Published in final edited form as:
Phys Ther. 2007 November ; 87(11): 1422–1432.

Age-Related Changes in Strength, Joint Laxity, and Walking Patterns: Are They Related to Knee Osteoarthritis?

Katherine S Rudolph, PT, PhD [Assistant Professor],
Department of Physical Therapy and Program in Biomechanics and Movement Science, University of Delaware, 301 McKinly Lab, Newark, DE 19716 (USA)

Laura C Schmitt, PT, PhD [Post-Doctoral Fellow], and
Department of Pediatrics, University of Cincinnati, College of Medicine; and Physical Therapist, Sports Medicine Biodynamics Center, Cincinnati Children's Hospital Medical Center, Cincinnati, Ohio

Michael D Lewek, PT, PhD [Assistant Professor]
Center for Human Movement Science, Division of Physical Therapy, University of North Carolina at Chapel Hill, Chapel Hill, NC

Abstract

Background and Purpose—Aging is associated with musculoskeletal changes and altered walking patterns. These changes are common in people with knee osteoarthritis (OA) and may precipitate the development of OA. We examined age-related changes in musculoskeletal structures and walking patterns to better understand the relationship between aging and knee OA.

Methods—Forty-four individuals without OA (15 younger, 15 middle-aged, 14 older adults) and 15 individuals with medial knee OA participated. Knee laxity, quadriceps femoris muscle strength (force-generating capacity), and gait were assessed.

Results—Medial laxity was greater in the OA group, but there were no differences between the middle-aged and older control groups. Quadriceps femoris strength was less in the older control group and in the OA group. During the stance phase of walking, the OA group demonstrated less knee flexion and greater knee adduction, but there were no differences in knee motion among the control groups. During walking, the older control group exhibited greater quadriceps femoris muscle activity and the OA group used greater muscle co-contraction.

Discussion and Conclusion—Although weaker, the older control group did not use truncated motion or higher co-contraction. The maintenance of movement patterns that were similar to the subjects in the young control group may have helped to prevent development of knee OA. Further investigation is warranted regarding age-related musculoskeletal changes and their influence on the development of knee OA.

Address all correspondence to Dr Rudolph at: krudolph@udel.edu.

Dr Rudolph and Dr Schmitt provided writing and project management. Dr Schmitt and Dr Lewek provided data collection. All authors provided data analysis. Dr Rudolph provided fund procurement, facilities/equipment, and institutional liaisons. Dr Lewek provided consultation (including review of manuscript before submission). The authors acknowledge William Newcomb, MD, for providing subject recruitment and Laurie Andrews, RTR, for assistance with radiographs.

This work was presented as a platform presentation at the Combined Sections Meeting of the American Physical Therapy Association; February 14, 2003; Tampa, Fla; as a platform presentation at XVth Biannual International Society of Electrophysiology and Kinesiology Conference; June 20, 2004; Boston, Mass; and at the Functional Fitness and the Science of Joint Preservation Course, University of Louisville, Louisville, Ky, April 28–29, 2006.

Symptomatic knee osteoarthritis (OA) is a worldwide problem[1-4] that produces substantial disability in middle-aged and older adults and leads to a tremendous economic burden on society.[5] The prevalence of OA among older individuals has led some authors[6] to regard its development as a normal part of aging. Loeser and Shakoor,[6] however, suggested that age-related changes in musculoskeletal tissue, such as muscle weakness and ligament laxity, do not directly cause OA, but may predispose individuals to develop the disease. It is possible that the manner in which people respond to these age-related changes in musculoskeletal tissues about the knee may be related to whether or not OA develops in the knees of older adults.

The features that are similar between older adults and people with knee OA include quadriceps femoris muscle weakness and altered knee movement during walking. Sarcopenia is well known in older adults and leads to quadriceps femoris muscle weakness,[7-11] which has been noted in people as early as 40 years of age.[9] Because both the development of knee OA[12] and quadriceps femoris muscle strength changes[9] are initiated during middle age, it is not surprising that quadriceps femoris muscle weakness has been implicated in the development of knee OA.[13-15]

Quadriceps femoris muscle weakness also is associated with adaptations in walking patterns that are theorized to put articular cartilage at risk. For instance, subjects with knee OA who have weaker quadriceps femoris muscles exhibit less stancephase knee motion during walking.[16] At self-selected walking speeds, it is the role of the quadriceps femoris muscles to control knee flexion during weight acceptance while the hamstring and gastrocnemius muscles are typically silent.[17] However, in the presence of quadriceps femoris weakness, which occurs with aging,[7-11] and in the presence of knee OA, either the hamstring or the gastrocnemius muscles may be required to assist with knee control.

Activation of muscles surrounding the knee can occur selectively and with precise timing that allows for normal knee motion, or, alternatively, activation can occur more generally as a global co-contraction pattern that could limit joint motion. We, therefore, have defined the movement strategy that involves both increased muscle co-contraction and reduced knee flexion during walking as a "stiffening strategy." Excessive muscle co-contraction can lead to excessive joint contact forces,[18] and reduced knee motion during weight acceptance can cause higher impact loads in the knee.[19,20] Older adults are known to walk with less knee flexion,[21] but whether they do so as a result of higher muscle co-contraction is unknown. However, not all older adults develop knee OA. If older adults who have not developed knee OA walk with a knee stiffening strategy, then the combination of reduced knee flexion and muscle co-contraction alone, in the presence of quadriceps femoris muscle weakness, is unlikely to contribute to the development of knee OA.

Another possible precursor to knee OA is excessive frontal-plane laxity, which is common in people with existing knee OA.[22-24] Specifically, greater frontal-plane knee laxity is observed in both the involved and uninvolved knees of people with OA compared with control subjects, suggesting that laxity may precede the development of knee OA.[22] Additionally, a significant correlation between frontal-plane laxity and age has been observed in individuals without evidence of knee OA.[22] This finding is consistent with the findings of other studies,[22,25,26] indicating that the material properties of ligaments of older adults can lead to excessive joint laxity. Because frontal-plane laxity has been related to high muscle co-contraction in individuals with knee OA,[24] it is plausible that normal age-related increases in joint laxity also may contribute to higher muscle co-contraction patterns and predispose individuals to develop knee OA. Whether older adults have greater frontal-plane knee laxity coupled with higher muscle co-contraction is not known.

In this study, we investigated the knee laxity, quadriceps femoris muscle strength (force-generating capacity), walking patterns, and muscle activation patterns in 3 age groups of people without symptomatic or radiographic knee OA to examine factors that are thought to contribute to the development of knee OA. The results are discussed in relation to characteristics of a group of people with knee OA. Because the older adults in our study did not have joint degeneration, we hypothesized that older adults who are healthy will have weaker quadriceps femoris muscles and increased frontal-plane knee laxity but will not exhibit greater muscle co-contraction patterns compared with young or middle-aged people.

Method

Subjects

Fifty-nine people were recruited from the community or were referred by a local orthopedic surgeon to participate in the study. All subjects signed an informed consent statement approved by the Human Subjects Review Board of the University of Delaware. Forty-four participants who reported no history of knee OA (confirmed by radiograph) or previous lower-extremity injury comprised 3 control groups (15 younger individuals [ages 18-25 years], 15 middle-aged individuals [ages 40-59 years], and 14 older individuals [ages 60-80 years]) (Tab. 1). The middle-aged individuals were matched by age and sex to 15 people with symptomatic, medial knee OA (Tab. 1). The subjects with medial knee OA were part of a larger study of people who were going to undergo a high tibial osteotomy. They had no history of knee ligament injury; however, those individuals with a history of meniscectomy were included. Data on some of the people with knee OA and data on the middle-aged control group have been reported previously.[24] Radiographic information, isometric quadriceps femoris strength, and kinematic, kinetic, and electromyographic (EMG) data during walking were collected from the moreinvolved limb of the subjects with OA and a randomly chosen limb of the control subjects. The test limb of the control subjects was chosen randomly to avoid any possible influence of limb dominance.

Procedure

Radiographs—The diagnosis of OA is based on the presence of knee pain in conjunction with age over 50 years and either radiographic evidence of OA (eg, osteophytes) or other symptoms such as stiffness or crepitus.[27] Although none of our control subjects complained of knee pain or stiffness, standing posterioranterior (approximately 30° of knee flexion) radiographs of the knees of the middle-aged and older control groups were obtained as an added precaution to rule out the presence of knee OA. Radiographs were not taken of the knees of the young subjects because they had no knee symptoms and no history of knee injury and were unlikely to have undiagnosed knee OA based on the above definition.

Varus and valgus stress radiographs were taken of the tested lower extremity in the middle-aged and older control groups as well as the OA group. Subjects were positioned supine on a radiograph table with the knee flexed to 20 degrees and the patella facing anteriorly. The x-ray tube was centered approximately 100 cm above the knee joint. A TELOS* stress device was used to apply a 150-N force in the varus or valgus direction (Fig. 1). Medial and lateral joint spaces were measured at the narrowest location in both compartments using calipers. X-ray beams were adjusted for magnification using a known distance from the TELOS device that was visible in every image. Medial and lateral joint laxities were calculated as described in Figure 1.[28] Interrater reliability was assessed by repeated testing on a subset of 8 subjects and showed high reliability for medial (intraclass correlation coefficient [ICC]=.96) and lateral laxity (ICC=.98).

*Austin & Associates, 1109 Sturbridge Rd, Fallston, MD 21047.

Skeletal alignment of the tested limb was measured with a standing long cassette radiograph for the middle-aged and older control groups and the OA group. Subjects stood, without footwear, with the tibial tubercles facing forward and the x-ray beam centered at the knee from a distance of 2.4 m. Alignment was measured as the angle formed by the intersection of the mechanical axes of the femur and tibia.[29-31] A knee was in varus alignment when the intersection of the lines was >0 degrees in the varus direction and was in valgus alignment when the intersection of the lines was >0 degrees in the valgus direction.[30]

Quadriceps femoris muscles function—Quadriceps femoris muscle force output was measured with an isokinetic dynamometer (Kin-Com 500H).[†] Each subject sat with the knee and hip flexed to 90 degrees, the knee joint axis aligned with the dynamometer axis, and the trunk fully supported. Thigh and hip straps secured each subject in the seat, while an ankle strap secured the shank to the dynamometer. Subjects performed a maximal volitional isometric contraction (MVIC) on which a supramaximal burst of electrical current (Grass S48 stimulator[‡]) (100 pulses/second, 600-microsecond pulse duration, 10-pulse tetanic train, 130 V) was applied. The burst superimposition was used for the measurement if the subjects were providing maximum activation of the quadriceps femoris muscles.[32] A central activation ratio (CAR) is a ratio between the highest volitional force (measured as the peak force before the electrical burst was applied) and the force achieved during the electrically elicited burst. Maximum volitional isometric contraction was measured as the highest volitional force (N) during the contraction and was normalized to body mass index (BMI) (N/BMI). In tests on 10 subjects who were healthy, repeated testing of the MVIC revealed an intraclass correlation coefficient (2,1) of .98.[33]

Gait and electromyographic (EMG) data—To determine knee motion during walking, the motions of the lower-extremity segments were collected by a 6-camera, passive, 3-dimensional motion analysis system (Vicon 512)[§] at 120 Hz. Cameras were calibrated to detect markers within a volume that was 1.5 × 2.4 × 1.5 m. Calibration residuals were kept below 0.6 mm. The cameras detected retroreflective markers (2.5 cm in diameter) placed on the tested lower extremity. Markers were placed bilaterally over the greater trochanters, the lateral femoral condyles, and lateral malleoli for identification of appropriate joint centers. Thermoplastic shells with 4 rigidly attached markers were used to track segment motion. The shells were secured on the posterior-lateral aspects of the thigh and shank. Previous work in our laboratory (unpublished data collected April 2002) has revealed good reliability for kinematic variables with ICCs ranging from .6343 to .9969. The ICCs for the kinematic variables used in the present study ranged from .9721 to .9969. Errors in estimating bone movement from skin mounted markers for sagittal and frontal plane motions are approximately 2 to 3 degrees during the stance phase of walking.[34,35] Vertical, medial-lateral, and anterior-posterior ground reaction forces were collected from a 6-component force platform (Bertec force platform, model 60905[‖]) and sampled at 1,920 Hz. Ground reaction force data were used to calculate moments about the knee and for determination of heel-strike and toe-off.

Electromyographic data were collected with a 16-channel electromyography system (model MA-300-16[#]) sampled at 1,920 Hz. After skin preparation, surface electrodes with parallel, circular detection surfaces (1.14 cm in diameter, 2.06 cm apart), a common mode rejection ratio (100 dB at 65 Hz), and a signal detection range of less than 2 μV for the built-in preamplifier were placed over the mid-muscle bellies of the lateral quadriceps femoris (LQ),

[†]Isokinetic International, 6426 Morning Glory Dr, Harrison, TN 37341.
[‡]Grass Instrument Division, Astro-Med Inc, 600 East Greenwich Ave, West Warwick, RI 02893.
[§]Oxford Metrics, 14 Minns Business Park, West Way, Oxford OX2 0JB, United Kingdom.
[‖]Bertec Corp, 6171 Huntley Rd, Ste J, Columbus, OH 43229
[#]Motion Lab Systems, 15045 Old Hammond Hwy, Baton Rouge, LA 70816.

medial quadriceps femoris (MQ), lateral hamstring (LH), medial hamstring (MH), lateral gastrocnemius (LG), and medial gastrocnemius (MG) muscles. Electromyographic data were recorded for 2 seconds at rest and during an MVIC for each muscle group for normalization.

Motion, force, and EMG data were collected simultaneously as subjects walked at a self-selected speed along a 9-m walkway for 10 trials. Walking speed was recorded from 2 photoelectric beams to ensure that speed did not vary more than 5% from their self-selected speed during the trials. Trials were only accepted if the subject walked at a consistent speed and walked across the force platform without adjusting their stride in any way to contact the force platform.

Data management—Marker trajectories and ground reaction forces were collected over the stance phase of one limb (heel-strike to toe-off on the force platform) and were filtered with a second-order, phase-corrected Butterworth filter with a cutoff frequency of 6 Hz for the video data and 40 Hz for the force-plate data. Sagittal- and frontal-plane knee angles and external knee moments were calculated with Euler angles and inverse dynamics, respectively (Visual 3D[**]). Data were analyzed using custom-written computer programs based on strict criteria (eg, thresholds for initial contact, time of peak adduction moment) to eliminate tester bias. Data were analyzed during the loading interval, which we defined as from 100 milliseconds prior to initial contact (to account for electromechanical delay)[36] through the first peak knee adduction moment. Data during the loading interval were time normalized to 100 data points and averaged across each subject's trials. Knee moments were normalized to body mass × height and are expressed as external moments. In addition to discrete variables, we calculated knee flexion excursion (from initial contact to peak knee flexion) and knee adduction excursion during loading.

All EMG data were band-pass filtered from 20 to 350 Hz. A linear envelope was created with full-wave rectification and filtering with a 20-Hz low-pass filter (eighth-order, phasecorrected Butterworth filter). The linear envelope was normalized to the maximum EMG signal obtained during a MVIC for each muscle.

Custom-designed software (Labview, version 8.0[††]), using the same kinematic and kinetic events as stated above, was used to analyze all EMG data. Magnitude of muscle activity and co-contraction between opposing muscle groups were analyzed over the loading interval after it was time normalized to 100 points. Magnitude of muscle activity was expressed as the average rectified value across the loading interval. *Co-contraction* was operationally defined as the simultaneous activation of a pair of opposing muscles and was calculated using an equation developed in our laboratory[37]:

$$\text{Average co-contraction value} = \frac{\left[\sum_{i=1}^{n} \frac{lowerEMGi}{bigberEMGi}(lowerEMGi + bigberEMGi)\right]}{n}$$

where *i* is the sample number and *n* is the total number of samples in the interval. Co-contraction values were averaged across the trials, and the average was used for analysis. This method does not identify which muscle is more active; rather, it represents a relative activation of 2 muscles while accounting for the magnitudes of both muscles. Co-contraction was calculated between the LQ and LH (LQH), MQ and MH (MQH), LQ and LG (LQG), and MQ and MG (MQG) muscles.

[**]C-Motion Inc, 15821-A Crabbs Branch Way, Rockville, MD 20855.
[††]National Instruments, 11500 N Mopac Expwy, Austin, TX 78759-3504.

Data Analysis

Group means and standard deviations were calculated for all data. One-way analysis of variance (ANOVA) was used to detect group differences in BMI, quadriceps femoris muscle strength and CAR, and radiograph variables. Because walking speed can influence lower-extremity kinematic and kinetic data,[38-40] analysis of covariance (ANCOVA), with walking velocity as a covariate, was used to detect group differences in kinematic and kinetic variables. For strength, radiograph, and kinematic and kinetic data, significance was established when $P \leq .05$. To detect group differences in magnitudes of muscle activity and in co-contraction variables, 95% confidence intervals were used to evaluate differences in the mean values.

Results

Subjects in the middle-aged control group had greater BMI values than the subjects in the young control group ($P=.030$), and subjects in the OA group had higher BMI values than the subjects in the young and older control groups ($P \leq .002$) (Tab. 1). The knees of the subjects in the middle-aged and older control groups were in less varus than the knees of the subjects in the OA group ($P<.001$) (Tab. 1).

Knee Laxity

Subjects in the OA group had significantly greater medial laxity than the subjects in the middle-aged and older control groups ($P=.001$) (Fig. 2). There were no differences in lateral laxity between the subjects in the OA group and the subjects in the middle-aged and older control groups ($P=.272$) (Fig. 2).

Quadriceps Femoris Muscle Strength

The subjects in the young control group produced greater volitional quadriceps femoris muscle force than the subjects in the older control group ($P<.001$) or the subjects in the OA group ($P<.001$) (Fig. 3). Subjects in the middle-aged control group generated more force than the subjects in the older control group ($P=.002$) or the subjects in the OA group ($P=.003$) (Fig. 3). There were no differences between the subjects in the older control group and the subjects in the OA group ($P=1.0$) or between the subjects in the young and middle-aged control groups ($P=.974$) (Fig. 3). No differences in CAR were observed among the control groups (young=0.93±.038 [$\bar{X}$±SD], middle-aged=0.93±.027, older=0.94±.052; $P=.84$).

Gait Characteristics

The subjects in the middle-aged control group walked faster than the subjects in the OA group ($P=.023$) and there were no other statistical differences among the groups (young control group=1.39±0.08 [$\bar{X}$±SD] m/s, middle-aged control group=1.51±0.15 m/s, older control group=1.45±0.10 m/s, OA group=1.38±0.12 m/s).

Results of kinematic and kinetic variables are shown in Table 2. Knee flexion excursion was not different among the young, middle-aged, and older control groups, but the OA group showed less knee flexion excursion compared with all 3 control groups ($P \leq .036$). The peak knee flexion moment was no different among the subjects in the young, middle-aged, and older control groups, but was reduced in subjects in the OA group compared with the subjects in the young ($P=.006$) and older ($P=.039$) control groups. There were no differences in frontal-plane knee motions or moments among the young, middle-aged, and older control groups. The subjects in the OA group exhibited greater adduction compared with the subjects in the young, middle-aged, and older control groups at initial contact ($P \leq .004$) and at peak adduction during loading ($P \leq .001$). The OA group showed greater adduction excursion compared with the young control group ($P=.049$) and the older control group ($P=.055$); however, the latter was not

statistically significant at the $P \leq .05$ level. The OA group showed greater peak knee adduction moments compared with the young, middle-aged, and older control groups ($P < .002$).

Muscle Activity

There was a large degree of variability in the muscle activation and co-contraction as is evident in the large range of the 95% confidence intervals shown in Figures 4 and 5. During loading response, there was a tendency for the subjects in the older control group to use higher lateral quadriceps femoris activity than the subjects in the young and middle-aged control groups and a tendency for higher medial gastrocnemius muscle activity in the subjects in the OA group and the older control group than in the subjects in the young and middle-aged control groups. However, the overlap of the 95% confidence intervals indicate that a larger sample size is needed to determine with more certainty whether the population means are different. In terms of muscle co-contraction, there were no differences among the control groups, although the OA group showed higher co-contraction than the subjects in the young control group in the LQG and MQG muscle pairs (Fig. 5).

Discussion and Conclusions

Most studies of age-related differences in movement and muscle activation patterns include samples of young subjects in their 20s and older adults over 60 years of age; yet, age-related changes in characteristics such as muscle strength or neuromuscular responses can occur in middle age[7-11] and may coincide with the development of knee OA. As a result, we intended to investigate characteristics in individuals who are healthy that are purported to be associated with the development of knee OA across a range of ages, including middle age. The novel nature of this approach and the findings of this study provide some insights into how changes in musculoskeletal function might establish an environment in which OA could develop. The results set the stage for future research into how age-related musculoskeletal changes might influence the development of knee OA.

The results of this study indicate that healthy aging was associated with a considerable loss of quadriceps femoris muscle strength in the older adults, although we did not observe increased frontal-plane laxity in those subjects. Despite quadriceps femoris muscle weakness, the older adults participating in this study did not adopt a knee stiffening strategy (ie, reduced knee motion and high muscle co-contraction) that we speculate may contribute to damage of articular cartilage. Despite the small sample size, these findings suggest that the older adults included in this study demonstrate movement strategies similar to those of younger individuals, which may have helped to prevent the development of knee OA as they aged; these findings, however, warrant further investigation.

As age-related muscle weakness develops, individuals must adapt their movements and muscle activity patterns to accommodate the diminished force-generating capacity of their aging muscles to maintain a certain level of function. As such, we propose that adaptations allowing for the continuation of normalized joint mechanics and muscle activation patterns are less likely to predispose the joint to articular cartilage damage. A failure to adapt to strength declines might contribute to the development of movement patterns similar to individuals with quadriceps femoris muscle weakness due to knee joint pathology.[41-43] Because the older adults in this study exhibited similar movement and muscle activity patterns to those in the younger age groups, it appears that they have discovered a successful approach to maintaining normal knee function despite their quadriceps femoris muscle strength decline.

In particular, the older control subjects exhibited significantly weaker quadriceps femoris muscles compared to the younger cohorts, yet they showed no differences in knee motion during weight acceptance compared with the young control subjects. Quadriceps femoris

muscle weakness has previously been associated with reduced knee motion during walking in the presence of joint pathology.[33,44] Electromyographic data suggest that the older adults in this study have compensated for the quadriceps femoris muscle weakness by selectively increasing quadriceps activity during loading. Although adequate muscle activity is necessary to ensure joint stability, too much activation can result in limited knee flexion and increased impact load on the knee.[19] Whether increased activation would be a positive or negative adaptation during walking, therefore, would depend on the end result of the muscle activity. The older adults in this study were able to maintain normalized knee motion, comparable to younger subjects, with increased quadriceps femoris activity. The ability to maintain normalized knee joint mechanics may have contributed to the lack of knee OA in this older adult cohort. Our conclusions are limited by the cross-sectional design of this study. A longitudinal study would be required to further investigate the effect of age-related musculoskeletal changes on movement strategies in terms of the development of knee OA.

Despite prior evidence of reduced stiffness and ligament strength with advancing age,[25] we were unable to detect increases in frontal plane laxity with aging in the control subjects. The OA group, however, exhibited increased frontal-plane laxity. Although subjects were carefully screened for a history of ligament injury, we included individuals with a history of meniscal damage in the OA group. The subjects with OA had no history of an incident ligamentous injury, and studies[45,46] suggest that meniscal injury in the absence of a traumatic event is a part of the degenerative process of knee OA. In individuals with knee OA, the presence of increased frontal plane laxity is known to degrade the relationship between strength and physical function.[47]

Because the older adults who were healthy did not have to cope with strength loss in a lax joint, they may have had the ability to adopt movement strategies that remain normalized and may be “joint sparing.” We can speculate that the subjects with OA did not have such an option, because they had to contend with strength loss in a lax joint, making joint stabilization a primary determinant in their adopted control strategy. These findings suggest that quadriceps femoris muscle weakness is associated with reduced stance-phase knee motion in the presence of other factors, such as increased knee laxity or pain, as was evident in the OA group. Such a speculation would suggest that age-related changes to musculoskeletal tissues alone are insufficient to lead to the development of knee OA, provided the aging individual has the means to maintain normal movement strategies.

The similarity in the knee motion among the control groups might be unexpected because other researchers[21] have shown that older adults walk with less knee motion during loading when walking at the same speed as younger subjects. It is possible that our finding of similar sagittal-plane knee kinematics among the young, middle-aged, and older control groups is due to a small number of subjects in our sample or related to our method of subject recruitment. Some of the older adults in our study were recruited from local fitness and senior centers and may represent a more active older adult compared with a typical older adult, and this may have enabled the older subjects in our study to better control more knee motion as the limb accepted weight. Future studies may consider measuring daily activity levels to account for potential influences on walking speeds and movement patterns.

It is interesting to note that differences in muscle co-contraction values were found only between the subjects with OA and young control subjects. In this study, the subjects with OA used greater lateral gastrocnemius muscle activity during loading and greater quadriceps femoris-gastrocnemius muscle co-contraction on the medial and lateral sides compared with the young control subjects, but no differences were observed between the subjects with OA and the middle-aged control subjects. This is in contrast to other work in our lab in which subjects with OA were found to use higher co-contraction between the quadriceps femoris-

gastrocnemius muscles on the medial side only compared with age-matched control subjects.[24] It is possible that people with pathologic conditions in the knee may limit knee motion through different muscle co-contraction strategies.[41,43] Additional research is required to delineate whether consistent differences in muscle activation patterns exist in people with knee OA or whether there are several strategies that people use to help control the knee in the face of a pathologic condition. Whether one strategy is more detrimental than another remains to be seen and should be further investigated.

There are several limitations to the study design that the readers should take into consideration. First, testers were not blinded to group assignment, which may have created bias in recording data. However, the use of the TELOS device to apply uniform stress during the stress radiographs reduced the influence of tester bias for this measure. Testers attempted to provide equivalent verbal encouragement to all subjects equally when testing quadriceps femoris muscle strength. In addition, the discomfort of the superimposed burst was motivation for all subjects to perform to their best ability to avoid repeat testing. During the movement analysis testing, similar instructions were provided to all subjects to walk at a comfortable speed. Custom-written computer algorithms were used to determine data points used in the analysis of kinematics, kinetics, and EMG data to reduce tester bias. Second, the distribution of male and female subjects in the groups was not the same and we did not account for the level of physical activity in the subjects in each group, both of which could have influenced the results. Finally, the subjects all walked faster than has been reported elsewhere,[48] and walking speed —although used as a covariate—may have influenced the results.

The finding that the older adults in our study used what can be considered a favorable movement pattern may suggest why they did not develop knee OA. We speculate that the manner in which middle-aged individuals compensate for age-related neuromuscular changes might influence the future integrity of the knee's articular cartilage. An alternative interpretation of our results is that the process of knee OA may cause changes in movement and muscle activation patterns. The absence of reduced knee motion and higher co-contraction in the middle-aged and older control subjects may have been a consequence of not having developed OA rather than the reason they did not develop OA. Resolution of such a question would entail a large-scale longitudinal study to track changes in movement and muscle activation patterns along with arthritic changes in large numbers of subjects.

Acknowledgements

This study was approved by the Human Subjects Review Board of the University of Delaware.

This study was funded, in part, by a grant (P20-RR16458) from the National Center for Research Resources (KSR), a component of the National Institutes of Health (NIH); a grant (T32HD007490) from the NIH (LCS, MDL); a PODS II grant from the Foundation for Physical Therapy (LCS, MDL); and a Doctoral Research Grant from the American College of Sports Medicine (MDL).

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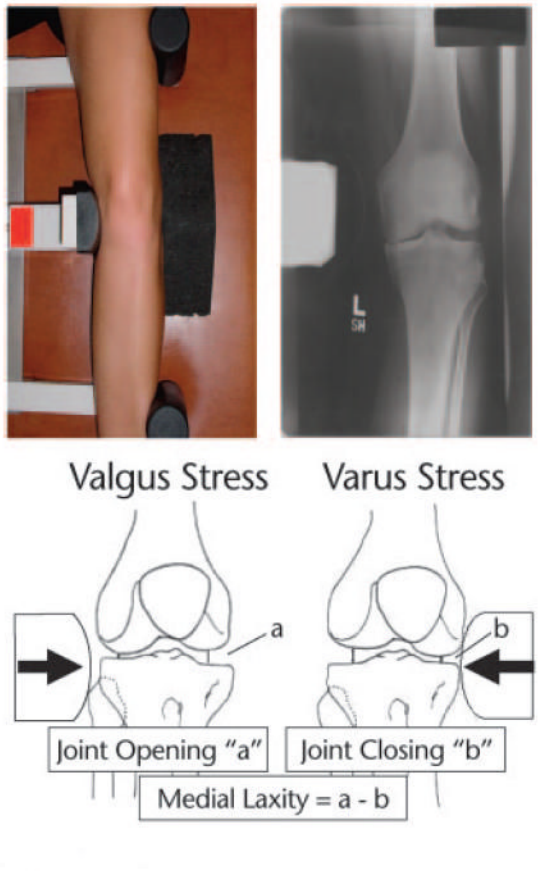

Figure 1.
Setup for stress radiographs. The top images show the limb alignment in the TELOS device (top left) and the resulting radiograph (top right), and the method of calculating medial laxity is shown in the lower images. Lateral laxity was calculated similarly but with subtraction of the lateral joint space in valgus from lateral joint space in varus.

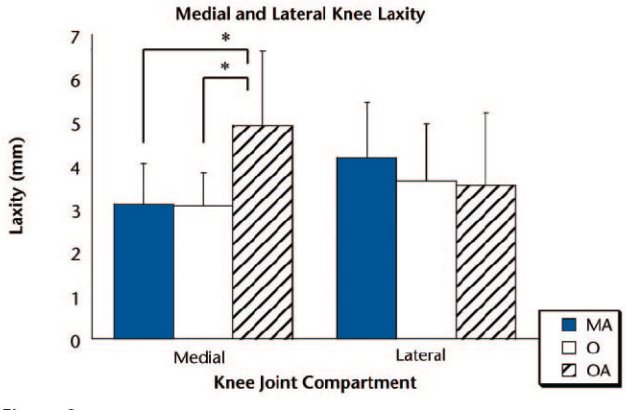

Figure 2.
Medial and lateral joint laxity. MA=middle-aged control group, O=older control group, OA=group with osteoarthritis. * *P*=.001. Error bars represent standard deviation.

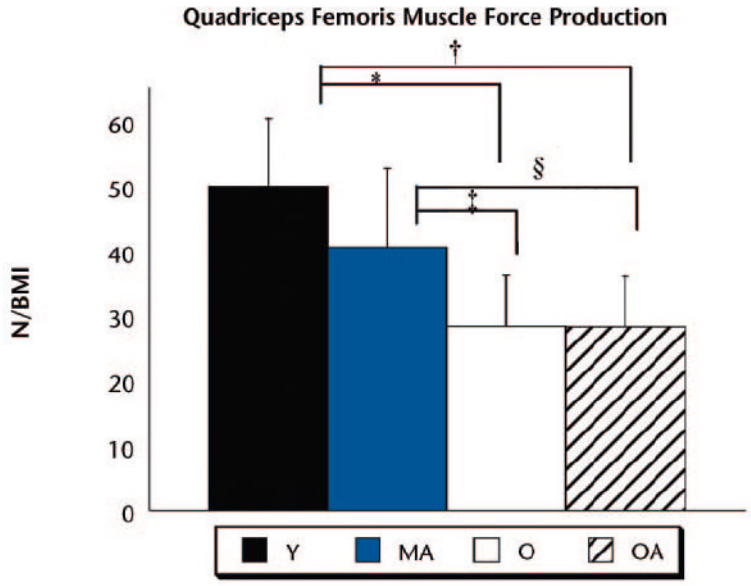

Figure 3.
Quadriceps femoris muscle force production. Y=young control group, MA=middle-aged control group, O=older control group, OA=group with osteoarthritis, N/BMI=highest volitional force during contraction normalized to body mass index. * *P*=.000, † *P*=.000, ‡ *P*=.002, § *P*=.003. Error bars represent standard deviation.

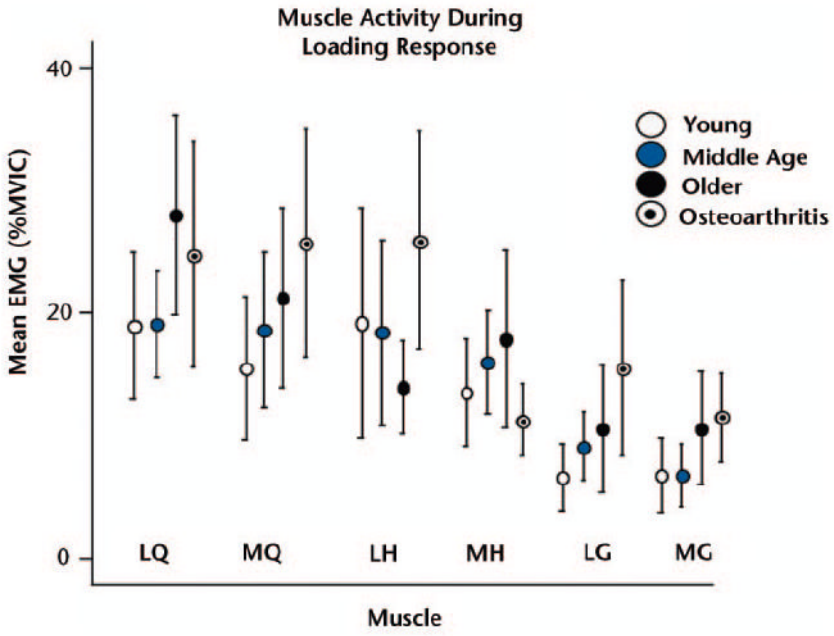

Figure 4.
Mean electromyographic (EMG) muscle activation during loading and 95% confidence interval (indicated by bars). MVIC=maximal voluntary isometric contraction, LQ=lateral quadriceps femoris muscle, MQ=medial quadriceps femoris muscle, LH=lateral hamstring muscle, MH=medial hamstring muscle, LG=lateral gastrocnemius muscle, MG=medial gastrocnemius muscle.

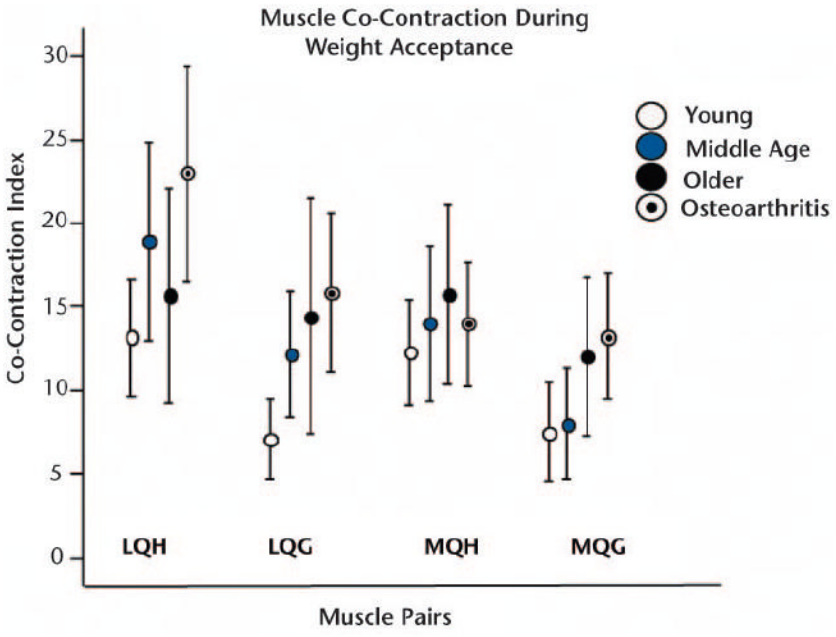

Figure 5.
Mean muscle co-contraction index during loading and 95% confidence interval (indicated by bars). LQH=lateral quadriceps femoris-lateral hamstring, MQH=medial quadriceps femoris-medial hamstring, LQG=lateral quadriceps femoris-lateral gastrocnemius, and MQG=medial quadriceps femoris-medial gastrocnemius muscle pairs.

Table 1

Group Characteristics

	Young Control Group (n=15)	Middle-aged Control Group (n=15)	Older Control Group (n=14)	Osteoarthritis Group (n=15)
Age (y), $\bar{X}$ (range)	20.6 (18-25)	49.2 (40-57)	68.8 (60-80)	49.2 (39-57)
Sex (female/male)	8/7	7/8	10/4	7/8
Body mass index (kg/m^2), $\bar{X}$ (SD)	24.3 (2.8),[ab]	28.7 (5.5)[a]	24.7 (2.5)[c]	30.7 (4.8),[bc]
Alignment (°), $\bar{X}$ (SD)	Not tested	0.1 (1.58) valgus[d]	1.0 (2.09) varus[d]	6.33 (2.39) varus[d]

[a] $P=.030$.

[b] $P=.001$.

[c] $P=.002$.

[d] $P<.001$.

Table 2

Mean Values (Adjusted for Walking Speed) and 95% Confidence Interval (in Parentheses) for Sagittal- and Frontal-Plane Kinematics and Kinetics

	Young Control Group (n=15)	Middle-aged Control Group (n=15)	Older Control Group (n=14)	Osteoarthritis Group (n=15)	*P*
Kinematics (°)					
Sagittal-plane knee angle at initial contact (negative is flexion)	−4.97 (−7.76, −2.18)	−3.59 (−6.48, −0.70)	−5.56 (−8.41, −2.70)	−4.68 (−7.50, −1.85)	.800
Knee flexion excursion during loading	16.75 (14.42, 19.09)	16.97 (14.55, 19.39)	17.94 (15.55, 20.33)	11.98 (9.62, 14.35)	≤.036[a]
Frontal-plane knee angle at initial contact(positive is adduction)	−0.434 (−2.5, 1.64)	−2.30 (−4.45, −0.15)	−0.833 (−2.96, 1.29)	4.83 (2.73, 6.93)	≤.004[a]
Peak frontal-plane knee angle during loading (positive is adduction)	2.62 (0.40, 4.84)	2.35 (0.05, 4.65)	2.18 (−0.10, 4.45)	9.93 (7.68, 12.18)	<.001[a]
Knee adduction excursion during loading	3.06 (1.99, 4.12)[b]	4.65 (3.54, 5.76)	3.00 (1.92, 4.10)[c]	5.10,[bc]	.049[b] .055
Kinetics (N·m/kg·m)					
Peak knee flexion moment during loading	−0.36 (−0.29, −0.44)[b]	−0.27 (−0.19, −0.35)	−0.33 (−0.25, −0.41)[c]	−0.17,[bc]	.006[b] .039[c]
Peak knee adduction moment during loading	0.28 (0.23, 0.32)[a]	0.33 (0.28, 0.37)[a]	0.26 (0.21, 0.31)[a]	0.45 (0.41, 0.50)[a]	≤.002[a]

[a] Osteoarthritis group different from all control groups.

[b] Osteoarthritis group different than young control group.

[c] Osteoarthritis group different than older control group.