

ACUTE EFFECTS OF STATIC STRETCHING ON PEAK TORQUE IN WOMEN

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ABSTRACT. Cramer, J.T., T.J. Housh, G.O. Johnson, J.M. Miller, J.W. Coburn, and T.W. Beck. Acute effects of static stretching on peak torque in women. *J. Strength Cond. Res.* 18(2):236–241. 2004.—The purpose of this study was to examine the effects of static stretching on concentric, isokinetic leg extension peak torque (PT) at 60 and 240°·s⁻¹ in the stretched and unstretched limbs. The PT of the dominant (stretched) and nondominant (unstretched) leg extensors were measured on a calibrated Cybex 6000 dynamometer. Following the prestretching PT assessments, the dominant leg extensors were stretched using 1 active and 3 passive stretching exercises. After the stretching, PT was reassessed. The results of the statistical analyses indicated that PT decreased following the static stretching in both limbs and at both velocities (60 and 240°·s⁻¹). The present findings suggested that the stretching-induced decreases in PT may be related to changes in the mechanical properties of the muscle, such as an altered length-tension relationship, or a central nervous system inhibitory mechanism. Overall, these findings, in conjunction with previous studies, indicated that static stretching impairs maximal force production. Strength and conditioning professionals should consider this before incorporating static stretching in preperformance activities. Future studies are needed to identify the underlying mechanisms that influence the time course of stretching-induced decreases in maximal force production for athletes and nonathletes across the age span.

KEY WORDS. Stretching-induced, dominant, nondominant, stretched, unstretched, maximal force production

INTRODUCTION

Static stretching is commonly performed prior to exercise (1, 6) and athletic events (4, 9). It is believed that increasing flexibility (increasing joint range of motion) will promote better performances and reduce the risk of injury during strenuous exercise (23, 24). A number of studies have used muscle stretching techniques to examine various aspects of muscle function including passive force production (14–16), stress-relaxation characteristics of muscle (17, 25, 26), neuromuscular reflex patterns (8, 10, 27), factors contributing to muscle damage (2, 13), and the mechanisms of increase in musculotendinous flexibility (14, 26). In addition, recent studies have examined the effects of static stretching on maximal isometric strength (3, 5, 7, 19) and concentric, isokinetic peak torque (PT) (21). Typically, pre-exercise stretching decreases isometric (3, 5, 7, 19) as well as dynamic muscle strength (12, 20–22, 28). This has implications for athletes involved in sports, such as powerlifting (21) and gymnastics (18), that require high levels of force production, and it has been suggested that performing static stretching prior to competition may hinder performance (7, 12, 18, 19, 21, 22, 28).

Two primary hypotheses have been proposed to explain the stretching-induced decrease in strength (3, 7, 11, 12, 19, 21, 22, 28): (a) mechanical factors involving the viscoelastic properties of the muscle, and (b) neural factors such as altered motor control strategies or reflex sensitivity. It has been suggested that the mechanical factors may include stretching-induced changes in the length-tension relationship of a muscle (7, 19, 21, 22). Other studies have indicated that decreases in muscle activation may partially account for the decreases in strength as a result of stretching the quadriceps femoris (5) and triceps surae muscles (3, 7). In addition, Avela et al. (3) studied the stretching-induced decrease in force production capabilities in both limbs by stretching the dominant plantar flexor muscles and using the contralateral limb as an unstretched control. Although Avela et al. (3) reported minimal effects of stretching on the unstretched limb, it was hypothesized that if the stretching-induced decrease in force production is mediated by a central nervous system (CNS) mechanism, it is possible that the unstretched limb may also be affected. Therefore, the purpose of this study was to examine the effects of static stretching on concentric, isokinetic leg extension PT at 60 and 240°·s⁻¹ in the stretched and unstretched limbs.

This study was designed to: (a) test the hypothesis of Nelson et al. (21) that the acute effects of static stretching on PT are velocity-specific during concentric, isokinetic leg extensions and (b) extend the findings of Avela et al. (3) by examining PT of the stretched and unstretched leg extensor muscles in women.

METHODS

Experimental Approach to the Problem

Fourteen women (mean age \pm SD = 22 \pm 1 year) who were recreationally active, but not involved in formal athletics, volunteered to participate in the investigation. The study was approved by the University Institutional Review Board for Human Subjects and all subjects completed a health history questionnaire and signed a written informed consent prior to testing.

Each subject completed a 5-minute warm-up at 50 W on a stationary cycle ergometer prior to the initial isokinetic testing. Before (pre) and after (post) the static stretching exercises, concentric isokinetic PT for extension of the dominant (based on kicking preference) and nondominant limbs were measured separately using a calibrated Cybex 6000 dynamometer (CYBEX Division of LUMEX, Inc., Ronkonkoma, New York) at randomly ordered velocities of 60 and 240°·s⁻¹. The subjects were in a seated position with a restraining strap over the pelvis

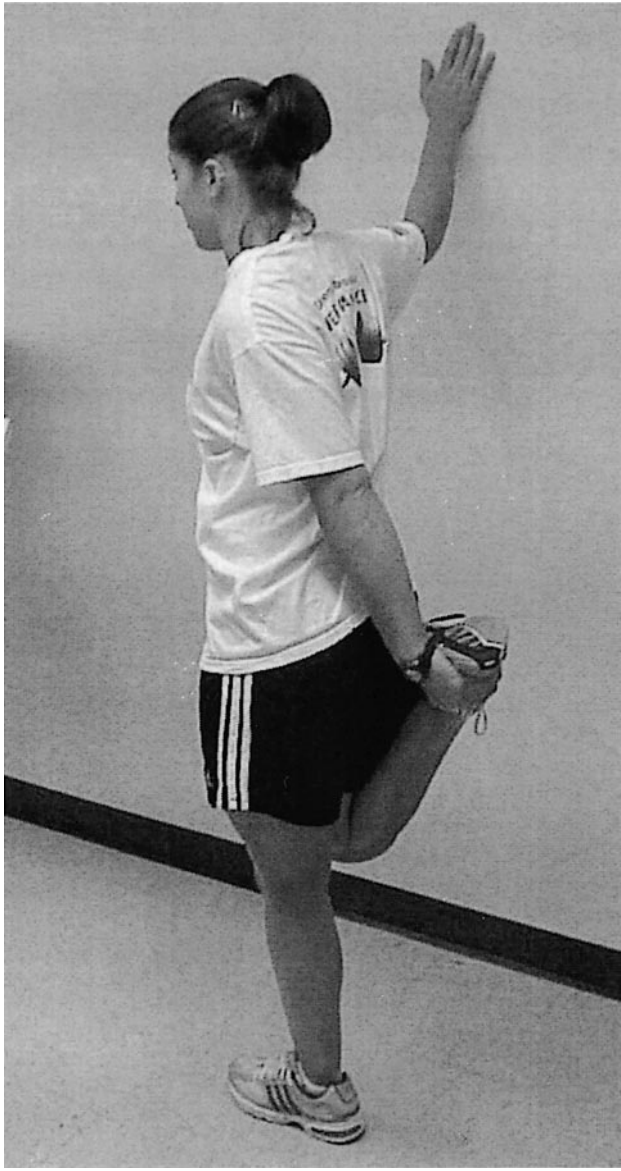


FIGURE 1. Example of the initial unassisted stretching exercise.

and trunk in accordance with the Cybex 6000 User's Guide (1991 *Cybex 6000 Testing and Rehabilitation User's Guide*, Cybex, Division of Lumex, Ronkonkoma, NY). The input axis of the dynamometer was aligned with the axis of the knee, while the contralateral leg was braced against the limb stabilization bar. Three submaximal warm-up trials preceded 3 maximal muscle actions at each velocity, with the highest PT selected as the representative score. A 2-minute rest was allowed between testing at each velocity, and a minimum of 5 minutes was allowed between testing for each limb. The joint angle at which PT occurred was provided by the Cybex 6000 software.

Immediately following the prestretching isokinetic tests, each subject underwent 4 static stretching exercises designed to stretch the leg extensor muscles of the dominant limb only, according to the procedures of Nelson et al. (21). Four repetitions of each stretching exercise were held for 30 seconds at a point of mild discomfort, but not

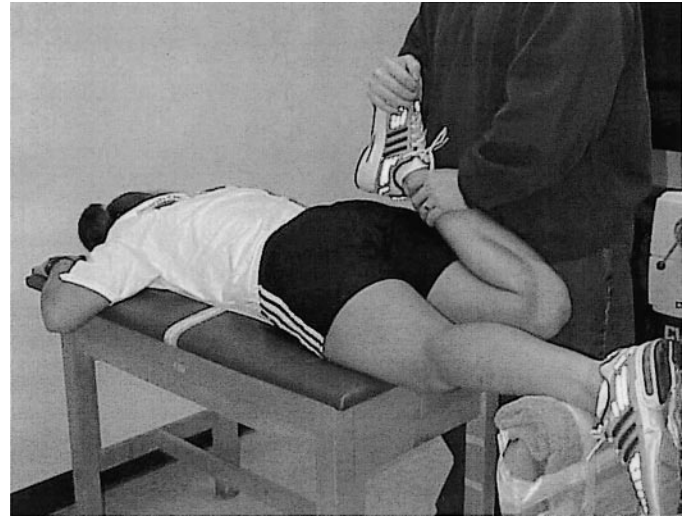


FIGURE 2. Example of the first assisted stretching exercise.

pain, as acknowledged by the subject. Between each stretching repetition, the leg was returned to a neutral position for a 20-second rest period. The total stretching time (mean \pm SD) was 16 ± 1 minutes.

Each subject performed an unassisted stretching exercise followed by 3 assisted stretching exercises. For the unassisted stretching exercise (Figure 1), the subject stood upright with one hand against a wall for balance. The subject then flexed the dominant leg to a knee joint angle of 90° . The ankle of the flexed leg was grasped by the ipsilateral hand, and the foot was raised so that the heel of the dominant foot approached the buttocks. Following the unassisted stretching exercise, the remaining stretching exercises were completed with the assistance of the primary investigator.

The first assisted stretching exercise (Figure 2) was performed with the subject lying prone on a padded table with her legs fully extended. The dominant leg was flexed at the knee joint and slowly pressed down so that the subject's heel approached the buttocks. If the heel was able to contact the buttocks, the knee was gently lifted off the supporting surface, causing a slight hyperextension at the hip joint, to complete the stretch. To perform the second assisted stretching exercise (Figure 3), the subject stood with her back to a table and rested the dorsal surface of her dominant foot on the table by flexing the leg at the knee joint. From this position, the dominant leg extensors were stretched by gently pushing back on both the knee of the flexed leg and the corresponding shoulder. The final assisted stretching exercise (Figure 4) began with the subject lying supine along the edge of the padded table with the dominant leg hanging off of the table. The dominant leg was flexed at the knee and the thigh was slightly hyperextended at the hip by gently pressing down on the knee. Immediately after the stretching exercises, each subject sat quietly for 4 ± 1 minutes (mean \pm SD) before performing the poststretching isokinetic tests for the dominant (stretched) limb, and 15 ± 3 minutes before testing the nondominant (unstretched) limb. Because the primary purpose of this study was to examine the effects of the stretching on the stretched limb, the isokinetic testing was always performed on the dominant limb first.



FIGURE 3. Example of the second assisted stretching exercise.

Previous test-retest reliability from our laboratory for PT during maximal, concentric isokinetic leg extensions indicated that, for 8 male subjects measured 48 hours apart, the intraclass correlation coefficients (R) ranged from 0.93–0.94 with no significant differences ($p > 0.05$) between mean values for test vs. retest at either velocity (60 and $240^\circ\cdot\text{s}^{-1}$).

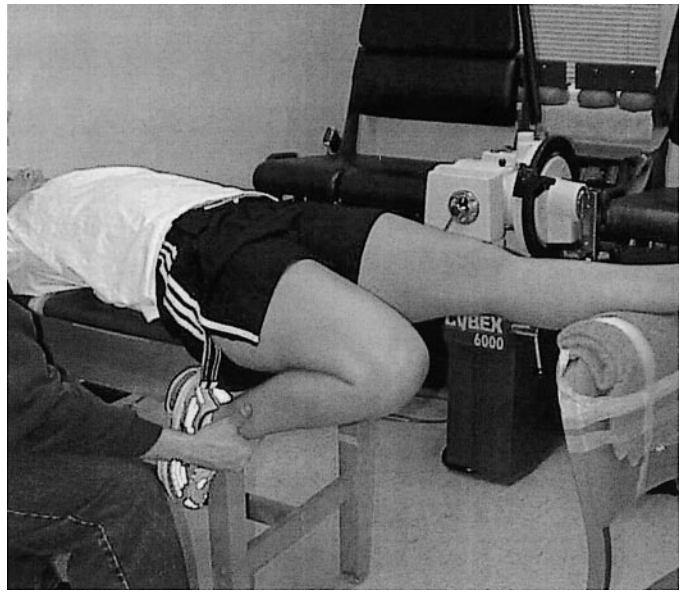


FIGURE 4. Example of the final assisted stretching exercise.

Two separate 3-way repeated measures ANOVAs (time [pre- vs. poststretching] \times limb [stretched vs. unstretched] \times velocity [$60^\circ\cdot\text{s}^{-1}$ vs. $240^\circ\cdot\text{s}^{-1}$]) were used to analyze the PT and joint angle at PT data. When appropriate, follow-up analyses included 2-way repeated measures ANOVAs and paired-samples t -tests. An alpha of $p \leq 0.05$ was considered statistically significant for all comparisons.

RESULTS

Table 1 contains the mean \pm SEM values for PT and joint angle at PT at pre- and poststretching. For PT, the analyses indicated no significant 3-way (time \times limb \times velocity) or 2-way (time \times limb; time \times velocity; limb \times velocity) interactions and no significant main effect for limb, but significant main effects for time and velocity. There was a decrease in PT collapsed across limb and velocity (Figure 5a) from pre- to poststretching. Furthermore, PT collapsed across time and limb was greater at $60^\circ\cdot\text{s}^{-1}$ than $240^\circ\cdot\text{s}^{-1}$ (Figure 5b).

For the joint angle at PT, the analyses indicated no significant interactions involving time (time \times limb \times velocity; time \times limb; time \times velocity), but a significant interaction for limb \times velocity and a significant main effect for time. Joint angle at PT collapsed across limb and velocity increased from pre- to poststretching (Figure 6a). Joint angle at PT collapsed across time for the dominant limb was greater than the nondominant limb at $60^\circ\cdot\text{s}^{-1}$,

Table 1. Peak torque (Nm) and joint angle at peak torque ($^\circ$) values.

Variable		Prestretching				Poststretching			
		Dominant leg		Nondominant leg		Dominant leg		Nondominant leg	
		$60^\circ\cdot\text{s}^{-1}$	$240^\circ\cdot\text{s}^{-1}$	$60^\circ\cdot\text{s}^{-1}$	$240^\circ\cdot\text{s}^{-1}$	$60^\circ\cdot\text{s}^{-1}$	$240^\circ\cdot\text{s}^{-1}$	$60^\circ\cdot\text{s}^{-1}$	$240^\circ\cdot\text{s}^{-1}$
Peak torque (Nm)	Mean	174.1	112.4	182.4	109.6	170.7	109.3	174.1	106.9
	SEM*	7.7	5.1	7.9	5.0	8.2	4.7	4.7	4.5
Joint angle at peak torque ($^\circ$)	Mean	65.1	5.29	62.2	57.9	64.1	57.1	57.1	59.1
	SEM*	1.0	3.0	1.3	1.8	1.5	1.6	1.3	1.4

* Standard error of the mean.

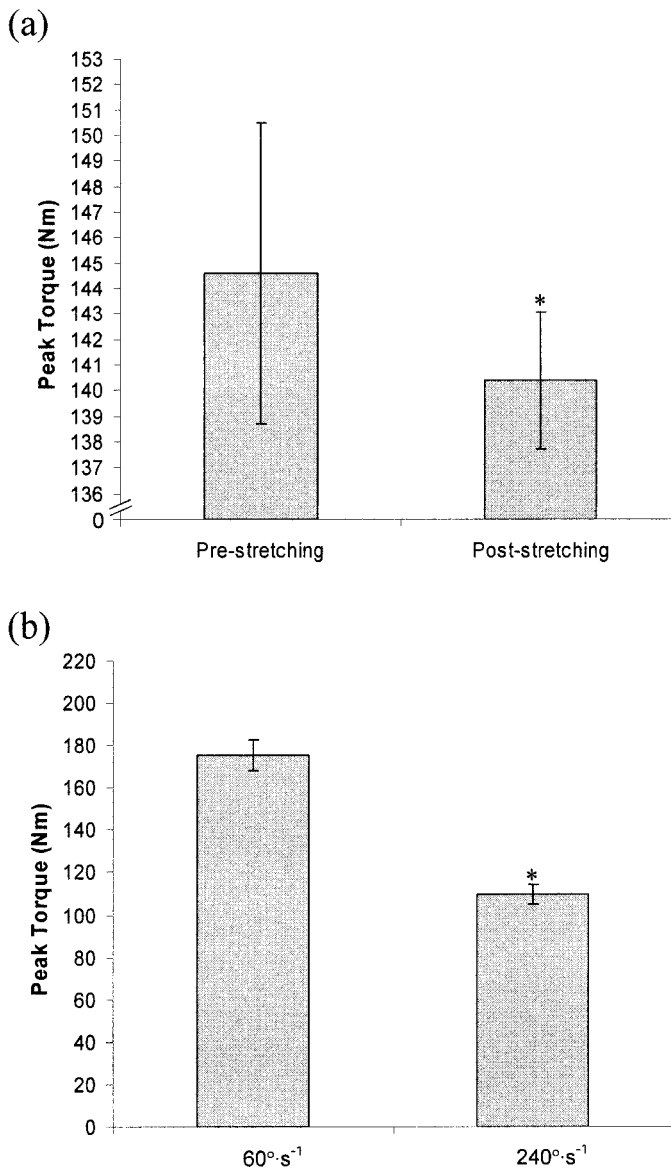


FIGURE 5. (a) The marginal means for peak torque collapsed across limb and velocity (Nm) decreased (* $p \leq 0.05$) from pre- to poststretching. (b) The marginal means for peak torque collapsed across time and limb (Nm) was greater (* $p \leq 0.05$) at $60^\circ \cdot s^{-1}$ than $240^\circ \cdot s^{-1}$. Values are mean \pm SEM.

while there were no differences between limbs at $240^\circ \cdot s^{-1}$ (Figure 6b). Furthermore, joint angle at PT collapsed across time was greater at $60^\circ \cdot s^{-1}$ than $240^\circ \cdot s^{-1}$ for both the dominant and nondominant limbs (Figure 6b).

DISCUSSION

The results of the present study were consistent with previous studies (5, 7, 12, 19, 21, 22) that have reported decreases in PT following static stretching (Figure 5). Recently, Nelson et al. (21) found decreases in PT at 60 and $90^\circ \cdot s^{-1}$ (7.2% and 4.5% decreases, respectively), but no change at 150, 210, or $270^\circ \cdot s^{-1}$ after an acute bout of static stretching. It was concluded that the decreases in PT after stretching were velocity-specific and occurred primarily under the high torque production conditions associated with the slower velocities (60 and $90^\circ \cdot s^{-1}$), but not the lower torque production conditions at the faster

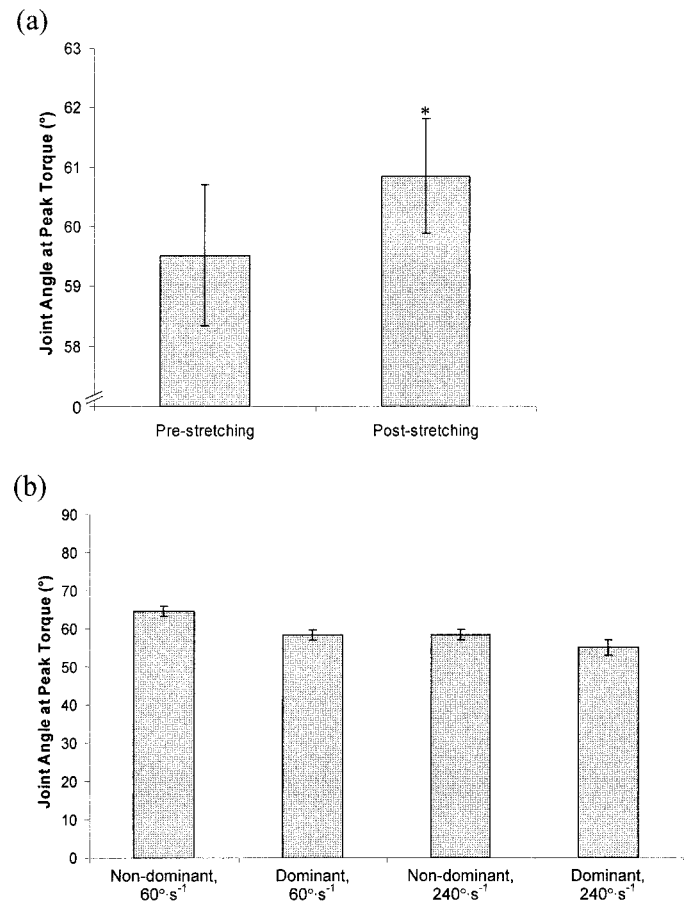


FIGURE 6. (a) The marginal means for joint angle at peak torque collapsed across limb and velocity (degrees) increased (* $p \leq 0.05$) from pre- to poststretching. (b) The marginal means for joint angle at peak torque collapsed across time (degrees) at $60^\circ \cdot s^{-1}$ were greater ($p \leq 0.05$) for the nondominant than the dominant limb, while at $240^\circ \cdot s^{-1}$ there was no difference ($p > 0.05$) between limbs. Joint angle at peak torque collapsed across time was greater ($p \leq 0.05$) at $60^\circ \cdot s^{-1}$ than $240^\circ \cdot s^{-1}$ for both the dominant and nondominant limbs. Values are mean \pm SEM.

velocities (150, 210, and $270^\circ \cdot s^{-1}$) (21). In the present study, however, the decreases in PT at $60^\circ \cdot s^{-1}$ (3.3%) and $240^\circ \cdot s^{-1}$ (2.6%) for the stretched leg suggested that stretching-induced decreases in PT may not be as velocity-specific as suggested by Nelson et al. (21).

Two hypotheses have been proposed for the stretching-induced decrease in force (or torque) production (3, 5, 7, 12, 19–22, 28): (a) mechanical factors involving the viscoelastic properties of the muscle that may affect the muscle's length-tension relationship, and (b) neural factors such as decreased muscle activation or altered reflex sensitivity. Recent studies (12, 19–22) have suggested that the primary mechanism underlying the stretching-induced decreases in force (after 10 minutes of recovery) is related to increased muscle compliance that may alter the muscle length-tension relationship, increase sarcomere shortening distance and velocity, and decrease force production due to the force-velocity relationship. A stretching-induced change in the length-tension relationship may also account for the increase in the joint angle at PT found in the present study. These findings were consistent with previous studies by Fowles et al. (7) and

Nelson et al. (19) that reported increases in the joint angle at which maximal isometric torque for plantar flexion or leg extension occurred after static stretching. Nelson et al. (21), however, found no stretching-induced changes in the joint angle at PT for maximal concentric isokinetic leg extension muscle actions at velocities ranging from 60–270°·s⁻¹.

It has also been hypothesized that neural factors contribute to the stretching-induced decrease in force, although the precise neural mechanisms have not been identified (3, 5, 7, 21). Fowles et al. (7) indicated that much of the decrease in force of the triceps surae, up to 15 minutes poststretching, was due to neural factors. Furthermore, Behm et al. (5) suggested that impaired muscle activation partially accounted for the stretching-induced decrease in force recorded from the quadriceps femoris muscles. A number of peripheral mechanisms have been proposed to explain the reduced muscle activation after stretching (3, 5, 7), including: (a) the autogenic inhibition of the Golgi tendon reflex, (b) mechanoreceptor and nociceptor afferent inhibition, (c) fatigue-induced inhibition, (d) joint pressure feedback inhibition due to excessive ranges of motion during stretching, and (e) stretch reflex inhibition originating from the muscle spindles. In addition, Avela et al. (3) suggested that a central nervous system (CNS) mechanism, such as “supraspinal fatigue,” may be responsible for the decreases in muscle activation and implied that decreases in force production from the unstretched contralateral limb would have supported this hypothesis. Unlike Avela et al. (3), however, the results of the present study showed the same pattern of stretching-induced decreases in force in both the stretched and unstretched limbs. Based on the hypothesis of Avela et al. (3), these findings suggest that the CNS may influence the decreases in force following an acute bout of static stretching.

PRACTICAL APPLICATIONS

The results of this study have implications for strength and conditioning coaches and women who perform static stretching prior to performance events. The stretching-induced decreases in strength may adversely affect the performance of athletes in sports that require high levels of force production. These findings, in conjunction with previous studies (12, 18, 19, 21, 22, 28, 29), indicated that static stretching impairs maximal force production. Strength and conditioning professionals should consider this before incorporating static stretching in preperformance activities. Future studies are needed to identify the underlying mechanisms that influence the time course of stretching-induced decreases in maximal force production for athletes and nonathletes across the age span.

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