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Influence of Antagonistic Hamstring Coactivation on Measurement of Quadriceps Strength in Older Adults

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Abstract

Introduction: We have a limited understanding of how antagonist muscle coactivation relates to measurement of strength in both individuals with and without knee osteoarthritis (KOA).

Objective: We sought to determine if hamstring coactivation during a maximal quadriceps activation task attenuates net quadriceps strength.

Design: Cross-sectional cohort analysis was conducted using data from the 60-month visit of the Multicenter Osteoarthritis Study (MOST).

Setting: Laboratory

Participants: A sample of 2328 community-dwelling MOST participants between the ages of 55 and 84 years, with or at elevated risk for KOA, completed the 60-month MOST follow-up visit. Of these, 1666 met inclusion criteria for the current study.

Interventions: Not applicable.

Main Outcome Measure(s): Quadriceps strength; percent combined hamstring coactivation (HC), medial HC, and lateral HC. Quadriceps and hamstring strength were assessed using an isokinetic dynamometer. Surface electromyography was used to assess muscle activation patterns. General linear models, adjusted for age, BMI, Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC), Kellgren-Lawrence (KL) grade and study site, modeled the relationship between antagonist hamstring coactivation and quadriceps strength.

Results: Men had significantly greater quadriceps strength ($p<.001$), history of knee injury ($p<.001$) and surgery ($p=.002$), and greater presence of varus malalignment ($p<.001$). Women had greater pain ($p<.001$) and proportion of KL grade ≥ 2 ($p=.017$). Sex-specific analyses revealed

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combined HC (p=.013) and lateral HC inversely associated with quadriceps strength in women (p=.023), but not in men (combined HC p=.320, lateral HC p=.755). A non-linear association was detected between quadriceps strength and medial HC. Assessment of quartiles of medial HC revealed the third quartile had reduced quadriceps strength when compared to the lowest quartile of coactivation in both men and women.

Conclusions: Hamstring coactivation attenuates measured quadriceps strength in women with or at elevated risk for KOA.

Level II Prospective study

Clinical trial registration number: NCT03033238

Keywords

Electromyography; Knee; Osteoarthritis; Epidemiology

INTRODUCTION:

Knee osteoarthritis (KOA) debilitates millions of people [1,2]. Joint destruction in KOA leads to increasing instability, which leads to higher muscle coactivation [3], the simultaneous contraction of agonist and antagonist muscles surrounding a joint [4]. Antagonist muscle coactivation during agonist contraction may be a stability strategy, both in individuals with knee joint disease and in those without knee joint disease. Ligaments are aided via adjustment of articular surface pressures and preservation of the joint's mechanical impedance [5]. Hence, coactivation may stabilize the knee to prevent falls, for example, after total knee arthroplasty [6]. However, coactivation also may contribute to excessive joint loading [7,8]. Prior work has revealed that thigh muscle coactivation occurs in moderate [9,10] and severe KOA [11]. This supports the hypothesis that individuals with KOA compensate for quadriceps weakness by coactivating hamstrings during movement.

Similarly, quadriceps weakness may be a risk factor for symptomatic [12,13,14,15,16] and progressive KOA and pain [17]. A meta-analysis revealed that individuals with quadriceps muscle weakness have an increased risk of developing radiographic, symptomatic, and self-reported KOA 14 years later [15]. However, the relationship between antagonist coactivation and quadriceps strength remains unclear.

Muscle strengthening may mitigate against KOA symptoms [18]. However, apparent quadriceps muscle weakness could indicate excessive hamstring coactivation. Thus, a more complete understanding of how antagonist muscle coactivation relates to measurement of strength in both individuals with and without KOA would be informative for formulating exercise prescriptions. Therefore, we sought to determine if hamstring coactivation during a maximal quadriceps activation task attenuates the magnitude of measured quadriceps strength in adults with or at risk for KOA.

METHODS:

Participants and Characteristics

This cross-sectional study was conducted with local institutional review board approval and followed US Federal Policy for the Protection of Human Subjects. Individuals were recruited into the Multicenter Osteoarthritis Study (MOST), a prospective longitudinal cohort study of community-dwelling adults between the ages of 50 and 79 years at baseline, with or at elevated risk for KOA (based on any of the following: overweight or obese compared with Framingham Study median weight for their age- and gender-specific group, [19] had a history of knee injury that made it difficult to walk for at least 1 week, or had a previous knee surgery), from April, 2003-April, 2005. Prior to study participation, all participants gave their written informed consent. All measurements took place at the 60-month follow-up visit from April 2009-December 2010. Enrollment in MOST was achieved through community-acquired sampling, as described previously [13,20]. Figure 1 depicts the inclusion and exclusion criteria for this analysis.

Assessments

Body mass index (BMI) was calculated from body mass and body height [20], Varus alignment was measured using hip-knee-ankle axis on full-limb radiographs with malalignment defined as ≥2° [21], Radiographic Kellgren-Lawrence (KL) grades were used for assessing KOA severity [22], Each participant's radiographs were scored by two independent readers (an experienced academically-based musculoskeletal radiologist and rheumatologists experienced in the interpretation of knee radiographs per study reading protocols) according to Kellgren-Lawrence scale [23], Readers were blinded to participant strength and coactivation levels. For cases in which the two readers disagreed on the presence of radiographic tibiofemoral OA, an adjudication panel of 3 experienced readers decided. The Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC) score [24,25] was used to measure pain.

Strength

We measured strength in the right lower limb, except in participants who had right total knee replacement, in which case the left side was measured. Quadriceps and hamstring strength was assessed using a computerized isokinetic dynamometer (Cybex 350, Medway, MA, USA). Briefly, four repetitions of alternating flexion/extension maximal strength efforts were performed at 60°/second. Strength measurements were excluded if the maximal quadriceps strength was measured to be less than 20Nm, given that all participants were independently ambulatory, which would require greater strength than this nominal value. Therefore, measurements this low indicated that participants did not give adequate effort for strength and coactivation testing. Further details of the strength testing protocol and exclusion criteria have been described previously [26,27,28].

Muscle Activation

Quadriceps and hamstring muscle activation levels were measured using a 4-channel sEMG system (Delsys Bagnoli, Boston, MA, USA). Measurements were made during the isokinetic

quadriceps strength test and normalized to the maximal extensor or flexor activation level, for quadriceps and hamstrings, respectively.

The protocol for sEMG followed internationally developed Surface Electromyography for the Non-Invasive Assessment of Muscle (SENIAM) standards [29]. The skin over the vastus medialis, vastus lateralis, semitendinosus and biceps femoris muscle mid-bellies were cleaned with isopropyl alcohol. Surface electrodes (41x20x5mm) were applied to the skin overlaying these muscles, positioned in line with the muscle fibers; thus, 1 cm recording bars were perpendicular to the muscle fibers. Hamstring sensors were placed midway between the ischial tuberosity and the lateral femoral condyle (biceps femoris) or medial condyle (semitendinosus). Sensors were placed in alignment from the anterior superior iliac spine to the medial collateral ligament (vastus medialis) or lateral patella (vastus lateralis). A reference electrode was placed over the bony prominence of the opposite ankle.

Differential bar electrodes had a fixed inter-electrode spacing of 1 cm, and were pre-amplified by a gain of 10. Variable post-amplification gains ranged from 100 to 10,000, and the signal was bandpass filtered between 20–450 Hz. Signals were collected at a sampling frequency of 1000Hz (National Instruments, Austin, TX, USA). Rectified sEMG signals were averaged across 200 ms moving windows throughout the middle 1400 ms of each 1500 ms contraction and standardized to the peak sEMG value obtained for each of the 4 muscles. To adjust for baseline noise, the square root of the difference between the squares of the measured sEMG and a period of sEMG measured at rest was used to assess all sEMG amplitudes as follows:

$$\text{corrected sEMG} = \sqrt{(\text{sEMG amplitude})^2 - (\text{mean baseline amplitude})^2}$$

Further, all sEMG signals were standardized to their maximal activation when acting as an agonist, thereby controlling for between-subject differences in impedance. This standardization allows each muscle's activation to be assessed as a percent of its maximum activation.

The mean hamstring activation during the knee extension strength testing periods was used to determine the hamstring coactivation values, considering medial, lateral, and combined hamstrings muscles. That is, antagonist amplitude equals the mean medial or lateral hamstring activation during repetition of maximal quadriceps torque, as a percent of the maximal medial or lateral hamstring activation during flexion contraction (i.e., when acting as agonist). If baseline amplitude (i.e., resting sEMG noise level) was greater than measured antagonist amplitude, hamstring coactivation was considered to be zero. Combined hamstring coactivation (CHC) was calculated as the root mean square of the medial (MHC) and lateral (LHC) hamstring coactivation levels [21], In all analyses, coactivation for each participant was defined as the median muscle coactivation level for the medial, lateral, and combined hamstrings respectively over the 4 peak strength repetitions. Figure 2 shows an example of the sEMG signals with hamstring coactivation during the strength 4 repetitions.

Statistical Analyses

Sex-specific univariate distributions were calculated for age, BMI, and hamstring coactivation, and frequencies were calculated for KL grade, WOMAC score, history of injury and surgery, and varus malalignment, χ^2 tests were conducted to determine sex differences in categorical variables. Pearson correlation analysis was conducted to determine linear relationships between quadriceps strength and hamstring coactivation. To model the relationship between hamstring coactivation and quadriceps strength, we constructed general linear models (GLM). We performed GLM analysis adjusted for age, sex, BMI, WOMAC scores, KL grade, and clinic site to determine if hamstring coactivation was associated independently with quadriceps strength. We then repeated these analyses stratifying by sex. Due to finding a non-linear association between MHC and strength, we used quartiles of MHC and adjusted for the same set of covariables in all participants and in each sex stratum. Analyses were completed using SAS version 9.4 (SAS, SAS Institute, Cary, NC, USA), with a significance level of $p<.05$.

RESULTS:

A total of 2328 participants completed the 60-month MOST visit, of whom 1666 (n=1579 right knee) met criteria for this study. We excluded 1 participant with non-OA, inflammatory arthritis, 2 participants with missing radiographs/KL grades, 4 participants with missing WOMAC values, and 655 participants with unreadable hamstring coactivation data. Hence, data from 1666 participants (985 female, 681 male) were used in analyses (Figure 1 and Table 1). The racial distribution of the sample was 147 (86.5%) White or Caucasian, 212 (12.3%) Black or African-American, 10 (0.6%) more than one race, 4 (0.2%) other, and 1 (0.06%) each Asian, American Indian or Alaskan Native, and don't know/refused to answer. Of the limbs studied 827 (49.6%) had radiographic KOA. Men had greater prevalence of varus malalignment ($\chi^2=76.1$, $p<.001$), history of knee injury ($\chi^2=21.9$, $p<.001$), and history of knee surgery ($\chi^2=9.5$, $p=.002$). Women had significantly higher WOMAC pain scores ($p<.001$) and greater proportion of KL grade ≥ 2 ($\chi^2=5.8$, $p=.017$). Men and women did not differ in age ($p=.273$) or BMI ($p=.547$).

Table 2 presents hamstring coactivation levels. Women demonstrated significantly greater levels of hamstring coactivation than men ($p< 0.001$) for combined, as well as medial and lateral hamstring coactivation separately.

Pearson correlation coefficients indicated significant inverse relationships between quadriceps strength and CHC ($r= -0.23$, $p<.001$), MHC ($r= -0.12$, $p<.001$), and LHC ($r= -0.22$, $p<.001$). Significant declines in extensor strength related to increasing KL grade ($r= -0.21$, $p<.001$), WOMAC pain ($r= -0.26$, $p<.001$), and age ($r= -0.35$, $p<.001$). However, quadriceps strength was positively correlated with BMI ($r= 0.072$, $p=.003$).

The GLM analyses evaluating relationships between hamstring coactivation and quadriceps strength are presented in Tables 3–5, for combined, only medial, and only lateral hamstring muscles, respectively. Study site was not associated with coactivation in any of the analyses (all $p>.280$). For CHC (Table 3), every 1% increase in median coactivation corresponded to a 0.141 Nm reduction in quadriceps strength across all participants ($p=.026$). However, sex-

specific analyses revealed this relationship occurred primarily in women, with a decline in peak extensor strength of 0.147 Nm per 1% increase in CHC (p=.013), but was not significant in men (p=.320).

After adjustment for covariates, MHC was not linearly related to measured quadriceps strength across all participants (p=.476), in men (p=.431), or in women (p=.805). However, a non-linear association was detected between quadriceps strength and MHC (Table 4) when analyzing coactivation quartiles. In women, the first quartile had 0% coactivation, the second quartile had 0.6–8.1% coactivation, the third quartile had 8.2–14.7% coactivation, and the fourth quartile had >14.7% coactivation. In men, the first quartile had 0% coactivation, the second quartile had 0.9 – 4.8% coactivation, the third quartile had 4.8–9.3% coactivation, and the fourth quartile had >9.3% coactivation. Those in the third quartile of MHC had reduced quadriceps strength compared to the lowest coactivation quartile, in all participants (p=.0003), in women (p=.022), and in men (p=.011).

Finally, for every 1% increase in LHC, measured quadriceps strength was 0.106 Nm lower in women (p=.023), but was not significantly different in men (p=.755) or when considering men and women combined (p=.116) (Table 5).

DISCUSSION:

This study revealed significant inverse associations between hamstring coactivation and measured quadriceps strength, and these associations were driven by the significant association in women. After adjustment for age, BMI, and WOMAC scores, greater CHC remained associated with lower measured quadriceps strength, indicating that net quadriceps torque measured was likely attenuated by antagonist coactivation, particularly in women. Both MHC and LHC were associated with reduced quadriceps strength, but with somewhat different relationships. Whereas LHC showed a significant relationship with quadriceps strength reductions, but only in women, MHC was associated with reduced strength in both men and women, but only in the third quartile (above average coactivation) compared to the first quartile (no coactivation). Together, these data demonstrate that antagonist muscle coactivation and net quadriceps strength are significantly associated in women, but not in men.

A secondary finding was that women had higher CMC, MHC, and LHC than men, consistent with previous studies that also found healthy women and women with KOA have greater hamstring antagonist coactivation than men [21,30,31]. Here, we report an inverse relationship between coactivation and quadriceps strength in women, but not in men. Women also have an elevated risk for cartilage loss [32], which is associated with strength loss [33,34]. Thus, these previous findings coupled with our results suggest the association between lower measured quadriceps strength and cartilage loss may be mediated by higher antagonist hamstring coactivation, particularly in women. This would need to be examined in future studies, as sex differences may be attributable to multiple factors. Morphological, anatomical, structural elasticity, and peripherally- and centrally-mediated pain mechanisms may be inherently different between women and men [30].

Lower levels of quadriceps strength are associated with and predict incident and progressive KOA [13,35,36,37], and may leave the knee joint vulnerable to injury [12]. Specifically, insufficient quadriceps strength leads to contact stress changes which are detrimental to articular cartilage [38] and can increase impulse loading on the knee joint during gait [39,40]. Thus, if damage to articular cartilage and increases in impulse loading are predictive of KOA, quadriceps strengthening could mitigate knee joint deterioration [12]. However, the results from the current study suggest that hamstring coactivation may be important to consider in those with or at risk for KOA, particularly when interpreting measures of quadriceps strength.

Specifically, measurement of net quadriceps torque, the magnitude of quadriceps torque minus concurrent hamstring torque, is frequently used as a measurement of quadriceps strength. Given this understanding, it is important to interpret quadriceps strength in the context of the degree to which hamstring coactivation attenuates net torque measurements, so that true quadriceps weakness can be differentiated from excess hamstring antagonist coactivation in developing therapeutic interventions. Some investigators have suggested that quadriceps strengthening is indicated to correct strength deficits [35,41,42]. However, older adults with or at risk for KOA may have adequate quadriceps strength, yet may have excessive hamstring coactivation that accounts for the lower magnitude of net quadriceps torque measured. In such cases, attention might be better directed towards addressing coordination of neuromuscular activation, rather than focusing on strengthening quadriceps muscles that may already have sufficient strength, but are working against excessive antagonist muscle activity.

Hamstring coactivation stabilizes the knee joint by opposing agonist contraction against a range of joint angular displacements and ligament loading [43]. By counteracting the quadriceps' anterior pull on the tibia and assisting the stabilization action of the ACL, hamstring coactivation likely maintains the distribution of articular contact stress within normal limits while also preventing ACL strain [5,21,43,44]. Therefore, our results are not meant to suggest that hamstring coactivation should be eliminated. In conjunction with strengthening the quadriceps, physiological levels of hamstring coactivation are necessary to mitigate against knee cartilage damage [40].

There were several design elements that strengthen ability to draw meaningful conclusions from this study. The relatively large sample size allowed sex-stratified analyses as well as sensitivity analyses to confirm both the main and sub-group findings. In addition, in this study, we measured sEMG and knee extensor torque bilaterally in the initial 321 participants. Neither the level of coactivation, nor the knee extensor torque significantly differed between limbs and including both lower limbs would have added covariance to analyses without contributing additional useful variance in the data. Therefore, in subsequent participants, these measurements were made in the right lower limb except in participants with a right TKA, in which case we measured the left lower limb. While isokinetic strength testing is not a functional activity performed in daily life, the standardization of joint angle, speed, testing conditions, placement of sEMG leads, and ability to encourage maximal agonist muscle activation strengthened ability to pool participant data and maximize clarity in interpretation of the data.

Study Limitations

The current study findings are generalizable to the population studied (i.e. with risk factors for knee OA, such as overweight, obese, or a history of knee injury or surgery) and the conditions of testing, however, limitations exist due to both design and measurement methods. For example, associations between antagonist coactivation and net quadriceps torque may have been influenced by body position during isokinetic testing. Specifically, testing participants while seated on the sEMG electrodes may have introduced noise during movement, potentially affecting the signal-to-noise characteristics. However, signals were adjusted for baseline noise and all participants were set-up similarly using standardized methodology.

Isokinetic dynamometers have been used in many studies of quadriceps and hamstring torque. This relies on the participant exerting maximal effort to measure maximal quadriceps strength. It is possible some participants provided less than maximal effort due to pain, unfamiliarity with the movement or machine, or fatigue. Additionally, a seated isokinetic quadriceps task is not a functional movement used in daily life. Thus, isokinetic quadriceps torque may be a suboptimal measurement of functional strength, although it allows for isolated strength assessment with reduced risk of compensatory movements and is convenient and reliable for large studies. In contrast, isometric quadriceps testing is an alternative that would require participants to produce maximal voluntary contractions. One coactivation study used isometric testing and showed individuals with radiographic knee OA had significantly lower quadriceps strength, but not hamstring strength, when compared to controls [45]. Further, there were no differences in coactivation between individuals with radiographic knee OA and controls. However, the authors of that study cautioned that their interpretation could change if an isokinetic test was used. Specifically, maximal voluntary contraction depends on the knee flexion angle, so it is possible hamstring coactivation differs between individuals with knee OA and controls at different flexion angles. Therefore, the study authors advocated the use of isokinetic testing as a follow up to their investigation [45]. Other tests aside from isokinetic or isometric exercise, such as walking, standing from a chair or some other frequent physical task, may better model coactivation during usual functional activities, but may also rely on greater compensatory mechanisms, such as hip extension, to avoid quadriceps activation.

Another potential limitation is that 28% of participants had sEMG data that were uninterpretable and therefore could not be included in analyses of coactivation. This analysis is part of a large, multicenter longitudinal study. The sEMG data collected for this analysis was captured from two sites and from more than 2,000 possibly eligible participants. Research nurses, not kinesiology-trained experts, obtained the sEMG data along with hundreds of other variables [13,20]. Nonetheless, no associations were found between missingness and participant characteristics. Antagonist coactivation was measured while seated for efficiency during a multi-hour visit with many other measurements in this study of over 2,000 individuals at two clinical sites. While focused study of coactivation during functional activities could provide different insights, studies using other modalities have produced conflicting results. A recent study [46] contradicted previous work, showing that greater lateral coactivation was associated with greater knee joint damage [47]. Hodges and

colleagues showed that coactivation of the medial muscles may be associated with medial tibial cartilage damage, and coactivation of the lateral muscles may protect against medial tibial cartilage loss [46]. Prolonged lateral muscle coactivation during walking was protective against cartilage volume loss over one year, in contrast to prolonged medial coactivation being associated with increased risk for medial tibial cartilage volume loss over one year. Hence, greater hamstring coactivation medially, but not laterally, may predict in which individuals knee OA will progress. However, it is plausible there is heterogeneity in medial and lateral muscle coactivation in those with and at risk for KOA [46,48].

Finally, we did not obtain subcutaneous fat measurements. Fat content can be higher in women [49], and different fat distributions between the anterior and posterior thigh could bias sEMG interpretation [50]. Fine wire [51]could be useful in reducing differences in signal amplitude between participants with varying amounts of subcutaneous adipose. Because women have greater thigh adipose tissue than men and, in this study, also had 50% greater coactivation than men, it is possible that this coactivation difference was even greater, considering the insulator between the leads and the motor end plates.

Conclusions

Hamstring coactivation was associated with lower measured quadriceps strength in women with or at risk for KOA. Women also demonstrated greater antagonist hamstring coactivation than men. These findings suggest that hamstring coactivation may attenuate measures of quadriceps strength, in a sex-dependent manner. Further research is needed to determine whether net quadriceps torque or hamstring coactivation account for elevated risk for knee joint deterioration, particularly in women.

Disclosures:

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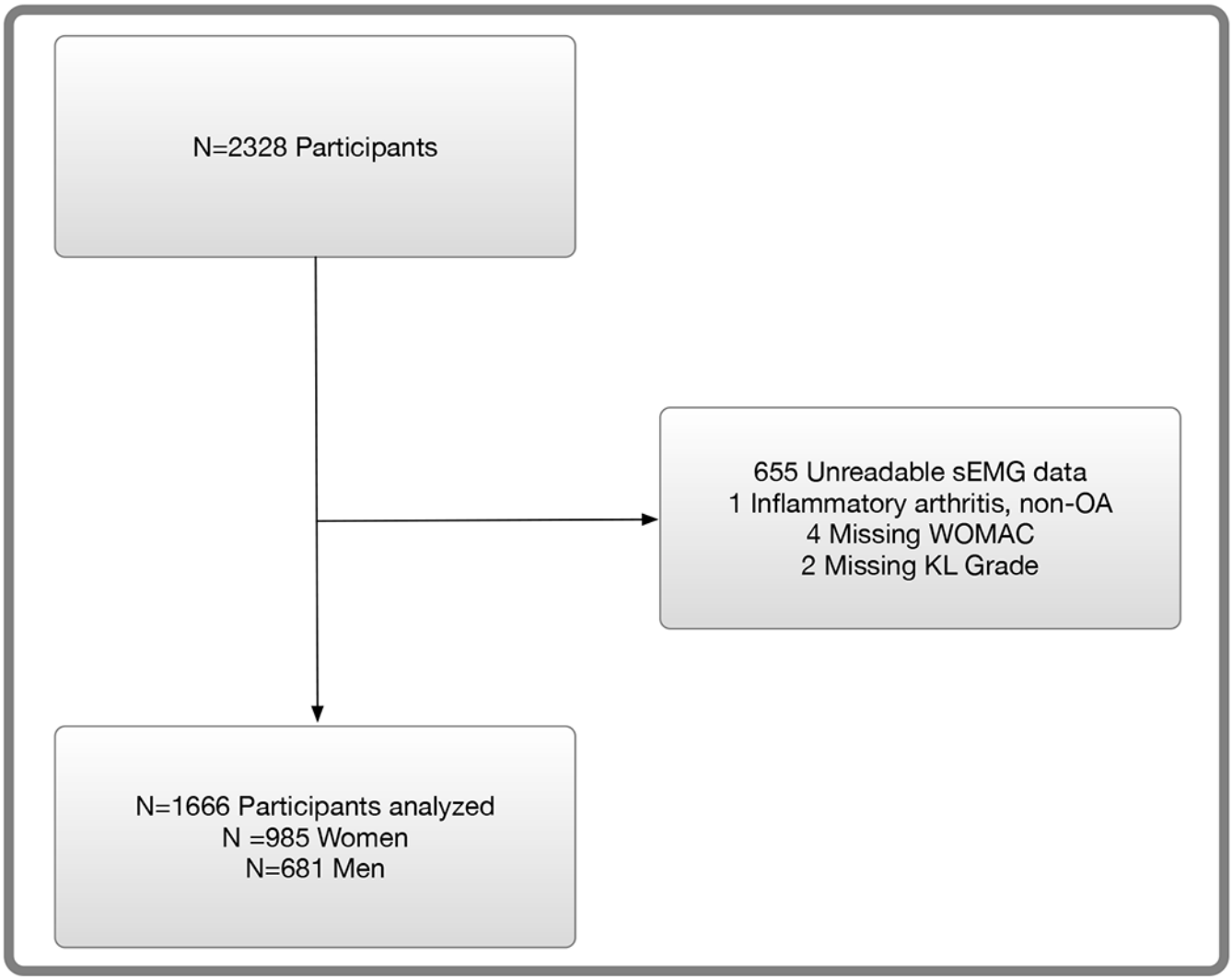

Figure 1.
Flow chart depicting the MOST participants included in and excluded from analyses.

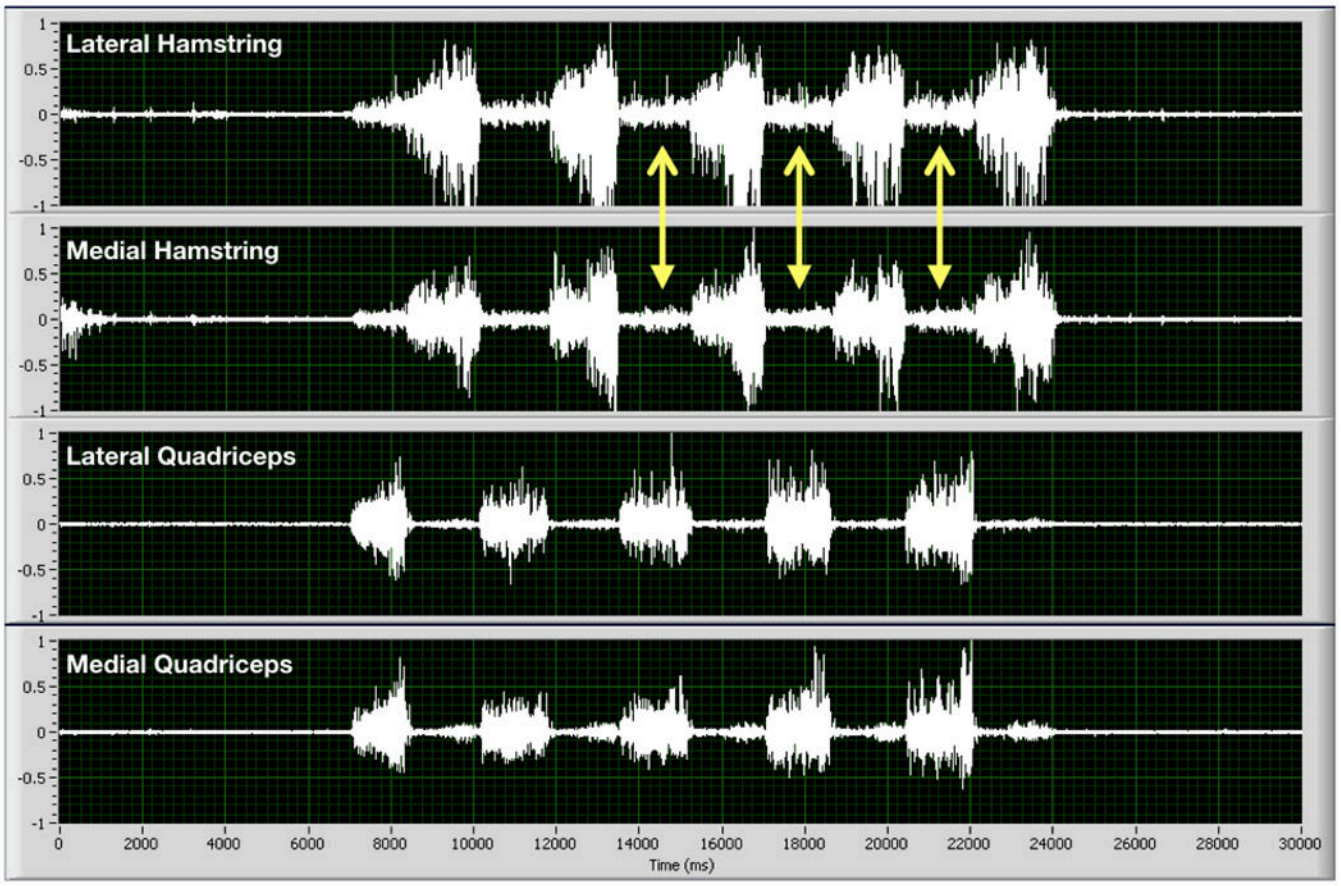

Figure 2.

Flexor activation (blue) during extensor bursts (red). Yellow arrows indicate hamstring coactivation during quadriceps maximal strength testing.

Table 1:

Participant Characteristics

	All Participants (n = 1666)	Men (n = 681)	Women (n = 985)
Age (years)	67.2 ± 7.6	67.0 ± 7.7	67.4 ± 7.5
BMI (kg/m^2)	30.6 ± 5.7	30.7 ± 5.3	30.5 ± 5.9
WOMAC Pain	2.5 ± 3.1	2.1 ± 2.8	2.8 ± 3.2 **
Varus Malalignment, n (%)	763 (46.6)	398 (59.5) **	365 (37.6)
Injury, n (%)	485 (29.1)	241 (35.4) **	244 (24.8)
Surgery, n (%)	263 (15.8)	130 (19.1) *	133 (13.5)
KL Grade ≥ 2, n (%)	827 (49.6)	314 (46.1)	513 (52.1) *
Quadriceps Strength (Nm)	90.4 ± 38.7	119.3 ± 38.1 **	70.4 ± 23.3

Means±SD and counts (percent) are presented. KL=Kellgren-Lawrence. Comparison between women and men:

* =p <.05;

** =p<.001

Table 2:

Hamstring Coactivation Levels

Hamstring Coactivation Level Distributions			
		Mean ± SD%	**p-value**
Combined Hamstrings	**Men**	10.9 ± 9.2	*p<.001*
	Women	16.6 ± 10.9	
Medial Hamstring	**Men**	6.4 ± 8.4	*p<.001*
	Women	9.8 ± 10.5	
Lateral Hamstring	**Men**	12.5 ± 11.7	*p<.001*
	Women	19.8 ± 13.7	

Table 3:

Association between Combined Hamstring Coactivation and Quadriceps Strength

	Covariate	β	Standard Error	R^2	p-value
Total					
	Age	−1.6	0.09		*<0.001*
	BMI	0.43	0.12		*<0.001*
	Sex, female	−45.3	1.4		*<0.001*
	WOMAC	−2.4	0.22		*<0.001*
	KL grade ≥2	−5.3	1.4		*<0.001*
	Combined Coactivation	**−0.14**	**0.06**		***0.026***
	Full Model			**0.54**	***<0.001***
Women					
	Age	−1.2	0.09		*<0.001*
	BMI	0.43	0.11		*<0.001*
	WOMAC	−1.8	0.21		*<0.001*
	KL grade ≥2	−5.3	1.4		*<0.001*
	Combined Coactivation	**−0.15**	**0.06**		***0.013***
	Full Model			**0.27**	***<0.001***
Men					
	Age	−2.1	0.17		*<0.001*
	BMI	0.45	0.25		0.070
	WOMAC	−3.6	0.48		*<0.001*
	KL grade ≥2	−5.2	2.7		0.054
	Combined Coactivation	**−0.14**	**0.14**		***0.320***
	Full Model			**0.27**	***<0.001***

BMI=body mass index; WOMAC=Western Ontario and McMaster Universities Osteoarthritis Index; KL=Kellgren-Lawrence

Table 4:

Association between Medial Hamstring Coactivation Quartiles and Quadriceps Strength

	Covariate	β	Standard Error	R^2	p-value
Total					
	Age	−1.6	0.009		*<0.001*
	BMI	0.43	0.12		*<0.001*
	Sex, female	−46.4	1.3		*<0.001*
	WOMAC	−2.4	0.22		*<0.001*
	KL grade ≥2	−5.4	1.4		*<0.001*
	Medial Coactivation				
	Highest Quartile	**1.5**	**1.8**		**0.408**
	Third Quartile	**6.2**	**1.7**		***<0.001***
	Second Quartile	**2.7**	**2.0**		**0.163**
	Full Model			**0.55**	***<0.001***
Women	Age	−1.2	0.9		*<0.001*
	BMI	0.41	0.11		*<0.001*
	WOMAC	−1.8	0.21		*<0.001*
	KL grade ≥2	−5.4	1.4		*<0.001*
	Medial Coactivation				
	Highest Quartile	**0.86**	**1.8**		**0.635**
	Third Quartile	**4.1**	**1.8**		***0.022***
	Second Quartile	**0.86**	**1.9**		**0.644**
	Full Model			**0.27**	***<0.001***
Men	Age	−2.1	0.17		*<0.001*
	BMI	0.48	0.25		0.052
	WOMAC	−3.5	0.48		*<0.001*
	KL grade ≥2	−5.4	2.7		0.044
	Medial Coactivation				
	Highest Quartile	**2.5**	**3.3**		**0.442**
	Third Quartile	**8.4**	**3.3**		***0.011***
	Second Quartile	**6.5**	**4.2**		**0.118**
	Full Model			**0.28**	***<0.001***

BMI=body mass index; WOMAC=Western Ontario and McMaster Universities Osteoarthritis Index; KL=Kellgren-Lawrence

Table 5:

Association between Lateral Hamstring Coactivation and Quadriceps Strength

	Covariate	β	Standard Error	R^2	p-value
Total					
	Age	−1.6	0.09		*<0.001*
	BMI	0.42	0.12		*<0.001*
	Sex, female	−45.6	1.4		*<0.001*
	WOMAC	−2.4	0.22		*<0.001*
	KL grade ≥2	−5.4	1.4		*<0.001*
	Lateral Coactivation	**−0.079**	**0.05**		**0.116**
	Full Model			**0.54**	***<0.001***
Women					
	Age	−1.2	0.09		*<0.001*
	BMI	0.42	0.11		*<0.001*
	WOMAC	−1.8	0.21		*<0.001*
	KL grade ≥2	−5.4	1.4		*<0.001*
	Lateral Coactivation	**−0.11**	**0.05**		***0.023***
	Full Model			**0.27**	***<0.001***
Men					
	Age	−2.1	0.17		*<0.001*
	BMI	0.43	0.25		.083
	WOMAC	−3.6	0.48		*<0.001*
	KL grade ≥2	−5.3	2.7		0.048
	Lateral Coactivation	**−0.035**	**0.11**		**0.754**
	Full Model			**0.27**	***<.001***

BMI=body mass index; WOMAC=Western Ontario and McMaster Universities Osteoarthritis Index; KL=Kellgren-Lawrence