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MUSCLE COACTIVATION: A GENERALIZED OR LOCALIZED MOTOR CONTROL STRATEGY?

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Abstract

Introduction—We examined generalized versus joint-specific influences on muscle coactivation.

Methods—Muscle coactivation was assessed during maximal isometric and isokinetic knee and elbow joint extension moments in 48 healthy subjects (27 men). Local (joint-specific) and generalized (person-specific) contributions were examined using a combination of statistical tests, including regression with generalized estimating equations (GEEs), exploratory factor analysis, and cluster analysis.

Results—GEEs produced similar significant coefficients for gender and joint; contraction type and test condition (angle or velocity) were not significant. Factor analysis indicated 2 joint-based factors, and cluster analysis indicated 2 groups of individuals, those with and without elevated coactivation at the knee and elbow. Women exhibited greater coactivation at both joints, but no consistent influences of angle or velocity were observed at either joint.

Conclusion—Muscle coactivation is a neuromuscular control response determined by local, joint-specific, and generalized, individual-specific influences.

Keywords

elbow; EMG; knee; motor control; muscle cocontraction

Muscle coactivation or cocontraction is the simultaneous activation of agonist and antagonist muscles. It is believed to be an important motor control strategy to improve joint stability^{1–3} and movement accuracy.⁴ In particular, coactivation of the hamstrings during quadriceps contraction (i.e., during extension moments) provides greater knee stability through joint compression and counteraction of the anterior shear induced by the pull of the

quadriceps on the tibia.^{1,2,5,6} In the upper extremity, muscle coactivation has been demonstrated to produce greater movement accuracy and reduced phase lag to external perturbations.⁴ This phenomenon has been studied during both sub-maximal, functional tasks^{7–12} and maximal isometric or isokinetic tasks,^{1,2,13–20} demonstrating its ubiquitous occurrence across activities.

Despite numerous studies of the degree of flexor muscle coactivation, particularly about the knee, it is not entirely clear to what degree this neuromuscular response is a generalized (i.e., central or feed-forward) motor control strategy vs. localized (i.e., feedback response based on joint-based afferent input). Although peripheral, joint-specific feedback is often assumed to be a major contributor to antagonist coactivation, some evidence supports the hypothesis of a generalized, centrally mediated motor control influence as well. Pathological agonist–antagonist coactivation in rhesus monkeys has been produced from damage to the globus pallidus of the basal ganglia.²¹ Similarly, dystonias, many of which involve abnormal muscle coactivation, are associated with damage to subcortical or brainstem regions involving the putamen, caudate nucleus, and thalamus.^{22,23} Interestingly, a classic study in monkeys concluded that antagonist coactivation results from activation of the premotor areas, which are distinct pathways from reciprocal muscle activation,⁴ and highlighted the complexity involved in muscle coactivation. More recently, force fluctuations in knee and elbow flexor muscles were found to be correlated, which are indicative of a central drive between these upper and lower extremity joints.²⁴ Last, although not universally observed, isometric ham-string coactivation during knee extension did not vary with mechanical variations, such as joint angle²⁵ or contraction velocity,^{14,26} despite the effects of joint reaction forces (i.e., compression and shear) produced by the quadriceps.^{18,27} These findings collectively suggest generalized motor control processes may play a role in normal muscle coactivation. However, no previous studies have attempted to characterize the degree to which normal antagonist coactivation is due to joint- or contraction-specific vs. generalized, individual-specific influences.

Thus, the primary purpose of this study was to assess muscle coactivation during extension moments between 2 joints within the same individual. To address this question, muscle coactivation during maximal isometric and isokinetic contractions about the knee and elbow joints were examined in a cohort of men and women. We hypothesized that muscle coactivation would exhibit both generalized (individual-specific) and localized (joint-specific) influences despite inherent differences in joint stability between the knee and elbow. Secondly, we hypothesized that, consistent with a generalized response, women would exhibit greater antagonist coactivation at both joints, based on trends observed in a previous study that may have been underpowered to determine significance.²⁸ This information may further our understanding of motor control strategies resulting in antagonist muscle coactivation to maintain joint stability and/or optimize movement accuracy during static and dynamic contractions.

METHODS

A total of 52 volunteers participated in the study, a component of a larger study described elsewhere.²⁹ All subjects provided written informed consent as approved by the local

institutional review board. There were technical problems with the electromyography (EMG) data collection in 4 subjects; this resulted in a total of 48 subjects with usable data, including 27 men (26 ± 6.1 years, 182.7 ± 5.6 cm, 86.3 ± 6.5 kg) and 21 women (22.6 ± 3.3 years, 169.2 ± 6.1 cm, 66.1 ± 12.3 kg). All subjects were in good general health, with no reports of any serious medical conditions, musculoskeletal pathology, or use of prescription medications.

Muscle Electromyography

Muscle activity was measured using surface EMG (Bagnoli 2.1; Delsys, Boston, Massachusetts) of the knee and elbow flexors (biceps femoris and biceps brachii, respectively) and extensors (vastus lateralis and triceps brachii, respectively). Each differential EMG electrode consisted of 2 self-contained silver bar electrodes ($1 \text{ cm} \times 2 \text{ mm}$) spaced 1 cm apart. The EMG signal was pre-amplified 10 times locally at the electrode and further amplified at either 1K or 10K gain as needed by the Bagnoli anti-aliasing bandpass filter (20–450 Hz). The skin was cleaned with rubbing alcohol before applying the EMG electrodes using commercially available double-sided adhesives (Delsys, Boston, Massachusetts). Electrodes were further secured with elastic-wrap bandages to minimize the risk of cables being caught during isokinetic testing. EMG electrodes were placed parallel to the muscle fiber orientations and midbelly of each respective muscle.

Muscle Coactivation

Coactivation occurred when the flexor muscles were active during extension moments (Fig. 1). Regions of antagonist activity were determined, excluding activity that was continuous with the prior or subsequent agonist contractions (i.e., flexion moments) to avoid including “agonist” activity in antagonist assessments. This is particularly important to consider during isokinetic testing where flexion and extension reciprocal contractions occur, and electromechanical delay can cause elevated EMG signals immediately prior to the initiation of the flexion agonist torque. Antagonist muscle EMG was standardized using the respective peak concentric muscle contraction for each test condition (i.e., isometric angle and isokinetic velocity). This provided percent of maximum values (%max).

There are several methods available to assess muscle coactivation. During submaximal efforts (e.g., functional tasks) a ratio of antagonist to agonist muscle activity is used frequently, that is, a coactivation index.^{30,31} This ratio (antagonist/agonist) is equivalent mathematically to estimating the level of antagonist muscle activity when the agonist is maximal (i.e., 100% active). However, during maximal strength testing, when the agonists are performing maximal voluntary contraction (MVC) either at a constant length or during a constant velocity contraction, the relative activity of antagonist muscles, normalized to their respective maximal activation under similar conditions, is also used as a measure of muscle coactivation.^{32,33} Although the coactivation index or ratio is particularly important for submaximal exertions to account for the level of agonist activation, the drawbacks of this approach include: (1) the necessity to assume which agonist muscles provide the best denominator for standardization, particularly when more than 1 agonist muscle may be active; and (2) not all agonists can be assessed readily using surface EMG (e.g., vastus intermedius of the quadriceps). Thus, for maximal contractions where an agonist and

antagonist phase is clearly differentiated, simply assessing the degree to which the antagonist is active provides a reasonable estimate of muscle coactivation.

Test Protocol

Subjects performed a 5-minute lower-body warm-up on a stationary cycle ergometer, pedaling at a self-selected comfortable pace prior to the knee strength testing, and a light upper-body warm-up using small (3–5-lb.) hand weights to perform 10–12 elbow flexion and extension contractions. All isometric and isokinetic testing was completed as described previously²⁹ using standard test positioning for knee and elbow joints on an isokinetic dynamometer (System 3; Biodex Medical Systems, Shirley, New York). Briefly, subjects were positioned and secured such that the respective joint center of rotation, that is, lateral femoral condyle or lateral epicondyle, was aligned with the dynamometer axis of rotation. Range-of-motion (ROM) limits were set from full extension (0°) to approximately 120° of knee flexion or 140° of elbow flexion.

Isometric torques were measured at 5 positions across the available knee or elbow ROM (15°, 35°, 55°, 75°, and 100° for knee; 15°, 45°, 60°, 90°, and 110° for elbow) using 1 of 3 randomly assigned testing orders to minimize possible effects of muscle fatigue. At each position, subjects performed 4 maximum contractions for flexion and extension, with 1-minute rest intervals between each contraction. Before beginning the isokinetic testing, a 5-minute rest was provided. Using the same joint ROM, 5 angular velocities (60°/s, 120°/s, 180°/s, 240°/s, and 300°/s) were then tested in 1 of 3 (randomly assigned) test orders to minimize order effects. Several submaximal trial repetitions were performed at each velocity for familiarization prior to the 4–7 maximal repetitions, with more repetitions at higher velocities. Isokinetic testing involved continuous contractions, alternating between flexion and extension throughout the full ROM. Between each test velocity, a 3-minute rest was provided to minimize the effects of fatigue.

Data Processing

The raw analog signals were sampled digitally at 1000 Hz using a 16-bit data acquisition board (National Instruments, Austin, Texas) and recorded using custom software (Lab-View, version 8.0; National Instruments). The digitized data were analyzed further with a 200-ms linear envelope (moving average), again using Lab-View v8.0. Torque results have been presented elsewhere.²⁹ The agonist and antagonist EMG activities were identified and averaged for each isometric and isokinetic contraction (LabView). Peak mean muscle activity was determined for each contraction condition (5 angle-specific isometric and 5 isokinetic) for each torque direction (flexion and extension) for each joint (knee and elbow). Thus, a total of 20 coactivation measures were extracted during extension moments for each subject. Baseline noise was accounted for by using nonlinear baseline correction (squared difference approach), as described previously.³⁴ Missing values ($n = 10$ data points, <1.0%) were imputed with NORM freeware software, using multiple imputations of incomplete multivariate data under a normal model (version 2.03).³⁵ These values were missing at random, as they were not dependent on any subject- or test-specific characteristics.

Statistical Analyses

Descriptive statistics were assessed for the 20 individual EMG coactivation variables (2 joints, 5 isometric conditions, and 5 isokinetic conditions) and across all angles (isometric mean) or all velocities (isokinetic mean) for each joint (SPSS, version 20; IBM, Armonk, New York). Regression analysis was used to predict which factors significantly influenced coactivation levels with generalized estimating equations (GEEs) to account for the correlations between repeated measures. Flexor EMG (%maximum) was the dependent variable, and the independent, predictor variables included: joint (knee vs. elbow); contraction type (isometric vs. isokinetic); condition nested within contraction type (5 angles or 5 velocities); and gender (men vs. women). To accommodate the correlations between the repeated-measures data, autoregression with a lag one correlation structure was utilized. β coefficients, their 95% confidence intervals, and Wald chi-square statistics were assessed for each predictor and their 2-way interactions. Significant gender differences, indicating central or individual-specific influences, and/or significant joint differences, indicating peripheral influences, were determined based on the respective β coefficients.

Pearson correlations were computed between the repeated flexor coactivation levels to determine whether further analysis using exploratory factor analysis was appropriate. If significant correlations were observed, principal axis factoring with varimax rotation and Kaiser normalization was used to determine the latent variables that best explained the observed variability in coactivation EMG. An optimal factor solution was determined based on eigenvalues >1.0 and examination of the scree plot using all 20 condition-specific coactivation measurements (knee and elbow, isometric, and isokinetic conditions). This analysis indicated which test condition (i.e., static vs. dynamic), joint (knee vs. elbow), or both, most strongly contributed to peripheral flexor muscle coactivation influences.

Cluster analysis using a 2-step clustering algorithm, the log-likelihood distance measure, and the Schwarz–Bayesian criterion (BIC) were used to group individuals based on their 20 elbow and knee and isometric and isokinetic antagonist coactivation variables. Cluster quality was assessed using the average silhouette value (a measure of cluster cohesion and separation). Between-group differences were confirmed using follow-up independent *t*-tests with modified Bonferroni tests. Between-cluster effect sizes (Cohen *d*) were calculated. For all statistics, significance was set at a $\alpha = 0.05$.

RESULTS

Generalized Estimating Equations

Muscle coactivation differed significantly between men and women and between the knee and elbow (Table 1), but contraction type and all 2-way interactions were non-significant. The β coefficients were of similar magnitude for the between-individual (gender) and within-individual (joint) differences. Accordingly, knee coactivation levels were higher than elbow coactivation levels by an absolute difference of 5.3% of maximum, after adjusting for all other model predictors across the isometric and isokinetic test conditions (Fig. 2). Similarly, coactivation was greater in women than in men by 6.0% of maximum, across test conditions, after adjusting for the remaining predictors.

Factor Analysis

Muscle coactivation levels were more strongly correlated among the within-joint comparisons than the between-joint comparisons (results not shown for brevity), indicating factor analysis was an appropriate statistical process to perform. The factor analysis results are shown in Table 2. A clear solution consisting of 2 factors was ascertained using principal axis factoring, based on the scree plot and eigenvalues,³⁶ explaining 60.1% of the total variance observed. Based on the resulting loading coefficients of the rotated solution, the 2 factors were unmistakably joint-specific and labeled— elbow and knee coactivation, respectively. Thus, the specific contraction conditions (i.e., isometric vs. isokinetic or specific angles or velocities) for each joint were not explanatory factors for muscle coactivation, consistent with the GEE results above.

Cluster Analysis

Two clusters were observed, with an average silhouette value of 0.4, indicating fair cohesion within clusters and separation between clusters. The clusters were differentiated primarily by consistent moderate (cluster 1) vs. high (cluster 2) antagonist coactivation observed in both knee and elbow flexors (Table 3). Between-cluster effect sizes (Cohen *d*) were consistently large across contraction types and joints, indicating an elevated generalized coactivation response in cluster 2. The proportions of men and women in each cluster were significantly different (Table 3), suggesting these individual-specific differences (i.e., a generalized tendency to coactivate flexor muscles during extension moments) were also associated partially with the previously observed gender differences. However, both subgroups contained men and women, and thus gender alone does not fully explain these generalized differences.

DISCUSSION

In this study, we examined normal muscle antagonist coactivation across multiple joints in a single cohort. Our main finding was that muscle coactivation is a result of both joint- and individual-specific influences, suggesting that both localized and generalized motor control strategies contribute to normal muscle coactivation.

Knee muscle coactivation was significantly greater than elbow muscle coactivation across testing conditions, which supports a localized, joint-specific influence on muscle coactivation. This observation is consistent with the premise that the elbow joint is inherently more stable than the knee. However, no consistent influences of joint angle or contraction velocity were apparent, which suggests that angle- and velocity-dependent agonist torque levels were not strongly influencing antagonist coactivation. This may be somewhat counterintuitive given the known differences in mechanical advantage between flexors and extensors and the variations in shear forces that occur with joint angle.^{37–39} This lack of variation among joint angles is consistent with several previous reports of coactivation at the knee,^{25,27} but in opposition to others.⁴⁰ Similarly, the lack of a consistent influence of contraction velocity on muscle coactivation observed in this study is consistent with several previous studies on the knee^{26,41} and another study of the elbow.¹⁴ Conversely, an increase in coactivation with increased velocity was observed previously¹⁶ at angles

approaching full extension. However, this study utilized angle-specific antagonist coactivation assessments, which may be problematic, given the wide range in error associated with electromechanical delays^{42–44} when assessed across multiple movement velocities. As the only increase in coactivation was observed near full knee extension, this may simply be a result of the activation needed to initiate the subsequent flexor contraction (i.e., due to the electromechanical delay). The larger sample size in our study in combination with the majority of the earlier findings provides further evidence that no consistent relationship exists between muscle coactivation and velocity during elbow or knee extension torque exertions.

The significant cluster results that show generalized differences in coactivation between 2 groups of individuals indicate that some degree of generalized facilitation or inhibition of muscle coactivation occurs in each subgroup. This supports the hypothesis that centrally driven, generalized motor control pathways are involved in normal muscle coactivation in addition to local joint influences. Indeed, this finding may be a further extension of the “common drive” hypothesis, that agonist–antagonist pairs are coactivated through a centrally mediated common descending drive.^{45,46} In this case, a common drive appears to be applied across joint regions rather than simply within a pair of opposing muscle groups.

On the other hand, because the cluster differences appeared to, in part, mirror the observed gender differences, we cannot fully rule out that peripheral differences between men and women, such as possible anatomical, structural elasticity, or morphological differences, were contributing to this generalized response. Clearly, many reasons may contribute to gender differences, including both peripherally and centrally mediated mechanisms. Future studies are needed to examine these differences. However, we can conclude that these results support our initial hypothesis, based on prior reports of tendencies for women to show greater coactivation,²⁸ as we were adequately powered to detect the differences in our larger cohort.

This finding of a generalized coactivation response may in turn drive future investigators to examine whether these strategies can be modified or adapted with training and intervention. Indeed, coactivation in osteoarthritis and stroke patient populations has garnered significant attention.^{10,30,47–49} It is possible that the generalized and joint-specific components that drive antagonist coactivation may respond differentially to intervention.

In addition to providing joint stability, coactivation is thought to contribute greater accuracy to fine motor activities.⁵⁰ Thus, although the knee may require greater coactivation to counteract the anterior shear forces of the quadriceps tendon, the elbow, a joint that is involved in more fine motor activities than the knee, may be hypothesized to require greater coactivation for accuracy. This relationship was not demonstrated overtly in this study, because the knee clearly had greater coactivation during extension moments. However, the task did not require significant fine motor control, as subjects merely had to exert maximal effort with little concern for task accuracy. Thus, these coactivation levels may be underestimating motor control strategies used for precision tasks, particularly for the elbow.

Several limitations should be highlighted that may affect the generalizability of this study. First, isokinetic and isometric contractions were examined and not functional tasks. Coactivation motor control strategies may differ when the musculoskeletal system is exposed to normal external perturbations. Further, values may differ from coactivation indices calculated during submaximal contractions, as they typically rely on standardization from 1 of the agonist muscles. However, our main purpose was to test for the presence of global and local influences, not to define normative levels of muscle coactivation. Thus, the control setting allowed by the isometric and isokinetic testing paradigm was useful.

Another potential limitation is that muscle activation is assessed indirectly using surface EMG, which may involve signal reduction and filtering due to subcutaneous adipose tissue or the measurement of aberrant electrical signals from other sources (e.g., cardiac, or cross-talk from nearby muscles). However, all signals were standardized to their own maximal signal to account for subject-specific variations in EMG amplitude. In addition, pilot testing was performed to ascertain whether cross-talk could be observed between flexor and extensor muscle groups. These pilot tests confirmed that signal cross-talk only occurred with significant deviations in standardized electrode placement. This, coupled with modeling studies, suggests a relatively low risk for cross-talk when electrodes are placed over the appropriate muscle bellies, differential signals are employed, and the interelectrode distance is small relative to the distance between muscles.^{51,52} Thus, we are confident our experimental approach maximized the reliability of our EMG signals.

In conclusion, we have assessed muscle coactivation across non-neighboring joints, the knee and the elbow, in the same cohort of individuals. Our results suggest that normal muscle coactivation is a neuromuscular control response determined largely by localized influences: the underlying mechanics of the joint of interest (but not the specific joint angle or contraction velocity). Secondly, person-specific variations in the propensity to use coactivation as a motor-control strategy across multiple joints indicate the presence of a generalized response.

Future studies are needed to more clearly define which, localized or generalized responses, may play the larger role and be more favorable targets for intervention in the presence of musculo-skeletal and/or neuromuscular pathology.

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Abbreviations

BIC	Schwarz–Bayesian criterion
EMG	electromyography

GEE	generalized estimating equation
MVC	maximum voluntary maximum
ROM	range of motion

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FIGURE 1.

An example of knee muscle EMG in 1 subject during an isometric knee flexion and extension strength test. Antagonist flexor muscle activity was assessed during knee extension (e.g., see region within dashed lines) and standardized by the peak flexor agonist EMG (processed using a 200-ms linear envelope).

FIGURE 2.

Mean (SE) antagonist muscle activation (% of maximum) is shown for **(a)** 5 isometric and **(b)** 5 isokinetic contractions for men (black triangles) and women (gray circles), for the biceps femoris (knee flexor, solid lines) and biceps brachii (elbow flexor, dashed lines), during extension. [Color figure can be viewed in the online issue, which is available at wileyonlinelibrary.com.]

Table 1

Parameter estimates for predicting muscle coactivation using generalized estimating equations (GEEs).

Parameter	β	Standard error	95% Wald confidence interval		Hypothesis test		
			Lower	Upper	Wald chi-square	df	P
Intercept	9.47	1.274	6.99	11.96	55.78	1	0.000*
Gender (men vs. women [†])	-6.00	1.44	-8.82	-3.18	17.43	1	0.000*
Joint (elbow [†] vs. knee)	5.25	1.90	1.52	8.97	7.62	1	0.006*
Contraction type (isometric vs. isokinetic)	-0.06	1.08	-2.17	2.05	0.00	1	0.954
Gender * joint	2.82	2.24	-1.58	7.21	1.58	1	0.209
Gender * contraction type	-0.09	1.12	-2.29	2.11	0.01	1	0.938
Joint * contraction type	1.96	1.04	-0.08	4.00	3.56	1	0.059
Isometric * angle 1	1.32	1.06	-0.76	3.40	1.55	1	0.213
Isometric * angle 2	0.40	0.85	-1.27	2.06	0.22	1	0.640
Isometric * angle 3	-0.46	0.71	-1.84	0.93	0.42	1	0.519
Isometric * angle 4	0.15	0.73	-1.27	1.57	0.04	1	0.834
Isokinetic * velocity 1	-0.95	0.80	-2.51	0.61	1.43	1	0.232
Isokinetic * velocity 2	0.87	0.71	-0.53	2.26	1.48	1	0.223
Isokinetic * velocity 3	-0.68	0.49	-1.63	0.28	1.93	1	0.164
Isokinetic * velocity 4	0.70	0.82	-0.91	2.30	0.72	1	0.395

* Significant at the $P < 0.05$ level.[†] Reference category.

Table 2

Muscle coactivation latent variable factor loadings for the 2-factor solution.

Joint	Contraction type	Test condition	Factor 1 elbow coactivation	Factor 2 knee coactivation
Elbow	Isometric	Angle 1 = 15°	0.83	0.02
		Angle 2 = 40°	0.68	0.15
		Angle 3 = 65°	0.80	-0.06
		Angle 4 = 90°	0.84	0.04
		Angle 5 = 110°	0.67	0.32
	Isokinetic	60°/s	0.73	0.33
		120°/s	0.79	0.18
		180°/s	0.80	0.16
		240°/s	0.71	0.29
		300°/s	0.92	0.20
Knee	Isometric	Angle 1 = 15°	0.15	0.55
		Angle 2 = 35°	0.14	0.43
		Angle 3 = 55°	0.02	0.73
		Angle 4 = 75°	0.12	0.83
		Angle 5 = 100°	0.11	0.72
	Isokinetic	60°/s	0.14	0.61
		120°/s	0.18	0.84
		180°/s	0.03	0.66
		240°/s	0.29	0.57
		300°/s	0.10	0.66

Principal axis factoring with varimax rotation was used; principal loadings (i.e., largest for each variable) are highlighted in bold for clarity.

Table 3

Cluster summary information including mean (SD), between-group *P*-values, and effect sizes (*d*).

Variable	Cluster 1 (<i>n</i> = 38, 79.2%)	Cluster 2 (<i>n</i> = 10, 20.8%)	<i>P</i> -value [*]	<i>d</i>
M:W ratio (% M)	25:13 (65.8% M)	2:8 (20% M)	0.013 [†]	-
Elbow isometric coactivation (% maximum)	4.8 (2.6)	13.3 (7.8)	0.014 [†]	1.6
Elbow isokinetic coactivation (% maximum)	4.1 (2.7)	12.8 (7.0)	0.011 [†]	1.6
Knee isometric coactivation (% maximum)	12.8 (5.7)	22.3 (8.9)	0.017 [†]	1.3
Knee isokinetic coactivation (% maximum)	10.7 (5.7)	22.2 (8.4)	0.007 [†]	1.5

* Based on Fisher exact test (2-sided) for men:women (M:W) distribution; independent t-test with equal variances not assumed for coactivation measures.

[†] Significant at the *P* < 0.05 level.