A prospective study of iliotibial band strain in runners

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Abstract

Background. Iliotibial band syndrome is the leading cause of lateral knee pain in runners. It is thought that pain develops from strain on the iliotibial band due to friction of the iliotibial band sliding over the lateral femoral epicondyle. The purpose of this study was to investigate mechanical strain in the iliotibial band as a possible causative factor in the development of iliotibial band syndrome.

Methods. From a large prospective study, female runners who incurred iliotibial band syndrome during the study were compared to a control group who incurred no injuries. Strain, strain rate and duration of impingement were determined from a musculoskeletal model of the lower extremity.

Findings. The results indicated that the iliotibial band syndrome subjects exhibited greater strain throughout the support period, but particularly at midsupport compared to the control group. Strain rate was significantly greater in the iliotibial band syndrome group compared to the control group and was greater in the involved limb of the iliotibial band syndrome group compared to their contralateral limb. However, there were no differences in the duration of impingement between the groups.

Interpretation. This study indicates that a major factor in the development of iliotibial band syndrome is strain rate. Therefore, we suggest that strain rate, rather than the magnitude of strain, may be a causative factor in developing iliotibial band syndrome. The effect size (>0.5) indicated that strain rate may be biologically significant in the etiology of iliotibial band syndrome.

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1. Introduction

Iliotibial band (ITB) syndrome is the leading cause of lateral knee pain in runners accounting for up to 12% of all running-related injuries (Taunton et al., 2002). The ITB is a sheet of connective tissue originating at the iliac crest and terminating at Gerdy's tubercle and the fibular head. The ITB runs down the lateral thigh and knee and passes over the lateral femoral epicondyle (LFE). Proximally, it acts as a lateral hip stabilizer resisting hip adduction. It originates in the fascial components of the gluteus maximus, gluteus medius, and tensor fasciae latae muscles. Distally, the ITB is attached to the supracondular tubercle of the femur, the lateral intramuscular septum and has fibers that articulate with the patella. It then courses down the lateral thigh and has its most distal attachment on Gerdy's tubercle of the tibia. Because of these attachments, movements such as increased femoral adduction and knee internal rotation, will likely lead to increased strain and tension on the ITB. It is believed that strain on the ITB and friction of the ITB sliding over the LFE is the cause of ITB syndrome (Orava, 1978; Noble, 1980).

Biomechanical factors that result in increasing the strain of the ITB are thought to contribute to the development of this injury. While a number of studies have investigated the kinematics of ITB syndrome (Messier et al., 1995; Orchard et al., 1996; Noehren et al., 2007), the relationship between running mechanics and ITBS is not well understood. Orchard et al. (1996) examined sagittal plane mechanics of

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runners with ITBS and reported no differences in sagittal plane knee motion between the injured and uninjured legs of runners with ITB syndrome. Messier et al. (1995) reported that runners with ITB syndrome exhibited twice the peak rearfoot motion compared to controls. However, Noehren et al. (2006) found that runners with a history of ITB syndrome exhibited decreased peak rearfoot eversion compared to healthy control subjects. These authors also reported that knee internal rotation was significantly greater in the ITB syndrome group as compared to the controls.

The symptoms associated with ITB syndrome appear as lateral knee pain and thus research has directed attention to knee mechanics. ITB syndrome has been associated with lateral knee pain that occurs just after heel strike at approximately 20° of knee flexion. In addition, ITB syndrome has been reported to be exacerbated with downhill running (Noble, 1980). Compared to level running, downhill running is associated with landing with an extended knee position and moving through greater knee flexion excursion. In addition, an impingement zone is thought to occur between 20° and 30° of knee flexion. The impingement zone is thought to result from the interaction of the ITB and the LFE. In this range, the distal fibers of the ITB are believed to compress with and slide over the LFE. However, differences in sagittal plane knee motion in patients with ITB syndrome have not been found (Orchard et al., 1996).

Two types of studies can be conducted when investigating the causative factors associated with injury. A retrospective study compares subjects that currently have the condition under investigation to healthy controls. While this is an acceptable design, a cause–effect relationship cannot be ascertained. A prospective study is one in which measures are determined before individuals obtain the injury and, once the injury is incurred, are compared to a group who did not obtain the injury. Thus, in a prospective study, causative factors may be determined.

The majority of studies on ITB syndrome are retrospective in nature. While these studies can establish association, prospective studies are needed to determine cause and effect. It has been suggested that strain in the ITB and factors associated with strain such as strain rate can result in injury (Miller et al., 2007). Animal models of tissue injury have utilized the magnitude of strain as an indicator of injury potential (e.g. Lieber and Friden, 1993, 1999; Brooks and Faulkner, 2001).

Noehren et al. (2007) suggested that kinematic parameters such as greater hip adduction and knee internal rotation would lead to greater ITB strain. In this paper, we used the kinematic data from these same subjects in a musculoskeletal model to predict ITB strain and impingement. Therefore, the purpose of this prospective investigation was to compare the pre-existing strain, strain rate and duration of impingement in the ITB between female runners who ultimately developed ITB syndrome to healthy

controls who developed no injuries. In addition, we compared these parameters of the involved and contralateral limbs of the ITB syndrome subjects and the right and left limbs of the control subjects. We hypothesized that runners who developed ITB syndrome will exhibit greater strain throughout the stance period of running, but particularly at touchdown and maximum knee flexion. In addition, they will exhibit greater strain rate and greater duration of impingement compared to the control subjects. Also, we expected that the contralateral limb values of the ITB syndrome group will be less than the involved limb, and equal to those of the healthy controls.

2. Methods

2.1. Subjects

Subjects were part of a large ongoing prospective investigation of lower extremity injuries in female runners. To be included in the study, all subjects ran a minimum of 20 miles a week, were between the ages of 18–45, and were free from any injuries at the time of data collection. Upon entry into the study, a detailed subject injury history was taken. Subjects ($n = 400$) were then followed monthly by e-mail for 2 years and reported any running-related injuries and monthly mileage.

An *a priori* power analysis, (beta = 0.80, alpha = 0.05, effect size = 0.8) was performed using pilot data collected in our laboratory (Ferber et al., 2003). The sample size calculation was based on the following variables: (1) peak rearfoot eversion; (2) rearfoot excursion; (3) peak knee internal rotation; and (4) peak tibial internal rotation. This procedure suggested that a minimum of 14 subjects per group would be needed to adequately power the study. Subsequently, 17 runners, diagnosed by a medical professional (i.e. Physicians, Physical Therapists, or Athletic Trainers), had developed ITB syndrome since starting the study. Subjects were excluded if they had any previous hip or knee injuries. These subjects were assigned to the ITB syndrome group. Seventeen mileage and age matched runners with no previous history of knee or hip injuries and who sustained no injuries during the follow-up period were chosen for the control group. At the time of the data collection, all subjects were asymptomatic exhibiting no pain. Table 1 presents the characteristics on which the groups were matched.

Table 1
Average values (SD) of the matching characteristics for the ITBS and control groups

	ITBS	Control
Age	26.8 (8.04)	28.5 (12.1)
Monthly mileage	96.2 (30.6)	99.3 (26.8)
Body mass index	21.9 (2.9)	22.1 (2.4)

2.2. Experimental set-up

Three-dimensional lower extremity kinematic data during running were collected at 120 Hz with a six camera motion capture system. A force plate, embedded in the center of the runway, sampling at 1080 Hz, was used to define foot contact. A series of photoelectric timing devices were used to monitor the running velocity.

2.3. Protocol

Prior to participation, each subject signed a consent form approved by the University's Human Subjects Compliance Committee. Retro-reflective markers were attached to the pelvis, thigh, shank and foot according to McClay and Manal (1999). All subjects wore a standard neutral running shoe. Subjects ran along a 25 m runaway at a speed of 3.7 m/s (±5%) striking a force plate at its center. The 3.7 m/s running speed was chosen because it represented the average training speed of the subjects. After collecting a standing calibration trial, five acceptable trials of both the right and left limbs were collected during the stance phase of running. An acceptable trial was one in which the subject contacted the force platform, without targeting, running within the time constraints.

2.4. Data analysis

Marker data were processed using Visual3D™ software (C-motion, Rockville, MD, USA). The start of stance corresponded with the first instance of the vertical ground reaction force above 20 N and the end corresponded with the last recorded instance of a vertical ground reaction force above 20 N. Using Visual3D™, all lower extremity segments were modeled as frustra of cones while the pelvis was modeled as a cylinder. The local coordinate systems of the pelvis, thigh, leg and foot were derived from the standing calibration trial. In addition, the segment ends were identified from the standing calibration trial in order to locate the segment origins. Coordinate data were low-pass filtered using a fourth-order Butterworth filter with an 8 Hz cutoff frequency. Six degrees-of-freedom for each segment were determined from the segment's set of reflective markers. Subsequently, lower extremity 3-D joint angles were calculated using an Xyz Cardan rotation sequence.

Since strain in the ITB cannot be calculated from the kinematics alone, ITB strain was calculated on a subject-specific basis using a model of the lower extremity derived from Software for Interactive Musculoskeletal Modeling (SIMM 4.0, MusculoGraphics, Santa Rosa, CA, USA; Delp et al., 1990; Delp and Loan, 1995). The model included the pelvis, sacrum, femur, tibia, fibula, patella, talus, calcaneus, and the metatarsals (Fig. 1). The ITB was defined as an elastic structure that originated on the iliac crest and terminated on Gerdy's tubercle. The model's hip and knee each had three degrees of rotational freedom. Bone sizes and muscle geometries in the SIMM model were
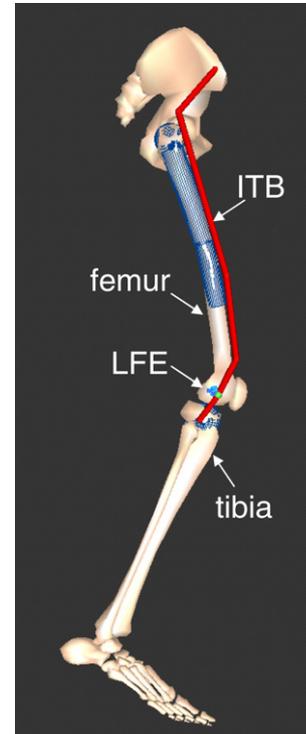

Fig. 1. Musculoskeltal model of the iliotibial band. Dark band along the lateral femur is the iliotibial band (ITB – ipiotibial band; LFE – lateral femoral epicondyle).

scaled to each subject's segment lengths. The segment lengths were obtained from the marker coordinates in the standing calibration trial. Inputs to the model were 3-D hip, knee and ankle joint angles during the stance phase of running. The model, therefore, exhibited the same kinematics as each subject as determined from the motion capture data. The model output consisted of the simulated length of the ITB at each time step during the stance phase of the running stride.

Strain was computed as

$$\text{Strain} = \frac{L_i - L}{L} = \frac{\Delta L}{L}$$

where L_i was the ITB length at time i during stance of the running trial and L is the ITB length in the standing calibration trial. Strain rate (SR) was defined as the slope of the strain-time profile from touchdown to mid-support as follows:

$$\text{SR} = \frac{\Delta \text{Strain}}{\Delta \text{time}}$$

where ΔStrain is the change in strain from touchdown to maximum knee flexion and Δt is the time from touchdown to maximum knee flexion. Impingement between the ITB and the LFE was modeled by defining a wrapping sphere whose surface was flush against the outer surface of the LFE. The duration of impingement was defined as the range of knee flexion angles during which the ITB interacted with the LFE. While friction is often related as a

factor in ITBS, we could not determine friction using this model. However, the duration of impingement is suggested to be the period in which higher friction values would be obtained.

2.5. Statistical analysis

The dependent measures, strain (at initial contact and peak knee flexion), strain rate and duration of impingement, were analyzed for both limbs of all subjects. Using an independent *t*-test, the injured limb of the ITB syndrome group was compared to the matched limb of the control group (e.g. if an ITB syndrome subject injured the right limb, the right limb of the age- and mileage matched control subject was selected) and the contralateral limb of the ITB syndrome group was compared to the comparable limb of the control group. Using a dependent *t*-test, comparisons were made between both limbs of the ITB syndrome group and the control group. In order to further evaluate mean differences, effect size (ES) was calculated to express such differences relative to the pooled standard deviation. ES was calculated as the mean difference between groups divided by the pooled standard deviation (Cohen, 1990). Cohen (1990) proposed that ES > 0.5 represents a clinically significant finding.

To further compare the model parameters of the involved and contralateral limbs of the ITB syndrome group and the right and left limbs of the control group, a symmetry index (SI) was used (Robinson et al., 1987). SI for the ITB syndrome group was computed as

$$ \mathrm{SI} = \frac{|X_{\mathrm{involved}} - X_{\mathrm{uninvolved}}|}{\frac{1}{2}(X_{\mathrm{involved}} + X_{\mathrm{uninvolved}})} \times 100 $$

where X_{involved} is the parameter for the injured limb and $X_{\mathrm{uninvolved}}$ is the parameter for the uninjured limb. For the control group, the right and left limbs were substituted for the involved and uninvolved limbs. A higher SI values indicates greater asymmetry and is reported as a percent.

In addition, we also calculated a Pearson Product Moment Correlation between the peak strain and strain rate parameters in the current study and the hip adduction and knee internal rotation values obtained in the Noehren et al. (2007) study.

3. Results

The means and standard deviations for all parameters are presented in Table 2. When comparing the involved limb of the ITB syndrome group to the matched limb of the control group, strain appeared greater in the ITB syndrome throughout all of stance although there was no statistically significant difference between groups (Fig. 2). At touchdown, strain was 7.3% versus 6.4% for the ITB syndrome and control limbs, respectively ($P = 0.26$; ES = 0.23). At maximum knee flexion, strains were 9.0% for the ITB syndrome group and 7.3% for the control group ($P = 0.11$; ES = 0.45). For strain rate, the involved limb of the ITB syndrome group was significantly greater than the matched limb of the control group ($P = 0.001$; ES = 1.91). The duration of impingement for the involved limb of the ITB syndrome group was 75% of the support period. The control limb duration exhibited a similar duration of 80% ($P = 0.11$; ES = 0.44).

Strain was not statistically greater for the involved limb of the ITB syndrome group compared to the uninvolved limb at touchdown and a maximum knee flexion (Fig. 3). The effect size, however, suggested a clinically significant difference at touchdown but not at maximum knee flexion. Strains at touchdown were 7.3% for the involved limb and 5.5% for the uninvolved limb ($P = 0.06$; ES = 0.51). Strain
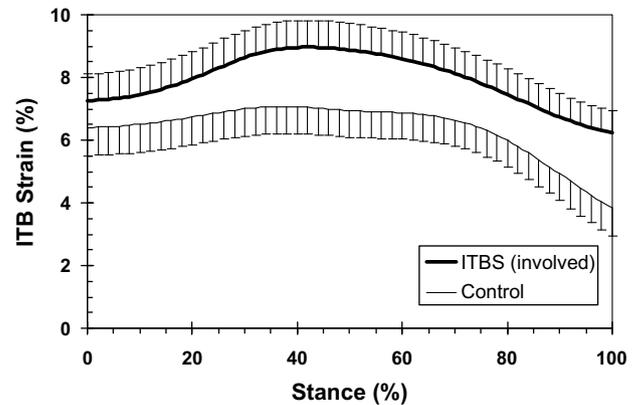

Fig. 2. Group means of iliotibial band strain during the stance period of the gait cycle comparing the involved limb of the ITBS group and the matched limb of the control group.

Table 2
Means (sd) of the dependent measures

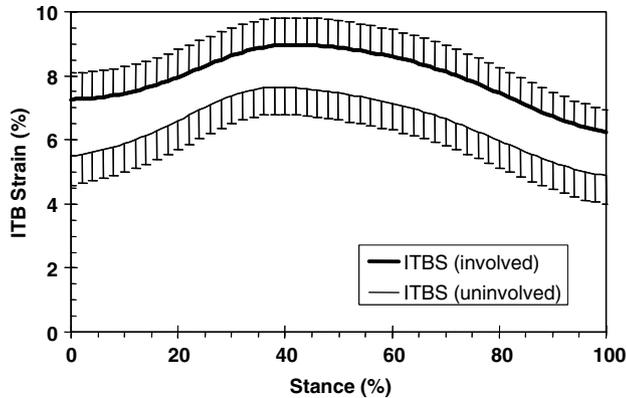| Parameters	ITBS		Control	
	Involved	Contralateral	Matched	Contralateral
Strain (%)				
at touchdown	7.3 (3.4)	5.5 (3.4)	6.4 (3.6)	6.3 (3.5)
at max. knee flexion	9.0 (3.4)	7.7 (3.7)	7.3 (3.4)	7.4 (3.3)
Strain rate (%/s)	25.1 (6.3)	20.0 (8.3)	12.4 (6.9)	13.2 (5.8)
Duration of impingement (%)	75 (10.6)	74 (8.1)	80 (8.5)	77 (9.4)

Fig. 3. Group means of iliotibial band strain during the stance period comparing the uninvolved and contralateral limbs of the ITBS group.

at maximum knee flexion was 9.0% for the involved limb and 7.7% for the uninvolved limb ($P = 0.11$; ES = 0.38). For strain rate, the involved limb was significantly greater than the uninvolved limb ($P = 0.03$; ES = 0.69). The duration of impingement for the involved limb and uninvolved limb were similar ($P = 0.50$; ES = 0.02). SI for the strain at touchdown, at maximum knee flexion, strain rate and duration of impingement was 27.9% (7.9), 15.4% (5.5), 22.6% (10.6), and 1.3% (3.2), respectively.

Comparing the uninvolved limb of the ITB syndrome group and the contralateral limb of the control group, there were no significant differences in strain at touchdown ($P = 0.28$; ES = 0.22), strain at maximum knee flexion ($P = 0.26$; ES = 0.23) and duration of impingement ($P = 0.13$; ES = 0.35). However, there was a significant difference in strain rate with the ITB syndrome uninvolved limb exhibiting a greater strain rate than the control limb ($P = 0.04$; ES = 0.91) (see Fig. 4).

The final comparison concerned the right and left limbs of the control group. There were no statistically significant differences in any of the parameters ($P > 0.05$) with no ES greater than 0.12. SI for the strain at touchdown, at max-
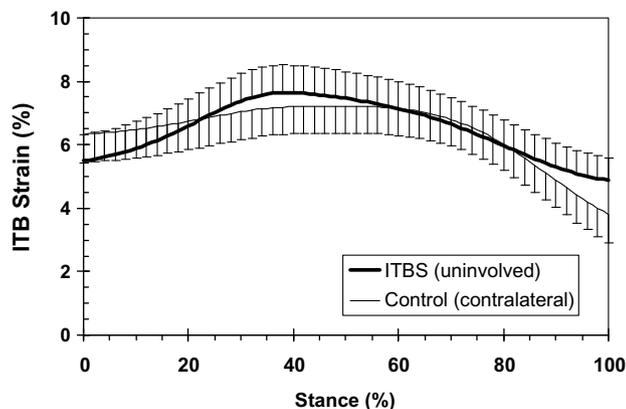

Fig. 4. Group means of iliotibial band strain during the stance period comparing the uninvolved limb of the ITBS group and contralateral control limb of the ITBS group.
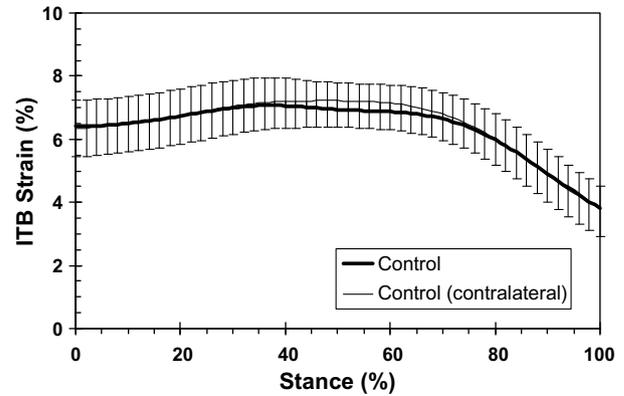

Fig. 5. Group means of iliotibial band strain during the stance period comparing the right and left limbs of the control group.

imum knee flexion, strain rate and duration of impingement was 1.1% (5.4), 1.2% (4.5), 6.5% (5.4), and 3.8% (2.7), respectively (see Fig. 5).

The correlation coefficients for relating peak strain to hip adduction and knee internal rotation were 0.34 ($R^2 = 0.12$) and 0.24 ($R^2 = 0.06$), respectively. Also showing a positive correlation was strain rate with hip adduction ($r = 0.42$, $R^2 = 0.18$) and with internal knee rotation ($r = 0.30$, $R^2 = 0.09$).

4. Discussion

The purpose of this investigation was to compare the pre-existing strain, strain rate and duration of impingement in the ITB between female runners who developed ITB syndrome to healthy controls. We hypothesized that runners who proceeded to develop ITB syndrome would exhibit greater ITB strain, strain rate and impingement than the non-injured control group. While strain appeared greater in the involved leg compared to the uninvolved and control limbs, these findings were not significant. Strain rate in the involved limb of the ITB syndrome group was significantly greater than the strain rate in their contralateral limb or either limb of the control group. Therefore, according to the results of the current modeling study, a major factor in the development of ITB syndrome appears to be strain rate. The effect sizes greater than 0.50 for all of the comparisons indicate that strain rate has biological significance. Thus, we can conclude that strain rate may be a cause of ITB syndrome. These results are consistent with Miller et al. (2007) with retrospective cases of ITB syndrome.

There are a myriad of other factors that have been related to ITB syndrome such as footwear, kinematics, and leg length discrepancy. However, these factors are indirect measures of internal loading. The present modeling methodology allowed for a more direct estimate of trauma to the ITB through the quantification of strain and strain rate. The kinematic differences exhibited by these runners, such as increased hip adduction and increased knee internal rotation (reported in Noehren et al., 2007) appear to

manifest themselves as increased strain rate on the ITB. These measures may be useful as unified measures of prospective injury potential that consider the combined effects of a wide range of kinematic and anthropometric influences.

The ITB is constituted primarily of collagen and functions very much like a tendon. As such, the ITB is viscoelastic and non-linear in behavior. Although we have no direct measure of tension in the ITB, strain rate in a viscoelastic material can be related to the tension in the tissue. Increased strain rate will necessarily increase the tension in the ITB as has been shown in various animal models in different tissue such as ligaments and tendons (Haut, 1983; Yamamoto and Hayashi, 1998). It would appear that the increased strain rate in the ITB, that we found prospectively in these subjects, would certainly have increased the risk of injury that they subsequently incurred.

Biomechanical factors, such as pronounced rearfoot eversion and weak hip abductors, may contribute to the development of ITB syndrome (Messier et al., 1995; Orchard et al., 1996; Fredericson et al., 2000). Proximally, the ITB acts as a lateral hip stabilizer resisting hip adduction. It originates in the fascial components of the gluteus maximus, gluteus medius, and tensor fasciae latae muscles. Distally, the ITB is attached to the supracondyle tubercle of the femur, the lateral intramuscular septum and has fibers that articulate with the patella. Because of these attachments, movements such as increased femoral adduction, will likely lead to increased tension on the ITB and possibly increased strain. These assumptions are partially supported by the results of this study. Although not statistically significant but with a moderate effect size, strain on ITB of the injured group was elevated more than that of both the contralateral limb of the injured group and the right and left limbs of the control group. The effect of this strain over multiple support periods during a prolonged run may have a cumulative effect on the ITB.

It has also been suggested that knee flexion from touchdown to maximum knee flexion may also result in increased strain and thus cause ITB syndrome. Miller et al. (2007) suggested that a more flexed knee at touchdown would lead to a greater duration of impingement and greater stress on the ITB. In this study, we found that knee flexion at touchdown in the ITB syndrome group was essentially the same in the control group (11.2° (4.3) and 12.8° (5.2), respectively) and the contralateral limb of the ITB syndrome group was the same as the involved limb. Similarly, at midsupport, the maximum knee angle of the involved limb of ITB syndrome group was less than the control group and less than their contralateral limb (42.5° (4.3) and 46.9° (5.4), respectively). These results are contrary to Miller et al. (2007) who reported increased knee angles. A prospective study reported the combined increase in hip adduction and knee IR in the ITB syndrome group that might increase ITB strain (Noehren et al., 2007). This notion was supported by Fairclough et al. (2006, 2007) who suggested that ITB syndrome is more related to impaired hip musculature than friction.

It has been theorized that friction of the ITB on the LFE is a primary causative factor in ITB syndrome (Orchard et al., 1996). While we did not directly measure ITB friction in this study, we used strain at key points in the stance phase and duration of impingement as surrogate measures of friction. It would seem likely that increased strain on the ITB and a greater duration of impingement would increase the friction on the LFE causing knee pain that is generally associated with ITBS. However, the results of this study indicate only little support for this theory. We found no differences in strain nor in duration of impingement.

We also hypothesized that the contralateral or uninvolved limb of the ITB syndrome group would exhibit strain, strain rate and duration of impingement characteristics similar to the right and left limbs of the control group but less than the involved limb. This hypothesis was somewhat supported. The differences in strain between the uninvolved limb of the ITBS group and the control limbs were minor with small effect sizes suggesting little biological significance. There were similar findings for the duration of impingement. In strain rate, however, the value for both limbs of the ITB syndrome group was greater than both limbs of the control group. The fact that there were large differences in the strain rate between both limbs of the ITB syndrome group suggests that the potential for bilateral ITB syndrome was apparent in the ITB syndrome group.

The degree of symmetry, as determined by SI, indicated that the duration of impingement was similar between the left and right limbs for both groups. This once again illustrates this parameter may be relatively unimportant in the development of ITB syndrome. However, there was greater asymmetry in the ITB syndrome group versus the control group for strain at touchdown, strain at maximum knee flexion and strain rate. For these parameters, the involved limb of the ITB syndrome group was greater than the uninvolved limb. The asymmetry for these parameters indicates that they may be key to the understanding of ITB syndrome. Thus, contrary to Miller (2006), this indicates that unilateral ITB syndrome may affect the contralateral limb. Secondarily, the asymmetry in the major parameters also indicates the model was sensitive enough to detect these differences. These results present a potential application of Musculoskeletal Modeling in clinical settings based on asymmetry in limb mechanics. In previous research, limb asymmetry in kinetic variables has distinguished between runners with retrospective tibial stress fractures and healthy runners (Zifchock et al., 2006). The present results suggest that asymmetry in ITB strain and strain rate are prospective risk factors for ITB syndrome, and could potentially be used in clinical settings as markers to identify runners at risk for developing ITB syndrome.

In the Noehren et al. (2006) study on the same subjects, hip adduction and knee internal rotation were suggested to increase strain in ITB syndrome subjects. Interestingly,

while strain rate appeared to be an indicator of later ITB syndrome in this study, this parameter showed a rather weak relationship with hip adduction and knee internal rotation as evidenced by R^2 values of 17% and 9%, respectively. Peak strain was not significantly different between groups and also revealed a weak relationship to peak hip adduction and knee internal rotation ($R^2 = 12\%$ and 6%, respectively). These correlations indicate that these particular kinematic parameters give only weak, indirect evidence of the internal loading of the ITB.

Several limitations of the present study should be noted. Most importantly, we had no direct measure of strain, strain rate or duration of impingement. All of these parameters were based on a model driven by the 3-D kinematics that were used as input into the model. Validation of the strain data would be difficult without invasive instrumentation of the ITB. However, the magnitudes of the modeled strains (6–9%) are within the range reported in the literature for *in vivo* strain sustained by elastic structures (5–12%; Monti et al., 2003) and are well beneath the failure strain of around 13% reported by Birnbaum et al. (2001) in mechanical testing of cadaver ITB. While tension in the ITB would have been an insightful measure, we could not calculate tension because we did not have a reliable measure of ITB stiffness for these subjects. In addition, we used a surrogate measure for LFE friction. Since we could not measure the friction directly, we deduced that greater friction would occur during the period of impingement.

5. Conclusions

This study modeled the strain on the ITB during running in a group that had ITB syndrome and a non-ITB syndrome control group. The ITB syndrome subjects were not injured at the time of data collection and only became injured at a point afterwards. Our results indicated that there were no significant differences in strain magnitude and in the duration of impingement. The major factor that demonstrated differences in injured and uninjured limbs was strain rate. The consistent differences found between the ITB syndrome involved limb and the matched control limb, between involved and uninvolved limbs of the ITB syndrome group and the lack of differences between the right and left control limbs indicate the strength of this strain model. It appears, therefore, that strain rate may be a predictor of ITB injury.

Acknowledgement

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