Inhibition of Maximal Voluntary Isokinetic Torque Production Following Stretching Is Velocity-Specific

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ABSTRACT

Recent research has shown that a regimen of stretching provides an acute inhibition of maximal force production by the stretched muscle group. To further characterize this phenomenon, the effect of an acute stretching regimen on maximal isokinetic knee-extension torque at 5 specific movement velocities (1.05, 1.57, 2.62, 3.67, and 4.71 rad·s⁻¹) was examined in 10 men and 5 women (22–28 years). Each person's 5 baseline maximal isokinetic knee-extension torques (dominant leg) were measured on a Cybex NORM dynamometer. Following the baseline torque measurements, the participants stretched the dominant quadriceps for 15 minutes using 1 active and 3 passive stretching exercises. Once the stretching exercises were completed, the maximal torque measurements were repeated. Poststretch maximal torque at 1.05 rad·s⁻¹ was significantly reduced (p < 0.05) from 218 ± 47 Nm (mean ± SD) to 199 ± 49 Nm (7.2% decrease). At 1.57 rad·s⁻¹, a similar decrease (p < 0.05) was also seen (204 ± 48 Nm vs. 195 ± 47 Nm; 4.5% decrease), but at the other velocities (2.62, 3.67, and 4.71 rad·s⁻¹), poststretch maximal torque was unaltered (p > 0.05). It appears, therefore, that the deleterious impact of stretching activities on maximal torque production might be limited to movements performed at relatively slow velocities.

Key Words: isokinetic contraction, flexibility, strength loss, warm-up


Introduction

Although stretching exercises designed to enhance flexibility are regularly included in both the training programs and the pre-event warm-up activities of most athletes, research exists that suggests pre-exercise stretching could have a negative impact on the performance of skills where success is related to maximal force or torque output (1, 3, 9). For example, Fowles and Sale (3) found that maximum isometric plantarflexion torque about the ankle joint was decreased by 28% immediately after the plantar flexors were passively stretched. A decrement in maximum torque was also present 60 minutes after stretching, albeit to a lesser degree (9%). In addition, stretching has been shown to elicit a strength deficit in concentric muscle actions. Kokkonen et al. (9) reported that a regimen of acute static stretching inhibited the 1 repetition maximum (1RM) lift of both knee extension (8% decrease) and knee flexion (7% decrease). The mechanism(s) responsible for such strength decrements following a bout of passive, static stretching, however, is (are) not clear. Acute increases in flexibility as a result of stretching have been attributed to both neurophysiological and mechanical factors (7). Consequently, a reduction in maximal force/torque output poststretching might be related to a change in neural or mechanical status.

Studies of static stretching have consistently reported decreases in motoneuron excitability during stretching as measured by the Hoffman-reflex (H-reflex; 2, 4). It is not apparent, however, if this depression in motoneuron excitability persists once a stretch is terminated. Some studies have found a significant mean decrease in the H-reflex immediately after stretching (1, 15), whereas others have not (4, 16). Until more conclusive evidence is available, it is possible that a reduction in maximal force output following stretching could be related to a decrease in neuromuscular function.

Nevertheless, we suggest that such a mechanism is probably not the prevailing cause of stretch-induced
force decrements. Although Avela et al. (1) and Thigpen et al. (15) found a depression of the H-reflex following termination of a stretch, neither of these studies provided sufficient evidence to suggest that this effect is sufficiently prolonged. Thigpen et al. (15) did not report how long after stretching the poststretch measurements were taken, and in the Avela et al. (1) study, a follow-up test 15 minutes poststretching revealed no significant difference in the strength of the H-reflex compared with the prestretch condition. Furthermore, in a subsample of 6 subjects, Avela et al. (1) observed that the H-reflex had almost completely recovered after 4 minutes. Moreover, the reflex might have recovered sooner had the authors imposed a more realistic stretching protocol in terms of the amount of stretching an individual would normally perform just prior to an event or exercise session. In an attempt to induce muscle spindle fatigue in the triceps surae, the muscle complex was subjected to prolonged, repeated passive stretching for 1 hour, whereas a typical protocol would subject a particular muscle to only several minutes of stretching. In the study by Kokkonen et al. (9), knee-extension and knee-flexion strength was reduced after stretching, although the knee extensors and flexors were stretched for only several minutes and the 1RM lift was achieved 10–15 minutes after the last stretch. Stretch-induced neuromuscular inhibition, therefore, was probably not the dominant mechanism behind the strength inhibition. Corroborating evidence has been provided by Fowles and Sale (3) who found that a poststretch decrement in maximum plantarflexion torque was present 60 minutes after stretching despite the fact that motor unit activation had virtually returned to prestretch values 15 minutes poststretch and had fully recovered after 30 minutes.

A possible mechanical mechanism, however, is a stretch-induced decrease in musculotendinous stiffness that has been demonstrated in some studies (11, 13) but not in others (5, 10). Wilson et al. (17) suggested that a stiff musculotendinous system allows for improved isometric and concentric force production. These investigators examined the relationship between active musculotendinous stiffness (stiffness of the muscle-tendon unit under stimulated conditions) and performance in a bench press-type movement under isometric, concentric, and eccentric conditions. They found a significant association between stiffness and maximum isometric force \((r = 0.63)\), maximum isometric rate of force development \((r = 0.78)\), maximum concentric rate of force development \((r = 0.65)\), and concentric work done \((r = 0.57)\). Their findings suggest that stiffness might influence performance the most in isometric contractions. No relationship between stiffness and maximum concentric force production was evident. However, the concentric test was performed with only 30% of maximum load, which would have allowed for a relatively fast movement velocity. It is possible that stronger relationships between stiffness and concentric performance exist at slower movement velocities; that is, in concentric contractions that more closely resemble isometric conditions. Likewise, the impact of passive muscle stretching on concentric force output may be more prominent at slower movement velocities that allow greater forces to be generated. In the study by Kokkonen et al. (9), the movement velocity was relatively slow because of the nature of the task. As mentioned previously, the major finding of this study was that stretching decreased knee-extension and knee-flexion strength as measured by the performance of a 1RM. As yet, no study has investigated if a similar effect of stretching on purely concentric performance exists at faster velocities of shortening. Such evidence would be of practical significance, as it would indicate that including stretching as part of a preactivity warm-up might compromise the performance of a wider range of activities. For example, if a similar effect is present at faster movement velocities, caution may also need to be exerted when including stretching as part of a warm-up in events such as swimming and cycle sprints where success is related to concentric power output. It was the purpose of this study, therefore, to further characterize the effects of an acute stretching regimen on concentric muscle performance by investigating the impact of stretching on maximal voluntary concentric knee-extension torque at 5 different movement velocities.

**Methods**

**Subjects**

Ten men and five women college students (age = 24 ± 6 years, mean ± SD) enrolled in professional physical education classes participated in the study. All individuals declared themselves free of any history of knee problems or pain and were naive to both the findings of Kokkonen et al. (9) and the purpose of the present study. The appropriate institutional review board approved the study, and each participant gave both written and oral consent before engaging in the experiment.

**Experimental Protocol**

Each participant performed 2 bouts (baseline and poststretching) of maximal voluntary isokinetic knee extensions with the dominant leg. Isokinetic torque was measured in the seated position on a Cybex NORM isokinetic device at 5 specific movement velocities (1.05, 1.57, 2.62, 3.67, and 4.71 rad·s\(^{-1}\)). The 2 measurement bouts were separated by 15 minutes of passive, static stretching of the dominant quadriceps muscle.

**Maximal Voluntary Isokinetic Torque Measurement Protocol**

Each participant was placed in an upright seated position and secured to both the Cybex NORM dyna-
mometer and corresponding chair according to company specifications in order to eliminate extraneous movements and to maintain a constant hip joint angle (90°). Following a quiet sitting period of 10 minutes, baseline maximal voluntary isokinetic torque measurements commenced. Four maximal voluntary isokinetic torque measurements were made at each of the 5 movement velocities with a 30-second rest period between each contraction. Each participant was randomly assigned to a counter-balanced design in terms of the specific testing sequence. All 4 measurements at a particular velocity were completed before the velocity was changed, and a minimum of 2 minutes was allowed to elapse before measurements were recorded at the next velocity. Each knee extension began at an initial knee angle of approximately 110° and went through a full range of motion to a final knee angle of 0°. The lower leg was then returned to the initial flexed position under 0 load in readiness for the next trial. Each contraction, therefore, was purely concentric and commenced from resting conditions.

On completion of the baseline measurements, the participant was released from the testing apparatus in order to undergo the stretching program. Following the stretching program, the participant was again secured to the apparatus in the exact same position used for the baseline tests, and the maximal voluntary isokinetic torque measurements were repeated using the same testing order and protocol as before. The NORM system sensitivity was set at 5 for all measurements as per company recommendations, and peak torque was calculated and adjusted for the effects of gravity by the NORM system software. Knowledge of individual or overall performances was not provided until the end of the study, and all participants were given strong verbal encouragement to do their best during each knee extension.

**Stretching Protocol**

The stretching program consisted of 3 passive, static stretching activities designed to stretch the quadriceps muscle group. The 3 exercises were preceded by a warm-up stretch performed by each individual. Each activity was performed 4 times either unaided (warm-up) or with assistance from 1 of the experimenters. When performing the assisted activities, the experimenter initially would push on the leg until a verbal acknowledgment of pain was received. The experimenter would then hold the participant's limb steady at that position for 30 seconds. At the end of the stretch, the leg was returned to a neutral position for 20 seconds, and then the stretch was repeated.

The warm-up stretch was performed by having the person first stand upright with 1 hand against a wall surface for balance. The person would then flex the knee joint to a 90° position. The ankle of the flexed leg was then grasped by the ipsilateral hand, and the foot raised so that the heel was as close as possible to the buttocks. The first assisted exercise was performed with the individual laying prone with the legs fully extended. An experimenter would then flex the individual's leg at the knee joint and slowly press the person's heel into the buttocks. As the heel contacted the buttocks, the knee was then lifted up off the supporting surface, causing hyperextension at the hip joint. The second activity started with the person lying supine along the edge of a table with the dominant leg hanging off of the table. An experimenter would flex the dominant leg at the knee and then proceed to hyperextend the leg at the hip. To perform the last activity, the participants stood with their back to a bench that was approximately level with the buttocks. They would then rest the dorsal side of the dominant foot on the bench by flexing at the knee joint. From this position, the experimenter would push backward on both the flexed knee and the corresponding shoulder.

**Statistical Analyses**

The isokinetic measurements for each velocity were analyzed using paired t-tests. The level of significance was set at \( p < 0.05 \) and was adjusted to cover for multiple comparisons using a Bonferroni adjustment (i.e., \( p \)-value divided by number of comparisons). Hence for significance at the 0.05 level to occur, the \( t \)-score needed to exceed the required \( t \)-score for the \( p < 0.01 \) level (i.e., 0.05 divided by 5).

**Results**

The results of the maximal voluntary isokinetic torque measurements for all of the movement velocities (1.05, 1.57, 2.62, 3.67, and 4.71 rad·s\(^{-1}\)) are presented in Figure 1. Following the stretching exercises, the mean maximal voluntary isokinetic torque for the velocities of 2.62, 3.67, and 4.71 rad·s\(^{-1}\) were not significantly different (\( p > 0.05 \)) from their respective prestretch values.

On the other hand, for the velocities of 1.05 and 1.57 rad·s\(^{-1}\), the mean maximal voluntary isokinetic torque measurement following the stretching exercises was significantly less (\( p < 0.05 \)) than the prestretch value. At 1.05 rad·s\(^{-1}\), the maximal torque had a mean 7.2% decrease (effect size = 1.32), and the mean decrease was 4.5% (effect size = 0.77) at 1.57 rad·s\(^{-1}\).

In addition to the maximal torque measurements, the knee angle at which the peak torque was achieved was compared between prestretch and poststretch conditions for each movement velocity. The mean knee angle at peak torque for all conditions is presented in Figure 2. As demonstrated in Figure 2, stretching had no influence (\( p > 0.05 \)) on the knee joint angle at which peak torque was achieved.
Figure 1. The mean (±SD) peak isokinetic torque before and after the stretching exercises for the movement velocities 1.05, 1.57, 2.62, 3.67, and 4.71 rad·s⁻¹. The asterisk indicates that the prestretching mean was significantly greater (p < 0.05) than the poststretching mean.

Figure 2. The mean (±SD) knee angle at which peak isokinetic torque was generated before and after the stretching exercises for the movement velocities 1.05, 1.57, 2.62, 3.67, and 4.71 rad·s⁻¹.

Discussion

It was the purpose of the present investigation to determine whether the reported inhibitory influence of acute muscle stretching on knee-extension 1RM was also manifested at a variety of movement velocities. The main finding was that a significant decrease in maximal voluntary isokinetic torque was seen at the 2 slowest velocities (1.05 and 1.57 rad·s⁻¹), with the greatest decrease (7.2%) occurring at the slowest velocity of 1.05 rad·s⁻¹. Interestingly, Kokkonen et al. (9) found that a knee-extension 1RM had an average decrease of 8.1% following an acute stretch, and the movement velocity during the 1RM test was between 0.79 and 1.05 rad·s⁻¹. Similar results, therefore, were obtained in the present study, lending support to the notion that the performance of a maximal concentric contraction under the conditions of a relatively slow movement velocity might be compromised if the contraction is performed after a bout of passive muscle stretching. A significant decrease in torque output of 4.5% was also seen at 1.57 rad·s⁻¹, but no differences were noted at the faster movement velocities of 2.62, 3.67, and 4.71 rad·s⁻¹. Consequently, we obtained no evidence to suggest that this effect persists at relatively fast velocities of shortening.

Although we cannot pinpoint the underlying mechanism that is responsible for our findings, it is possible that a poststretch decline in active musculotendinous stiffness caused the noted force decrement at the slowest movement velocities. However, because of our incapability of obtaining an accurate measure of musculotendinous stiffness under stimulated or active conditions due to the technical difficulties involved, we can only speculate about this potential mechanism. As previously mentioned in the introduction, Wilson et al. (17) found the strongest relationship between stiffness and force production to exist under isometric conditions. In concentric contractions, therefore, any decrease in stiffness might have a greater influence on the ability of the muscle to generate force at slower shortening velocities when the conditions for cross-bridge attachment more closely resemble those conditions under which isometric contractions are performed.

A logical explanation as to why an association between stiffness and muscle performance might be more pronounced at slower movement velocities can be formulated from the proposals of Wilson et al. (17). These investigators theorized that in maximal isometric and concentric contractions, a more compliant musculotendinous unit might less effectively develop force because of the contractile component operating under compromised length and velocity conditions. Specifically, a more compliant unit might initially allow the contractile component to shorten at a faster rate, and this would continue until the elastic components reached their limit of stretch. In addition, for a given level of activation, greater compliance might allow the contractile component to shorten more and operate at a less favorable point on the length/tension curve. It is tempting to suggest, therefore, that in the present study the stretching protocol reduced active musculotendinous stiffness sufficiently to allow the contractile component to shorten farther and at a faster rate, thus reducing force output. If these proposed mechanisms are valid, it is logical that they would be more influential at slower movement velocities. For concentric contractions, the force/velocity relationship shows
that for a given increase in shortening velocity the greatest drop in force occurs at the slowest velocities. Also, the higher forces that can be generated at slow velocities would cause a greater extension of the elastic structures, thereby allowing a greater decrease in contractile component length.

With regard to an increased shortening velocity, however, it could be argued that this effect should last only until the elastic structures reach their limit of stretch and become effectively stiff, thereby affecting the rate of force development, rather than peak torque. Indeed, Wilson et al. (17) noted that stiffness was more highly correlated with initial rate of force development than any other isometric and concentric performance variable. Nevertheless, peak torque may still be compromised by such a mechanism if the point at which the elastic structures become fully stretched occurs after the contractile component has shortened sufficiently so that it no longer operates on the plateau region of the length/tension curve. Furthermore, a short period of relatively fast contractile component shortening might compromise the ability of the cross-bridges to attach and produce force, even after the point of maximum stretch.

It has been demonstrated that isometric force is diminished if the isometric contraction takes place immediately after a period of active muscle shortening compared with a pure isometric contraction at an equivalent muscle length (6, 14). Although the mechanism behind this phenomenon is not known, Sugi and Tsuchiya (14) found that in a single fiber active stiffness under isometric conditions postshortening was less than the stiffness developed in the nonshortening condition. This outcome indicates that the decrease in force is related to a reduction in the number of attached cross-bridges. A similar disruption of optimal cross-bridge attachment may have occurred in the present study if the stretching treatment decreased active stiffness sufficiently to allow an initial period of rapid shortening. We must mention again, however, that we had no means of measuring the active stiffness of the quadriceps muscle-tendon complex. The aforementioned speculations, therefore, remain to be tested by future research.

Another potential mechanism for the decrease in poststretch torque output might be a reduced neural activation of the quadriceps. Some studies have shown decreases in motoneuron excitability during stretching as measured by the H-reflex (2, 4). It seems unlikely, however, that this effect would have persisted for the amount of time that was allowed to elapse between the final stretching exercise and the first isokinetic torque measurement. Following the stretching protocol, the participants were first secured to the chair of the dynamometer and then sat quietly for 10 minutes before the testing began. As discussed in the introduction, studies by Avela et al. (1) and Fowles and Sale (3) suggest that any neural inhibition would have sufficiently recovered by this stage. Furthermore, the fact that a reduction in peak torque was not consistently found across all movement velocities is evidence against such a mechanism. Nevertheless, we cannot readily discount the possibility of neuromuscular inhibition playing a role, because no electromyographic data was collected.

Regardless of any limitations, however, this study contributes to the growing body of evidence that acute stretching can inhibit a muscle's maximal force production. This body of evidence not only includes the work of Kokkonen et al. (9) on knee joint muscles, but also includes work on the ankle joint (1, 3), work with ballistic stretching exercises (8), and work involving the vertical jump (12). Moreover, it should be noted that the poststretch force decrement occurs even though conventional opinion suggests that warm-up activities, learning effects, and placebo effects (i.e., subjects expect improvement because it is still commonly thought that stretching improves performance) would lead to improvement.

The goal of future research in this area should be to explicate the underlying mechanisms responsible for the detrimental influence of preactivity stretching on performance. Furthermore, future work might investigate how robust this effect is across a variety of skilled activities. The present study suggests that any negative impact is of greater concern in those activities performed at slower velocities of movement where success is related to maximum force or torque output. Yet Nelson et al. (12) have shown vertical jump performance to be impaired by a prior bout of static stretching. A decrease jump height was evident regardless of whether the jump was performed with or without a stretch-shortening action. These seemingly contradictory findings might be explained by the fact that jumping is a much more skilled activity than performing maximal isokinetic knee extensions. The multijoint actions of the vertical jump and the sequential timing of segmental motion require a high degree of coordination for optimal performance. It is possible that the influence of stretching on force and power output might also be related to the complexity of the task.

Clearly, much more work needs to be done to establish how universal the deleterious effect of stretching is on the performance of activities that demand a high force and/or power output. It is recommended, therefore, that applied studies be conducted to investigate the direct impact of stretching on a variety of activities in order to establish the degree to which the negative influence of preactivity stretching can be generalized.

**Practical Applications**

This research points out 2 important facts concerning muscle performance following stretching. First, it sup-
ports the previous works that have shown prior stretching to decrease a muscle’s capacity to generate force. Second, this study suggests that the inhibitory relationship between acute stretching and force production is velocity-specific, with the greatest decrements seen at slower movement velocities. Therefore, in events such as weightlifting, where success is related to maximum force or torque output, rather than power output, it is probably not advisable to include intense stretching exercises as part of the warm-up, as this might degrade performance. However, the same caution may not be warranted in activities that rely on concentric power output such as sprint cycling.

References