Effect of Knee Joint Angle and Contraction Intensity on Hamstrings Coactivation

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[1]*School of Public Health, Physiotherapy and Sports Science, University College Dublin, Dublin, IRELAND;* [2]*Institute for Sport and Health, University College Dublin, Dublin, IRELAND; and* [3]*School of Electrical, Electronic Communications Engineering, University College Dublin, Dublin, IRELAND*

ABSTRACT

WU, R., E. DELAHUNT, M. DITROILO, M. M. LOWERY, and G. DE VITO. Effect of Knee Joint Angle and Contraction Intensity on Hamstrings Coactivation. *Med. Sci. Sports Exerc.*, Vol. 49, No. 8, pp. 1668–1676, 2017. **Purpose**: This study investigated the effect of knee joint angle and contraction intensity on the coactivation of the hamstring muscles (when acting as antagonists to the quadriceps) in young and older individuals of both sexes. **Methods**: A total of 25 young (24 ± 2.6 yr) and 26 older (70 ± 2.5 yr) healthy men and women participated. Maximal voluntary isometric contraction of the knee extensors and flexors was assessed at two knee joint angles (90° and 60°, 0° = full extension). At each angle, participants performed submaximal contractions of the knee extensors (20%, 50%, and 80% maximal voluntary isometric contraction), whereas surface EMG was simultaneously acquired from the vastus lateralis and biceps femoris muscles to assess the level (EMG root-mean-square) of agonist activation and antagonist coactivation. Subcutaneous adipose tissue in the areas corresponding to surface EMG electrode placements was measured via ultrasonography. **Results**: The contractions performed at 90° knee flexion demonstrated higher levels of antagonist coactivation (all $P < 0.01$) and agonist activation (all $P < 0.01$) as a function of contraction intensity compared with the 60° knee flexion. Furthermore, after controlling for subcutaneous adipose tissue, older participants exhibited a higher level of antagonist coactivation at 60° knee flexion compared with young participants ($P < 0.05$). **Conclusions**: The results of the present study suggest that 1) the antagonist coactivation is dependent on knee joint angle and contraction intensity and 2) subcutaneous adipose tissue may affect the measured coactivation level likely because of a cross-talk effect. Antagonist coactivation may play a protective role in stabilizing the knee joint and maintaining constant motor output. **Key Words:** AGING, SEX, KNEE JOINT STABILITY, SUBCUTANEOUS ADIPOSE TISSUE, SURFACE EMG, MUSCLE ACTIVATION

Accurate coordination of agonist and antagonist muscles is essential for the efficient execution of human movements (3). Indeed, most voluntary muscle contractions involve a pattern of activation of agonist muscles with concurrent inhibition of antagonist muscles (i.e., reciprocal innervation), which is an important reflex mechanism underlying neuromuscular control (33). Under specific circumstances, such as during voluntary isometric contraction, high velocity limb displacement, and precise motor control, a synchronized cocontraction of agonist and antagonist muscles (i.e., coactivation) is required to ensure joint stability (34).

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This coactivation mechanism is integral to the maintenance of knee joint stability during locomotion and other activities of daily living (3,16,37). This process is likely modulated through proprioceptive pathways originating in the ligamentous and musculotendinous units of the knee joint (3). Such proprioceptive pathways, at least for the knee joint musculature, play a protective role in stabilizing the joint by regulating the neural drive to agonist and antagonist muscles (3,37). It has been proposed that during voluntary contraction of the knee extensors (KE), the hamstrings (i.e., antagonist muscles) could provide opposing mechanical contraction of the agonist muscles in response to variations in joint angular displacement and ligamentous loading, thus maintaining joint stability, equalizing pressure distribution over the articular surfaces, and preventing ligamentous damage (3,13,32,37).

It is logical to propose that the level of coactivation of antagonist muscles (i.e., knee flexors [KF]) could depend on the magnitude of joint angular displacement (i.e., joint angle) and the intensity of contraction of the agonist muscles. Previous studies conducted on young individuals, observed that the level of antagonist coactivation was higher at a more flexed knee joint angle than at a more extended angle (21,23) and increased as a function of the torque generated by the KE muscles (2,22). Aging may also influence the

level of antagonist coactivation with several studies reporting that older individuals attempt to generate maximal effort by adopting a strategy of enhanced antagonist coactivation level when compared with their younger counterparts (15,25). By contrast, other studies have observed that the level of coactivation remain unchanged with aging (5,40). Similarly, it has been illustrated that after a resistance-training program, older individuals either exhibit a reduction (11) or remain constant (31) in the level of antagonist coactivation around the knee joint. There remains a need to clarify the age-related adaptation in motor strategy in response to changes in knee joint angle and contraction intensity. A clearer understanding of these strategies may assist in the design of exercise interventions to counteract age-related deficiencies in motor control.

The assessment of the magnitude of antagonist coactivation is typically achieved by expressing surface EMG (EMG amplitude) activity in the antagonist muscle as a percentage of its maximal activity when acting as an agonist (8,16). However, an inherent limitation associated with surface EMG amplitude measurement is the possible "cross-talk" contamination originating from the adjacent muscles (16,39). Moreover, it is well known that the amount of cross-talk is influenced by subcutaneous adipose tissue covering the muscle under consideration (36,39). Indeed, several simulation and experimental studies have suggested that higher amounts of subcutaneous adipose tissue are associated with greater cross-talk contamination in surface EMG recordings (9,24,36). In this regard, it is logical to suspect that surface EMG recordings could be disproportionately affected if the amount of subcutaneous adipose tissue is not duly considered when assessing the level of antagonist coactivation in different populations. In the present study, therefore, we aimed to quantify the effect of age, knee joint angle, and contraction intensity on the coactivation level of the hamstring muscles (acting as antagonists at varying levels of isometric contraction of the KE), with the amount of subcutaneous adipose tissue taken into account. We hypothesized that 1) the antagonist coactivation (hamstrings) on the knee joint would be dependent on both joint angular position and contraction intensity, 2) older individuals would be characterized by a higher level of antagonist coactivation than the young, and 3) the amount of subcutaneous adipose tissue would affect the observed differences between young and older men and women, whereby thicker subcutaneous adipose tissue would correspond to a higher level of antagonist coactivation.

METHODS

Participants. A total of 25 young (12 men and 13 women) and 26 older (13 men and 13 women) healthy individuals participated voluntarily. Young participants were characterized by a lower level of BMI than older participants ($P < 0.05$). The age and main physical characteristics of the participants are detailed in Table 1. All participants were evaluated as being "medically stable" as per the criteria proposed by Greig et al. (10). None of the participants had performed strength training or engaged in competitive sports within the last 5 yr. All participants provided written informed consent, and the study was approved by the University College Dublin Human Research Ethics Committee.

TABLE 1. Participants characteristics and subcutaneous adipose tissue thickness.

	Young Men	Young Women	Older Men	Older Women
	n = 12	*n* = 13	*n* = 13	*n* = 13
Age (yr)	24.6 ± 2.7	23.4 ± 2.4	70.1 ± 1.8	69.5 ± 3.2
Stature (cm)	182.6 ± 6.6	165.5 ± 5.7	178.2 ± 4.5	162.5 ± 5.5
Body mass (kg)	77.7 ± 16.0	62.9 ± 10.2	82.6 ± 10.8	64.6 ± 8.0
BMI ($kg \cdot m^{-2}$)	23.1 ± 3.7	23.0 ± 3.8	25.6 ± 2.9	24.5 ± 3.0
Subcutaneous adipose tissue thickness				
Vastus lateralis (mm)	7.6 ± 2.4	12.9 ± 3.5	8.2 ± 2.8	12.0 ± 4.1
Biceps femoris (mm)	10.1 ± 4.1	15.7 ± 4.3	7.8 ± 1.7	14.5 ± 4.0

Values are presented as mean ± SD; *n* = number of participants.

Experimental design. Participants attended two laboratory testing sessions separated by at least 3 d. On each visit, participants performed a series of maximal and submaximal isometric contractions on an isokinetic dynamometer at one of two different predetermined knee joint angles (90° or 60°, where 0° represents full knee joint extension). The order of joint angular position was randomized. Each test session began with a 5-min warm-up performed on a cycle ergometer at a work rate of 50–100 W. Participants then performed 1) maximal voluntary isometric contraction (MVIC) of the KE, 2) MVIC of the KF, and 3) three submaximal isometric contractions of the KE corresponding to 20%, 50%, and 80% of predetermined knee extension MVIC, in a randomized order. Surface EMG signals were recorded, simultaneously, from the vastus lateralis (VL) and biceps femoris (BF) muscles during the aforementioned measurements. In addition, subcutaneous adipose tissue thickness of the VL and the BF muscles covering the area where the surface EMG electrodes were attached was obtained (via ultrasonography) during the first laboratory test session to investigate its effect on surface EMG amplitude.

Subcutaneous adipose tissue thickness. A real-time B-mode ultrasound imaging unit (DP-6600; Mindray Bio-Medical Electronics Co., Ltd., ShenZhen, China) with a 40-mm, 7.5-MHz linear-array probe was used to measure subcutaneous adipose tissue covering the VL and the BF muscles. The measurements were taken with the participant in a supine and prone position, respectively, with the knee joint fully extended and muscles relaxed. Images were taken at the sites corresponding to the position of the VL and the BF surface EMG electrodes. The mean value of the two best quality images from each muscle was used for further analysis. Subcutaneous adipose tissue thickness was defined as the distance between the skin surface and the muscle belly (29) and was determined using computer software (AutoCAD 2015; Autodesk, Inc., San Rafael, CA) (Fig. 1).

Maximal and submaximal isometric contraction. After completion of the warm-up, an isokinetic dynamometer (Biodex Medical System, Inc., Goleta, CA) was used to perform the MVIC of the KE and the KF for each participant's right leg. Participants were seated comfortably on the

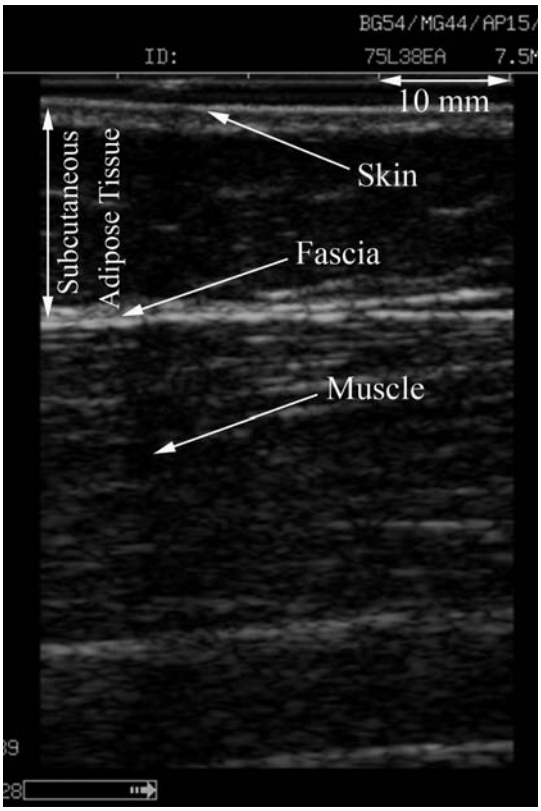

FIGURE 1—Ultrasound image of subcutaneous adipose tissue thickness covering the area where the surface EMG electrodes were attached on BF.

dynamometer chair and firmly strapped at the thigh, pelvis, and torso with the hip joint flexed to 80° (where 0° represents full hip joint extension) and knee joint flexed to 90° or 60° (the adopted knee joint angle for each laboratory test session was randomized). The rotational axis of the dynamometer was aligned to the lateral femoral condyle, and the lower leg was strapped to the machine lever arm 2 cm superior to the lateral malleolus. After familiarization with the testing procedures, each participant performed a minimum of three maximal attempts for knee extension and flexion, respectively, with a 3-min rest between each attempt. Participants were required to exert force "as strongly and as quickly as possible" for 3 s, whereas online visual feedback and strong verbal encouragement were provided to facilitate maximal effort. An additional attempt was allowed when torque variation was higher than 5% between the best two attempts. After correction for gravity, the greatest torque attained by each participant for knee extension and flexion was used for future analysis. The participants then performed the three submaximal isometric contractions at the predetermined target intensities (i.e., 20%, 50%, and 80% MVIC of the KE, respectively). The order of contraction intensity was again randomized to minimize the potential effect of skill acquisition. Participants were instructed to match a horizontal target torque band (bandwidth = ±5% target torque) as displayed on the computer monitor and were verbally encouraged to sustain the torque output "as steady as possible" for approximately 7 s at each contraction intensity. A rest period of either 5 min (after 20% or 50% MVIC) or 10 min (after 80% MVIC) was followed to avoid accumulation of fatigue.

Surface EMG. Surface EMG was recorded during the assessment of maximal and submaximal contractions from the VL and the long head of the BF muscles. These were used as representative of the EMG activation of the KE and KF muscle groups, respectively (8). The skin was first shaved, lightly abraded, and cleaned with ethyl alcohol before two Ag/AgCl bipolar electrodes (Blue Sensor N-00-S; Ambu Mdeicotest A/S, Ølstykke, Denmark) were placed with a 20-mm interelectrode distance according to the SENIAM guidelines (12). An additional ground electrode was placed over the skin of the patella. Transparent films were used to record the position of the electrodes relative to anatomical reference points (i.e., border of patella) and skin marks (i.e., freckles or scars). This allowed replication of electrode positioning during the second laboratory test session.

Data acquisition and analysis. Raw EMG (sampling frequency = 2048 Hz) and torque (sampling frequency = 512 Hz) signals were collected synchronously, amplified with a gain of 1000, and stored on a PC using a 16-bit A/D converter data acquisition system (Biopac System, Inc.) for postprocessing.

All collected signals were processed using custom written programs (MATLAB R2014a; Mathworks, Natick, MA). Torque signals were offline low-pass filtered with a cutoff frequency of 15 Hz using a zero-phase, fourth-order, Butterworth filter. MVIC values of the KE and the KF were established as the greatest 1-s average reached within any single torque recording. Surface EMG signals were band-pass filtered between 10 and 500 Hz (zero-phase, fourth-order, Butterworth filter). To quantify the surface EMG amplitude at maximal and submaximal isometric contractions, the root-mean-square (RMS) amplitude was estimated over a 1-s epoch corresponding to the MVIC of the highest torque attained and over the middle 3 s of each submaximal contraction to avoid the transient phase. For the level of hamstrings antagonist coactivation, the BF RMS (EMG amplitude) at each contraction intensity was normalized with respect to BF RMS obtained during the MVIC of the KF at the same joint angle (8). For the level of quadriceps agonist activation, the VL RMS (EMG amplitude) at each contraction intensity was normalized with respect to the VL RMS obtained during the MVIC of the KE at the same joint angle.

Statistical analysis. Statistical analysis was performed using SPSS 20.0 (IBM Ireland Ltd., Dublin, Ireland). A repeated-measures ANOVA on knee joint angle was performed to compare the dependent variable of the MVIC of the KF. A four-factor repeated-measures ANOVA (age × sex × joint angle × contraction intensity) was performed on KE isometric torque, EMG coactivation of antagonist, and activation of agonist muscles during isometric contractions of the KE. A Greenhouse–Geisser correction was used when the assumption of sphericity was violated. When a significant interaction

was detected, pairwise comparisons were performed with Fisher's least significant difference. In addition, the association between subcutaneous adipose tissue thickness and EMG activation was evaluated via Pearson's correlation. When a significant correlation was revealed, a repeated-measures ANCOVA was performed adopting subcutaneous adipose tissue as a covariate. Effect size was evaluated using eta-squared (η^2) and interpreted as small (0.01), moderate (0.06), and large (0.14) (7). Statistical significance was set at $P < 0.05$.

RESULTS

Muscle strength for knee extension and flexion. Young participants exhibited higher KE torque values compared with older participants ($F_{1,47} = 28.0$, $\eta^2 = 0.37$, $P < 0.001$); in addition, men were characterized by higher KE torque values compared with women ($F_{1,47} = 41.8$, $\eta^2 = 0.47$, $P < 0.001$). A significant main effect was observed for joint angle, whereby larger torque values were recorded at 60° than at 90° of knee flexion ($F_{1,47} = 4.8$, $\eta^2 = 0.09$, $P < 0.05$) (Table 2). Submaximal voluntary contraction torque values were also normalized to the MVIC of the KE performed at the same angle. Supplemental Digital Content 1 (http://links.lww.com/MSS/A893) depicts the % MVIC values achieved at each contraction intensity (i.e., 20%, 50%, and 80% MVIC) for both young and older men and women at 90° and 60° of knee flexion, respectively (see Figure, Supplemental Digital Content 1, http://links.lww.com/MSS/A893). Regarding the MVIC of the KF, young men and women had larger torque values than older men and women ($F_{1,47} = 4.8$, $\eta^2 = 0.09$, $P < 0.01$), and higher KF torque values were produced at 60° than 90° of knee flexion for both sexes ($F_{1,47} = 5.8$, $\eta^2 = 0.11$, $P < 0.05$) (Table 2).

Antagonist coactivation. When the effect of subcutaneous adipose tissue is not accounted for in the statistical analysis, a significant main effect for joint angle ($F_{1,47} = 40.7$, $\eta^2 = 0.46$, $P < 0.001$) was observed, as well as a significant joint angle by contraction intensity interaction ($F_{2.3,106.0} = 14.5$, $\eta^2 = 0.24$, $P < 0.001$). A higher level of antagonist coactivation was found at 90° compared with 60° of knee flexion at all contraction intensities regardless of age (all $P < 0.001$) (Fig. 2A). Moreover, a higher level of coactivation was also observed at 90° compared with 60° of knee flexion for both young ($P < 0.001$) and older ($P < 0.01$) participants, with no difference between young and older participants at each angle (Fig. 2B). In addition, a sex–contraction intensity interaction ($F_{1.6,72.6} = 14.2$, $\eta^2 = 0.23$, $P < 0.001$) was observed. Women had a greater coactivation level than men (all $P < 0.01$), which increased at a greater proportion to increasing contraction intensity than in men (all $P < 0.001$) (Fig. 3A).

Subcutaneous adipose tissue. A main effect of sex was observed for subcutaneous adipose tissue thickness of the VL ($F_{1,47} = 5.9$, $\eta^2 = 0.34$, $P < 0.001$) and the BF ($F_{1,47} = 5.9$, $\eta^2 = 0.43$, $P < 0.001$), respectively, indicating that women had a greater subcutaneous adipose tissue thickness for both VL (36.5%, 12.5 ± 3.8 vs 7.9 ± 2.6 mm) and BF (40.8%, 15.1 ± 4.1 vs 8.9 ± 3.3 mm) when compared with men (Table 1). Moreover, subcutaneous adipose tissue thickness (covering the area where the surface EMG electrodes were attached on BF) was correlated with the level of antagonist coactivation (i.e., BF) for all contraction intensities and both measured joint angles (i.e., 90° and 60° of knee flexion) (all $P < 0.05$) (Fig. 4). On the other hand, no correlation was observed between subcutaneous adipose tissue thickness of the VL and agonist activation. Therefore, an ANCOVA analysis was performed on antagonist coactivation adopting subcutaneous adipose tissue thickness of the BF as a covariate ($F_{1,46} = 7.8$, $\eta^2 = 0.15$, $P < 0.01$). When subcutaneous adipose tissue thickness was included in the analysis, the previously observed main effect for sex disappeared, indicating that the level of BF coactivation did not differ between men and women across the contraction intensities (Fig. 3B). Furthermore, the ANCOVA analysis showed that coactivation level was higher in the older participants but only at 60° of knee flexion ($F_{1,46} = 4.2$, $\eta^2 = 0.08$, $P < 0.05$) (Fig. 2C).

Agonist activation. An interaction between contraction intensity and joint angle ($F_{1.4,65.8} = 21.9$, $\eta^2 = 0.32$, $P < 0.001$) indicated that 90° of knee flexion was characterized by a higher magnitude of VL activation compared with 60°, which was also proportionate to contraction intensity (all $P < 0.001$) (Fig. 2D). Moreover, an age–joint angle interaction ($F_{1,47} = 8.5$, $\eta^2 = 0.15$, $P < 0.01$) showed that a more pronounced difference between joint angles (90° and 60° of knee flexion) was observed in young ($F_{1,47} = 62.2$, $\eta^2 = 0.57$,

TABLE 2. Muscle strength for knee extension and flexion.

	Joint Angle	Young Men	Young Women	Older Men	Older Women
20% MVIC of the KE (N·m)	60°	52.1 ± 14.7	34.9 ± 6.6	35.0 ± 8.3	20.3 ± 5.4
	90°	45.9 ± 14.0	30.8 ± 9.7	33.8 ± 9.2	21.6 ± 4.7
50% MVIC of the KE (N·m)	60°	125.6 ± 31.3	81.6 ± 12.9	85.2 ± 17.4	50.5 ± 12.3
	90°	112.8 ± 34.5	74.6 ± 19.8	81.3 ± 23.3	53.5 ± 12.0
80% MVIC of the KE (N·m)	60°	200.5 ± 50.1	128.1 ± 22.8	135.4 ± 28.3	81.0 ± 19.0
	90°	181.9 ± 56.1	118.1 ± 33.4	131.9 ± 37.3	86.1 ± 21.0
100% MVIC of the KE (N·m)	60°	251.4 ± 60.2	156.2 ± 30.7	173.8 ± 38.3	104.9 ± 23.7
	90°	233.6 ± 66.9	150.2 ± 39.0	166.3 ± 45.6	108.2 ± 23.4
100% MVIC of the KF (N·m)	60°	132.3 ± 39.9	77.0 ± 11.3	84.3 ± 19.5	55.3 ± 9.0
	90°	107.1 ± 36.7	62.9 ± 15.8	62.8 ± 17.8	43.7 ± 8.5

Muscle strength values are presented as mean ± SD. Statistical analysis revealed that for muscle strength of the KE, young participants had greater values than older participants ($P < 0.001$), men had greater values than women ($P < 0.001$), and 60° exhibited greater values than 90° of knee flexion ($P < 0.05$). For the KF MVIC, young participants had greater values than older participants ($P < 0.01$) and 60° exhibited greater values than 90° of knee flexion ($P < 0.05$) for men and women.

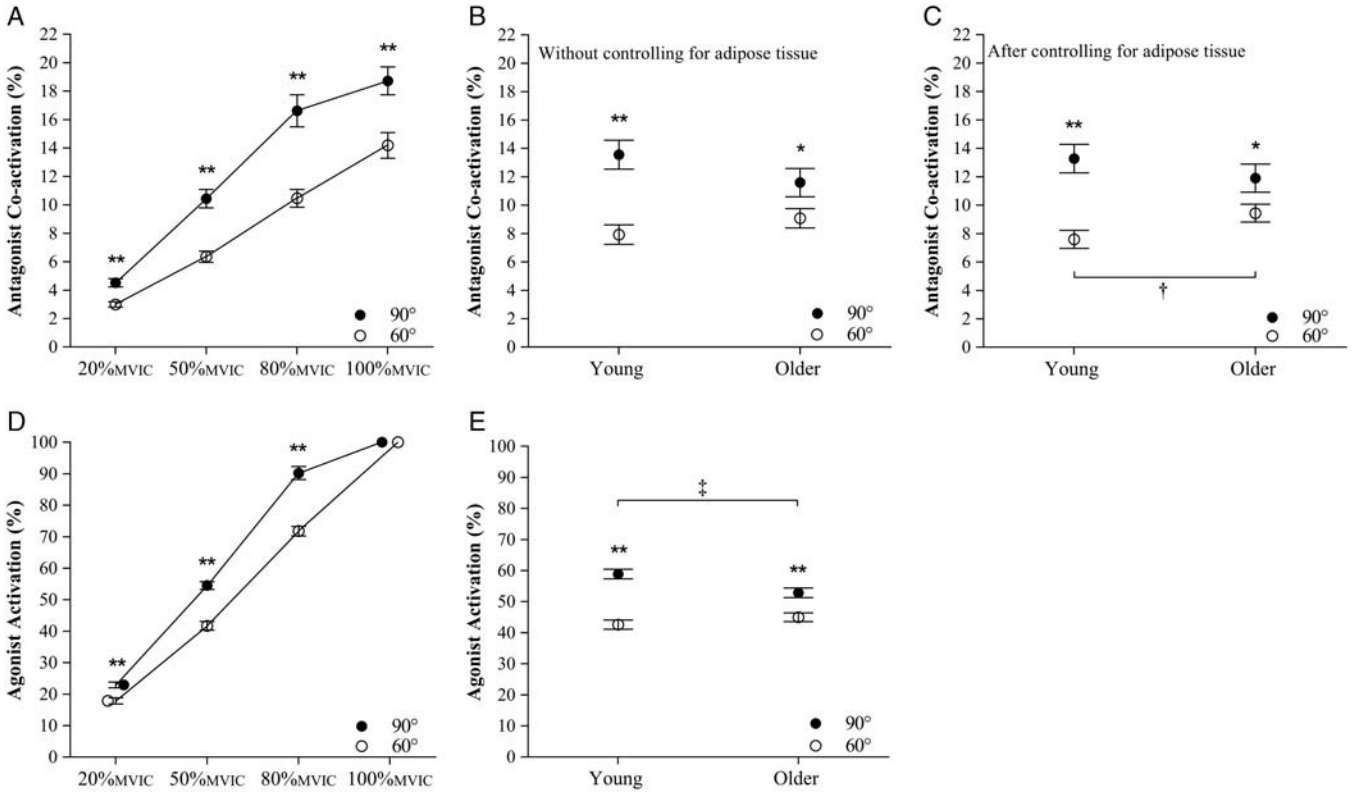

FIGURE 2—A and D, Comparisons, regardless of age, between 90° and 60° of knee flexion as a function of contraction intensities for antagonist coactivation (A) and agonist activation (D) are shown. B and C, Results for antagonist coactivation at both 90° and 60° of knee flexion in young and older participants without (B) and after (C) controlling for subcutaneous adipose tissue. E, Results for agonist activation comparing older and young participants. All values were mean ± SE. *$P < 0.01$, **$P < 0.001$ for 90° compared with 60° of knee flexion. †$P < 0.05$, ‡$P < 0.01$ for young compared with older participants.

$P < 0.001$) compared with older ($F_{1,47} = 15.1$, $\eta^2 = 0.24$, $P < 0.001$) participants (Fig. 2E).

DISCUSSION

In the present study, the effect of knee joint angle on the neural drive to agonist and antagonist muscles at varying contraction intensities was comprehensively examined, for the first time, comparing young and older individuals of both sexes. Moreover, subcutaneous adipose tissue was specifically measured and included in the analysis to investigate the potential effect it could have on the surface EMG amplitude response.

Our study revealed that 1) a higher level of antagonist coactivation and agonist activation during isometric contraction of the KE was observed at 90° compared with 60° of knee

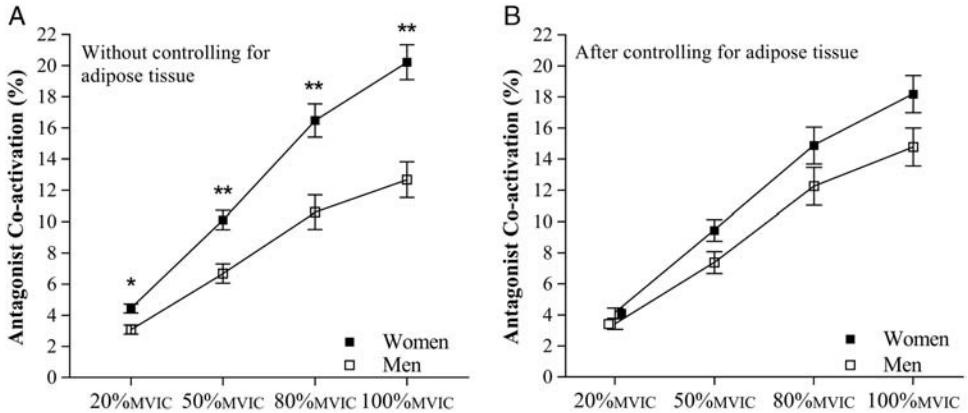

FIGURE 3—Antagonist coactivation (mean ± SE) in men and women at varying contraction intensities without (A) and after (B) controlling for subcutaneous adipose tissue. *$P < 0.01$, **$P < 0.001$ for men compared with women.

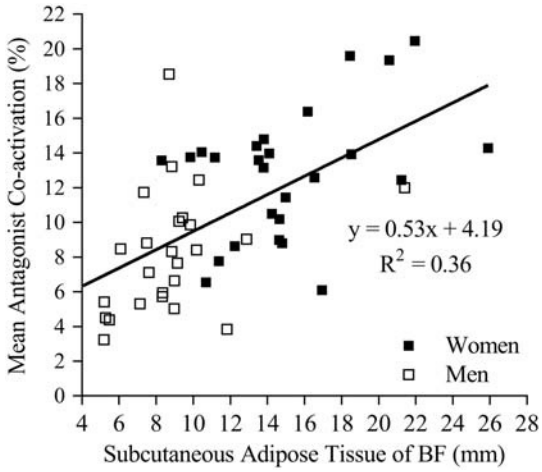

FIGURE 4—Correlation between antagonist coactivation (average of four contraction intensities at two joint angles) and subcutaneous adipose tissue thickness covering the BF.

flexion (Figs. 2A and 2D) and 2) after controlling for the effect of BF subcutaneous adipose tissue, older participants were characterized by a higher level of antagonist coactivation at 60° of knee flexion compared with young participants (Figs. 2B and 2C), whereas the higher levels of antagonist coactivation previously observed in the women disappeared (Figs. 3A and 3B).

Effect of contraction intensity and joint angle. Increased activation of agonist muscles and coactivation of antagonist muscles as a function of contraction intensity during isometric knee extension contraction has previously been detailed (2,21,22). During a voluntary contraction, muscle torque is modulated by a combination of motor unit (MU) recruitment and changes in MU firing rate (26). Net knee joint torque, as presented in this study, is the result of agonist (i.e., KE) and antagonist (i.e., KF) torques. It has been purported that antagonist-generated opposing torques are augmented in proportion to increased joint torque, such as at the knee joint (3,13) and ankle joint (6). To generate higher levels of desired joint torque, a greater number of recruited MU and increased MU firing rates are required, thus resulting in a rise in EMG activity of both agonist and antagonist muscles.

It has been suggested that antagonist coactivation (i.e., hamstrings) could reduce the amount of anterior–posterior joint shear force produced by quadriceps contraction, thus helping to preserve knee joint stability (3). Because of the movement of the distal patellar tendon insertion as the knee joint angle changes, the magnitude of this shear force varies with knee angular position (1). As often observed, the KE torque generated by individuals is smaller at knee joint angles close to full knee extension and/or flexion (4,23). The knee joint moment arm has been found to be maximal between 30° and 70° of knee flexion (where 0° represents full knee joint extension) and to decrease at knee flexion angles > 70° (17). Therefore, at more knee-flexed positions, the knee joint and ligament strain might be increased as a consequence of the higher antagonist muscle force to compensate for the mechanical disadvantage of the shorter moment arm to maintain constant force output (3). Indeed, our study reported greater antagonist coactivation combined with a lower KE muscle torque at 90° when compared with 60° of knee flexion (Fig. 2A and Table 1). This result is consistent with those of Krishnan et al. (21), who reported a higher antagonist coactivation level of both medial and lateral hamstring muscles at 90° in comparison with 30° of knee flexion during several submaximal isometric contractions of the KE (10%, 20%, 30%, and 50% MVIC). Kubo et al. (23) also observed higher coactivation levels of BF at more flexed knee positions (100° and 110°) than at more extended positions (40°–80°) during maximal isometric knee extension.

Such variation in pattern, in which the antagonist coactivation level heightened at an increased knee-flexed position (i.e., 90° of knee flexion), is also commensurate with the activation level of the prime mover (i.e., agonist muscles). In the present study, 90° knee flexion was characterized by a higher activation level of the agonist muscles (i.e., KE) compared with 60° knee flexion, which is in general accordance with previous studies performed on the same muscle group (4,21,23,38). This in part is attributed to an increased Iα afferent input to the motor neuron pool of the quadriceps associated with a longer muscle length (i.e., 90° of knee flexion) (4,23). However, other factors are likely to be involved in explaining this response. For instance, it is logic to consider that the KE muscles have to overcome the additional torque generated by the hamstrings at 90° of knee flexion, which is expected to counteract shear forces at the knee at that angle. In addition, another factor to justify this observation could be linked to an increased central drive as previously suggested (21). Therefore, our results lend support to the original hypothesis proposed by Baratta et al. (3), suggesting that the observed changes in antagonist coactivation as a function of joint angle and contraction intensity may play an important role in maintaining knee joint stability by providing synergistic action to counteract the agonist's increasingly destabilizing influence.

Effect of sex and age. In the present study, no sex-related difference was found for antagonist (i.e., KF) coactivation in both young and older individuals after controlling for the effect of subcutaneous adipose tissue (Fig. 3B). Indeed, several previous studies have observed no significant effects of sex on the hamstrings antagonist coactivation in children, young individuals, and older individuals (8,18,22,40). Therefore, we suggest that at least for the knee joint musculature, the adaptation of antagonist coactivation level as a function of contraction intensity and joint angle is sex independent. In addition, after including the subcutaneous adipose tissue in the analysis, we observed a higher level of KF coactivation in older participants when compared with their younger counterparts, but only at 60° of knee flexion (Fig. 2C). Several previous studies have reported inconsistent results, indicating that older individuals either exhibit an increase (15,25) or remain unchanged (5,40) in the coactivation

level of antagonist muscle around the knee joint. Our results seem to explain, at least partially, this discrepancy via the existence of subcutaneous adipose tissue covering the examined muscles because of its well-known effect on the amount of EMG cross-talk contamination originating from muscles adjacent to those under consideration (16,27,36). Therefore, we recommend that when surface EMG amplitude is used as a measure of muscle activity across populations characterized by different levels of fatness, the effect of subcutaneous adipose tissue should be also considered. It is important to say that in many studies, the torque contribution from the coactivation of the KF has been estimated from the EMG activity (5,6). This antagonist "extra" force is usually added to the measured MVIC of the KE to obtain the "true agonist MVIC" and to estimate the level of specific force (5,6). It is then obvious that a possible cross-talk contamination will have a significant impact on these assessments. During voluntary movement, healthy young individuals can generate knee joint torque by optimally coordinating the concurrent activation of agonist–antagonist muscles. On the contrary, older individuals might generate a desired torque with a different neural strategy, which adopts an enhanced antagonist coactivation (14). The higher levels of antagonist coactivation observed in elderly persons have been frequently ascribed to the reduction in reciprocal inhibition with aging (14,19). This assumption has been supported by Kido et al. (19), who examined the short-latency reciprocal inhibition in 22 participants (age 22–82 yr) from the soleus muscle by stimulating the common peroneal nerve, as well as in 21 participants (age 22–82 yr) from the tibialis anterior muscle by stimulating the tibial nerve. Accordingly, they reported a negative relationship between the magnitude of inhibition and age and, therefore, concluded that reciprocal reflex inhibition through the Iα inhibitory interneuron diminished with age. In the present study, when a similar level of agonist activation was observed between young and older participants, the elderly persons were characterized by a higher level of antagonist coactivation (i.e., 60° of knee flexion) (Figs. 2C and 2E). Therefore, we could suggest that the age-related decrease in reciprocal inhibition may account for the increased antagonist coactivation observed in our older individuals at 60° of knee flexion. What remains to be explained is why this difference was not present at 90°. It has been proposed that hamstring muscles attenuate anterior translation of the tibia and anterior cruciate ligament (ACL) tension by counteracting the contraction of the quadriceps (3,13). However, it has been shown that this reflexive activation of the hamstrings is more applicable for the terminal 60° before full knee extension (35) because the ACL can only provide tension between the tibia and the femur from 60° of knee flexion to near full extension during isometric contraction of the KE (32). This may help to explain why the significant difference between young and older participants was observed only at 60° of knee flexion but not at 90° (Fig. 2C). It could be speculated that older individuals adopt the strategy of heightened antagonist EMG activity at 60° flexion, as a compensation for the age-related deterioration in the mechanical properties of knee joint ligaments (28), to ensure the integrity of the musculoskeletal system and knee joint stability during voluntary contraction (3,37). On the other hand, when knee extension contraction takes place at 90°, the ACL is unloaded and the tibia translates posteriorly with respect to the femur (13,35). Therefore, age-induced reduction in knee joint ligaments properties may have a lesser effect on joint stability, hence resulting in a similar coactivation level at 90° of knee flexion between young and older participants.

This age-related deterioration in mechanical properties around the knee joint may also contribute to explain the smaller difference observed, in the level of agonist activation between 90° and 60° of knee flexion, in older compared with young participants (Fig. 2E). The agonist activation (i.e., KE) at longer muscle length (i.e., 90° of knee flexion) is usually potentiated by the activation of Iα afferent excitatory input originating from the muscle spindles (4). However, part of the stretch imposed on the muscle–tendon unit could be also absorbed by the tendon itself (30). Therefore, an age-related increase in joint tendon/ligaments compliance can cause less stretch being transmitted to muscle spindles at longer muscle lengths, resulting in a decreased Iα afferent excitatory input to the α motor neuron pool in response to muscle stretching in older individuals (20,30).

Obviously, there are limitations to this investigation. First, only VL and BF muscles were measured to represent the quadriceps and hamstrings muscle group, respectively, although this approach has been previously adopted by numerous studies (8, 18, 40 among many others). In addition, another limitation relates to the way the subcutaneous adipose tissue was measured especially in respect to the potential pressure difference regarding the BF subcutaneous adipose tissue (covering the area where the surface EMG electrodes were attached). It is acknowledged that extra pressure is inevitably produced on the tissue (covering the hamstrings) in a seated position, although it was difficult to obtain subcutaneous adipose tissue thickness in a seated position by using the ultrasound measurement adopted in the present study. On the other hand, we think that the way we approach this should have minimized this problem.

In summary, the results of the present study confirmed that neural drive is dependent on joint angle and torque level. We observed that the level of antagonist coactivation and agonist activation were higher at 90° compared with 60° of knee flexion, with both antagonist coactivation and agonist activation increasing as a function of contraction intensity. In addition, after controlling for the effect of subcutaneous adipose tissue, older participants exhibited a higher level of antagonist coactivation at 60° of knee flexion compared with young participants, whereas the previously observed sex difference disappeared. The results indicate that the presence of subcutaneous adipose tissue in different populations would affect the observed antagonist coactivation level, measured by surface EMG, possibly because of the volume-conducted cross-talk. In addition, our study indicates that the

mechanical properties of knee joint tendon/ligaments may also account for the age-related changes in neural drive to agonist and antagonist muscles. Overall, these conclusions could lend support to the original hypothesis formulated in previous studies, which suggests that agonist–antagonist coactivation may represent a protective mechanism that serves to stabilize the knee joint and maintain constant motor output. Our approach can contribute to a better understanding of the age-related decline in neuromuscular function, for instance, in relation to the quantification of the specific force decline in older individuals.

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