Balance Test score									
Anterior	−0.06	<0.01	.75	−0.02	<0.01	.94	−0.04	<0.01	.86
Posteromedial	0.13	0.02	.50	0.18	0.03	.37	0.12	0.01	.56
Posterolateral	0.06	<0.01	.77	0.12	0.01	.55	0.06	<0.01	.77

Table 4. Correlations Between Ankle Joint Laxity and Balance and Cartilage Deformation After Accounting for Body Weight in Healthy Control Individuals

	Deformation								
	Overall			Medial			Lateral		
Variable	*r*	R^2	*P*	*r*	R^2	*P*	*r*	R^2	*P*
Ankle joint laxity									
Anterior	0.13	0.02	.52	0.26	0.07	.17	−0.08	0.01	.66
Posterior	0.07	<0.01	.73	0.11	0.01	.59	−0.01	<0.01	.99
Inversion	0.16	0.03	.42	0.07	<0.01	.70	0.18	0.03	.34
Eversion	0.11	0.01	.59	0.33	0.11	.08	−0.21	0.04	.27
Static balance, time-to-boundary minima mean									
Medial-lateral	−0.05	<0.01	.81	−0.19	0.04	.33	0.15	0.02	.45
Anterior-posterior	−0.16	0.03	.41	−0.33	0.11	.09	0.10	0.01	.60
Star Excursion Balance Test score									
Anterior	0.12	0.01	.55	0.17	0.03	.38	0.01	<0.01	.95
Posteromedial	0.13	0.02	.49	0.03	<0.01	.87	0.23	0.05	.24
Posterolateral	0.36	0.13	.06	0.20	0.04	.30	0.42	0.18	.03

loading. Postural instability could represent greater shearing forces being applied to the talar cartilage from excessive ML rocking of the talus in the mortise. It is also possible that greater ML shifts of the center of pressure, in conjunction with altered tibiotalar kinematics, could have shifted the location of peak contact strain.[18] If static postural control is to be used as a potential therapeutic target to slow ankle PTOA progression, further investigation is needed to better understand how postural instability influences talar cartilage behavior.

We identified no associations between SEBT reach distances and cartilage deformation magnitude in this CAI sample, which was consistent with previous research.[24] More specifically, no relationships between SEBT reach distances and T2-mapping (ie, cartilage composition scores) was seen in a CAI sample.[24] However, in that same sample, we observed an association between longer ML time to stabilization, calculated during a single-legged jump landing, and worse cartilage composition.[24] Although task and outcome differences may have explained the contrary findings, it was also possible that more precise laboratory-oriented measures of postural control (ie, instrumented measures) better captured factors that influenced cartilage loading behavior.

Clinically, our results suggested that mechanical instability and poor static balance could be the targets of intervention strategies in an effort to improve cartilage loading behavior and ultimately slow ankle PTOA development. Currently, external ankle supports (eg, bracing, taping) are conservative options for enhancing mechanical stability of the ankle, but these tools do not alter ankle kinematics.[25] Thus, it is unlikely that they would improve the altered in vivo loading patterns observed in those with CAI.[18,19] Lateral ligament reconstruction (eg, the Broström-Gould procedure) decreased both anterior translation and internal rotation of the talus while weight bearing.[26] At this time, surgical reconstruction appears to be the best option for restoring ligament integrity at the ankle, but prospective exploration is needed to determine the effect of surgical reconstruction on direct measures of cartilage health. However, anterior cruciate ligament reconstruction is not effective in preventing PTOA at the knee joint[27]; hence, this invasive approach may not address the PTOA risk at the ankle. Also, it is important to refine the clinical assessment technique for identifying subtalar joint instability to evaluate treatment effects on residual joint laxity more accurately in the clinical setting.[28] Balance training[29] and manual therapy[30] have consistently improved postural control in those with CAI. Therefore, these practices should be used clinically. Future research is warranted to determine whether improved postural control mitigates cartilage degeneration in those with CAI and whether other interventions might also improve direct measures of cartilage health (ie, composition or loading behavior or both). Most importantly, quantification of the cumulative effect of surgical reconstruction and robust neuromuscular rehabilitation (eg, balance training and manual therapy) on a variety of outcomes, including cartilage health, is needed, as no such data currently exist.

CONCLUSIONS

Greater inversion laxity and poor static postural control were moderately associated with greater talar cartilage deformation after a 2-minute static loading protocol in those with CAI. Given these relationships, improving mechanical stability and postural control should be targeted during intervention strategies in an effort to restore or improve cartilage loading behavior. Further evaluation is required to determine how improving common CAI mechanical and sensorimotor impairments affects cartilage loading behavior in individuals with CAI.

REFERENCES

1. Hootman JM, Dick R, Agel J. Epidemiology of collegiate injuries for 15 sports: summary and recommendations for injury prevention initiatives. *J Athl Train*. 2007;42(2):311–319.
2. Anandacoomarasamy A, Barnsley L. Long term outcomes of inversion ankle injuries. *Br J Sports Med*. 2005;39(3):e14; discussion e14. doi:10.1136/bjsm.2004.011676
3. Harrington KD. Degenerative arthritis of the ankle secondary to long-standing lateral ligament instability. *J Bone Joint Surg Am*. 1979;61(3):354–361.
4. Wikstrom EA, Song K, Tennant JN, Dederer KM, Paranjape C, Pietrosimone B. T1ρ MRI of the talar articular cartilage is increased in those with chronic ankle instability. *Osteoarthritis Cartilage*. 2019;27(4):646–649. doi:10.1016/j.joca.2018.12.019
5. Liess C, Lüsse S, Karger N, Heller M, Glüer C-C. Detection of changes in cartilage water content using MRI T2-mapping in vivo.

Osteoarthritis Cartilage. 2002;10(12):907–913. doi:10.1053/joca.2002.0847
6. Song K, Pietrosimone B, Blackburn JT, Padua DA, Tennant JN, Wikstrom EA. Acute talar cartilage deformation in those with and without chronic ankle instability. *Med Sci Sports Exerc*. 2021;53(6):1228–1234. doi:10.1249/MSS.0000000000002572
7. Van Ginckel A, Almqvist F, Verstraete K, Roosen P, Witvrouw E. Human ankle cartilage deformation after different in vivo impact conditions. *Knee Surg Sports Traumatol Arthrosc*. 2011;19(1):137–143. doi:10.1007/s00167-010-1159-4
8. Song K, Wikstrom EA. Plausible mechanisms of and techniques to assess ankle joint degeneration following lateral ankle sprains: a narrative review. *Phys Sportsmed*. 2019;47(3):275–283. doi:10.1080/00913847.2019.1581511
9. Gribble PA, Delahunt E, Bleakley CM, et al. Selection criteria for patients with chronic ankle instability in controlled research: a position statement of the International Ankle Consortium. *J Athl Train*. 2014;49(1):121–127. doi:10.4085/1062-6050-49.1.14
10. Hubbard TJ, Kaminski TW, Vander Griend RA, Kovaleski JE. Quantitative assessment of mechanical laxity in the functionally unstable ankle. *Med Sci Sports Exerc*. 2004;36(5):760–766. doi:10.1249/01.mss.0000126604.85429.29
11. Hertel J, Olmsted-Kramer LC. Deficits in time-to-boundary measures of postural control with chronic ankle instability. *Gait Posture*. 2007;25(1):33–39. doi:10.1016/j.gaitpost.2005.12.009
12. Hertel J, Miller SJ, Denegar CR. Intratester and intertester reliability during the Star Excursion Balance Tests. *J Sport Rehabil*. 2000;9(2):104–116. doi:10.1123/jsr.9.2.104
13. Song K, Jang J, Nolte T, Wikstrom EA. Dynamic reach deficits in those with chronic ankle instability: a systematic review and meta-analysis. *Phys Ther Sport*. 2022;53:40–50. doi:10.1016/j.ptsp.2021.11.004
14. Song K, Pietrosimone BG, Nissman DB, Wikstrom EA. Ultrasonographic measures of talar cartilage thickness associate with magnetic resonance-based measures of talar cartilage volume. *Ultrasound Med Biol*. 2020;46(3):575–581. doi:10.1016/j.ultrasmedbio.2019.12.005
15. Dancey CP, Reidy J. *Statistics Without Maths for Psychology*. Pearson/Prentice Hall; 2007.
16. Golditz T, Steib S, Pfeifer K, et al. Functional ankle instability as a risk factor for osteoarthritis: using T2-mapping to analyze early cartilage degeneration in the ankle joint of young athletes. *Osteoarthritis Cartilage*. 2014;22(10):1377–1385. doi:10.1016/j.joca.2014.04.029
17. Hertel J. Functional anatomy, pathomechanics, and pathophysiology of lateral ankle instability. *J Athl Train*. 2002;37(4):364–375.
18. Bischof JE, Spritzer CE, Caputo AM, et al. In vivo cartilage contact strains in patients with lateral ankle instability. *J Biomech*. 2010;43(13):2561–2566. doi:10.1016/j.jbiomech.2010.05.013
19. Caputo AM, Lee JY, Spritzer CE, et al. In vivo kinematics of the tibiotalar joint after lateral ankle instability. *Am J Sports Med*. 2009;37(11):2241–2248. doi:10.1177/0363546509337578
20. Lee S, Yoon YC, Kim JH. T2 mapping of the articular cartilage in the ankle: correlation to the status of anterior talofibular ligament. *Clin Radiol*. 2013;68(7):e355–e361. doi:10.1016/j.crad.2013.01.023
21. Wikstrm EA, Hubbard TJ. Talar positional fault in persons with chronic ankle instability. *Arch Phys Med Rehabil*. 2010;91(8):1267–1271. doi:10.1016/j.apmr.2010.04.022
22. Wikstrom EA, Song K, Tennant JN, et al. Gait biomechanics and balance associate with talar and subtalar T1ρ relaxation times in those with chronic ankle instability. *Med Sci Sports Exerc*. 2022;54(6):1013–1019. doi:10.1249/MSS.0000000000002867
23. Hertel J. Sensorimotor deficits with ankle sprains and chronic ankle instability. *Clin Sports Med*. 2008;27(3):353–370; vii. doi:10.1016/j.csm.2008.03.006
24. Golditz T, Welsch GH, Pachowsky M, Hennig FF, Pfeifer K, Steib S. A multimodal approach to ankle instability: interrelations between subjective and objective assessments of ankle status in athletes. *J Orthop Res*. 2016;34(3):525–532. doi:10.1002/jor.23039
25. Kobayashi T, Saka M, Suzuki E, et al. The effects of a semi-rigid brace or taping on talocrural and subtalar kinematics in chronic ankle instability. *Foot Ankle Spec*. 2014;7(6):471–477. doi:10.1177/1938640014543357
26. Wainright WB, Spritzer CE, Lee JY, et al. The effect of modified Broström-Gould repair for lateral ankle instability on in vivo tibiotalar kinematics. *Am J Sports Med*. 2012;40(9):2099–2104. doi:10.1177/0363546512454840
27. Luc B, Gribble PA, Pietrosimone BG. Osteoarthritis prevalence following anterior cruciate ligament reconstruction: a systematic review and numbers-needed-to-treat analysis. *J Athl Train*. 2014;49(6):806–819. doi:10.4085/1062-6050-49.3.35
28. Medina McKeon JM, Hoch MC. The ankle-joint complex: a kinesiologic approach to lateral ankle sprains. *J Athl Train*. 2019;54(6):589–602. doi:10.4085/1062-6050-472-17
29. McKeon PO, Ingersoll CD, Kerrigan DC, Saliba E, Bennett BC, Hertel J. Balance training improves function and postural control in those with chronic ankle instability. *Med Sci Sports Exerc*. 2008;40(10):1810–1819. doi:10.1249/MSS.0b013e31817e0f92
30. Hoch MC, McKeon PO. Joint mobilization improves spatiotemporal postural control and range of motion in those with chronic ankle instability. *J Orthop Res*. 2011;29(3):326–332. doi:10.1002/jor.21256

Address correspondence to Kyeongtak Song, PhD, ATC, 325 Sports Science Complex, 50 Yonsei-ro, Seodaemun-gu, Seoul, 03722, Republic of Korea. Address email to ktsong@yonsei.ac.kr.