THE KINEMATICS OF OVERGROUND SPRINTING IN TRACK AND FIELD ATHLETES WITH PREVIOUS HAMSTRING INJURIES

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The purpose of this study was to examine the kinematic characteristics of lower extremity and long head of the biceps femoris (BF ih) muscle length during overground sprinting in track and field athletes with previous unilateral hamstring strain injuries. Ten male college sprinters with a history of hamstring injury performed a maximum effort sprint on an athletic track. Three-dimensional kinematic data were recorded during sprinting, and the hip and knee joint angles and musculotendon length of the biceps femoris muscle were calculated. The previously injured limb displayed delayed peak hip flexion and increased knee flexion compared with the uninjured limb, placing the BF ih muscle at decreased length during the terminal swing phase of sprinting.

KEYWORDS: sprinting, hamstring strain injury, previous injury, hip, knee, musculotendon length.

INTRODUCTION: Hamstring strain injury is extremely common in many sports, with high reinjury rates (Orchard, Seward, & Orchard, 2013). Most acute hamstring strains occur during sprinting and involve the long head of the biceps femoris (BF ih), whereas the semitendinosus and semimembranosus muscles are less frequently injured (Askling, Tengvar, Tarassova, & Thorstensson, 2014). A history of hamstring strain injury has been reported as a strong predictor of future injury (Hagglund, Walden, & Ekstrand, 2006). Several previous studies have described running kinematics (Lee, Reid, Elliott, & Lloyd, 2009) and activation patterns (Daly, Persson, Twycross-Lewis, Woledge, & Morrissey, 2016; Silder, Thelen, & Heiderscheit, 2010) of previously injured limbs. One study found that previously injured athletes had significantly reduced BF ih muscle activation ratios with respect to the ipsilateral gluteus maximus muscle in the late swing phase of treadmill running (Daly et al., 2016). They also suggested that the decrease in the BF ih muscle activity may have contributed to the increased anterior pelvic tilt and hip flexion occurring during late swing, resulting in increased hamstring length. On the other hand, Silder et al. (2010) did not find any significant differences in activation patterns and musculotendon length of the BF ih muscle during treadmill sprinting between previously injured and uninjured limbs. Some of this disagreement may stem from differences in methodological factors. Considering the mechanisms of the hamstring strain injury occurring during high-speed or high-intensity situations such as sprinting (Brooks, Fuller, Kemp, & Reddin, 2006), an investigation should be conducted in athletes performing overground sprinting at submaximal sprinting speed. Therefore, the purpose of this study was to examine the kinematic characteristics of the lower extremity and the BF ih muscle during overground sprinting in track and field athletes who had returned to sport following unilateral hamstring strain injury.

METHODS: Ten male college sprinters (mean age, 19.9 ± 0.3 years; 172.0 ± 3.9 cm; 65.9 ± 7.1 kg) with a history of unilateral hamstring strain injury were recruited. All participants had already returned to practice and had been participating in track and field at the time of this study. The time period after injury was 2-61 months, with a time to return to sports of 1-6 months.
The measurement area was set on an athletic track. After sufficient warm-up, each participant performed a maximum effort sprint from the starting line set approximately 40 m from the centre of the measurement area with attached passive markers. Three-dimensional kinematic data of 34 reflective markers were recorded at 200 Hz using a 12-camera passive marker system (MAC3D system, Motion Analysis Corporation, Santa Rosa, CA, USA). Reflective markers were placed on the upper and lower extremities of each subject using a modified Helen Hayes marker set (Kadaba, Ramakrishnan, & Wootten, 1990) for a total of 29 anatomical landmarks.

A stride was defined as the time from ground contact of the foot to the next ground contact of the same foot. The sprinting velocity was calculated by computing the horizontal speed of the centre of mass during a sprinting gait cycle. A single sprinting gait cycle for each limb was analysed. A three-dimensional musculoskeletal model comprising 53 bone segments and 155 degrees of freedom (nMotion musculos; NAC Image Technology, Inc., Japan) was used for the calculation of the hip and knee joint angles and musculotendon length of the BFlh muscle. The musculoskeletal modelling and inverse kinematics calculation were based on a previous study (Higashihara, Nagano, Takahashi, & Fukubayashi, 2015). The BFlh musculotendon length was normalised to length in the upright posture. The data from the sprinting gait cycle were resampled to 101 points (representing the gait cycle from 0 to 100% in 1% increments) by interpolation (IGOR Pro 4.04J; WaveMetrics, Inc. USA). The sprinting motion was divided into four phases: the stance phase, beginning at foot strike and ending at toe-off; the early-swing phase, beginning at toe-off and ending at maximum knee flexion during swing; the mid-swing phase, beginning at maximum knee flexion during swing and ending at maximum hip flexion; and the late-swing phase, beginning at maximum hip flexion and ending at foot strike.

Differences in the hip and knee joint angles and BFlh musculotendon length between limbs at each time point of the sprinting gait cycle (% of the gait cycle) were determined using two-way repeated measures analysis of variance (ANOVA). Bonferroni’s post hoc analysis was conducted if ANOVA showed statistically significant main or interaction effects. Effect sizes (ES) were calculated using a partial correlation ratio (partial $\eta^2$). Statistical significance was defined as $p < 0.05$.

**RESULTS:** The mean sprinting velocity was 9.4 ± 0.2 m/s. Statistically significant interaction effects were found in the hip and knee angles (hip: $F = 3.772$, $\eta^2 = 0.295$, $p < 0.001$; knee: $F = 2.369$, $\eta^2 = 0.208$, $p < 0.001$). Hip flexion angle was significantly lower during mid-swing (at 59–70% of the sprinting gait cycle, maximum difference $5^\circ$, $p < 0.05$) and significantly higher before foot contact (at 97–100% of the sprinting gait cycle, maximum difference $4^\circ$, $p < 0.05$) in the previously injured limb, compared with the uninjured limb (Figure 1(a)). The time of the peak hip flexion of the previously injured limb (77.6 ± 2.6% gait cycle) occurred significantly later than that of the uninjured limb (75.9 ± 3.0% gait cycle, $\eta^2 = 0.86$, $p < 0.01$). The knee flexion angle was significantly higher in the previously injured limb than in the uninjured limb at 78–86% of the sprinting gait cycle (maximum difference $6^\circ$, $p < 0.05$, Figure 1(b)). Statistically significant interaction effects were found in the musculotendon length of the BFlh ($F = 2.949$, $\eta^2 = 0.247$, $p < 0.001$). The normalized BFlh length showed a significant reduction in the previously injured limb compared with that of the uninjured limb during the late-swing phase (at 81-86% of the sprinting gait cycle, maximum difference 3%, $p < 0.05$, Figure 1(c)).

**DISCUSSION:** The results of the present study showed that the previously injured limb displayed a delay of peak hip flexion during mid-swing and increased knee flexion during the terminal swing compared with those in the uninjured limb, which may result in decreased BFlh muscle length during the terminal swing of sprinting. It has been shown that athletes who sustained a previous unilateral hamstring injury tend to develop peak knee flexion torque at a greater knee flexion angle (i.e., shorter muscle length) during isokinetic strength testing compared with the uninjured limb (Brockett, Morgan, & Proske, 2004). Therefore, the interlimb difference observed in this study may reflect a shorter optimal fibre length of the

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previously injured hamstring muscles. The decreased BFih length may be due to a reduction in the lengthening capacity of the previously injured hamstring muscle. Athletes may unconsciously limit lengthening of the hamstring muscles by controlling knee extension during the terminal swing. These kinematic characteristics were observed in athletes following rehabilitation and return to full competition and training. It has been speculated that the neuromuscular inhibition of voluntary hamstring activation occurs following hamstring strain injury and that this inhibition has a detrimental effect on hamstring recovery by limiting hamstring exposure to eccentric stimuli at long muscle lengths during rehabilitative exercise (Fyfe, Opar, Williams, & Shield, 2013). Neuromuscular inhibition at longer muscle lengths may impede the rehabilitation process by limiting adaptations within the previously injured muscle. Future work should seek to clarify whether activation deficits of the hamstring muscles are observed and whether these are a risk factor for hamstring strain re-injury.

![Graphs showing hip flexion angle, knee flexion angle, and biceps femoris muscle length](image)

**Figure 1:** Mean and standard deviation of the hip flexion angle (a), knee flexion angle (b), and the biceps femoris muscle length normalised to the lengths in the upright posture (c) during the sprinting gait cycle. Asterisks denote values that were significantly different between the uninjured limb (dashed line) and injured limb (solid line) (p < 0.05).
CONCLUSION: In this study, we examined the kinematic characteristics of the lower extremity and BFlh muscle length during overground sprinting in athletes with previous unilateral hamstring strain injuries. Significant asymmetries in sagittal hip and knee movement were detected in the injured limb during the terminal swing, likely placing the hamstring muscles at decreased length during this phase of sprinting. The present study provides a better comprehension of kinematic characteristics of the lower extremity and hamstring muscles during sprint performance following hamstring strain injury. This information will contribute to rectification rehabilitation strategies, reducing injury/reinjury risk.

REFERENCES


ACKNOWLEDGEMENTS: The authors thank NAC Image Technology, Inc. for providing the musculoskeletal modelling software. This study was funded by a research fellowship grant provided by the Japanese Society for the Promotion of Science for Young Scientists (grant number 11J00261).