THE EFFECTS OF FATIGUE ON PATHOMECHANICS AND ELECTROMYOGRAPHY IN FEMALE RUNNERS WITH ILIOTIBIAL BAND SYNDROME

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ABSTRACT

The Effects of Fatigue on Pathomechanics and Electromyography in Female Runners with Iliotibial Band Syndrome

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The etiology of iliotibial band syndrome (ITBS) is not fully understood, however, dysfunction at the hip and decreased resistance to fatigue have been suggested to contribute to development of the syndrome. The objective of this study was to investigate differences in hip abductor strength and fatigue resistance, hip muscle activation timing and hip joint kinematic, kinetic and joint coupling patterns in female runners with and without ITBS. In addition, this study examined the effects of a run to exertion on these variables. Twelve female runners with ITBS and 20 healthy female runners participated in this study. Gluteus medius strength and electromyographic (EMG) data were collected during isometric testing. In addition, EMG data from the gluteus medius and tensor fascia latae muscles as well as 3-dimensional kinematic, kinetic and joint coupling data were collected during overground running. All data were collected prior-to and following a run to exertion. Prior to the run to exertion, with runners in a "fresh" state, there were no differences in hip abductor strength, kinematic joint coupling and terminal swing phase muscle activation timing between runners with ITBS and healthy runners. In a "fresh" state, ITBS runners demonstrated less resistance to fatigue at their gluteus medius muscle than did the healthy runners. As a result of exertion, runners with ITBS demonstrated decreased peak hip adduction angles during the stance phase of running gait. There were no group-by-exertion interactions for peak hip internal rotation angles, hip abductor and external rotator moments, kinematic joint coupling or hip abductor strength. There was a main effect of exertion for hip abductor moments, hip external rotator moments and hip abductor strength whereby both healthy and injured runners demonstrated 3.8, 4.2 and 7.3% decreases respectively following the run to exertion. In addition, there was a main effect of exertion on hip frontal/knee transverse plane kinematic joint coupling during the first half of loading where runners demonstrated a 7.3% increase in joint coupling values following the run to exertion. Our data did not detect group-by-exertion interactions or main effects of group or exertion with respect to terminal swing muscle activation timing. There was a significant group-by-exertion interaction when examining fatigue resistance. In a fresh-state, runners with ITBS demonstrated less resistance to fatigue than their healthy counterparts. Following the run to exertion, these differences did not exist. The results of this study suggest that currently symptomatic runners with ITBS demonstrate a potentially compensatory pattern of decreased stance phase hip adduction as compared with healthy runners. Hip internal rotation, abductor moments, external rotator moments or kinematic joint coupling do not appear to discriminate between the two groups. The results of this study also suggest that hip abductor strength may not be as large of a factor in the development of ITBS as previously thought. Instead, this muscle's endurance, or its ability to resist fatigue may play a larger role.

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CHAPTER 1

INTRODUCTION

Running is an aerobic activity that provides participants with cardiovascular, musculoskeletal, and psychological benefits[1] as well as contributing towards weight loss/maintenance. It is also a convenient form of exercise that requires very little equipment, is easily accessible and has minimal financial costs. Therefore it has become an increasingly popular activity for athletes as well as the general population. Increasing enrollment in races around the country evidences the continued rise in running popularity. In 1970 only 127 runners participated in the New York City marathon. Last year, in 2009, more than 40,000 runners participated. However, along with a rise in popularity comes increased prevalence of injury. Running is a high impact, repetitive sport with annual injury rates reaching up to 52% of participants.[2] During a 30-minute run, an estimated 5000 foot strikes occur.[3] With each foot strike, forces twice the body weight are exerted on the body[4] requiring increased muscle activity for shock attenuation. The combination of muscle fatigue, high impact forces and increased exposures (via a larger number of foot strikes), coupled with faulty mechanics is thought to contribute to running injuries. While considerable research has been conducted in the areas of running biomechanics, additional work examining the relationship between biomechanics and injury mechanisms is still necessary.

Iliotibial band syndrome (ITBS) is the second most common running injury[5] and the leading cause of lateral knee pain in runners[6]. Although the cause is unclear, the incidence of ITBS has increased from 4.3% in 1981 to 8.4% in 2000.[5] A better etiological understanding of ITBS is needed to decrease the incidence of ITBS and improve treatment. Advancing our understanding, applying new knowledge towards injury prevention, and tailoring interventions to the individual, will also decrease associated medical costs (doctor visits, surgical intervention, physical therapy etc.) and keep runners active in their sport. Ultimately, a more in-depth understanding of ITBS will allow runners to receive the many health benefits associated with regular cardiovascular exercise.

Running Biomechanics

The following will serve as a basic review of the fundamentals of running gait and components that differentiate running from walking.

During both walking and running, the gait cycle is defined from foot strike (initial contact) to ipsilateral foot strike and is further divided into periods of stance and swing. Stance phase, describing the period of time where the foot is in contact with the ground, begins at initial contact and ends when the foot leaves the ground (toe off). Swing phase, describing the period of time when the foot is in the air for limb advancement, extends from toe off until the subsequent ipsilateral foot strike. During walking gait, stance is further broken down into periods of double and single limb support. Running thus becomes differentiated from walking by the absence of double limb support and the presence of double float. Double float is a period where neither foot is in contact with the ground and occurs twice during the running gait cycle. At the point in a gait cycle when an individual progresses from exhibiting periods of double stance to exhibiting periods of double float, they have transitioned from walking to running. Figure 1-1 diagrams when, during the walking and running gait cycles, periods of double limb support and double float occur.

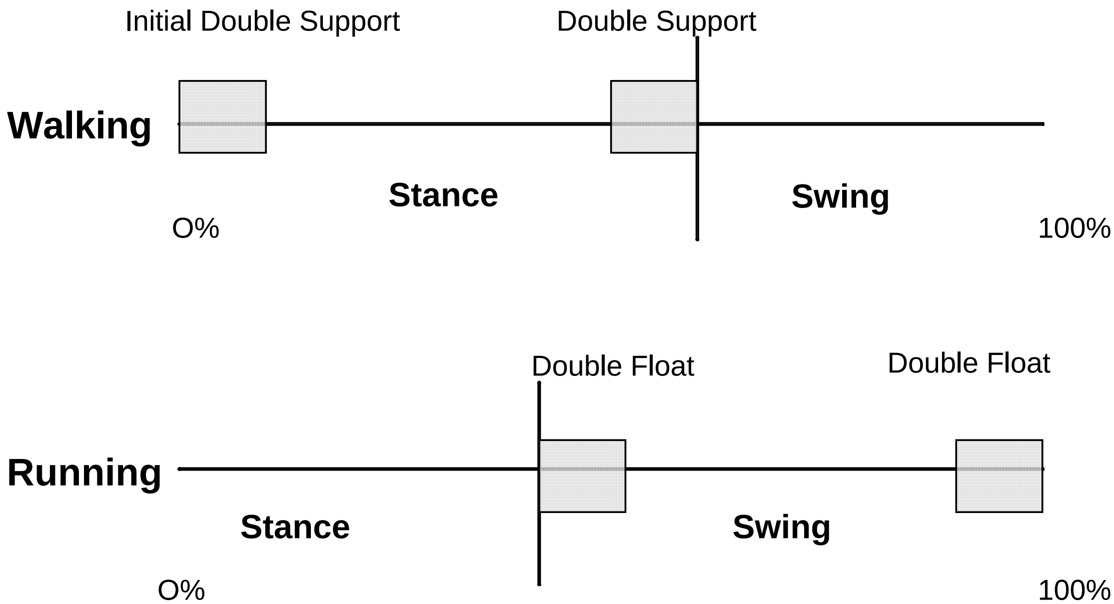

Figure 1-1. The gait cycle. Walking includes periods of *double support* as compared to running which includes periods of *double float*.

During healthy walking gait, initial contact typically occurs at the heel. During running, however, runners employ various foot strike patterns resulting in kinematic and kinetic differences. For example, initial contact can occur at the heel (rearfoot strike pattern), in the middle 1/3 of the foot (midfoot strike pattern) or at the distal 1/3 of the foot (forefoot strike pattern). Approximately 80% of runners are rearfoot strikers.[7] With this in mind, the remainder of this document will discuss running biomechanics with respect to a rearfoot strike pattern.

There are temporal-spatial, kinematic, kinetic and electromyographic differences between walking and running gait. For example, running results in time-distance parameter changes that include, but are not limited to; increased step/stride length, increased cadence and decreased cycle time. These parameters combined result in increased velocity when compared with walking gait.[8]

Joint kinematic changes are significant during running gait when compared with walking. In general, joint excursions increase, the body's center of mass lowers, and the trunk moves into increased anterior lean during running.[7,8] At the hip, specific joint changes include delayed timing of maximal hip extension,[7] increased maximal hip flexion during swing and increased hip adduction during loading.[7,8] Transverse plane motion at the hip is of similar pattern and excursion to that seen during walking.[7,8] At the knee, kinematic changes are primarily in the sagittal plane with runners demonstrating similar patterns to walkers, yet with increased excursion. Running peak knee flexion angles during loading response and swing phase reach values of 45º and 90º respectively as compared with 20º and 60º during walking.[7] Similar to the knee, reported ankle kinematic changes are primarily in the sagittal plane with runners demonstrating a dorsiflexion bias throughout the gait cycle. Additionally, during walking *plantar flexion* occurs after initial contact as the anterior tibialis eccentrically lowers the forefoot to the ground. During running, however, immediate increased *dorsiflexion* occurs as the body's weight is transferred onto the stance limb.[7]

Joint kinetic differences are also noted when comparing walking and running gait. Ground reaction forces differ primarily in the vertical direction. During walking, the vertical ground reaction force curve demonstrates a traditional "double bump pattern", with the first and second bump indicating loading response and pushoff respectively. This typical double bump pattern, however, is not seen during running gait. Instead, an initial impact peak occurs during loading response with a prolonged second peak following for the duration of stance.[8] The data in Figure 1-2 represent vertical ground reaction force data during walking and running gait. Anterior-posterior ground reaction forces differ most in the presence of an additional "breaking" force seen early on in the running stance phase. Both vertical and anterior-posterior ground reaction forces are of increased amplitude during running gait, however, the degree of amplitude is dependent on the speed at which the runner is moving.[8] Typically, sagittal and frontal plane joint moments at the hip, knee, and ankle demonstrate similar patterns in walking and running, but with larger amplitudes seen during running gait.[7,8]

When comparing electromyographic (EMG) activity during walking and running, increased signal amplitude during running has been well documented in the literature.[9-13] There are likely two main causes of this increase in amplitude. First, increased muscular forces are necessary to generate the larger joint excursions seen during running.[8] Second, large external forces and moments require a response for the control of joint motion.[7,8] In addition to amplitude changes, EMG profile differences have been seen when comparing walking with running. Earlier onset activation has been consistently documented in the hamstrings, calf and anterior tibialis muscles.[9-11] There have been conflicting reports in the literature with regards to gluteus maximus, gluteus medius and rectus femoris EMG profiles. Gazendam and colleagues[11] documented similar gluteus medius, maximus and rectus femoris profiles during walking and running. On the contrary, Nilsson and colleagues[9] documented delayed rectus femoris activation while both Novacheck[7] and Ounpuu[8] documented prolonged gluteus medius activity during running gait. Contrary to previously mentioned findings of similar gluteus maximus profiles during walking and running, Lieberman et al.[13] documented earlier onset of the gluteus maximus muscle during running as compared with walking.

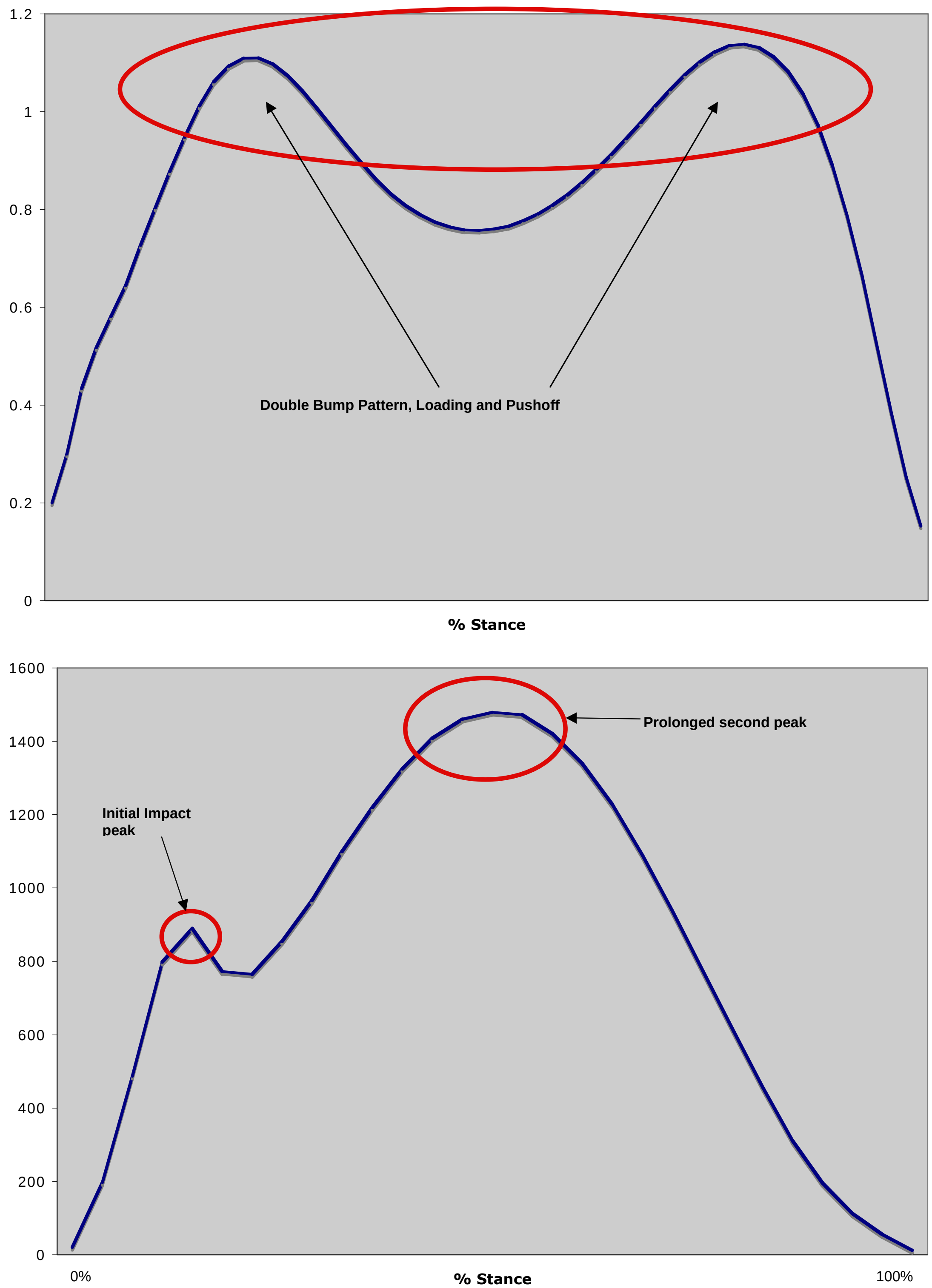

Figure 1-2. Representative Vertical Ground Reaction Force data during walking (top) and during running (bottom) gait.

Iliotibial Band Syndrome

The iliotibial band (ITB) is a fibrous sheath extending down the lateral side of the femur. It attaches to the linea aspera by means of the intermuscular septum and continues until its insertion into Gerdy's Tubercle.[14] Distal to the lateral femoral epicondyle, the iliotibial band is free from bony attachment.[14] In a cadaveric study with the preserved knees in full extension, the posterior fibers of the iliotibial band were found to lie over or just anterior to the lateral femoral epicondyle.[15] While it receives proximal contributions from the gluteus maximus muscle, the ITB is primarily a continuation of the tensor fascia latae (TFL) muscle. The TFL originates anteriorly at the external lip of the iliac crest and continues one-third the distance down the lateral thigh until it inserts into the iliotibial band.[16] When contracted, the TFL flexes, internally rotates and abducts the hip joint. During gait, it assists both the gluteus medius and minimus to stabilize the pelvis in the coronal plane.[17]

Iliotibial band friction syndrome was first thoroughly described by Renne[18] as an irritation of the iliotibial band caused by back and forth rubbing over the lateral femoral epicondyle during repetitive knee flexion and extension as seen during running or marching exercises. Noble[19] expanded on this description with further detail on clinical examination and musculoskeletal findings. At approximately 30° of knee flexion, the iliotibial band is thought to move from its position anterior to the lateral femoral epicondyle, to a more posterior position. During this motion, "friction" is thought to occur between the posterior fibers of the ITB and the lateral femoral epicondyle.[14] This friction causes a localized irritation and thus results in the complaints of lateral knee pain commonly associated with ITBS. During running, knee flexion up to 45 degrees can occur during loading response[7], thereby increasing the likelihood of ITBS in this population of athletes. Early literature in support of the *friction* theory, reported the presence of a bursa over the lateral femoral epicondyle.[18] However, more recently, MRI and cadaveric studies have not supported these findings.[20-22]

Fairclough et al.[22] recently provided an alternate etiology of ITBS. The authors suggest that *compression* rather than friction is occurring between the iliotibial band and the lateral femoral epicondyle. In a cadaveric study, the authors document the presence of adipose tissue containing Pacinian corpuscles beneath the iliotibial band. The presence of Pacinian corpuscles, deep subcutaneous mechanoreceptors that sense global pressure[23], support the theory that *compression* rather than *friction* is occurring between the lateral femoral epicondyle and the iliotibial band. In either case, the pathogenesis of ITBS involves an irritation and resultant inflammation deep to the posterior fibers of the iliotibial band and surrounding the lateral femoral epicondyle.[20,21]

Orchard et al.[15] describe an "impingement zone" as a range of knee angles where contact occurs between the iliotibial band and the lateral femoral epicondyle. The authors compare this *zone* to the "painful arc" seen in patients with subacromial impingement syndrome and describe a similar disease progression. Contact occurs between the iliotibial band and the lateral femoral epicondyle causing a localized inflammatory response. This inflammation then increases the zone of impingement (range of angles at which contact between the two structures will occur) and leads to a progressive worsening of symptoms.

Etiological factors

A number of etiologic factors have been associated with iliotibial band syndrome including improper training (running on banked surfaces, excessive mileage or a sudden increase in mileage, hill training), inexperience, biomechanical differences, anatomical differences (short stature, limb length inequality, lighter weight) and strength impairments.[6,14,24-27] For detailed descriptions of these findings, the reader is referred to the referenced readings. Additional etiologic factors include strength impairments, kinetic and kinematic alterations, fatigue related issues and gender. While symptoms of ITBS present themselves at the knee, recent developments in the literature have found many of these impairments and alterations occurring at the hip. The following sections will serve as a review of these etiological factors as they relate to this study.

The Role of the Hip in Runners with ITBS

Dysfunctions at the hip including decreased strength, altered kinematics, and altered kinetics have been linked to iliotibial band syndrome. Weakness in the hip abductor muscles,[28,29] trends towards weak hip external rotator muscles[28] and increased peak hip abductor moments (reflecting decreased ability to control femoral adduction) [30] have been documented in runners with ITBS. Furthermore, Fairclough et al.[22] have suggested that there is a relationship between impaired *function* of the hip musculature and ITBS whereby a primary dysfunction of the lateral hip muscles results in compressive forces on the tissues beneath the ITB and hence, secondary pain over the lateral femoral epicondyle.

Additionally, both prospective and retrospective studies have documented increased stance phase hip adduction angles in runners with ITBS.[25,30-32] These findings demonstrate the importance of continued exploration into the role that the hip plays in the development of ITBS.

During loading response, vertical ground reaction forces can reach 2.2 times body weight in 23 milliseconds[4] with resultant hip adduction motion.[7] At this time, a large contribution from the hip abductors, specifically the gluteus medius muscle, is required to absorb this shock and overcome large external adductory moments.[7] Additionally, the tensor fascia latae and the upper fibers of the gluteus maximus assist with hip abduction.[16] Inability of the hip muscles to counter this external adduction moment, whether due to weakness or neuromuscular dysfunction, is suggested to be a factor in the development of ITBS.[29,30,33] The increased stance phase hip adduction found by Noehren et al.[25] is consistent with other studies' findings of hip abductor weakness[29] and of increased hip adduction moments[30]. These findings strongly indicate impairments of the hip as an etiological source of ITBS, particularly the eccentric control of the lateral hip muscles during loading response. Due to the inconsistencies in the literature, further research into lower extremity biomechanical differences in runners with ITBS is necessary.

Hip muscle strength and function

The role of hip muscle strength has not been fully investigated in runners with ITBS, yet research has indicated that strength impairments at the hip may be associated with the development of ITBS. Niemuth et al. conducted a study that looked at hip muscle strength in runners with overuse injuries.[28] Using a hand-held dynamometer, the authors compared bilateral hip strength of 6 antagonistic muscle groups between a group of injured (including ITBS) and non-injured runners. Peak force values from the hip abductors/adductors, hip internal/external rotators and hip flexors/extensors were normalized to the subjects' weight and utilized to compare between groups and between limbs. A 2-way mixed ANOVA was conducted for the factors of injury status and leg (both groups) and duration of symptoms and leg (experimental group only). While no strength differences were found between legs in the uninjured control group, there were significant differences found between the injured and uninjured limb of the experimental group. Specifically, the authors found significantly weaker hip abductors and hip flexors on the injured limb and significantly stronger hip adductors on the uninjured limb. Although not significant, the authors also found a trend towards weaker external rotators on the injured limb. As a retrospective study, we are unable to imply a causal relationship between hip weakness and overuse running injuries, yet we may note that an association exists between the two.

These findings by Niemuth et al.[28] of decreased hip abductor strength in the injured limb of runners with overuse injuries support the findings of Fredericson et al.[29] whose study examined hip muscle strength in runners with ITBS. Isometric hip abductor strength was compared between a group of runners with ITBS and a non-injured control group. Following isometric strength testing, the injured runners received a six-week rehabilitation program including anti-inflammatory modalities and exercises (iliotibial band stretching and gluteus medius muscle strengthening). Paired *t*-tests were utilized to compare hip abductor strength between the injured limb and the non-injured limb and to compare pre- and post- rehabilitation strength measures in the injured limb. Two-sample unequal-variance *t*-tests compared hip abductor strength in the treatment patients to hip abductor strength in the control group. As Niemuth et al. found, prior to the intervention, all injured participants demonstrated significantly greater gluteus medius weakness on their injured side. There was also significantly weaker gluteus medius muscle strength in the injured group (both injured and non-injured limbs) as compared to the healthy runners. Following the 6-week rehabilitation period, participants who received treatment demonstrated significant strength gains and 22/24 were able to return to running. The findings of hip muscle strength deficits in runners with ITBS give further justification of the need for future studies in this area.

Hip abductor weakness is proposed to result in kinematic alterations including increased hip adduction and knee valgus angles during running gait.[29,31,33] While no study has directly correlated hip strength deficits with kinematic alterations, the previously mentioned literature documenting kinematic deviations in runners with ITBS seems to support this theory. *Therefore, continued focus on the role of the hip, both muscularly and biomechanically, is of great importance in runners with ITBS.*

Kinematic and Kinetic Changes in Runners with ITBS

Multisegmental alterations in joint kinematics and kinetics have been documented in runners with ITBS. Earlier studies tended to focus on kinematic and kinetic data from the knee and ankle[14,15] while more recently, deviations at the hip have been studied and documented[25,32,34]. To date, six biomechanical studies of runners with ITBS have been published.[14,15,25,27,32,34] The following will serve as a review of these studies.

Messier et al.[14] explored the relationships between the anthropometric, biomechanical, strength and training variables in runners with and without ITBS. Over two testing sessions, the investigators collected kinematic and kinetic running gait data, isokinetic knee strength and endurance measures, information from a training questionnaire, and various anthropometric measures. Frontal plane kinematic values including calcaneal to tibial angles (termed pronation/supination) and calcaneal-to-vertical angles (eversion/inversion) were calculated during a 15-minute treadmill run. Kinetic measures including vertical, anterior-posterior and medial-lateral peak ground reaction forces and time to critical peak forces were calculated during overground running.

A preliminary linear discriminant function analysis followed by a final discriminant function analysis (utilizing only variables which were significant at a 0.10 level in the preliminary analysis) revealed training variable differences between the groups. Specifically, runners with ITBS ran more miles, trained at a faster pace, spent less time with the current training protocol, spent more time running on a track, and spent more time swimming (for cross training) than the control group. There were no differences between runners who ran on asphalt, dirt, crowned roads, hills or level/trail surfaces nor were there differences in those participants who performed running or cycling as their form of cross training. Anthropometric measures found that runners with ITBS were of significantly shorter stature than those without ITBS. Isokinetic strength measures at 60°/sec revealed significantly decreased peak flexion torque and an increased flexion/extension ratio in the ITBS group. In addition, participants with ITBS were found to perform less extension work during the first and last six repetitions at 240°/sec.

Extension average power was greater in the ITBS runners, and flexion peak torque/BW and work were less in the ITBS group.

Kinematic data revealed a decreased calcaneal-to-vertical touchdown angle (decreased inversion at initial contact) and increased maximum supination velocity in runners with ITBS. No significant differences were found in maximum or total pronation, pronation angle for 10% of stance, maximum or total eversion, eversion angle for 10% of stance, time to maximum pronation, maximum pronation velocity or initial velocity. Kinetic variables revealed a significantly reduced maximal braking force in runners with ITBS.

Orchard et al. performed a two-part biomechanical study looking at the anatomy of the iliotibial band in cadaveric specimens as well as looking at the sagittal plane knee kinematics of nine recreational runners with unilateral ITBS.[15]

In the cadaveric portion of the study, iliotibial band width, length and its spatial relationship with the lateral femoral epicondyle was examined in eleven cadaveric legs. The authors found large variations among iliotibial band widths (23-51mm) and the spatial relationship between the iliotibial band and the lateral femoral epicondyle (5/11 bands laid over the lateral femoral epicondyle, 6/11 ranged from 3-9 mm anterior).

Sagittal plane kinematic data were collected with and without participants wearing a 0.5 cm heel lift in the affected shoe while running on a treadmill for two minutes. During both test conditions (with and without the heel lift), comparisons between the involved and uninvolved limb revealed no significant differences in sagittal plane flexion angles at the hip, knee or ankle. The results of this study, however, are limited in their applicability for multiple reasons. The lack of statistically significant differences in joint flexion angles may be attributed to the absence of a control group. Biomechanical differences may be present in both lower extremities and not detected when they are compared against each other as they would be if compared against an appropriately matched control group. This thought is supported by a later study, which found sagittal plane kinematic differences at the knee when comparing runners with ITBS to healthy runners.[27]

In a third study, Miller et al. compared kinematic data between 8 runners with ITBS and 8 age-matched controls.[27] Data were collected every two minutes while participants ran until voluntary exhaustion on a treadmill. A two-by-two factorial ANOVA was performed to compare start and end kinematic data between groups.

At the beginning of the run, runners with ITBS demonstrated significantly greater minimal knee flexion angles, maximal ankle dorsiflexion velocity, and maximum foot adduction than control runners. Following the run, runners with ITBS demonstrated significantly greater knee flexion angles, maximum foot inversion, minimum thigh flexion velocity and maximum knee internal rotation velocity than their non-injured counterparts. This study was the first to examine transverse plane motion in runners with ITBS, yet their findings of increased tibial internal rotation velocity are supported by later work documenting increased knee internal rotation motion in runners with ITBS[25,32]. Based on the anatomical insertion of the iliotibial band at the lateral tibia, internal tibial rotation may increase strain at the ITB and compressive forces between the ITB and the lateral femoral epicondyle.

Contrary to findings by Noehren et al.[25] and Ferber et al.[32], Miller et al. found no significant differences at the hip. The use of a treadmill during data collection may explain the differences between studies, as treadmill running has been shown to alter running kinematics.[35,36] Overall, this kinematic evaluation measured ample variables and was well designed. The authors provide insight into kinematic changes that occur following an exhaustive run, valuable information for this injury population that has not been studied elsewhere.

Noehren et al. conducted a prospective study comparing lower extremity kinematics and kinetics between a group of female runners who developed ITBS and healthy controls.[25] Three-dimensional lower extremity kinematic and kinetic data were collected bilaterally during overground running. Runners wore a neutral running shoe with retroreflective markers attached externally. Of note, this method of capturing tibio-calcaneal motion via external markers has been shown to overestimate average tibio-calcaneal motion.[37,38] At the time of data collection, all runners were free from injury. Participants were then followed for a period of 4 years via e-mail, and any running injuries were recorded. From the original sample size of 400 runners, 18 developed ITBS. These runners were included in the study and compared to an age and mileage matched control group. Kinematic variables including peak rearfoot eversion, knee internal rotation, hip adduction and knee flexion motion at initial contact along with tibial internal rotation and femoral rotation (relative to the room) were compared between the injured leg of the ITBS group and the right leg of the control group. The authors also compared peak rearfoot eversion, knee external rotation and hip abduction moments. Independent t-tests were utilized to test the hypotheses with significance set at $p<0.05$. The authors found significantly greater peak hip adduction and tibial internal rotation angles in the group of runners with ITBS. In addition, the authors noted a trend towards decreased peak eversion in the ITBS group. No significant kinetic differences were found between groups.

In a later study, the same group of researchers conducted a retrospective analysis comparing kinematic and kinetic data between 35 runners with a history of ITBS and 35 healthy runners.[32] Methodology was consistent with the previously described study.[25] Kinematic and kinetic data including: peak rearfoot eversion angle and invertor moment, peak knee internal rotation angle and external rotator moment, peak hip adduction and abductor moment and peak knee flexion were extracted from the first 60% of stance. A one-way ANOVA was used to compare between groups. Consistent with their prospective findings, the authors documented increased hip adduction and knee internal rotation in runners with ITBS. Previously the authors had documented a trend towards decreased peak rearfoot eversion in ITBS runners, however, this retrospective analysis found no differences in peak ankle eversion angles. As documented in their previous prospective study, the authors did not find differences in hip or knee moments, however contrary to their previous work; their retrospective study did find increased ankle invertor moments in the injured runners.

Finally, in a recent study, Grau and colleagues compared overground kinematic data between 18 runners with ITBS and 18 healthy control runners.[34] Three-dimensional kinematic data were collected while runners ran barefoot across a dense foam surface. Group comparisons were made with respect to peak angular, range of motion and maximum velocity values for hip flexion and adduction, knee flexion, ankle dorsiflexion and ankle eversion. Additional timing variables calculating the maximal angular excursion as a percentage of the foot "rollover process" were calculated. In contrast to much of the previous work, the authors found *decreased* hip adduction values in the ITBS group, both at their peak and nearly throughout the "rollover process". Additional group differences included decreased hip range of motion, decreased abduction velocity, decreased maximum hip flexion velocity and decreased maximum knee flexion velocity in the injured runners.

In summary, the biomechanical findings at the ankle, knee and hip in runners with ITBS are both limited and conflicting. Ferber et al. found increased rearfoot eversion angles and eversion excursion in runners with ITBS.[26] These findings were not supported by either Messier et al.[14], who found no significant difference in total eversion motion or eversion velocity, nor by Noehren et al.[25] who found a trend towards decreased rearfoot eversion angles in runners with ITBS.

Similar conflicting finding were discovered at the knee. While Orchard et al.[15] and Grau et al.[34] found no significant stance phase difference in sagittal plane knee flexion angles during fresh-state running, Miller et al.[27] found increased maximal knee flexion angles in the knees of runners with ITBS following a run to exertion.

The hip's involvement in runners with ITBS remains unclear due to similar conflicting findings in published studies. Studies have documented no significant differences in hip kinematics during fresh and fatigued-state running [27], while other conflicting work has documented decreased[34] or increased[25,32] levels of stance phase hip adduction. It is clear that further investigation into hip biomechanics in runners with ITBS is needed.

One explanation for the varied results found in the literature may be the mixed methodologies used to collect the data. For example, Miller et al.[27] examined kinematics during treadmill running, yet examined the effect of a run to exertion. Other studies have examined overground running, but with no fatiguing protocol[15,25,32,34] and/or no control group[15]. Others examined barefoot running[34], some included both genders[15,34] and some just female runners[25,32]. Each of these methodological variations limits the generalizability of the existing research at large.

The Effects of Fatigue

An additional component that is lacking from much of the existing ITBS research is on the effect of fatigue. Understanding the effect of fatigue is of great importance in all running research. However, anecdotally, runners with ITBS complain of symptoms after 2-3 miles of running, making an understanding of the effect of fatigue even more important in this population. Kinetic, kinematic and electromyographic changes have been well-documented following fatigue in both healthy and injured runners.[27,39-42] During marathon or half-marathon preparation, high weekly mileage and "long" training runs (ranging in distance from 8-23 miles) require runners to push their bodies beyond the point of fatigue. Surprisingly, fatigue alters running biomechanics in as little as 15 minutes of running,[41] impacting both the casual and long distance runner. Additionally, fatigue-related changes, such as delayed muscle activation and altered force attenuation[3,39] have been suggested to increase injury risk. Miller and colleagues[27] are the only authors to-date who have examined the effect of a run to exertion on runners with ITBS. This study, however, is limited in its generalizability based on their collection of data during treadmill running. Additionally, no study has examined the effect of exertion on joint kinetics. Therefore, further examination into the effect of fatigue on *overground* running kinematics and kinetics is necessary.

In one study examining biomechanics and fatigue, Derrick et al.[41] compared impacts and joint kinematics in healthy runners during a fatiguing treadmill run. Accelerometers were utilized to calculate peak impact at the head and lower extremity while electrogoniometers measured peak angles and velocities of knee flexion/extension and rearfoot inversion/eversion at initial contact. Data from the beginning, middle and end of the run were collected and compared using a one-way repeated measures ANOVA. The authors documented fatigue-related changes in both impact and kinematic data. Following the fatiguing run, they found significantly increased peak impact acceleration at the leg in addition to significantly increased shock attenuation. Significant kinematic changes following the exhaustive run included increased knee flexion angle at initial contact, increased knee flexion velocity, increased rearfoot inversion angle at initial contact and increased rearfoot velocity. Their findings of increased shock attenuation contradict a later study,[3] however based on their results, the authors conclude that fatigue-related kinematic alterations may increase injury risk.

Nordin and Frankel[43] have proposed that strenuous exercise and resultant muscular fatigue cause both gait alterations and a decreased ability to absorb shock. This statement has been supported by findings of decreased shock attenuation following a fatiguing run.[3] In this study, Mercer et al.[3] examined impacts and shock attenuation during and after a fatiguing run. With accelerometers mounted to the leg and skull, participants performed a “maximal graded exercise test”. This test involved incremental increases in treadmill speed and grade over the course of a run until the participant could no longer continue. Data were collected prior-to and following the exertion protocol. As mentioned previously, the authors found a 12% decrease in shock attenuation following the fatiguing run. While there is no conclusive evidence linking these fatigue-related changes to injury, decreased shock attenuation implies that the body is subjected to higher levels of forces and therefore greater risk of injury following a run to fatigue.

In a study of 90 injury-free female runners, Gerlach et al.[42] examined the effect of fatigue on ground reaction force (GRF) data during treadmill running. Following a warm-up run, fresh-state GRF data were collected via force plates imbedded in the treadmill while participants ran at their 5K race pace. Participants were then "fatigued" using a "modified discontinuous VO_{2max}" protocol during a treadmill run. The modified discontinuous VO_{2max} protocol progressively increases treadmill speed and gradient until voluntary exhaustion. Participants' oxygen uptake, respiratory exchange ratio and heart rate were monitored as criteria for the achievement of VO_{2max}. A repeat collection of GRF data was performed following the run to fatigue. Runners were then followed prospectively for 12 months to document any occurrence of musculoskeletal injury. Runners who developed injuries in the year following data collection as well as runners with a documented injury in the year preceding data collection were categorized as "injured" runners. Kinetic variables calculated from GRF data included peak impact force, impact loading rate and peak active force (pushoff). A 2-way repeated measures ANOVA was used for the comparison of injured/non-injured runners as well as pre/post-fatigue dependent variables. Following the run to fatigue, peak impact force and peak loading rates both decreased significantly. There were no differences in peak force at pushoff between the fatigued and non-fatigued state. Kinetic changes may be associated with decreased cadence and increased step length found in the post-fatigue state. No significant interactions were found between injury status (combining retrospective and prospective injuries) and pre/post-fatigue kinetics. However, when examining only runners with a retrospective history of running injury, subjects were found to have less of a decline in peak impact force and peak loading rates than healthy runners. The authors conclude that previous injury may affect a runner's ability to compensate for fatigue putting them at further risk for re-injury.

Christina et al.[40] examined the effect of localized muscular fatigue on vertical ground reaction forces (VGRF) and ankle joint kinematics during treadmill running. Kinetics and kinematics were measured immediately following a localized fatiguing protocol to ankle dorsiflexors or ankle invertors. This protocol involved 15 repetitions of ankle dorsiflexion or inversion with resistance set at varying percentages of their one-repetition maximum until reaching a point at which they could not move against gravity (dorsiflexion protocol) or until they were unable to reach a pre-determine force target (inversion protocol). Following the dorsiflexor fatiguing protocol, runners demonstrated significantly less dorsiflexion at initial contact and a significantly increased rate of rise of the peak impact force. Following localized invertor fatigue, there were significant decreases in peak impact forces, peak push-off forces, and the rate of decline of the impact force. No significant differences were found for the rate of rise of peak push-off force or sagittal/frontal plane ankle kinematics. An additional regression analysis showed a direct relationship between ankle dorsiflexion angle at initial contact and the loading rate of the impact force. The authors conclude that fatigue-related changes may result in increased internal loads and contribute to the development of injury.

A recent study similarly examined sagittal plane running kinematics following a localized knee flexor/extensor and ankle plantar/dorsiflexor fatiguing protocol.[44] This protocol involved participants performing repeated isokinetic knee flexion/extension and ankle plantar/dorsiflexion exercise until they were unable to achieve 30% of their maximum torque. The authors found that following ankle fatigue, the angle of dorsiflexion at initial contact decreased. These findings supported those of Christina et al.[40] The authors also found an increased angle of knee flexion at initial contact following the knee fatiguing protocol. These findings support the previous work of Derrick et al.[41] who also found increased knee flexion angles at initial contact in fatigued runners. These studies all support a link between fatigue, kinematic alterations and an increased risk of injury. Therefore, it is important to consider these findings when examining and treating injured runners.

Fatigue has also been found to cause electromyographic alterations during running gait. Nyland et al.[39] measured kinematics, kinetics and lower extremity onset muscle activation timing following a fatiguing protocol in nineteen healthy female athletes. Once fatigued, participants were asked to perform three "run and rapid stop" trials while impact forces, surface electromyography and joint kinematics during landing were collected. During the "run and rapid stop" trials, onset muscle activation of the rectus femoris, vastus lateralis, biceps femoris and medial hamstring muscles tended to be delayed in the fatigued state as compared with the non-fatigued state. While the maximal knee flexion angle tended to occur earlier during the fatigued condition, there were no significant differences in the remaining kinematic and kinetic variables. The findings of delayed muscle onset activation are important. A delay in the muscle's ability to become activated may alter the shock absorptive capacity of the muscle causing increased stress on the bones, muscles and ligaments. Muscles are most active prior-to initial contact and during loading response,[7] therefore, complete and timely muscular recruitment is important to absorb the large external forces being exerted on the body upon impact.

Only two studies to-date have examined fatigue-related changes in runners with ITBS.[27,45] In the first study, kinematic data were collected prior-to and following a 20-minute treadmill run.[27] Using a two-by-two factorial ANOVA, pre- and post-fatigue comparisons were made between a group of runners with ITBS and a healthy control group. Following the run, the authors found significantly greater knee flexion angles, maximum foot inversion, minimum thigh flexion velocity, and maximum knee internal rotation velocity in runners with ITBS. These findings are similar to those of Derrick et al.[41] who discovered increased knee flexion and rearfoot inversion angles at initial contact in healthy fatigued runners. In a second study by the same group[45], variability of kinematic joint coupling was examined on the same group of runners prior-to and following their 20-minute treadmill run. Using a 2x2 factorial ANCOVA with time and group as a factor, time as a repeated measure and speed as a covariate, the authors did not find any main effect of time indicating that variability did not change over the course of the run for both the injured and healthy runners. The authors did, however, find post-fatigue differences between the two groups. Details of this study are provided in the following section on joint coupling.

As demonstrated in the above-referenced studies, fatigue causes significant changes in kinematic, kinetic and muscle electromyography during running in both healthy and injured runners. These changes, including decreased shock attenuation,[3] delayed EMG onset activation,[39] and kinematic/kinetic alterations,[40,41] are thought to contribute to injury risk. However, much of the fatigue-related running research has been conducted on healthy runners with only one study examining the effect of fatigue on runners with ITBS. With such strongly accepted anecdotal evidence that symptoms of ITBS present themselves after 20-30 minutes of running, further examination into the effects that fatigue has on running biomechanics in this group of runners is necessary.

Joint Coupling

Single joint kinematic alterations have been extensively documented in the injured population.[14,15,26,27,39] However, the relationship between these alterations and injuries fails to provide researchers with a complete understanding of running injury pathology. Therefore, biomechanical running studies have also examined *interactions* between joint motions (excursion and timing) and their relationships to injury. These interactions, often referred to as joint coupling, can be quantified in a variety of ways. Measurement variables include: timing of joint motions, joint range of motion excursions and relative timing of two motions (within or between joints).

Investigators can examine joint coupling variables at discrete times during the gait cycle or continuously throughout the gait cycle.[46] The following review will describe both the discrete and continuous methods utilized to quantify joint coupling during gait.

Discrete joint coupling relationships are commonly measured in terms of timing of joint motion or ratios of joint excursion. Timing descriptors typically include the time to reach a peak angular value, for example time to reach peak knee flexion and peak rearfoot eversion.[47] Timing differences between motions or segments are then calculated as the time to peak distal motion (eversion) minus the time to peak proximal motion (knee flexion). These differences can then be described and compared between the injured and uninjured populations.

Excursion ratios provide a discrete measure of the *relative motion* occurring between two joints. The ratios are calculated as the total angular motion at the distal segment divided by the total angular motion at the proximal segment. For example, when measuring rearfoot eversion and knee flexion, the peak value of rearfoot eversion excursion would be divided by the peak value of knee flexion excursion. This provides the reader with a ratio indicating equal or unequal excursion between the two segments. An excursion ratio value equal to 1 shows equal excursion between the two segments. A ratio value greater than 1 indicates greater excursion occurring distally while a ratio value less than 1 indicates greater excursion occurring proximally.[47]

Quantitative measures of continuous joint coupling include the continuous relative phase (CRP) and vector coding approaches. The CRP measure utilizes the dynamic systems theory approach. This measure plots joint angle (angular position) versus joint velocity (angular velocity) in a “phase-plane”.[48] A “phase angle” for each segment or joint is then obtained from this phase plane. The “relative phase” is calculated as the differences in phase angles between the two segments or joints. This calculation allows the researcher to obtain the relative phase throughout the entire movement cycle rather than at discrete points in time. Limitations of the CRP approach include the assumption that the data of interest are sinusoidal. Aside from sagittal plane measures of hip and knee flexion/extension, many joint motions that occur at the lower extremity during running are non-sinusoidal.[49] Therefore, the use of CRP can generate erroneous results in this population. Another limitation of the CRP measure is the need to normalize data. Normalization of data is performed to minimize the influence of varying movement amplitudes[47] thus avoiding the possibility that one segment or motion will dominate the CRP[48]. However, normalization is not performed consistently between studies and therefore comparison of CRP data is limited.[46]

Vector coding is another measurement tool utilized to describe continuous joint coupling. It is the chosen methodology for the studies described in this document due to the inherent limitations of the CRP measure. In the vector coding process, an angle-angle diagram (Figure 1-3) is constructed from the two motions of interest (i.e. hip abd/adduction and knee internal/external rotation). Using the following equation, $\Theta = |\tan^{-1}(y_2-y_1/x_2-x_1)|$, angles of the trajectory between two successive data points are calculated for all data points over the period of interest (i.e. loading response). The mean angular value for the period of interest is then calculated. This provides a continuous excursion ratio coupling angle with a range of 0°-90°. A 45° angle indicates equal amounts of movement between the two segments. An angle greater than 45° indicates greater amounts of distal motion while an angle less than 45° indicates greater amounts of proximal motion.[46,47,49]

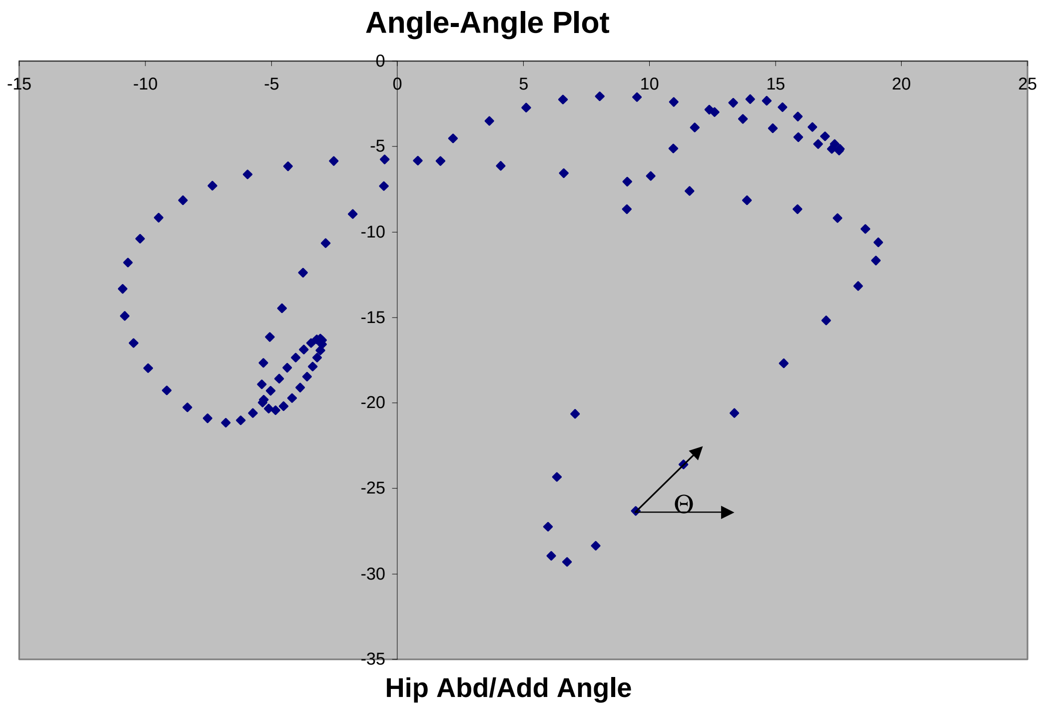

Figure 1-3. Angle-angle plot constructed from hip abd/adduction and knee internal/external rotation motion during a representative gait cycle. Θ indicates vector coding angle between two successive data points.

Joint coupling has been examined in both the injured and uninjured population. Dierks et al.[47] studied lower extremity joint coupling relationships in a group of 40 uninjured recreational runners. In their work, 20 male and 20 female runners between the ages of 18 and 45 years old were asked to run along a 25-meter runway while 3-dimensional forceplate and motion data were collected. Joint timing, excursion ratios, vector coding and continuous relative phase data were calculated and averaged across the trials. The variables of interest included: rearfoot eversion (EV), tibial internal rotation (TIR), knee flexion (KF), and knee internal rotation (KIR). Indicating relatively greater synchronicity, the authors found smaller timing differences in EV-TIR, EV-KF and TIR-KF as compared to EV-KIR and TIR-KIR. The authors found EV/TIR and EV/KIR excursion ratios were each approximately 2.0. This value indicates more relative eversion excursion than TIR or KIR excursion. Using GRF data, the authors defined four phases of stance for the calculation of vector coding and continuous relative phase values. The first phase, loading response was defined from heel-strike to impact peak. Phase 2 was defined from impact peak to the maximum vertical ground reaction force (VGRF). Phase 3 extended from maximum VGRF to half the distance to toe-off and phase 4 extended from the end of phase 3 to toe-off. Vector coding results displayed greater distal segment excursion during stance (i.e. values greater than 45°) for most coupling relationships. The one exception was relationships involving KE/KF. These relationships demonstrated greater proximal excursion (values less than 45°). At mid-stance, values were approximately 45°, indicating relatively equal amounts of motion during this time. Continuous relative phase values ranged between -44.2° and 48.6° with 0° indicating completely in-phase and 180° indicating completely out-of-phase relationships. Phase 3 was the most in-phase while the transitional phases 1 and 4 were the most out-of-phase. This study gives the reader a comprehensive understanding of kinematic joint coupling patterns in a healthy runner described with both discrete and continuous measures.

Abnormal joint coupling and variability of coupling is thought to contribute to injuries in the athletic population.[48,49] For example, Hamill et al.[48] performed two studies measuring CRP values in a group of runners thought to be *at risk for* patellofemoral pain (Q-angle greater than 15°) and in a group of individuals *with* patellofemoral pain. These individuals were compared to groups of individuals at *low-risk* for patellofemoral pain (Q-angle less than 15°) and *painfree* subjects respectively. In the first study comparing “at-risk” to “low-risk” individuals, participants performed ten trials of overgound running along a 35-meter runway with an imbedded force platform. In the second study, runners with and runners without patellofemoral pain ran on a treadmill at three velocities (2.5, 3.0 and 3.5m/sec). In both studies, three-dimensional data were collected and stance phase kinematic variables included; thigh adduction/abduction, thigh flexion/extension, tibial rotation and foot inversion/eversion. CRP coupling patterns and variability were calculated and compared between groups. In the first study, no differences in coupling patterns or variability were found between groups (at-risk/low-risk runners). This finding is not surprising as all subjects involved in this study were asymptomatic. For the purpose of this review, more details will be provided on the second study involving symptomatic participants. In this study, the authors compared the CRP and the variability of the CRP at each running velocity. In addition to the kinematic variables mentioned previously, the authors also measured thigh internal/external rotation and tibial internal/external rotation coupling for this study. They found decreased variability of the CRP coupling during stance in the patellofemoral pain group as compared to the asymptomatic group. The between-group differences were most pronounced during the transitional periods from stance to swing and from swing to stance. The decreased variability amongst the patellofemoral pain participants indicates greater repeatability of segment coupling, a trait that may predispose the individual to injury. In other words, the authors describe decreased CRP variability as an indication of a non-healthy state. They suggest that injured runners may establish constrained movement patterns to avoid painful movement patterns.

In another study, Heiderscheit et al.[49] compared the variability of stride characteristics (stride duration and length) and joint coordination in eight female runners with patellofemoral pain and eight non-injured female controls. Bilateral 3-dimensional kinematic data were collected during treadmill running. Kinematic coupling variables included thigh rotation/leg rotation, thigh flexion/leg flexion, knee rotation/ankle inversion, knee flexion/ankle inversion and knee flexion/ankle dorsiflexion. Vector coding was used to examine coupling measures of interest and the average standard deviation of the data was used to examine variability. A three factor (group x limb x speed) repeated measures ANOVA was performed to compare the mean stride duration, mean stride length, coefficients of variation (CV) of stride duration and length and each coupling parameter. The authors found stride duration to be similar across both groups. Unlike the findings by Hamill et al.[48], these authors did not find significant differences in coordination variability between groups when measured across the stride cycle. However, when analyzed within specific stride regions, the patellofemoral pain group was found to have decreased variability in thigh rotation/leg rotation coupling near initial contact when compared to the non-injured control group. This finding was consistent with previous work[48], however, in this study, significance was only found in one of the multiple variables measured.

Only one study to-date has examined kinematic joint coupling in runners with ITBS.[45] This work utilized the CRP measure to compare variability between gait phases (stance and swing), discrete instances (initial contact), and fatigued state (fresh vs. fatigued) in healthy runners and runners with ITBS. The coupling variables of interest included: thigh abduction/adduction (abd/add) with tibial internal/external rotation

(IR/ER), thigh abd/add with foot inversion/eversion(inv/ever), tibial IR/ER with foot inv/ever, knee flexion/extension (flex/ext) with foot abd/add and knee abd/add with foot inv/ever. A 2x2 factorial ANCOVA with time and group as a factor, time as a repeated measure and speed as a covariate was utilized to examine the variability of the CRP measures. Study results showed no main effect of time indicating that variability did not change over the course of the run. During stance and swing, runners with ITBS did, however, show increased knee flex/ext-foot abd/add variability during fresh-state running. At the end of the run they demonstrated less stance-phase variability in thigh abd/add-foot inv/ever while variability of this measure was increased during swing. Thigh abd/add-tibial IR/ER variability was decreased in both the stance and swing phases of running gait. Additionally, swing-phase variability of thigh abd/add-foot inv/ever was decreased in runners with ITBS. The results of this study do not consistently support previous work by Hamill et al.[48] or Heiderscheit et al.[49] who have theorized that variability of movement patterns are decreased in injured runners. It does, however, demonstrate altered variability amongst runners with ITBS and support the need for further work in this area.

Research has primarily focused on kinematic coupling between the knee and the ankle while less attention has been paid to coupling at and between the hip and knee. During the stance phase of gait, hip adduction motion is coupled with hip and knee internal rotation. Alterations in these motions, have been documented in runners with ITBS, both during fresh-state, non-fatigued[25] and fatigued-state[27] running. If the excursion, velocity, or timing of joint motions is altered, normal kinematic coupling may change. Specific to the runner with ITBS, where alterations in hip adduction and knee internal rotation have been documented[25,27,45], discoordination in hip adduction and hip/knee internal rotation coupling patterns may result in greater torsional forces at the knee, placing increased strain on the iliotibial band. Further, findings that the motions of interest have been altered as a result of fatigue suggest that there may be some link between fatigue and altered joint coupling. While Miller et al.[45] did not find an effect of time on *variability* the CRP measure, the CRP measure cannot be used to compare the original kinematic time-series data, as vector coding will allow. To-date no studies have examined the effect of joint coupling on vector coding values at and between the hip and knee in runners with ITBS. Fatigue and its related kinematic alterations may result in abnormal joint coupling, alterations that are thought to lead to injury. Whether from altered kinematics or altered coupling patterns, both increased strain at the iliotibial band and increased contact with the lateral femoral epicondyle may lead to localized inflammation and progress to ITBS. Therefore, further knowledge is required regarding the kinematic coupling relationships at the hip and knee in runners with ITBS.

Electromyography

Background and Utility

Electromyography is a commonly used tool for the capture of neuromuscular electrical signal during tasks such as running or walking gait. The electromyographic signal is then analyzed to provide the investigator information about task-specific muscle function. During a muscle contraction, muscle fibers emit an electrical signal termed the motor unit action potential (MUAP). A MUAP is the most basic unit of the EMG signal.[50] Electrodes positioned over or within the contracting muscle detect each successive MUAP to provide scientists with EMG signal for the duration of the muscle contraction.

The EMG signal can be detected via surface or indwelling electrodes. Surface electrodes are placed on the skin over the muscle while fine-wire, or indwelling, electrodes are inserted via a needle into the muscle belly. Surface electrodes can be further divided into active or passive electrodes. Active electrodes house pre-amplifiers at the skin surface, a step that decreases impedance from the skin-electrode interface and improves electrical signal. Passive electrodes do not have this skin-surface pre-amplification and will not be discussed further in this document. Indwelling electrodes can be further divided into “needle” or “wire” configurations. Both needle and fine-wire electrodes contain insulated wires within a needle cannula, however, needle electrodes require that the needle remain in the muscle belly after insertion and for the duration of testing. Due to its limited use with dynamic activities such as running, the needle electrode will not be discussed further. The fine-wire electrodes allow investigators to insert and remove the needle cannula. When the needle is removed, the wire remains in the muscle for the duration of testing. The tip of the wire has no insulation and thus becomes the “detection” surface.[51] Once data collection is complete, the fine-wire is easily removed from the muscle.

There are advantages and disadvantages to both surface and fine-wire electrodes. As with many methodological choices, it is often these advantages and disadvantages that are weighed by the researcher and dictate electrode choice. The advantages of surface electrodes include the obvious non-invasive nature of the detection device and its ease of use. The major disadvantage to this electrode selection is its limited application for use over superficial muscles and its increased risk of detecting crosstalk from nearby muscles.[51] The advantage of using an indwelling electrode is its application for deeper muscles that would be unable to be accurately detected via surface electrode. The clear disadvantage of the indwelling electrode is its invasive nature. Additionally, once inserted, the fine-wire electrode cannot easily be repositioned if not correctly located. Therefore, participants are sometimes faced with more than one needle insertion prior to the determination that the needle is in the muscle of interest.

Depending on the research or clinical question being asked, there are multiple methods utilized to process raw EMG data. Graphically, the EMG signal is a series of sinusoidal waves whose amplitudes cross zero, resulting in both positive and negative values (Figure 1-4a). Because of this sinusoidal pattern and the random nature of the EMG signal, a simple mathematical mean has no meaningful interpretation.[51] Due to the biphasic nature of this waveform, processing the EMG signal involves a first step called rectification. Rectification either eliminates or inverts all negative signal leaving only positive values (Figure 1-4b).[51] The rectified signal can then be analyzed by integration (calculating the area under the signal), calculating means, calculating the root-mean-square (RMS) value or tabulating the number of times the signal crosses zero or has directional changes.[51] Using the Fast Fourier transform technique, the EMG signal can also be transformed from the time domain into the frequency domain. At this time, frequency analyses to determine the mean, median, and bandwidth are appropriate and meaningful.[51]

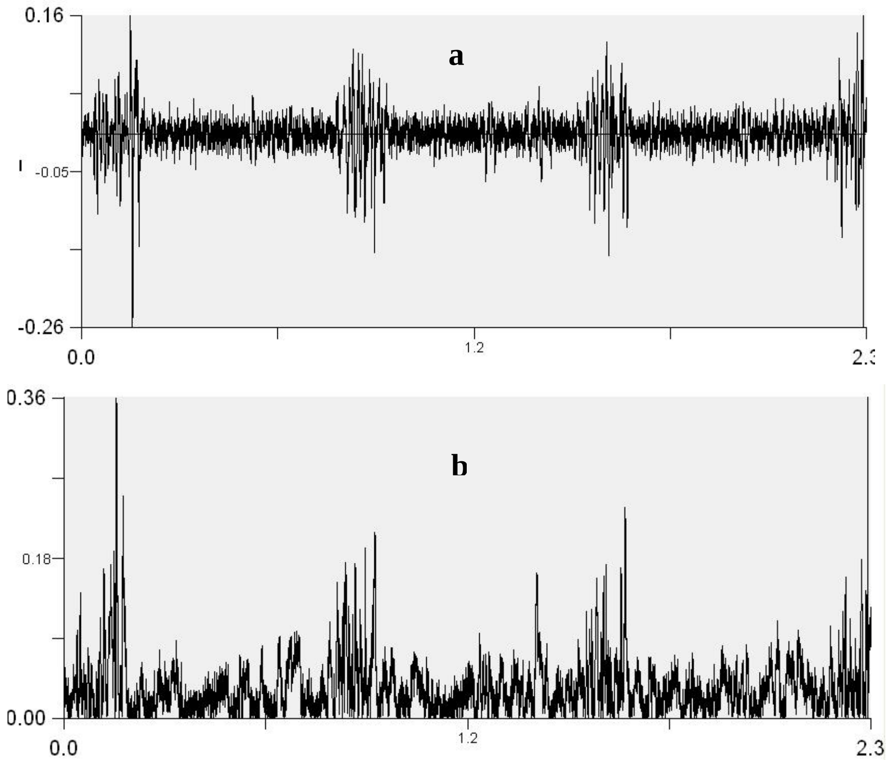

Figure 1-4. a. Representative raw EMG data. Sinusoidal pattern crosses zero with resultant positive and negative values. b. Representative rectified EMG data. Inversion of all negative signal leaves only positive values.

The EMG signal has three primary applications. The first two involve analyzing signal amplitude. Signal amplitude can be either used to describe muscle force production or to indicate activation/cessation timing. A third common use involves analyzing EMG spectral variables (frequency) and lends itself to document localized muscular fatigue.

Over the past several decades, the EMG signal has provided investigators with a wealth of information regarding normal and pathologic muscle activity. There are, however, multiple factors that can affect the EMG signal, resulting in incorrect data interpretation. Therefore, its application and interpretation must be done with caution.

When visually examining the EMG signal in conjunction with simultaneously collected force data, the relationship between signal amplitude and force generation is apparent. However, multiple factors can affect the EMG signal thus altering the force/signal relationship. Therefore, this relationship is not exact and in most cases should not be described as such in the literature. Factors affecting the EMG signal include but are not limited to: electrode application (i.e. location relative to the motor point, position relative to muscle fiber orientation), human physiology (i.e. blood flow, subcutaneous tissue between electrode and muscle, muscle fiber composition), and instrumentation issues (i.e. crosstalk, electrode specifications).[51] These factors can directly affect signal characteristics and make interpretation of the signal/force relationship difficult.

Determining muscular activation and cessation should also be done with caution. Of primary concern in this analysis is the effect of crosstalk. When capturing and analyzing EMG signal to determine the timing of a muscle's onset/cessation, the analysis of low-amplitude signal is necessary. Therefore, electrical activity from nearby muscles, or "crosstalk" may be detected and improperly analyzed.[51] Another factor to consider when calculating muscular activation and cessation is conduction velocity. Depending on the electrode location relative to the motor point (greater distance from the motor point increases signal to electrode time delay) and muscular fatigue, conduction velocity may affect your activation/cessation timing making comparisons between muscles or subjects difficult.[51]

A final utility of the EMG signal is the interpretation of spectral data as an indication of muscle fatigue. One characteristic of fatiguing muscle is a shift in the power spectrum to lower frequencies.[51,52] This frequency shift can be monitored during a sub-maximal isometric contraction to provide an index of muscle fatigue. This frequency shift, or compression, can be explained by shape alterations of the MUAP. Over the course of a sustained muscle contraction, the MUAP increases in duration.[51,52] Alterations in the shape of the MUAP (associated with increased MUAP duration) lead to a power spectral shift to lower frequencies. An additional mechanism that causes this spectral shift during an isometric contraction greater than 30% of maximal voluntary contraction is lactic acid accumulation within the muscle.[51] It has been documented in the literature that this frequency shift also results from synchronization in the firing of motor units.[52] More recently, however, this factor has been refuted. Arguments against the idea that motor unit synchronization leads to a frequency shift are most strongly based on the knowledge that during a muscle contraction, synchronization does not occur frequently or in a consistent enough nature to result in compression of the frequency spectrum.[51] While the mean or median frequency shift is a reliable tool to document fatigue in a muscle, as with analyzing EMG amplitude or activation/cessation, there are factors that will affect the MUAP shape and thus alter the median frequency value. These factors include, but are not limited to: electrode configuration (filtering parameters) and muscle physiology (i.e. muscle fiber diameter, intra-muscular temperature, blood flow, muscle fiber conduction velocity).[51] During anisometric contractions each of these factors are likely to affect MUAP shape. Therefore, the analysis of spectral modification to quantify muscle fatigue should only be done during isometric contractions.[51]

Electromyography Repeatability and Reliability

The repeatability of surface electromyography has been shown to be superior to fine-wire electromyography when measuring phasic muscle activity during gait.[53] Specifically, the reproducibility (repeatability of EMG measures within a test run) and reliability (repeatability of EMG measures within a session, between runs) of surface EMG was significantly higher than that of fine-wire EMG when measuring the rectus femoris, vastus lateralis, semitendinosis and gastrocnemius during overground walking. While still superior to wire electrodes, constancy (repeatability of EMG measures between test days) was fair for surface electrode data in this study. The authors analyzed repeatability using the variance ratio. This ratio measures the repeatability of waveforms with a value of zero indicating similar waveforms and a value of one indicating dissimilar waveforms. In this case, phasic muscle activity during gait is represented as a waveform and measured for similarity.

Kadaba et al.[54] documented good repeatability for the use of surface electromyography on muscles at the hip during overground walking. Specifically, the authors examined EMG data from the gluteus medius and gluteus maximus while collecting data from a total of ten lower extremity muscles. Data were collected three times each day, with three separate days of data collection each one-week apart. The coefficient of multiple correlations (CMC) was utilized as a measure of within- and between-day repeatability. The CMC is a tool to evaluate the similarity of waveforms used to express the gait cycle. When waveforms are similar, the CMC tends to 1, when dissimilar, it tends to 0. The authors report gluteus medius within-day CMC values of 0.854 ± 0.061 and between-day values of 0.838 ± 0.062. They also reported gluteus maximus within-day CMC values of 0.851 $\pm$ 0.060 and between-day values of 0.820 $\pm$ 0.084. Tensor fascia latae data were not collected in this study

There has been little documented as to the reliability and repeatability of EMG data during running. In one study, Gollhofer and colleagues[55] examined the within-trial, day-to-day and week-to-week reproducibility of the integrated EMG signal at the gastrocnemius and soleus muscles during treadmill running. They found reliability coefficients of the total integrated EMG activity ranging from 0.47 (week-to-week comparison of the gastrocnemius muscle) to 0.97 (day-to-day comparison of the soleus muscle). Within a trial, gastrocnemius and soleus muscle reliability was very good (0.94 and 0.95 respectively). In addition, six of the eight measures demonstrated good repeatability (reliability coefficients >0.84) of the gastrocnemius and soleus muscles. Smoliga et al.[56] also examined the reliability of lower extremity electromyography data during running. Integrated EMG, RMS, maximum M-wave value, and median power frequency were compared from the 9th to the 10th minute of EMG data collected from the vastus lateralis, semimembranosus, gluteus maximus and rectus femoris muscles during treadmill running. ICC values ranged from 0.394 (RMS EMG data from the vastus lateralis muscle) to 0.990 (median power frequency data from the semimembranosus). In general, the study found the vastus lateralis to have the poorest reliability (<0.80 for 3 of 4 measures) while the remaining three lower extremity muscles had considerably more reliability associated with them (<0.80 for 3 of 12 combined measures). Median power frequency was found to have the greatest reliability while RMS EMG data had the poorest reliability. While there are few studies that have examined the reliability of EMG data collection during running gait, these two above-mentioned studies show us that in general, good within-day, between-day and between-week reliability is attainable for the muscles of the lower extremity.

Electromyography During Running Gait

Whether to control motion at the hip or elsewhere, proper neuromuscular control with timely recruitment of lower extremity muscles is of great importance during running gait. Neuromuscular re-education is a commonly used treatment technique that focuses on improving muscular control of human motion. Improved muscular activation timing and altering abnormal movement patterns are often goals of this therapy.

While many studies have described what is “normal” lower extremity electromyographic activity and kinematic movement patterns during running, for the purpose of this document, only hip electromyographic patterns (gluteus maximus, gluteus medius, tensor fascia latae) will be discussed. During running gait, both the gluteus medius and gluteus maximus muscles are active from approximately 90%- 115% of the running gait cycle.[11,57] In the gluteus medius muscle, signal amplitude was not affected by increasing running speed.[11] In the gluteus maximus muscle, increased running speed resulted in a linear increased in amplitude.[11] Gazendam et al.[11] also documented a second peak of activity occurring during midswing (60-84% of the gait cycle) in the gluteus maximus and at the stance-swing transition (30-50% of the gait cycle) in the gluteus medius muscle. This second burst of activity was not supported in the findings of Mann et al.[57] As compared to the gluteus medius muscle, the tensor fascia latae (TFL) muscle has a delayed onset with earlier cessation of activity during pre-loading and loading

(95%- 112% of the gait cycle).[58] A second period of activity during early swing (40-50% of the gait cycle) was also documented.[58]

The three hip muscles examined above (gluteus maximus, gluteus medius and tensor fascia latae) were all active during pre-loading and through the loading portion of stance. It seems intuitive that activation of these stabilizing muscles in anticipation of weight acceptance is an important tactical move for runners. Proper activation timing of these muscles prepares the runner to accept weight and absorb shock during the period of running gait where the largest amount of vertical force is exerted on the body. This is likely a large factor in injury prevention. EMG analysis in runners thus becomes an excellent tool for the evaluation of hip muscle dysfunction, or to examine the effect of fatigue on muscle function. In an electromyographic study of the gluteus medius and tensor fascia latae muscles during walking, Gottschalk and colleagues[59,60] documented phasic activation of the gluteus medius muscle with the posterior fibers activating first, followed by the middle and then anterior fibers. This is followed by activation of the tensor fascia latae, which is most active during full stance. In a runner with impaired gluteus medius muscle function, this activation pattern may become altered with a delay in the activation of the gluteus medius muscle. A delay of the gluteus medius muscle may require earlier activation and increased demands on the tensor fascia latae muscle for the control of eccentric hip adduction. While both the tensor fascia latae and the gluteus medius muscles act to abduct and stabilize the hip, the tensor fascia latae is an internal rotator while the posterior fibers of the gluteus medius work to externally rotate the hip.[16] Delayed activation or weakness of the gluteus medius muscle, coupled with early activation of the tensor fascia latae muscle may result in net hip adduction and internal rotation as well as increased hip adduction moments. Increases in hip adduction and internal rotation will lengthen the iliotibial band, put undue strain on its fibers and may result in increased contact distally at the lateral femoral epicondyle (Figure 1-5). The resultant delay in activation may result in kinematic alterations (i.e. increased hip adduction) and thus diminish the system's shock absorption capacity. To date, no study has addressed the activation timing of the lateral hip musculature during running in individuals with ITBS. Further, no study has examined the effect of a fatiguing run on gluteus medius "fatigue resistance" (rate of the median frequency shift to lower frequencies) in this population. *Therefore, further investigation into this area is necessary.*

Gender Differences in Running

Female runners are twice as likely to suffer from ITBS and three times as likely to sustain a gluteus medius injury as their male counterparts.[5] Gender differences have not only been shown in such retrospective analyses of running injuries, but have also been shown to exist in lower extremity kinematics and kinetics during running and landing activities.[61-64] In one study examining running gait, Ferber et al.[61] found differences in both hip and knee kinematics between male and female runners. In their study, the authors examined hip and knee peak angles, peak moments, peak angular velocities and negative work during the first 60% of the stance phase of running gait. Participants ran along a 25-meter runway at a speed of 3.65m/s and were positioned such that they would strike a forceplate during the run. Female runners were found to exhibit significantly greater peak hip adduction angles, hip frontal plane negative work and hip adduction velocity than their male counterparts. In addition, female runners demonstrated significantly greater peak knee abduction angles, peak hip internal rotation angles, greater hip transverse plane negative work and peak hip external rotation velocity as compared to men. This well designed and thoroughly described study provides findings that are important when considering both design and interpretation of running studies, specifically when investigating ITBS. Gender differences have not been found exclusively during running tasks. Lephart et al.[64] reported similar findings when comparing lower extremity kinematics and vertical ground reaction forces during a single-leg landing task. Kinematic and kinetic data were collected on and compared between 15 male and 15 female athletes. Peak angular displacement and time to peak angular displacement for hip flexion, rotation and abduction as well as knee flexion and lower leg rotation were calculated. In addition, maximum vertical force and time to maximum vertical force were examined. Similar to the findings by Ferber et al.[61], Lephart et al.[64] discovered significantly greater hip internal rotation and decreased lower leg internal rotation during a vertical landing in female athletes. They also discovered decreased knee flexion in females as compared to males.

In addition to kinematic and kinetic gender differences, Zazulak et al.[65] discovered differences in hip muscle activity during a vertical landing task in female athletes. The authors examined gluteus maximus, gluteus medius and rectus femoris ensemble peak and ensemble mean electromyographic values in 13 female and 9 male athletes. Electromyographic data was analyzed in the 200ms prior to and the 250ms following landing. The authors found significantly decreased peak gluteus maximus activation in female athletes during the post-contact phase of landing. In addition, females demonstrated increased peak rectus femoris activation in the pre-contact phase of landing.

Based on the previously mentioned structural, kinematic and kinetic gender differences, participant gender must be taken into account when examining these variables. Given the documented gender differences as well as the increased prevalence of ITBS and gluteus medius injuries in the female running population, this study will be limited to female runners.

Summary of Current Literature

Running is a popular form of exercise due to its cardiovascular and psychological benefits as well as its convenience and low cost. However, running is a high impact and repetitive sport with injuries affecting more than 50% of its participants on an annual basis.[2] Iliotibial band syndrome (ITBS) is the second most common running injury.[5] Many factors are thought to contribute to its development including faulty mechanics, strength deficits, fatigue and neuromuscular dysfunction. When running, forces twice the runner's weight are exerted on their body with each foot strike.[4] The result of this force is a greater demand on lower extremity muscles for proper shock absorption as evidenced by increased EMG amplitudes seen during running.[9-13] This demand is particularly important at the abductor muscles of the hip that are required to overcome large external adductor moments during the loading phase of running gait. Failure to stabilize at the hip, whether based on decreased strength, muscle fatigue, or altered neuromuscular recruitment, may have consequential effects up and down the kinetic chain. These effects can include alterations in joint kinematics, kinetics, or joint coupling including increased hip adduction or knee internal rotation motion, such as seen in runners with ITBS.[25,31,49,66] Kinematic alterations such as these may lengthen the iliotibial band, put undue strain on its fibers, and result in increased contact distally at the lateral femoral epicondyle (Figure 1-5).

Fatigue is a likely contributor to a runner's inability to maintain adequate stability at the hip and subsequently the development of ITBS. When examined in a general running population, fatigue has been shown to result in kinetic, kinematic and electromyographic changes.[27,39-42]. Only two studies to-date have examined the effects of fatigue on runners with ITBS and both have documented resultant kinematic changes.[27,45] Additionally, when examining the healthy athlete, some noted fatigue-related kinematic alterations, such as delayed muscle activation[39] and altered force attenuation[3], are thought to put runners at risk for injury. To further magnify the problem, fatigue has been shown to alter running biomechanics in as little as 15 minutes of running[41], impacting both casual and long distance runners. Maintaining proper hip mechanics in the face of fatigue thus seems a reasonable goal for the prevention and treatment of ITBS. However, prior to making this statement with any certainty, further examination into the effects of fatigue on hip mechanics in female runners with ITBS is necessary.

It has been well documented that there are gender differences with respect to lower extremity biomechanics and injury. In addition, many of these differences have been documented at the hip, with female runners being twice as likely to develop ITBS and three times as likely to sustain a gluteus medius injury than their male counterparts.[5] During running, hip joint kinematics have been shown to differ between male and female runners with females exhibiting greater hip adduction and internal rotation angles.[61]

Based on the documented gender differences as well as the increased prevalence of ITBS and gluteus medius injuries in the female running population, this study will be limited to female runners.

Figure 1-5 represents the conceptual framework of this study. There are two primary study objectives. The first will be to will be to compare fresh-state hip muscle strength, electromyographic and joint coupling data between runners with ITBS and healthy runners. The second will be to examine the effects that fatigue has on hip muscle strength, electromyography and hip joint kinematics, kinetics, and coupling in runners with ITBS as compared with uninjured runners.

Specific Aims

Specific Aim 1: To determine whether differences exist between runners with ITBS and healthy controls with respect to gluteus medius function (strength, fatigue resistance, and electromyographic onset activation timing), tensor fascia latae activation timing and kinematic joint coupling.

Hypothesis 1.1: Runners with ITBS will exhibit significantly different gluteus medius and tensor fascia latae activation timing as compared to healthy controls during speed-controlled overground running.

Hypothesis 1.2: Runners with ITBS will exhibit significantly different gluteus medius fatigue resistance and gluteus medius strength during isometric testing as compared to healthy controls during speed-controlled overground running.

Hypothesis 1.3: Runners with ITBS will exhibit significantly altered hip frontal-hip transverse plane and hip frontal-knee transverse plane kinematic joint coupling as compared to healthy runners during speed-controlled overground running.

Specific Aim 2: To determine the effect of performing a run to exertion (17/20 rating on Borg's Rating of Perceived Exertion) on hip joint kinematics and kinetics, and joint coupling in runners with ITBS as compared to healthy controls during speed-controlled overground running.

Hypothesis 2.1: As a result of exertion, runners with ITBS will have a greater change in stance phase peak hip adduction and internal rotation as compared to healthy controls during speed-controlled overground running.

Hypothesis 2.2: As a result of exertion, runners with ITBS will have a greater change in stance phase peak hip abductor and external rotator moments as compared to healthy controls during speed-controlled overground running.

Hypothesis 2.3: As a result of exertion, runners with ITBS will have a greater change in stance phase hip frontal-hip transverse plane and hip frontal-knee transverse plane kinematic joint coupling as compared to healthy controls during speed-controlled overground running.

Specific Aim 3: To determine the effect of performing a run to exertion (17/20 rating on Borg's Rating of Perceived Exertion) on gluteus medius function and tensor fascia latae activation timing in runners with ITBS as compared to those in healthy controls.

Hypothesis 3.1: As a result of exertion, runners with ITBS will have a greater change in gluteus medius and tensor fascia latae activation timing during overground running.

Hypothesis 3.2: As a result of exertion, runners with ITBS will have a greater change in gluteus medius fatigue resistance and gluteus medius strength during isometric testing.

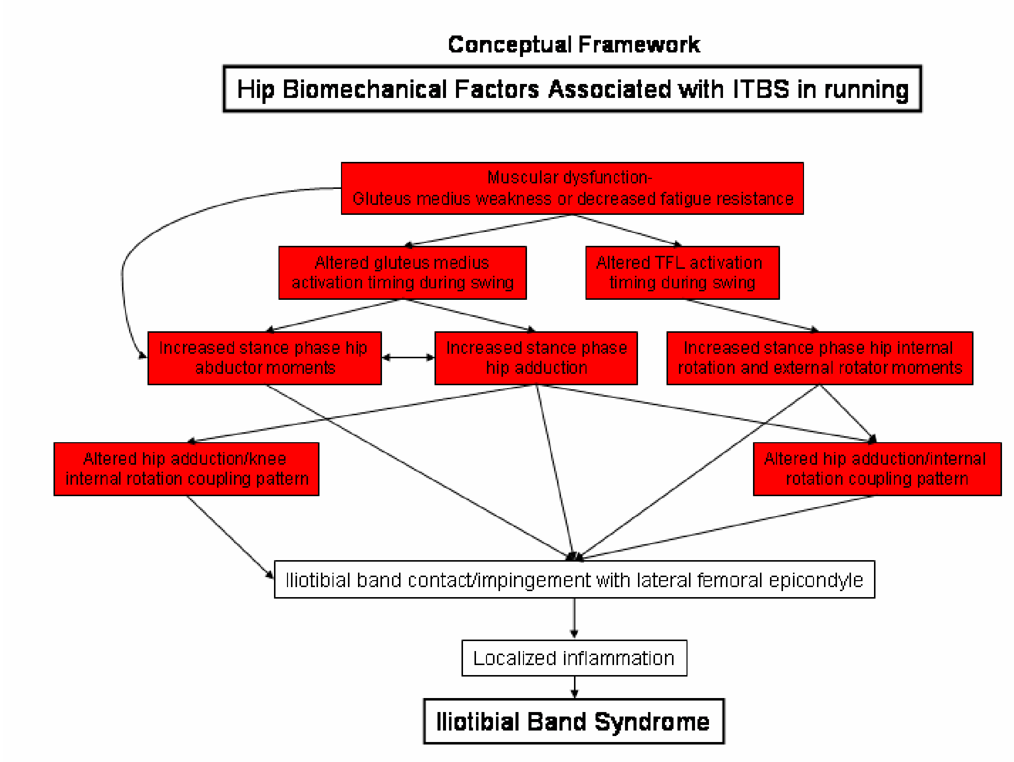

Figure 1-5. Conceptual framework. Fatigue's effect on muscular function and subsequent lower extremity alterations in runners with ITBS. Red boxes indicate factors to be examined in this study.

CHAPTER 2

HIP MUSCLE FUNCTION AND KINEMATIC JOINT COUPLING IN FEMALE RUNNERS WITH ILIOTIBIAL BAND SYNDROME

Introduction

Ilitobial band syndrome (ITBS) is the second most common running injury and the leading cause of lateral knee pain in runners.[5] The iliotibial band (ITB) is a fibrous sheath extending down the lateral side of the femur and continuing until its insertion into Gerdy's Tubercle.[14] The pathogenesis of ITBS involves an irritation and resultant inflammation deep to the posterior fibers of the iliotibial band at the location of the lateral femoral epicondyle.[20,21] There have been multiple theories as to the cause of this irritation. Originally, this irritation was proposed to be caused by back and forth rubbing of the ITB over the lateral femoral epicondyle during repetitive knee flexion and extension, such as seen during running.[18] At approximately 30° of knee flexion, the iliotibial band is thought to move from its position anterior to the lateral femoral epicondyle, to a more posterior position. During this motion, friction is thought to occur between the posterior fibers of the ITB and the lateral femoral epicondyle.[14] This friction causes a localized irritation and thus results in the complaints of lateral knee pain commonly associated with ITBS. During running, knee flexion up to 45 degrees can occur during loading response[7], thereby increasing the likelihood of ITBS in this population of athletes. The, "friction theory" has resulted in ITBS also being termed iliotibial band friction syndrome. Early literature in support of the friction theory, reported the presence of a bursa over the lateral femoral epicondyle.[18] However, more recently, MRI and cadaveric studies have not supported these findings.[20-22] An alternate theory suggests that *compression* rather than friction is occurring between the ITB and the lateral femoral epicondyle.[22] A cadaveric study showed that there was adipose tissue containing Pacinian corpuscles, deep subcutaneous mechanoreceptors that sense global pressure[23], beneath the iliotibial band.[22,67] While there is not sufficient evidence to confirm either theory, it is well-accepted that the pathogenesis of ITBS involves a localized irritation and inflammation deep to the ITB at the location of the lateral femoral epicondyle.

While symptoms of ITBS present themselves at the lateral knee, recent findings in the literature have found many associated impairments occurring at the hip. Weakness in the hip abductor and external rotator muscles[28,29] and increased peak hip abductor moments (reflecting decreased ability to control femoral adduction)[30] have been documented in runners with ITBS. Furthermore, Fairclough et al.[22] have suggested that there is a relationship between impaired function of the hip musculature and ITBS. They propose that a primary dysfunction of the lateral hip muscles will likely manifest as abnormal hip kinematics resulting in compressive forces on the tissues beneath the ITB and secondary pain over the lateral femoral epicondyle. During the typical loading response of running, vertical ground reaction forces can reach 2.2 times body weight in 23 milliseconds[4] with resultant hip adduction motion.[7] At this time, a large contribution from the hip abductors, specifically the gluteus medius muscle, is required to absorb this shock and overcome large external adduction moments.[7] Additionally, the tensor fascia latae and the upper fibers of the gluteus maximus assist with hip abduction.[16] Inability of the hip muscles to counter this external adduction moment, whether due to weakness or neuromuscular dysfunction, is suggested to be a factor in the development of ITBS.[22,29] In fact, both prospective and retrospective studies have documented increased stance phase hip adduction angles in runners with ITBS[25,32] with hip abductor weakness as a proposed contributor[25,29,32].

In addition to a muscle's strength, its capacity to resist fatigue may also play a role in its ability to function. One characteristic of fatiguing muscle is a measured shift of the EMG power spectrum to lower frequencies during an isometric contraction.[52,68] This frequency shift can be monitored during a sub-maximal isometric contraction to provide an index of muscle fatigue, termed "fatigue resistance". The power spectral shift to lower frequencies can be explained by shape alterations of the motor unit action potential (MUAP), whereby the MUAP increases in duration and thus changes its shape over the course of a sustained muscle contraction.[52,68] Additional mechanisms that are proposed to cause this spectral shift include lactic acid accumulation within the muscle[68] or synchronization in the firing of motor units[52]. Whether as a result of motor unit synchronization, lactic acid accumulation, or shape alterations in the MUAP, measurement of the power spectral shift can serve as a useful index of a muscle's ability to resist fatigue.

Another area of biomechanical consideration in runners with ITBS is regarding the coupling of motions that occur between joint motions. Abnormal joint coupling and variability of coupling is thought to contribute to injuries in the athletic population.[45,48,49] Miller and colleagues have examined the *variability* of joint coupling in runners with ITBS, however, no study has reported on the joint coupling *patterns* in a similar cohort of runners. During running gait, hip adduction is naturally coupled with both hip and knee internal rotation. Studies have documented increased knee internal rotation velocity[27] as well as increased peak hip adduction and knee internal rotation angles[25] in runners with ITBS. Discoordination in the hip adduction/hip internal rotation or hip adduction/knee internal rotation coupling pattern may result in greater torsional forces at the knee, place increased strain on the iliotibial band and/or increased contact between the ITB and the lateral femoral epicondyle. Each of these biomechanical alterations may lead to localized inflammation and progress to ITBS.

While researchers have examined hip abductor strength in runners with ITBS, these measures are taken during isometric or isokinetic contractions and lack the functional specificity of data collected during running itself. To-date, no studies have reported on gluteus medius onset activation timing during overground running in individuals with ITBS. Additionally, while fatigue is likely to play a role in the capacity of the gluteus medius to resist hip adduction, there is a lack of literature to describe how its fatigue resistance differs in individuals with ITBS. Finally, the lack of knowledge regarding joint coupling in runners with ITBS, warrants further investigation into this area. Therefore the objective of this study was to determine whether differences exist between runners with ITBS and healthy controls during speed-controlled overground running with respect to gluteus medius function (strength, fatigue resistance, and electromyographic onset activation timing), tensor fascia latae activation timing and kinematic joint coupling at and between the hip and knee. Three hypotheses were stated for this study. First, it was hypothesized that during overground running, individuals with ITBS would exhibit significantly different gluteus medius and tensor fascia latae activation timing during terminal swing as compared to healthy controls. The second hypothesis was that runners with ITBS would exhibit significantly different gluteus medius isometric strength and fatigue resistance (smaller linear slope of median frequency vs. time plot) during isometric testing as compared to healthy controls. Lastly, it was hypothesized that runners with ITBS would exhibit significantly different hip frontal-hip transverse plane and hip frontal-knee transverse plane kinematic joint coupling than healthy controls.

Methods

Participants

Female runners are twice as likely to suffer from ITBS and three times as likely to sustain a gluteus medius injury in comparison to their male counterparts.[5] Therefore, 20 healthy female runners (28.9 ±6.1 yrs; 1.6± 0.09 m; 56.8± 5.2 kg) and 12 female runners with a current diagnosis of ITBS (32.4± 7.9 yrs; 1.7 ± 0.06 m; 60.6± 5.0 kg) were included in this study. While the groups did not differ with respect to age (P=0.17), runners in the ITBS group tended to be taller (P=0.01) and have greater mass (P=0.06) than the control runners. All participants were (1) female; (2) aged 18-50 years old; (3) rearfoot strikers; (4) running ≥ 15 miles/week; and (5) able to run one at least 9-minute mile. Additionally, all runners were free from all neuromuscular and musculoskeletal disorders for 6 months prior to data collection, with the exception of current symptoms of ITBS in the injured runners. All injured runners received a diagnosis of unilateral ITBS by a doctor or physical therapist. This protocol was approved by the Hospital for Special

Surgery and Temple University Institutional Review Boards. Prior to data collection, informed consent was obtained from each participant.

Procedures

EMG Instrumentation

For the capture of fatigue resistance and muscular onset timing, EMG data were collected using a 16 channel MA-300 EMG System (Motion Lab Systems, Baton Rouge,LA). Disposable surface gel electrodes with a 20mm interelectrode distance were placed on the gluteus medius and tensor fascia latae muscles of the injured limb in the ITBS group or the dominant limb of the control group. Lower extremity dominance was established as the limb that the runner reported they would use to kick a soccer ball. This self-report method of determining lower extremity dominance was found to have 97.7% agreement with task performance and a 96% test-retest agreement {{145 Coren, Stanley 1978}}. Electrodes were applied in parallel with the direction of the muscle fibers and in locations as described by Perotto et al.[69](Table 2-1). Once in place, the electrodes were connected via snap leads to the MA-411 EMG pre-amplifier (Motion Lab Systems, Baton Rouge,LA). Correct electrode placement was verified through resisted muscular testing as described by Perotto et al.[69](Table 2-1). EMG data were sampled at 4800 Hz, bandpass filtered at 10-2000Hz and pre-amplified with a x20 gain. For the purposes of determining muscular onset timing, a 5- second maximal voluntary isometric contraction (MVIC) and a 2-second resting trial were acquired prior to further testing.

Table 2-1. EMG Electrode location and placement verification activity[69]

Muscle	Electrode location	Placement verification activity
Gluteus Medius	One inch distal to the midpoint of the iliac crest	Thigh abduction with the participant sidelying
Tensor Fascia Latae	Two fingerbreadths anterior to the greater trochanter	Thigh abduction with hip flexion

Strength Testing

Isometric gluteus medius strength testing was performed using the Biodex System 4 (Biodex Medical Systems, Shirley, NY). As described in Kendall and McCreary[16], participants were positioned in sidelying with the bottom leg flexed at the hip and knee. So as to best isolate the gluteus medius from the rest of the hip abductors (tensor fascia latae, gluteus minimus), the participant's pelvis was rotated slightly forward with the test limb held in a position of approximately 15° hip abduction with slight extension. The dynamometer attachment was positioned 3cm proximal to the lateral femoral epicondyle and participants were stabilized using a strap positioned at the level of their pelvis. Three 5-second MVIC contractions were performed with 30-second rests between trials.

Fatigue Resistance

Following the collection of MVIC strength data, measures of gluteus medius fatigue resistance were collected on the dominant limb of control runners and on the injured limb of runners with ITBS. Between strength and fatigue resistance testing conditions, participants remained in the Biodex machine so their inter-test position remained the same. Participants were asked to perform a 60-second isometric hip abduction contraction at 50% of their previously determined MVIC strength measure while EMG data of the gluteus medius muscle were collected. During data collection, visual feedback displayed on the Biodex computer monitor in the form of a line graph was given to assist participants in the maintenance of a 50% MVIC contraction.

Running Trials

Following the capture of strength and gluteus medius fatigue resistance data, runners were given a minimum 5-minute rest while they were prepared for the collection overground running data. During that time, passive retroreflective markers were placed on specified locations at the participant's trunk, pelvis and lower extremities (Figure 2-1).

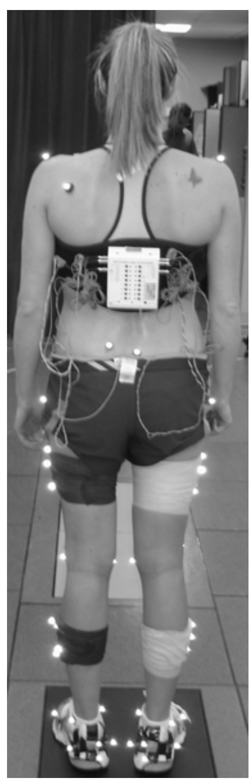

Figure 2-1. Six-degree of freedom marker set with electrodes and EMG pack in place.

A 12-camera Motion Analysis Corporation system (Motion Analysis Corporation, Santa Rosa, CA) was utilized for calibration of the field and capturing three-dimensional digitized data. Video data were collected at 120Hz. During overground running data collection, participants wore neutral, laboratory-provided running shoes (New Balance 1061, New Balance; Boston, MA). To begin overground running data collection, participants were asked to stand at the center of the data collection volume for the capture of a static calibration trial. They were then asked to move their hip through an approximate 20° arc of abduction/adduction and flexion/extension motion for the identification of a functionally-determined hip joint center[70]. Next, markers used solely for anatomical definitions were removed so that only tracking markers remained during running trials. Participants then ran along a 30 m runway through the data capture volume for the collection of five acceptable overground running trials. An acceptable trial was defined as one where EMG signal quality was good (i.e. no noise or motion artifact), kinematic data were available for the identification of gait events and calculations of kinematic joint coupling values, and the runner maintained a speed of 3.35 m/sec (±10%). To ensure consistency across trials, velocity was recorded with two photoelectric timers placed 4 meters apart. When the runner broke the first photoelectric beam, a timer began. The timer then turned off when the runner broke the second photoelectric beam providing real-time feedback to the investigative team. If necessary, feedback was then provided to the runner for them to adjust their speed to the desired velocity.

Data Analysis

Strength

Using Biodex System 4 software, peak hip abductor torque values were averaged across the three isometric strength-testing trials and the mean utilized for data analysis. As mentioned previously, data from the injured limb of participants with ITBS and from the dominant limb of control participants were utilized for data analysis.

Fatigue Resistance

Several of the participants were not able to sustain an isometric contraction at 50% of their MVIC for 60 seconds. Once they relaxed their hip abductor muscles, the frequency of the muscle contraction would vary and affect the EMG signal's frequency content. Therefore, only the first 30 seconds of the fatigue resistance data collection were analyzed.

Using custom code written in MatLab (The MathWorks, Natick, MA), raw EMG data were initially processed in 213 ms time segments (bins) and transformed into the frequency domain using the fast Fourier transform technique. The median power frequencies were then obtained for 1.91second bins of data and plotted versus time. The linear slope of this plot was obtained and used to express the gluteus medius "rate of fatigue" (Figure 2-2). This methodology has been thoroughly described by Kondraske et al.[71] and Naeije et al.[52]. Its accuracy in detecting the myoelectric spectral shift has been verified by DeAngelis et al.[72]

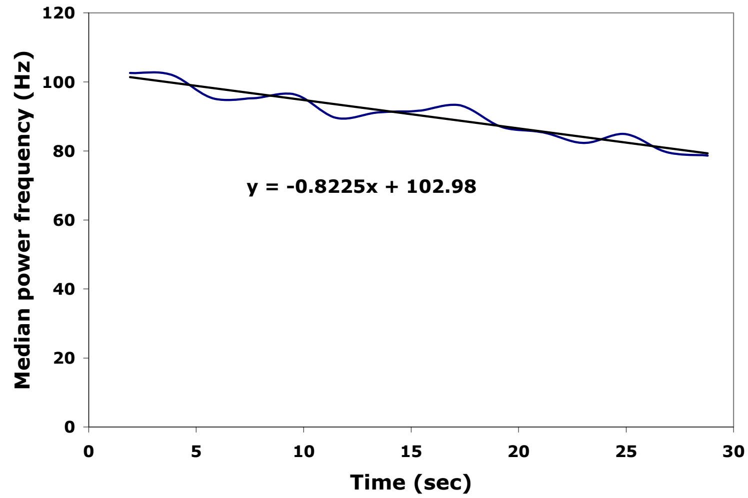

Figure 2-2. Representative plot of median power frequency vs. time. Linear slope of this curve (-0.8225) indicates gluteus medius "rate of fatigue".

Muscle Activation Timing

MVIC, resting and overground running EMG data were processed and analyzed using a customized code written in Visual 3-D (C-Motion, Inc; Rockville, MD). To eliminate baseline voltage offset, the mean of the raw EMG signal was subtracted on a trial-by-trial basis. EMG data were then full-wave rectified and a linear envelope was created using a low pass second-order Butterworth filter with phase correction and a frequency cutoff of 20Hz. To determine a threshold for the onset of muscle activity, the peak activity from all trials (MVIC and dynamic running trials), and the mean EMG value of the resting trial were calculated. Ten percent of the difference between the maximum value and the mean resting value was then added to the mean resting value and considered the threshold value. During a running trial, when EMG activity ascended above this threshold value, the muscle was considered "on". Gluteus medius and tensor fascia latae onset during terminal swing, expressed as a percent of the gait cycle, was utilized as the outcome of interest.

Kinematic Joint Coupling

Joint coupling motions of interest included hip abduction-adduction/hip internal-external rotation and hip abduction-adduction/knee internal-external rotation. Coupling variables were chosen based on the thought that aberrations in these motions would lengthen the ITB and cause increased compression at the level of the lateral femoral epicondyle or increased strain within the structure itself.

To account for the varying functional demands over the stance phase of running gait, coupling was examined over four specific phases of stance: the first and second halves of both loading and propulsion. Loading was functionally defined from initial contact to the time of first peak knee flexion. Propulsion was functionally defined from the time of first peak knee flexion to toe off. Vector coding was used to describe the magnitude of joint coupling between the motions of interest. As described by Heiderscheit et al.[49] and Dierks et al.[47], an angle-angle diagram (Figure 2-3) is constructed from the two motions of interest. Using the equation, $\Theta = |\tan^{-1}(y_2\text{-}y_1/x_2\text{-}x_1)|$, angles of the trajectory between two successive data points with respect to the horizontal were calculated for all data points over the period of interest (i.e. first half of loading phase) (Figure 2-4). The mean angular value for the period of interest is then calculated. This provides a continuous excursion ratio coupling angle with a range of 0°-90°. A 45° angle indicates equal amounts of movement between the two segments. An angle greater than 45° indicates greater amounts of distal motion while an angle less than 45° indicates greater amounts of proximal motion.[46,47,49] In the case of hip abduction/adduction and hip internal/external rotation, hip abduction/adduction is defined as the proximal motion.

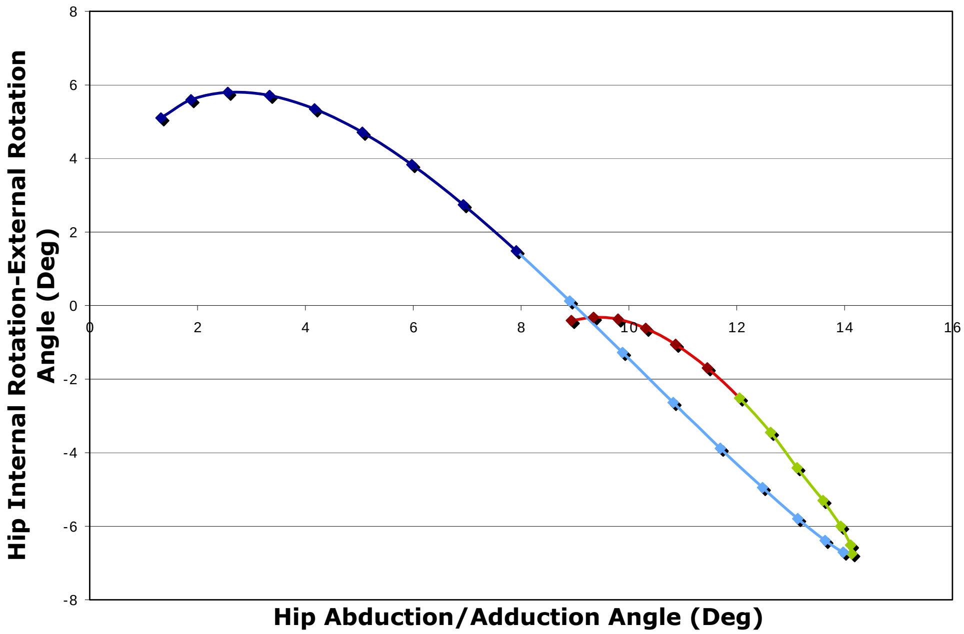

Figure 2-3. Angle-angle plot constructed from hip abd/adduction and internal/external rotation motion from one representative trial. Red and green data represent first and second halves of loading respectively. Light and dark blue data represent first and second halves of push-off respectively.

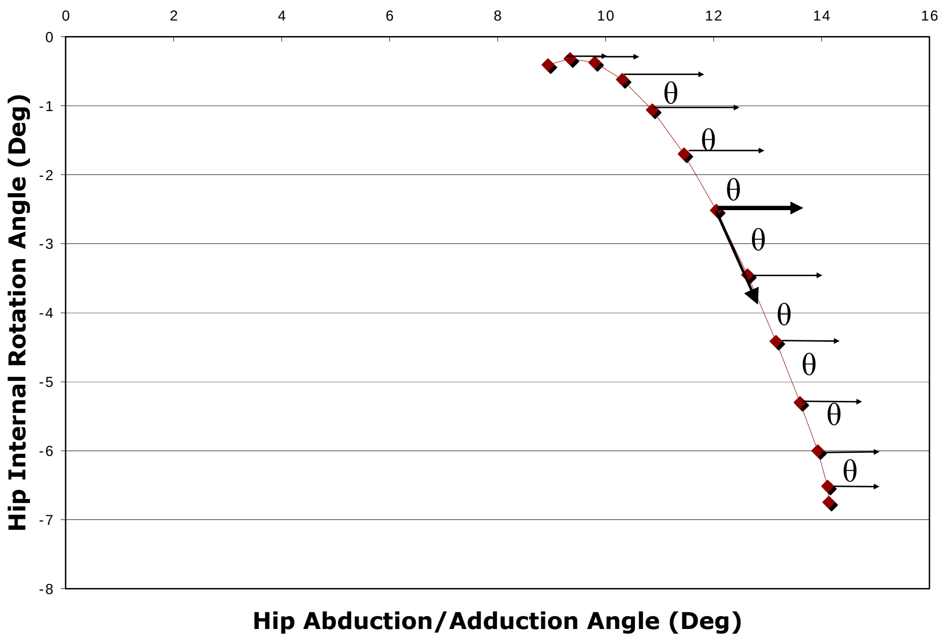

Figure 2-4. Angle-angle plot created during loading phase of running. Vector coding angular values Θ are calculated from trajectories between each successive data point with respect to horizontal. Values are then averaged over the time period of interest.

Statistical Analysis

Two-tailed independent t-tests were used to compare strength, EMG and joint coupling variables of interest between runners with ITBS and healthy runners. An a-priori significance level of $p<0.05$ was established for all analyses, and a trend was operationally-defined at $0.05<p\leq0.10$.

Results

Gluteus Medius Strength

There was not a significant difference in hip abduction strength between runners with ITBS and uninjured runners (P=0.59). On average, isometric hip abductor peak torque values were 78.2 ± 14.3 N-m for the injured runners and 75.7 ± 12.5 for the uninjured control runners.

Gluteus Medius Fatigue Resistance

Due to a methodological error, fatigue resistance data from 4 uninjured control runners were collected at sampling rates of ≤ 1200Hz. Due to possible undersampling of data and to avoid any aliasing, these data points were removed from analysis. Therefore, the fatigue resistance analysis was conducted on a sample of 16 uninjured runners and 12 runners with ITBS. Even at this smaller sample size, there was a significant difference in the "rate of fatigue" (linear slope of the median power frequency vs. time plot) between runners with ITBS and their healthy counterparts (P=0.01). The mean linear slope of the median power frequency vs. time plot was -0.996 ± 0.51 in injured runners as compared with -0.546 ± 0.35 in uninjured runners. The steeper slope seen in runners with ITBS indicates that their gluteus medius muscle demonstrates characteristics of fatigue at a higher rate than uninjured runners.

EMG Onset

Due to the high impact nature of running, raw EMG data from each runner were examined for the presence of motion artifact prior to data analysis. Based upon this visual examination, gluteus medius data from 2 and tensor fascia latae data from 3 uninjured control runners were subsequently excluded from statistical analyses. Means and standard deviations for terminal swing onset activation of the gluteus medius and tensor fascia latae muscles are provided in Table 2.2. With a sample size of 18 uninjured control runners and 12 runners with ITBS, our data did not demonstrate a significant difference in gluteus medius terminal swing activation timing between groups (P=0.38). Similarly, with 17 uninjured controls and 12 runners with ITBS, our data did not reveal a significant difference in tensor fascia latae terminal swing activation timing between groups (P=0.41).

Table 2-2. Gluteus medius and tensor fascia latae activation timing during terminal swing expressed as a percent of the gait cycle.

	Gluteus Medius -% gait cycle, mean (SD)	Tensor Fascia Latae- % gait cycle, mean (SD)
ITBS	90.1 (7.7)	95.3 (6.0)
Uninjured Controls	92.2 (5.0)	93.5 (6.3)

Kinematic Joint Coupling

There were no significant differences in hip abduction/adduction- hip internal/external rotation or hip abduction/adduction- knee internal/external rotation joint coupling values between runners with ITBS and healthy controls across the defined phases of stance. During the second half of loading, however, a trend (P=0.06) was noted in hip frontal-knee transverse plane kinematic joint coupling. During this phase of stance, runners with ITBS demonstrated coupled motion at a nearly 1:1 ratio (coupling angle of 44.8º ± 15.6º) while healthy runners' demonstrated a tendency towards increased transverse plane knee excursion (coupling angle of 55.0º ± 13.9º). Table 2-3 shows vector coding means and standard deviations as well as significance values for both healthy and injured runners across the phases of stance.

Table 2-3. Kinematic joint coupling means and standard deviations for hip abduction/adduction (abd/add) –hip internal/external rotation (IR/ER) as well as hip abd/add- knee IR/ER across the phases of stance. P values indicate between group measures for each phase of stance.

		First Half Loading		Second Half Loading		First Half Propulsion		Second Half Propulsion	
Hip abd/add-Hip IR/ER vector coding values (°)	ITBS mean (SD)	43.6(13.7)	P=0.72	40.6(14.5)	P=0.55	21.3(9.9)	P=0.54	40.2(11.3)	P=0.09
	Control mean (SD)	41.7(14.6)		43.7(13.7)		23.4(8.7)		33.1(10.7)	
Hip abd/add-Knee IR/ER vector coding values (°)	ITBS mean (SD)	58.5(13.9)	P=0.45	44.8(15.6)	P=0.06	21.0(7.3)	P=0.30	53.5(14.8)	P=0.91
	Control mean (SD)	54.5(15.3)		55.0(13.9)		26.0(15.4)		52.9(15.1)	

Discussion

While symptoms of ITBS typically occur at the knee, dysfunction originating at the hip has come forth in recent years as a potential etiologic factor in the syndrome's development. This study examined hip abductor strength, fatigue resistance and activation timing as well as kinematic joint coupling in female runners with ITBS and their healthy counterparts.

Previous studies have examined hip muscle strength in runners with ITBS, however, findings have been contradictory[28,29,73] leaving researchers and clinicians without conclusive evidence. Our current study found that hip abductor strength in the involved limb of runners with ITBS was not significantly different from that of a non-injured control group. These findings were consistent with those of Grau et al.[73], who, using similar testing methodology, found there to be no differences in isometric or isokinetic strength between runners with ITBS and healthy controls. Our findings are, however, in contrast to previous studies[28,29], which have documented decreased hip abductor strength in runners with ITBS or in a cohort of injured runners *including* those with ITBS. In our study, as well as in the study by Grau and colleagues, an isometric torque-testing machine was utilized for strength testing. This is as compared to a handheld dynamometer, which was utilized in the contrasting studies. Isometric torque testing devices have been shown to have higher intrarater and interrater reliability than hand-held dynamometers.[74] Hand-held dynamometers are limited by the strength of the tester and, even if utilized with stabilization straps, are subject to error if any shifting in the dynamometer head occurs during the isometric contractions. These limitations and methodological differences may explain the contrasting study findings. An additional factor that may explain differences in study findings was that participants in our study were not excluded based on participation in physical therapy or performance of any hip-strengthening regimen. While only one participant had initiated physical therapy at the time of data collection, the remaining participants were not screened to determine if they were performing any targeted hip abductor exercises independent of a therapist's guidance. While this is a potential limitation of our study, it is also a likely representation of the true clinical population that physicians, physical therapists or athletic trainers may see in their facilities. It has been suggested that weakness of the hip abductor muscles may result in increased hip adduction during the stance phase of running gait.[25,29] Based on our study findings, hip abductor strength deficits may play less of a role in the presentation of ITBS than previously thought.

While differences in isometric hip abduction strength were not found between runners with ITBS and healthy controls, there was a significant difference in the ability of the runners' gluteus medius muscle to resist fatigue. Our study findings suggest that the hip abductors of runners with ITBS demonstrate less resistance to fatigue then hip abductors of healthy runners. Based on the clinical presentation of ITBS, the idea that the injured runners' hip abductors are not "weaker" but less resistant to fatigue is of particular interest. Symptoms of ITBS tend not to be present at the initiation of a run, but arise and/or worsen after 20-30 minutes of running. These results, combined with the clinical presentation or ITBS, suggest that hip abductor endurance or fatigue resistance may play a larger role in the syndrome's etiology than strength alone.

Strength and fatigue resistance data were collected during isometric contractions. The gluteus medius muscle, however, functions in an eccentric capacity during loading response. Therefore, an additional purpose of this study was to examine the activation timing of the gluteus medius and tensor fascia latae muscles during terminal swing as the muscle is preparing itself for the large eccentric demands placed on it during loading response. Our study found that terminal swing gluteus medius activation occurred, on average, at 90 and 92% of the gait cycle in injured and uninjured runners respectively. The tensor fascia latae muscle activated at 95 and 94% of the cycle in the same groups. These data are consistent with Mann and colleagues' findings that the gluteus medius and tensor fascia latae muscles activate at 90 and 95% of the gait cycle respectively.[57] Proper activation timing of these muscles prepares the runner to accept weight and absorb shock during the period of running gait where the largest amount of vertical force is exerted on the body. Contrary to our hypothesis, gluteus medius and tensor fascia latae terminal swing activation timing were not significantly different between injured and healthy runners during fresh-state, non-fatigued running. It was expected that runners with ITBS would demonstrate delayed activation timing, which would result in increased hip adduction angles during stance. Our data, however, do not suggest that hip abductor activation timing is associated with injury status. It is possible that other neuromuscular mechanisms such as the duration or amplitude of activation are different between groups; however, these variables were not examined in this study. In addition, these data were collected with runners in a non-fatigued state. It is also possible that fatigue may induce neuromuscular changes in activation timing that are not seen during fresh-state running. This will be examined in future analyses

It has been proposed that abnormal joint coupling and variability of coupling is related to overuse running injuries.[45,48,49] Therefore, this study examined whether differences in hip joint coupling existed between female runners with ITBS and an uninjured control group. When comparing between groups, there were no significant differences in hip frontal- hip transverse plane or hip frontal- knee transverse plane joint coupling values. Based on results from recent studies documenting altered hip adduction and knee internal rotation angles in runners with susceptibility for ITBS[25,32,34], differences in joint coupling were expected between the experimental groups. Yet, the absence of a difference between coupling values in the two groups were not in support of our original hypothesis and suggest that any kinematic alterations in the injured runners did not result in changes in the degree of joint coupling occurring between and within joint motions. It is possible that within the injured population, changes in joint kinematics are occurring simultaneously between the motions of interest and therefore are not affecting the degree of coupling between motions. By design, the vector coding technique is utilized to examine joint coupling as a continuous measure, a means with which to provide investigators and clinicians a description of joint coupling across the gait cycle. In contrast, extracting peak values of stance-phase joint motions indicates a joint's end range, but gives no indication as to the timing or excursion of this motion. Therefore, it is possible that the differences in peak joint motions previously documented in runners with ITBS are not enough to effect vector coding values. Further, it should be noted that many of the studies that have related kinematic alterations to ITBS have been conducted in runners who were healthy at the time of data collection and has either a retrospective or prospective history of ITBS. Conversely, the current study was conducted on runners with current symptoms of ITBS.

Conclusion

Understanding the role that the hip plays in the development of ITBS is important to appropriately target musculoskeletal or neuromuscular interventions. When measured in a fresh, non-fatigued state, hip abductor strength and onset activation timing as well as kinematic joint coupling at the hip and knee do not appear to be related with ITBS. Runners with ITBS do, however, demonstrate decreased resistance to fatigue in their gluteus medius muscle. This suggests that endurance of the hip abductors may be a factor in the presentation of the syndrome. Future analyses will be directed at examining the effect of fatigue on neuromuscular, strength and kinematic variables in runners with ITBS.

CHAPTER 3

THE EFFECTS OF FATIGUE ON LOWER EXTREMTY KINEMATICS, KINETICS AND JOINT COUPLING IN FEMALE RUNNERS WITH ILIOTIBIAL BAND SYNDROME

Introduction

Running is an aerobic activity that provides participants with cardiovascular, musculoskeletal, and psychological benefits[1] as well as contributing towards weight loss/maintenance. It is also a convenient form of exercise that requires very little equipment, is easily accessible, and inexpensive. Therefore it has become an increasingly popular activity for athletes as well as the general population. Increasing enrollment in races around the country is evidence of the continued rise in running popularity. In 1970, only 127 runners participated in the New York City marathon, while, in 2009, more than 40,000 runners participated. However, along with a rise in popularity comes increased prevalence of injury. Running is a high impact, repetitive sport with annual injury rates reaching up to 52% of participants.[2] Iliotibial band syndrome (ITBS) is the second most common running injury[5] and the leading cause of lateral knee pain in runners[6]. Although the cause is unclear, the incidence of ITBS has increased from 4.3% in 1981 to 8.4% in 2000.[5] Iliotibial band friction syndrome was first thoroughly described by Renne[18] as an irritation of the iliotibial band caused by back and forth rubbing over the lateral femoral epicondyle during repetitive knee flexion and extension as seen in running or marching exercises. The friction caused by the back and forth rubbing is though to result in a localized irritation, leaving runners with symptoms of lateral knee pain. Early literature in support of the friction theory, reported the presence of a bursa over the lateral femoral epicondyle.[18] However, more recently, MRI and cadaveric studies have not supported these findings.[20-22] Fairclough et al.[22] recently provided an alternate etiology of ITBS. The authors suggest that *compression,* rather than friction, is occurring between the iliotibial band and the lateral femoral epicondyle. In a cadaveric study, the authors document the presence of adipose tissue containing Pacinian corpuscles, deep subcutaneous mechanoreceptors that sense global pressure[23], beneath the iliotibial band. The presence of Pacinian corpuscles seem to support the theory that *compression* rather than *friction* is occurring between the lateral femoral epicondyle and the iliotibial band. One kinematic alteration that may result in increased compressive forces is the presence of increased stance phase hip adduction. Excessive angles of hip adduction are thought to increase the length of the ITB[25,32] and may therefore result in distal compression at the lateral femoral epicondyle. Current research has suggested that there are frontal plane stance phase kinematic differences between runners with ITBS and healthy runners[25,32]. This demonstrates the importance of continued exploration into the role of the hip in the development of ITBS.

Single joint kinematic alterations have been extensively documented in the injured population.[14,15,26,27,39] However, the relationship between these alterations and injuries is unclear and thus fails to provide researchers with a complete understanding of running injury pathology. Therefore, biomechanical running studies have also examined *interactions* between joint motions (excursion and timing) and their relationships to injury. Alterations of these interactions, often referred to as joint coupling, and the variability of their patterns have been associated with running injury.[48,49] It has been suggested that decreased variability of motion, particularly within an injured population, indicates that runners have limited patterns of movement, making them susceptible to overuse injuries.[45,48,49] Miller and colleagues[45] examined the variability of joint coupling patterns in runners with ITBS and healthy runners. Following a fatiguing run, runners with ITBS were found to demonstrate less variability of thigh abduction/adduction-foot inversion/eversion and a trend towards less variability in thigh abduction/adduction-tibial internal/external rotation coupling. The findings of decreased variability in coupling patterns involving hip adduction/abduction and tibial internal/external rotation suggest that alterations of these coupled motions may be a factor for injury development in runners with ITBS. Hip adduction and internal rotation both naturally occur as coupled motions during the stance phase of gait. However, altered timing or excursion of either phasic motion may lead to abnormal stretching and loading of the musculoskeletal system. This is supported by several previous studies of runners who are susceptible to ITBS. Miller et al.[27] found that runners with ITBS exhibit increased knee internal rotation velocity while Noehren et al.[25] and Ferber et al.[32] both found increased peak hip adduction and knee internal rotation in runners with ITBS. While previous work has focused upon peak rotations at these joints, it is possible that discoordination in the hip adduction/hip internal rotation or hip adduction/knee internal rotation coupling patterns may result in greater torsional forces at the knee, placing increased strain on the ITB. While Miller and colleagues examined the *variability* of joint coupling, no study has examined the joint coupling patterns of runners with ITBS. Therefore further investigation into this area is needed.

An additional factor that is of great importance during any running research is the effect that fatigue plays on changes in gait mechanics. Runners with ITBS typically complain of symptoms after 2-3 miles of running which suggests that fatigue may influence the onset of symptoms. Fatigue-related changes in kinetics and kinematics have been well-documented in both healthy and injured runners following as little as 15 minutes of running.[27,39-42] In addition, fatigue-related changes, such as delayed muscle activation and altered force attenuation[3,39] have been suggested to increase injury risk. Miller and colleagues [27,45] are the only authors, to-date, who have examined the effect of a run to exertion on runners with ITBS. Their studies, however, are limited in their generalizability as data were collected during treadmill running which is known to result in different kinematic patterns from those seen during overground running[75,76]. Additionally, no study has examined the effect of exertion on joint kinetics or joint coupling patterns in this population of runners. Therefore, the purpose of this study was to determine the effect of a run to exertion on hip joint kinematics, kinetics and joint coupling patterns in runners with ITBS, as compared to healthy controls. It was hypothesized that, as a result of exertion, individuals with ITBS would exhibit significantly greater hip joint kinematic, kinetic, and joint coupling changes as compared to healthy controls.

Methods

Participants

Female runners are twice as likely to suffer from ITBS and three times as likely to sustain a gluteus medius injury in comparison to their male counterparts[5]. In addition, kinematic and kinetic gender differences specific to the hip have been documented during running.[61] Based on this information, this study was limited to female runners. The sample consisted of 20 healthy female runners (28.9 ±6.1 yrs; 1.6± 0.09 m; 56.8± 5.2 kg) and 12 female runners with a current diagnosis of ITBS (32.4± 7.9 yrs; 1.7 ± 0.06 m; 60.6± 5.0 kg). While the groups did not differ with respect to age (P=0.17), runners in the ITBS group tended to be taller (P=0.01) and have greater mass (P=0.06) than the control runners. All participants were (1) female; (2) aged 18-50 yrs old; (3) rearfoot strikers; (4) running ≥ 15 miles/week; and (5) able to run one at least 9-minute mile. Additionally, all runners were free from all neuromuscular and musculoskeletal disorders for 6 months prior to data collection, with the exception of current symptoms of ITBS in the injured runners. All injured runners received a diagnosis of unilateral ITBS by a doctor or physical therapist. This protocol was approved by the Hospital for Special Surgery and Temple University Institutional Review Boards. Prior to data collection, informed consent was obtained from each participant.

Procedures

Instrumentation

A 12-camera motion analysis system (Motion Analysis Corporation, Santa Rosa, CA, USA) was used to calibrate the field and collect kinematic and kinetic data. Passive, retroreflective markers were placed in specified locations on the trunk, pelvis and lower extremities for the tracking of segment coordinate systems during dynamic running trials (Figure 3-1). These markers were used to define and track a model that included a trunk, pelvis, thighs, shanks, and hindfeet. The trunk was defined and tracked by markers placed on the sternal notch, C7, right and left ASIS, and sacrum. The pelvis was defined and tracked by the right and left ASIS and sacrum. The thighs were defined by a functionally-determined hip joint center, medial and lateral femoral condyles, and tracked by an array cluster on the thigh. The shanks were defined by medial and lateral femoral condyles, the tibial tuberosity, medial and lateral malleoli, and tracked by an array cluster on the shank. The hindfeet were defined by the distal toe, proximal and distal calcaneus (aligned based on the bisection of the posterior calcaneus), and tracked by the proximal, distal, medial and lateral calcaneus.

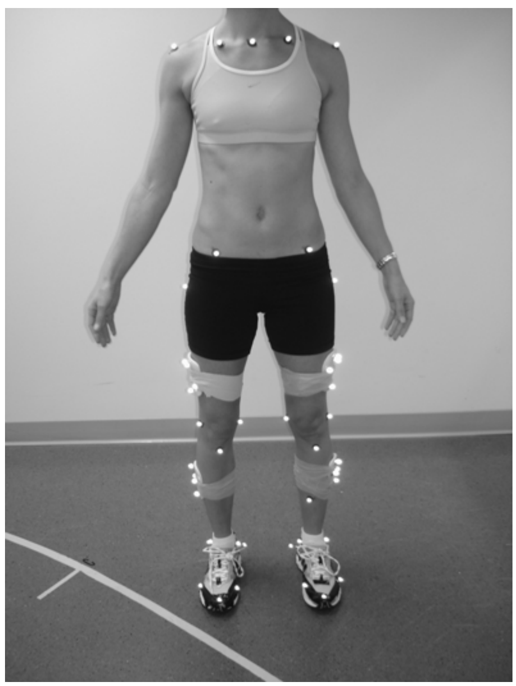

Figure 3-1. Six degree of freedom marker set utilized to define and track segment motion.

All testing was conducted with participants wearing a neutral, laboratory-provided running shoe (New Balance, 1062; Boston, MA, USA) with customized holes cut from the heel cup to allow marker placement on the calcaneus. Video and analog data were collected at 120 Hz and 4800Hz respectively.

Three-dimensional motion analysis

To begin data collection, runners stood at the center of the data collection volume for the capture of a static calibration trial. They were then cued to move their hip through an approximate 20° arc of abduction/adduction and flexion/extension motion for the identification of a functionally-determined hip joint center[70]. Next, markers used solely for anatomical definitions were removed so that only tracking markers remained during running trials. In a fresh state, participants ran along a 30-meter runway through the data capture volume for the collection of five acceptable overground running trials. An acceptable trial was defined as one where the runner struck at least one force plate and maintained a running speed of 3.35 m/sec (±10%). To ensure consistency across trials, velocity was recorded with two photoelectric timers placed 4 meters apart. When the runner broke the first photoelectric beam, a timer began. The timer then turned off when the runner broke the second photoelectric beam providing real-time feedback to the examiners. If necessary, feedback was then provided to the runner for them to adjust their speed to the desired velocity. Participants were then asked to perform a run to exertion. This run was performed on a treadmill with participants cued to run at a pace that approximated their 5-kilometer race pace. Prior to beginning the run to exertion, runners were educated on the use of the Borg CR10 scale for pain and the Borg Rating of Perceived Exertion Scale (RPE) to rate exertion.[77] Every three minutes throughout the course of the run, participants were asked to provide a verbal pain and RPE rating. Runners were considered fatigued when they reached a 17/20 on the Borg RPE scale or rated their pain a 6/10 on the CR10 scale. Five acceptable trials of fatigued-state overground running data were then collected in the same manner as previously described.

Data Processing

Three-dimensional motion analysis

Three-dimensional motion data from the dominant limb of uninjured controls and the injured limb in runners with ITBS were processed and analyzed using customized code written in Visual 3-D (C-Motion, Inc; Rockville, MD, USA) and LabView (National Instruments; Austin, TX, USA). Lower extremity dominance was established as the limb that the runner reported they would use to kick a soccer ball. This self-report method of determining lower extremity dominance was found to have 97.7% agreement with task performance and a 96% test-retest agreement {{145 Coren, Stanley 1978}}. Kinematic and kinetic data were smoothed using low-pass fourth order Butterworth filters at 8Hz and 50Hz respectively. For each variable measured, data from the 5 trials were averaged for statistical analysis.

Kinematic, kinetic and joint coupling variables were chosen based on the thought that aberrations in these movements would lengthen the ITB and cause increased compression at the level of the lateral femoral epicondyle or increased strain within the structure itself. Kinematic and kinetic variables of interest included peak stance phase values of hip adduction and internal rotation angles and hip abductor and external rotator joint moments (internally-referenced). Joint coupling motions of interest included hip abduction-adduction/hip internal-external rotation and hip abduction-adduction/knee internal-external rotation. To account for the varying functional demands over the stance phase of running gait, coupling was examined over four specific phases of stance: the first and second halves of both loading and propulsion. Loading was functionally defined from initial contact to the time of first peak knee flexion. Propulsion was functionally defined from the time of first peak knee flexion to toe off. Vector coding was used to describe the magnitude of joint coupling between the motions of interest. As described by Heiderscheit et al.[49] and Dierks et al.[47], an angle-angle diagram (Figure 3-2) is constructed from the two motions of interest. Using the equation, $\Theta = |\tan^{-1}(y_2\text{-}y_1/x_2\text{-}x_1)|$, angles of the trajectory between two successive data points with respect to the horizontal were calculated for all data points over the period of interest (i.e. first half of loading phase) (Figure 3-3).

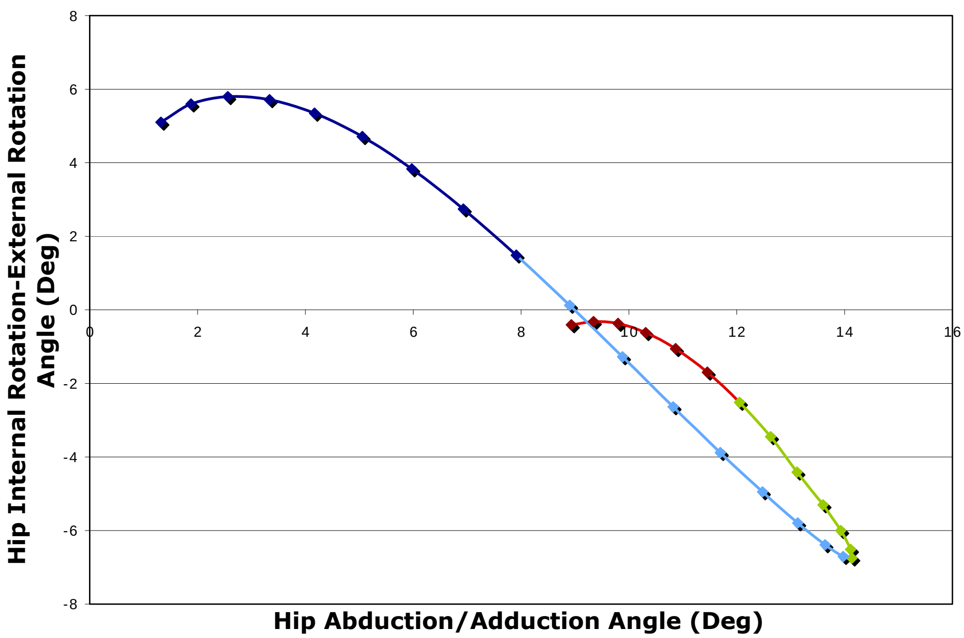

Figure 3-2. Angle-angle plot constructed from hip abd/adduction and internal/external rotation motion from one representative trial. Red and green data represent first and second halves of loading respectively. Light and dark blue data represent first and second halves of push-off respectively.

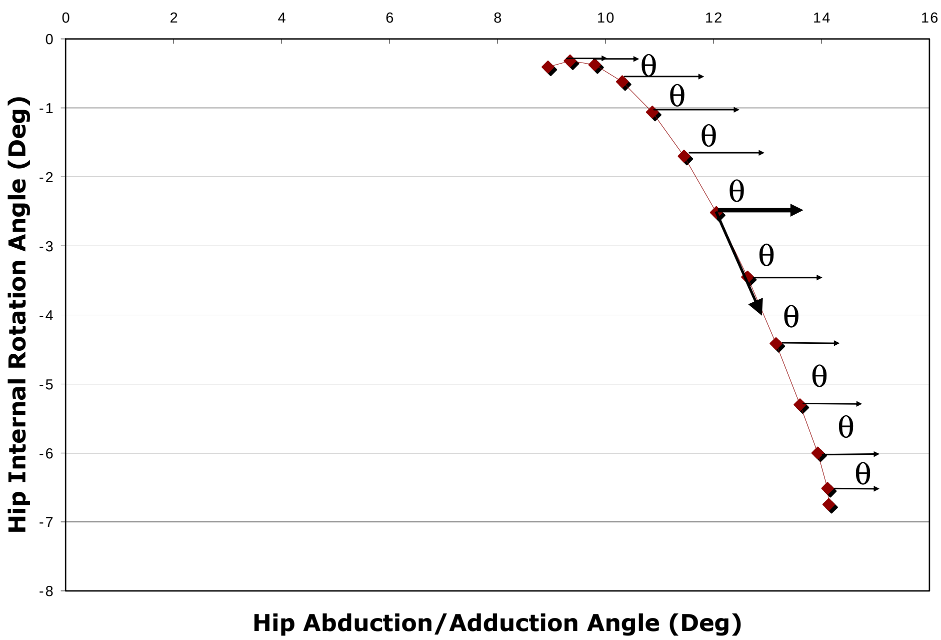

Figure 3-3. Angle-angle plot created during loading phase of running. Vector coding angular values Θ are calculated from trajectories between each successive data point with respect to horizontal. Values are then averaged over the time period of interest.

The mean angular value for the period of interest is then calculated. This provides a continuous excursion ratio coupling angle with a range of 0°-90°. A 45° angle indicates equal amounts of movement between the two segments. An angle greater than 45° indicates greater amounts of distal motion while an angle less than 45° indicates greater amounts of proximal motion.[46,47,49] In the case of hip abduction/adduction and hip internal/external rotation, hip abduction/adduction is defined as the proximal motion.

Statistical Analysis

A 2-way ANOVA was used to examine the effects of exertion on kinematic, kinetic and joint coupling variables in the two experimental groups. When a significant group-by-exertion interaction was detected, post-hoc comparisons were made using independent t-tests between groups. A significance level of $p \leq 0.05$ was established for all analyses.

Results

Treadmill Run to Exertion

There were no significant differences between groups with respect to the duration or the speed of the treadmill runs. The run to exertion lasted an average of 25.6± 8.3 and 24.7 ± 7.0 minutes for ITBS and healthy runners respectively. Runners with ITBS performed their run to exertion at an average pace of 3.1 ± .44 m/sec, while healthy runners ran at a pace of 3.2 ± .29 m/sec. For all healthy runners, the treadmill run was terminated as a result of exertion (reaching a Borg RPE rating of 17/20). For 2 of the 12 ITBS runners, the treadmill run was terminated due to pain (Borg CR10 rating of 6/10). For these runners, their Borg RPE rating was 14-15/20 and 15/20 (indicating "hard") on the Borg RPE scale at the time their run to exertion was terminated. Over the course of the run, pain ratings of the healthy runners increased from an average of 0.3/10 at the start of the run to 1.25/10 at the end, noting symptoms such as "side-stitches", muscular soreness etc, but no symptoms that were suggestive of repetitive use musculoskeletal injuries. Pain ratings for the injured runners ranged from an average 0.25/10 at the start of the run to 2.7/10 at the end, attributed mainly to the ITBS symptoms.

Kinematics and Kinetics

There was a significant group-by-exertion interaction with respect to stance phase peak hip adduction angles (P=0.02) (Figure 3-4). Post-hoc t-tests analyzing simple main effects demonstrated that there were no differences between groups in the fresh state (P=0.27). In the fatigued state, however, there was a significant difference between groups (P=0.03) where, on average, runners with ITBS were in 3º less stance phase peak hip adduction than the healthy runners. This translates to an 18.5% average difference between groups.

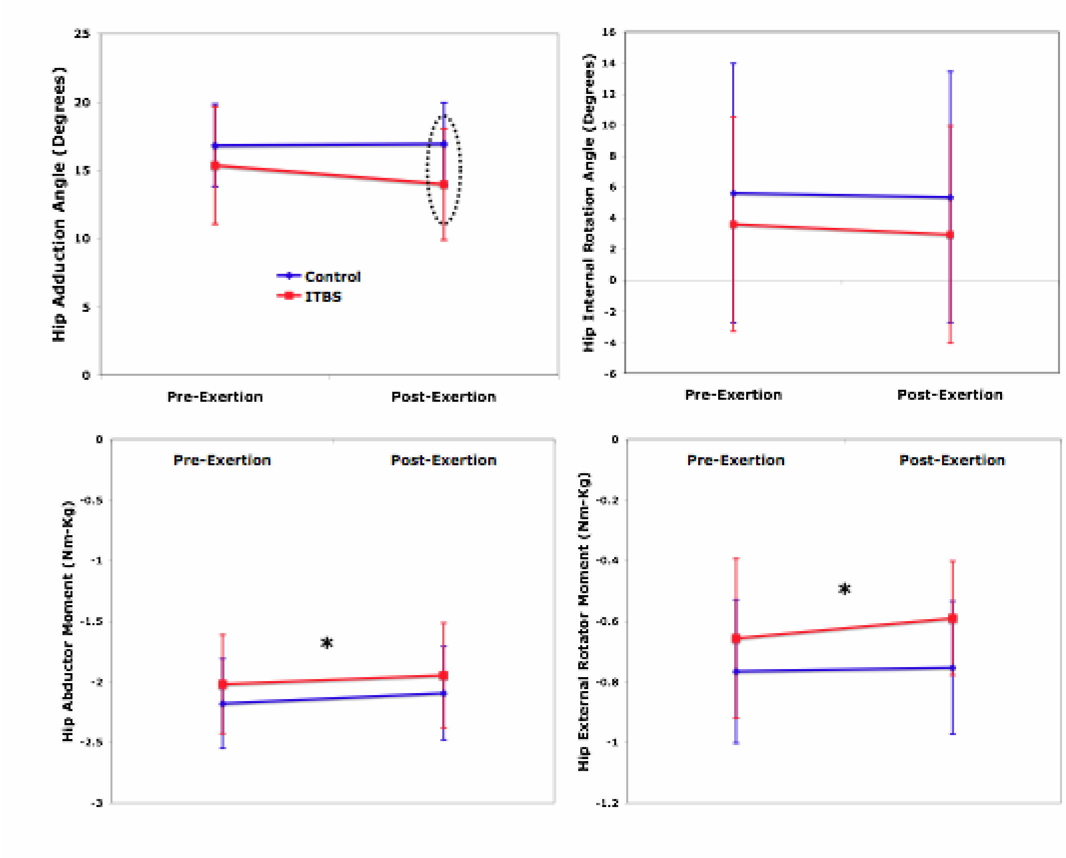

Figure 3-4. Peak stance phase hip angles and moments during overground running, prior-to and following a run to exertion. indicates significant simple main effect, * indicates significant main effect of exertion

There was no significant group-by-exertion interaction (P=0.52) (Figure 3-4), nor any main effects of group (P=0.44) or exertion (P=0.13) on peak hip internal rotation angles.There were no significant group-by-exertion interactions for peak stance phase hip abductor (P=0.82) or external rotator moments (P=0.18) (Figure 3-4). There was, however, a main effect of exertion for both variables (P=0.01 and P=0.05, respectively). Following the run to exertion, both healthy and injured runners demonstrated significantly decreased joint moments. Hip abductor moments decreased by 3.8% while external rotator moments decreased by 4.2% following the run to exertion. There was no main effect of group on hip joint moments.

Kinematic Joint Coupling

There were no significant group-by-exertion interactions for stance phase *hip* abduction/adduction-*hip* internal/external rotation joint coupling during the first and second halves of loading (P=0.30 and P= 0.70, respectively) or the first and second halves of propulsion (P= 0.73 and P=0.71, respectively) (Figure 3-5). In addition, there were no significant main effects of group or exertion. When examining stance phase *hip* abduction/adduction and *knee* internal/external rotation joint coupling, there were no significant group-by-exertion interactions across both the first and second halves of loading (P=0.90 and P=0.20, respectively) or the first and second halves of propulsion (P=0.22 and P=0.78, respectively) (Figure 3-6). There was no main effect of group across the phases, yet there was a main effect of exertion during the first half of loading (P=0.01). Following the run to exertion, joint coupling values during the first half of loading increased from an average of 56.0 to 60.1 degrees, an increase of 7.3%.

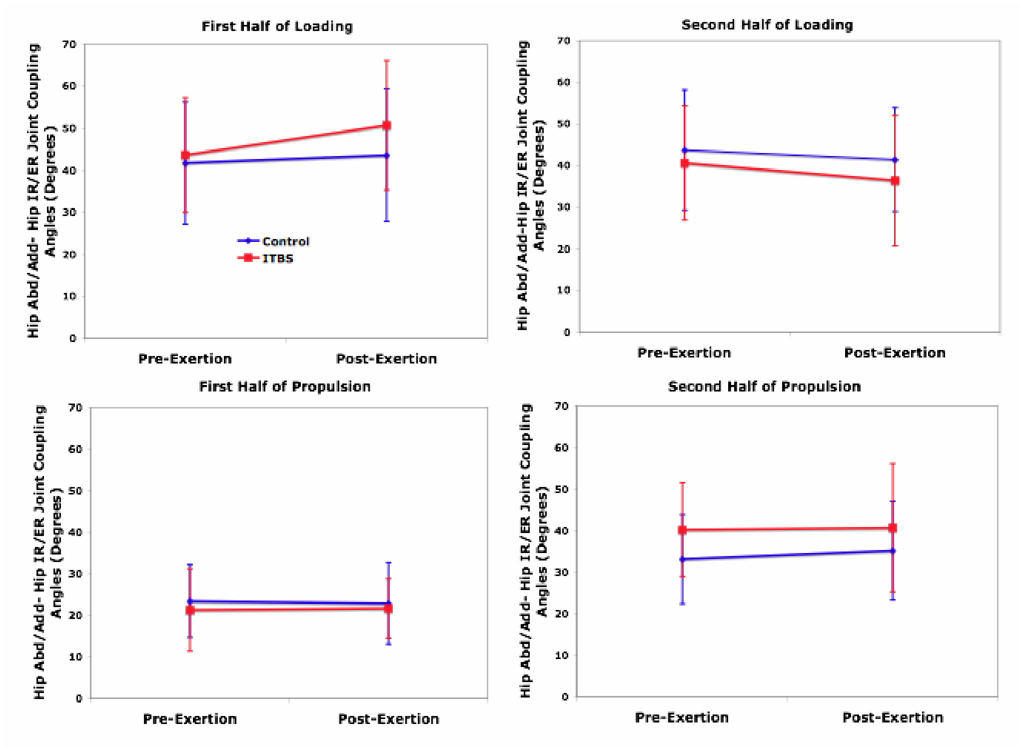

Figure 3-5. Hip abduction/adduction- Hip internal/external rotation joint coupling angles during the stance phase of overground running.

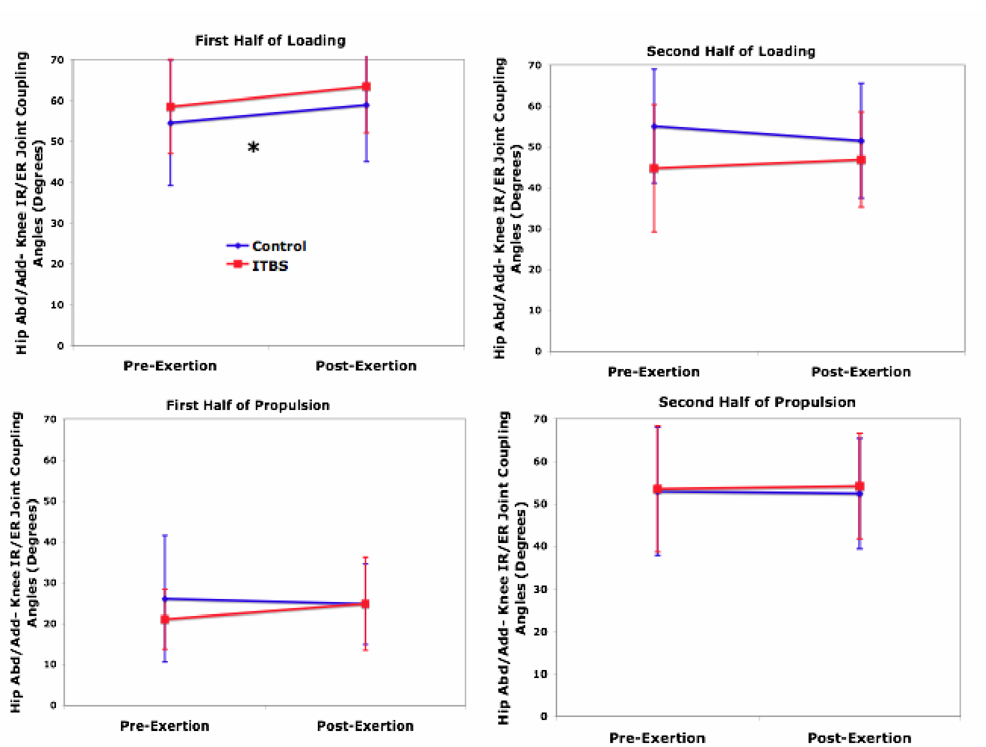

Figure 3-6. Hip abduction/adduction- Knee internal/external rotation joint coupling angles during the stance phase of overground running. * indicates significant main effect of exertion

Discussion

The aim of this study was to determine the effect of performing a run to exertion on hip joint kinematics, kinetics and joint coupling patterns in runners with ITBS as compared to healthy controls. It was hypothesized that, as a result of exertion, runners with ITBS would exhibit significantly greater hip joint kinematic, kinetic and joint coupling changes than their healthy counterparts.

In support of the hypothesis, there was a significant group-by-exertion interaction noted with respect to stance phase peak hip adduction angles. Following a run to exertion, runners with ITBS demonstrated a 9.1% decrease in stance phase peak hip adduction angles as compared with their healthy counterparts who demonstrated no significant change in their frontal plane hip mechanics. This suggests that over the course of a run to exertion, runners with ITBS are altering their gait such that they *decrease* their peak stance phase hip adduction angles. Further, this demonstrates an average difference of 18.5% between groups, with injured runners in significantly less hip adduction than uninjured runners. Previous work has supported these findings of decreased hip adduction in runners with ITBS.[34] Yet other findings of increased hip adduction angles[25,26] in runners who have a history of ITBS or in those who go on to have ITBS contradict our results. However, our runners and those examined in the study by Grau and colleagues[34] examined currently injured runners. It is therefore possible that the adjustments we noted are present as an attempt to prevent or relieve symptoms associated with ITBS. Decreased angles of hip adduction may result in decreased length of the ITB, decreased strain within its fibers, and decreased contact distally at the lateral femoral epicondyle. Runners who are currently injured may have subconsciously made this stance phase accommodation to relieve painful symptoms over the course of their run.

This study did not identify a significant group-by-exertion interaction when examining peak stance phase hip internal rotation angles, nor peak stance phase hip abductor and external rotator moments suggesting that, with regards to these variables, exertion does not effect these runners differently. In addition, with respect to hip internal rotation angles, there were no significant main effects of group or time. It had been theorized that potential dysfunction of the gluteus medius muscle, whose posterior fibers function to externally rotate the hip, may result in excessive hip internal rotation. Yet the findings of this study suggest that this may not be the case in runners with current symptoms of ITBS. In fact, while it was not significant, the runners with ITBS demonstrated slightly lower peak hip internal rotation angles as compared to healthy runners. Similarly, there was no main effect of group on peak hip abductor or external rotator moments. Noehren et al.[25] reported similar findings when examining hip abductor moments in healthy female runners and female runners with ITBS. There was, however, a significant effect of exertion on hip abductor and external rotator moments whereby both groups of runners demonstrated a decrease in their joint moments following the run to exertion. This suggests that the demands on their hip abductor and external rotator musculature may have been reduced over the course of the run. The source of this reduction is unclear. There was no effect of exertion on hip adduction or internal rotation angles in the pooled group of ITBS and healthy runners. Thus, it could be hypothesized that another kinematic alteration, such as lateral trunk lean, which was not examined in this study, may contribute to these changes.

Alterations in joint coupling patterns and the variability of joint coupling patterns have been associated with running injury[48,49] and specifically with ITBS[45]. However, this study sought to specifically examine the effects of exertion on joint coupling values across the loading and propulsive phases of stance. The results show that runners with ITBS are not affected differently by exertion than healthy runners with respect to frontal and transverse plane hip joint coupling or with frontal plane hip and transverse plane knee joint coupling. These findings are interesting in light of the existing studies of runners with susceptibility to ITBS, which has documented single joint kinematic alterations involving these motions[25,27,32,34]. However, these previous studies have examined runners in their fresh, non-exerted state. Further, some studied runners who were not symptomatic at the time of data collection, but had a retrospective or prospective history of ITBS. Using the continuous relative phase method, Miller and colleagues examined the *variability* of kinematic coupling during an exhaustive run in individuals with current ITBS symptoms[45]. The authors reported similar findings of no interactions between group and time for various coupling measures between the hip and knee as well as between the knee and foot.

This study is the first to report overground running kinematic and kinetic alterations at the hip as a result of exertion in runners with ITBS. Presently, there is not a consensus in the literature with respect to the frontal plane hip biomechanical differences between healthy runners and those with ITBS. However, this study's findings of decreasing stance phase peak hip adduction angles in an exerted state are supported by the findings by Grau and colleagues[34] who documented decreased hip adduction angles during overground running in a population of runners with ITBS who were presently symptomatic. It has previously been suggested that hip abductor weakness may result in increased stance phase hip adduction angles in runners with ITBS.[29,32] However, based on the current findings of *decreased* stance phase hip adduction in runners with ITBS, it seems counterintuitive that hip abductor weakness has played any role in the exertion-related kinematic alterations. Further, the posterior fibers of the gluteus medius muscle, a primary hip abductor, act to externally rotate the hip. Weakness at this muscle would not only presumably present in the form of increased hip adduction, but also in increased hip internal rotation angles during stance, neither of which were seen in this study. These findings suggest that hip abductor weakness is not the sole cause of iliotibial band syndrome. Conversely, these finding may have implications that support changes in clinical treatment of runners with ITBS. While many physical therapists and clinicians seek to strengthen the hip abductor musculature and provide immediate neuromuscular re-education or gait re-training to decrease hip adduction motion, the results of this study suggest that this education may not be so clearly indicated as symptomatic runners are already making these modifications. It should not go without mentioning, however, that based on the cross-sectional nature of this study, which examined currently injured runners, the presumption of cause and effect based on the kinematic findings described here are limited.

Conclusion

Exertion was found to decrease the stance phase peak hip adduction angles in female runners with a current diagnosis ITBS. Exertion was not shown to affect females with ITBS differently than healthy controls with respect to stance phase peak hip internal rotation, peak hip abductor moments or peak hip external rotator moments. However, as a group, runners with ITBS and healthy runners demonstrated decreased hip abductor and external rotator moments following the run to exertion. Exertion did not affect stance phase hip frontal-transverse plane or hip frontal-knee transverse plane joint coupling differently between healthy female runners and those currently symptomatic with ITBS. When examined as a group, runners with ITBS and healthy runners all demonstrated decreased hip frontal-knee transverse plane coupling during the first half of loading with a bias towards greater transverse plane knee excursion.

CHAPTER 4

HIP MUSCLE ELECTROMYOGRAPHIC AND STRENGTH CHANGES FOLLOWING A RUN TO EXERTION IN FEMALE RUNNERS WITH ILIOTIBIAL BAND SYNDROME

Introduction

Ilitobial band syndrome (ITBS) is the second most common running injury and the leading cause of lateral knee pain in runners.[5] The iliotibial band (ITB) is a fibrous sheath extending down the lateral side of the femur and continuing until its insertion into Gerdy's Tubercle.[14] The pathogenesis of ITBS involves an irritation and resultant inflammation deep to the posterior fibers of the iliotibial band at the location of the lateral femoral epicondyle.[20,21] It is thought that this irritation stems from either friction or compression occurring between the two structures during repeated knee flexion/extension, such as seen during running or cycling. Most often, symptoms of ITBS present themselves at the lateral knee; however, recent findings in the literature have found also implicated strength impairments at the hip.[28,29] During loading response, a large contribution from the hip abductors, specifically the gluteus medius muscle, is required to absorb shock and overcome large external adduction moments.[7] Additionally, the tensor fascia latae and the upper fibers of the gluteus maximus assist with hip abduction.[16] Inability of the hip muscles to counter this external adduction moment, whether due to weakness or neuromuscular dysfunction, is suggested to be a factor in the development of ITBS.[22,29,30] In fact, both prospective and retrospective studies have documented increased stance phase hip adduction angles in runners with ITBS[25,32] with hip abductor weakness as a proposed contributor.[25,29,32]

A characteristic of ITBS that differentiates its symptoms from those of other overuse running injuries is the relative absence of symptoms during the early stages of running. Runners tend to complain of ITBS symptoms after 2-3 miles of running. This suggests that fatigue-related biomechanical changes may be associated with ITBS pathogenesis. Fatigue-related kinematic, kinetic and electromyographic changes have been documented in both healthy and injured runners.[27,39,41,42,79] Yet little is known as to the effects of fatigue on hip muscle function in runners with ITBS. One characteristic of fatiguing muscle is a shift of the EMG power spectrum from higher to lower frequencies during a submaximal isometric contraction.[52,68] This frequency shift can be monitored to provide an index of muscle fatigue, which we will term "fatigue resistance". Fatigue resistance is a particularly useful measure for runners with ITBS, especially at the gluteus medius muscle due to the high demands placed on this muscle during loading response. To-date, no study has thoroughly examined hip abductor muscle function in runners with ITBS. Therefore, the purpose of this study was to determine the effect of performing a run to exertion on gluteus medius function (fatigue resistance and isometric strength) as well as gluteus medius and tensor fascia latae terminal swing activation timing in runners with ITBS as compared to healthy controls. It was hypothesized that as a result of exertion, runners with ITBS would have a greater change in gluteus medius fatigue resistance and gluteus medius strength during isometric testing. It was also hypothesized that as a result of exertion, runners with ITBS would have a greater change in gluteus medius and tensor fascia latae activation timing during overground running.

Methods

Participants

Twenty healthy female runners (28.9 ±6.1 yrs; 1.6± 0.09 m; 56.8± 5.2 kg) and 12 female runners with a current diagnosis of ITBS (32.4± 7.9 yrs; 1.7 ± 0.06 m; 60.6± 5.0 kg) were included in this study. While the groups did not differ with respect to age (P=0.17), runners in the ITBS group tended to be taller (P=0.01) and have greater mass (P=0.06) than the control runners. All participants were (1) female; (2) aged 18-50 yrs old; (3) rearfoot strikers; (4) running ≥ 15 miles/week; and (5) able to run one at least 9-minute mile. Additionally, all runners were free from all neuromuscular and musculoskeletal disorders for 6 months prior to data collection, with the exception of current symptoms of ITBS in the injured runners. All injured runners received a diagnosis of unilateral ITBS by a doctor or physical therapist. This protocol was approved by the Hospital for Special Surgery and Temple University Institutional Review Boards. Prior to data collection, informed consent was obtained from each participant.

Procedures

EMG Instrumentation

For the capture of fatigue resistance and muscular onset timing, EMG data were collected using a 16 channel MA-300 EMG System (Motion Lab Systems, Baton Rouge,LA). Disposable surface gel electrodes with a 20mm interelectrode distance were placed on the gluteus medius and tensor fascia latae muscles of the injured limb in the

ITBS group or the dominant limb of the control group. Lower extremity dominance was established as the limb that the runner reported they would use to kick a soccer ball. This self-report method of determining lower extremity dominance was found to have 97.7% agreement with task performance and a 96% test-retest agreement {{145 Coren, Stanley 1978}}. Electrodes were applied in parallel with the direction of the muscle fibers and in locations as described by Perotto et al.[69](Table 4-1). Once in place, the electrodes were connected via snap leads to the MA-411 EMG pre-amplifier (Motion Lab Systems, Baton Rouge,LA). Correct electrode placement was verified through resisted muscular testing as described by Perotto et al.[69](Table 4-1). EMG data were sampled at 4800 Hz, bandpass filtered at 10-2000Hz and pre-amplified with a x20 gain. For the purposes of determining muscular onset timing, a 5- second maximal voluntary isometric contraction (MVIC) and a 2-second static baseline trial were acquired prior to further testing.

Table 4-1. EMG Electrode location and placement verification activity[69]

Muscle	Electrode location	Placement verification activity
Gluteus Medius	One inch distal to the midpoint of the iliac crest	Thigh abduction with the participant sidelying
Tensor Fascia Latae	Two fingerbreadths anterior to the greater trochanter	Thigh abduction with hip flexion

Strength Testing

Isometric gluteus medius strength testing was performed using the Biodex System 4 (Biodex Medical Systems, Shirley, NY). As described in Kendall and McCreary[16], participants were positioned in sidelying with the bottom leg flexed at the hip and knee. So as to best isolate the gluteus medius from the rest of the hip abductors (tensor fascia latae, gluteus minimus), the participant's pelvis was rotated slightly forward with the test limb held in a position of approximately 15° hip abduction with slight extension. The dynamometer attachment was positioned 3cm proximal to the lateral femoral epicondyle and participants were stabilized using a strap positioned at the level of their pelvis. Three 5-second MVIC contractions were performed with 30-second rests between trials.

Fatigue Resistance

Following the collection of MVIC strength data, measures of gluteus medius fatigue resistance were collected on the dominant limb of control runners and on the injured limb of runners with ITBS. Between strength and fatigue resistance testing conditions, participants remained in the Biodex machine so their inter-test position remained the same. Participants were asked to perform a 60-second isometric hip abduction contraction at 50% of their previously determined MVIC strength measure while EMG data of the gluteus medius muscle were collected. During data collection, visual feedback displayed on the Biodex computer monitor in the form of a line graph was given to assist participants in the maintenance of a 50% MVIC.

Running Trials

Following the capture of strength and gluteus medius fatigue resistance data, runners were given a minimum 5-minute rest while they were prepared for the collection of EMG onset data during overground running. During that time, passive retroreflective markers were placed on specified locations on the trunk, pelvis and lower extremities (Figure 4-1).

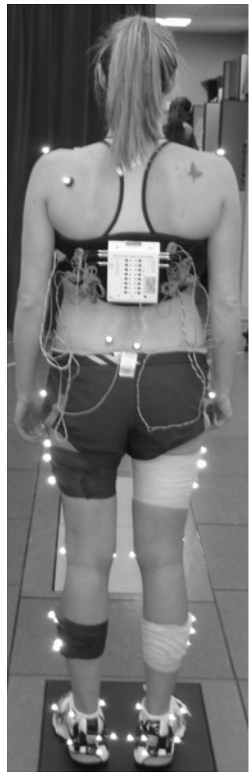

Figure 4-1. Six-degree of freedom marker set with electrodes and EMG pack in place.

A 12-camera Motion Analysis Corporation system (Motion Analysis Corporation, Santa Rosa, CA) was utilized for calibration of the field and capturing three-dimensional digitized data. During overground running data collection, participants wore a neutral, laboratory-provided running shoe (New Balance 1061, New Balance; Boston, MA). Video and analog data were collected at 120 Hz and 4800Hz respectively. To begin overground running data collection, runners stood at the center of the data collection volume for the capture of a static calibration trial. They were then asked to move their hip through an approximate 20° arc of abduction/adduction and flexion/extension motion for the identification of a functionally-determined hip joint center.[70] Next, markers used solely for anatomical definitions were removed so that only tracking markers remained during running trials. In a fresh state, participants ran along a 30 m runway through the data capture volume for the collection of five acceptable overground running trials. An acceptable trial was defined as one where EMG signal quality was good (i.e. no noise or motion artifact), kinematic data were available for the identification of gait events, and the runner maintained at a speed of 3.35 m/sec (±10%). To ensure consistency across trials, velocity was recorded with two photoelectric timers placed 4 meters apart. When the runner broke the first photoelectric beam, a timer began. The timer then turned off when the runner broke the second photoelectric beam providing real-time feedback to the examiners. If necessary, verbal feedback was then provided to the runner for them to adjust their speed to the desired velocity. Participants were then asked to perform a run to exertion. This run was performed on a treadmill with participants cued to run at a pace that approximated their 5-kilometer race pace. Prior to beginning the run to exertion, runners were educated on the use of the Borg CR10 scale for pain and the Borg Rating of Perceived Exertion Scale (RPE) to rate exertion.[77] Every three minutes throughout the course of the run, participants were asked to provide a verbal pain and RPE rating. Runners were considered fatigued when they reached a 17/20 on the Borg RPE scale or rated their pain a 6/10 on the CR10 scale. Five acceptable trials of fatigued-state overground running data were then collected in the same manner as previously described. Once fatigued-state overground running data collection was complete, retroreflective markers were removed from the participants and isometric strength and fatigue resistance testing were recollected.

Data Analysis

Strength

Using Biodex System 4 software, peak hip abductor torque values were averaged across the three isometric strength-testing trials and the mean utilized for data analysis. As previously mentioned, data from the injured limb of participants with ITBS and from the dominant limb of control participants were utilized for data analysis.

Fatigue Resistance

Several of the participants were not able to sustain an isometric contraction at 50% of their MVIC for 60 seconds. Once they relaxed their hip abductor muscles, the frequency of the muscle contraction would vary and affect the EMG signal's frequency content. Therefore, only the first 30 seconds of the fatigue resistance data collection were analyzed.

Using a customized code written in MatLab (The MathWorks, Natick, MA), raw EMG data were initially processed in 213 ms time segments (bins) and transformed into the frequency domain using the fast Fourier transform technique. The median power frequencies were then obtained for 1.91second bins of data and plotted versus time. The linear slope of this plot was obtained and used to express the gluteus medius "rate of fatigue" (Figure 4-2).

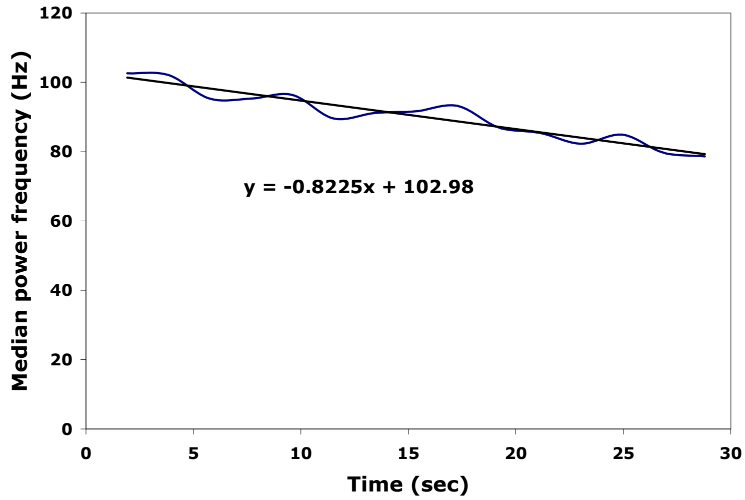

Figure 4-2. Representative plot of median power frequency vs. time. Linear slope of this curve (-0.8225) indicates gluteus medius "rate of fatigue".

This methodology has been thoroughly described by Kondraske et al.[71] and Naeije et al.[52]. Its accuracy in detecting the myoelectric spectral shift has been verified by DeAngelis et al.[72]

Muscle Activation Timing

MVIC, resting and overground running EMG data were processed and analyzed using a customized code written in Visual 3-D (C-Motion, Inc; Rockville, MD). To eliminate any baseline voltage offset, the mean of the entire raw EMG signal was subtracted on a trial-by-trial basis. EMG data were then full-wave rectified and a linear envelope was created using a low pass second-order Butterworth filter with phase correction and a frequency cutoff of 20Hz. To determine a threshold for the onset of muscle activity, the peak activity from all trials (MVIC and dynamic running trials) and the mean EMG value of the resting trial were calculated. Ten percent of the difference between the maximum value and the mean resting value was added to the mean resting value and considered the threshold value. During a running trial, when EMG activity ascended above this threshold value, the muscle was considered "on". Gluteus medius and tensor fascia latae onset during terminal swing, expressed as a percent of the gait cycle, was utilized as the outcome of interest.

Statistical Analysis

A 2-way ANOVA was used to examine the effects of exertion on strength and electromyographic variables in the two experimental groups. When a significant group-by-exertion interaction was detected, post-hoc comparisons were made using independent t-tests between groups. A significance level of $p \leq 0.05$ was established for all analyses and a trend was operationally-defined at $0.05 < p \leq 0.10$.

Results

Treadmill Run to Exertion

There were no significant differences between groups with respect to the duration or the speed of the treadmill runs. The run to exertion lasted an average of 25.6± 8.3 and 24.7 ± 7.0 minutes for ITBS and healthy runners respectively. Runners with ITBS performed their run to exertion at an average pace of 3.1 ± .44 m/sec, while healthy runners ran at a pace of 3.2 ± .29 m/sec. For all healthy runners, the treadmill run was terminated as a result of exertion (reaching a Borg RPE rating of 17/20). For 2 of the 12 ITBS runners, the treadmill run was terminated due to pain (Borg CR10 rating of 6/10).

For these runners, their Borg RPE rating was 14-15/20 and 15/20 (indicating “hard”) on the Borg RPE scale at the time their run to exertion was terminated. Over the course of the run, pain ratings of the healthy runners increased from an average of 0.3/10 at the start of the run to 1.25/10 at the end, noting symptoms such as “side-stitches”, muscular soreness etc, but no symptoms that were suggestive of repetitive use musculoskeletal injuries. Pain ratings for the injured runners ranged from an average 0.25/10 at the start of the run to 2.7/10 at the end, attributed mainly to the ITBS symptoms.

Gluteus Medius Strength

There was no significant group-by-exertion interaction (P=0.78) for hip abductor strength (Figure 4-3). There was no main effect of group (P=0.53), however, there was a significant main effect of exertion (P=0.01). Following the run to exertion, both healthy and injured runners demonstrated a 7.3% decrease in hip abductor strength.

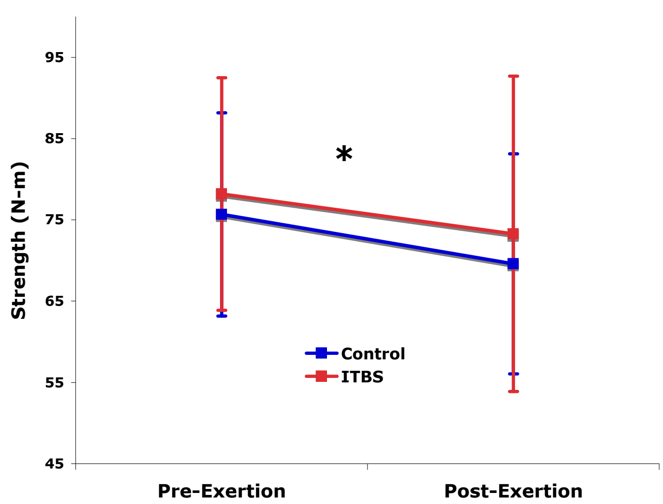

Figure 4-3. Hip abductor strength, prior-to and following a run to exertion. * indicates significant main effect of exertion

Gluteus Medius Fatigue Resistance

Due to a methodological error, fatigue resistance data from 4 uninjured control runners were collected at sampling rates of ≤ 1200Hz. Due to possible undersampling of data and to avoid any aliasing, these data points were removed from analysis. In addition, post-exertion data from one control runner was not available for analysis as it was discovered after data collection that the ground electrode had become disconnected. Therefore, the fatigue resistance analysis was conducted on a sample of 15 healthy runners and 12 runners with ITBS. There was a significant group-by-exertion interaction (P=0.04) with respect to the “rate of fatigue” (linear slope of the median power frequency vs. time plot). Post-hoc t-tests analyzing simple main effects demonstrated that there was a significant difference between groups in the fresh state (P=0.01) but there were no differences when in the exerted state (P=0.72) (Figure 4-4). The presence of a pre-exertion difference with no post-exertion difference was surprising to the investigators. Therefore, further investigation into exertion-related changes in the magnitude of frequency values was performed. Paired t-tests were utilized to examine the initial median frequency values for injured and healthy runners prior-to and following the run to exertion. In healthy runners, initial median frequency values did not demonstrate a significant change from pre- to post-exertion state (P=0.99) while in the injured runners, there was a significant decrease in initial median frequency values as a result of exertion (P=0.01) (Figure 4-5).

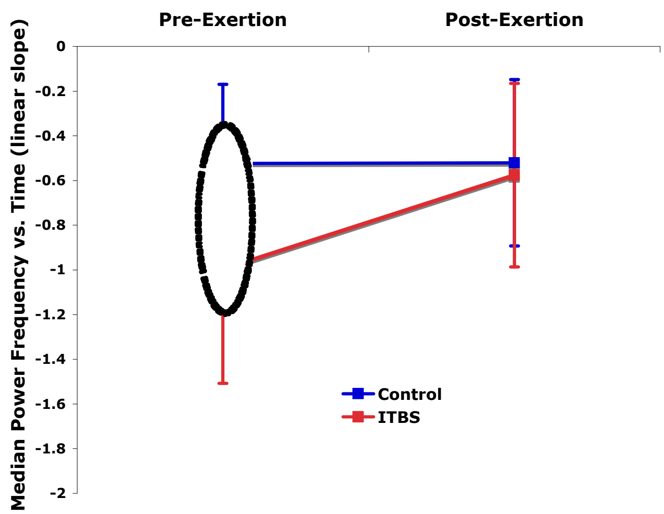

Figure 4-4. Gluteus medius fatigue resistance prior-to and following a run to exertion. indicates significant simple main effect

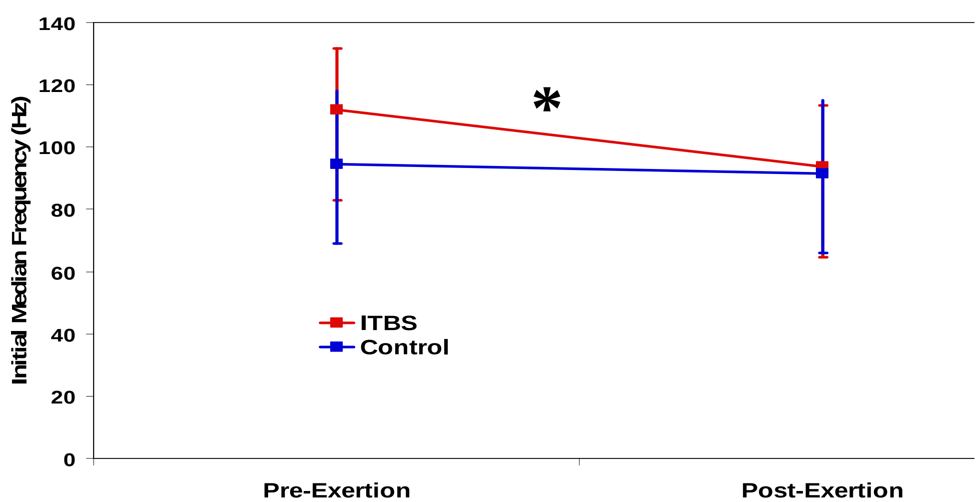

Figure 4-5. Initial median frequency prior-to and following a run to exertion. * indicates significant difference between pre- and post-exertion values for ITBS runners

EMG Onset

Due to the high impact nature of running, prior to data analysis raw EMG data from each runner were examined for the presence of motion artifact that could not be corrected-for using filtering techniques. Based upon this visual examination, gluteus medius data from 2 and tensor fascia latae data from 4 uninjured runners were subsequently excluded from statistical analyses. Means and standard deviations for pre- and post-exertion terminal swing onset activation of the gluteus medius and tensor fascia latae muscles are provided in Table 4-2.

Table 4-2. Gluteus medius (GM) and tensor fascia latae (TFL) terminal swing activation timing as a percent of the gait cycle, pre- and post-exertion.

	GM-pre % gait cycle (SD)	GM-post % gait cycle (SD)	TFL-pre % gait cycle (SD)	TFL-post % gait cycle (SD)
Control	92.2 (5.0)	91.8 (5.0)	93.5 (6.3)	95.6 (2.4)
ITBS	90.1 (7.7)	93.8 (6.2)	95.3 (6.0)	95.8 (4.5)

With a sample size of 18 uninjured control runners and 12 runners with ITBS, our data did not demonstrate a significant group-by-exertion interaction (P=0.19), nor any main effects of group (P=0.97) or exertion (P=0.28) with respect to gluteus medius terminal swing activation timing (Figure 4-6). With 16 uninjured controls and 12 runners with ITBS, our data did not reveal a significant group-by-exertion interaction (P=0.52), nor any main effects of group (P=0.36) or exertion (P=0.34) with respect to tensor fascia latae terminal swing activation timing (Figure 4-7).

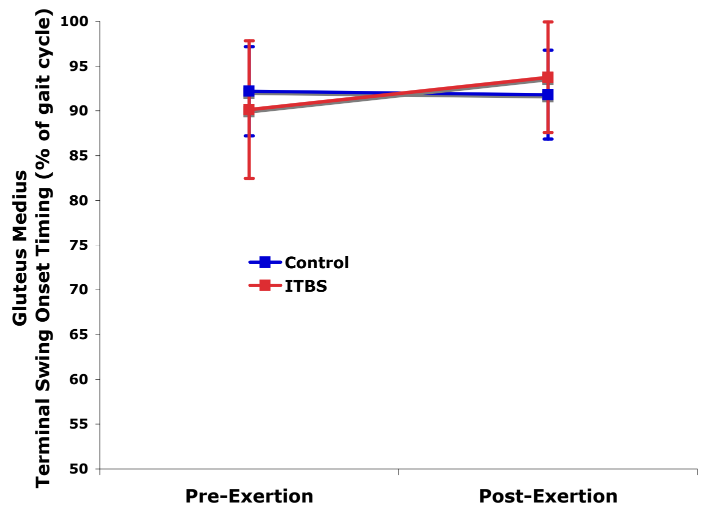

Figure 4-6. Gluteus medius onset activation timing prior-to and following a run to exertion.

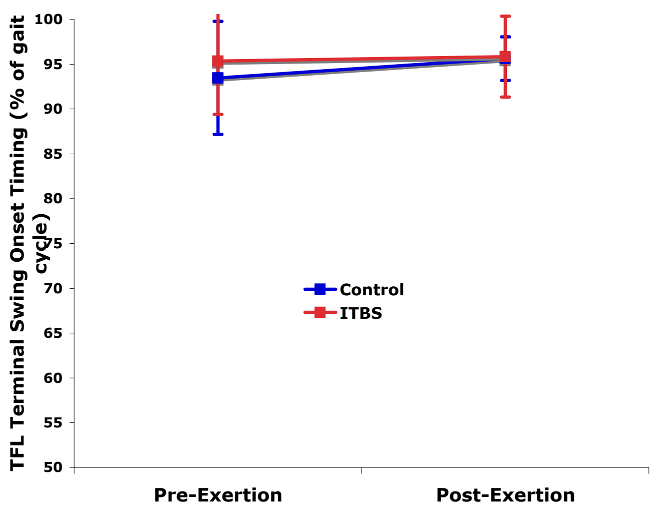

Figure 4-7. Tensor fascia latae onset activation timing prior-to and following a run to exertion.

Discussion

The aim of this study was to determine the effects of performing a run to exertion on hip muscle electromyography and hip abductor strength. It was hypothesized that, as a result of exertion, runners with ITBS would exhibit significantly different electromyographic and strength changes than uninjured runners. In support of our hypothesis, we found that runners with ITBS exhibited significantly different changes in their gluteus medius rate of fatigue during isometric testing. The direction of this change was, however, of interest. While in healthy runners the gluteus medius rate of fatigue remained largely unchanged from a pre- to post-exertion state, the runners with ITBS demonstrated a decrease in the slope of their median power frequency vs. time plot following the run to exertion. A decrease in the slope of the median frequency vs. time curve suggests that the gluteus medius muscle of runners with ITBS is more resistant to fatigue when in an exerted state. This finding was surprising to the investigators and prompted further investigation into the magnitude of the EMG signal's frequency component. It was found that in a post-exertion state, runners with ITBS demonstrated a significantly lower initial median frequency value than when in a non-fatigued state. This was in contrast to the control runners who demonstrated no change in their initial median frequency value from pre- to post-exertion. The decreased magnitude of the initial median frequency in injured runners suggests that a fatigue-related frequency shift had already occurred and therefore was not detected when examined during isometric testing. Therefore, the finding of decreased gluteus medius initial median frequency magnitude following a run to exertion in the injured runners supports the notion that the gluteus medius muscle of runners with ITBS is less resistant to fatigue than that of their healthy counterparts.

Contrary to our hypothesis, this study did not identify a significant group-by-exertion interaction when examining gluteus medius strength and gluteus medius and tensor fascia latae terminal swing activation timing. In addition, there were no significant main effects of group or of time when examining terminal swing onset activation timing. When collapsed across groups, the gluteus medius muscle became active on average at 91.4 and 92.6% of the gait cycle when in pre- and post-exertion states. The tensor fascia latae became active on average at 94.1 and 95.7% of the gait cycle when in pre- and post-exertion states. These ranges are consistent with findings from Mann and colleagues[57] who documented gluteus medius and tensor fascia latae activation at 90 and 95% of the gait cycle respectively. Our study is the first of its kind to examine the effects of exertion on hip muscle onset timing. The gluteus medius and tensor fascia latae muscle activate during terminal swing to provide hip joint abductor stability prior-to and during loading response.[57] Based on our study findings, it does not appear that the gluteus medius or tensor fascia latae muscles are early or delayed in their onset. Early activation would have indicated that the muscles were being overworked or utilized in a compensatory manner. A delay in their activation may have indicated a dysfunction that would alter kinematics and potentially act as a source of symptoms. The results from our study, however, do not support either of these scenarios and instead suggests that these muscles are becoming active at the appropriate time. Previous studies have documented alterations in stance phase hip kinematics present in runners with ITBS. The results of our study do not suggest that activation timing of the gluteus medius and tensor fascia latae muscles are to blame. It is possible that it is the total activation timing as a percent of the gait cycle or the timing of stance phase cessation (rather than swing phase activation) that differentiates injured from uninjured runners. Future studies should hypothesize on and examine these variables.

Following the run to exertion, both control runners and those with ITBS demonstrated a statistically significant decrease in hip abductor strength. The direction of this change is not surprising as it suggests that the run to exertion fatigued the gluteus medius muscle and decreased its ability to generate torque. This change, however, was consistent across groups and did not differentiate injured from uninjured runners.

Fairclough et al.[22] have suggested that there is a relationship between impaired *function* of the hip musculature and ITBS whereby a primary dysfunction of the lateral hip muscles results in compressive forces on the tissues beneath the ITB and hence, secondary pain over the lateral femoral epicondyle. Our data suggest that the lateral knee pain experienced in runners with ITBS is more likely associated with a decreased ability of the gluteus medius muscle to resist fatigue, than with a decreased ability for this muscle to generate a maximal torque (as seen during isometric testing) or by alterations in the terminal swing onset timing of the gluteus medius or tensor fascia latae muscles. As runners with ITBS tend to experience symptoms of pain when in an exerted state, future studies should explore other factors that may be affected by decreased muscular endurance.

CHAPTER 5

DISCUSSION

Review of Specific Aims

Running is a popular form of exercise due to its cardiovascular and psychological benefits as well as its convenience and low cost. However, running is a high impact and repetitive sport with injuries affecting more than 50% of its participants on an annual basis.[2] Iliotibial band syndrome (ITBS) is the second most common running injury.[5] Many factors are thought to contribute to its development including faulty mechanics, strength deficits, fatigue and neuromuscular dysfunction. When running, forces twice the runner's weight are exerted on their body with each foot strike.[4] The result of this force is a greater demand on lower extremity muscles for proper shock absorption as evidenced by increased EMG amplitudes seen during running.[9-13] This demand is particularly important at the abductor muscles of the hip that are required to overcome large external adduction moments during the loading phase of running gait. Failure to stabilize at the hip, whether based on decreased strength, muscle fatigue, or altered neuromuscular recruitment, may have consequential effects up and down the kinetic chain. These effects can include alterations in joint kinematics, kinetics, or joint coupling including increased hip adduction or knee internal rotation motion, such as seen in runners with ITBS.[25,49] Kinematic alterations such as these may lengthen the iliotibial band, put undue strain on its fibers, and result in increased contact distally at the lateral femoral epicondyle.

The objective of this study was to examine differences in hip muscle function and kinematic joint coupling in female runners with ITBS as compared with uninjured control runners. In addition, this study sought to examine the effects of a run to exertion on hip muscle strength, electromyography and hip joint kinematics, kinetics and coupling in runners with ITBS and uninjured control runners.

The specific aims and hypotheses for this study were:

Aim 1: To determine whether differences exist between runners with ITBS and healthy controls with respect to gluteus medius function (strength, fatigue resistance, and electromyographic onset activation timing), tensor fascia latae activation timing and kinematic joint coupling.

Hypothesis 1.1: Runners with ITBS will exhibit significantly different gluteus medius and tensor fascia latae activation timing as compared to healthy controls during speed-controlled overground running.

Hypothesis 1.2: Runners with ITBS will exhibit significantly different gluteus medius fatigue resistance and gluteus medius strength during isometric testing as compared to healthy controls during speed-controlled overground running.

Hypothesis 1.3: Runners with ITBS will exhibit significantly altered hip frontal-hip transverse plane and hip frontal-knee transverse plane kinematic joint coupling as compared to healthy runners during speed-controlled overground running.

Aim 2: To determine the effect of performing a run to exertion (17/20 rating on Borg's Rating of Perceived Exertion) on hip joint kinematics and kinetics, and joint coupling in runners with ITBS as compared to healthy controls during speed-controlled overground running.

Hypothesis 2.1: As a result of exertion, runners with ITBS will have a greater change in stance phase peak hip adduction and internal rotation as compared to healthy controls during speed-controlled overground running.

Hypothesis 2.2: As a result of exertion, runners with ITBS will have a greater change in stance phase peak hip abductor and external rotator moments as compared to healthy controls during speed-controlled overground running.

Hypothesis 2.3: As a result of exertion, runners with ITBS will have a greater change in stance phase hip frontal-hip transverse plane and hip frontal-knee transverse plane kinematic joint coupling as compared to healthy controls during speed-controlled overground running.

Aim 3: To determine the effect of performing a run to exertion (17/20 rating on Borg's Rating of Perceived Exertion) on gluteus medius function and tensor fascia latae activation timing in runners with ITBS as compared to those in healthy controls.

Hypothesis 3.1: As a result of exertion, runners with ITBS will have a greater change in gluteus medius and tensor fascia latae activation timing during overground running.

Hypothesis 3.2: As a result of exertion, runners with ITBS will have a greater change in gluteus medius fatigue resistance and gluteus medius strength during isometric testing.

Summary of Results

Twenty healthy female runners and 12 female runners with a current diagnosis of ITBS enrolled in this study. While the groups did not differ with respect to age, runners in the ITBS group tended to be taller and weigh more than the control runners. All participants were between the ages of 18 and 50 years old. All were rearfoot strikers, running a minimum of 15 miles per week and were able to run at least one 9-minute mile. In addition, runners in the control group were free from all neuromuscular and musculoskeletal disorders for 6 months prior to data collection, while runners in the ITBS group were free from all neuromuscular and musculoskeletal disorders, with the exception of ITBS, for 6 months prior to data collection.

Aim 1

Previous studies have examined hip muscle strength in runners with ITBS, however, findings have been contradictory[28,29,7334] leaving researchers and clinicians without conclusive evidence. Our current study found that hip abductor strength in the involved limb of runners with ITBS was not significantly different from that of a non-injured control group. These findings were consistent with those of Grau et al.[73] who, using similar testing methodology, found there to be no differences in isometric or isokinetic strength between runners with ITBS and healthy controls. It has been suggested that weakness of the hip abductor muscles may result in increased hip adduction during the stance phase of running gait.[25,29,32] Based on our study findings, hip abductor strength deficits may play less of a role in the presentation of ITBS than previously thought.

While differences in isometric hip abduction strength were not found between runners with ITBS and healthy controls, there was a significant difference in the ability of the runners' gluteus medius muscle to resist fatigue. Our study findings suggest that the hip abductors of runners with ITBS demonstrate less resistance to fatigue then hip abductors of healthy runners. Based on the clinical presentation of ITBS, the idea that the injured runners' hip abductors are not "weaker" but less resistant to fatigue is of particular interest. Symptoms of ITBS tend not to be present at the initiation of a run, but arise and/or worsen after 20-30 minutes of running. These results, combined with the clinical presentation or ITBS, suggest that hip abductor endurance or fatigue resistance may play a lager role in the syndrome's etiology than strength alone.

Strength and fatigue resistance data were collected during isometric contractions. The gluteus medius muscle, however, functions in an eccentric capacity during loading response. Therefore, an additional purpose of this study was to examine the activation timing of the gluteus medius and tensor fascia latae muscles during terminal swing as the muscle is preparing itself for the large eccentric demands placed on it during loading response. Our study found that terminal swing gluteus medius activation occurred, on average, at 90 and 92% of the gait cycle in injured and uninjured runners respectively. The tensor fascia latae muscle activated at 95 and 94% of the cycle in the same groups. These data are consistent with Mann and colleagues' findings that the gluteus medius and tensor fascia latae muscles activate at 90 and 95% of the gait cycle respectively.[57] Proper activation timing of these muscles prepares the runner to accept weight and absorb shock during the period of running gait where the largest amount of vertical force is exerted on the body. Contrary to our hypothesis, gluteus medius and tensor fascia latae terminal swing activation timing were not significantly different between injured and healthy runners during fresh-state, non-fatigued running. It was expected that runners with ITBS would demonstrate delayed activation timing, which would result in increased hip adduction angles during stance. Our data, however, do not suggest that hip abductor activation timing is associated with injury status. It is possible that other neuromuscular mechanisms such as the duration or amplitude of activation are different between groups; however, these variables were not examined in this study.

When comparing between groups, there were no significant differences in hip frontal- hip transverse plane or hip frontal- knee transverse plane joint coupling values. Based on results from recent studies documenting altered hip adduction and knee internal rotation angles in runners with susceptibility for ITBS[25,32,34], differences in joint coupling were expected between the experimental groups. Yet, the absence of a difference between coupling values in the two groups were not in support of our original hypothesis and suggest that any kinematic alterations in the injured runners did not result in changes in the degree of joint coupling occurring between and within joint motions. It is possible that within the injured population, changes in joint kinematics are occurring simultaneously between the motions of interest and therefore are not affecting the degree of coupling between motions. Further, it should be noted that many of the studies that have related kinematic alterations to ITBS have been conducted in runners who were healthy at the time of data collection and has either a retrospective or prospective history of ITBS. Conversely, the current study was conducted on runners with current symptoms of ITBS.

The results of this study suggest that, when measured in a fresh, non-fatigued state, hip abductor strength and onset activation timing as well as kinematic joint coupling at the hip and knee do not appear to be related with ITBS. Runners with ITBS do, however, demonstrate decreased resistance to fatigue in their gluteus medius muscle. This suggests that endurance of the hip abductors may be a factor in the presentation of the syndrome.

Aim 2

The purpose of this aim was to determine the effect of performing a run to exertion on hip joint kinematics, kinetics and joint coupling patterns in runners with ITBS as compared to healthy controls. There was a significant group-by-exertion interaction noted with respect to stance phase peak hip adduction angles. Following a run to exertion, runners with ITBS demonstrated a 9.1% decrease in stance phase peak hip adduction angles as compared with their healthy counterparts who demonstrated no significant change in their frontal plane hip mechanics. This suggests that over the course of a run to exertion, runners with ITBS are altering their gait such that they *decrease* their peak stance phase hip adduction angles. In light of previous findings by Noehren et al.[25] who found that runners who go on to have ITBS symptoms have increased hip adduction angles, the direction of this change is surprising. However, it is possible that these adjustments are present as an attempt to prevent or relieve symptoms associated with ITBS. Decreased angles of hip adduction may result in decreased length of the ITB, decreased strain within its fibers, and decreased contact distally at the lateral femoral epicondyle. Runners who are currently injured may have subconsciously made this stance phase accommodation to relieve painful symptoms over the course of their run.

This study did not identify a significant group-by-exertion interaction when examining peak stance phase hip internal rotation angles, nor peak stance phase hip abductor and external rotator moments suggesting that, with regards to these variables, exertion does not effect these runners differently. In addition, with respect to hip internal rotation angles, there were no significant main effects of group or time. Similarly, there was no main effect of group on peak peak hip abductor or external rotator moments. Noehren et al.[25] reported similar findings when examining hip abductor moments in healthy female runners and female runners with ITBS. There was, however, a significant effect of exertion on hip abductor and external rotator moments whereby both groups of runners demonstrated a decrease in their joint moments following the run to exertion. This suggests that the demands on their hip abductor and external rotator musculature may have been reduced over the course of the run. The source of this reduction is unclear. There was no effect of exertion on hip adduction or internal rotation angles in the pooled group of ITBS and healthy runners. Thus, it could be hypothesized that another kinematic alteration, such as lateral trunk lean, which was not examined in this study, may contribute to these changes.

The results of this study show that runners with ITBS are not affected differently by exertion than healthy runners with respect to frontal and transverse plane hip joint coupling or with frontal plane hip and transverse plane knee joint coupling. These findings are interesting in light of the existing studies of runners with susceptibility to ITBS, which has documented single joint kinematic alterations involving these motions[25,27,32,34]. However, these previous studies have examined runners in their fresh, non-exerted state. Further, some studied runners who were not symptomatic at the time of data collection, but had a retrospective or prospective history of ITBS.

This study is the first to report overground running kinematic and kinetic alterations at the hip as a result of exertion in runners with ITBS. Presently, there is not a consensus in the literature with respect to the frontal plane hip biomechanical differences between healthy runners and those with ITBS. However, this study's findings of decreasing stance phase peak hip adduction angles in an exerted state are supported by the findings by Grau and colleagues[34] who documented decreased hip adduction angles during overground running in a population of runners with ITBS who were presently symptomatic. It has previously been suggested that hip abductor weakness may result in increased stance phase hip adduction angles in runners with ITBS.[29,32] However, our findings suggest that hip abductor weakness is not the sole cause of iliotibial band syndrome. These finding may have implications that support changes in clinical treatment of runners with ITBS. While many physical therapists and clinicians seek to strengthen the hip abductor musculature and provide immediate neuromuscular re-education or gait re-training to increase hip adduction motion, the results of this study suggest that this education may not be so clearly indicated as symptomatic runners are already making these modifications.

Aim 3

The purpose of this aim was to determine the effects of performing a run to exertion on hip muscle electromyography and hip abductor strength. It was found that runners with ITBS exhibited significantly different changes in their gluteus medius rate of fatigue during isometric testing. In healthy runners, the gluteus medius rate of fatigue remained largely unchanged from a pre- to post-exertion state, however runners with ITBS demonstrated a decrease in the slope of their median power frequency vs. time plot following the run to exertion. A decrease in the slope of the median frequency vs. time curve suggests that the gluteus medius muscle of runners with ITBS is more resistant to fatigue when in an exerted state. However, in a post-exertion state, runners with ITBS demonstrated a significantly lower initial median frequency value than when in a non-fatigued state. This was in contrast to the control runners who demonstrated no change in their initial median frequency value from pre- to post-exertion. The decreased magnitude of the initial median frequency in injured runners suggests that a fatigue-related frequency shift had already occurred and therefore was not detected when examined during isometric testing. Therefore, the finding of decreased gluteus medius initial median frequency magnitude following a run to exertion in the injured runners supports the notion that the gluteus medius muscle of runners with ITBS is less resistant to fatigue than that of their healthy counterparts.

This study did not identify a significant group-by-exertion interaction when examining gluteus medius strength and gluteus medius and tensor fascia latae terminal swing activation timing. In addition, there were no significant main effects of group or of time when examining terminal swing onset activation timing. When collapsed across groups, the gluteus medius muscle became active on average at 91.4 and 92.6% of the gait cycle when in pre- and post-exertion states. In pre- and post-exertion states, the tensor fascia latae became active on average at 94.1 and 95.7% of the gait cycle. These ranges are consistent with findings from Mann and colleagues[57] who documented gluteus medius and tensor fascia latae activation at 90 and 95% of the gait cycle respectively. Our study is the first of its kind to examine the effects of exertion on hip muscle onset timing. The gluteus medius and tensor fascia latae muscle activate during terminal swing to provide hip joint abductor stability prior-to and during loading response.[57] Based on our study findings, it does not appear that the gluteus medius or tensor fascia latae muscles are early or delayed in their onset. Early activation would have indicated that the muscles were being overworked or utilized in a compensatory manner. A delay in their activation may have indicated a dysfunction that would alter kinematics and potentially act as a source of symptoms. The results from our study, however, do not support either of these scenarios and instead suggests that these muscles are becoming active at the appropriate time. Previous studies have documented alterations in stance phase hip kinematics present in runners with ITBS. The results of our study do not suggest that activation timing of the gluteus medius and tensor fascia latae muscles are to blame. It is possible that it is the total activation timing as a percent of the gait cycle or the timing of stance phase cessation (rather than swing phase activation) that differentiates injured from uninjured runners. Future studies should hypothesize on and examine these variables.

Following the run to exertion, both control runners and those with ITBS demonstrated a statistically significant decrease in hip abductor strength. The direction of this change is not surprising as it suggests that the run to exertion fatigued the gluteus medius muscle and decreased its ability to generate torque. This change, however, was consistent across groups and did not differentiate injured from uninjured runners.

Limitations

There are multiple limitations of this study that should be considered in future work. Despite our best efforts at minimizing motion artifact, running is inherently a high impact activity. Therefore, it is possible that EMG signal which included motion artifact were contained in data analysis. The use of wireless EMG, rather than our non-telemetered system may have helped to minimize motion artifact. Following the run to exertion, there was also the chance of perspiration gathering beneath the electrodes. To minimize the effect of perspiration under the electrodes, we utilized separate snap-leads rather than applying the pre-amplifier directly over the muscle. Nonetheless, there is no guarantee that perspiration beneath the electrodes did not affect the EMG signal.

Our sample consisted of females between the ages of 18 and 50 years old. While this age range increases the generalizability of our data, running mechanics are known to change with age.[80-82] Future studies should consider narrower age ranges.

An additional limitation of our study is that data were collected with all runners wearing standardized footwear. This selection was made based on the knowledge that choice of footwear interacts with lower extremity mechanics[83-85], therefore, letting runners utilize their own footwear may have added a confounding variable. Our data were collected with runners wearing a “neutral” running shoe. If runners are accustomed to running in footwear with pronatory or supinatory control, their mechanics have been affected by running in laboratory-provided shoes. Limiting inclusion criteria to runners who were accustomed to wearing neutral running shoes would have minimized this effect.

Lastly, the design of our treadmill run to exertion may have resulted in our runners reaching aerobic fatigue rather than true muscular fatigue. We asked runners to perform the run at their estimated 5-kilometer race pace. In addition, we chose to qualify them as "exerted" based solely on their self-perceived rating of exertion. The addition of a VO2 max protocol as a criterion for fatigue or by having them perform a slower, but longer treadmill run may have assured us that our runners were not simply aerobically fatigued.

Future Research

Electromyographic, kinematic and strength data collected during this study will be further analyzed to further investigate associations between these factors and ITBS. Further examination of the gluteus medius and tensor fascia latae EMG data may include: the muscles' total activation timing throughout the gait cycle, their timing of cessation following loading response, and their timing of peak activity during loading response. Future research should consider EMG examination of other lower extremity muscles including the hip adductors and gluteus maximus in a population of runners with ITBS.

It has been proposed that a relationship exists between hip abductor strength and lower extremity kinematics[25,28,29,31,32], yet no studies have examined this relationship. Strength and kinematic data collected during this study should be examined to investigate whether a relationship exists between the two variables. In addition, future research should include the collection and analysis of data on male runners. Finally, additional runners with ITBS will continue to be recruited so that more data may be collected and statistical power increased for the underpowered variables.

Future research should consider a prospective study design that follows runners who are currently symptomatic with ITBS until they have been symptom-free for 6 months. Kinematic, kinetic, EMG, strength data and each variable's response to exertion should be examined to determine whether, and to what extent, lower extremity mechanics change when symptoms of ITBS are not present. This information will give clinicians insight into the differences between running mechanics at the time of injury and mechanics that will likely be present upon completion of physical therapy.

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