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Authors

Segal, NA
Nevitt, MC
Welborn, RD
et al.

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The association between antagonist hamstring coactivation and episodes of knee joint shifting and buckling

N.A. Segal[†,*], **M.C. Nevitt**[‡], **R.D. Welborn**[§], **U.-S.D.T. Nguyen**[||,¶], **J. Niu**[||], **C.E. Lewis**[#], **D.T. Felson**[||], and **L. Frey-Law**[§] **for the MOST Investigative Group**

N.A. Segal: segal-research@uiowa.edu; M.C. Nevitt: mnevitt@psg.ucsf.edu; R.D. Welborn: robert-welborn@uiowa.edu; U.-S.D.T. Nguyen: UyenSa.Nguyen@umassmed.edu; J. Niu: niujp@bu.edu; C.E. Lewis: celewis@uabmc.edu; D.T. Felson: dfelson@bu.edu; L. Frey-Law: laura-freylaw@uiowa.edu

[†]The University of Kansas, Kansas City, KS, United States

[‡]University of California San Francisco, San Francisco, CA, United States

[§]The University of Iowa, Iowa City, IA, United States

[||]Boston University School of Medicine, Boston, MA, United States

[¶]University of Massachusetts Medical School, Worcester, MA, United States

[#]University of Alabama at Birmingham, Birmingham, AL, United States

SUMMARY

Objective—Hamstring coactivation during quadriceps activation is necessary to counteract the quadriceps pull on the tibia, but coactivation can be elevated with symptomatic knee osteoarthritis (OA). To guide rehabilitation to attenuate risk for mobility limitations and falls, this study evaluated whether higher antagonistic open kinetic chain hamstring coactivation is associated with knee joint buckling (sudden loss of support) and shifting (a sensation that the knee might give way).

Design—At baseline, median hamstring coactivation was assessed during maximal isokinetic knee extensor strength testing and at baseline and 24-month follow-up, knee buckling and shifting

[*]Address correspondence and reprint requests to: N.A. Segal, Department of Rehabilitation Medicine, The University of Kansas, 3901 Rainbow Boulevard, Mailstop 1046, Kansas City, KS 66160, United States. Tel: 1-913-588-6944.

Competing interest statement

The authors have no professional relationships with companies or manufacturers who will benefit from the results of the present study.

Author contributions

Responsibility for the integrity of the work as a whole: NA Segal.
Study concept and design: NA Segal, MC Nevitt.
Acquisition of data: M Nevitt, DT Felson, CE Lewis, L Frey Law.
Analysis and interpretation of data: NA Segal, MC Nevitt, L Frey Law, RD Welborn, J Niu, US Nguyen.
Drafting of the manuscript: NA Segal, RD Welborn, MC Nevitt.
Critical revision of the manuscript for important intellectual content: all authors.
Statistical Expertise: J Niu.
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was self-reported. Associations between tertiles of co-activation and knee (1) buckling, (2) shifting and (3) either buckling or shifting were assessed using logistic regression, adjusted for age, sex, knee OA and pain.

Results—1826 participants (1089 women) were included. Mean ± SD age was 61.7 ± 7.7 years, BMI was 30.3 ± 5.5 kg/m^2 and 38.2% of knees had OA. There were no consistent statistically significant associations between hamstring coactivation and ipsilateral prevalent or incident buckling or the combination of buckling and shifting. The odds ratios for incident shifting in the highest in comparison with the lowest tertile of coactivation had similar magnitudes in the combined and medial hamstrings, but only reached statistical significance for lateral hamstring coactivation, OR(95%CI) 1.53 (0.99, 2.36).

Conclusions—Hamstring coactivation during an open kinetic chain quadriceps exercise was not consistently associated with prevalent or incident self-reported knee buckling or shifting in older adults with or at risk for knee OA.

Keywords

Muscle activation; Knee; Osteoarthritis; Epidemiology

Introduction

Osteoarthritis (OA) represents joint failure, with loss of joint protective mechanisms. Coordination of contraction and relaxation of the agonist and antagonist muscles that bridge the joint become increasingly important in preventing episodes of instability during movement. Even before activity-related pain and mechanical instability develop, episodes of buckling or shifting may occur.

Buckling, episodes of sudden loss of postural support across the knee upon weight acceptance, contributes to significant functional limitations and increases risk for falls[1]. Factors cross-sectionally associated with buckling include quadriceps weakness, anterior cruciate ligament (ACL) tears, the presence and severity of radiographic tibiofemoral OA, a history of injury, obesity and pain in adjacent joints[2–4]. Buckling is a functionally significant impairment that can limit mobility and restrict participation in activities[5]. Several studies have indicated that independent of knee pain, age, sex, and BMI, buckling significantly limits physical function[1,4,6,7]. Shifting, a sensation that the knee joint will give way when it does not actually do so, is closely related and may be a symptom that precedes development of buckling. Shifting confers the same negative consequences as knee buckling—falls and fear of falls—limiting mobility and participation in activities[7].

Individuals with knee OA, particularly those with knee pain, have an increased risk for falls and fall-related injuries and fractures[8–14], and many fall during an episode of knee buckling[1]. Fear of falling has an adverse impact on quality of life and physical function above and beyond these injuries[15,16]. Falls can be prevented and balance confidence restored through exercise-based interventions[17,18]. Prevention of buckling through neuromuscular training is a potential focus for interventions to prevent falls and functional

limitations in people with, or at risk for, knee OA. However, for exercise-based interventions to be effective, the underlying cause of falls should be targetted[15].

Coactivation of the hamstrings during quadriceps contraction is necessary for joint stability, even in individuals without knee OA—serving to dynamically counteract the anterior pull of the quadriceps on the tibia, through assisting the passive stabilizer, the ACL[19–24]. Older adults with knee OA demonstrate higher levels of muscle coactivation around the knee than those without OA, as well as reduced knee range of motion during gait[25,26]. Both coactivation and reduced range of motion may be compensations intended to "stiffen" the joint, particularly for those with a sense of instability[26–28].

Normally, agonist activation and antagonist coactivation occur in distinct on/off cycles. However, in those with severe tibiofemoral OA, coactivation occurs throughout the stance phase of gait[29]. It is unknown whether abnormal levels of coactivation are adaptive for inducing a sense of joint stability or maladaptive, destabilizing the joint through reducing the net knee extensor torque, precipitating buckling or shifting episodes. However, there is evidence that coactivation may be modified with directed rehabilitation[30]. Additionally, muscle coactivation is, in part, a generalized motor control strategy that some individuals appear to be more prone to utilize, even when assessed using open kinetic chain strength testing[31]. This assessment of muscle coactivation during an open kinetic chain test condition is a relatively simple assay that could be assessed in clinical rehabilitation settings. Therefore, advancing understanding of coactivation patterns in those with or at risk for buckling or shifting may enable design of effective rehabilitative interventions.

In this study, we measured knee extensor and flexor muscle activity during isokinetic strength testing, using surface electro-myography (sEMG) as an indicator of knee muscle coactivation. We then tested the hypotheses that activation level of the hamstrings during a maximal voluntary quadriceps isokinetic contraction would be higher in (1) participants who reported prevalent knee joint buckling or shifting at baseline (cross-sectionally) and (2) those who developed incident buckling or shifting at 24-month follow-up (longitudinally).

Methods

Participants

The Multicenter Osteoarthritis Study (MOST), is a cohort study investigating risk factors for knee osteoarthritis in 3026 individuals between the ages of 50–79. Baseline for our study, which took place at the 60-month MOST visit, included 1826 participants who met the eligibility criteria (Fig. 1). Details on selection and subject exclusion have been described previously[32]. In brief, participants were recruited for MOST if they reported factors suggesting either preexisting knee OA (i.e., frequent knee pain) or were at elevated risk for knee OA (i.e., history of knee injury or surgery or BMI>25), while maintaining a distribution of age and sex in proportion to that of the US population. At baseline, age, sex, and history of injury or surgery were assessed by questionnaire and varus malalignment (≥2°) was measured using hip–knee–ankle axis on full-limb radiographs. Body mass index (BMI), Kellgren–Lawrence (KL) grade, and Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC) pain scores were measured at baseline and follow-up clinic

visits[33]. The WOMAC pain scale was used to evaluate participants' knee pain[34,35]. Knee radiographs were obtained at baseline for all participants for assessment of OA using KL grading[36].

Hamstring coactivation

Coactivation of the hamstring muscles was assessed during isokinetic knee extensor strength testing on a Cybex 350 isokinetic dynamometer (CSMi, Stoughton, MA) using a 4-channel Bagnoli surface electromyography (sEMG) system (Delsys, Boston, MA). The isokinetic strength testing protocols have been described in detail previously[37,38]. Briefly, four repetitions of alternating flexion and extension maximal strength efforts were performed at 60°/s. The chair seat and back were placed at 85° and the dynamometer tilt was 0°. Testing began with the knee flexed to near 90°. Three warm up repetitions were performed at 50% of maximal effort. Participants were instructed to fully extend the knee and then pull the leg back to approximately 90° flexion. Following a 5-s rest period, four extension and flexion repetitions were recorded at maximal effort[38]. Initially, sEMG was measured bilaterally, but reductions in clinic time allocated for the examination necessitated unilateral measurements, resulting in a total of 1826 right and 257 left thighs assessed.

Prior to the muscle activation and strength measurements, participants completed a warm-up consisting of 20-m walk and chair stand tests, as described previously[32,39]. Participants were asked to remove their shoes and wore shorts that allowed access to the thigh muscles. While standing, four Delsys 2.1 sEMG sensors (Ag–AgCl bar electrodes separated by 1 cm) were applied to the anterior and posterior thighs (Fig. 2) after being cleaned and slightly abraded with rubbing alcohol pads.

A disposable gel sEMG reference pad was placed over the lateral malleolus of the ankle. Placement of the sensors followed Surface ElectroMyoGraphy for the Non-Invasive Assessment of Muscles (SENIAM) recommendations, although in standing rather than lying down to minimize effort and time delays for participants. Specifically, hamstring sensors were placed mid-way between the ischial tuberosity and the lateral femoral condyle (biceps femoris) or the medial condyle (semitendinosus). The quadriceps sensors were placed along the line from anterior superior iliac spine (ASIS) to the medial collateral ligament (vastus medialis) or the lateral patella (vastus lateralis).

Muscle activation of the quadriceps and hamstrings was collected during the isokinetic strength testing. Signals were collected at 1000 Hz using a 12-bit National Instruments (NI) USB DAQ card, after anti-aliasing 20–450 Hz bandpass filtering (Delsys), using a custom LabView data collection program (NI, Austin, TX) and saved digitally for later offine processing. All EMG signals were post-processed using a 200 ms root mean square (RMS) window using a custom LabView program. The average sEMG amplitude across the duration of each knee extensor contraction was calculated. Each contraction window was defined as 1400 ms and visually applied to the center of the full contraction (1500 ms = 90 ° at 60 °/s). The sEMG was not able to be time locked with the iso-kinetic testing equipment. Coactivation of the hamstrings only during the repetition achieving maximal isokinetic knee extensor torque was used as the variable of interest in these analyses. An example of the raw

and post-processed sEMG signals obtained during isokinetic strength testing is depicted in Fig. 3.

Because of the reciprocal maximal activation of the quadriceps and hamstrings muscles during this strength testing protocol, the agonist and antagonist phases of activation are clearly identifiable. Agonist muscle activation is indicated by the dashed arrows in Fig. 3, occurring when the muscles of interest are acting for torque generation. Antagonist muscle coactivation is indicated by the solid arrows, occurring when the muscles of interest are opposing the primary torque. To compare sEMG signals across individuals, all signals were standardized to their maximal activation when acting as an agonist. This standardization controls for between-subject differences in impedance and enables assessment of each muscle's activation level as a percentage of maximum (%max). Baseline “noise” (not shown) in the sEMG signal is typically small, but if not appropriately accounted for can result in erroneous assessments of muscle activation[40]. To adjust for baseline noise, the square root of the difference of the squares, or the power baseline subtraction method as outlined in Equation (1), was used.

$$\text{Hamstring Antagonist Coactivation} = \sqrt{\left\{(\text{Antagonist Amplitude})^2 - (\text{Mean Baseline Amplitude})^2\right\}} \quad (1)$$

“Antagonist amplitude” is the mean activation of the medial (or lateral) hamstrings during the repetition of maximal knee extensor torque, as a percent of the maximal medial (or lateral) hamstring activation observed when it was acting as an agonist (during flexion contractions)[40,41]. “Baseline amplitude” is the mean baseline amplitude as a percent of the maximal medial (or lateral) hamstring activation obtained during quiet rest. If the “Baseline amplitude” was greater than “Antagonist amplitude”, which can occur with slight decreases in baseline noise and little to no coactivation, then hamstring antagonist coactivation was considered to be zero (as negative coactivation values are non-physiologic). The “combined hamstring” coactivation was calculated as the RMS of the medial and lateral hamstring coactivation levels (see Equation (2)).

$$\text{Combined Hamstring Coactivation} = \sqrt{\frac{\left\{(\text{Medial Hamstring Coactivation})^2 + (\text{Lateral Hamstring Coactivation})^2\right\}}{2}} \quad (2)$$

Outcomes: buckling and shifting

Participants completed a self-administered questionnaire about buckling and shifting history at baseline and 24-month follow-up. For buckling, participants were asked “In the past 3 months, has either of your knees buckled or given way at least once?” Those who answered affirmatively were considered to have experienced knee buckling and asked to provide details about the buckling episodes, including which knee (right and/or left) and the frequency of episodes. To assess whether participants experienced a sensation of shifting that did not involve the knee actually buckling, we also asked all participants, “In the past 3

months has either knee felt like it was shifting, slipping, or going to give way but didn't actually do so?" Those who answered affirmatively were considered to have experienced knee shifting. These questions were repeated at the 24-month follow-up visit and only those participants who reported not having the symptom at baseline, but having the symptom at 24-month follow-up were considered to have developed the incident outcome longitudinally. In addition to determining associations between antagonist hamstring coactivation and each of the outcomes independently, we also assessed relationships with the combined outcome, "either buckling or shifting." The six outcomes studied are summarized in Fig. 4.

Statistical analyses

For qualifying participants, univariate distributions (means and standard deviations or medians and interquartile ranges) were calculated for age, body mass index, medial, lateral and composite hamstring antagonist coactivation. Frequencies were calculated for sex, Kellgren–Lawrence grade, and all variables relating to shifting and buckling and χ^2 analyses were used to compare differences in distributions of categorical variables between men and women. Univariate distributions and frequencies were compared between qualifying participants and non-qualifying participants (as described in Fig. 1). Generalized estimating equations (GEE) were used to determine whether statistically significant associations existed between the three hamstring antagonist coactivation variables (medial, lateral and combined hamstring coactivation) and age, WOMAC knee pain score, KL grade, varus alignment, knee surgery and knee injury history. Analyses were completed with and without adjustment for those variables found to be covariates, to clarify the reasons for associations detected.

Odds ratios (OR) and 95% confidence intervals (CI) were calculated using logistic regression GEE models, controlling for age, sex, KL grade ≥2 and WOMAC knee pain and the interdependence between limbs within participants to address the hypotheses that coactivation level of the hamstrings during an isokinetic knee extensor task is higher: in those who report ipsilateral knee joint shifting, buckling or the combination, (1) cross-sectionally at baseline (the 60-month MOST visit) and (2) longitudinally at 24-month follow-up (the 84-month MOST visit) in those without the symptoms at baseline. The independent variable was tertile of median hamstring antagonist coactivation (medial, lateral or combined) with the lowest tertile being the reference group.

The dichotomous dependent variable was the presence of bucking, shifting, or either buckling or shifting. No statistically significant interactions between sex-specific tertile of coactivation and sex were detected (all *P*-value for interaction term were >0.40), and therefore the interaction term was dropped. A *P*-value for the trend of the associations across coactivation tertiles and the outcomes were calculated. Confirmatory analyses treated the independent variables (coactivation) as continuous and sensitivity analyses were conducted using the outcome of 2 or more episodes within the past 3 months in order to assess consistency of findings with a more robust outcome variable (e.g., repetitive bucklers). Analyses were performed using SAS 9.2 (SAS Inc, Cary, NC).

Results

A total of 2083 limbs (830 knees from 737 men and 1253 from 1089 women) were studied. At baseline, participants' mean ± SD age was 67.4 ± 7.7 years and BMI was 30.7 ± 5.8 kg/m^2. Knees were KL grade 0 or 1 in 55.3% and KL ≥ 2 in 42.8% (KL grade missing for 1.9%). The mean ± SD WOMAC Knee Pain was 2.6 ± 3.1. A history of knee injury was reported by 30.6% and knee surgery by 21.3% of participants. Table I describes the characteristics of the participants by sex. There were no statistically significant differences between knees and participants eligible and the 2577 knees and 504 ineligible participants with regard to sex or KL grade. Table I presents sex-specific cut-offs for tertiles of hamstring coactivation and rates of outcomes. Combined hamstring coactivation levels were not significantly associated with history of surgery (P = 0.408), history of knee injury (P = 0.473) or varus malalignment (P = 0.921). However, higher hamstring coactivation levels were significantly associated with greater age (P = 0.003), female sex (P = <0.001), KL grade≥2 (P = 0.001), and higher WOMAC Pain (P = 0.002).

Prevalent buckling

At baseline, prevalent buckling was not associated with tertiles of coactivation for the medial, lateral, or combined hamstrings (results for combined hamstring coactivation in Table II). The absence of an association persisted whether buckling was defined as one episode or two or more episodes in the past 3 months (data not shown).

Incident buckling

At 24 months following the muscle coactivation measurements, incident buckling was present in 95 of the 1695 limbs (5.6%) that did not have buckling at baseline (overall for all analyses). Report of incident buckling was not associated with tertiles of hamstring coactivation after adjustment for covariates (Table II). However, the middle tertile of medial hamstring coactivation had a lower odds for incident buckling, OR = 0.48 (95% CI: 0.26, 0.92) compared with the lowest tertile of coactivation (P = 0.002).

Prevalent shifting

Report of prevalent knee joint shifting did not vary in a statistically significant manner across tertiles of medial or combined hamstring coactivation at baseline after adjustment for covariates (Table III). While, in the unadjusted analyses, the middle tertile of medial hamstring coactivation had a decreased odds for prevalent shifting (Table IIIb) and the highest tertile of lateral hamstring coactivation had an increased odds of prevalent shifting (Table IIIc), these were no longer statistically significant after adjustment and there was not a statistically significant trend across the tertiles.

Incident shifting

There was a trend towards report of incident knee joint shifting being associated with tertiles of hamstring coactivation after adjusting for covariates. The point estimates for highest tertiles of combined, medial and lateral hamstring coactivation were similarly elevated with respect to the lowest tertile and for lateral coactivation, there was a statistically significant

trend across tertiles for elevated odds of incident shifting with higher coactivation (P = 0.049).

Prevalent buckling or shifting

Overall, report of prevalent buckling or shifting was not associated with combined, medial or lateral hamstring coactivation level after adjustment for covariates (Table IV). While, in the unadjusted analyses, the highest tertile of combined hamstring coactivation had an increased risk for prevalent buckling or shifting in comparison with the lowest tertile, OR = 1.34 (95% CI: 1.01, 1.79) and the middle tertile of medial hamstring coactivation had a decreased risk for buckling or shifting in comparison to the lowest tertile, OR = 0.70 (95% CI: 0.53, 0.93), these findings were no longer statistically significant after adjustment and there were not statistically significant trends across the tertiles.

Incident buckling or shifting

Report of incident buckling or shifting was not associated with combined, medial or lateral hamstring coactivation after adjustment (Table IV). However, in unadjusted analyses, the middle tertile of medial hamstring coactivation was associated with a decreased risk of incident buckling or shifting compared with the lowest tertile of coactivation (Table IVb), OR = 0.65 (95% CI: 0.44, 0.96).

Discussion

This study tested our hypothesis that coactivation level of the hamstrings during an isokinetic knee extensor task is higher in those who reported prevalent knee joint buckling or shifting at baseline and incident knee joint buckling or shifting at 24-month follow-up. In a population with or at risk for knee OA, we found that 7.9% and 15.3% reported prevalent buckling and shifting, respectively at baseline and 5.6% and 11.6% reported incident buckling and shifting, respectively, at follow-up. We did not find a consistent statistically significant association between coactivation and ipsilateral prevalent or incident buckling or shifting. Despite considering buckling in several ways (single or multiple episodes in the past 3 months, both at baseline and at 24-month follow-up, and treating coactivation data as continuous and stratified by tertile), no systematic relationships with coactivation level were detected.

Although there was a reduced odds for incident buckling, shifting or the combination in the middle tertile of coactivation in comparison with both the high and low tertiles of coactivation, this finding did not reach statistical significance. While no linear dose effect is apparent, this consistent U-shaped relationship may suggest that a certain degree of coactivation of the hamstrings is protective against these symptoms and that having excessive or insufficient hamstring coactivation could confer elevated risk for these symptoms. In several cases, after controlling for the presence of knee OA (KL grade ≥2) and WOMAC knee pain, relationships between coactivation and shifting, as well as the combination of shifting and buckling were attenuated (Tables III and IV). This suggests that baseline pain score and the presence of knee OA may contribute to both coactivation as well as to buckling and shifting, confounding the association.

Knee pain is a prominent symptom in individuals with knee OA[5,42,43]. Many individuals with knee OA and knee pain also experience knee instability, including buckling[1,4]. One study by Felson *et al.* found that 14.1% of knees with pain experienced buckling while only 2.1% of knees with no pain experienced buckling[1]. That study also found that the prevalence of buckling increases with the severity of pain. Our study confirms these previous findings regarding pain, while adding information regarding the strength of the associations. An early study of the MOST cohort that assessed potential risk factors for buckling found male sex, knee injury history, and knee pain intensity to be associated with a greater risk for buckling[5]. Hamstring coactivation was found to be associated with female sex and to have no significant association with knee injury history in the current study, consistent with the lack of association between hamstring coactivation and knee buckling. However, knee pain again was closely associated with buckling in the current study of this same cohort 5 years later, even after controlling for age, sex, hamstring coactivation level and the presence of knee OA (KL grade ≥2).

To evaluate the significance of the correlation between coactivation and shifting, it is important to realize that buckling and shifting are related and can co-exist[7]. When a joint begins to shift, people may compensate for the perceived loss of stability by redistributing weight to the contralateral leg, grasping a railing, or sitting. Joints can buckle when compensatory mechanisms are unavailable or when shifting occurs too rapidly for the patient to compensate.

To our knowledge, the current study is the first to examine associations between antagonist hamstring coactivation and buckling. However, buckling has been previously assessed in several other settings. A study of individuals from a community setting found that 12% (278 out of 2351) had experienced at least one episode of buckling in the past 3 months and 13% fell during that episode[1]. We also know that knee buckling plays a significant role in falls in older adults, as a recent study found that the recovery limb during a fall shows a significant amount of knee buckling[44]. These studies have revealed that buckling is common and it is associated with mobility safety and functional limitations. Our study found a very similar prevalence of buckling, while also evaluating a new outcome, shifting.

While we hypothesized that understanding how coactivation affects knee joint buckling or shifting could potentially be useful for designing rehabilitation programs, we did not detect a relationship between coactivation and risk for buckling. The finding that moderate coactivation potentially confers protection against incident knee joint shifting in comparison with lower or higher coactivation levels could be examined in future studies aimed at clarifying levels of coactivation that are beneficial. However, our data do not support that hamstring coactivation during an isokinetic knee extensor task is an indicator of either a compensatory mechanism in response to more frequent buckling or a motor control strategy that precipitates shifting or buckling. In addition, as coactivation levels were measured in MOST only at the 60-month visit, whether buckling and shifting preceded coactivation could not be assessed.

Accordingly, the interpretation of how or whether coactivation strategies should be modified remains unclear. Some researchers have concluded that the increased coactivation in

individuals with knee OA could be a stabilizing mechanism in response to laxity on the medial side of the joint[26]. That same study suggested that the coactivation, though stabilizing, should be altered because it could amplify joint destruction by increasing joint compression. Other investigators have proposed that altering coactivation could destabilize the knee, limiting walking[27]. Furthermore, coactivation could have a negative impact in those with knee joint buckling and shifting. Hamstring coactivation reduces net knee extensor moment, possibly resulting in a lower threshold for the knee to give way, i.e., less quadriceps extensor torque to resist a sudden knee flexion moment. Thus, while coactivation during gait often increases with knee OA severity, it remains unclear whether coactivation adversely contributes to, or is a positive compensation for, joint pathology. To reconcile these issues, it will be necessary to assess the patterns of coactivation over time—determine ranges of coactivation during functional activities in older adults without knee OA as well as levels in those with knee OA that are associated with buckling/shifting episodes and levels that precede worsening of joint morphology.

Buckling and/or shifting were assessed through self-report and participants may not accurately recall events. However, since participants are unlikely to be aware of their coactivation status, inaccurate recall is unlikely to be systematically associated with coactivation and thus unlikely to introduce bias. In addition, recall relies on our older population of participants remembering specific events which may or may not have had memorable outcomes and may have been precipitated by obstacle navigation or changes in surface conditions. Also, coactivation was assessed during an isolated open kinetic chain strength assessment, which allowed normalization to maximal activation and was feasible in this wired sEMG study, but may not translate to coactivation strategies used during functional activities, particularly closed kinetic chain activities. Although evidence of a tendency to use coactivation as a generalized motor control strategy in open kinetic chain tasks has been demonstrated[31], this does not rule out the possibility that coactivation during functional tasks may differ from open kinetic chain coactivation assessment.

Despite limitations, the large sample size permitted useful assessment of potential associations, while providing sufficient statistical power to control for multiple covariates, including age, pain, sex, and KL grade. Few, if any cohorts of this size have ever examined muscle coactivation using objective measures, such as sEMG. While muscle activation was not assessed during walking, it provides some insight into motor control strategies used during an open kinetic chain task in which the knee may be perceived as unstable. In conclusion, patient recall of knee buckling and shifting was not associated with hamstring coactivation during an open kinetic chain quadriceps strength test. This study advanced understanding of the inter-relatedness of pain, knee OA and coactivation strategies and demonstrated the absence of an association between hamstring coactivation and knee buckling or shifting.

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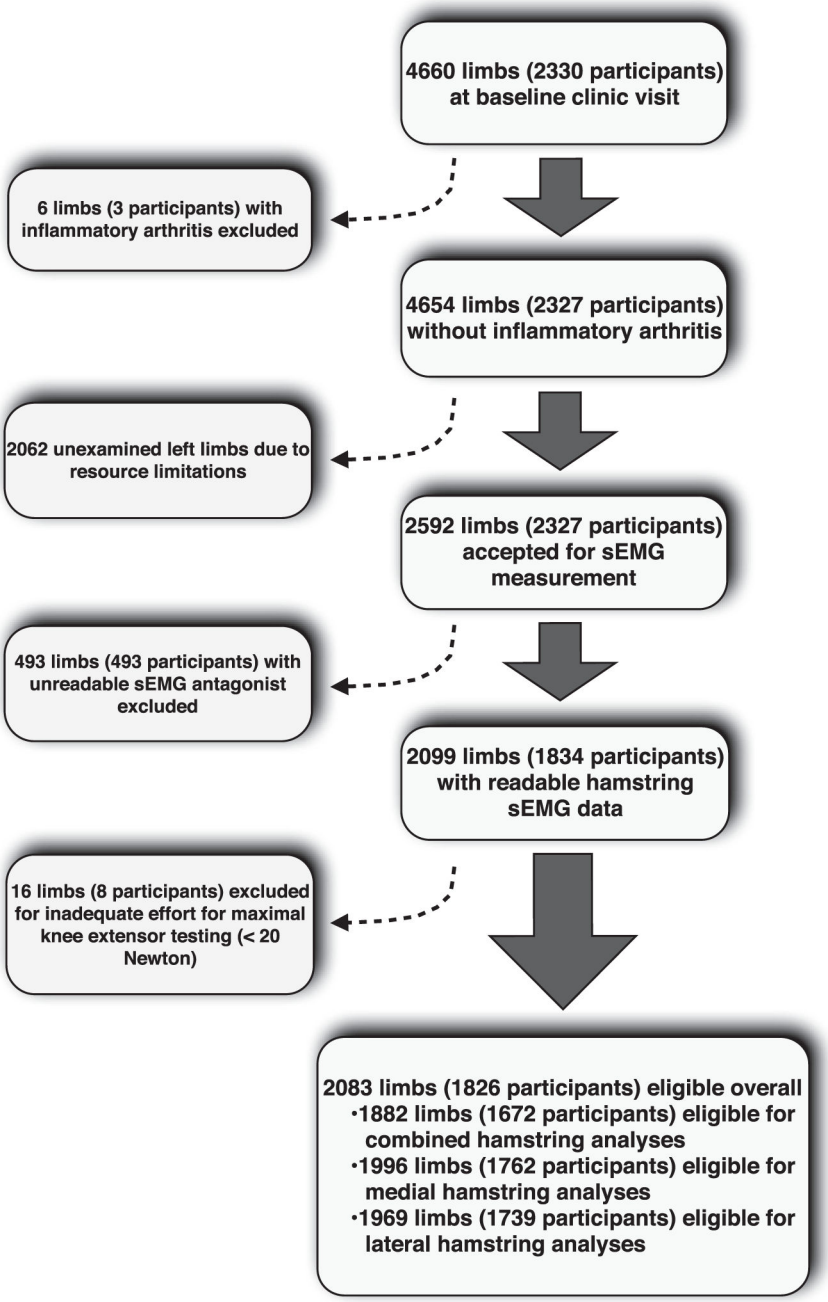

Fig. 1.
Participant enrollment and eligibility criteria.

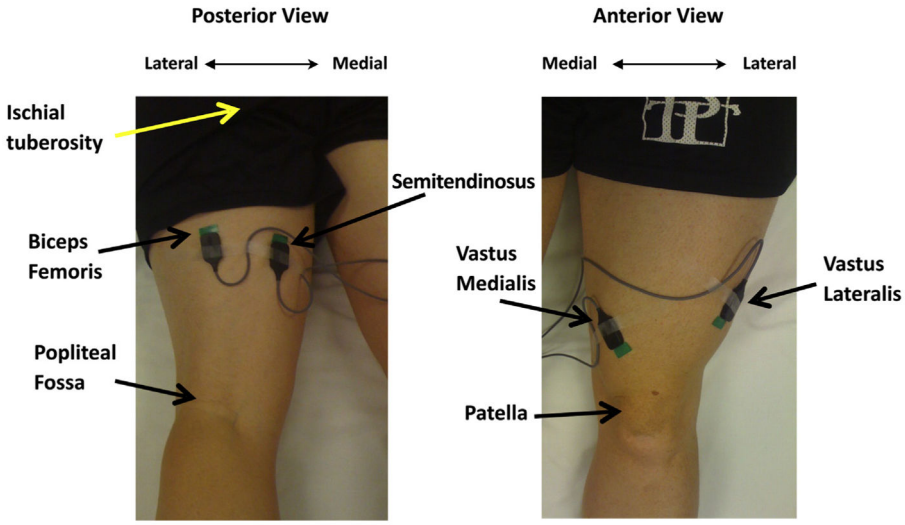

Fig. 2. Placement of sEMG Electrodes

Lateral posterior thigh (biceps femoris): The first sensor was applied halfway down the posterior thigh, between the ischial tuberosity and the lateral femoral condyle, oriented vertically in line with muscle. *Medial posterior thigh (semimembranosis):* The second sensor was applied vertically, mid-way down the posterior thigh, between the ischial tuberosity and the medial condyle. *Lateral anterior thigh (vastus lateralis):* The third sensor was placed on the lateral anterior thigh, approximately one-third of the distance between the lateral patella and the anterior superior iliac spine (ASIS), in line with the vastus medialis muscle. *Medial anterior thigh (vastus medialis):* The fourth sensor was placed on the anterior medial thigh, approximately one-quarter of the distance between the medial collateral ligament and the ASIS. The sensor was placed at an angle of approximately 30° to the line of the femur, in line with the vastus medialis muscle.

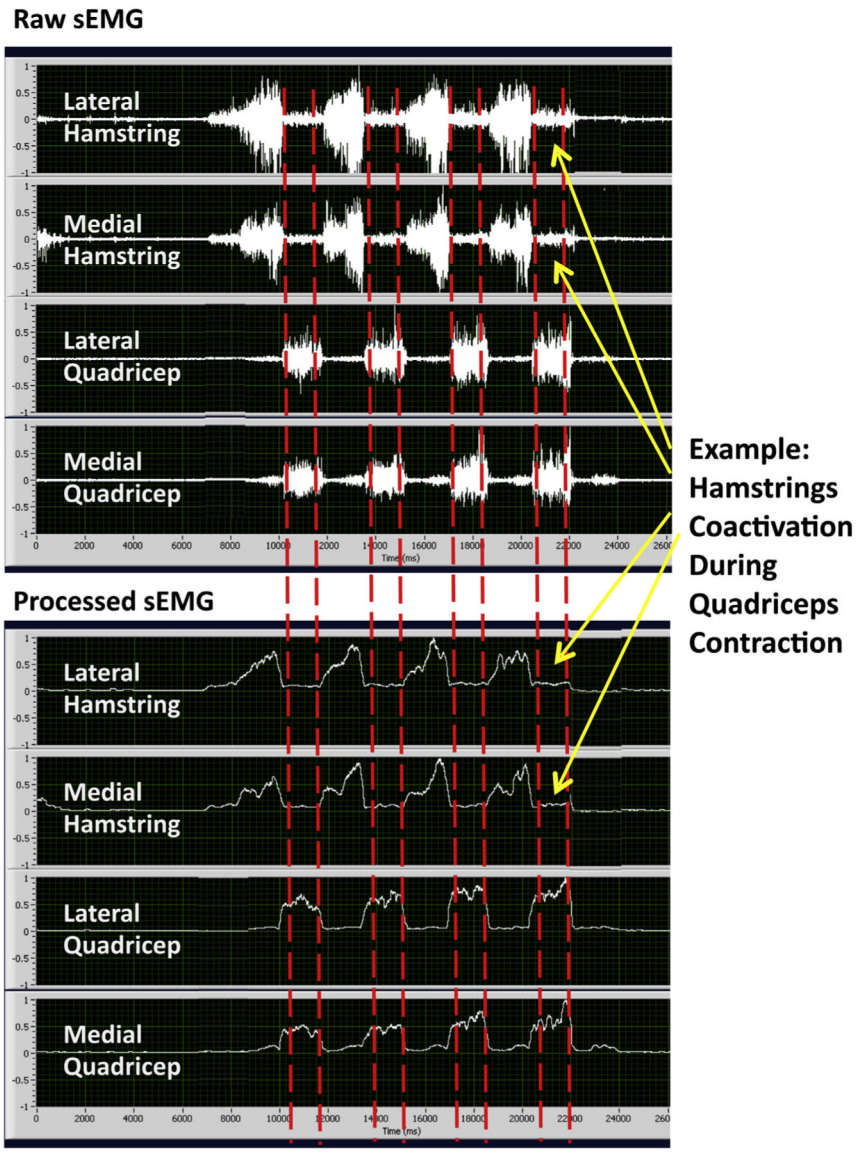

Fig. 3. Example of sEMG Data for Hamstring Coactivation During Extensor Strength Testing

The arrows indicate examples of hamstring muscle coactivation during periods of quadriceps activation.

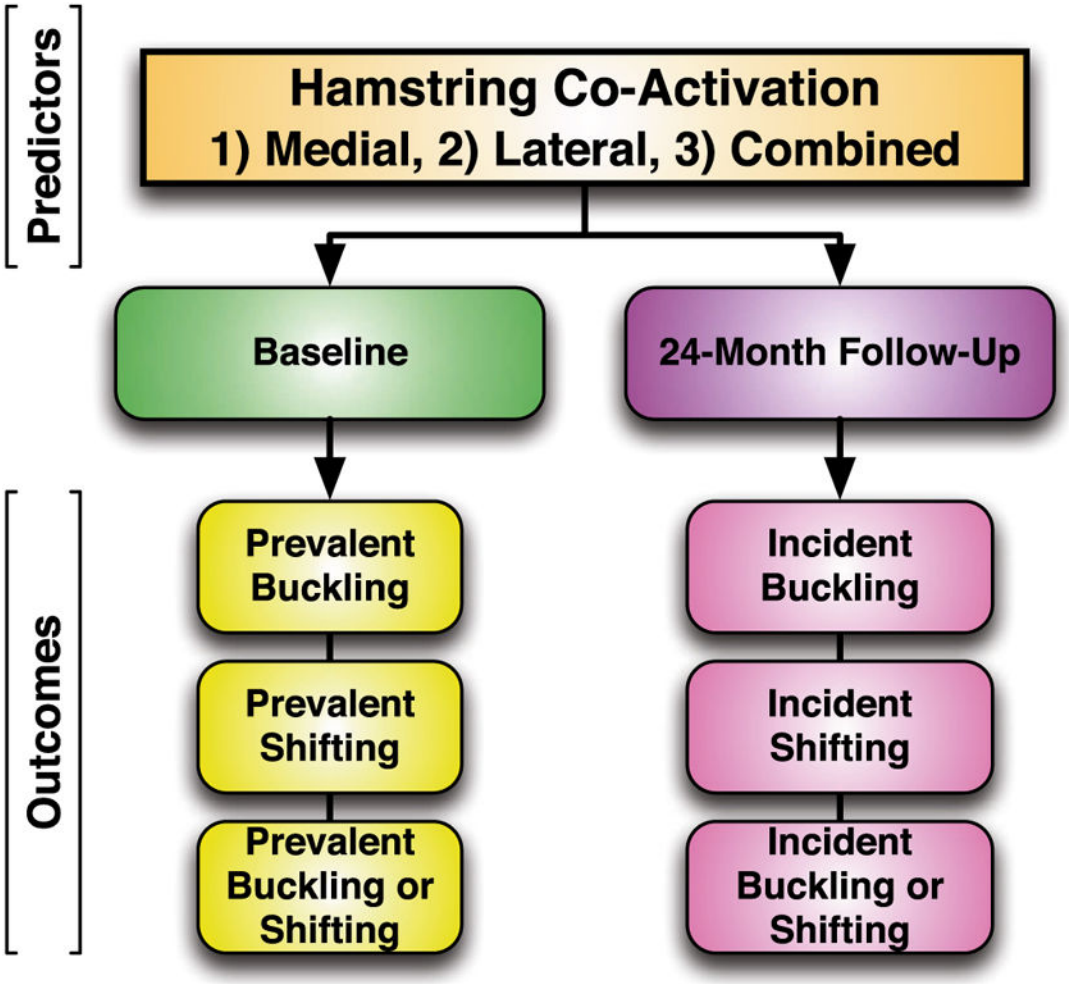

Fig. 4.
Study outcome measurements.

Table I

Baseline coactivation levels for combined, medial, and lateral hamstrings. Sex specific tertiles of median % coactivation for combined, medial, and lateral hamstring coactivation. Prevalence of buckling and shifting at baseline and follow-up

Participant characteristics	
Labels	**All Participants 2083 limbs from 1826 participants**
Baseline mean combined hamstring coactivation	14.4 ± 10.8% {Men, 10.9 ± 9.3%; Women, 16.9 ± 11.1%; $P < 0.001$}
Baseline mean medial hamstring coactivation	8.8 ± 10.3% {Men, 6.4 ± 8.8%; Women, 10.3 ± 10.9%; $P < 0.001$}
Baseline mean lateral hamstring coactivation	17.1 ± 13.8% {Men, 12.6 ± 11.7%; Women, 20.2 ± 14.3%; $P < 0.001$}

	Tertile	**Cut points in men**	**Cut points in women**
Combined hamstring tertiles of coactivation (% coactivation)	1	0.00–6.30%	0–11.44%
	2	6.32–12.22%	11.46–19.26%
	3	12.27–82.80%	19.28–79.80%
Medial hamstring tertiles of coactivation (% coactivation)	1	0.00–0.00%	0.00–4.11%
	2	0.94–7.64%	4.14–12.85%
	3	7.69–92.96%	12.88–99.07%
Lateral hamstring tertiles of coactivation (% coactivation)	1	0.00–6.91%	0.00–13.63%
	2	6.93–14.02%	13.64–23.51%
	3	14.02–99.76%	23.54–99.55%

	Baseline	**Follow-up for those without the symptom at baseline**
Ipsilateral knee buckling at least once in the past 3 months? (%)	7.9% (164/2082)	5.6% (95/1695)
Ipsilateral knee shifted at least once in the past 3 months? (%)	15.3% (318/2082)	11.6% (181/1565)
Ipsilateral knee buckled or shifted at least once in the past 3 months? (%)	19.8% (412/2082)	13.7% (203/1480)

Table IIa

Association between knee buckling and combined hamstring coactivation

	Tertiles of coactivation	Buckling (%)*	Crude OR (95% CI)	Adjusted OR†,‡ (95% CI)
Prevalent buckling at baseline	1 (*n* = 626)	44 (7.0)	1	1
	2 (*n* = 627)	54 (8.6)	1.25 (0.83, 1.88)	1.47 (0.92, 2.35)
	3 (*n* = 628)	52 (8.3)	1.20 (0.80, 1.81)	1.11 (0.68, 1.80)
Incident buckling at 24-month follow-up	1 (*n* = 517)	29 (5.6)	1	1
	2 (*n* = 509)	22 (4.3)	0.76 (0.43, 1.34)	0.70 (0.38, 1.27)
	3 (*n* = 503)	36 (7.2)	1.30 (0.78, 2.15)	1.14 (0.67, 1.96)

*Median antagonist coactivation averaged 12.9 ± 8.9% in participants who reported prevalent buckling and 12.3 ± 10.9% in participants who did not report buckling at baseline.

†Models adjusted for age, sex, TF ROA (KL grade ≥2 vs <2), WOMAC knee pain.

‡Linear trend from adjusted model $P = 0.8367$ for prevalent buckling, $P = 0.4199$ for incident buckling.

Table IIb

Association between knee buckling and medial hamstring coactivation

	Tertiles of coactivation	Buckling (%)*	Crude OR (95% CI)	Adjusted OR†,‡ (95% CI)
Prevalent buckling at baseline	1 (*n* = 706)	67 (9.5)	1	1
	2 (*n* = 623)	46 (7.4)	0.78 (0.52, 1.15)	1.12 (0.71, 1.77)
	3 (*n* = 666)	46 (6.9)	0.74 (0.50, 1.09)	0.97 (0.61, 1.52)
Incident buckling at 24-month follow-up	1 (*n* = 556)	38 (6.8)	1	1
	2 (*n* = 520)	14 (2.7)	0.38 (0.2, 0.7)	0.48 (0.26, 0.92)
	3 (*n* = 549)	41 (7.5)	1.10 (0.7, 1.74)	1.29 (0.79, 2.09)

*Median antagonist coactivation averaged 5.80 ± 8.63% in participants who reported prevalent buckling and 6.83 ± 10.4% in participants who did not report buckling at baseline.

†Models adjusted for age, sex, TF ROA (KL grade ≥2 vs <2), WOMAC knee pain.

‡Linear trend from adjusted model $P = 0.9965$ for prevalent buckling, $P = 0.1345$ for incident buckling.

Table IIc

Association between knee buckling and lateral hamstring coactivation

	Tertiles of coactivation	Buckling (%)*	Crude OR (95% CI)	Adjusted OR†,‡ (95% CI)
Prevalent buckling at baseline	1 (*n* = 655)	47 (7.2)	1	1
	2 (*n* = 656)	57 (8.7)	1.28 (0.85, 1.92)	1.32 (0.84, 2.07)
	3 (*n* = 657)	51 (7.8)	1.12 (0.75, 1.68)	0.87 (0.54, 1.41)
Incident buckling at 24-month follow-up	1 (*n* = 537)	29 (5.4)	1	1
	2 (*n* = 535)	27 (5.1)	0.93 (0.54, 1.60)	0.87 (0.50, 1.53)
	3 (*n* = 527)	33 (6.3)	1.17 (0.70, 1.95)	0.94 (0.54, 1.62)

*Median antagonist coactivation averaged 15.0 ± 12.3% in participants who reported prevalent buckling and 14.6 ± 13.9% in participants who did not report buckling at baseline.

†Models adjusted for age, sex, TF ROA (KL grade ≥2 vs <2), WOMAC knee pain.

‡Linear trend from adjusted model $P = 0.4517$ for prevalent buckling, $P = 0.9483$ for incident buckling.

Table IIIa

Association between knee shifting and combined hamstring coactivation

	Tertiles of coactivation	Shifting (%)	Crude OR (95% CI)	Adjusted OR*,† (95% CI)
Prevalent shifting at baseline	1 (*n* = 626)	84 (13.4)	1	1
	2 (*n* = 627)	97 (15.5)	1.20 (0.88, 1.65)	1.21 (0.85, 1.74)
	3 (*n* = 628)	105 (16.7)	1.31 (0.95, 1.80)	1.28 (0.89, 1.84)
Incident shifting at 24-month follow-up	1 (*n* = 484)	51 (10.5)	1	1
	2 (*n* = 473)	47 (9.9)	0.93 (0.61, 1.40)	1.05 (0.67, 1.64)
	3 (*n* = 454)	70 (15.4)	1.53 (1.03, 2.25)	1.44 (0.93, 2.26)

*Models adjusted for age, sex, TF ROA (KL grade ≥2 vs <2), WOMAC knee pain.

†Linear trend from adjusted model $P = 0.1804$ for prevalent shifting, $P = 0.0845$ for incident shifting.

Table IIIb

Association between knee shifting and medial hamstring coactivation

	Tertiles of coactivation	Shifting (%)	Crude OR (95% CI)	Adjusted OR*,† (95% CI)
Prevalent shifting at baseline	1 (*n* = 706)	117 (16.6)	1	1
	2 (*n* = 623)	76 (12.2)	0.69 (0.50, 0.95)	0.80 (0.56, 1.15)
	3 (*n* = 666)	107 (16.1)	0.96 (0.72, 1.28)	1.18 (0.85, 1.64)
Incident shifting at 24-month follow-up	1 (*n* = 513)	60 (11.7)	1	1
	2 (*n* = 497)	46 (9.3)	0.78 (0.52, 1.16)	0.97 (0.62, 1.51)
	3 (*n* = 493)	69 (14.0)	1.21 (0.84, 1.75)	1.42 (0.93, 2.16)

*Models adjusted for age, sex, TF ROA (KL grade ≥2 vs <2), WOMAC knee pain.

‡Linear trend from adjusted model $P = 0.2467$ for prevalent shifting, $P = 0.0748$ for incident shifting.

Table IIIc

Association between knee shifting and lateral hamstring coactivation

	Tertiles of coactivation	Shifting (%)	Crude OR (95% CI)	Adjusted OR*,† (95% CI)
Prevalent shifting at baseline	1 (*n* = 655)	87 (13.3)	1	1
	2 (*n* = 656)	103 (15.7)	1.24 (0.91, 1.69)	1.08 (0.76, 1.55)
	3 (*n* = 657)	114 (17.4)	1.39 (1.02, 1.89)	1.31 (0.92, 1.86)
Incident shifting at 24-month follow-up	1 (*n* = 505)	50 (9.9)	1	1
	2 (*n* = 498)	51 (10.2)	1.04 (0.69, 1.56)	1.05 (0.67, 1.64)
	3 (*n* = 470)	73 (15.5)	1.67 (1.13, 2.46)	1.53 (0.99, 2.36)

*Models adjusted for age, sex, TF ROA (KL grade ≥2 vs <2), WOMAC knee pain.

†Linear trend from adjusted model $P = 0.1334$ for prevalent shifting, $P = 0.0486$ for incident shifting.

Table IVa

Association between knee buckling or shifting and combined hamstring coactivation

	Tertiles of coactivation	Buckling or shifting (%)	Crude OR (95% CI)	Adjusted OR*,† (95% CI)
Prevalent buckling or shifting at baseline	1 (*n* = 626)	108 (17.25)	1	1
	2 (*n* = 627)	126 (20.10)	1.24 (0.93, 1.64)	1.33 (0.96, 1.86)
	3 (*n* = 628)	136 (21.66)	1.34 (1.01, 1.79)	1.34 (0.96, 1.88)
Incident buckling or shifting at 24-month follow-up	1 (*n* = 461)	62 (13.5)	1	1
	2 (*n* = 447)	49 (11.0)	0.79 (0.53, 1.17)	0.85 (0.56, 1.30)
	3 (*n* = 428)	76 (17.8)	1.38 (0.96, 1.98)	1.29 (0.85, 1.94)

*Models adjusted for age, sex, TF ROA (KL grade ≥2 vs <2), WOMAC knee pain.

†Linear trend from adjusted model $P = 0.1414$ for prevalent buckling or shifting, $P = 0.1614$ for incident buckling or shifting.

Table IVb

Association between knee buckling or shifting and medial hamstring coactivation

	Tertiles of coactivation	Buckling or shifting (%)	Crude OR (95% CI)	Adjusted OR*,† (95% CI)
Prevalent buckling or shifting at baseline	1 (*n* = 706)	154 (21.8)	1	1
	2 (*n* = 623)	102 (16.4)	0.70 (0.53, 0.93)	0.88 (0.64, 1.22)
	3 (*n* = 666)	134 (20.1)	0.91 (0.70, 1.18)	1.14 (0.84, 1.56)
Incident buckling or shifting at 24-month follow-up	1 (*n* = 483)	71 (14.7)	1	1
	2 (*n* = 471)	47 (10.0)	0.65 (0.44, 0.96)	0.79 (0.52, 1.21)
	3 (*n* = 468)	79 (16.9)	1.17 (0.83, 1.65)	1.36 (0.92, 2.02)

*Models adjusted for age, sex, TF ROA (KL grade ≥2 vs <2), WOMAC knee pain.

†Linear trend from adjusted model $P = 0.3677$ for prevalent buckling or shifting, $P = 0.0503$ for incident buckling or shifting.

Table IVc

Association between knee buckling or shifting and lateral hamstring coactivation

	Tertiles of coactivation	Buckling or shifting (%)	Crude OR (95% CI)	Adjusted OR*,† (95% CI)
Prevalent buckling or shifting at baseline	1 (*n* = 655)	116 (17.7)	1	1
	2 (*n* = 656)	134 (20.4)	1.23 (0.93, 1.63)	1.15 (0.83, 1.59)
	3 (*n* = 657)	142 (21.6)	1.31 (0.99, 1.73)	1.20 (0.87, 1.66)
Incident buckling or shifting at 24-month follow-up	1 (*n* = 478)	60 (12.6)	1	1
	2 (*n* = 469)	57 (12.2)	0.97 (0.66, 1.42)	0.98 (0.64, 1.48)
	3 (*n* = 447)	76 (17.0)	1.43 (0.99, 2.06)	1.28 (0.85, 1.92)

*Models adjusted for age, sex, TF ROA (KL grade ≥2 vs <2), WOMAC knee pain.

†Linear trend from adjusted model $P = 0.3485$ for prevalent buckling or shifting, $P = 0.1891$ for incident buckling or shifting.