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Stretch Could Reduce Hamstring Injury Risk During Sprinting by Right Shifting the Length-Torque Curve

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Abstract

Ruan, M, Li, L, Chen, C, and Wu, X. Stretch could reduce hamstring injury risk during sprinting by right shifting the length-torque curve. *J Strength Cond Res* 32(8): 2190–2198, 2018— It was hypothesized that static stretch would shift the length-torque curve to the right, which may reduce the risk of muscle strain injuries. The purpose of this study was to evaluate the acute effects of static stretching of hamstring (SSH) on the risk of hamstring injury during sprinting indicated by the shift of the length–torque relationship. Twelve female college athletes (age: 20.8 ± 0.7 years; height: 1.61 ± 0.05 m; body mass: 54.25 ± 4.22 kg) participated in this study. Subjects performed overground sprinting under 2 conditions: after warm-up with 4 × 30 seconds SSH or after warm-up without SSH. Three-dimensional kinematic and kinetic data and electromyography of biceps femoris long head (BFlh), rectus femoris, and vastus medialis were collected during testing. The maximum length of BFlh during late swing phase increased after SSH with large effect size and close to statistically significant ($p = 0.05$, $d = 1.22$), but the knee flexion torque at the peak length did not change significantly. Static stretching of hamstring significantly reduced peak values of both horizontal ($d = 1.46$) and vertical ($d = 1.79$) ground reaction forces, and BFlh's activation level during the preactivation (late swing) phase ($p = 0.05$, $d = 2.16$). The results indicated that the length of BFlh–knee torque relationship and the length of BFlh–hip torque relationships during the late swing phase and initial stance phase were shifted to the right after SSH, which may reduce risk of hamstring strain injuries. We suggest that preactivity static stretching should not be simply removed and participators should give priority to stretch muscles that are vulnerable to strain injuries.

Key Words angle–torque relationship, tension–length relationship, muscle strain injuries, biomechanics

Introduction

Hamstring strain injuries (HSIs), accounting for 37% of all muscle injuries (7), are the most prevalent injury in sports involving high-speed sprinting. Because of the great impact of HSIs, tremendous scientific efforts have been made to identify their potential risk factors (29). Several risk factors have been proposed, including nonmodifiable factors such as age (30) and previous injuries (30) and modifiable factors such as shortened optimum muscle length, strength imbalance (2), flexibility (13), and fatigue (29). Although the cause-and-effect relationships between these proposed risk factors and HSIs have not been established, it is proposed that greater flexibility may reduce the risk of muscle strain injuries by allowing muscles to absorb more energy during lengthening (46). At the same time, reduced hamstring flexibility was identified as a significant independent risk factor for HSIs in elite soccer players (12). Accordingly, pre-exercise stretching exercises are regularly recommended because of its acute effect on improving compliance of the muscle-tendon unit (MTU) (20).

However, the effects of pre-exercise stretching exercises on preventing muscle strain injuries have not been adequately studied (23). A recent review study (4) revealed that 8 studies showed some preventive effectiveness of stretching, whereas 4 showed no effect. It is notable that the majority of randomized trials showed some efficacy. There was some evidence in the literature providing explanations for why stretching could impact the risk of sustaining a muscle strain injury. Static stretching has been shown to increase tendon extensibility (18), which provides a "shock-absorber" mechanism that rapidly absorbs mechanical energy during rapid and forceful eccentric contraction (33). The increased

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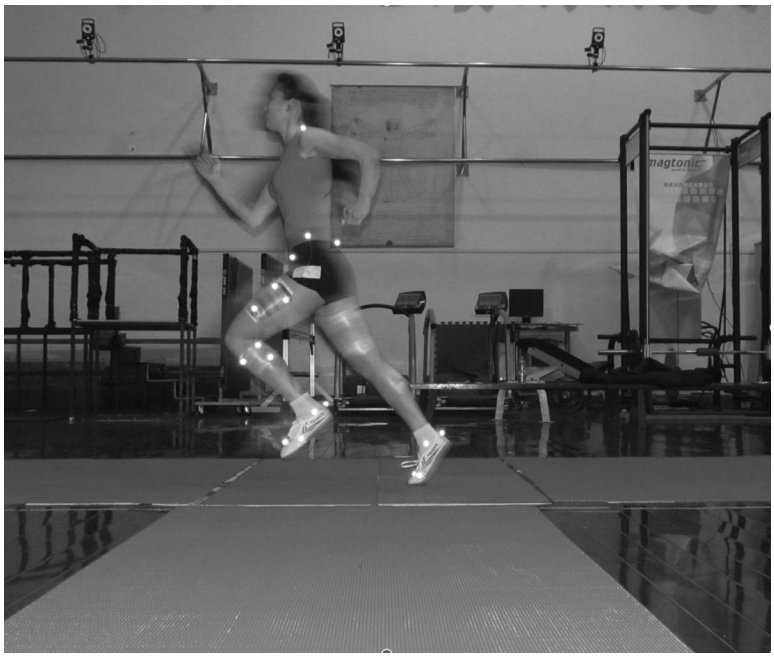

Figure 1. A synthetic rubber track was placed on the laboratory floor and the force platform.

compliance would shift the angle (length)-torque (tension) curve to the right (14,24). This rightward shift may contribute to minimizing eccentric contraction-induced muscle damage because of reduced sarcomere strain and limited myofibrillar disruption during eccentric contractions. Additionally, this shift of angle–torque relationship would allow greater force production at longer muscle lengths with enhanced ability to resist excessive muscle elongation (14,24). Hence, it was hypothesized that static stretching may decrease the susceptibility to a muscle strain injury because of the shift in the angle–torque relationship (23). Although the rightward shift of the angle–torque relationship was observed during slow concentric (24) or isometric contractions (14,24) after stretching, it remains a question whether the rightward shift of the angle–torque relationship would occur during sprinting after stretching because the effects of stretching are velocity specific (28) and joint angle specific (27). Because injuries to the long head of biceps femoris (BFlh) constitute over 80% of all HSIs (8) and BFlh produces both hip extension torque and knee flexion torque during running, (knee) angle–torque relationship measured in isokinetic contractions should be changed to BFlh length–hip torque and BFlh length–knee torque relationship during sprinting.

Although some authors (3) suggested that most hamstring injuries in sprinting occur while sprinting at or close to the maximum speed, no substantial data support this statement. Considering more than 70% of sprints in competitive soccer matches were performed over distance less than 10 m (21) and hamstring plays an important role in horizontal force production during sprint acceleration phase (26), 10 m was chosen as the sprinting distance in the present study. Therefore, the purpose of this study was to evaluate the acute effects of static stretching of hamstring (SSH) on risk of HSIs during sprint acceleration. Specifically, BFlh length–hip torque and BFlh–knee torque relationships during sprinting with and without SSH were compared. We hypothesized that the length–torque relationships would shift to the right during sprinting after SSH.

Table 1. Biomechanical variables during sprinting.*

Variables	Prestretching	Poststretching	p	Effect size
Length at IC (m)	0.450 ± 0.01	0.455 ± 0.01	0.165	1.22
Peak length (m)	0.465 ± 0.01	0.470 ± 0.01	0.075	1.22
Knee torque at peak length ($Nm \cdot kg^{-1}$)	−1.21 ± 0.13	−1.18 ± 0.13	0.423	0.57
Stance duration time (s)	0.14 ± 0.01	0.15 ± 0.01	0.009	2.45
Peak Fx (BW)	−0.58 ± 0.17	−0.49 ± 0.13	0.028	1.46
Peak Fz (BW)	3.18 ± 0.48	2.91 ± 0.21	0.05	1.79

*Peak Fx = peak value of horizontal ground reaction force during early stance phase; Peak Fz = peak value of vertical ground reaction force during early stance phase.

Methods

Experimental Approach to the Problem

A within-subject experimental design was used to evaluate the effects of SSH on risk of hamstring strain injury during sprinting. Subjects visited the

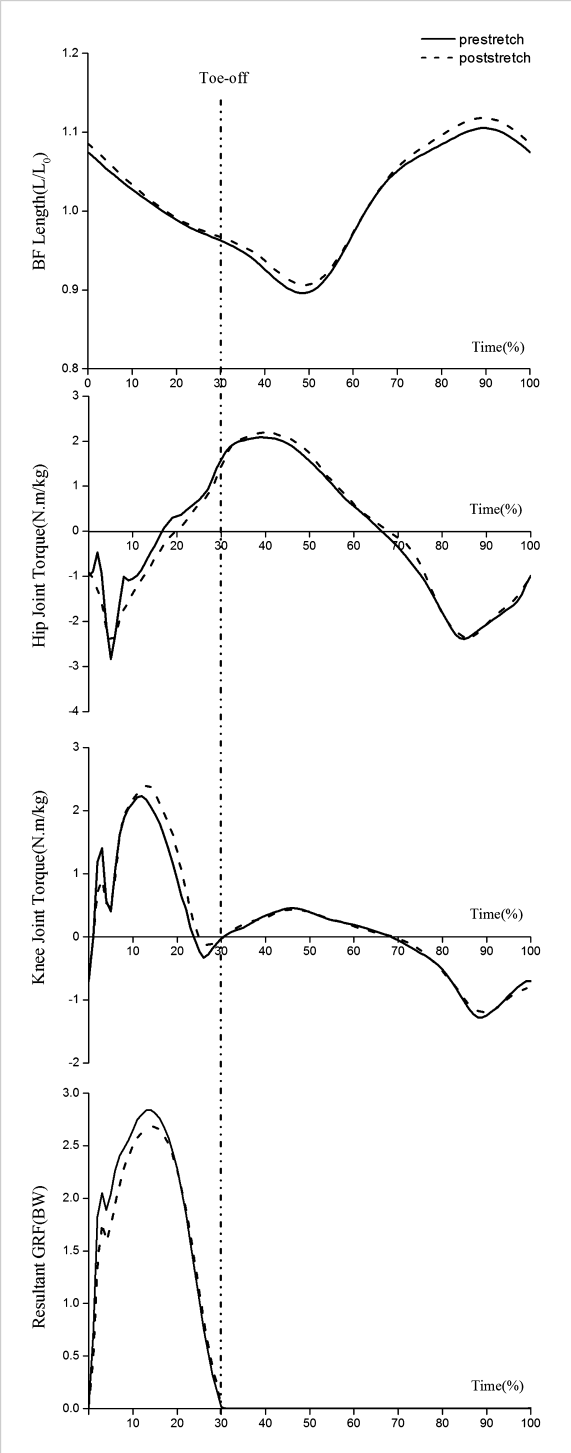

Figure 2. Hip torque, knee torque, BF-long head length relative to the resting length (standing), and vertical GRF during a running gait cycle (prestretch: 0.516 ± 0.03 seconds; poststretch: 0.523 ± 0.04 seconds). A positive hip torque represents flexion torque, and a positive knee torque represents an extension torque. BF = biceps femoris; GRF = ground reaction forces.

laboratory on 3 different days, with 48 hours between the 2 testing days. Day 1 was used for subjects' familiarization with testing procedures. Day 2 and day 3 were used for data collection under the 2 experimental conditions for sprinting: (a) warm-up with no stretch (prestretching condition) and (b) warm-up with SSH (poststretching condition). The order of the 2 experimental conditions was random balanced across all subjects: 6 subjects performed condition 1 first, and the other 6 subjects performed condition 2 first.

Subjects

Twelve female college athletes (mean ± *SD*, age: 20.8 ± 0.7 years; height: 1.61 ± 0.05 m; body mass: 54.25 ± 4.22 kg; personal bests in 100 m: 13.3 ± 0.65) who had received no less than 3 years of sports training were recruited to participate in this study. The participants with a history of lower limb injury were excluded. All participants had experience with static stretching exercises, but had no knowledge about the purpose or hypotheses of the current study. The Ethics Committee of Shanghai University of Sport approved the project, and participants signed informed consent forms before participation. Subjects wore spandex shorts, spandex shirts, and the same type of athletic shoes (cross-training shoes) provided by the laboratory during data collection.

Procedures

Before the data collection on testing days, wireless EMG electrodes (Trigno wireless, Delsys, Boston, USA) were attached on biceps femoris (BF), rectus femoris (RF), and vastus medialis (VM) of participants' dominant leg after the skin was shaved and then cleaned with alcohol. The dominant leg was determined based on the preferred jumping leg in a single-leg vertical jump. The rectangular (25 × 12 × 7 mm) electrodes were placed over the muscle belly aligned with muscle fiber orientation and were secured with strapping tape to minimize motion artifact.

Retroreflective markers (14 mm) were attached bilaterally on subjects' acromioclavicular joints, iliac crest, anterior superior iliac spines, posterior superior iliac spines, greater trochanters, medial and lateral epicondyles of the knee, medial and lateral malleoli, the first and fifth metatarsal heads, heels, and the second toes. Additional rigid plates with 4 markers were attached to bilateral thighs and legs. Full details regarding marker placement could be found in our previous study (35). Participants performed a static calibration trial with all markers presented. The calibration markers, including the greater trochanters, medial and lateral epicondyles of the knee, and medial and lateral malleoli, were then removed before warm-up. After the calibration trial, participants completed a 5-minute jogging on a treadmill as a warm-up, followed by performing 3 vertical jumps, with the highest jump used for EMG normalization (35). Stretch protocol was conducted, followed by sprinting testing. Participants

TABLE 2. Average EMG of BF, RF, and VM.*†

	Pre-stretching	Post-stretching	*p*	Effect size
BF_aEMG0	1.46 ± 0.88	0.86 ± 0.39	0.050	2.16
BF_aEMGT	0.96 ± 0.46	0.79 ± 0.56	0.361	0.81
RF_aEMG0	0.22 ± 0.15	0.18 ± 0.08	0.263	0.82
RF_aEMGT	0.55 ± 0.57	0.35 ± 0.19	0.319	1.15
VM_aEMG0	0.51 ± 0.24	0.44 ± 0.20	0.166	0.32
VM_aEMGT	0.89 ± 0.42	1.13 ± 0.46	0.261	0.54

*BF = biceps femoris; RF = rectus femoris; VM = vastus medialis.

†aEMG0 represents preactivation phase (from 100 ms before IC–IC). aEMGT represents stance phase (from IC to toe-off).

started from 10 m away from the force platform and ran forward with the maximum effort without targeting the force platform. Trials were counted as successful when participant's dominant foot stepped on the force platform. Subjects performed 3 successful trials within 5–10 minutes after the stretch. All tests on day 2 and day 3 were performed in the afternoons of October. The room temperature was controlled by air conditioning and was kept at 22° C during all tests.

Stretching Protocol. We used a passive static straight-leg raise to stretch the participants' hamstrings. Participants lay supine on a mat with the contralateral hip and knee stabilized in full extension. The investigator then pushed the stretching leg and took her into full knee extension and maximum hip flexion until the participant reported discomfort. All stretches were held for 30 seconds and repeated 4 times with a 10-second rest between the stretches (28,35). The procedure was performed firstly on the dominant leg and then on the nondominant leg. In the no-stretch protocol, participants rested for 5 minutes before they performed the sprinting task.

Date Collection. The surface EMG signals were collected using wireless Ag electrodes with a parallel bar arrangement (contact area 1 × 10 mm, 10 mm interelectrode distance) (Trigno wireless) and preamplified closed to the detection site (Common Mode Rejection Ratio, CMRR > 80 dB, bandpass = 20–450 Hz). All acquired EMG data were recorded using Delsys EMG works acquisition software at a sampling rate of 2,000 Hz.

In addition to EMG system, a 16-camera motion analysis system (200 Hz; Vicon Motion Analysis, Oxford, United Kingdom) and a force platform (1,000 Hz; Kistler Instruments, Winterthur, Switzerland) were employed to simultaneously record the EMG signals, the 3D kinematics, and ground reaction forces (GRF), respectively, during the testing (Figure 1).

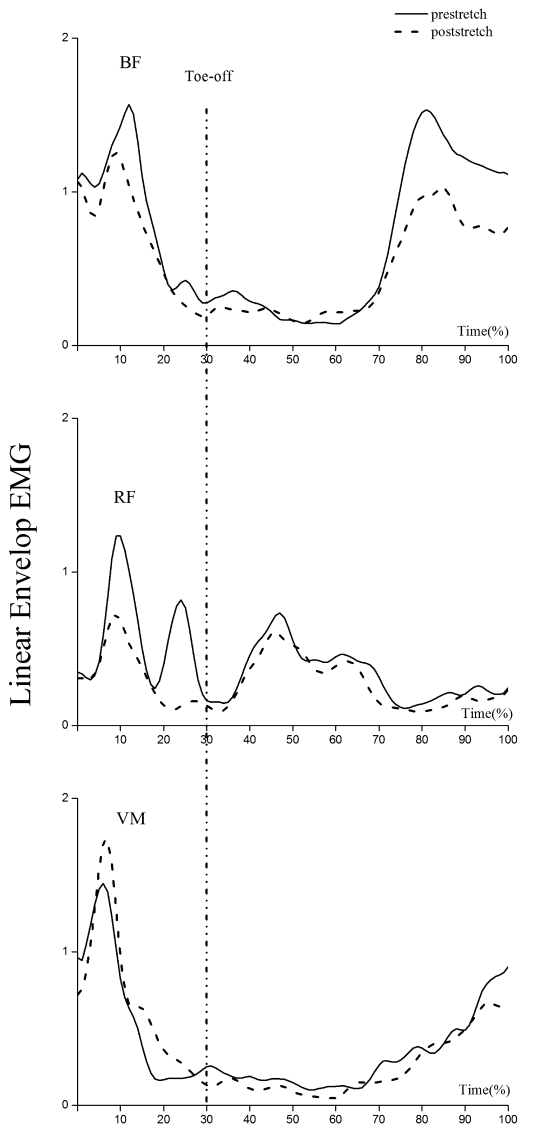

Figure 3. Linear envelop EMG of BF, RF, and VM. BF = biceps femoris; RF = rectus femoris; VM = vastus medialis.

Data Reduction. The raw data were processed with a 3D biomechanical analysis suite, Visual 3D (C-Motion, Germantown, MD, USA) to compute the 3D kinematic and kinetic variables, and the EMG variables. The 3D marker coordinates and GRF signals were smoothed using a fourth-order Butterworth low-pass filter with cutoff frequencies of 10 and 100 Hz, respectively. The instantaneous horizontal velocity of the center of mass at the initial foot-contact (IC) on the force platform during sprinting was calculated to quantify speed. The fastest trial for each participant was reduced for analysis. The 3D angular kinematics was computed using a Cardan sequence (X-Y-Z), and a right-hand rule was used to determine the polarity

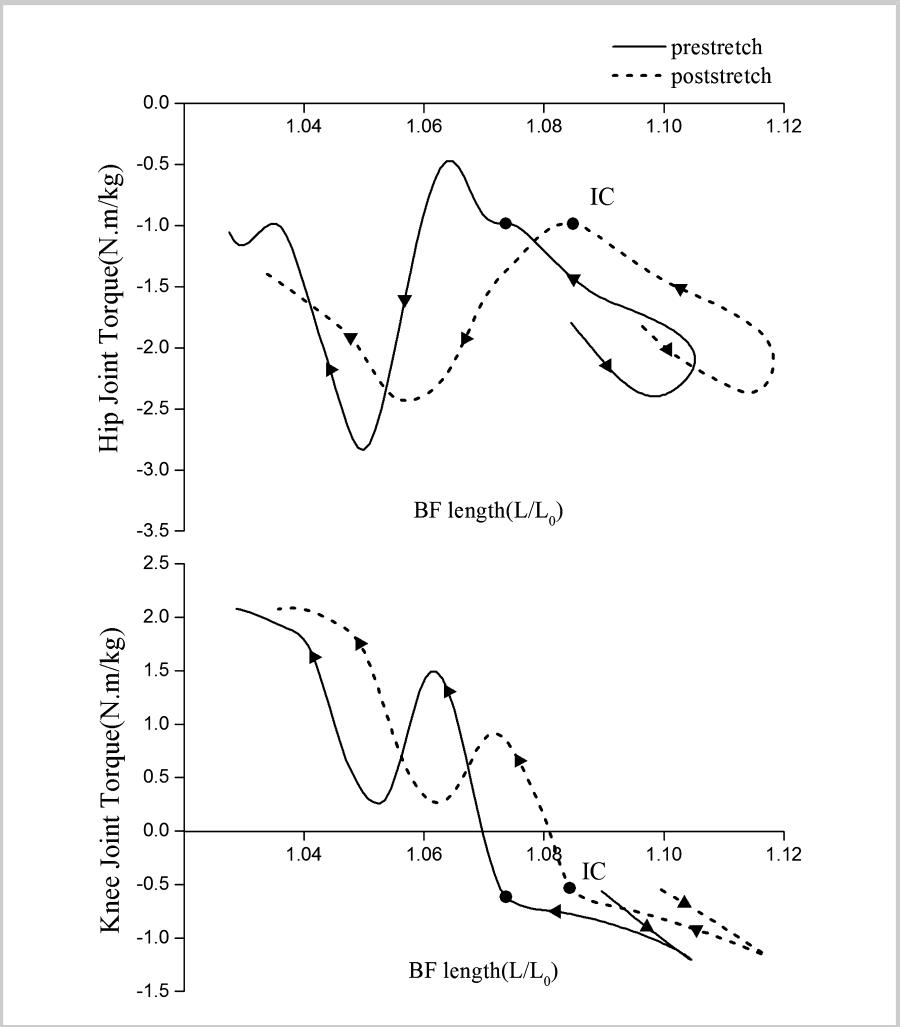

Figure 4. BF length–knee torque relationship, and BF length–hip torque relationship from 100 ms before IC to 50 ms after the initial contact. A positive hip torque represents flexion torque, and a positive knee torque represents an extension torque. BF = biceps femoris; IC = initial foot-contact.

of the angular variables. An inverse dynamics approach was used to calculate the joint torque. The joint torque was normalized to the body mass, and the GRF were normalized to the body weight. The 3D marker coordinates were processed with OpenSim (NCSRR, Stanford, CA, USA) to calculate the kinematics of BFlh. Muscle-tendon unit lengths of BFlh are determined solely by the positions of muscle origins and insertions.

Raw EMG signals were full-wave rectified and filtered using a moving root-mean-squared (RMS) filter with a window size of 50 ms. The maximum RMS values of the EMG signal of each of the 4 muscles in vertical jump testing were used to normalize the EMG of the respective muscle during sprinting. The normalized EMG signals were then integrated into 2 time intervals: from 100 ms before foot contact for the preactivation phase or late swing phase, from foot contact to toe-off for the stance phase. The integrated EMGs were further divided by the respective time intervals to obtain average EMG (aEMG) values.

Statistical Analyses

The results were expressed as mean ± *SD*. A paired-samples T-test was applied to compare the results with and without SSH. Statistical significance was set as $p \leq 0.05$. Statistical analysis was performed using SPSS software (version 19.0; SPSS, Chicago, IL, USA). Effect size (d) was evaluated based on Cohen (5) for paired t-test. Where $0.2 < d < 0.5$, as small, $0.5 < d < 0.8$, as medium, and $0.8 < d$, as large.

Results

The mean running speed was not significantly changed after SSH (prestretching: 5.84 ± 0.18; poststretching: 5.79 ± 0.23, $p > 0.05$). The maximum length of BP, which occurred during the late swing phase (Table 1 and Figure 2), increased after SSH with large effect size and close to statistically significant ($p = 0.05$, $d = 1.22$). The knee flexion torque at the peak length reduced after SSH with medium effect size also not statistically significant ($p > 0.05$, $d = 0.57$, Table 1 and Figure 2). Peak values of both horizontal ($d = 1.46$) and vertical ($d = 1.79$) GRF during the initial stance phase were significantly reduced ($p < 0.05$) after SSH, and the total duration time was significantly increased ($p < 0.05$, $d = 2.45$, Table 1).

Static stretching of hamstring significantly ($p = 0.05$, $d = 2.16$, Table 2 and Figure 3) reduced BF's activation level during the preactivation phase, but not the stance phase ($p > 0.05$, $d = 0.81$, Table 2 and Figure 3). Rectus femoris ($p > 0.05$, $d = 0.81$ and 1.15 for preactivation and stance phase, respectively, Table 2 and Figure 3) and VM's ($p > 0.05$, $d = 0.32$ and 0.54 for preactivation and stance phase, respectively, Table 2 and Figure 3) activation level did not change significantly regardless of phase.

Changes in BFlh length–hip joint torque and BFlh length–knee joint torque relationship during the late swing (100 ms before IC–IC, about 20% cycle time) early stance phase

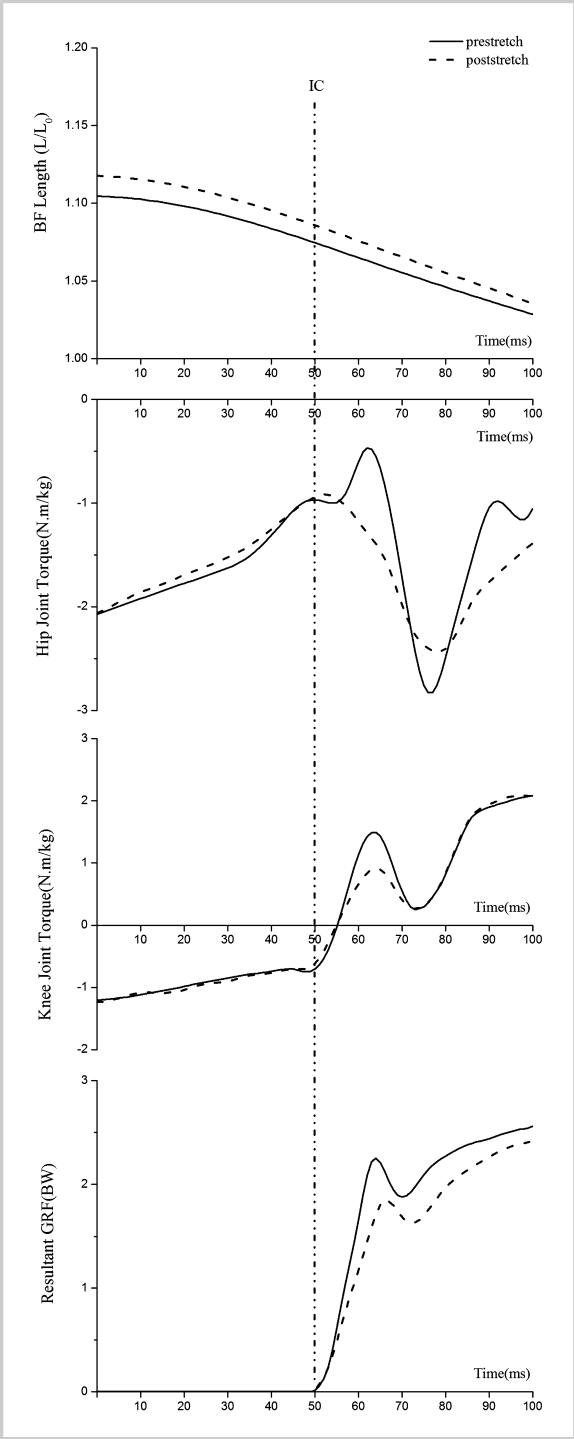

Figure 5. Biceps femoris long head length, hip joint torque, knee joint torque, and resultant GRF during swing-stance transition phase (from 50 ms before IC to 50 ms after IC). GRF = ground reaction forces; IC = initial foot-contact.

(from IC 50 ms after IC, about 10% cycle time) are shown in Figure 4. Both BFlh length–hip and BFlh length–knee torque relationships showed right shifts after SSH when BFlh was longer than 1.05 L/L_0, which corresponds to the late swing phase and the first 5% cycle time (about 25 ms) of stance phase. The very brief lengthening period observed at the beginning of the selected period accompanied with extension torque at hip joint and flexion torque at knee joint. Biceps femoris long head was shortening during most of this period. This shortening was accompanied with hip extension torque throughout and with initial knee extension torque but changed to knee extension torque soon after the IC.

Prestretching and poststretching comparisons in BF length, hip joint torque, knee joint torque, and resultant GRF during the swing-stance transition phase (50 ms before IC to 50 ms after IC) are shown in Figure 5.

Discussion

The main observations in this study were that (a) the peak knee flexion torque during the late swing phase of sprinting did not change significantly but the BF's length at which the peak knee flexion torque occurred increased after SSH; (b) Peak values of both horizontal and vertical GRF during the initial stance phase were significantly reduced ($p < 0.05$) after SSH; (c) The length-torque curves observed in this study indicated a rightward shift in both hip joint and knee joint after SSH was occurred in both late swing phase and initial stance phase, which supported our hypothesis. To the best of our knowledge, this is the first study to show that a right shift in the length (angle)–torque relationship after SSH could also be occurred during sprinting.

Static stretching has a neural component in which a decrease in the amplitude of the EMG during maximal voluntary contraction after stretching has been observed (6). The results of present study also confirmed this neural component would occur in the poststretching dynamic movement. Because less activated muscle would absorb less energy before it was stretched to failure (10), the neural component of stretching may have a negative impact on preventing HSIs. On the other hand, static stretching also has a mechanical component in which the compliance of MTU increased after stretching (20,24). It was proposed that the rightward shift in length–tension relationship was attributed to this mechanical component (24). More specifically, this mechanical component may include increased muscle fascicle resting length (40) and reduce tendon stiffness (18). The increased muscle resting length may partially offset the negative impact on presenting HSIs associated with the neural component, and reduced tendon stiffness may even created an advantageous situation for counteracting potentially injurious muscle elongations.

There is a debate in the literature regarding which specific mechanical parameter that causes muscle strain injury (42). Although many animal studies (19) supported that the strain, not force, is the direct cause of muscle damage, Warren et al. (45) reported that the muscle injury is related most closely to the peak force during the lengthening. This inconsistency may be due to the different initial length during eccentric

contraction (16). The muscle initial lengths of 0.85 or 0.9 L_0 used in Warren et al's study may be applicable for HSIs occurred during high-speed sprinting. It was proposed (34) that the excessive strain would not be the direct cause for HSIs during sprinting because they likely occur near optimal lengths rather than 25 or 12.5% strain beyond the optimal lengths applied in animal studies (19). On the other hand, the force applied on hamstring during sprinting may reach 10 times the body weight or 50% larger than the maximal isometric force (39). Therefore, it is reasonable to hypothesize that the extreme force could be the direct cause for HSIs.

Besides debate on specific mechanical parameter in which phase hamstrings are most susceptible to injury is also disputed, some studies (41,48) suggested hamstrings are most susceptible to injury during the late swing phase of sprinting because an eccentric contraction of the hamstring was observed during the late swing phase. The results of the present study also confirmed that the eccentric contraction of the hamstring occurred during the late swing phase. However, it might not be correct to assume that the hamstrings are at their most susceptible to injury in the late swing phase of sprinting just because the MTU of hamstring is stretched during this phase (34). During high intensity dynamic movements, the MTU is lengthened but the muscle fibers may remain isometric or may even shorten during the eccentric contraction (15). As running speed increases, there is a requirement during the swing phase for hamstring increased force production to counter the propulsion contraction of the hip flexors and quadriceps muscles. Eventually, whether the muscle fibers may undergo lengthening or not depends on the interaction between muscle force and external force. A recent study showed that forced lengthening of fully activated, isometrically contracted muscle could disrupt myofibrillar ultrastructure, whereas even small, low-velocity shortening of muscle fiber preceding a stretch could protect against stretch-induced muscle damage (37). Therefore, stretching exercise may reduce the risk of hamstring strain injury during the swing phase through 2 ways: (a) reduced tendon stiffness could increase the possibility for muscle fibers to undergo some shortening at the expense of a lengthening tendon during the swing phase; (b) the rightward shift in the length-tension curve could decrease the possibility for muscle fiber to undergo some lengthening during the late swing phase because of enhanced force production at longer MTU length.

Other scholars (31,39) suggested the initial stance phase (5% of stance phase) (39) was the highest-risk period for HSIs because the hamstrings have to work hard to counteract high knee extension torque and hip flexion torque produced by knee extensors, hip flexors, and the ground reaction force. During the initial stance phase, in addition to encountering knee extensors, the hamstrings have to generate knee flexion torque to counteract the effect of GRF and generate hip extension torque to push the ground backwards (39). Orchard (31) argued that the GRF is the likely cause for HSIs in sprinting based on 2 reasons: (a) the GRF causes other tissue injuries in running; (b) the risk of muscle strains is very low during open chain activities. Other scholars (47) argued that HSIs are not likely occurred in the stance phase because the hamstrings are in a concentric contraction. The results of the present study also confirmed that a concentric contraction of the hamstring was occurred during the stance phase. However, a sudden increase in hip joint torque and knee joint torque was observed after IC (Figure 5). Therefore, it is reasonable to predict that a concentric contraction (MTU) we observed during uninjured trials may change to an eccentric contraction if the joint torques were increased further. Sun et al. (39) argue that knee torque and hip torque during initial stance phase may have been underestimated because a sudden increase in hip and knee torque may be removed as a noise spike. More importantly, we do not know whether the muscle fibers of hamstring were lengthening or not during the initial stance phase. Ishikawa and Komi (17) found fascicle length of gastrocnemius was shorten and then lengthened because of GRF loading during stance phase of sprinting. It is very likely that the muscle fibers of BFlh are also working with similar patterns. The results of the present study showed that the peak GRF in the early stance phase was reduced significantly after SSH and accordingly, the peak value of joint torque at hip and knee was also reduced significantly. In addition, force productions of BF at longer muscle lengths were augmented. Therefore, the results of the present study suggested that SSH may be able to reduce the risk of HSIs occurred during the initial stance phase.

This rightward shift has also been observed after eccentric strength training (25), which has been shown to reduce the risk of subsequent HSIs (2). On the contrary, the leftward shift has been associated with age (22) and previous hamstring injury (32), both of which have been recognized as major risk factors for HSIs (9). The high hamstring injury recurrence may be attributed to a shorter MTU length for active tension in the previously injured muscle (11). A shift in the length–tension relationship could be a result of training or it could reflect the presence of residual scar tissue at the MTU (11). Eccentric strength training and stretching after HSIs may effectively restore optimum MTU length for active tension to normal length, thereby reducing the risk of re-injury (11). It has been proposed that the adaptation process during a period of eccentric training involves additional sarcomeres in series in muscle fibers. In this regard, SSH stretching would be a plausible acute intervention although eccentric training would be an obvious chronic intervention (23).

The current study has several limitations. Although the knee flexion torque may provide some information about force production of the hamstring when no significant EMG changes in RF and VM occurred after SSH, no direct assessment of hamstring strength and tendon stiffness was

applied in the current study. Additionally, changes in fiber lengths, tendon stiffness, and pennation angle after SSH were not measured in the current study and we evaluated the injury risk based on uninjured trials. Certainly, further studies with direct assessment of hamstring force and muscle fibers and tendon length in vivo are needed to determine the mechanisms associated with SSH impact on HSIs. The participants recruited in this study were female recreational athletes and the speeds were much lower compared with male sprinters in a previous study (7.77 ± 0.11) (48), but the speeds measured in the present study were already within the scope of sprinting for elite female soccer players during competitive matches (sprinting $> 5.4\ m \cdot s^{-1}$) (21). Although male athletes were not recruited in this study, we believe the results of this study may also be generalized to male athletes in considering female athletes may be more tolerant of static stretching than male athletes (44). However, cautions should be given when generalizing the results to sprint races because the crouch start would produce greater running velocity, maximal power, and ground reaction force during the acceleration phase compared with the standing start (38).

In summary, we have observed a rightward shift of length–torque relationship during the late swing phase and a reduced peak GRF during the early stance phase after effective static stretching. A rightward shift of torque–length relationship may increase the possibility for muscle fibers to shorten before eccentric contraction and decrease the possibility for muscle fiber being overstretched during late swing phase. Hamstring strain injuries are thought to occur with the hamstring in a lengthened position; the rightward shift in the length-tension curve would be advantageous for counteracting potentially injurious muscle elongations (23). A reduced peak GRF during the initial stance phase may decrease demand on force production of hamstring. Overall, static stretch may reduce the risk of hamstring injuries during the late swing and the initial stance of sprinting.

Practical Applications

Because of stretch-induced muscle force and power deficit, the American College of Sports Medicine (1) has suggested that static stretching during the warm-up period should be removed from sport activities that heavily relied on muscular strength and power. However, stretch-induced muscle force and power deficit does not necessarily reduce athletic performance. On the contrary, our previous study (35) showed that antagonist stretching of the hamstrings significantly enhanced jumping performance. And the present study also showed that SSH did not reduce sprinting speed. More importantly, the present study provided evidence that SSH could reduce hamstring injury risk during sprinting by right shifting the length-torque curve. Therefore, we suggest that from the perspective of injury prevention and performance, static stretching during the warm-up period should not be simply removed from sport activities that heavily relied on muscular strength and power. Even if stretch-induced muscle force deficit may reduce some sports performance, practitioners should give priority to stretch muscles that are vulnerable to strain injuries and keep it in mind that effects of static stretch may only last 10–20 minutes (36) and short duration stretching (e.g., 2 sets of 15 seconds) may not sufficient to alter the properties of the MTU (43).

Acknowledgments

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