Knee Extension and Flexion Weakness in People With Knee Osteoarthritis: Is Antagonist Cocontraction a Factor?

Author
G. Lloyd, David, L. Heiden, Tamika, R. Ackland, Timothy

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Knee Extension and Flexion Weakness in People With Knee Osteoarthritis: Is Antagonist Cocontraction a Factor?

Quadiceps weakness is one of the most common and disabling impairments seen in individuals with knee osteoarthritis (OA). Sufficient quadiceps and hamstrings strength, both isometric and dynamic, is essential for undertaking basic activities of daily living such as standing and walking. Muscle strength testing has revealed that those with knee OA have a 25% to 45% loss of knee extension strength and a 19% to 25% loss of knee flexion strength, compared with similarly aged controls. There are 3 factors thought to contribute to knee extension and flexion weakness in those with knee OA: muscle atrophy, failure of voluntary muscle activity, and apparent weakness from increased antagonist muscle cocontraction.

Decreases in muscle cross-sectional area (CSA) have been established in subjects with early OA degenerative changes and in those with severe knee OA. Ikeda et al found that women with early degenerative changes in the knee joint had reductions in quadiceps CSA of up to 12%, compared with age-matched women without radiographic changes. Fink et al found atrophy of type 2 fibers and type 1 fibers of the vastus medialis in patients undergoing total joint arthroplasty. Importantly, a smaller lean-muscle CSA in the diseased limb, compared to the contralateral limb, in subjects with OA contributes to muscle weakness but is not the only factor involved.

Several investigators have examined failure of voluntary muscle activity as a contributing factor in reduced knee extension strength. Failure of voluntary activity is the measure of an individual’s ability to fully activate their muscles during maximal voluntary contractions. Reductions in muscle activation are due to either an inability to recruit all motor units or a reduction in the motor unit discharge rate. In healthy adults, simulated failure of voluntary activity by saline injection into the knee joint to cause effusion resulted in decreased isometric extension strength and knee extension torque during strength testing and increased muscle cocontraction.

* STUDY DESIGN: Controlled laboratory study, cross-sectional data.
* OBJECTIVES: To investigate isometric knee flexion and extension strength, failure of voluntary muscle activation, and antagonist cocontraction of subjects with knee osteoarthritis (OA) compared with age-matched asymptomatic control subjects.
* BACKGROUND: Quadiceps weakness is a common impairment in individuals with knee OA. Disuse atrophy, failure of voluntary muscle activation, and antagonist muscle cocontraction are thought to be possible mechanisms underlying this weakness; but antagonist cocontraction has not been examined during testing requiring maximum voluntary isometric contraction.
* METHODS: Fifty-four subjects with knee OA (mean ± SD age, 65.6 ± 7.6 years) and 27 similarly aged control subjects (age, 64.2 ± 5.1 years) were recruited for this study. Isometric knee flexion and extension strength were measured, and electromyographic data were recorded, from 7 muscles crossing the knee and used to calculate cocontraction ratios during maximal effort knee flexion and extension trials. The burst superimposition technique was used to measure failure of voluntary activation.
* RESULTS: Knee extension strength of subjects with knee OA (mean ± SD, 115.9 ± 6.7 Nm) was significantly lower than for those in the control group (152.3 ± 9.6 Nm). No significant between-group difference was found for failure of voluntary muscle activation, or the cocontraction ratios during maximum effort knee flexion or extension.
* KEY WORDS: burst superimposition, OA, quadiceps strength, voluntary muscle activation
traction during gait. As such, failure of voluntary activity is thought to contribute to disability and has been proposed as an important element in the pathogenesis and further progression of knee OA.

The results of investigations examining failure of voluntary muscle activity in subjects with knee OA have been somewhat mixed, depending on disease severity and the measurement methods used. Two methods have been employed to assess failure of voluntary activity in subjects with knee OA: the burst superimposition technique and the twitch interpolation technique. In these measures, electrical stimuli are superimposed onto a voluntarily contracted muscle group, thereby recruiting any inactive motor units and subsequently generating additional force. Each method uses a different type of stimulus. The twitch interpolation technique uses a single pulse on various levels of muscle contractions, which is calculated as 1 – (superimposed twitch force at maximum voluntary isometric contraction ÷ twitch force at rest) and reported as a percentage of voluntary activation. The burst superimposition technique uses a train of stimuli and is calculated as the ratio between the maximal volitional effort prior to stimulus and the total force produced with the stimulus, termed the central activation ratio (CAR). A CAR of 1.0 implies that the muscle is fully activated through voluntary contraction, with no additional force obtained from the stimulus. Although both methods have been used in subjects with knee OA, the burst superimposition technique is the recommended technique during maximal contractions, due to trains of stimuli having greater sensitivity to activation deficiencies than superimposed single or double stimuli.

A loss of knee extension and/or flexion strength, often interpreted as muscle weakness, may be a consequence of increased antagonist muscle activation. Knee muscle cocontraction is thought to occur to stabilize joints and decrease anterior/posterior translation. For example, during isometric knee extension strength tests, the increases in joint torque production coincide with the increases in antagonist (hamstring) muscle activity, thereby generating a flexion moment that counteracts the extension moment generated by the quadriceps. This hamstring antagonist cocontraction appears as a loss of knee extension strength. In healthy adults (20–30 years of age), hamstring antagonist cocontraction during maximum isometric extension is between 5% to 10% of maximum activation. Others have found that elderly female subjects (mean age, 69.5 years) have approximately 15% more hamstring antagonist cocontraction during maximal isometric knee extension trials than younger female subjects (mean age, 22.8 years). So the aging process alone results in greater levels of hamstring antagonist cocontraction. Subsequently, subjects with knee OA who tend to be older must be compared with similarly aged, unaffected people.

Increased agonist-antagonist cocontraction levels have been reported in subjects with knee OA while walking and performing other activities of daily living. During activities of daily living, subjects with knee OA have greater hamstring antagonist cocontraction than their healthy counterparts, most likely in compensation for weak quadriceps or because of pain. Therefore, it is possible that they use this mechanism to protect the knee joint and minimize pain during isometric strength testing, which in turn would decrease the measured knee extension and flexion strength, giving the appearance of weakness. Lewek et al. observed low knee extension strength in subjects with knee OA and, by using the burst superimposition technique, showed that failure of voluntary activity was not exhibited in their cohort. They then suggested that the apparent decrease in knee extension strength was due to muscle atrophy. However, they did not examine the possibility for hamstring antagonist cocontraction. To date, the level of antagonist cocontraction during maximum voluntary isometric contractions (MVICs) has not been assessed in subjects with knee OA, especially in regard to low isometric knee extension strength. Antagonist cocontraction could be a factor contributing to apparent knee extension and flexion weakness reported in this population.

The purpose of this study was to examine knee extension and flexion strength, failure of voluntary muscle activation for knee extension, and antagonist cocontraction during maximal effort isometric knee flexion and extension in subjects with knee OA. Consistent with what has been reported previously, we hypothesized that subjects with knee OA would have reduced isometric knee extensor and knee flexor strength compared with age-matched asymptomatic controls. We also hypothesized that subjects with knee OA would not display greater failure of voluntary activity during maximal voluntary isometric knee extension compared to same-aged controls. However, we hypothesized that subjects with knee OA would exhibit higher levels of antagonist cocontraction during maximum effort isometric knee flexion and extension trials than the age-matched controls.

**METHODS**

Fifty-four subjects diagnosed with knee OA (30 female) were recruited through public advertisement and local orthopaedic outpatient clinics. These subjects had radiographic signs of knee OA, body mass index less than 35 kg/m², morning knee stiffness less than 30 minutes, could walk unassisted, and had not received steroid injections in the previous 6 months or regular physiotherapy in the previous 12 months. Subjects with knee OA were required to fill in the Knee Osteoarthritis Outcomes Survey (KOOS) as part of the inclusion criteria and were included based on an average score of less than 70 for each of 4 subscales: pain, symptom, activities of daily living, and quality of life. Difficulties associated with responses to the KOOS...
have previously been used as inclusion criteria when examining subjects with knee OA. Twenty-seven (18 female) asymptomatic controls, in the same age range as those with knee OA, were enlisted through local newspaper advertisements and university mailing lists. People diagnosed with any form of arthritis were excluded from the control group. Both those with knee OA and age-matched controls were screened and excluded if they suffered from any neuromuscular disease, cardiovascular disorder, or recent surgery or injury (within last 2 years) to the back or lower extremities. All test procedures were approved by the University of Western Australia Human Research Ethics Committee, the rights of all subjects were protected, and subjects gave their informed, written consent prior to testing.

Electromyographic (EMG) data were recorded using a 16-channel EMG system (Delsys, Boston, MA), with custom-made, in-lead, double-differential preamplifiers (input impedance, >1 MΩ; bandwidth, 20–450 Hz; common-mode rejection ratio, >140). After cleaning and abrasion of the skin, ClearTrace Ag/AgCl disposable circular (25-mm diameter) surface EMG electrodes (ConMed Corporation, Utica, NY) were applied with an interelectrode distance of 30 mm, in line with the muscle fibers over the midmuscle bellies. The following muscles of the test limb were tested: the quadriceps (rectus femoris, vastus lateralis, vastus medialis), the hamstrings (biceps femoris and semimembranosus), and the gastrocnemius (medial gastrocnemius and lateral gastrocnemius). The test limb was the most painful or functionally limited limb in subjects with knee OA, and for the controls, their test limb was selected to ensure matching with the body mass and height of the subjects with knee OA. A reference electrode was placed over the head of the fibula. The raw EMG data were checked for artifacts after placement of electrodes and prior to data collection.

Quadriceps, hamstrings, and gastrocnemius strength, and failure of voluntary muscle activation of the test limb were measured isometrically using an isokinetic dynamometer (Biodex Medical Systems, Inc, Shirley, NY). Subjects were seated with a hip angle of 90° and a knee angle of 90° for the knee strength testing. During the gastrocnemius testing, the knee angle was set at 65°. The subjects were secured with Velcro straps to reduce movement of the body and test limb. The knee joint was visually aligned with the dynamometer axis of movement and the device locked at 90° of knee flexion for quadriceps and hamstring testing. Following familiarization and warm-up trials, the subject underwent 3 MVICs for each of the quadriceps, hamstrings, and gastrocnemius muscles. Subjects were instructed to maximally extend the knee for quadriceps MVC testing, maximally flex the knee while maximally plantar flexing the ankle for gastrocnemius MVC trials. A computer provided real-time feedback and displayed a visual target line for each trial. The visual target line was set just above each subject’s maximum force attained during the warm-up trials and was adjusted as needed to ensure a maximum effort was undertaken. To motivate the subject, verbal encouragement was provided during each trial. EMG and torque data were sampled simultaneously at 2000 Hz. Subjects were given 2 minutes rest between each trial, to prevent fatigue. EMG data were normalized to the maximum value obtained for each muscle during the agonist MVIC trial. The trial with the strongest recorded torque for each muscle group was used in determining stimulus amplitude and target line values for the failure of voluntary activation testing described below. The muscle cocontraction values were calculated during the maximum torque trial to avoid stimulus artifact that would occur during the burst superimposition testing, affecting the EMG signal. The cocontraction was calculated across a 400-millisecond period, when the maximum torque was achieved and when the force was stable.

We chose to use hip and knee angles of 90° during knee strength testing for 4 reasons. Firstly, we did not want to expose the subjects with knee OA to too many strength tests, as well as electrical stimulation tests, over long periods. Secondly, even though the greatest knee extension strength occurs between 60° and 70°, with hip at 90°, the greatest maximum voluntary quadriceps activation is possible at knee and hip angles of 90°. Thirdly, the hamstrings tend to have the greatest activation with knee and hips angles at 90°. Finally, and most importantly, we wanted to utilize the same angle used by Lewek et al to examine the role of cocontraction in the possible absence of failure of voluntary activity.

To test failure of voluntary muscle activity, 2 aluminium-plate electrodes (70 × 50 mm), covered with saline-soaked pads, were attached proximally over the vastus lateralis and distally over the vastus medialis. Stimulation was performed by a Grass S88 square pulse stimulator, with a Grass Model SIU5A stimulus isolation unit (Grass Instruments, West Warwick, RI). The amplitude of the stimulation was adjusted to produce a torque equivalent to 25% of MVIC when applied to the resting muscle of each subject. A response equivalent to 20% to 25% maximum torque has been suggested adequate to measure failure of voluntary muscle activity. Initial stimuli began at low amplitudes and were increased over 3 or 4 trials until a sufficient response (20% to 25% of maximum torque) was elicited. Following stimulus familiarization and amplitude determination, the subjects were instructed to undertake an MVIC and aim for a visual target line on the computer screen in front of them. The visual target was adjusted between trials as needed, to ensure that subjects were continuing to work at a maximum effort in each trial. During the MVICs, the stimulus (amplitude, 100–150 V; pulse duration, 600 microseconds; pulse interval, 10 milliseconds; train duration, 100 milliseconds) was delivered automatically by computer 350 milliseconds after
the visual target line had been reached. The 350-millisecond interval was selected to ensure the likelihood of applying the stimuli when the subjects’ MVIC torques were stable. This is particularly important for the subjects with knee OA, as they may not have been able to hold an MVIC for any longer durations. The trial was not used if a subject did not reach the visual target line. Each subject completed 3 extension MVIC trials with the stimulus superimposed over the quadriceps. CAR values were used to express the level of inhibition and were calculated by dividing the subject’s voluntary torque immediately prior to stimulation by the torque value with the stimulus applied.25

Raw EMG and torque data were processed using custom-written software (Matlab, Version R2007a; The MathWorks, Inc, Natick, MA). Raw EMG data were full-wave rectified and filtered with a second-order, low-pass Butterworth, with a 6-Hz cutoff to create linear envelopes. EMG data were normalized for amplitude to maximum EMG values calculated from the Biodex trials. Raw torque data were filtered using a second-order, low-pass Butterworth filter with a 2-Hz cutoff. Knee extension and flexion strength were taken from the trial with the greatest measured torque.

Cocontraction has 2 components: the ratio of agonist and antagonist activation, and the total amount of activation of the agonists and antagonists. Therefore, we constructed 2 variables—cocontraction ratios (CCR) and net activation—that were examined during the maximum knee flexion and extension trials. CCRs used in this experiment were the ratio of hamstring (semimembranosus, biceps femoris) to quadriceps (vastus lateralis, vastus medialis, rectus femoris), and the ratio of flexion (semimembranosus, biceps femoris, medial gastrocnemius, lateral gastrocnemius) to extension (vastus lateralis, vastus medialis, rectus femoris). These ratios differed in that the hamstring-quadriceps CCR used only 2 flexors (semimembranosus and biceps femoris), while the flexion-extension CCR included the medial gastrocnemius and lateral gastrocnemius. These ratios were calculated as follows: if agonist mean EMG > antagonist mean EMG, CCR = 1 – (antagonist mean EMG / agonist mean EMG); else, CCR = (agonist mean EMG / antagonist mean EMG) – 1.

In these equations, if agonists were more active than the antagonists, the CCR would be above zero, and vice versa. Maximum cocontraction would be represented with a CCR equal to zero, while a minimum cocontraction is indicated with a CCR of 1 or –1. In addition to the CCR, net muscle activation values (ie, the sum of all agonist and antagonist activity) were calculated from the normalized EMG data for each flexion and extension trial.

Between-group differences for age, height, body mass, and KOOS subscales were examined using t tests. Isometric knee flexion and extension torque were examined between groups, using an analysis of covariance (ANCOVA) to account for the effect of body mass that was entered as the covariate. Regression analysis between body mass and torque showed equivalent slopes between the groups for extension (P = .691) and flexion (P = .437) tests; thus the assumption of homogeneity of slopes was not violated. The covariate and dependant variables were significantly correlated (P<.001) and scatter plots with regression lines fitted showed the existence of nonzero y intercepts, indicating the need for the use of the ANCOVA. Because body mass and muscle activation data were not significantly correlated, an analysis of variance (ANOVA) was sufficient to compare between group differences on the remaining dependant variables (flexion-extension CCR, hamstring-quadriceps CCR, net muscle activation for flexion and extension strength, and failure of voluntary activity). All statistical analyses were done using SPSS for Windows, Version 12.0 (SPSS, Inc, Chicago, IL). The analysis of 6 dependant variables required multiple statistical tests. A Bonferroni adjustment (0.05/6) resulted in a value of 0.008; therefore, a slightly higher significance level of P<.01 was used, as Bonferroni adjustment is known to be conservative.49

## RESULTS

**Table 1.** Demographics (Mean ± SD) and t Test Comparison of Subjects With Knee OA and Controls

<table>
<thead>
<tr>
<th></th>
<th>OA*</th>
<th>Controls†</th>
<th>t Score</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>65.6 (75)</td>
<td>64.2 (61.9)</td>
<td>0.82</td>
<td>.42</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.70 (0.09)</td>
<td>1.70 (0.09)</td>
<td>-0.12</td>
<td>.90</td>
</tr>
<tr>
<td>Body Mass (kg)</td>
<td>81.4 (14.2)</td>
<td>71.3 (13.8)</td>
<td>3.06</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>28.1 (4.2)</td>
<td>24.4 (3.6)</td>
<td>3.88</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>KOOS Pain (0-100)‡</td>
<td>575 (18.2)</td>
<td>96.2 (6.2)</td>
<td>14.04</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>KOOS symptom (0-100)‡</td>
<td>536 (176)</td>
<td>93.7 (8.1)</td>
<td>14.04</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>KOOS ADL (0-100)‡</td>
<td>60.0 (20.0)</td>
<td>96.6 (5.7)</td>
<td>12.46</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>KOOS QOL (0-100)‡</td>
<td>33.7 (15.8)</td>
<td>88.4 (15.3)</td>
<td>14.92</td>
<td>&lt;.01</td>
</tr>
</tbody>
</table>

Abbreviations: ADL, activities of daily living; BMI, body mass index; KOOS, Knee Osteoarthritis Outcomes Survey; OA, osteoarthritis; QOL, quality of life.

* n = 54 (female, n = 30).
† n = 27 (female, n = 18).
‡ Lower KOOS scores indicate greater functional declines and increase levels of pain and symptoms.

Age and height did not differ significantly between the groups, but the control subjects were 12.5% lighter than the subjects with knee OA (Table 1). KOOS scores revealed significant differences between the groups on all 4 subscales (Table 1). These values are...
similar to those previously reported for individuals with moderate OA (grade 2–3). Gender ratios were different between the control and OA groups. An analysis examining the effect of gender and group on each of the dependant variables showed nonsignificant interactions for gender ($P > .05$); thus the observed differences were not due to the affect of gender.

Using body mass as a covariate, the knee extension strength of the subjects with knee OA was significantly less ($P = .026$) than that of the control group. The covariate of body mass was significantly associated with knee extension and knee flexion strength ($P < .01$).

Mean ± SD quadriceps CAR values were $0.942 ± 0.041$ and $0.952 ± 0.033$ for the knee OA and control groups, respectively, and no significant difference existed between the groups ($P = .267$).

There were no significant differences between the control subjects and subjects with knee OA in the hamstring-quadriceps CCR, flexion-extension CCR, or net muscle activation measured in either the isometric knee flexion or extension trials (TABLE 2). Individual muscle activation during the flexion and extension MVICs (FIGURE 2) revealed no differences between the controls and subjects with OA.

**DISCUSSION**

As hypothesized, and as shown previously, the maximum knee extension strength of subjects with knee OA was less than that of control subjects. The knee flexion strength, although reduced, was not significantly lower in this group of subjects with knee OA, and there was no evidence to suggest a failure of voluntary quadriceps activity. In addition, our hypothesis that the magnitude of antagonist cocontraction during maximum effort isometric knee flexion and extension trials would differ from controls was not supported.

Previous studies have shown a large variation in the percentage deficit in knee extension strength of subjects with knee OA compared with healthy adults (26%–45%). In individuals with moderate to severe knee OA, these strength deficits are related to functional declines and the amount of voluntary quadriceps activation failure. Our 23% reduction in knee extension strength is smaller than that previously reported, but still substantially different to controls. The difference between our knee extension strength findings and those previously reported could be due to differences in the testing methods used, the reporting of torque values, or alterations in the muscle moment arms of the subjects with knee OA.

Methodologically, some previous studies have had insufficient rest between trials, no visual feedback or verbal encouragement, no warm-up trials and have used different knee angles than those used in this investigation. Additionally, differences could be caused by different units of measure and normalization procedures. The units of measurement for strength should reflect muscle generated torque (Nm), which is a product of force and the lever arm distance (m). Strength measurements, within the knee OA literature, have, in some cases, reported only the applied force (N), thus neglecting limb length differences between subjects. Another concern with some studies is the use of “ratio normalization” of torque by simply dividing the torque measure by body mass or body mass index (BMI). This ratio normalization fails to eliminate the confounding effects of a subject’s body size and structure on the peak torque values for 2 reasons: it assumes a linear relationship (in this case, one between peak torque and BMI or body mass), and (2) a y intercept of zero, which is rarely the case in biological experiments. ANCOVA has been effective for treating body mass as a confounding factor of knee strength.

**TABLE 2**

<table>
<thead>
<tr>
<th>Knee extension trials (mean ± SD)</th>
<th>Knee OA Subjects*</th>
<th>F Value</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hamstring-quadriceps CCR</td>
<td>$0.74 ± 0.17$</td>
<td>0.78 ± 0.12</td>
<td>1.510</td>
</tr>
<tr>
<td>Flexion-extension CCR</td>
<td>$0.07 ± 0.15$</td>
<td>0.78 ± 0.12</td>
<td>0.354</td>
</tr>
<tr>
<td>Net activation</td>
<td>$2.01 ± 0.37$</td>
<td>1.97 ± 0.31</td>
<td>0.272</td>
</tr>
</tbody>
</table>

Knee flexion trials (mean ± SD):

| Hamstring-quadriceps CCR         | $0.89 ± 0.13$    | 0.91 ± 0.05 | 0.826 | .339 |
| Flexion-extension CCR           | $0.77 ± 0.15$    | 0.90 ± 0.05 | 0.885 | .350 |
| Net activation                   | $2.30 ± 0.54$    | 2.12 ± 0.34 | 3.336 | .072 |

Abbreviations: ANOVA, analysis of variance; CCR, cocontraction ratio; net activation, activity summation of all muscles; OA, osteoarthritis.

* Values are mean ± SD.
between subjects with knee OA and controls in this study. We chose to use the burst superimposition technique due to its recommendation for use when undertaking maximal strength trials. Studies of muscle activation in individuals with knee OA using this method have provided mixed results, possibly due to methodological, disease status, and participant age differences. Three studies of muscle inhibition in subjects with end-stage knee OA, using the burst superimposition technique, have reported CAR values substantially less than the 0.94 to 0.95 values considered normal for healthy older adults. Stevens et al found a CAR value of 0.85 for the involved limb of subjects with knee OA, but this was not significantly different to the subject’s uninvolved limb. Mizner et al found a mean preoperative CAR value of 0.87 in subjects with knee OA but did not compare their data to a control group. Peterson et al reported CAR values of 0.76 in their knee OA group; however, this value was determined using the methods of Stackhouse et al, where the curvilinear relationship between CAR and maximal voluntary contraction are modeled using a submaximal contraction, making a comparison between their results and the present study impossible. The disparity between these previous studies and the results of the current investigation may be due to the differences in disease severity of the subject groups. However, alternative reasons could be due to differences in the stimulus amplitude and the knee flexion angle used during testing.

It is difficult to compare the precise values of voluntary activation failure between studies due to differences in the stimulus parameters used. Studies that examined voluntary activation failure in subjects with knee OA, using the burst superimposition technique, have failed to clarify how their voltage level was determined for testing and have used the same voltage for each subject, despite possible differences in body composition. Interestingly, Stevens et al did not report the voltage used during their

in unaffected subjects and subjects with knee joint pathologies. This is because it removes the linear effect of the covariate on torque and does not assume a zero y-intercept, making it the ideal solution in statistical tests of torque data.

Smaller muscle moment arms in those with knee OA could affect the maximum extension moment. However, there is no evidence to suggest that subjects with knee OA have changes in, or smaller, knee flexion and extension moment arms.

Failure of voluntary muscle activity was not responsible for the measured reductions in knee extension strength

![Bar chart showing muscle activation in OA subjects and controls](image-url)
testing. Based on differences in subject response to the stimulus due to muscle mass and adipose tissue, we believe that determining the appropriate voltage for each individual was necessary to ensure that all inhibited motoneurons were activated when the stimulus was applied. Additionally, given that our subjects had CAR of 0.95, a stimulus producing 25% of maximum torque from “at rest” would have been more than sufficient to maximally stimulate the muscles.

Differences in knee flexion angles used in studies of voluntary activation failure could be another reason for the differences found among studies. Several studies of voluntary quadriiceps activation failure have used a knee flexion angle of 75°, due to reductions in the knee flexion range of their participants, compared to the 90° knee flexion angle used in the current investigation. Failure of voluntary muscle activity measured at knee flexion angles of less than 80° has been shown in subjects who were normal, with quadriiceps activation levels of 85% compared to 95% when tested at knee flexion angles greater than 80°. This may account for the differences between the current investigation and these aforementioned studies of subjects with knee OA. Indeed, our study is similar to the study by Lewek et al, enrolling subjects with moderate knee OA and using a 90° knee flexion angle with the burst superimposition technique. This may explain the agreement of our results (CAR, 0.94) with those of Lewek et al, who also failed to find muscle inhibition in a group of subjects with knee OA (CAR, 0.93) compared with controls. Importantly to the current study, Lewek et al then surmised that the lower knee extension strength was due to muscle atrophy. However, they did not examine the possibility of hamstring cocontraction reducing the apparent isometric knee extension strength.

To our knowledge, this is the first examination of antagonist cocontraction during maximal isometric extension did not differ between controls and subjects with knee OA (22%-26%), nor were there any differences in the net muscle activation level between groups. These hamstring cocontraction values were higher than those recorded for healthy adults in studies using identical muscle groups (9%-13%). This is not surprising, as antagonist cocontraction of the hamstrings during maximum isometric knee extension has been shown to be greater in older women (40%) as compared to young women (20%). Our cocontraction values are much lower than those previously found for healthy older women. This difference in cocontraction level between the current investigation and that of Macaluso et al may be due to our use of biceps femoris, medial hamstrings, and 3 quadriceps muscles, where they only used biceps femoris and vastus lateralis to examine antagonist cocontraction. In isokinetic knee extension, the antagonist cocontraction of the biceps femoris, a more lateral hamstring muscle, has been found to have greater values than that measured from the medial hamstring. If this variation in medial and lateral hamstring muscle activity also applies to an isometric contraction, then the use or inclusion of other flexor and extensor muscles in the calculation of cocontraction, could account for these differences. Antagonist quadriceps activity, in the knee flexion trials in this study, did not differ between controls and subjects with knee OA and does not differ with age.

The subjects with knee OA in the present study demonstrated reduced knee extensor strength in the absence of voluntary muscle activation failure and hamstring antagonist cocontraction. These results suggest that muscle atrophy was the most likely cause of quadriceps weakness. However, the knee extension strength of the subjects with knee OA in this study may have been better than in some studies. A lack of testing and reporting standards makes comparisons between studies difficult. Given the moderate severity level of our knee OA cohort, large declines in strength and high levels of inhibition were expected. It is possible that a group of subjects with knee OA, with even greater decline in quadriceps strength compared to controls, could exhibit both failure of voluntary muscle activation and increased levels of antagonist cocontraction; but this has not been measured. Alternatively, subjects with greater knee varus or valgus deformity may exhibit different levels of failure of voluntary activity and/or cocontraction.

The lack of difference in failure of voluntary muscle activation and antagonist cocontraction between the controls and subjects with knee OA suggests that the knee joint deterioration does not alter the level of muscle activation needed in an isometric task. It is important to note that these results may only be applicable to this task, and that in more functional tasks, antagonist cocontraction, failure of voluntary activation, and muscle atrophy may all contribute to knee extension weakness.

There are several limitations of this study. Firstly, although patients were physician diagnosed or waiting for knee replacement surgery, we were unable to obtain radiographic data for OA grading. However, the self-perceived data obtained from the KOOS was similar to that of subjects with moderate OA. Secondly, the collection of EMG data and the calculation of cocontraction ratios during the MVIC trials and not during the stimulation trials could have affected the results; but the protocols within each trial were the same (visual target line and verbal encouragement, and sufficient rest between trials to provide consistency in the testing). The rationale behind using the MVIC trials for the collection of cocontraction data was to ensure that EMG data were adequately sampled while the subject was producing a steady MVIC.

Because antagonist cocontraction is altered in the gait patterns of subjects with knee OA, it is thought that a more dynamic isokinetic test may reveal different results. Due to the differences shown in voluntary muscle activation failure based
on the knee flexion angle, it is possible that antagonist cocontraction levels may also differ between subjects with knee OA and controls at knee flexion angles other than 90°. Clearly, further investigation is required to examine the levels of antagonist cocontraction in subjects with knee OA at different knee angles and during more dynamic tasks such as isokinetic strength testing.

CONCLUSION

This study revealed no differences between the subjects with OA and control subjects in muscle cocontraction and failure of voluntary quadriceps muscle activation during maximal isometric knee extension trials. These findings suggest that altered muscle cocontraction and failure of voluntary quadriceps muscle activation are not associated with the reductions in isometric knee extension torque exhibited in this group of subjects with knee OA.

KEY POINTS

FINDINGS: Reducions in isometric knee extension strength in subjects with knee OA are not due to quadriceps muscle inhibition. Further, antagonist cocontraction is not increased in subjects with knee OA and is not thought to contribute to the isometric knee extension weakness.

IMPLICATIONS: The findings of this investigation highlight the need for pure muscle-strengthening exercises in individuals with knee OA.

CAUTION: These results may be specific to this group of subjects with knee OA. Additionally, increases in antagonist cocontraction could potentially occur during more dynamic strength testing or activities.

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