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Older Adults Overcome Reduced Triceps Surae Structural Stiffness to Preserve Ankle Joint Quasi-Stiffness During Walking

Rebecca L. Krupenevich,[1] William H. Clark,[1] Gregory S. Sawicki,[2] and Jason R. Franz[1]
[1]University of North Carolina at Chapel Hill and North Carolina State University; [2]Georgia Institute of Technology

Ankle joint quasi-stiffness is an aggregate measure of the interaction between triceps surae muscle stiffness and Achilles tendon stiffness. This interaction may be altered due to age-related changes in the structural properties and functional behavior of the Achilles tendon and triceps surae muscles. The authors hypothesized that, due to a more compliant of Achilles' tendon, older adults would exhibit lower ankle joint quasi-stiffness than young adults during walking and during isolated contractions at matched triceps surae muscle activations. The authors also hypothesized that, independent of age, triceps surae muscle stiffness and ankle joint quasi-stiffness would increase with triceps surae muscle activation. The authors used conventional gait analysis in one experiment and, in another, electromyographic biofeedback and *in vivo* ultrasound imaging applied during isolated contractions. The authors found no difference in ankle joint quasi-stiffness between young and older adults during walking. Conversely, this study found that (1) young and older adults modulated ankle joint quasi-stiffness via activation-dependent changes in triceps surae muscle length–tension behavior and (2) at matched activation, older adults exhibited lower ankle joint quasi-stiffness than young adults. Despite age-related reductions during isolated contractions, ankle joint quasi-stiffness was maintained in older adults during walking, which may be governed via activation-mediated increases in muscle stiffness.

Keywords: Achilles tendon, activation, aging, plantar flexor, biofeedback

Compared with young adults, older adults often display a characteristic reduction in propulsive power at the ankle during the push-off phase of walking.[1,2] Although colloquially described in the biomechanics literature as a product of ankle joint velocity and net joint moment, ankle power output in walking is generated through the complex interaction between neural drive, triceps surae muscle contractile behavior, and series elastic response from the Achilles tendon.[3,4] Ankle joint quasi-stiffness, defined as the slope of the ankle moment–angle relation,[5] is a function of all active and passive structures spanning the ankle joint and represents an aggregate measure of this interaction. Our research group recently provided empirical evidence that ankle joint quasi-stiffness (1) can be actively regulated through the activation-dependent modulation of soleus length–tension behavior and (2) represents contributions from both the Achilles tendon and triceps surae muscles.[6] These activation-dependent effects on muscle–tendon and ankle joint mechanics may be altered in older adults due to age-related changes in the structural properties, and thus, the functional behavior of the Achilles tendon and triceps surae muscles. Indeed, although specific cause–effect relations have yet to be established, we posit that the inability to maintain triceps surae muscle–tendon unit stiffness during walking may result in slower walking speeds and poor walking economy. While translationally important, the effects of age on ankle joint quasi-stiffness are relatively unknown. Addressing this gap will further our basic understanding of triceps surae muscle–tendon interaction as a potential mechanism for age-associated changes in walking performance and may inform the future development of assistive devices designed to augment ankle joint behavior.[7]

Ankle joint quasi-stiffness during walking, largely a function of muscle–tendon unit stiffness, arises from a tuned neuromechanical interaction between Achilles tendon stiffness and triceps surae muscle stiffness. Thus, changes in ankle joint quasi-stiffness may occur through changes in Achilles tendon stiffness that are not proportionately met by changes in triceps surae muscle stiffness (and vice versa). Indeed, recent modeling and experimental data suggest that ankle joint quasi-stiffness is systematically regulated by triceps surae muscle stiffness via changes in triceps surae muscle activation.[6,8] Furthermore, this activation-dependent modulation of triceps surae muscle stiffness occurs in response to simulated changes in Achilles tendon stiffness when the task demand requires requisite ankle joint quasi-stiffness to preserve joint function, for example, during walking.[8]

Aging is associated with changes in muscle and tendon structural properties,[9,10] and thus, may contribute to altered ankle joint quasi-stiffness in older adults. Given its aggregate nature, age-related changes in ankle joint quasi-stiffness may be attributed to changes in the Achilles tendon moment arm, neural drive, triceps surae muscle length–tension behavior, and/or Achilles tendon series elasticity. The latter of these is a likely factor; despite conflicting reports in the comparative biomechanics literature, the majority of human studies have found that Achilles tendon stiffness decreases by up to ~40% in old age[9–11] in older versus young adults. Theoretically, decreased Achilles tendon stiffness would contribute to decreased ankle–joint quasi-stiffness. Age-related reductions in maximum muscle force-generating

Krupenevich, Clark, and Franz are with the Joint Department of Biomedical Engineering, University of North Carolina at Chapel Hill, Chapel Hill, NC, USA; and North Carolina State University, Raleigh, NC, USA. Sawicki is with the George W. Woodruff School of Mechanical Engineering, Georgia Institute of Technology, Atlanta, GA, USA. Franz (jrfranz@email.unc.edu) is corresponding author.

potential are also highly prevalent,[12,13] potentially scaling down active contributions to muscle stiffness, and thus, ankle joint quasi-stiffness. However, no study to our knowledge has quantified the extent to which ankle joint quasi-stiffness differs between young and older adults during tasks spanning isolated contractions to more functional activities, such as walking.

Therefore, this work leveraged 2 separate but complementary experiments designed to determine whether older adults maintain ankle joint quasi-stiffness during walking compared to young adults and, if so, how. In experiment 1, we quantified age-related differences in ankle joint quasi-stiffness during the stance phase of walking. Based on known structural changes within the triceps surae due to age, we hypothesized that older adults would walk with lower ankle joint quasi-stiffness than young adults. However, ankle joint quasi-stiffness in walking is governed not only by structural properties, but also by actively mediated changes in muscle length–tension behavior that may also be fundamentally altered due to age. Accordingly, in experiment 2, we leveraged isolated contractions and a novel electromyographic biofeedback paradigm to prescribe matched activation levels in young and older subjects and thereby remove this potential for the activation-dependent modulation of ankle joint quasi-stiffness. Here, we first hypothesized that, in both groups, the slope of the relation between muscle force and fascicle length (ie, muscle stiffness) and that of the relation between ankle moment and ankle angle (ie, ankle joint quasi-stiffness) would increase with triceps surae muscle activation. Second, we hypothesized that, at matched triceps surae muscle activations, older adults would exhibit decreased ankle joint quasi-stiffness compared with young adults. Conversely, if ankle joint quasi-stiffness during walking is unaffected by age, our cumulative results would suggest that older adults maintain requisite ankle joint mechanics via a local adaptive mechanism of increased triceps surae muscle activation.

Methods

We provide methodological details for each of 2 experiments, referred to below as experiment 1, comprised of measurements during walking, and experiment 2, compromised of measurements during isolated contractions.

Participants

In this study, 10 young (5 males and 5 females; 23 [2] y, 66.2 [10.4] kg, 1.73 [0.10] m) and 12 older (5 males and 7 females; 75 [5] y, 68.6 [11.1] kg, 1.51 [0.24] m) adults participated in experiment 1, whereas 9 young (4 males and 5 females; 23 [3] y, 70.7 [10.4] kg, 1.76 [0.12] m) and 8 older (4 males and 4 females; 72 [4] y, 69.1 [18.6] kg, 1.68 [0.09] m) adults participated in experiment 2. Three young subjects and 3 older subjects participated in both experiments. Prior to participation in either experiment, the subjects were screened and excluded if they reported injury or fracture to the lower extremity within the previous 6 months, neurological disorders affecting the lower extremity, or currently taking medications that cause dizziness. All subjects provided written informed consent according to the University of North Carolina Biomedical Sciences Institutional Review Board.

Experiment 1 (Walking)

Prior to the data collection, we established each subject's preferred overground walking speed using 2 infrared timing gates placed 2 m apart in the center of a 10-m walkway. The participants were instructed to walk at a normal and comfortable pace. The preferred walking speed was averaged across 3 trials. The subjects then walked on an instrumented treadmill (Bertec Corp, Columbus, OH) for 5 minutes to acclimate to treadmill walking. Following the acclimation period, the subjects walked on the treadmill at their preferred walking speed for 1 minute, while ground reaction forces were measured at 1000 Hz and 3D positions of 31 retroreflective markers were recorded using a 14-camera motion capture system (Motion Analysis Corp, Santa Rosa, CA). Ground reaction forces and marker position data were filtered using a fourth-order low-pass Butterworth filter with cutoff frequencies of 100 Hz and 6 Hz, respectively. A 7-segment, 18 degree of freedom model of the pelvis and lower limbs was created for each subject and scaled using marker positions from a static standing trial.[14] The hip joint was defined using functional hip joint centers.[15] A global optimization inverse kinematics routine calculated lower extremity joint kinematics, moments, and powers.[16] All strides during the 1-minute trial (55 [5] strides per subject) were averaged to produce a representative time-normalized stride for each outcome measure per subject. The outcome variables for each subject were then calculated using their representative time-normalized stride.

Ankle joint quasi-stiffness during walking was calculated for 3 subphases during the stance phase (dorsiflexion, dualflexion, and plantar flexion[5,17]) to characterize the distinct energy storage and return phases of the ankle during walking. The dorsiflexion phase was defined as the point of local minimum ankle angle after heel contact to 30% of the stride, the dualflexion phase was defined as 30% of the stride to the point of maximum ankle moment, and the plantar flexion phase was defined as the point of maximum ankle moment to the point of maximum ankle angle.[5] Ankle joint quasi-stiffness was then calculated for each phase as the slope of the best-fit line.

Experiment 2 (Isolated Contractions)

The subjects first walked for 6 minutes at 1.25 m/s on a treadmill to precondition their triceps surae and allow their movement patterns to stabilize prior to performing dynamometer tasks.[18] The subjects then performed 3 maximum voluntary isometric plantar flexor contractions (MVIC) while seated in a dynamometer (Biodex Medical Systems, Shirley, NY) with their trunk flexed to 85°, knee flexed to 20°, and ankle in a neutral position (0°). A single electrode (Trigno; Delsys Inc, Natick, MA) on the soleus was used to extract a reference maximum activation from the MVIC trials. The electrode site was prepped by shaving and abrading the skin.

The subjects then performed a series of isolated plantar flexion tasks while using biofeedback to match a prescribed soleus activation.[6] Specifically, the subjects performed isokinetic eccentric contractions of the triceps surae muscles at 30°/s through a 25° range of motion (10° of plantar flexion to 15° of dorsiflexion). Ankle angular velocity of 30°/s is comparable with ankle angular velocity during the midstance phase of walking when the triceps surae muscles are being eccentrically loaded in dorsiflexion.[19] During isokinetic contractions, instantaneous rectified soleus activation was presented to subjects as a "dot" on a screen. The screen also displayed a horizontal line representing their 75% MVIC ankle moment. We asked the subjects to keep the dot as close to the line as possible during the biofeedback tasks (Figure 1). A second condition ("Passive," 0% MVIC) was also performed; during this condition, the dynamometer moved the ankle joint at 30°/s through the prescribed range of motion without active subject resistance.

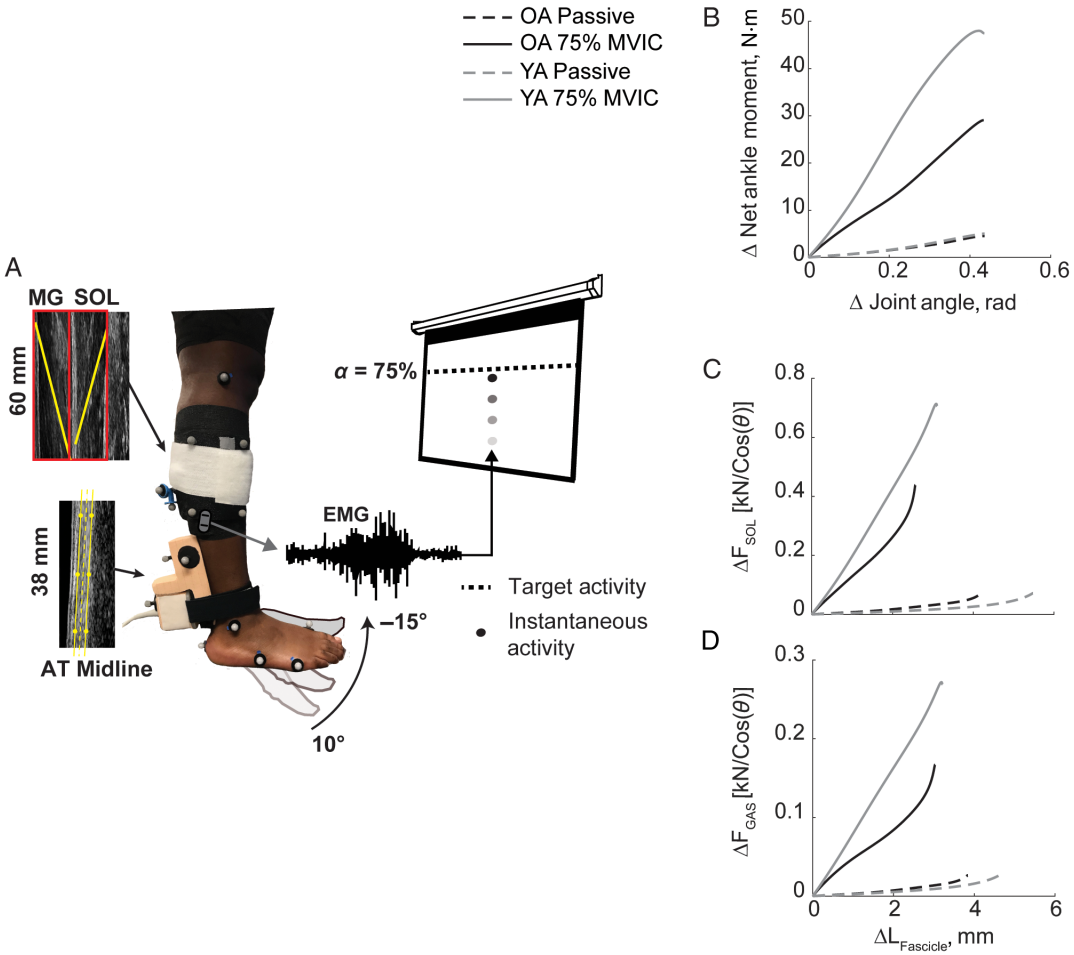

Figure 1 — (A) Our experimental design used electromyographic biofeedback to prescribe a matched soleus activation (75% MVIC) across both age groups during eccentric action of the triceps surae muscles. Instantaneous rectified soleus activation was presented to subjects as a "dot" on a screen. The screen also displayed a horizontal line representing their 75% MVIC ankle moment. We asked subjects to keep the dot as close to the line as possible during the biofeedback tasks. Ultrasound imaging captured triceps surae muscle fascicle length and pennation angle changes. (B) Group mean ankle joint quasi-stiffness (top) quantified via the relation between the change in ankle joint angle and the change in net ankle moment. (C) Group mean soleus muscle stiffness (middle) quantified via the relation between the change in ΔF_{SOL}, equal to longitudinal muscle force divided by the cosine of pennation angle, and the change in soleus muscle $\Delta L_{Fascicle}$. Group mean gastrocnemius stiffness (bottom) quantified via the relation between the change in ΔF_{GAS}, equal to longitudinal muscle force divided by the cosine of pennation angle, and the change in gastrocnemius muscle $\Delta L_{Fascicle}$. EMG indicates electromyography; MVIC, maximum voluntary isometric contraction; ΔF_{SOL}, soleus muscle fascicle force; $\Delta L_{Fascicle}$, fascicle length; ΔF_{GAS}, gastrocnemius muscle fascicle force.

Three trials were collected for each condition (passive rotation and 75% MVIC). The participants rested for at least 1 minute between trials and conditions and were allowed to practice the biofeedback tasks prior to the data collection.

Subjects

Soleus and gastrocnemius fascicle lengths and subject-specific Achilles tendon moment arms during isokinetic conditions were obtained using ultrasound imaging. Detailed techniques for image collection and processing were published previously[6] and are briefly summarized here. A 10 MHz, 60 mm linear array ultrasound transducer (LV7.5/60/128Z-2, Telemed Echo Blaster 128; Telemed, Vilnius, Lithuania) operating at 61 frames per second recorded cine B-mode images of a 65-mm deep longitudinal cross-section of the gastrocnemius and soleus, through the mid belly of the medial gastrocnemius. The time series of gastrocnemius soleus fascicle length and pennation angle were obtained using UltraTrack.[20] The pennation angle was defined as the angle between the fascicle and deep aponeurosis. Longitudinal soleus muscle length was defined as the product of the fascicle length and the cosine of the pennation angle. Subject-specific moment arms were determined using a second 38 mm transducer (L14-5W/38; Ultrasonix Corp, Richmond, BC), operating at 70 frames per second, placed on the Achilles free tendon distal to the soleus muscle–tendon junction.[21] The Achilles tendon moment arm was defined as the perpendicular distance from the Achilles tendon midline to the transmalleolar axis while the ankle was in a neutral position. The transmalleolar axis was defined as the shortest distance between the tendon line of action and the transmalleolar axis.

The electromyography data were demeaned, full-wave rectified, and band-pass filtered (20–450 Hz). The net ankle moment was recorded at 1000 Hz in sync with the ultrasound and motion capture data. Gravity was corrected for in the dynamometer data using a built-in zeroing procedure that subtracts the weight of the foot from the measured torque data. An 8-camera motion

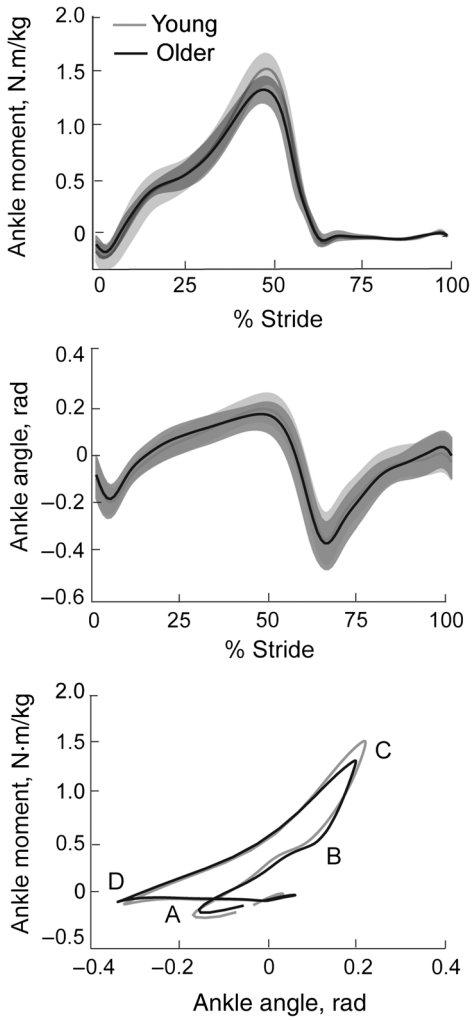

Figure 2 — Group mean sagittal plane ankle joint moments (top), angles (middle), and the relation between these outcomes (bottom) for young (gray) and older (black) adults walking at their self-selected speeds plotted over an average gait cycle from heel strike to heel strike. Gray shaded regions indicate ±1SD from the mean (see Supplementary Figure 1 [available online] for ankle joint quasi-stiffness variance). Positive values indicate an internal plantar flexor moment or dorsiflexion. Quasi-stiffness was calculated as the slope of the relation between ankle joint moment and ankle joint angle for 3 distinct portions of the stance phase: dorsiflexion (A–B), dual-flexion, (B–C), and plantar flexion (C–D).

capture system (Motion Analysis Corp) recorded 3D positions of 14 retroreflective markers placed on the right leg and each ultrasound transducer. Marker position data were used in an inverse kinematics routine to estimate the ankle angle.[16]

Individual gastrocnemius and soleus muscle forces were calculated by scaling the triceps surae muscle force, defined as the net ankle moment divided by the subject-specific moment arm, by the relative physiological cross-sectional area of the gastrocnemius (24%) and soleus (63%).[22] The same physiological cross-sectional area values were used for young and older adults because previous reports have indicated that the relative cross-sectional area of the triceps surae muscles is preserved with age.[22] Muscle stiffness was then calculated as the change in muscle force divided by the change in muscle length (calculated as the slope of the best fit line across the entire matched range of motion).

Statistical Analysis

The primary outcome measure for experiment 1 was ankle joint quasi-stiffness during 3 subphases of walking: dorsiflexion, dualflexion, and plantar flexion. Two-tailed independent samples *t* tests were performed to test for the effect of age on ankle joint quasi-stiffness during walking. The primary outcome measures for experiment 2 were soleus and gastrocnemius muscle stiffness and ankle joint quasi-stiffness. A 2-way mixed factorial analysis of variance tested for the effect of age (old vs young) and activation (75% MVIC activation vs passive rotation) on muscle stiffness and ankle joint quasi-stiffness. Post hoc *t* tests compared the differences due to age at each level of activation (ie, passive and 75% MVIC activation). We defined significance using an alpha level of .05 for all comparisons. The effect sizes in both experiments were calculated using Cohen *d*. Values of $d > 0.20$, 0.50, and 0.80 indicate small, moderate, and large differences, respectively.[23]

Results

For experiment 1, the average preferred walking speed was unaffected by age and was 1.33 (0.17) m/s for young adults and 1.28 (0.16) m/s for older adults. Compared with the young adults, the older adults walked with 11% smaller peak ankle moment (1.56 [0.14] N·m/kg vs 1.37 [0.12] N·m/kg, $P = .002$, $d = 1.46$), but no difference in peak dorsiflexion (0.22 [0.06] rad vs 0.21 [0.0.6] rad, $P = .52$, $d = 0.17$) or peak plantar flexion (–0.33 [0.11] rad vs –0.35 [0.10] rad, $P = .65$, $d = 0.19$).

The assumption of normality for primary outcome variables in experiment 1 was tested and met using the Shapiro–Wilk test. We found no difference in ankle joint quasi-stiffness between the young and older adults during the dorsiflexion (2.91 [0.74] $N{\cdot}m{\cdot}kg^{-1}{\cdot}rad^{-1}$ vs 2.59 [0.86] $N{\cdot}m{\cdot}kg^{-1}{\cdot}rad^{-1}$, $P = .38$, $d = 0.40$), dualflexion (9.02 [2.42]$N{\cdot}m{\cdot}kg^{-1}{\cdot}rad^{-1}$ vs 8.93 [5.93] $N{\cdot}m{\cdot}kg^{-1}{\cdot}rad^{-1}$, $P = .96$, $d = 0.02$), or plantar flexion (3.08 [0.63] $N{\cdot}m{\cdot}kg^{-1}{\cdot}rad^{-1}$ vs 2.68 [0.53] $N{\cdot}m{\cdot}kg^{-1}{\cdot}rad^{-1}$, $P = .12$, $d = 0.69$) phases of stance (Figure 2, Table 1).

For experiment 2, the results of the Shapiro–Wilk test for normality indicated that the assumption of normality was met for all primary outcome variables except for soleus and gastrocnemius stiffness during passive rotation, likely due to the presence of some outliers (Figure 3). However, an examination of the skewness and kurtosis statistics suggest that normality of data is a reasonable assumption for these variables.

Compared with the young adults, the older adults exhibited 54% smaller MVIC torque (86 [32] N·m vs 49 [18] N·m, $P = .01$, $d = 1.43$). The subjects successfully and similarly matched the 75% MVIC target within a range of ±5% (young: 74% [4%] MVIC, older: 81% [6%] MVIC, $P = .31$, $d = 1.37$). We found an age × condition interaction for ankle joint quasi-stiffness ($F_{1,15} = 6.63$, $P = .02$, $\eta_p^2 = .31$; Figure 1A and 1B), with significant main effects for condition ($F_{1,15} = 65.50$, $P < .001$, $\eta_p^2 = .81$) and age ($F_{1,15} = 6.65$, $P = .02$, $\eta_p^2 = .31$). Post hoc comparisons revealed no difference in ankle joint quasi-stiffness between the young and older adults during passive rotation (young: 12.2 [5.9] N·m/rad, older: 10.7 [5.5] N·m/rad, $P = .59$, $d = 0.26$; Figure 3A, Table 1), but greater ankle joint quasi-stiffness in the young versus older adults at 75% MVIC activation (young: 120.0 [51.3] N·m/rad, older: 66.5 [29.1] N·m/rad, $P = .02$, $d = 1.28$; Figure 3B, Table 1). In addition, the Achilles tendon moment arm was not different between groups (young: 43.3 [6.7] mm, older: 41.9 [5.7] mm, $P = .69$, $d = 0.23$).

Table 1 Summary Statistics and Effect Sizes for Pairwise Comparisons of Primary Outcomes

Experiment 1 (walking)	Young (n = 10)	Older (n = 12)	*P* value	Cohen *d* (95% CI)
Ankle joint quasi-stiffness, N·m·kg^{-1}·rad^{-1}				
Dorsiflexion phase	2.91 (0.74)	2.59 (0.86)	.376	0.40 (–0.45 to 1.24)
Dualflexion phase	9.02 (2.42)	8.93 (5.93)	.960	0.02 (–0.82 to 0.86)
Plantar flexion phase	3.08 (0.63)	2.68 (0.53)	.124	0.69 (–0.17 to 1.55)
Experiment 2 (isolated contractions)	**Young (n = 9)**	**Older (n = 8)**	***P* value**	**Cohen *d* (95% CI)**
Passive rotation				
Ankle joint quasi-stiffness, N·m/rad	12.2 (5.9)	10.7 (5.5)	.594	0.26 (–0.69 to 1.22)
Gastrocnemius stiffness, N/mm	6.4 (3.4)	7.3 (5.2)	.675	0.21 (–0.75 to 1.16)
Soleus stiffness, N/mm	15.3 (9.5)	18.1 (10.6)	.572	0.28 (–0.68 to 1.23)
75% MVIC				
Ankle joint quasi-stiffness, N·m/rad	120.0 (51.3)	66.5 (29.1)	**.020**	1.28 (0.24 to 2.33)
Gastrocnemius stiffness, N/mm	109.8 (66.7)	53.1 (29.9)	**.043**	1.04 (0.08 to 2.12)
Soleus stiffness, N/mm	277.5 (131.6)	154.4 (82.3)	**.038**	1.12 (0.10 to 2.14)

Abbreviations: CI, confidence interval; MVIC, maximum voluntary isometric contraction. Note: Summary statistics shown are mean (SD). Bold values indicate significance at $p < .05$.

We found an age × condition interaction for soleus ($F_{1,15} = 5.18$, $P = .04$, $\eta_p^2 = .26$; Figure 1C) and gastrocnemius stiffness ($F_{1,15} = 4.82$, $P = .04$, $\eta_p^2 = .24$; Figure 1C), with significant main effects for condition (SOL: $F_{1,15} = 51.95$, $P < .001$, $\eta_p^2 = .78$, GAS: $F_{1,15} = 3.33$, $P < .001$, $\eta_p^2 = .68$) and age (SOL: $F_{1,15} = 5.08$, $P = .04$, $\eta_p^2 = .26$, GAS: $F_{1,15} = 4.91$, $P = .04$, $\eta_p^2 = .25$). Post hoc comparisons revealed no difference in soleus (young: 15.3 [9.5] N/mm, older: 18.1 [10.6] N/mm, $P = .57$, $d = 0.28$) or gastrocnemius stiffness (young: 6.4 [3.4] N/mm, older: 7.3 [5.2] N/mm, $P = .68$, $d = 0.21$) between young and older adults during passive rotation (Figure 3C and 3E), but greater soleus (young: 277.5 [131.6] N/mm, older: 154.4 [82.3] N/mm, $P = .04$, $d = 1.12$) and gastrocnemius (young: 109.8 [66.7] N/mm, older: 53.1 [29.9] N/mm, $P = .04$, $d = 1.04$) stiffness in young versus older adults at 75% MVIC activation (Figure 3D and 3F).

Discussion

We designed this series of 2 experiments to examine how age-related differences in triceps surae structure influence ankle joint mechanical function across tasks spanning isolated contractions to walking. We anticipated differences in ankle joint quasi-stiffness during walking to arise from known structural changes within the triceps surae muscles and Achilles tendon due to age. Contrary to our hypothesis, we determined that ankle joint quasi-stiffness during the stance phase of walking was not different between the young and older adults. To examine the extent to which muscle activation can modulate ankle joint quasi-stiffness, our second experiment compared isolated passive stretches with active stretches at matched activation levels in young and older subjects. As hypothesized, we found that older and young adults could modulate ankle joint quasi-stiffness via activation-dependent changes in triceps surae muscle length–tension behavior. Also as hypothesized, at a matched activation level, older adults exhibited lower ankle joint quasi-stiffness than young adults. Altogether, these findings suggest that age-related decreases in ankle joint quasi-stiffness may be mitigated, at least in part, via activation-mediated increases in triceps surae muscle stiffness.

Our older adult subjects walked with smaller peak ankle joint moments compared to young adults, agreeing well with hallmark age-related reductions in mechanical output from the triceps surae.[1,2,24,25] However, despite smaller ankle joint moments, the young and older adults walked at similar speeds, with similar stance phase values of ankle joint quasi-stiffness. Our findings are similar to those of Crenna and Frigo,[17] who found no difference in ankle joint quasi-stiffness between young (aged <50 y) and older (aged 65–85 y) adults, and Collins et al,[26] who found no age effect among older adult women (range 65–91 y) during the plantar flexion phase of walking despite characteristic age-related reductions in peak ankle moment. Some age-related changes in gait mechanics[27] and muscle and tendon structural properties[28] have been shown to occur as early as middle age, and most occur before the age of 65 years. Therefore, our finding of no difference in ankle joint quasi-stiffness during walking between distinct groups of young and older adults provides new support for this mechanism. One interpretation of this finding is that, despite underlying neural and musculoskeletal changes due to age, maintaining ankle joint quasi-stiffness is necessary to meet the task demands of walking.[4] Indeed, the prioritization of ankle joint quasi-stiffness is widely observed, even in those with gait pathology, and thus more obvious and acute limitations afflicting the triceps surae muscles. For example, individuals with hemiparesis following stroke or with a total ankle replacement walk with ankle joint quasi-stiffness largely indistinguishable from that in healthy controls.[29,30] However, given the relatively complex nature of how ankle joint quasi-stiffness may be modulated during gait, it is challenging to distinguish the presence of age-related adaptive responses based on this outcome measure alone.

Unlike during walking, age had a significant effect on ankle joint quasi-stiffness and triceps surae muscle stiffness during isolated contractions at a matched activation level. Specifically, at 75% MVIC, the older adults averaged 44% less ankle joint quasi-stiffness accompanied by 44% less soleus muscle stiffness, and 52% less gastrocnemius stiffness compared to young adults. The differences in stiffness between the gastrocnemius and soleus muscles are due to the different force-generating capacities of each muscle, herein based on the relative physiological cross-sectional area.[22] Given that the

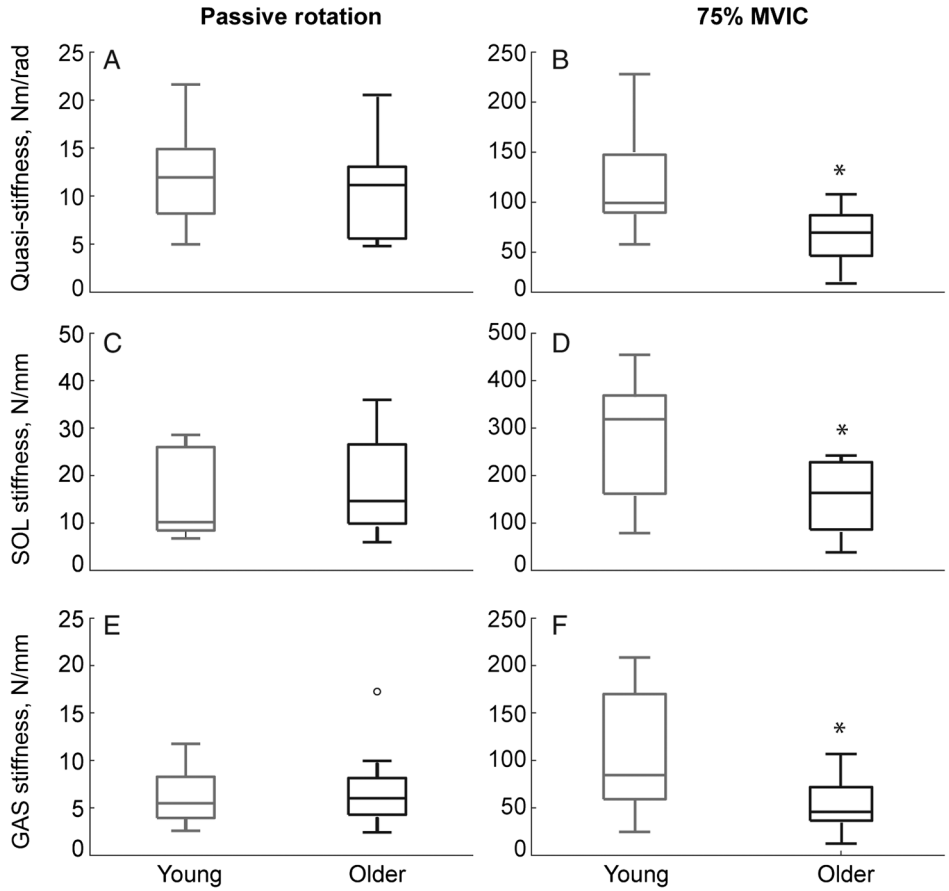

Figure 3 — Box and whisker plots of ankle joint quasi-stiffness (A and B), soleus muscle stiffness (C and D), and gastrocnemius muscle stiffness (E and F) during passive ankle rotation (left column) and at 75% maximum voluntary isometric contraction activation (right column) in young and older adults. Horizontal lines within each box indicate the median, the boundaries indicate the 25th and 75th percentiles, the whiskers indicate the highest and lowest values, and the open circle (○) indicates an outlier. SOL indicates soleus; GAS, gastrocnemius. *Significant pairwise comparison between young and older adults ($P < .05$).

isolated contractions were performed using an ankle joint range of motion that resembled the range of motion during the stance phase of walking, the principal difference between these measurements and those measured during walking is the manner in which activation regulated the triceps surae muscle length–tension behavior. Conceptually, altered muscle stiffness across a fixed muscle-tendon unit length change (ie, fixed ankle joint range of motion) may emerge from between-group differences in the rise in muscle–tendon unit force and/or in fascicle lengthening. Our data would suggest that the significant age-related reduction in muscle stiffness was more related to a smaller rise in muscle–tendon unit force than to increased fascicle lengthening across the range of motion tested.

Unfortunately, we are unable to distinguish between muscle and tendon contributions to the measured rise in muscle–tendon unit force. We offer 2 potentially interrelated explanations for our findings. First, older adults generally have a smaller capacity for generating maximum triceps surae muscle force than young adults.[12,13] Age-related changes in human muscle length–tension behavior have not been well-documented in the literature. Nevertheless, a reduction in maximum force-generating potential would ostensibly act to scale down each triceps surae muscle's length–tension curve at a prescribed level of activation. Here, eccentric action of the triceps surae muscles at a matched 75% MVIC activation elicited the same relative amount of fascicle lengthening in older and younger adults. Accordingly, we would anticipate a smaller rise in force in older than young adults as a consequence of their scaled-down length–tension relation, which, thereby, would elicit reduced stiffness at the individual muscle and ankle joint levels. Second, given evidence suggesting that Achilles tendon stiffness decreases with age,[9] it is equally plausible that the smaller rise in muscle–tendon unit force in older adults at 75% MVIC activation can be explained in part by smaller tendon force contributions.

Conversely, in the absence of muscle activation, the young and older adults exhibited similar mechanical behavior at the muscle and ankle joint levels. In fact, consistent with earlier studies, we found relatively negligible values of triceps surae muscle stiffness and ankle joint quasi-stiffness during passive rotation across the range of motion tested.[6,31] It is possible that our selected dorsiflexion range of motion (ie, 15°) failed to sufficiently stretch the triceps surae during passive rotation. Thus, our data during passive rotation do not necessarily conflict with the notion of a more compliant Achilles tendon in old age.[9] Indeed, in the absence of muscle activation, the subjects' Achilles tendons may have been operating at or below the toe region of the stress–strain relation. This is consistent with a recent comparative study showing no effect of age on tendon behavior near the toe region.[32]

There are several limitations in these studies. First, the soleus and gastrocnemius muscles are composed of different regions that vary in architecture. We only imaged the posteromedial region of these muscles, which may not be representative of the other regions, and ultrasound imaging may not adequately capture their 3-dimensional behavior. In addition, age-related differences in relative motor unit recruitment of the triceps surae muscles may contribute to age-related differences in ankle joint quasi-stiffness not accounted for this in this study. We did not measure triceps surae muscle activation or length–tension behavior during walking trials in experiment 1. However, age-associated increases in soleus activation during walking have been previously observed in experimental studies[33,34] and in simulation studies in response to increased Achilles tendon compliance.[8] Length–tension data would be very difficult to reliably capture at matched activations during walking, which is one of the key strengths of our isolated contractions protocol in experiment 2. We also note that the older adults in these studies were healthy and active. Therefore, these results may not generalize to older adults who are less mobile or who have various pathologies that may preclude their ability to preserve ankle joint quasi-stiffness during walking. Nevertheless, our inclusion/exclusion criteria were selected to minimize the effects of potentially confounding variables, such as chronic disease or past history of injury. Our sample sizes were relatively small, increasing the chance of type II statistical error. This study was a focused study intended to inform larger-scale studies. Therefore, although we did not make adjustments for multiple comparisons, we provide a Supplementary Material (available online) that includes all individual subject data supporting the primary outcomes reported in this study. Finally, because they were collected at different times, the majority of our subject cohort differed between experiments 1 and 2. Nevertheless, the small intersubject variance in our primary outcomes (see Figure 2 and Supplementary Figure 1 [available online]) suggests that our findings are generalizable across samples of young and older adults. However, our mechanistic explanations, drawn from findings spanning both studies, should be interpreted conservatively.

In summary, we found that, despite age-related reductions in ankle joint quasi-stiffness during controlled conditions at a matched level of muscle activation, young and older adults did not exhibit differences in ankle joint quasi-stiffness during walking. Our collective findings suggest that, during normal walking, older adults may maintain quasi-stiffness via a local adaptive response of increasing triceps surae activation. This apparent local adaptive response may be a stop gap, given upper limits on muscle recruitment, that warrants further study. These findings contribute to our basic understanding of triceps surae muscle–tendon interaction and its role in age-related changes in walking performance.

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