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The Effects of Plantar Fasciitis on Multi-segment Foot Running Gait Kinematics

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THE EFFECTS OF PLANTAR FASCIITIS ON MULTI-SEGMENT FOOT RUNNING

GAIT KINEMATICS

by

Robin L. Bauer

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ABSTRACT

THE EFFECTS OF PLANTAR FASCIITIS ON MULIT-SEGMENT FOOT RUNNING
GAIT KINEMATICS

by

Robin L. Bauer

University of Wisconsin-Milwaukee, 2012
Under the Supervision of Professor Stephen C. Cobb

Plantar fasciitis is a common lower extremity injury caused by mechanical overload that affects 10% of all runners. Despite its commonality, research results investigating the etiology of the condition and the most efficacious treatment have been equivocal. A potential limitation of previous research assessing the mechanical changes associated with plantar fasciitis may be the modeling of the foot as a single segment. To date no study has investigated running kinematics in individuals with plantar fasciitis using a multi-segment foot model. Sonography has also reported plantar fascia thickening and degeneration associated with plantar fasciitis in non-athletic populations; however it has not been used to investigate the plantar fascia in runners with plantar fasciitis. Therefore, the primary purpose of this study was to compare running kinematics between runners with plantar fasciitis and uninjured runners using a six foot segment model. The secondary purpose was to investigate differences in plantar fascia thickness between the

two groups. Fifteen runners with plantar fasciitis (age: 30 ± 8.74 yrs, mass: 67.98 ± 8.20 kg) and 15 age, gender and mileage matched uninjured runners (age: 29.33 ± 6.53 yrs, height: 170.52 ± 7.78 cm, mass: 68.07 ± 9.99 kg) were recruited. Data collection included foot structure assessment, ultrasound imaging, and running gait analysis. Stance phase was separated into 4 subphases, and MANOVAs ($\alpha \leq 0.05$) were performed to assess between-subject ROM differences for the functional articulations (rearfoot complex, calcaneocuboid, and calcaneonavicular complex, medial and lateral forefoot, and 1st metatarsophalangeal complex). Independent t-tests ($\alpha \leq 0.05$) were conducted to investigate differences in plantar fascia thickness.

Results revealed calcaneocuboid eversion ROM during phase 1 ($p = 0.003$) and plantar fascia thickness ($p = 0.004$) were significantly greater in the plantar fasciitis group. The increased eversion excursion of the calcaneocuboid in the plantar fasciitis group may suggest decreased lateral midfoot stability. Although the results of this study advance the understanding of the effect of plantar fasciitis on running gait, additional study of the influence of extrinsic and intrinsic foot musculature and foot strike pattern are warranted before conclusions regarding the effect of plantar fasciitis on running gait can be drawn.

I dedicate this work to Brian, Mom, Dad, Lindsey and Amy

Thank you for your unending encouragement, support, and love.

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CHAPTER 1: INTRODUCTION

Background

Overuse syndromes are the most frequently occurring injuries that affect runners (Messier & Pittala, 1988). Among them, plantar fasciitis is the third most frequently diagnosed injury (Taunton, Ryan, Clement, McKenzie, Lloyd-Smith, et al., 2002), affecting 10% of all runners. In one retrospective study on the prevalence of lower extremity injuries among runners, plantar fasciitis was the most common incurred injury (Williams, McClay, & Hamill, 2001). Plantar fasciitis is not, however, limited to runners. General population epidemiological studies have suggested that plantar fasciitis affects an estimated one in ten people at some point in their lifetime (Crawford, 2005). Furthermore, approximately one million patient visits to office-based physicians and to hospital outpatient departments per year are estimated to be for plantar fascia related symptoms (Riddle & Schappert, 2004). Plantar fasciitis is also one of the most common overuse injuries suffered by military personnel (Roy, 2011; Scher et al., 2009), and the most common cause of chronic heel pain (Irving, Cook, Young, & Menz, 2008). In addition to heel pain, plantar fasciitis also has a significant negative impact on general health-related quality of life (Irving et al., 2008). Specifically, patients with plantar fasciitis become more socially isolated, lack the energy to participate in their usual activities, and generally demonstrate a decreased ability to perform a broad range of physical tasks when compared to those without the condition (Irving et al., 2008). Coupled with reduced mobility leading to inactivity and weight gain, and therefore an increased risk for numerous chronic diseases, plantar fasciitis is a serious public health problem (Irving et al., 2008; Young, Rutherford, & Niedfeldt, 2001).

Despite the commonality of plantar fasciitis, the etiology of the injury is not well understood, and evidence supporting the effectiveness of current treatment options is limited (Cole, Seto, & Gazewood, 2005; Roos, Engstrom, & Soderberg, 2006). Most authors agree that plantar fasciitis is a multifactorial problem (Allen & Gross, 2003; Wearing, Smeathers, Urry, Hennig, & Hills, 2006) and there is general clinical consensus that mechanical dysfunction is the primary contributing factor. The mechanical dysfunction is theorized to cause repetitive microtrauma and overuse that ultimately progresses to plantar fasciitis (Glazer & Hosey, 2004). Anatomical pathoetiological factors that may lead to the mechanical dysfunction during gait and ultimately plantar fasciitis include: reduced ankle dorsiflexion range of motion (Cornwall & McPoil, 1999; Kibler, Goldberg, & Chandler, 1991; Labovitz, Yu, & Kim, 2011; Riddle, Pulisic, Pidcoe, & Johnson, 2003); excessive pronation (Middleton & Kolodin, 1992; Taunton, Clement, & McNicol, 1982; Wearing et al., 2006) leg length discrepancy (Glazer & Hosey, 2004; Krivickas, 1997; Subotnick, 1985); increased first metatarsophalangeal joint motion (Wearing et al., 2004); and weak intrinsic foot muscles (Allen & Gross, 2003; Kibler et al., 1991; Wearing et al., 2007). Overtraining and inadequate shoewear have also been reported to lead to the development of plantar fasciitis (Glazer & Hosey, 2004; Taunton et al., 1982). To date, however, results of the studies have been largely inconsistent. In addition to the previously mentioned factors, results of experimental studies aimed at identifying the dysfunction during gait, however, have been equivocal (Wearing et al., 2006). Specifically, several studies have observed changes in gait (Chang, van Emmerik, & Hamill, 2007; Wearing et al., 2007), however, a recent rearfoot model investigating female runners with plantar fasciitis did not report kinematic

differences between participants with and without plantar fasciitis (Pohl, Hamill, & Davis, 2009).

Although the anatomical and biomechanical factors associated with plantar fasciitis are unclear, the functions of the plantar fascia are well documented (Sarrafian, 1987; Taunton et al., 1982; Wearing et al., 2006). The plantar fascia provides support during the stance phase of gait, maintains the medial longitudinal arch during midstance, and aids in re-supination of the foot during late midstance and propulsion (Taunton et al., 1982). Biomechanical analyses have shown that elongation of the arch during midstance increases tension within the plantar fascia (Sarrafian, 1987). The increased tension within the plantar fascia aids in locking the midtarsal joints to prepare the foot for propulsion. During the propulsive phase the plantar fascia functions like a windlass to aid in resupination of the foot and thus provide stability for the arch prior to toe-off , (Taunton et al., 1982). Specifically, the windlass mechanism occurs during toe extension when the plantar fascia is wound around the metatarsal heads, thereby shortening its effective length and increasing tension within the fascia (Hicks, 1954). The activation of this windlass mechanism, however, is thought to occur only when sufficient tension is produced within the plantar fascia (Hicks, 1954).

Some authors have theorized that both dysfunction of the windlass mechanism during gait and decreased tension within the plantar fascia may occur as a result of degeneration of the fascia associated with plantar fasciitis (Wearing et al., 2004; Wu, Chang, Mio, Chen, & Wang, 2011). Supporting the theories, walking gait studies have reported compensatory changes in gait in individuals with plantar fasciitis in the first metatarsophalangeal joint angle (Wearing et al., 2004) and in the forefoot (Chang et al.,

2007). Additionally, in a study examining the stiffness of the plantar fascia using elastography, Wu et al. (2011) reported a significant decrease in plantar fascia stiffness in individuals with plantar fasciitis. Several authors have also reported evidence of degeneration in the plantar fascia associated with plantar fasciitis using ultrasound imaging. The studies have revealed a hypoechoic appearance and adaptive thickening of the plantar fascia, which signifies microtears and degeneration, respectively (Cardinal, Chhem, Beauregard, Aubin, & Pelletier, 1996; Lemont, Ammirati, & Usen, 2003; Wearing et al., 2006). In previous studies, the threshold for plantar fasciitis has been suggested to be a thickness of greater than 4.0 mm. However, these studies have used participants with a wide range of age and activity level, so it is unknown if the thresholds would apply to younger very active groups (Cardinal et al., 1996; Fabrikant & Park, 2011; Karabay et al., 2007). Together, these studies suggest that structural changes in the plantar fascia that occur with plantar fasciitis may lead to decreased tension and compensatory changes during gait; however, these effects are unknown in runners. Due to the anatomical structure and function of the plantar fascia, the compensatory changes during gait may occur in multiple segments of the foot.

As previously stated, mechanical dysfunction is theorized to be a primary factor associated with plantar fasciitis, but these mechanical changes are not well understood. A limitation of the majority of previous studies may be the modeling of the entire foot as a single rigid segment, tracking only the rearfoot complex. Increased forefoot pronation has been reported in patients with plantar fasciitis during walking, suggesting the single segment model may be an oversimplification of the foot (Chang et al., 2007). Moreover, Pohl et al. (2009) reported that individuals with plantar fasciitis did not have different

static rearfoot positions but they did have differences in the static arch position. This observation may further support the argument that mechanical changes associated with plantar fasciitis may occur in the midfoot and forefoot segments. The contribution of distal foot motion on foot function during gait has also been reported by Arndt et al. (2007) and Cobb et al. (2009), who investigated multi-segment foot kinematics during jogging and walking, respectively. Arndt et al. (2007) demonstrated significantly increased midfoot mobility with in-vivo running kinematics, while Cobb et al. (2009) showed significant differences in motion distal to the calcaneus between participants with typical and low arch structure during walking. These results and the fact that midfoot, forefoot, and hallux motion during gait may directly influence tension in the plantar fascia, emphasize the need to analyze foot function with a multi-segment model.

With respect to treatment of plantar fasciitis, 90-95% of the reported patients diagnosed with plantar fasciitis receive conservative treatment (O'Malley et al., 2000; Wearing et al., 2006). The primary conservative treatment options include shoe inserts, custom molded orthotics, stretching and strengthening exercises, and custom-made night splits (Cole et al., 2005; P. F. Davis, Severud, & Baxter, 1994; Kogler, Solomonidis, & Paul, 1996; Roos et al., 2006). Even with successful conservative treatment, the median duration of symptoms is 12 months, with a range of 6 to 96 months (Irving et al., 2008) and a significant minority has ongoing debility (Davis et al., 1994). Moreover, a long-term follow-up study on plantar fasciitis cases reported that after four years only 80% of the patients were completely pain-free (Wolgin, Cook, Graham, & Mauldin, 1994). Although the symptoms of plantar fasciitis may ultimately be resolved, the fact that

patients diagnosed with the condition are typically affected for a prolonged period of time suggests that current conservative treatment protocols are not sufficiently effective.

In addition to conservative treatment, 5-10% of plantar fasciitis patients require surgical intervention (O'Malley et al., 2000). Fasciotomy, both partial and complete surgical sectioning of the plantar fascia, is the most common surgical intervention (Tweed, Barnes, & Allen, 2009). Surgical outcomes for plantar fasciitis patients however, are equivocal with mixed reports of activity level and disability following the surgery (Kitaoka et al., 1997; O'Malley et al., 2000). Short-term outcomes of fasciotomy are reported to be 71% successful in patients with heel pain (Kitaoka, Luo, & An, 1997; Leach, Seavey, & Salter, 1986), but the long-term consequences of altering the structure of the plantar fascia are not well understood. Although fasciotomy may temporarily relieve symptomatic complaints in individuals suffering from plantar fasciitis (Davies, Weiss, & Saxby, 1999), there is growing concern that fasciotomy may lead to biomechanical changes including a decrease in arch height and a reduction in the structural stability of the foot (Huang, Kitaoka, An, & Chao, 1993; Kitaoka et al., 1997; Tweed et al., 2009). These structural changes that occur following surgery have been hypothesized to change gait mechanics that may lead to the development of additional joint/soft tissue pathology over time. More recently, endoscopic plantar fascia release has been proposed as a better alternative to the traditional open approach because it speeds recovery and enables patients to return to activity faster (Boyle, Witt, & Riegger-Krugh, 2003); however, even with this improved surgical approach, 2 - 35% of patients have continued symptoms (Schepsis, Leach, & Gorzyca, 1991).

The lack of understanding of the pathoetiological factors associated with plantar fasciitis could be a contributing factor to the ineffectiveness of current treatment protocols. Until the pathoetiological factors of plantar fasciitis are identified, it will be difficult to develop interventions that effectively treat the condition. Another contributing factor to the lack of understanding of the etiological factors associated with plantar fasciitis is the lack of a gold standard diagnostic test for plantar fasciitis. Currently, plantar fasciitis is most commonly a clinical diagnosis (Wearing et al., 2006). The characteristic clinical signs and symptoms used to diagnose plantar fasciitis include: complaint of a sharp, localized pain at the base of the heel where the plantar fascia attaches to the calcaneus (Cole et al., 2005; McBryde, 1984; Singh, Angel, Bentley, & Trevino, 1997; Tountas & Fornasier, 1996); and a gradual onset of pain that is typically worse with the first few steps in the morning and following periods of inactivity (Buchbinder, 2004; Cole et al., 2005; Kibler et al., 1991). While the signs and symptoms are characteristic of plantar fasciitis, they may also be present in other conditions such as heel spurs and tarsal tunnel syndrome. The presence of fluid collection at the origin of the plantar fascia has also been observed in several studies, which is attributed to repetitive microtears that result from the mechanical stress on the plantar fascia (Akfirat, Sen, & Gunes, 2003; Gibbon & Long, 1999; Sabir, Demirlenk, Yagci, Karabulut, & Cubukcu, 2005). Results of these studies suggest that, when used in conjunction with a clinical exam, ultrasound imaging may be a useful clinical tool to aid in the diagnosing plantar fasciitis.

Purpose

Given the lack of understanding of mechanical effects and structural changes in the plantar fascia in runners, the current study had two purposes. The primary purpose was to compare foot kinematics in runners with plantar fasciitis and injury-free runners using a multi-segment foot model. The secondary purpose of the study was to investigate the differences in plantar fascia thickness between runners with plantar fasciitis and uninjured runners. It was hypothesized that participants with plantar fasciitis would have altered kinematics during the mid and late stance phases of gait. Specifically, it was hypothesized that in patients with plantar fasciitis, the medial midfoot and forefoot would be more mobile during midstance, and the first metatarsophalangeal joint would exhibit increased extension in late stance due to degeneration and decreased stiffness of the plantar fascia.

Delimitations

1. Data were collected on runners and therefore any generalizations made from the findings are limited to this population. The results are not applicable to the general population diagnosed with plantar fasciitis. Specifically, plantar fasciitis in the sedentary population develop due to different factors. The appearance and structure of the plantar fascia in runners compared to that of sedentary individuals is also unknown.

2. This study utilized common clinical symptoms of plantar fasciitis as inclusionary criteria. No specific diagnostic imaging, or physician diagnosis, was included. However, a Certified Athletic Trainer performed a physical exam to rule out all other potential causes of heel pain.

3. Participants ran in a flat sandal, not necessarily built for running, in order to use the multi-segment foot model. Although all participants were provided adequate time to warm-up in the sandal, it is unknown whether they altered their gait to adjust to running in the sandal.

Assumptions

1. Participants honestly answered the questions on the background and activity level questionnaires.

2. All lower extremity segments are rigid bodies.

3. The surface marker based multi-segment foot model kinematics represent the bone movement of each functional articulation it is representing.

4. The motion of the foot in the sandal is not different than the motion of the foot in a running shoe.

Limitations

1. Surface marker based models are not invasive so they cannot directly measure bone movement. Because the markers are placed on the surface of the skin, there will be error due to skin and adipose tissue movement that cannot be completely eliminated. This soft tissue movement of the foot and leg will be reduced with the use of a liquid adhesive, adhesive tape, four-marker marker clusters, and a rigid body reconstruction optimization procedure.

2. This is a retrospective study looking at the mechanical changes in runners with plantar fasciitis. The cause of any mechanical changes can only be interpreted from the results as potential contributing factors.

Significance

The results of the study enhanced understanding of the mechanical effects of plantar fasciitis on running gait mechanics. Previous studies have only analyzed rearfoot or rearfoot-forefoot kinematics, both of which ignore the midfoot motion of the foot. However, more recently the movement and importance of these midfoot bones has been identified in individuals without plantar fasciitis during walking and running gait studies. To capture the movement of the multiple bones of the foot during running, it was necessary to use a multi-segment foot model. Specifically, use of a multi-segment foot model allowed for identification of potential mechanical dysfunction in the forefoot, midfoot, and hallux segments during running gait that may result from plantar fasciitis. In addition to understanding the kinematics of the foot, this study also investigated the differences in plantar fascia thickness between runners with plantar fasciitis and uninjured runners. Moreover, it contributed to the understanding of the etiology of plantar fasciitis and may contribute to the development of more effective treatment protocols.

CHAPTER 2: REVIEW OF LITERATURE

Introduction

The primary purpose of the current study was to compare foot kinematics in runners with plantar fasciitis and injury-free runners using a multi-segment foot model. This Review of Literature begins with an overview of the anatomy and functional role of the plantar fascia during gait. The anatomy of the plantar fascia is crucial to understanding its function, and knowledge of its function is critical to understanding the mechanical mechanisms theorized to cause plantar fasciitis. Following the overview, the histopathology of plantar fasciitis and the theorized relationship between the histopathology and mechanical function are discussed. The next two sections review the pathoetiological factors theorized to contribute to the development plantar fasciitis and the mechanical effects of plantar fasciitis on gait. Finally, the role of a multi-segment foot model in advancing and understanding foot function is discussed. These final sections will bring the Review of Literature full circle, providing the necessary background information to explain the importance of utilizing a multi-segment foot model to investigate the mechanical effects of plantar fasciitis on running gait.

The secondary purpose of the study was to investigate differences in plantar fascia thickness between runners with plantar fasciitis and uninjured runners; therefore, the final section of this Review of Literature discusses the current clinical and diagnostic methods of diagnosing plantar fasciitis.

Structure and Function of the Plantar Fascia

Anatomy of the Plantar Fascia

The plantar fascia is a broad, flat, fibrous, tendon-like structure that covers the sole of the foot (Uden, Boesch, & Kumar, 2011) (Figure 1). It consists of non-contractile irregularly ordered collagen fibers with minimal elastic properties that span the transverse tarsal, tarsometatarsal, and metatarsophalangeal joints (Nordin & Frankel, 2001). The plantar fascia originates at the medial tubercle of the calcaneus then divides distally into superficial and deep segments at the metatarsal heads. The superficial fibers insert into the skin, while the deep segments attach to the transverse metatarsophalangeal ligaments and to the plantar surface of the proximal digits (Marieb, 2001).
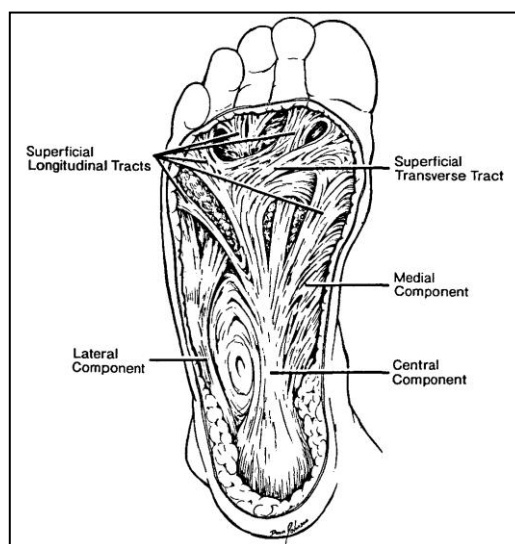
Figure 1. Medial, central and lateral bands of the plantar fascia (Brasile & Hedrick, 1996).

The plantar fascia is a continuous structure and is often described as three separate components or bands (medial, lateral, central bands) (Hedrick, 1996) (Figure 1). The medial band is a very thin structure, forming the investing fascia of the abductor hallucis muscle. Although virtually nonexistent at the proximal end, it becomes larger as

it courses distally along the medial sole to join the dorsal fascia of the foot (Hedrick, 1996). The lateral band is a more substantial component of the plantar fascia that originates at the lateral margin of the medial tubercle of the calcaneus, extends toward the cuboid and inserts into the base of the fifth metatarsal (Brasile & Hedrick, 1996). Its thickness and development, however, are variable (Brasile & Hedrick, 1996; Cralley, Schuberth, & Fitch, 1982; Hiramoto, 1983). In some individuals the band is thick and fully developed, whereas in approximately 12% of individuals, it is completely absent (Dylevsky, 1988). Due to this variability, the significance and importance of the lateral band are not well understood. Lastly, the central band originates at the plantar aspect of the medial process of the medial calcaneal tuberosity (Brasile & Hedrick, 1996; Mitchell, Meyer, & Krueger, 1991) and receives fibers from the Achilles tendon and plantaris tendons proximally (Brasile & Hedrick, 1996). It invests the central plantar muscles and resembles the palmar aponeurosis of the palm of the hand, but is tougher, denser, and more elongated (Maffulli, Binfield, Moore, & King, 1999). At its origin, the central band is approximately 1.5 to 2 cm wide. It then expands into a triangular shape as it divides distally into five longitudinally oriented bands along the sole of the foot (Bojsen-Møller, 1976; Hedrick, 1996). It is this band that spans the medial longitudinal arch of the foot (Cornwall & McPoil, 1999; Roxas, 2005) and is considered to be the major component of the plantar fascia both structurally and functionally (Hiramoto, 1983; Pontious, Flanigan, & Hillstrom, 1996). All references to the plantar fascia in this document refer to the central band since it is considered to be the most structurally and functionally significant of the three bands (Wearing et al., 2006).

Functions of the Plantar Fascia

The plantar fascia is a thick and tenacious structure that provides stability to the multiple joints of the foot, supports the medial longitudinal arch, and protects the sole of the foot from injury (Maffulli et al., 1999). During static stance, the weight of the body is supported almost entirely by the passive structures of the foot with the plantar fascia functioning as the primary support (Basmajian & Stecko, 1963). During gait, the plantar fascia functions to maintain the medial longitudinal arch and to aid in re-supination of the foot in the late midstance and propulsive stance subphases (Lisowski, 2004; Michaud, 1997). Specifically, during midstance, tension in the plantar fascia increases as the arch elongates (Sarrafian, 1987). This increased tension in the plantar fascia provides stability to the mid-tarsal joints and assists in re-supination of the foot during late midstance and the propulsive subphase of stance so that the foot can function as a rigid lever at toe-off (Taunton et al., 1982).

Weight-bearing Mechanisms/Theories

It is well known that the plantar fascia contributes to arch maintenance during both static stance and gait (Wearing et al., 2006). However, because it is not possible to directly measure the plantar fascia in-vivo without invasive techniques (Kim & Voloshin, 1995; Wright & Rennels, 1964), much of the current understanding of how the plantar fascia functions during stance was developed from in-vitro studies (Huang et al., 1993; Sarrafian, 1987; Wright & Rennels, 1964). From these studies, authors have demonstrated that the plantar fascia serves as a critical structure in supporting the medial longitudinal arch during loading conditions. Specifically, the medial longitudinal arch under load is typically described as functioning similar to that of a beam and a truss

(Hicks, 1954; Hicks, 1955; Sarrafian, 1987). Lake (1938) was the first to develop theories of how the foot functions under loading conditions, and Hicks (1955) experimentally confirmed the theories with an in-vitro cadaver study. When the foot is described as a beam mechanism, the structures forming the medial longitudinal arch (calcaneus, talus, navicular, cuneiforms, and medial three metatarsals) represent the beam. During weightbearing, the beam experiences bending strain, as it functions to maintain the structure of the arch. Specifically, the inferior surface of the medial longitudinal arch (the plantar ligaments of the foot) is placed under tension, while the superior surface of the medial longitudinal arch (the articulating bones of the arch) is under compression.

In addition to functioning like a beam when loaded, the arch also functions as a truss. A truss is composed of two wooden struts that are under compression, connected by a rope or a tie rod that is under tension. When described as a truss, the arch represents the triangular structure, the heel and forefoot represent the two struts, and the plantar fascia functions as the tie rod (Hicks, 1955; Nordin & Frankel, 2001). It is a mechanical truss, with the plantar fascia providing support and allowing movement via elongation and shortening when the structure is loaded. As a truss, the plantar fascia functions to modify the stiffness of the arch in relation to the weight-bearing load (Vogler & Bojsen-Moller, 2000). In an experimental study investigating the effect of loading during flat standing and toe-standing loading conditions on fresh foot amputation specimens, Hicks (1955) demonstrated that the arch functions similar to both a beam and truss during flat standing, but primarily a truss during late stance and toe off.

During flat standing, the foot functioned like a truss and a beam simultaneously (Hicks, 1955). Specifically, the medial longitudinal arch functioned like a truss, as

tension in the plantar fascia limited the amount of elongation of the arch that occurred during weight-bearing, but also prevented the arch from completely flattening (Hicks, 1955). The heel and forefoot (the two struts) of the arch were also loaded in compression to resist the tensile forces of the plantar fascia. The foot also functioned like a beam during static stance. Under vertical loading conditions the arch flattened in relation to the amount of weight placed on the body (Hicks, 1955), which is similar to a beam that bends as it is stressed with more weight. Hicks theorized that the two mechanisms function in conjunction with one another to support the medial longitudinal arch during midstance of the gait cycle.

During toe-off in gait, the windlass mechanism pulls the foot into a ray-flexed position that raises the medial longitudinal arch (Hicks, 1955). Unlike flat stance in which support of the arch is provided by both beam and truss mechanisms, in the toe-standing position, Hicks (1955) noted support for the arch is solely due to the function of the truss mechanism. Therefore, in a toe-standing position without the support of the beam mechanism, the plantar fascia may play an even greater role in arch support. Finally, because the windlass mechanism was observed in cadaver feet, Hicks (1955) theorized that the raising of the arch observed in a toe-standing position was primarily the result of support provided by the plantar fascia via the windlass mechanism rather than from the action of arch-raising muscles. In another study, Hicks (1954) mimicked toe-standing by extending the first metatarsophalangeal joint in an in-vivo radiographic study. Extension of the first metatarsophalangeal joint resulted in sliding of the phalanx on the dorsum of the metatarsal head that pulled on the plantar pads and which wrapped the plantar fascia around the heads of the metatarsals like a cable being wound on to a

windlass (Hicks, 1954). When this was performed, the plantar fascia did not shift distally because of its strong attachment to the calcaneum. Instead the windlass shifted and pulled the metatarsal heads proximally toward the calcaneus. In a subsequent in-vitro experiment, Hicks (1955) confirmed that the plantar fascia was strong enough to perform the arch-raising mechanism or the "windlass" mechanism during the toe-off phase of walking (Figure 2). Although the plantar fascia attaches to all of the metatarsal heads and each goes through extension at heel lift, the hallux is thought to be the greatest contributor to the windlass due to its greater range of motion compared to the lesser digits (Hicks, 1953).
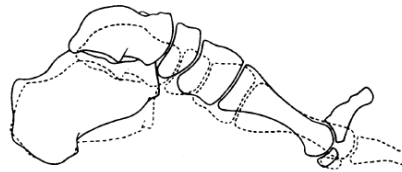
Figure 2. Demonstration of the windlass mechanism (Hicks, 1954).

In-vitro Studies

Following the development of the truss and beam theories to explain arch function and the experimental evidence of the truss and beam mechanisms during load-bearing conditions provided by Hicks (1955), researchers became interested in determining the contribution of the plantar fascia to arch maintenance during weight-bearing activities.

The majority of the research studies on the plantar fascia and its role in maintaining arch support have been in-vitro studies. Most of these studies have focused on understanding and quantifying the amount of load the plantar fascia can sustain (Huang et al., 1993; Wright & Rennels, 1964). In a significant landmark study, Wright and Rennels (1964) tested three cadaver feet in an apparatus that loaded each specimen and measured the elongation of the arch during several loading conditions up to a maximum load of 200 pounds. The majority of the change in arch length occurred with the smaller loads of 50 and 100 pounds, compared to the larger loads of 150 and 200 pounds. With the numerical data from these experiments, the authors calculated the stress-strain relationship for each specimen, which was an indication of the modulus of elasticity. As the load increased, the moduli of elasticity increased demonstrating that the specimens became stiffer with more load.

Approximately 30 years later, Huang et al. (1993) performed a similar experiment with 12 fresh-frozen human cadaveric feet. The feet were loaded with 230, 460, and 690 Newtons (51.6, 103.2, and 154.7 pounds respectively). Similar to Wright and Rennels (1964), the authors measured the horizontal displacement of the arch with each load to determine the stiffness of each specimen. In addition, Huang et al. (1965) also measured the arch displacement before and after sequential sectioning of the plantar fascia, the plantar ligaments, and the spring ligament. The authors reported that the greatest reduction in arch stiffness (25%) occurred after re-sectioning the plantar fascia compared to re-sectioning the spring ligament and the long and short plantar ligaments. Moreover, with the re-sectioning of all four tissue structures, the arch still retained 63% of its original stiffness. From this, they concluded that the plantar fascia is the greatest

contributor to arch maintenance, but also that the other supporting structures (the plantar ligaments and the spring ligament) contribute to the stability of the medial longitudinal arch.

A limitation of the first two studies is that they only loaded the foot in a flat stance condition, which is not a full indication of how the plantar fascia acts during the entire stance phase. In a different approach to understanding the role of the plantar fascia, Salathe et al. (1986) developed a 12-segment two-dimensional mathematical model of the foot to predict the tension that occurs in the plantar fascia during terminal stance. The authors analyzed the foot as a statically indeterminate structure, predicting that the greatest tension within the fascia would occur when the heel was raised off the ground and the windlass mechanism was activated. According to the previously described theory of the truss mechanism, the plantar fascia is the primary supportive structure maintaining the arch during this phase. Results of the Salathe et al. (1986) study revealed large loads on the plantar fascia and metatarsal heads during heel off. The greater the flexibility of the metatarsal heads during heel off, the larger the load on the metatarsal heads, which was deflected from the plantar fascia (E. P. Salathe, Jr., Arangio, & Salathe, 1986). Thus, in a normal-functioning foot during heel off, the plantar fascia applies a large amount of load onto the metatarsal heads, assisting in toe-off during the terminal subphase of stance.

Both the Wright and Rennels (1964) and Huang et al. (1993) in-vitro studies quantified the load bearing capacity of the plantar fascia and contributed to enhancing the knowledge of the role of the plantar fascia in maintaining arch support in different loading conditions. Although both studies provide experimental evidence of the importance of the plantar fascia to maintaining support of the arch, both studies have

several limitations that must be considered. Wright and Rennels (1964) did not include the contribution of arch-supporting structures other than the plantar fascia. As demonstrated by Huang et al. (1993), however, other passive elements contribute to arch maintenance. Furthermore, because of the nature of the studies, neither Wright and Rennels (1964) nor Huang et al. (1993) were able to determine the contribution of the extrinsic and intrinsic muscles of the foot. These structures may also be vital in supporting the arch and dissipating load, therefore the quantification of the plantar fascia's contribution from both studies may be overestimations. Finally, both studies used static loading of cadaveric specimens so the results may not be generalizable to dynamic loading of in-vivo tissues. Even with these limitations, however, it is clear that the plantar fascia contributes significantly to the support of the arch during loading. Subsequent in-vitro studies have also revealed that the plantar and spring ligaments, in conjunction with the plantar fascia, are important in storing energy and providing support to the arch (Ker et al., 1987; Kitaoka et al., 1997).

Static and Quasi-static In-vivo Studies

To address the limitation of the generalizability of results of studies performed on in-vitro tissues to in-vivo tissues, several researchers have performed in-vivo studies to quantify the contribution of the plantar fascia in maintaining the arch (Kim & Voloshin, 1995; Wright & Rennels, 1964). In the previously discussed in-vitro study, Wright and Rennels (1964) also performed an in-vivo study. The authors measured the length of the arch using radiographs after applying increasing loads in fifty-pound increments up to 200 pounds to a subject seated in a chair. Consistent with the in-vitro results, the authors

reported a significant increase in the arch length during the smaller loads and little change during the larger loads. Again, the limited change during the larger loads was attributed to stiffening of the plantar fascia. From these data, Wright and Rennels (1964) concluded that the resulting tension in the plantar fascia was approximately 47% of the weight placed on the subject's tibia. Thirty years later, Kim and Voloshin (1995) quantified the load bearing capacity of the plantar fascia in an in-vivo experiment that utilized a viscoelastic model of the foot which included additional supporting structures (the intrinsic muscles of the foot and the tendons of the extrinsic muscles) of the arch. They employed an accelerometric technique to develop a simple biomechanical model to analyze the load bearing mechanism of the foot during the stance phase of gait. The model was used to analyze change in the maximum acceleration on the ankle following plantar fascia release. The foot without the plantar fascia generated a higher acceleration than the model with the plantar fascia, emphasizing its importance in attenuating shock. The authors noted that surgical release of the plantar fascia modified the dynamic behavior of the foot due to the reduction of the dynamic load-bearing capacity of the ankle. The results suggested that the plantar fascia contributes approximately 14% of the total load on the foot, which is significantly less than the 47% suggested by Wright and Rennels (1964). However, both studies once again confirm that plantar fascia contributes a significant amount to maintaining the integrity of the arch.

In addition to loading during midstance, authors have also investigated static loading during the toe-off position. The results of the studies have revealed that the plantar fascia is relaxed until heel lift, when digital extension initiates the previously mentioned windlass mechanism (Vedi et al., 1999; Williams et al., 1999). Results of

these studies have suggested that the windlass effect is engaged at approximately 20° of first metatarsophalangeal joint extension (Vedi et al., 1999; Williams et al., 1999). As previously stated, with the increased tension, the windlass mechanism is believed to contribute to the raising of the arch to increase the stability of the foot in preparation for the propulsive phase of gait (Bohsen-Moller, 1979; Rush et al., 2000).

Previous in-vitro studies quantified the contribution of the plantar fascia to maintaining arch support, but were unable to measure the activity of the foot's intrinsic muscles during gait (Huang et al., 1993; Wright & Rennels, 1964). Therefore, although in-vitro studies have improved the understanding of the effect of loading on the plantar fascia, the results may not be generalized to in-vivo tissues. The in-vivo studies more accurately depicted the contribution of the plantar fascia in maintaining arch support; however, the studies have also been limited by the inability to quantify support of dynamic structures such as intrinsic and extrinsic muscles of the foot.

Contribution of Dynamic Structures in Maintaining Arch Stability

The plantar fascia is the main static stabilizer of the arch during gait. However, in addition to the other static stabilizers previously mentioned there are also a number of dynamic stabilizers that may also contribute to the arch support. Dynamic control of the foot and ankle is accomplished through actions of 12 extrinsic and 19 intrinsic muscles. The extrinsic muscles are the strongest and most important in providing active control of the foot during gait (Nordin & Frankel, 2001). Moreover, the muscles of the leg also enable an efficient transfer of muscle force to the floor during normal gait and ensure a smooth progression of body weight from heel contact to toe off. The soleus and gastrocnemius muscles are important during midstance of gait, acting eccentrically to

slow the forward motion of the tibia over the foot. In addition to the gastrocnemius and soleus, the tibialis posterior, a strong inverter of the foot and ankle, also acts as a dynamic supporter of the medial longitudinal arch. The tibialis posterior primarily functions to invert the subtalar joint which assists in locking the midfoot joints during mid and late stance phases to ensure rigidity of the foot during toe-off (Nordin & Frankel, 2001).

Several authors have attempted to measure the contribution of the extrinsic and intrinsic muscles during gait through in-vitro and in-vivo studies (Basmajian & Stecko, 1963; Reeser et al., 1983; Thordarson et al., 1995). Some of the earliest studies used electromyography to determine the role of these supporting foot and leg muscles in maintaining the arch. The studies suggested that the muscles contribute very little to arch support (Basmajian & Bentzon, 1954; Basmajian & Stecko, 1963; Reeser et al., 1983). Basmajian and Stecko (1963) used indwelling electrodes to study the activity in six leg and foot muscles (tibialis anterior, tibialis posterior, peroneus longus, flexor hallucis longus, abductor hallucis, and flexor digitorum brevis) of 20 subjects. The subjects were placed in a seated position and EMG activity was assessed during loading conditions of 100, 200, and 400 pounds. With the smaller loads, little muscle activity was observed, so the authors concluded that the posture was maintained primarily by the passive structures of the foot. However, the 400 pound load required an increase in muscle support. With these results, the authors concluded that static structures are the primary support for arch maintenance under normal loading conditions, while the dynamic muscles are reserved for excessive loads, including the take-off phase of walking (Basmajian & Stecko, 1963). A limitation to this study, however, was the methods employed. Although the results enhance understanding of the role of muscle support during various loading conditions,

they cannot be applied directly to gait because the subjects were in a seated position. Moreover, the authors reported technical difficulties with assessing the activity of the tibialis posterior muscle.

Thirty years later, Thordarson et al. (1995) evaluated the role of the leg muscles in addition to the plantar fascia in supporting the medial longitudinal arch in 12 fresh cadaveric specimens. The authors investigated the contribution of the dynamic support provided to the longitudinal arch during the stance phase of gait, applying plantar loads of 0, 350, and 700 Newtons (0, 78.5, and157 pounds) to the muscles' tendons (posterior tibialis, flexor digitorum longus, flexor hallucis muscle, peroneus longus, peroneus brevis, and Achilles tendon). Each was tensioned separately while the angular relationships of the first metatarsal, navicular, and talus were recorded using a 3-dimensional motion analysis system. Thordarson et al. (1995) also evaluated the contribution of the plantar fascia in supporting the arch. The authors loaded the foot while the ankle was in neutral position and with the toes in a dorsiflexed position. They confirmed that the plantar fascia had the most significant arch-supporting function in the sagittal plane: a 3.6° improvement at 350 N and a 2.3° improvement at 700 N. Moreover, results also demonstrated the dynamic contribution of the tendons of the foot, particularly of the tibialis posterior which consistently supported the arch at the 350 and 700 N loads.

Although these landmark studies have advanced the knowledge about the contribution of dynamic structures in supporting the arch during static stance, the results still may not be generalizable to gait. These limitations have led to additional research investigating muscular support provided by the extrinsic and intrinsic foot muscles during toe-standing (Hamel et al., 2001; Salathe & Arangio, 2002; Salathe et al., 1986; Sharkey

et al., 1998; Tansey & Briggs, 2001). In the studies, authors have used toe-standing to simulate the propulsion phase of gait, just before toe-off. Although the intrinsic muscles appear to be relatively quiet during midstance, their contribution appears to be equally important to that of the plantar fascia as the heel is elevated from the ground during terminal stance (Hamel et al., 2001; Tansey & Briggs, 2001). Several authors have used biomechanical models to investigate the role of extrinsic muscles in support of the arch during terminal stance (Salathe & Arangio, 2002; Salathe et al., 1986). In one study, Salathe and Arangio (2002) used a biomechanical model of the foot that included the extrinsic muscles (tendo calcaneus, tibialis posterior, halluces longus, digitorum longus, peroneus brevis, and peroneus longus), tendons, and ligaments. The study modeled the contribution of the structures during different applied loading conditions. They reported that under load, the muscles change the support distribution among the metatarsal heads and decrease the tension within the plantar fascia, particularly the portion extending to the medial rays. There was an associated increase in the force exerted by the muscles during the toe-standing condition, which is thought to help in maintaining balance in addition to maintaining the medial longitudinal arch. From this model, the authors concluded that the muscles of the foot actively support the arch during toe-off and decrease the load borne by the plantar fascia (Salathe & Arangio, 2002). However, there are several limitations to this model that should be considered. Although it provides insight to the function of the muscles during gait, it cannot precisely represent the human foot. Specifically, there are other modifications that should be considered during terminal stance since the geometry of the foot changes and the windlass mechanism (Hicks, 1954) may result in an increase of force onto the plantar fascia. Additionally, this model

excluded the role of the intrinsic muscles of the foot, which are theorized to stabilize the longitudinal arch and support it similarly to the plantar fascia (Mann, 1992). The results of Salathe and Arangio (2002) have also been different from those of experimental studies that have investigated the distribution of support under the metatarsal heads (Cavanagh, 1987; Viladot, 1992). Limitations to these studies could be a lack of understanding of the relationship between the anatomical and physiological structures that comprise the foot, and their role in support distribution during gait.

Mechanical Properties and Histopathology

Mechanical Properties of the Plantar Fascia

The plantar fascia consists of noncontractile irregularly ordered collagen fibers with minimal elastin properties (Uden et al., 2011) that allow it to provide support for the arch and passively elongate and shorten during gait (Wright & Rennels, 1964). Specifically, the elastin fibers have a low modulus of elasticity, which are theorized to allow a relatively large deformation of the arch when the fascia is initially loaded, (Brasile & Hedrick, 1996; Gefen, 2003; Wright & Rennels, 1964). The collagen fibers, however, have a higher modulus of elasticity and are theorized to contribute to the increased tension in the plantar fascia as deformation of the arch continues (Wright & Rennels, 1964). Although in-vitro studies have been successful in measuring the strain in the plantar fascia under different loading conditions (Kitaoka et al., 1994; Sharkey, et al., 1998; Wright & Rennels, 1964), these experimental results, using instrumented mechanical apparatuses to load the foot, are not completely applicable to dynamic gait.

As a follow-up to these in-vitro studies, Gefen (2003) performed an in-vivo study of the plantar fascia during barefoot walking, using a digital radiographic fluoroscopy imaging system. Lateral images of the foot were assessed to evaluate the plantar fascia's transient length during the latter half of walking. The plantar fascia was shown to undergo continuous elongation throughout the stance phase, reaching a deformation of 9-12% between the initial and final positions. Specifically, a rapid elongation during midstance was observed, followed by a slower elongation until toe-off. Furthermore, the plantar fascia in this study demonstrated an increased stiffness during the early stages of weight acceptance, contributing to the overall increase in the stability of the arch during dynamic loading. These results support those of the in-vitro studies described earlier (Sharkey, Ferris, & Donahue, 1998; Wright & Rennels, 1964).

Stiffness of the Plantar Fascia

Changes to stiffness of the plantar fascia are central to the theorized histopathology of plantar fasciitis. How the stiffness of the plantar fascia changes with plantar fasciitis, however, is not well understood. The traditional theory has been that plantar fasciitis is associated with an increase in the stiffness of the plantar fascia (Cardinal et al., 1996). However, this theory is based on plantar fasciitis resulting from chronic inflammation, which leads to inadequate healing of the structure and a corresponding increase in stiffness. With respect to inflammation, in a review of histological analysis of the plantar fascia following fifty cases of heel spur surgery for chronic plantar fasciitis, Lemont et al. (2003) suggested that there is no objective

histologic evidence to support the presence of inflammation in plantar fasciitis (Lemont et al., 2003).

Recent research has provided a counter-argument to that of the chronic inflammation pathoetiology. Several authors have suggested that plantar fasciitis may instead be associated with degeneration of the plantar fascia, and that may result in a reduction in stiffness (Lemont et al., 2003). The recent studies suggest that plantar fasciitis may be primarily the result of degeneration within the structure, and that chronic inflammation is a possible secondary mechanism (Wearing et al., 2006). In support of this argument, Grasel et al. (1999) examined magnetic resonance images of the plantar fascia of patients clinically diagnosed as having plantar fasciitis. Inflammation was ruled out as a cause of the condition because of the linearity and low prevalence of signal intensity within the fascia. Instead, the study concluded that the changes within the plantar fascia were perifascial edema due to microtears in the plantar fascia at its origin. More recent studies utilizing ultrasound to image the plantar fascia in patients with plantar fasciitis have also shown evidence of degeneration within the plantar fascia. Karabay, Toros, and Hurel (2007) used ultrasonographic imaging to evaluate 23 cases of plantar fasciitis. The images depicted a thickening at the proximal portion of the fascia, hypoechoic changes, and presence of perifascial fluid. Plantar fascia thickness has been reported to be 4 mm or larger in individuals with plantar fasciitis, compared to 2-2.5 mm in asymptomatic individuals (Cardinal et al., 1996). Specifically, this increased thickness and hypoechoic appearance are likely related to the underlying fiber degeneration process of microtears. The repetitive movement and constant loading that occurs particularly during running have been theorized to contribute to the progression of degeneration. The

fascia may be overloaded and overused during running, leading to subsequent degenerative changes in the connective tissues (Cardinal et al., 1996; Karabay et al., 2007; Ribeiro et al., 2011).

With respect to changes in plantar fascia stiffness associated with plantar fasciitis, a recent study by Wu et al. (2011) that used elastography to image the plantar fascia demonstrated a loss of stiffness in the plantar fascia in patients with plantar fasciitis Specifically, the authors evaluated 13 individuals with plantar fasciitis and 40 healthy individuals who were divided into young (18-50 years) and old (> 50 years). Stiffness was indicated by the intensity of the various color components (red, green and blue) on the sonoelastogram. Their results demonstrated a significantly greater intensity of blue and green colors in the plantar fascia of those with plantar fasciitis and of the healthy older adults. The combination of, and increase in these colors, is indicative of a softening, or loss of stiffness, of the plantar fascia. Furthermore, the similarity of the color scheme between the older healthy adults and the individuals with plantar fasciitis is a demonstration of the age-related changes that occur in the structure.

The recent evidence of degeneration and decreased stiffness within the plantar fascia in patients with plantar fasciitis is contrary to the traditional theory of chronic inflammation and an increase in stiffness of the structure (Cardinal et al., 1996; Karabay et al., 2007; Wearing et al., 2007; Wearing et al., 2004). This shift in theory may be very important to developing effective treatment programs because treatment for degenerative, more mobile structures may be different than for an inflamed and stiff structure.

Pathoetiology

In order to develop effective programs and protocols to more effectively prevent and treat plantar fasciitis, respectively, an understanding of the pathoetiological factors associated with the histopathology is critical. While most authors agree that the cause of plantar fasciitis is multifactorial, consisting of a combination of anatomical, biomechanical, and environmental factors, the roles of the different factors are not well understood (Arangio, Chen, & Salathe, 1998; Wearing et al., 2006). This Review of Literature will address the anatomical and biomechanical factors associated with plantar fasciitis. While environmental factors such as footwear and training errors may be important contributing factors to plantar fasciitis, they are beyond the scope of this project and therefore will not be reviewed.

Anatomical Factors

Foot Structure

Although foot structure is theorized to influence loading on the plantar fascia, results of experimental studies investigating the role of foot structure in the development of plantar fasciitis have been equivocal (Arangio et al., 1998; Pohl et al., 2009; Wearing et al., 2006). While most of the literature has focused on the association between low arches and plantar fasciitis, some authors have reported high-arch foot structures as a contributing factor to plantar fasciitis. Low-arched foot structures have been suggested to increase tensile load within the plantar fascia, thereby increasing the risk of microdamage and subsequent development of plantar fasciitis (Huang et al., 2004; Kwong et al., 1998; Rome et al., 2001; Pohl et al., 2009; Taunton et al., 1982). Conversely, high arches are

believed to be associated with decreased mobility of the foot, which may increase the stress on the plantar fascia due to poor shock absorption, thereby increasing the risk of microdamage and subsequent development of plantar fasciitis (Williams et al., 2001). As previously stated, however, results of experimental studies have been inconsistent. For example, in a retrospective study of high and low arched type runners with various lower extremity injuries, Williams et al. (2001) reported that of the 13 runners with plantar fasciitis eight had high arches and 5 had low arches. Arch height in the study was quantified using the arch ratio (the ratio of the height of the dorsum of the foot at 50% foot length to the truncated foot length). Similarly, in a retrospective study on the factors associated with the development of plantar fasciitis in athletes, Rome et al. (2001) used calipers to measure navicular height during standing. Results of the study did not reveal significant differences in arch shape of patients with and without plantar heel pain. A year later, another retrospective study of running related injuries byTaunton, Ryan, Clement, McKenzie, Lloyd-Smith, et al. (2002) reported that only 30 of the 159 patients with plantar fasciitis (19%) had either high or low visually assessed arch structure.

Conversely, other studies have shown that a lowered arch is more frequently associated with individuals who have plantar fasciitis (Prichasuk, 1994; Shama, Kominsky, & Lemont, 1983; Wearing et al., 2007). In a retrospective study, (Shama et al., 1983) reported that 81% of 52 patients with heel pain showed radiographic evidence of foot pronation. Similarly, in another retrospective study, Prichasuk (1994) observed significantly lower calcaneal pitch, assessed via a radiograph in 82 patients with heel pain compared to a non-injured group. The study concluded that pes planus, or lowered arches, was an important factor in the development of plantar fasciitis. Finally, (Wearing

et al., 2007) analyzed arch shape and plantar fascia thickness of patients with and without plantar fasciitis using weight-bearing radiographic during quiet bi-pedal stance and sonography, respectively. The authors found that arch shape was significantly correlated with the sonographic images of fascial thickness, accounting for approximately 80% of the variance in the sagittal thickness of the symptomatic fascia.

One major factor in the inconsistency between studies may be the way in which arch structure has been assessed. Authors in the previous studies have used subjective visual assessment (Taunton et al., 2002), navicular height (Pohl et al., 2009; Rome et al., 2001), and radiographs (Prichasuk, 1994; Shama et al., 1983; Wearing et al., 2007) to quantify foot structure. One source of inconsistency may be that the varying methods used to quantify foot structure assessed different aspects of foot structure. If so, the differing aspects of foot structure may have differing effects on foot function. Another source of inconsistency may be the reliability and/or validity of the methods used to compute foot structure. The inter-tester reliability of visual observation utilized to classify foot posture has been reported as poor (Cowan, Robinson, Jones, Polly, & Berrey, 1994). Additionally, many of the other measures have good intra-tester reliability, but poor inter-tester reliability (Rome et al., 2001; Taunton et al., 2002). A third factor that may contribute to the inconsistency in the results is that static measurements of arch height, which most of the research studies have incorporated, may not be related to dynamic movement (Sahin, Ozturk, & Atici, 2010). Likewise, the results of the dynamic movement of the arch when studied using various surface-marker based techniques have also been equivocal (Messier & Pittala, 1988; Pohl et al., 2009; Warren, 1984; Warren & Jones, 1987); however, the methodical differences between the studies

limit the ability to directly compare the results. Continued research investigating the dynamic movement of the arch is necessary in order to further understand the effect of foot structure on foot function. Finally, a fourth potential factor in the inconsistency of the results is whether the study was retrospective or prospective. The retrospective studies have primarily associated low arch foot structure with plantar fasciitis (Pohl et al., 2009; Prichasuk, 1994; Shama et al., 1983; Wearing et al., 2007), so it is possible that the increased mobility of the plantar fascia in plantar fasciitis caused the lowering of the arch (Wearing et al., 2007).

Leg Length Discrepancy

Leg length discrepancy is often cited as a contributing factor to plantar fasciitis because it is theorized to cause increased pronation during gait, and therefore increased tension on the plantar fascia (Glazer & Hosey, 2004; Krivickas, 1997; Messier & Pittala, 1988). In a retrospective analysis of factors associated with plantar fasciitis, a leg length discrepancy of 0.63 cm was found in 53% of the plantar fasciitis group compared to only 21% of the control group (Messier & Pittala, 1988). Although leg length differences may be a contributing factor with plantar fasciitis, a limitation to these studies is the lack of methodical information regarding the way the authors measured the leg lengths. Furthermore, although radiographs are the gold standard to assess leg length discrepancy, they are not feasible in many cases due to radiation exposure and cost. Moreover, relevance of the clinical methods is limited due to lack of reliability (Glazer & Hosey, 2004; Krivickas, 1997; Messier & Pittala, 1988).

Ankle Range of Motion

Limited ankle dorsiflexion has been suggested to contribute to the development of plantar fasciitis. Specifically, tight gastrocnemius and soleus muscles may contribute to decreased motion in the ankle during gait, thereby placing additional stress on the plantar fascia (Labovitz et al., 2011). Limited ankle dorsiflexion during gait may force the forefoot to compensate with dorsiflexion and pronation, placing repetitive longitudinal stress on the plantar fascia (Riddle et al., 2003). Many studies investigating risk factors associated with plantar fasciitis have reported a strong relationship between decreased ankle dorsiflexion mobility and plantar fasciitis (Cornwall & McPoil, 1999; Kibler et al., 1991; Labovitz et al., 2011; Riddle et al., 2003). The studies have shown that individuals with less than 10° of ankle dorsiflexion may be at a greater risk for developing plantar fasciitis compared to those with normal passive dorsiflexion range of motion of the ankle. In a retrospective study on the functional biomechanical deficits in runners with plantar fasciitis, Kibler et al. (1991) recruited 43 competitive or recreational athletes who were clinically diagnosed with unilateral plantar fasciitis. The principle athletic activity for these participants was running long distances or jogging. The researchers compared the asymptomatic limb with the symptomatic limb, reporting that the majority of runners with plantar fasciitis showed a lack of passive dorsiflexion with the knee extended in the symptomatic limb versus the asymptomatic limb. This deficiency was hypothesized to cause excessive pronation during gait that ultimately contributed to development of plantar fasciitis (Kibler et al., 1991). Over a decade later, Riddle et al. (2003) conducted a matched case-control study to compare fifty patients with a clinical diagnosis of unilateral plantar fasciitis. Passive ankle dorsiflexion range of motion was measured with

the knee extended while the participants were lying in the prone position. Nearly half of the participants with plantar fasciitis had less than 5° of motion, while the majority of the participants with plantar fasciitis had less than 10° of ankle range of motion (Riddle et al., 2003). The authors suggested that the risk of plantar fasciitis increased as the range of ankle dorsiflexion decreased. In a recent retrospective study, Labovitz et al. (2011) compared passive ankle range of motion between a group of participants with plantar fasciitis and a control group. The authors looked at the entire range of motion, at the ankle, with the knee both extended and flexed. They reported a significant difference in the measurements between the groups. Over 96% of the plantar fasciitis group participants were deficient in ankle range of motion (Labovitz et al., 2011).

Conversely, in a retrospective study on biomechanical factors associated with female runners with a history of plantar fasciitis, Pohl et al. (2009) demonstrated a significant increase in ankle dorsiflexion in the plantar fasciitis group. The authors suggested the reason for the differing results may be that the runners in the current study had a history of plantar fasciitis, but were pain-free at the time of data collection and had been undergoing physical therapy. Since increasing range of motion at the ankle is one of the most common therapeutic exercises for plantar fasciitis patients, the authors theorized that the results likely reflected the increase in range of motion that occurred as the result of receiving treatment (Pohl et al., 2009).

Based on the results of the current literature on ankle range of motion, it is suggested that at least four to 10° of dorsiflexion is required for the stance phase of a normal walking gait pattern. An angle that is less than 10° constitutes equines and is theorized to result in subtalar joint compensation during weightbearing, thus leading to

abnormal pronation and increased plantar fascia stress during gait (Cornwall & McPoil, 1999). However, a limitation to these studies is the measurement of passive range of motion since it many not directly translate to dynamic differences during gait. Although most studies agree that there is an association between passive ankle dorsiflexion range of motion and plantar fasciitis, the relation to dynamic movement, particularly running, is unclear. Moreover, another limitation of these studies is comparing the ankle dorsiflexion range of motion of the unaffected limb to the affected limb in participants with unilateral plantar fasciitis. There may be compensatory changes in the asymptomatic foot that may result in decreased range of motion.

First Metatarsophalangeal Joint Range of Motion

In addition to range of motion of the ankle joint, range of motion of the first metatarsophalangeal joint has also been investigated as a potential contributing factor to the development of plantar fasciitis. As stated previously, extension of the toes and in particular the first metatarsophalangeal joint is important to supporting the arch during gait through initiation of the windlass mechanism (Allen & Gross, 2003). Several authors have indicated that reduced passive first metatarsophalangeal joint extension range of motion is associated with patients with plantar fasciitis patients (Allen & Gross, 2003; Creighton & Olson, 1987). A retrospective study by Creighton and Olson (1987) compared first-toe metatarsophalangeal joint flexion and extension range of motion in runners with and without plantar fasciitis. Results of the study indicated that runners with plantar fasciitis had a significant decrease in active and passive extension and passive flexion of the first-toe metatarsophalangeal joint. Contrary to this finding, a retrospective

study by Allen and Gross (2003) did not find a significant difference in first-toe

metatarsophalangeal joint between the unilateral plantar fasciitis group and the control

group. More recently, Labovitz et al. (2011) reported no significant difference in first

metatarsophalangeal joint extension range of motion in a group of plantar fasciitis

participants compared to a control group.

Despite the inconsistency in these results for individuals with plantar fasciitis, the

traditional theory is that a decrease in passive extension of the first metatarsophalangeal

joint range of motion is associated with a corresponding increase in stiffness of the

plantar fascia. However, recent sonography images of the plantar fascia suggest that there

is instead a softening of the plantar fascia rather than an increase in its stiffness (Wu et

al., 2011). Specifically, toe extension causes the plantar fascia to be wound around the

metatarsal heads, thereby shortening its effective length and increasing tension within the

fascia (Hicks, 1954). The function of this increased tension is to provide stability for the

arch as the foot re-supinates and prepares for toe-off (Taunton et al., 1982). The

activation of the windlass mechanism, however, only occurs when there is sufficient

tension within the fascia (Hicks, 1954). Therefore, based on the recent discovery of a

decrease in stiffness in the plantar fascia in individuals with plantar fasciitis Wu et al.

(2011), it is likely that the windlass mechanism might not be sufficiently activated. Gait

analysis that assessed toe extension range of motion may provide further insight to the

effect of plantar fasciitis on first metatarsophalangeal joint function.

Most authors agree that the cause of plantar fasciitis is multifactorial and consists

of a combination of anatomical, biomechanical, and environmental factors; however, the

role of these different factors is not well understood (Arangio et al., 1998; Wearing et al.,

2006). A limitation of most studies is that they are retrospective, so the causality of plantar fasciitis cannot be determined. Moreover, the anatomical and biomechanical factors discussed have largely been static measurements that may not be directly applicable to function. Although these factors provide insight into the development of plantar fasciitis, a dynamic assessment may be necessary to fully understand their contribution to gait. Furthermore, this knowledge may contribute to development of more effective treatment protocols for plantar fasciitis.

Biomechanical Factors

Extensive research has been done on the static measurements of range of motion and arch structure, two variables theorized to be associated with plantar fasciitis. However, there has been limited research on the mechanical effect of plantar fasciitis during gait, and how these static measurements are associated with dynamic function. In order to better understand the relevance of mechanical changes associated with plantar fasciitis, normal function of the foot during gait will be briefly reviewed. During the stance phase of the gait cycle, the foot the normal undergoes a supination-pronation-supination movement cycle. At initial contact, the foot is in a supinated position to provide a stable base of support as the foot makes contact with the ground. This is followed by a period of pronation during the mid-portion of the stance phase. Pronation transitions the foot from a relatively rigid structure to a more mobile structure that functions to contribute to shock absorption and allow the foot to adapt to uneven terrain. Following this mid-portion of stance, the foot supinates again to provide a relatively rigid lever for push-off (Perry, 1992).

Although there is good general agreement that the plantar fascia plays an important role of supporting the medial longitudinal arch throughout the stance phase of gait, the role has largely been based on theory Wearing et al. (2004) and inferred from in-vitro (Erdemir et al., 2004; Huang et al., 1993; Wright & Rennels, 1964) and in-vivo static loading studies (Kim & Voloshin, 1995; Wright & Rennels, 1964), and simulation studies (Arangio et al., 1998; E. P. Salathe & Arangio, 2002). Only a limited number of studies have investigated the mechanical effects of plantar fasciitis on gait (Messier & Pittala, 1988; Pohl et al., 2009; Taunton et al., 2002; Wearing et al., 2004).

Of these studies, many have involved imaging to describe measures of arch movement. These studies have indicated that the plantar fascia undergoes a length change of approximately 6 mm through the stance phase of gait (Kayano, 1986; Wearing et al., 2006). Other studies employing surface mounted goniometry have indicated that the arch alternates through periods of elongation and shortening during stance phase (Yang et al., 1985). Collectively, the studies have reported that the plantar fascia initially lengthens with heel contact, shortens throughout midstance, lengthens at heel lift, and then rapidly shortens during late terminal stance as the arch is raised to prepare the foot for propulsion (Kayano et al., 1986; Yang et al., 1985). Nearly 20 years later, Wearing et al. (2004) measured the plantar fascia directly in-vivo with digital fluoroscopy, comparing the sagittal movement of the medial longitudinal arch in participants with plantar fasciitis and a control group. The authors did not find a significant change in arch movement associated with chronic plantar fasciitis, but they concluded instead that the arch mechanics may influence the severity of plantar fasciitis, once the condition is present (Wearing et al., 2004). In addition to dynamic arch mechanics, Wearing et al. (2004) also

investigated first metatarsophalangeal joint motion using lateral radiographs of the foot during walking. They reported that patients with plantar fasciitis had a greater peak metatarsophalangeal joint angle during stance compared to asymptomatic participants. Wearing et al. (2004) also assessed fascial thickness with sonographic imaging, and compared the measurements between groups. They found that the peak metatarsophalangeal joint angle significantly correlated with an increase in fascial thickness in only the symptomatic foot of the plantar fasciitis group participants (Wearing et al., 2004). These results are consistent with the idea that a loss of stiffness in the plantar fascia is associated with plantar fasciitis. As previously stated, recent studies using sonoelastography have shown a decrease in stiffness of the plantar fascia that is associated with plantar fasciitis (Wu et al., 2011), while others reported degeneration and adaptive thickening using ultrasound imaging (Cardinal et al., 1996; Lemont et al., 2003; Wearing et al., 2006). Together, these result in a loss of integrity of the structure, ultimately affecting motion at the first metatarsophalangeal joint. These factors may all contribute to change in joint angle that Wearing et al. (2004) reported. Although the above studies have improved understanding of the dynamic movement of the plantar fascia throughout the stance phase, they are not clinically feasible to conduct due to cost and radiation exposure.

Surface-based two-and three-dimensional motion analysis studies have more recently investigated the motion of the plantar fascia during gait, with contrasting results to those of the earlier studies. The surface-marker based studies found the arch to elongate until approximately 75-80% of the stance phase, followed by shortening until toe-off (Cashmere, Smith, & Hunt, 1999; Chang et al., 2007; Hunt, Smith, & Torode,

2001). The technique employed during these studies likely accounts for the vast differences in results. While the more recent two and three dimensional surface-based marker studies are affected by some skin movement error, the surface mounted goniometry used by Wearing et al. (2006) may have been prone to larger error due to its greater mass. Warren and Jones (1987) performed a two-dimensional gait analysis to compare running kinematics in runners with plantar fasciitis, runners who were currently pain-free but had a history of plantar fasciitis, and non-injured runners with no history of plantar fasciitis. Each subject was filmed in both a barefoot and running shoe condition while running on a treadmill. The film was used to record the runners' footstrike type and to measure the calcaneal pronation. The authors found that runners with plantar fasciitis pronated more than non-injured runners; however, they had only moderate success in correctly classifying subjects into their proper groups. Although an improvement from the static studies, this study is limited due to the projection errors associated with two-dimensional gait assessment. A year later, Messier and Pittala (1988) compared rearfoot kinematics of competitive and recreational runners who were not injured with runners who had plantar fasciitis. Subjects ran on a treadmill for five minutes in their normal training shoes, while two-dimensional data was collected from contrasting markers that were placed on the subject's legs and heel counters. The results were in agreement to those found by Warren and Jones (1987) in that plantar fasciitis is associated with excessive rearfoot movement.

Contrary to previous findings on plantar fasciitis and rearfoot pronation, a more recent retrospective study by Pohl et al. (2009) did not reveal differences in rearfoot motion between a healthy group and a plantar fasciitis group. Pohl et al. (2009)

investigated rearfoot kinematics during the stance phase of walking gait in 20 female

runners with plantar fasciitis using a surface marker based rearfoot model. The

participants ran overground while kinematic data was recorded using a three-dimensional

motion capture system and force plate. The authors did not find significant differences in

peak dorsiflexion, peak eversion, time to peak eversion, and the eversion excursion,

between the plantar fasciitis and control groups. While the three-dimensional analysis is

an improvement over the previous two-dimensional studies, there are still several

limitations associated with the foot model used. They only tracked rearfoot motion,

potentially masking kinematic differences within the midfoot. The potential importance

of midfoot motion to foot function has been reported by Arndt et al. (2009); Cobb et al.

(2009) who investigated multi-segment foot kinematics during jogging and walking,

respectively. Arndt et al. (2007) demonstrated increased midfoot mobility with in vivo

running kinematics through an invasive in-vivo kinematic study on four male

participants. Bone pins were inserted in nine foot segments and segment motion was

tracked during several jogging trials. Significant motion in the talonavicular joint was

observed in all three planes of motion (Arndt et al., 2007). Cobb et al. (2009) showed a

significant difference in motion distal to the calcaneus between typical and low arch

participants during walking. The authors investigated stance phase kinematics between

participants with low-mobile versus typical foot postures using a multi-segment medial

foot model. Four functional articulations were tracked, with significant differences

observed in the calcaneonavicular complex abduction excursion during midstance

between the two groups (Cobb et al., 2009). The results of these two studies demonstrate

important differences in midfoot motion that cannot be ignored. To further emphasize the

importance of investigating midfoot function, Pohl et al. (2009) suggested that plantar fasciitis may be more related to midfoot pronation rather than rearfoot pronation. Increased forefoot pronation has previously been associated with plantar fasciitis in walking (Chang et al., 2007), further indicating the importance of tracking foot motion distal to the calcaneus. Analysis of midfoot and forefoot motion during running using a multi-segment foot model may inform researchers of potential associations of foot motion in runners with plantar fasciitis compared to injury-free runners.

Another important consideration in evaluating the result of Pohl et al. (2009) is the fact that the variables were computed over the entire stance phase versus examining kinematics within subphases of stance. Based on previously developed theories of foot function during gait (Brasile & Hedrick, 1996; Hicks, 1955) and previous in-vitro and in-vivo studies (Hicks, 1954; Hicks, 1955; Vedi et al., 1999; Williams et al., 1999), assessing the stance phase of gait as a single phase may potentially mask differences that may occur during the stance subphases. As previously mentioned, several authors have demonstrated that the foot functions differently throughout the stance phase. During flat standing, the medial longitudinal arch functions similar to both a beam and truss, but primarily a truss during late stance and toe off (Hicks, 1955). Moreover, in-vivo models using magnetic resonance imaging revealed that the plantar fascia is relaxed until heel lift, when digital extension initiated the windlass mechanism (Vedi et al., 1999; Williams et al., 1999).

The results from the invasive in-vivo and in-vitro studies, in addition to the gait analysis studies, suggest that runners with plantar fasciitis may exhibit altered kinematics in the distal segments of the foot during mid and late stance phase. However, the effect of

plantar fasciitis during running with a surface-marker based multi-segment foot model has not yet been examined. In order to develop and implement training programs that effectively reduce runner's risk of developing plantar fasciitis it is important to first understand the mechanical changes during gait associated with plantar fasciitis.

Multi-segment Foot Model

Recently, a number of multi-segment foot models have been developed in order to characterize foot kinematics. The multi-segment models began as single segment and rearfoot complex models, but have since progressed to models that include up to six segments (Rankine, Long, Canseco, & Harris, 2008). The single segment models treat the foot as a single rigid segment, an over-simplification of the foot. The first marker-based rearfoot segment models compared the motion of the calcaneus and the tibia, using a variety of optoelectronic capture systems (Kepple, Stanhope, Lohmann, & Roman, 1990; Moseley, Smith, Hunt, & Gant, 1996). These were also an over-simplification. Both models ignore the distal segments of the foot, which have recently shown significant motion to occur (Cobb et al., 2009; Lundgren et al., 2008; Nester et al., 2007).

Some of the first multi-segment foot models were two- foot segment models that defined a forefoot segment in addition to the traditionally defined hindfoot and tibia segments (Davis, Zifchock, & Deleo, 2008; Hunt et al., 2001; Kitaoka et al., 2006). These models had the ability to analyze the distal motion of the foot, which the previous rearfoot complex models ignored; however, there was still considerable disagreement in the results among the models. Moreover, there were differences in the methods between foot models, including marker position and number of markers used, particularly with the

number of metatarsal markers. Each model was also used on different clinical populations, so it is difficult to compare results among the studies. Hunt et al. (2001) employed a two foot-segment model (leg, rearfoot, and forefoot) to look at the change in height of the medial longitudinal arch during the stance phase of walking in twelve male participants. These authors noted that the frontal, transverse, and sagittal planes of motion in the forefoot segment were in agreement with previously reported forefoot motion from bone pin studies. This finding was important in that it established the concurrent validity of marker based multi-segment foot models as well as confirmed the importance of the joints distal to the calcaneus.

In addition to the previously mentioned two foot segment models, several three foot segment models that define tibia/fibula, hindfoot, forefoot, and hallux segments have also been developed (Carson, Harrington, Thompson, O'Connor, & Theologis, 2001; Cornwall & McPoil, 1999, 2002; Kidder et al., 1996; Myers, Wang, Marks, & Harris, 2004). More recently, Jenkyn, Anas, and Nichol (2009) developed a four foot-segment foot model to analyze the foot and ankle complex in walking. Their model subdivided the foot into hindfoot, midfoot, medial forefoot, and lateral forefoot segments. The study reported that hindfoot and forefoot pronation in the frontal plane coincided with the dropping of the medial longitudinal arch during midstance, and arch raising in the late stance and swing phase (Jenkyn et al., 2009). This model is unique because of its separation of medial and lateral forefoot segments, defining them as two separate segments rather than a rigid segment. Recently, Wolf et al. (2008) suggested that this separation of the medial and lateral forefoot is important due to the segments acting independently of one another. Recently, Cobb et al. (2009) developed a four foot model

that defined rearfoot complex, calcaneonavicular complex, medial forefoot, and the first metatarsophalangeal complex functional articulations to compare different foot structures during walking. The study reported differences in motion in the segments distal to the calcaneus between participants with differing foot structure, further signifying the importance of tracking the motion of the foot beyond the rearfoot. Similarly, Arndt et al. (2007) demonstrated significant increased midfoot mobility, but with in-vivo running kinematics. The use of multi-segment models enables researchers to identify differences between foot structures and pathologies, the medial and lateral forefoot segments, and the motion occurring in the midfoot. All of these components may be important to analyze in future models.

Collectively, the many multi-segment foot models have led to the development of a six-segment multi-segment model that is used in the current study. It expands upon these current multi-segment foot models by including medial and lateral midfoot segments in addition to medial and lateral forefoot segments (Bauer, Joshi, Klinkner, & Cobb, 2011; Cobb, James, Hjertstedt, & Kruk, 2011). The application of a multi-segment foot model to plantar fasciitis may improve understanding of the mechanical effect of plantar fasciitis on gait.

Diagnosis

Clinical Diagnosis

A contributing factor in the inconsistent results between previous studies investigating the effect of plantar fasciitis on gait mechanics is the lack of a gold standard

diagnostic test for plantar fasciitis. Plantar fasciitis is typically a clinical diagnosis based on the presence of signs and symptoms characteristic of the injury. Pain at the heel, especially during the first few steps in the morning or after being inactive for an extended period of time are the most common symptoms used to diagnose plantar fasciitis (Alshami, Babri, Souvlis, & Coppieters, 2007; Cole et al., 2005; Karabay et al., 2007; Wearing et al., 2006). Another common symptom reported by athletes, especially runners, is an initial decrease in heel pain early with activity followed by a return of symptoms at the end of prolonged activity (Karabay et al., 2007).

Diagnostic Imaging

While the presence of plantar fasciitis can be determined using the traditional clinical method of diagnosis for plantar fasciitis, it is difficult to differentiate other potential causes of heel pain. Heel pain may be caused from numerous conditions in addition to plantar fasciitis, including tarsal tunnel syndrome, entrapment of plantar nerves of the foot, calcaneal fracture, rupture of the plantar fascia, and atrophy of the heel fat pad (Alshami et al., 2007). Definitive differential diagnosis often requires some form of diagnostic imaging in addition to the clinical examination. Plain radiographs can rule out calcaneal stress fracture (Roxas, 2005) or show calcaneal spurring and calcifications within the plantar soft tissue (Cosca & Navazio, 2007). However, not all cases of plantar fasciitis are associated with the development of calcaneal heel spurs. In fact, $15 - 25\%$ of asymptomatic individuals have been reported to show radiographic evidence of heel spurs, while many patients with plantar fasciitis do not reveal presence of heel spurs.

Additionally, the cost and radiation exposure limit the clinical usefulness of radiographs, and they are not able to assess soft tissues (Young et al., 2001).

More recently, researchers and clinicians have begun using ultrasound to investigate the mechanical properties of the plantar fascia. Studies of the uninjured persons have revealed the plantar fascia to be hyperechoic with a striated appearance resulting from the longitudinal fibers orientation (Cardinal et al., 1996). Studies using ultrasound to image the plantar fascia of patients with plantar fasciitis have revealed adaptive thickening at the origin, a hypoechoic appearance resulting from tissue degeneration, and presence of fluid collection (Cardinal et al., 1996; Karabay et al., 2007; Sahin et al., 2010; Wearing et al., 2004; Wu et al., 2011). Specifically, Karabay et al. (2007) evaluated 23 cases with plantar fasciitis, using ultrasonographic imaging to analyze the structure. Their images depicted a thickening at the proximal portion of the fascia, hypoechoic changes, and presence of perifascial fluid. Thickening in plantar fasciitis patients have been reported to be 4 mm or larger, compared to 2-2.5 mm in asymptomatic persons (Cardinal et al., 1996). This thickening and hypoechoic appearance are theorized to occur from the repetitive movement and constant loading during weightbearing activities, including running. During running, the plantar fascia may be aggravated from the overload and overuse, leading to subsequent degenerative changes in the connective tissues (Karabay et al., 2007; Ribeiro et al., 2011).

In addition studying the changes in tissue thickness and appearance, Wu et al. (2011) used elastography to evaluate the stiffness of the plantar fascia in a group of both healthy young and old adults, as well as individuals with plantar fasciitis. The results indicated an associated decrease in stiffness in healthy older adults and individuals with

plantar fasciitis (Wu et al., 2011). As mentioned previously, these findings are contrary to the traditional thought of an increase in stiffness associated with plantar fasciitis, but may explain some of the inconsistencies between the theorized mechanical effects of plantar fasciitis on gait and the results of experimental studies. This softening of the plantar fascia may weaken the structure by reducing the loading capacity and its ability to maintain the arch's rigidity. This loss of stability may further lead to mechanical changes during the stance phase of gait.

These studies suggest that when used in conjunction with the traditional clinical exam, ultrasound imaging may be a useful diagnostic tool for definitively diagnosing plantar fasciitis. Moreover, the fact that ultrasound is noninvasive, relatively inexpensive, and does not involve radiation exposure, suggests that sonography may also be a useful imaging technique for assessing treatment programs effectiveness (Cardinal et al., 1996)

Conclusions

Most researchers agree that plantar fasciitis is multifactorial pathology with a mechanical overload component. It is a degenerative condition, affecting 10% of all runners, and lasting for 6-18 months. It is also a large public health problem and debilitating condition because of the importance the plantar fascia plays during gait. The plantar fascia functions to maintain the medial longitudinal arch and to aid in re-supination of the foot during the late midstance and propulsive stages of gait. Biomechanical analyses have shown an increase in tension of the plantar fascia as the arch elongates during midstance, which is theorized to increase tension within the plantar fascia.

Researchers have identified various risk factors for developing plantar fasciitis; however, results have been equivocal. Several anatomical factors, including foot structure, leg length, ankle range of motion, and first metatarsophalangeal joint range of motion have been associated with plantar fasciitis, but the results have been inconsistent. Moreover, biomechanical factors including foot pronation during gait have also been identified as risk factors.

A limitation of previous research investigating the mechanical changes associated with plantar fasciitis may be modeling the foot as a single rigid segment. Recent studies using multi-segment foot models have reported significant midfoot and forefoot motion during gait. To date no study has investigated running kinematics in patients with plantar fasciitis using a six segment foot model. Further analysis of this motion during running can enhance understanding of plantar fasciitis.

Plantar fasciitis is most commonly a clinical diagnosis, but clinicians and researchers have started using ultrasound as a diagnostic tool. Ultrasound imaging could help definitively diagnose plantar fasciitis in conjunction with a clinical exam, and lead to improved prevention strategies and treatment programs for these patients.

CHAPTER 3: METHODS

Participants

Forty-nine participants went through an initial phone screening. Of these, sixteen did not qualify based on the initial screening, one did not qualify based on the physical exam, one was unable to complete the gait trials due to a pain level of eight, and one participant had data that was not unable to be tracked. Of the forty-nine, thirty participants (15 plantar fasciitis and 15 control) were fully collected and analyzed. Fifteen runners with plantar fasciitis and fifteen age, gender, and mileage matched-controls were recruited for this study (Table 1). Runners were chosen because of the prevalence of plantar fasciitis among this population. Specifically, plantar fasciitis is the third most frequently diagnosed running injury (Taunton, Ryan, Clement, McKenzie, & Lloyd-Smith, 2002), affecting 10% of all runners (Baxter, 1994).

Table 1. Mean (SD) of demographic information for plantar fasciitis and control group participants

Variable	Plantar Faciitis	Control
Gender	m = 8, f = 7	m = 8, f = 7
Age, years	30.00 (8.74)	29.27 (6.44)
Height, cm	170.60 (8.25)	170.52 (7.78)
Mass kg	67.98 (8.20)	68.07 (9.99)

Previous research reported a large effect size for maximum first metatarsophalangeal joint angle during gait between a plantar fasciitis and control group (Effect size = 1.1) (Wearing et al., 2004). Based on these previous data, to achieve a

power of 0.8 with α = 0.05, 10 subjects per group were required to compare the metatarsophalangeal joint angle. In addition, previous research from an ongoing footwear study in the Musculoskeletal Injury Biomechanics Laboratory at UW-Milwaukee, using the same kinematic variables as the proposed study, reported a range of small to large effects sizes between footwear conditions. Based on these data, to achieve a power of 0.8 with α = 0.05, 7-39 subjects were needed per group to compare kinematic variables. Therefore, based on previous research, and sufficient power to detect a moderate effect size (Effect size = 0.25) in the kinematic variables between participants with plantar fasciitis and a control group, 15 subjects per group were needed to achieve a power of 0.8 with α = 0.05 (Wearing et al., 2004). Participants were recruited: (1) from the University of Wisconsin-Milwaukee community via announcements in the College of Health Sciences classrooms; and (2) from the posting and emailing of flyers both on campus and to appropriate businesses and organizations in the community.

Inclusionary/Exclusionary Criteria

Plantar Fasciitis Group

To be eligible for participation in the plantar fasciitis group, runners were required to meet the following criteria: have the presence of the common clinical signs and symptoms used to diagnose plantar fasciitis for a minimum of six weeks (Roos et al., 2006; Wearing et al., 2004); be 18-45 years old; be habitually running at least10 miles per week at the time of the study (Messier & Pittala, 1988); and have a body mass index (BMI) of less than 30 kg/m^2. The BMI criteria was selected due to obesity, classified as a

BMI of 30 kg/m^2 or greater, having been established as a risk factor for plantar fasciitis in previous studies (Buchbinder, 2004; Riddle et al., 2003). Of the fifteen participants tested, the group consisted of 12 left and 3 right feet. If plantar fasciitis was bilateral, then the most severe limb was tested.

Participants were not required to have a diagnosis of plantar fasciitis from an allied health professional. Rather, the common clinical signs and symptoms utilized by allied health professionals to diagnose plantar fasciitis (Allen & Gross, 2003; Filippou et al., 2004; Karabay et al., 2007) were used as inclusionary criteria for participation in the study. Specific inclusionary criteria included a complaint of tenderness to palpation of the medial calcaneal tubercle and the medial aspect of the proximal portion of the plantar fascia, or pain along the plantar fascia as it courses under the arch toward the metatarsal heads (Cornwall & McPoil, 1999; Karabay et al., 2007). Additionally, the participants must have reported that the pain from the plantar fasciitis: was present upon weightbearing immediately following prolonged periods of inactivity (Allen & Gross, 2003; Karabay et al., 2007); and gradually decreased throughout the day with ordinary walking; and worsened with prolonged activity (Cardinal et al., 1996). Furthermore, participants were required to have the signs and symptoms for a minimum of six weeks. Based on previous studies, the six-week criteria was determined to be sufficient time for degenerative changes in the plantar fascia to occur and to avoid recruiting patients with plantar fasciitis that were potentially in the acute inflammatory response stage of tissue healing (Wearing et al., 2004).

The upper age range limit was selected due to results of a study by Wu et al. (2011) that showed similar changes in the plantar fascia stiffness assessed with

ultrasound imaging in healthy older adults ages 50 and older and a group of younger patients with plantar fasciitis. Imaging of the older adults over 50-years old showed a softening of the plantar fascia that was similar to that seen in individuals with plantar fasciitis (Wu et al., 2011). To avoid the presence of age-related changes that would potentially mask differences between healthy individuals and runners with plantar fasciitis, individuals older than 45-years old were excluded. Finally, the minimum mileage criteria of 10-miles per week, is consistent with previous studies looking at runners with plantar fasciitis (Messier & Pittala, 1988).

In addition to the inclusionary criteria, the exclusionary criteria were based on patients' previous injury and general medical history. Specific exclusionary criterion included current injuries other than plantar fasciitis, pregnancy, and a history of: lower extremity surgery on the injured side; inflammatory or connective tissue disease such as osteoarthritis, rheumatoid arthritis, and Marfan Syndrome; a diagnosed foot deformity such as hallux valgus; a neurologic systemic disorder that would predispose an individual to heel pain and/or muscle weakness; and diabetic neuropathy (Wearing et al., 2007). The exclusionary criteria were selected because of potential that they may cause changes in foot function similar to those associated with plantar fasciitis.

Previous or current treatment was not an exclusionary criterion for the study. The rationale for not excluding potential participants based on current/previous treatments was the fact that there is limited evidence supporting the effectiveness of any current treatment modalities for plantar fasciitis (Buchbinder, 2004; Crawford & Thomson, 2003; Irving et al., 2008; Wolgin et al., 1994). Therefore, individuals who were seeking different treatment or therapy for their plantar fasciitis were included if: (1) there was no

change in treatment protocol for six weeks leading up to their testing session; and (2)

they continued to have the clinical signs and symptoms of plantar fasciitis. Although

current treatment was not used as exclusionary criteria, any treatment that the participants

were receiving was documented. The common treatment interventions that the

participants received included plantar fascia stretching, ankle range of motion stretching,

icing, therapeutic ultrasound, massage under the arch and heel, orthotic devices and/or

heel cushions, foot muscle strengthening, night splints, and cortisone shots.

Control Group

The control group runners were age (± 5 years) (Allen & Gross, 2003), gender-

matched with the plantar fasciitis group participants. For mileage matching, runners were

further grouped according to average weekly mileage (10-20, 20-30, and 30+ miles per

week). In addition to the exclusionary criteria for the plantar fasciitis group, the control

group could not have a history of plantar fasciitis or a lower extremity injury within the

previous six months. These criterions were established to eliminate factors that may

induce mechanical dysfunction and mask differences between groups. The limb of the

control group that was tested was side-matched to their matched plantar fasciitis group

participant

Study Protocol

Initial Phone Screening

Participants completed a 10 minute initial phone screening assessment. For

plantar fasciitis group participants, the interview consisted of questions pertaining to the

inclusionary criteria, the symptomatic complaints of their condition, and a brief medical

history (Appendix E). The participant was asked to report the magnitude of their pain,

based on a 0-10 pain scale ("0" was "no pain" and "10" was "worst pain ever"), during the

first few steps of walking after arising in the morning and after prolonged periods of

inactivity (Wearing et al., 2007). The control group participants completed the same

questionnaire, with the exception of the questions pertaining to the current plantar

fasciitis injury.

Visit One

     After qualifying based on the initial phone assessment, the participants reported to

the Musculoskeletal Injury Biomechanics Laboratory (Enderis Hall room 132) for Visit 1.

Visit 1 consisted of a brief physical exam for the plantar fasciitis group participants to

rule out other potential causes of heel pain. The visit also included ultrasound imaging

and foot structure assessment of participants in both groups. This testing session lasted

approximately 45 minutes. Prior to beginning the testing session, all participants were

informed of the study procedures and were asked to read and sign an informed consent

approved by the University's Institutional Review Board.

Physical Exam

     To confirm the presence of plantar fasciitis and rule out other common causes of

heel pain, a Certified Athletic Trainer examined the potential plantar fasciitis group

participants. To confirm the presence of plantar fasciitis, the examiner palpated the

proximal insertion of the plantar fascia, as well as passively extended the first

metatarsophalangeal joint. If a pain was elicited on either test the presence of plantar fasciitis was confirmed. Other potential sources of heel pain that were ruled out through manual muscle testing and palpation of relevant structures in the foot included tarsal tunnel syndrome, calcaneal stress fracture, heel pad syndrome, and tibialis posterior and Achilles tendinopathy (Aldridge, 2004).

Ultrasound Assessment 1

Participants received an ultrasound assessment in the Physical Activity and Health Research Lab, in Enderis Hall, Room 434. Three successive ultrasound images of both feet were captured, following a protocol similar to that reported by Rathleff, Moelgaard, and Olesen (2011). The measurement method has been established as having moderate-high intra- and inter-tester reliability for imaging the plantar fascia. Ultrasound imaging consisted of positioning the patient lying in a prone position with the ankle in a neutral dorsiflexion/plantar flexion position and the toes extended to near end range. The thickness of the proximal attachment of each participant's right and left plantar fascia were imaged using an ultrasound machine equipped with a 4.0 cm wide transducer head and 12 MHz transducer (Vivid-i, General Electric Healthcare; Waukesha, WI) and a scan depth of 2.5 cm. The examiner applied ultrasonic gel to the transducer and to the patient's skin. The foot was placed in a neutral position with the toes extended to apply tension to the plantar fascia (Figure 3).

Figure 3. Set-up for ultrasound imaging.

Imaging of the plantar fascia consisted of real time scanning of longitudinal sonographic

images. To obtain the scans, the ultrasound transducer was positioned approximately 0.05

cm medial to the proximal attachment of the plantar fascia on the calcaneal tubercle.

While in a seated position, the examiner slowly moved the transducer laterally across the

participant's foot until a clear image of the plantar fascia was displayed on the screen

(Figure 4). At this point, the examiner froze and saved the image. The procedure was

repeated until three successful images were collected. A successful image was based on

the clarity and positioning of the images. Specifically, clearly defined borders, and proper

alignment of the calcaneus and the plantar fascia were the criteria needed for each

successful image. Still images were saved in DICOM (Digital Imaging and

Communications in Medicine) format, exported onto DVDs, and post-processed using

AccessPoint Software from Freeland Systems (North Venice, FL). After all of the images

for the participants were collected, they were batch-read in order to increase the examiner

repeatability and reliability. The images were then sent to Dr. Kenneth Lee, a Radiologist

at the University of Wisconsin-Madison for over-reading. If consented, participants were

notified if any incidental findings were found in the images and were encouraged to follow up with their physician.

The thickness of the proximal insertion of the plantar fascia for each image was measured until the measurements were within 0.5 mm of each other (Figure 4). Hypoechoic appearance was also noted for each image. The ultrasound measurement was used to investigate differences in plantar fascia thickness between runners with plantar fasciitis and uninjured runners since it is unknown if there is an adaptive thickening associated with runners.
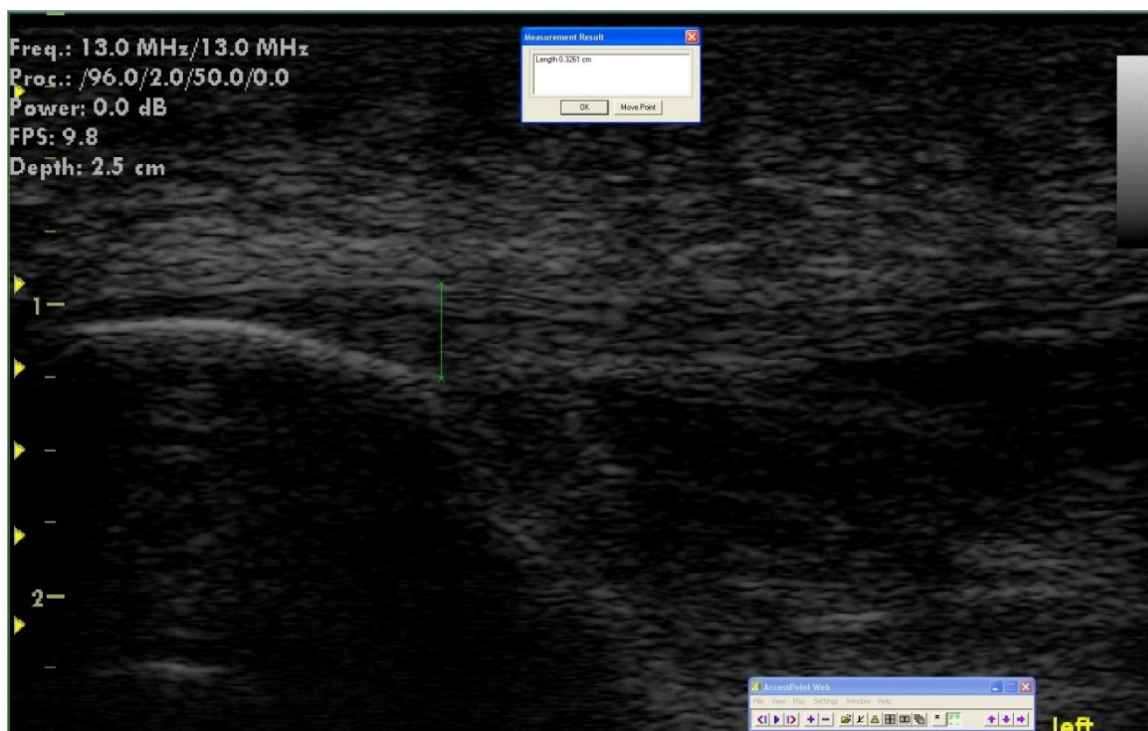
Figure 4. Representative ultrasound image of the left plantar fascia of an asymptomatic individual demonstrating the procedures that will be used to quantify the diameter of the plantar fascia.

Foot Structure Assessment

Arch structure was determined based on the navicular index measure assessed using the digital photographic measurement method (DPMM). The navicular index was calculated as a ratio of the navicular tubercle and the truncated foot length (distal toe – posterior calcaneus) (Figure 5). The participant's foot was positioned using the protocol outlined in Cobb et al. (2011). Briefly, the DPMM procedures consisted of the examiner identifying three anatomic landmarks (navicular tuberosity, medial mallelous, and the metatarsophalangeal joint) on the foot to be measured using a ballpoint pen (Figure 5). The medial border of the participant's foot to be measured was positioned in the center and along the front edge of a custom built measuring platform that was placed on a measurement scale. The leg was aligned vertically in the frontal and sagittal planes while digital photographs were captured during a 10% weightbearing condition. Next, two depth measurements of the dorsum of the foot and the metatarsophalangeal joint were obtained using sliding calipers. The measurements were used as inputs in the subsequent data analysis to correct for out of plane perspective errors.
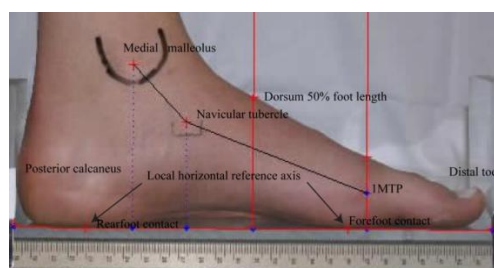
Figure 5. Digital photographic measurement method.

The pictures were then uploaded to a customized software program (Matlab v. 7.6.0, The Mathworks Inc, Natick, MA) where seven anatomic landmarks, two reference points, and a scale factor were identified to compute the navicular index. The anatomic

landmarks included the heel tip, navicular tubercle, first metatarsophalangeal joint, toe

tip, dorsum of the first metatarsophalangeal joint, dorsum height at 50% of the foot

length, and medial malleolus (Figure 5). The reference points were the rearfoot and

forefoot contact points, and the scale factor was computed through the digitizing of two

points of known length on a ruler positioned on the platform. To facilitate identification

of the dorsal landmarks, the software program plotted reference lines at 50% of total foot

length and at the first metatarsophalangeal joint, perpendicular to the local horizontal

reference axis (the line connecting the rearfoot and forefoot contact points) (Figure 5).

Perspective errors of out-of-plane anatomic landmarks were corrected by linearly

adjusting the scaling factor by the percentage distance out of plane using the following

formula: $\%\text{Error} = [1 - (L1/L2)] * 100$, where L1 is the plane-to-camera distance and L2

is the landmark-to-camera distance (Cobb et al., 2011).

Visit Two

Ultrasound Reliability

To assess the reliability of the examiner during the study, twenty percent of the

participants were randomly selected to have a second ultrasound exam. This exam lasted

approximately 20 minutes. The images were collected at the same time of day ($\pm$ 3 hours)

as the first images. The testing session was performed as per the protocol mentioned

previously. The reliability of the examiner was assessed on the images collected during

the two visits. The measurements of the images between the two sessions were defined as

reliable if the measurements were within one digital pixel (about 0.11 mm).

Gait Analysis

The running gait analysis was conducted in the Musculoskeletal Biomechanics Laboratory (Enderis 132). Prior to beginning the gait analysis, the participants were given time to walk and run in the sandal (Maui and Sons, Pacific Palisades, CA) that they would wear during the gait analysis trials. If the runners in the plantar fasciitis group were symptomatic due to inactivity prior to the gait analysis, they were encouraged to run on a treadmill to further warm-up until the pain subsided (Karabay et al., 2007; Messier & Pittala, 1988; Uden et al., 2011; Wearing et al., 2007). The participants were able to begin the gait trials only when they reported that they had a pain level of 2 on the 0-10 pain scale. This criterion was established to ensure the participants were not symptomatic and to avoid any effect of pain on their gait.

Participants completed 10 successful running trials at 4.0 ($\pm$10%) m/s along a runway with a force plate mounted in the middle of the runway. The runners ran at 4.0 ($\pm$10%) m/s to limit any variability in gait kinematics due to different running speeds. A successful running trial was defined as a trial during which initial contact and toe-off occurred on the force plate. Following each trial, participants in the plantar fasciitis group were asked to report their pain based on the 0-10 scale described previously. Since it is unknown whether the pain associated with plantar fasciitis changes foot kinematics during running, the gait trials were discontinued if the patient experienced a pain level of 3 or greater. Only one participant discontinued the trials due to a pain level of 8 during the gait trials. The data for this participant was discarded and another participant was recruited.

Multi-segment Foot model

A six foot segment model was used to quantify foot motion during the gait

analysis. The model partitioned the foot into six different segments (hallux, medial

forefoot [first and second metatarsals], lateral forefoot [fourth and fifth metatarsals]

navicular, cuboid, and calcaneus) and also defined a leg segment. During the gait

analysis, technical marker clusters consisting of four 6.4 mm markers were placed either

on the skin or on custom built wands on each of the segments of interest. The technical

markers identified six functional articulations: rearfoot complex (RC, formed by the leg

and calcaneus segments), calcaneonavicular complex (CNC, formed by the calcaneus and

navicular segments), calcaneocuboid joint (CC, formed by the calcaneus and cuboid

segments), medial forefoot (MFF, formed by the navicular and medial forefoot

segments), lateral forefoot (LFF, formed by the cuboid and lateral forefoot segments),

and 1st metatarsophalangeal complex (MTP, formed by the hallux and the medial

forefoot segments) (Figure 6).
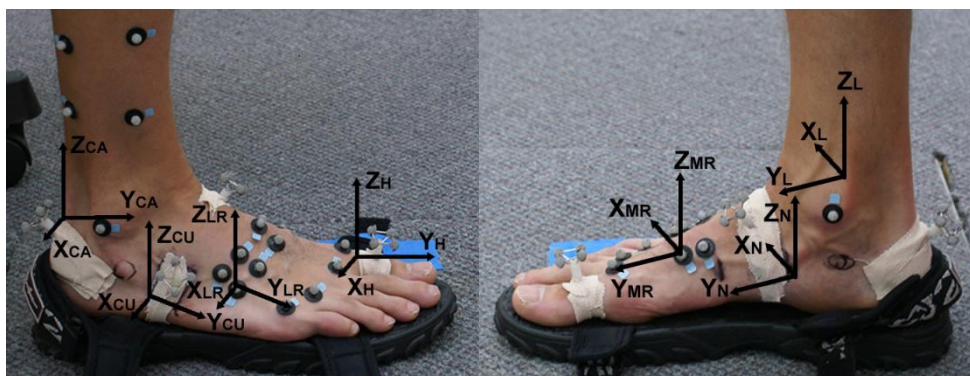
Figure 6. Technical and anatomical markers. The anatomical markers were the markers on the metatarsal heads, the malleoli, and tibial tuberosity (not shown). Additional anatomical landmarks on the calcaneus, navicular, hallux, and cuboid segments were identified using a Davis Pointer. **Left Figure**: cancaleus (X_{CA}, Y_{CA}, Z_{CA}), cuboid (X_{CU}, Y_{CU}, Z_{CU}), lateral rays (X_{LR}, Y_{LR}, Z_{LR}), and hallux (X_H, Y_H, Z_H) anatomical coordinate systems. **Right figure**: Leg (X_L, Y_L, Z_L), navicular (X_N, Y_N, Z_N), and medial rays (X_{MR}, Y_{MR}, Z_{MR}) anatomical coordinate systems. All of the anatomical coordinate systems were defined using the appropriate anatomical landmarks.

In contrast to other studies, the multi-segment foot model used in the current study has been demonstrated to be reliable during both walking and running gait (Bauer et al., 2011). All functional articulations across all three planes were very repeatable (correlation coefficients of ≥ 0.70), except the calcaneonavicular transverse plane (correlation coefficient $= 0.64$) and the lateral forefoot frontal plane (correlation coefficient $= 0.56$) which were moderately repeatable (Bauer et al., 2011). All technical marker clusters were secured to the participant using liquid adhesive (Mastisol, Ferndale Laboratories, Inc, Ferndale, MI), double sided adhesive electrode washers (In-Vivo Metric, Healdsburg, CA), and tape (Elastikon, Johnson & Johnson, New Brunswick, NJ). The technical marker clusters were placed on areas where skin movement relative to the underlying bone was minimal and not covered by the sandals worn by the participants.

Prior to performing the gait trials, an anatomical calibration procedure was completed to identify relevant anatomical landmarks on each segment and to define local coordinate systems within each segment. The procedure was a static trial in which the 3D position of additional anatomical reference landmarks on the foot and leg were identified using either 6.4 mm retro-reflective markers (Figure 6) or a Davis pointer. The participant was placed in a seated position for the calibration trial so that compensatory movements due to abnormal foot posture/mobility were not captured. The anatomical reference markers were then removed prior to the performance of the gait trials.

Three dimensional positions of the technical marker clusters were captured at 200 Hz with a 10-camera Eagle system (Motion Analysis Inc, Santa Rosa, CA). A force plate (Advanced Mechanical Technology, Inc., Watertown, MA) sampling at 1000 Hz mounted near the center of the run-way identified initial contact and toe-off events of the

stance phase. Following completion of the calibration and running trials, Cortex software (Motion Analysis Inc, Santa Rosa, CA) was used to reconstruct the 3D position of each reflective marker. A custom written software program (Matlab v. 7.6.0, The Mathworks Inc., Natick, MA) was then used to filter the data, and reconstruct the 3D position of each segment using the calibrated anatomical system technique with a single value decomposition optimization procedure (Cappozzo, 1984). The single value decomposition is an optimization procedure that was incorporated to further minimize skin movement errors that occur during dynamic movements. The anatomical reference markers were reconstructed using the single value decomposition optimization procedure and used to define six functional articulations, compute anatomical axes of rotation, and compute clinically relevant joint angles for each of the functional articulations using the joint coordinate system technique (Grood & Suntay, 1983). The data were time normalized to 100% stance, and ensemble averaged from five of the ten trials. Due to the variability of gait, a minimum of 5 trials are required to collect reliable data. The five most consistent trials were used for ensemble averaging. The consistency of the trials was determined by visual assessment of the time-series plots. Finally, the Matlab program calculated joint range of motion in four different subphases of stance. The subphases were defined as phase 1 (0-20% of stance), phase 2 (21-50% of stance), phase 3 (51-75% of stance) and phase 4 (76-100% of stance) (Ferber et al., 2005). All motions were distal segment moving on the proximal segment with the exception of the rearfoot complex, which was the proximal moving on the distal segment.

Statistical Analysis

The Statistical Package for the Social Sciences version 13.0 (SPSS Inc., Chicago, IL) was used to perform the statistical analyses. Joint angles of the functional articulations within each of the four stance subphases were computed. For each stance subphase, three separate MANOVAs were performed to analyze between-subject sagittal, frontal and transverse planes range of motion differences for three of the functional articulations (RC, CNC, and CC). Three separate MANOVAs were performed to analyze between-subject sagittal plane range of motion for the remaining three functional articulations (MFF, LFF, and MTP). The independent variables in the MANOVAs were the group (plantar fasciitis and control) and the dependent variables were the range of motion in each plane (sagittal, frontal, transverse). To minimize the number of variables within each MANOVA, a preliminary analysis was performed to determine whether a primary motion occurred for each joint. If there was less than 1° of motion, it was eliminated from the analysis within the MANOVA. Follow-up independent t-tests were used to investigate significant MANOVA omnibus F ratios. The significance level for all of the tests was $\alpha \leq 0.05$.

The secondary purpose of the study was to investigate differences in plantar fascia thickness between runners with plantar fasciitis and uninjured runners. To assess this relationship, independent t-tests were performed to compare differences in plantar fascia thickness between the control and plantar fasciitis groups. A dependent t-test was performed to compare differences in plantar fascia thickness between the plantar fasciitis group injured foot versus the uninjured foot.

CHAPTER 4: RESULTS

Foot Structure

Prior to performing the gait kinematic statistical analysis, differences in foot structure between the injured and uninjured groups were investigated. The preliminary investigation was performed to determine if foot structure should be included as a co-variate in the subsequent analyses. This was deemed important due to the theorized relationship between foot structure and plantar fasciitis (Huang et al., 2004; Kwong et al., 1988; Pohl et al., 2009; Rome et al., 2001; Taunton et al., 2002; Wearing et al., 2006; Williams et al., 2001) and previous studies that have revealed kinematic gait differences between participants with differing foot structures (Arndt et al., 2007; Cobb et al., 2009). Independent t-tests were used to assess differences between the groups. One control group participant's images could not be digitized, so the data was discarded from the foot structure comparison. Results revealed no significant difference in arch structure between groups ($F_{1,27} = 0.361$ p = 0.553). Based on descriptive data collected from a group of uninjured individuals a NI of less than 0.204 (the ratio 1SD below the mean of the descriptive data) may be classified as a low arch, a NI between 0.204 and 0.268 (ratios within ± 1 SD of the mean of the descriptive data) may be classified as typical arch, and a NI of greater than 0.268 (the ratio 1 SD above the mean of the descriptive data) may be classified as a high arch. Using the above criteria, both groups would be classified as having typical arch structure (plantar fasciitis mean: 0.229 ± 0.031; control mean: 0.232 ± 0.028).

Multi-Segment Foot Kinematics

Phase 1

The MANOVA results for the CC ROM between the runners in the plantar fasciitis group and the control group was significant ($F_{3,26} = 4.042$, $p = .017$). MANOVA results were not significant for RC, CNC, MFF, LFF, or MTP ROM between groups during phase 1 (Appendix G). Follow-up independent t-tests for the CC joint omnibus F-ratio revealed significantly increased eversion excursion ($F_{1,28} = 10.514$, $p = 0.003$) in the plantar fasciitis group (mean: $-3.63 \pm 2.73°$) versus the control group (mean: $-1.16 \pm 1.14°$). The control group landed in a more everted position, with very little frontal plane movement in phase 1. In comparison, the plantar fasciitis group landed in a less everted position, then everted to the end of phase 1. There were no other significant findings in the CC joint ROM (Table 2, Figure 7).

Table 2. Mean (SD) sagittal, frontal, and transverse plane ROM for the calcaneocuboid joint during phase 1

		Plantar Fasciitis	**Control**	**p-value**
Sagittal Plane	Dorsiflexion	4.44 (3.74)	4.91 (2.73)	p = 0.701
Frontal Plane	Eversion	3.63 (2.73)	1.16 (1.14)	p = 0.003*
Transverse Plane	Adduction	3.63 (1.89)	3.28 (2.14)	p = 0.645

*Significantly different from control group ($p < 0.05$)
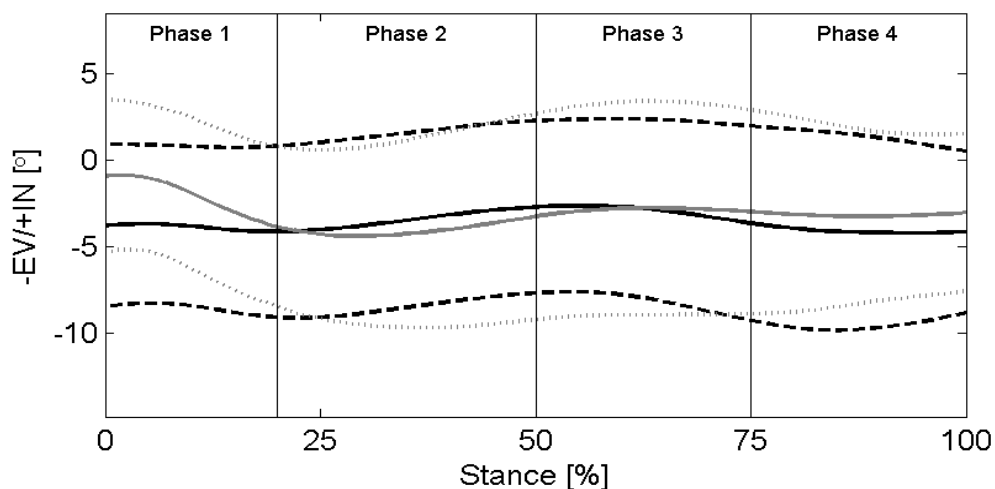
Figure 7. Frontal plane calcaneocuboid joint stance phase kinematics (mean ± 1 SD) for the control (black lines) and plantar fasciitis (gray lines) group participants.

Phase 1 (0-20% of stance) defined the early period of stance phase. During this phase, the CC (calcaneocuboid) eversion excursion was significantly greater in the plantar fasciitis group compared to the control group (mean difference: 2.47°). While the CC is everting during this time, the RC (rearfoot complex) and lateral midfoot (CC) are in contact with the ground and the foot is transitioning from a relatively rigid structure to a mobile structure. With the CC motion, the control group landed in a more everted position, and then exhibited very little frontal plane movement during the phase (1.16 ± 1.14°). In comparison, the plantar fasciitis group landed in a less everted position, then everted through the end of the phase (3.63 ± 2.73°) (Figure 9, Appendix I).

Phase 2

MANOVA results did not reveal significant group differences for any of the variables within the functional articulations during phase 2 (Appendix H). Phase 2 (21-

50% of stance phase) defined the midstance period of stance phase. Both groups began

phase 2 in a dorsiflexed MFF position, and continued to dorsiflex to the end of phase 2,

with the plantar fasciitis group in slightly greater dorsiflexion (Figure 11, Appendix I).

For the CNC, the plantar fasciitis group began in a greater inverted position compared to

the control group. However, the plantar fasciitis group went through slight inversion

before everting to the end of phase 2, ending in a similar position as they started in. The

control group had less inversion at the beginning, and instead everted to the end of the

phase, to a more neutral CNC position (Figure 10, Appendix I).

Phase 3

MANOVA results did not reveal significant group differences for any of the

variables within the functional articulations during phase 3 (Appendix H). Phase 3 (51%-

75% of stance phase) defined the late stance period of stance phase. The plantar fasciitis

group started in a greater inverted CNC position compared to the control group; however,

both groups displayed a similar pattern of eversion through the rest of the phase, with the

plantar fasciitis group resulting in slightly increased eversion excursion. Both groups also

followed a similar pattern for the MFF. They started in a dorsiflexed position, then

plantarflexed to the end of the phase.

Phase 4

MANOVA results were not significant for the RC, CC, CNC, MFF, LFF, or MTP

ROM between groups during phase 4 (Appendix H). Phase 4 (76-100% of stance phase)

defined the terminal stance period of stance phase. Both groups exhibited a similar pattern of MTP (first metatarsalphalangeal complex) motion. They begin in an extended position, and then extended to ≈90% of the phase, where they started to flex. The plantar fasciitis group started and ended with slightly less extension, so the overall motion was greater (Figure 11, Appendix I).

Ultrasound

Twenty percent of the participants (6 randomly selected participants) underwent repeat scans within one week of the first scan (± 3 hours). Intra-reader and intra-scanner measurements were assessed. All measurements of the reliability session were within one digital pixel of the first session.

Independent t-test results revealed the thickness of the plantar fasciitis group participants was significantly greater (mean: 4.64 ± 1.07 mm) than the limb-matched control group participants (mean: 3.75 ± 0.54 mm) ($F_{1,28} = 6.650$, $p = 0.007$). Although the mean of the control group plantar fascia thickness was less than 4.0 mm, there were three control group participants who had a thicker plantar fascia (over 4.0 mm) but were asymptomatic (Range: 4.372 mm – 4.772 mm). In addition, there were six plantar fasciitis group participants who did not demonstrate thickening of the plantar fascia greater than 4 mm (Range: 3.153 mm – 3.969 mm).

The dependent t-test comparing the thickness of the plantar fascia between the plantar fasciitis group injured foot versus the uninjured foot revealed the thickness of the injured foot was significantly greater than the uninjured foot (mean: 3.66 ± 0.56 mm) ($F_{1,28} = 6.776$, $p = 0.004$).

Results of an independent t-test that assessed the non-injured foot in the plantar fasciitis group and the limb-matched control were not significant ($F_{1,28} = 0.019$, p = 0.659). The thickness of the non-injured side was slightly less (mean: 3.75 ± 0.54 mm) than that of the control group.

CHAPTER 5: DISCUSSION

The primary hypothesis of this study was that participants with plantar fasciitis would have altered kinematics during the mid and late stance phases of gait. Specifically, it was hypothesized that the medial midfoot (calcaneonavicular complex) and forefoot would be more mobile during midstance, and that the first metatarsophalangeal joint would exhibit increased extension in late stance due to a decreased stiffness and degeneration of the plantar fascia. These hypotheses were not supported by the current study. Although the plantar fasciitis group demonstrated greater medial midfoot and forefoot motion during midstance and increased first metatarsalphalangeal complex extension during late stance compared to the uninjured group, the differences were not statistically significant. However, there were significant differences between the groups in the calcanealcuboid joint during early stance (phase 1).

The secondary hypothesis of this study was that the thickness of the plantar fascia would be significantly greater in runners with plantar fasciitis compared to uninjured runners. This hypothesis was supported by the data.

Multi-segment Foot Kinematics

Phase 1

The increased eversion excursion of the CC in the plantar fasciitis group may suggest decreased lateral midfoot stability as eversion is a component of pronation which functions to increase the mobility of the foot. However, the role of the plantar fascia in supporting the lateral longitudinal arch is unknown. All of the previous plantar fascia

(Kayano et al., 1986; Sarrafian, 1987; Wright & Rennels, 1964) and plantar fasciitis (Messier & Pittala, 1988; Pohl et al., 2009; Taunton et al., 2002) studies have focused exclusively on the contribution of the plantar fascia to medial longitudinal arch stability and the effect of plantar fasciitis on medial longitudinal arch function (Huang et al., 2004; Wearing et al., 2004), respectively. Similarly, there is no previous gait research to which to compare the results of the current study since all the studies have either ignored the midfoot altogether (Chang et al., 2007; Messier & Pittala, 1988; Pohl et al., 2009; Taunton et al., 2002) or have performed walking gait trials rather than running (Chang et al., 2007). However, if the rearfoot and midfoot act as a constrained tarsal mechanism as proposed by (Huson et al., 2000), a loss of rearfoot complex stability (RC) may result in a loss of medial (CNC) and lateral (CC) midfoot stability. The decreased midfoot stability could also affect both the medial and lateral longitudinal arch (formed by the calcaneus, cuboid, and 4th and 5th metatarsals) stability. In the current study, however, only lateral midfoot (CC) motion was significantly different between the groups. One potential reason that the medial midfoot (CNC) motion was not significantly affected may be that it was not on the ground during early stance and therefore, not loaded to the same degree as the rearfoot complex and the lateral midfoot. Moreover, it is possible that the extrinsic and intrinsic foot musculature were able to compensate for a decrease in medial longitudinal arch stability caused by the loss of plantar fasciitis stiffness. The musculature may not, however, be well positioned to compensate for decreases in lateral longitudinal arch stability. The majority of the larger intrinsic and extrinsic foot muscles that function as dynamic stabilizers are either located on the medial side or have attachment to the medial foot.

Regarding the RC (rearfoot complex) eversion ROM, there was no statistically significant difference between groups. The participants in the plantar fasciitis group demonstrated only slightly greater RC eversion excursion during phase 1 (mean: -7.39 ± 3.12°) compared to the control group (mean: -5.92 ± 2.51°). Visual inspection of the frontal plane RC graphs suggests that although the plantar fasciitis group participants landed in a greater inverted position compared to the control group, both groups underwent a similar eversion range of motion during the subphase. As previously stated, one explanation for lack of statistical significance may the positioning of the extrinsic and intrinsic foot musculature. The failure to reach statistical significance may also have been due in part to the large variability within groups (plantar fasciitis group eversion ROM range: 2.91° – 12.349°; control group eversion ROM range: 0.045° – 19.83°) (Figure 8, Appendix I).

Differences in foot strike patterns may have contributed to the variability. Visual assessment of the runners during the gait analysis session suggested that some runners were rearfoot strikers, while others were midfoot/forefoot strikers. Previous studies have shown that foot strike pattern affects the kinematics of healthy runners (Lieberman et al., 2010; William et al., 2000). Due to the exploratory nature of the current study, however, foot strike pattern was not used as inclusionary/exclusionary criteria. This may, however, be important criteria to consider in future studies. It is unclear if the runners with plantar fasciitis were natural midfoot/forefoot runners or if they changed their foot strike from a rearfoot to the mid/forefoot either intentionally or unintentionally to reduce their heel pain during running. If the runners did alter their foot strike pattern due to pain, it was likely an adaptation that occurred over time versus during the running trials since all of

the participants were currently running a minimum of 10 miles/week. Additionally, some of the variability across participants may have been due to the unfamiliarity with the sandal used in the study. However, a previous study that investigated the kinematics differences between running in shoes versus sandals, reported no differences in rearfoot eversion excursion between the conditions (Barnes et al., 2010). This study used both gait sandals and shoes with a neutral foot bed, which was similar to the flat, neutral foot bed of the sandal used in the current study. It should be noted, however, that no study has analyzed the midfoot and forefoot kinematic differences between a running shoe and sandal due to the impracticality of placing retroreflective markers on the distal segments foot in a running shoe. Therefore, it is unknown whether the distal foot segments function differently in a running shoe versus a sandal.

Phase 2

It was hypothesized that a softening of the plantar fascia in the plantar fasciitis group (Wu et al., 2011) would result in increased midfoot and forefoot ROM during midstance. However, this hypothesis was not supported. Rather, there were no significant differences between groups in the midfoot and forefoot. The range of motion in the medial midfoot (plantar fasciitis eversion ROM: $1.62 \pm 1.76°$; control group ROM: $1.60 \pm 0.99°$; plantar fasciitis inversion ROM: $1.47 \pm 2.53°$; control group inversion ROM: $0.45 \pm 0.68°$) and medial forefoot (plantar fasciitis dorsiflexion ROM: $6.54 \pm 2.88°$; control group dorsiflexion ROM: $6.00 \pm 2.06°$) were very similar.

The results of the current study are inconsistent with those of Chang et al (2007). Chang et al (2007) observed increased forefoot motion in the plantar fasciitis group

participants compared to healthy controls during walking. However, the differences between the results of the current study and the Chang et al. (2007) study may be explained by the differences in methodology. Chang et al. (2007) assessed walking gait while the current study investigated running gait. Recent studies comparing the two gait modes have indicated that the foot functions differently in walking versus running gait (Arndt et al., 2012, Cobb et al., 2012). In a preliminary study, Arndt et al. (2012) investigated in-vivo multi-segment foot kinematics during walking and running in healthy adults. The authors demonstrated that the foot, particularly the midfoot, was more mobile during walking but revealed increased rigidity during running. Furthermore, in another preliminary study, Cobb et al. (2012) demonstrated increased foot mobility during walking compared to running in different shoe conditions. In addition, the previous study analyzed forefoot motion by assessing the movement of the forefoot relative to the calcaneus (Chang et al., 2007), which is different than the six-segment foot model used in the current study which tracked the specific motion of the medial and lateral midfoot.

The previously mentioned increased rigidity of the foot in running gait during walking gait is likely the result of increased activity of the dynamic stabilizers of the foot (the intrinsic and extrinsic foot muscles). If this is the case, our hypotheses regarding midfoot motion may not have been supported in part because the increased muscular activity may have compensated for the loss of plantar fascia stiffness during running

Phase 3

It was hypothesized that there would be an increase in the medial midfoot and altered medial forefoot motion in the plantar fasciitis group during this phase due to the

softening of the plantar fascia (Wu et al., 2011); however, this was not supported. Rather, there were no significant differences in medial midfoot (CNC) motion (plantar fasciitis group eversion ROM: $3.58 \pm 1.60º$; control group eversion ROM: $3.03 \pm 1.66º$) or medial forefoot (MFF) motion (plantar fasciitis group plantarflexion ROM mean $\pm$ SD: $-7.84 \pm 2.3°$; control group plantarflexion ROM mean $\pm$ SD: $-7.95 \pm 2.71°$) between groups.

Phase 4

It was hypothesized that participants in the plantar fasciitis group would demonstrate increased first metatarsophalangeal joint complex ROM during the terminal stance of running gait; however, this was not supported. Rather, there were no significant differences between groups during phase 4.

As previously mentioned, during terminal stance the plantar fascia simulates a windlass by wrapping around the metatarsal heads, locking the midtarsal joints and raising the arch. Therefore, it was expected that the softening of the plantar fascia associated with plantar fasciitis (Wu et al., 2011) would affect the motion at the first metatarsophalangeal joint during late stages of running gait. The results of the current study, however, did not support this theory. Rather, there were no significant kinematic differences between groups during phase 4.

These results are also inconsistent with those from a previous study that, demonstrated an increase in the peak metatarsophalangeal joint angle ($\approx 4º$ mean difference) in individuals with plantar fasciitis compared to uninjured participants during terminal stance of walking gait(Wearing et al., 2004). The mean difference in metatasophalangeal joint range of motion between the groups in the current study was

less than 2º. Additionally, the peak angle (≈30º) occurred at ≈90% of stance phase for both groups (Figure 11, Appendix I). However, as seen in the figure, there was also large variability in the joint ROM within the plantar fasciitis group (range: 4.04º - 24.71º), which may have contributed to the lack of statistical significance (Table 6, Appendix H). Further inspection of the plantar fasciitis group data revealed that two participants had very low extension ROM (4.04º and 5.83º) during the phase. Potential reasons for this may have been that over time the participants altered their gait to avoid pain or discomfort associated with first metatarsophalangeal (MTP) joint extension during late stance. As previously stated, another explanation may be that the muscular activity (dynamic stabilizers) was sufficient to compensate for the decreased stiffness of the plantar fascia.

With respect to the inconsistency in the results of the current study compared to those of Wearing et al. (2004), differences in methodology between the previous studies may partially explain the disparity between the findings. Wearing et al. (2004) used two-dimensional digital fluoroscopy to record dynamic lateral radiographs of the foot (Wearing et al., 2004), compared to the three-dimensional six-segment surface marker based foot model used in the current study. It is unknown if the two methods of analyzing foot motion can be compared. However, a limitation to this previous data collection was that only the initial 80% of stance was analyzed, because the frequency response of the image intensifying system resulted in a blurring of the images beyond this point (Wearing et al., 2004). Since the current study revealed a peak angle at ≈90% of stance, it is difficult to compare the peak angles between the studies. Moreover, participants in the previous study were assessed during walking gait (Wearing et al., 2004). As previously

mentioned, foot function during walking gait cannot be compared directly to foot function during running (Arndt et al., 2012 ; Cobb et al., 2012).

Ultrasound

The results of the ultrasound measurements supported the hypothesis and were consistent with previous findings (Cardinal, 1996; Karabay et al., 2007). There was an associated increase in plantar fascia thickness in those with plantar fasciitis (mean: 4.64 ± 1.07 mm). Previous studies have reported that a thickness of ≥ 4.0 mm in patients with the traditional clinical signs and symptom of the pathology was an indication of plantar fasciitis (Cardinal, 1996; Huang et al., 2004; Karabay et al., 2007). In the current study, 4.0 mm was not used as criteria because it was unknown if this thickness would be appropriate for younger more active individuals. In this population, there may be an adaptive thickening due to the increased load experienced during running, which has not been investigated prior to the current study.

Before making generalizations regarding the effect of plantar fasciitis on plantar fascia thickness in runners, a number of important factors should be considered. These data suggest that although the average thickness of the plantar fasciitis group was greater than 4.0 mm, and the average thickness of the control group was less than 4.0 mm, this threshold may not be most appropriate criteria for runners. Rather, a percentage difference of the plantar fasciitis group that is compared to either a large descriptive data set of uninjured runners or to the uninvolved limb, together with a clinical exam that confirms that presence of the clinical signs and symptoms of the condition may be more

appropriate. Additionally, it may also be important to include additional sonographic measures such as echogenicity, stiffness, and fluid level, in addition to thickness since those measurements have previously been reported to be associated with plantar fasciitis (Cardinal, 1996; Karabay et al., 2007).

Second, age has also been shown to be associated with an increase in fascia thickness and a softening of the plantar fascia (Wu et al., 2011). Wu et al (2011) examined the plantar fascia of participants who were 50 years old and older without plantar fasciitis and compared them to a group of individuals with plantar fasciitis. Results revealed similar softening of the plantar fascia in both groups. In an attempt to avoid recruiting participants with age- related plantar fascia changes that may have masked differences associated with plantar fasciitis, the current study excluded individuals greater than 45 years old. However, since Wu et al. (2011) only recruited participants in the older age group who were 50 and older, the point at which the age-related changes in the plantar fascia begin is unknown. If the changes begin prior to the age of 45, it is possible that some of the control participants in the current study had age-related changes to the plantar fascia similar to those associated with plantar fasciitis. In the current study, the three participants in the control group who had a thickness of greater than 4.0 mm were between 32 and 45 years old. Stiffness was not assessed in the current study so it is unclear whether this was a factor. Furthermore, it is unclear whether the thickness associated with the runners was degenerative or adaptive from running. In comparison to Wu et al., (2011), the average plantar fascia thickness of the younger healthy subjects (2.4 ± 3.0 mm) was much less than those of the current study (3.7 ± 0.5 mm) in a relatively similar age group (Mean age for both studies ≈30 years). These data

suggest that it is possible for an adaptive thickness to occur during running. Moreover, these factors may further suggest the need for other sonographic measures (echogenicity, fluid, or elasticity) of the plantar fascia in addition to thickness assessment. These measurements would enhance the understanding of the structure of the plantar fascia. In addition, there may be a need to develop relative difference criteria and/or criteria that consider age and/or activity level.

Third, although previous research has shown a significant difference between the uninjured foot in the plantar fasciitis group compared to the control group (Fabrikant et al., 2001; Wearing et al., 2004), the current study did not show similar findings. Plantar fascia thickness in the asymptomatic limb (mean: 3.66 ± 0.56 mm) of the plantar fasciitis group was not significantly different than that of the control group (mean: 3.75 ± 0.54 mm). Although it is difficult to compare data from the current study to the previous studies due to the differing age and activity levels of the participants, these data may further suggest that there is a healthy adaptive thickening of the plantar fascia that occurs in response to the repetitive loading during running.

Other Factors

It is well-known that the plantar fascia is a main contributor of medial longitudinal arch support during both static stance and walking gait (Wearing et al., 2006). The contribution of the plantar fascia to medial longitudinal arch support during running gait, however, has not been investigated. Results of the current study suggest that other than calcaneocuboid joint eversion ROM during early running stance, there are no significant running gait kinematic differences between runners with and without plantar

fasciitis. However, prior to making generalizations regarding the effect of plantar fasciitis on foot function during running, a number of important factors should be considered. First, the role of dynamic stabilizers in supporting the medial longitudinal arch during running gait has not been established. Other authors have investigated intrinsic foot muscle activity during loading conditions, demonstrating increased muscle activity in the supporting foot muscles with an associated increase in load (Salathe & Arangio, 2002; Thordarson et al., 1995). Salathe and Arangio (2002) also reported that this increased load reduces the amount of load on the plantar fascia. There was an associated increase in the force exerted by the muscles during the toe-standing condition, which is thought to help in maintaining balance in addition to maintaining the medial longitudinal arch. With their mathematical model, these authors concluded that the muscles of the foot actively support the arch during toe-off and decrease the load borne by the plantar fascia (Salathe & Arangio, 2002). Kibler et al. (1991) also found that high deficits of plantar flexor muscle strength deficits were associated with plantar fasciitis (Kibler et al., 1991).

More recently, Chang et al. (2012) used MRI to estimate the volume of the tibialis posterior and plantar intrinsic foot muscles between the affected and unaffected limb of participants with unilateral chronic plantar fasciitis. Their results revealed that the forefoot volumes of the plantar intrinsic foot muscles in the affected limb were significantly smaller in participants involved versus uninvolved foot. Rearfoot, total foot volume, and tibialis posterior size were similar between groups (Chang et al., 2012). The atrophy of the forefoot in the plantar fasciitis group suggests potential dysfunction may occur during gait. The forefoot muscles may fatigue faster because of their smaller volume, so changes in kinematics may not occur until this point. In conjunction with the

evidence that pain associated with plantar fasciitis decreases with the first few minutes of running and returns with prolonged activity (Cardinal et al., 1996; Karabay et al., 2007), these results may suggest that changes in running gait may not be evident until the foot musculature becomes fatigued. However, the population studied in the Chang et al. (2012) was older (44.9 ± 8.4 years) and had no minimum activity-level requirement compared to the current study (30.0 ± 8.7 years), but participants were excluded if they had a BMI of > 35 kg/m^2. The age and activity level of the participants in this previous study may limit the generalization of the results since the effect of physical activity on the foot musculature is unknown (Buchbinder, 2004). It is possible that the repetitive loading of running may result in hypertrophy or limited atrophy and weakening of the foot musculature.

Second, although degenerative changes within the plantar fascia have been associated with plantar fasciitis (Lemont et al., 2003; Wu et al., 2011), when the degenerative changes begin and when the changes are sufficient to affect function has not been established. Participants in the plantar fasciitis group in the current study were required to have symptoms for at least six weeks. The average length of symptoms in the current study (5.67 ± 4.85 months) was less than the average duration of 10-12 months in the Wu et al. (2011) study. Although previous research has suggested that six weeks is a sufficient amount of time for mechanical changes to occur within the plantar fascia (Wearing et al., 2004), it is possible that the hypothesized kinematic differences were not observed in the current study because the degeneration had not reached the threshold at which function was affected.

Limitations

Multi-segment Foot Kinematics

There are a number of limitations to this study that should be considered prior to drawing conclusions from these data. First, all of the participants in the study were runners that ran at least 10 miles per week. Second, participants were excluded if BMI was greater than 30 kg/m^2. As previously mentioned, it is unknown whether obesity and/or inactivity contribute differently to the development of plantar fasciitis (Buchbinder, 2004), therefore, the results of the current study may not be applicable to individuals with plantar fasciitis who are less active and/or obese.

Third, although all participants walked and ran in the sandal provided by the lab prior to beginning the running gait trials, it is possible that participants altered their kinematics due to not being used to the sandal. Furthermore, as previously stated the kinematics in the distal foot segments may be different running in a sandal versus a running shoe. Fourth, the fact that foot strike pattern was not an inclusionary/exclusionary criterion is another limitation of the current study. Previous data has shown that foot strike does affect foot/rearfoot running kinematics (De Wit et al., 2000; Lieberman et al., 2010; Williams et al., 2000), so it is unknown how this affected the joint angles. However, no previous study has determined whether plantar fasciitis is associated with a specific foot strike pattern, or a change in foot strike pattern, therefore it was not feasible to use foot strike pattern as an inclusionary/exclusionary criteria for the current study.

Ultrasound

There were also limitations associated with the ultrasound testing. Specifically, it is unknown whether activity level and time of day affect the changes in thickness or appearance of the plantar fascia; therefore, the re-test of participants for the reliability portion of the study may have been strengthened if both measures were assessed during the same day (Rathleff et al., 2010). In addition, the absence of guidelines for the participants to follow prior to the ultrasound examination and the fact that the time of day at which sonography assessment was performed was not standardized may also be limitations of the study. These may be important omissions if activity performed prior to assessment and/or diurnal changes do influence the thickness of the plantar fascia (Rathleff et al., 2010). While sonography assessment was always performed prior to gait analysis, standardizing the time of day and activities prior to testing was not feasible due to variability associated with access to the sonography system and the availability and training schedules of the participants.

Directions for Future Research

This was the first study to investigate the effect of plantar fasciitis on multi-segment foot kinematics during running. While the study has advanced the understanding of how plantar fasciitis affects foot function, further investigation into a number of areas related to the pathology and its effect on function are warranted. Specifically, two areas that require further research are that of the progressive degenerative process associated with plantar fasciitis and the relationship between the progressive degenerative process and foot function. Future research should focus on the sonographic and gait assessment of patients grouped according to the duration of plantar fasciitis symptoms. Additionally,

future studies could partition groups based on running experience. The pathoetiology and effect of plantar fasciitis on gait mechanics in recreational or less experienced runners may be different than in elite or very experienced runners. It is possible that the plantar fasciitis may be more of an inflammatory pathology in the recreational runners, versus a more degenerative condition in the experienced or elite runners.

Another area that requires further investigation is the effect of prolonged running on the foot mechanics of runners with plantar fasciitis. This would allow the researcher to determine whether the pain associated with plantar fasciitis resulted in participants' alteration in gait pattern over time. If differences in kinematics are noted between the groups after the prolonged run, it may be that the foot muscles are unable to compensate for a loss of stiffness in the plantar fascia when fatigued. Additionally, since foot strike pattern has previously been shown to affect foot kinematics (De Wit et al., 2000; Lieberman et al., 2010; Williams et al., 2000), future research may need to group plantar fasciitis and control participants according to foot strike pattern. It might also be interesting to see if runners develop the plantar fasciitis as midfoot/forefoot strikers or if they changed their foot strike pattern because of the pain associated with plantar fasciitis.

Finally, since orthotics have been used as a successful conservative treatment for individuals with plantar fasciitis in several studies (Cole et al., 2005; Davis et al., 1994; Kogler et al., 1996; Roos et al., 2006), an orthotic intervention would be an appropriate follow-up. It would be interesting to investigate whether the increased eversion calcaneocuboid joint excursion during early stance that was present in runners with plantar fasciitis would be altered with orthotic intervention.

Summary

The primary hypothesis of the study was that runners with plantar fasciitis would demonstrate statistically significant distal foot kinematic differences during mid and late stance phases of gait. However, this hypothesis was not supported. Rather, there was a significant difference in the calcanealcuboid (CC) joint eversion during early stance. The increased eversion excursion of the CC in the plantar fasciitis group may suggest decreased lateral midfoot stability. The secondary hypothesis was supported, in that the ultrasound images revealed statistically significant thickening in the plantar fasciitis group. Although this study has advanced the understanding of the effect of plantar fasciitis on running gait, additional study of the influence of extrinsic and intrinsic foot musculature and foot strike pattern are warranted. It is possible that muscular activity was sufficient to compensate for the decreased stiffness of the plantar fascia, so that changes in kinematics in individuals with plantar fasciitis may not be evident until the dynamic muscular support becomes fatigued. Additionally, previous studies have shown that foot strike pattern affects the kinematics of healthy runners so this may be important criteria to consider in future studies.

REFERENCES

Akfirat, M., Sen, C., & Gunes, T. (2003). Ultrasonographic appearance of the plantar fasciitis. *Clin Imaging, 27*(5), 353-357.

Aldridge, T. (2004). Diagnosing heel pain in adults. [Review]. *Am Fam Physician, 70*(2), 332-338.

Allen, R. H., & Gross, M. T. (2003). Toe flexors strength and passive extension range of motion of the first metatarsophalangeal joint in individuals with plantar fasciitis. *J Orthop Sports Phys Ther, 33*(8), 468-478.

Alshami, A. M., Babri, A. S., Souvlis, T., & Coppieters, M. W. (2007). Biomechanical evaluation of two clinical tests for plantar heel pain: the dorsiflexion-eversion test for tarsal tunnel syndrome and the windlass test for plantar fasciitis. [Evaluation Studies]. *Foot Ankle Int, 28*(4), 499-505. doi: 10.3113/FAI.2007.0499

Arangio, G. A., Chen, C., & Salathe, E. P. (1998). Effect of varying arch height with and without the plantar fascia on the mechanical properties of the foot. [Comparative Study Research Support, Non-U.S. Gov't]. *Foot Ankle Int, 19*(10), 705-709.

Arndt, A., Wolf, P., Liu, A., Nester, C., Stacoff, A., Jones, R., . . . Lundberg, A. (2007). Intrinsic foot kinematics measured in vivo during the stance phase of slow running. [Clinical Trial]. *J Biomech, 40*(12), 2672-2678. doi: 10.1016/j.jbiomech.2006.12.009

Basmajian, J.V., Bentzon, J.W. (1954). An electromyographic study of certain muscles of the leg and foot in the standing position. Surg, Gynec., and Obstet., 98, 662-666.

Basmajian, J. V., & Stecko, G. (1963). The role of muscles in arch support of the foot: an electromyographic study. *Journal of Bone and Joint Surgery, 45A*, 1184-1190.

Bauer, R., Joshi, M., Klinkner, T., & Cobb, S. (2011). *A multi-segment foot model based on in-vivo and in-vitro stereophotogrammetric studies and clinical theories of dynamic foot function: Running gait reliability.* Paper presented at the American Society of Biomechanics annual meeting.

Baxter, D. E. (1994). The heel in sport. *Clin Sports Med, 13*(4), 683-693.

Bojsen-Møller, F., Flagstad, K.E. (1976). Plantar aponeurosis and internal architecture of the ball of the foot. J Anat,121, 599-611.

Boyle, K. L., Witt, P., & Riegger-Krugh, C. (2003). Intrarater and interrater reliability of the beighton and horan joint mobility index. *J Athl Train, 38* (4), 281-285.

Brasile, F. M., & Hedrick, B. N. (1996). The relationship of skills of elite wheelchair basketball competitors to the international functional classification system. *Therapeutic Recreation Journal, Second Quarter*, 114-127.

Buchbinder, R. (2004). Clinical practice. Plantar fasciitis. [Review]. *N Engl J Med, 350*(21), 2159-2166. doi: 10.1056/NEJMcp032745

Cappozzo, A. (1984). Gait analysis methodology. *Human Movement Science, 3*, 27-50.

Cardinal, E., Chhem, R. K., Beauregard, C. G., Aubin, B., & Pelletier, M. (1996). Plantar fasciitis: sonographic evaluation. *Radiology, 201*(1), 257-259.

Carson, M. C., Harrington, M. E., Thompson, N., O'Connor, J. J., & Theologis, T. N. (2001). Kinematic analysis of a multi-segment foot model for research and clinical applications: a repeatability analysis. *Journal of Biomechanics, 34*(10), 1299-1307.

Cashmere, T., Smith, R., & Hunt, A. (1999). Medial longitudinal arch of the foot: stationary versus walking measures. *Foot and Ankle International, 20*(2), 112-118.

Chang, R., van Emmerik, R., & Hamill, J. (2007). *Kinematics and anti-phase coordination of the rearfoot-forefoot couple in chronic plantar fasciitis.* Paper presented at the 10th Annual International conference on foot biomechanics and orthotic therapy.

Cobb, S. C., James, C. R., Hjertstedt, M., & Kruk, J. (2011). A digital photographic measurement method for quantifying foot posture: validity, reliability, and descriptive data. *J Athl Train, 46*(1), 20-30. doi: 10.4085/1062-6050-46.1.20

Cobb, S. C., Tis, L. L., Johnson, J. T., Wang, Y. T., Geil, M. D., & McCarty, F. A. (2009). The effect of low-mobile foot posture on multi-segment medial foot model gait kinematics. *Gait and Posture, 30*(3), 334-339. doi: S0966-6362(09)00161-1 [pii]10.1016/j.gaitpost.2009.06.005

Cole, C., Seto, C., & Gazewood, J. (2005). Plantar fasciitis: evidence-based review of diagnosis and therapy. *Am Fam Physician, 72*(11), 2237-2242.

Cornwall, M. W., & McPoil, T. G. (1999). Plantar fasciitis: etiology and treatment. *Journal of Orthopaedic and Sports Physical Therapy, 29*(12), 756-760.

Cornwall, M. W., & McPoil, T. G. (2002). Motion of the calcaneus, navicular, and first metatarsal during the stance phase of walking. *Journal of the American Podiatric Medical Association, 92*(2), 67-76.

Cosca, D. D., & Navazio, F. (2007). Common problems in endurance athletes. [Review]. *Am Fam Physician, 76*(2), 237-244.

Cowan, D. N., Robinson, J. R., Jones, B. H., Polly, D. W., Jr., & Berrey, B. H. (1994). Consistency of visual assessments of arch height among clinicians. *Foot and Ankle International, 15*(4), 213-217.

Cralley, J. C., Schuberth, J. M., & Fitch, K. L. (1982). The deep band of the plantar aponeurosis of the human foot. *Anat Anz, 152*(2), 189-197.

Crawford, F. (2005). Plantar heel pain and fasciitis. [Review]. *Clin Evid*(13), 1533-1545.

Crawford, F., & Thomson, C. (2003). Interventions for treating plantar heel pain. [Review]. *Cochrane Database Syst Rev*(3), CD000416. doi: 10.1002/14651858.CD000416

Creighton, D., & Olson, V. L. (1987). Evaluation of range of motion of the first metatarsophalangeal joint in runners with plantar faciitis*. *J Orthop Sports Phys Ther, 8*(7), 357-361.

Davies, M. S., Weiss, G. A., & Saxby, T. S. (1999). Plantar fasciitis: how successful is surgical intervention? *Foot Ankle Int, 20*(12), 803-807.

Davis, I. S., Zifchock, R. A., & Deleo, A. T. (2008). A comparison of rearfoot motion control and comfort between custom and semicustom foot orthotic devices. *Journal of the American Podiatric Medical Association, 98*(5), 394-403.

Davis, P. F., Severud, E., & Baxter, D. E. (1994). Painful heel syndrome: results of nonoperative treatment. *Foot and Ankle International, 15*(10), 531-535.

Dylevsky, I. (1988). Connective tissues of the hand and foot. [Review]. *Acta Univ Carol Med Monogr, 127*, 5-195.

Fabrikant, J. M., & Park, T. S. (2011). Plantar fasciitis (fasciosis) treatment outcome study: plantar fascia thickness measured by ultrasound and correlated with patient self-reported improvement. [Comparative Study]. *Foot (Edinb), 21*(2), 79-83. doi: 10.1016/j.foot.2011.01.015

Filippou, D. K., Kalliakmanis, A., Triga, A., Rizos, S., Grigoriadis, E., & Shipkov, C. D. (2004). Sport related plantar fasciitis. Current diagnostic and therapeutic advances. *Folia Med (Plovdiv), 46*(3), 56-60.

Gefen, A. (2003). The in vivo elastic properties of the plantar fascia during the contact phase of walking. [Research Support, Non-U.S. Gov't]. *Foot Ankle Int, 24*(3), 238-244.

Gibbon, W. W., & Long, G. (1999). Ultrasound of the plantar aponeurosis (fascia). *Skeletal Radiol, 28*(1), 21-26.

Glazer, J. L., & Hosey, R. G. (2004). Soft-tissue injuries of the lower extremity. *Prim Care, 31*(4), 1005-1024. doi: 10.1016/j.pop.2004.07.007

Grood, E. S., & Suntay, W. J. (1983). A joint coordinate system for the clinical description of three-dimensional motions: application to the knee. *Journal of Biomechanical Engineering, 105*(2), 136-144.

Hamel, A.J., Donahue, S.W., Sharkey, N.A. (2001). Contributions of active and passive toe flexion to forefoot loading. Clin Orthop, 393, 326-334.

Hicks, J. H. (1953). The mechanics of the foot I: The joints. *Journal of Anatomy, 87*, 345-357.

Hicks, J. H. (1954). The mechanics of the foot II. the plantar aponeurosis. *Journal of Anatomy, 88*, 25-30.

Hicks, J. H. (1955). The foot as a support. *Acta Anat (Basel), 25*(1), 34-45.

Hiramoto, Y. (1983). Shape of the fibular part of the plantar aponeurosis in Japanese. [Comparative Study]. *Okajimas Folia Anat Jpn, 60*(5), 329-337.

Huang, C. K., Kitaoka, H. B., An, K. N., & Chao, E. Y. S. (1993). Biomechanical evaluation of longitudinal arch stability. *Foot and Ankle, 14*(6), 353-357.

Hunt, A. E., Smith, R. M., & Torode, M. (2001). Extrinsic muscle activity, foot motion and ankle joint moments during the stance phase of walking. *Foot and Ankle International, 22*(1), 31-41.

Irving, D. B., Cook, J. L., Young, M. A., & Menz, H. B. (2008). Impact of chronic plantar heel pain on health-related quality of life. [Research Support, Non-U.S. Gov't]. *J Am Podiatr Med Assoc, 98*(4), 283-289.

Jenkyn, T. R., Anas, K., & Nichol, A. (2009). Foot segment kinematics during normal walking using a multisegment model of the foot and ankle complex. *Journal of Biomechanical Engineering, 131*(3), 034504. doi: 10.1115/1.2907750

Karabay, N., Toros, T., & Hurel, C. (2007). Ultrasonographic evaluation in plantar fasciitis. [Comparative Study]. *J Foot Ankle Surg, 46*(6), 442-446. doi: 10.1053/j.jfas.2007.08.006

Kayano, J. (1986). Dynamic function of medial foot arch. *J. Jpn. Orthop. Ass., 60*(11), 1147-1156.

Kepple, T. M., Stanhope, S. J., Lohmann, K. N., & Roman, N. L. (1990). A video-based technique for measuring ankle-subtalar motion during stance. *Journal of Biomechanical Engineering, 12*, 273-280.

Ker, R.F., Bennett, M.B., Bibby, S.R., Kester, R.C., Alexander, R.M. (1987). The spring in the arch ofthe human foot. Nature, 325, 147-149.

Kibler, W. B., Goldberg, C., & Chandler, T. J. (1991). Functional biomechanical deficits in running athletes with plantar fasciitis. [Comparative Study Research Support, Non-U.S. Gov't]. *Am J Sports Med, 19*(1), 66-71.

Kidder, S., Abuzzahab, F., Dow, A., Ortiz, T., Harris, G., & Johnson, J. (1996). Repeatability of kinematic data in normal foot and ankle motion. *Gait and Posture, 4*(2), 180-180.

Kim, W., Voloshin, A.S. (1995). Role of plantar fascia of the load bearing capacity of the human foot. J Biomech, 28, 1025-1033.

Kitaoka, H. B., Crevoisier, X. M., Hansen, D., Katajarvi, B., Harbst, K., & Kaufman, K. R. (2006). Foot and ankle kinematics and ground reaction forces during ambulation. *Foot and Ankle International, 27*(10), 808-813. doi: 942125 [pii]

Kitaoka, H. B., Luo, Z. P., & An, K. N. (1997). Mechanical behavior of the foot and ankle after plantar fascia release in the unstable foot. *Foot and Ankle International, 18*(1), 8-15.

Kogler, G. F., Solomonidis, S. E., & Paul, J. P. (1996). Biomechanics of longitudinal arch support mechanisms in foot orthoses and their effect on plantar aponeurosis strain. *Clin Biomech (Bristol, Avon), 11*(5), 243-252.

Krivickas, L. S. (1997). Anatomical factors associated with overuse sports injuries. *Sports Med, 24*(2), 132-146.

Labovitz, J. M., Yu, J., & Kim, C. (2011). The role of hamstring tightness in plantar fasciitis. *Foot Ankle Spec, 4*(3), 141-144. doi: 10.1177/1938640010397341

Lake, N.C. (1938). The foot. (2 ed.). London: Balliere, Tindall & Cox.

Leach, R. E., Seavey, M. S., & Salter, D. K. (1986). Results of surgery in athletes with plantar fasciitis. *Foot Ankle, 7*(3), 156-161.

Lemont, H., Ammirati, K. M., & Usen, N. (2003). Plantar fasciitis: a degenerative process (fasciosis) without inflammation. *J Am Podiatr Med Assoc, 93*(3), 234-237.

Lisowski, F.P. (2004). A Guide to Dissection of The Human Body. (2nd ed.). Singapore: World Scientific Publishing Company.

Lundgren, P., Nester, C., Liu, A., Arndt, A., Jones, R., Stacoff, A.,Lundgren, P., Nester, C., Liu, A., Arndt, A., Jones, R., Stacoff, A.,

Maffulli, N., Binfield, P. M., Moore, D., & King, J. B. (1999). Surgical decompression of chronic central core lesions of the achilles tendon. *The American Journal of Sports Medicine, 27*(6), 747-752.

Mann, R.A. (1992). Overview of Foot and Ankle Biomechanics, Disorders of the Foot and Ankle, M.H. Jahssm ed., W.B. Saunders, Philadelphia, 1, 385-408.

Marieb, E.N. (2001). Human Anatomy and Physiology. (5th ed). San Francisco: Benjamin Cummings.

McBryde, A. M., Jr. (1984). Plantar fasciitis. *Instr Course Lect, 33*, 278-282.
Messier, S. P., & Pittala, K. A. (1988). Etiologic factors associated with selected running injuries. *Med Sci Sports Exerc, 20*(5), 501-505.

Middleton, J. A., & Kolodin, E. L. (1992). Plantar fasciitis-heel pain in athletes. *J Athl Train, 27*(1), 70-75.

Michaud, T.C. (1997). Foot Orthoses and Other Forms of Conservative Foot Care. Massachusetts: Lippincott, Williams & Wilkins.

Mitchell, I. R., Meyer, C., & Krueger, W. A. (1991). Deep fascia of the foot. Anatomical and clinical considerations. [Review]. *J Am Podiatr Med Assoc, 81*(7), 373-378.

Moseley, L., Smith, R., Hunt, A., & Gant, R. (1996). Three-dimensional kinematics of the rearfoot during the stance phase of walking in normal young adult males. *Clinical Biomechanics, 11*(1), 39-45.

Myers, K. A., Wang, M., Marks, R. M., & Harris, G. F. (2004). Validation of a multisegment foot and ankle kinematic model for pediatric gait. *IEEE Trans Neural Syst Rehabil Eng, 12*(1), 122-130.

Nester, C. J., Liu, A. M., Ward, E., Howard, D., Cocheba, J., Derrick, T., & Patterson, P. (2007). In vitro study of foot kinematics using a dynamic walking cadaver model. *Journal of Biomechanics, 40*(9), 1927-1937.

Nordin, M., & Frankel, V. H. (2001). *Basic Biomechanics of the Musculoskeletal System* (3rd ed.). Philadelphia: Lippincott Williams & Wilkins.

O'Malley, M.J., Page, A., Cook, R. (2000). Endoscopic plantar fasciotomy for chronic heel pain. Foot Ankle Int, 21, 505-510.
Perry, J. (1992). *Gait Analysis: Normal and Pathological Function*. Thorofare, NJ: SLACK Incorporated.

Pohl, M. B., Hamill, J., & Davis, I. S. (2009). Biomechanical and anatomic factors associated with a history of plantar fasciitis in female runners. *Clin J Sport Med, 19*(5), 372-376. doi: 10.1097/JSM.0b013e3181b8c27000042752-200909000-00004 [pii]

Pontious, J., Flanigan, K. P., & Hillstrom, H. J. (1996). Role of the plantar fascia in digital stabilization. A case report. *Journal of the American Podiatric Medical Association, 86*(1), 43-47.

Prichasuk, S. (1994). The heel pad in plantar heel pain. *Journal of Bone and Joint Surgery.British Volume, 76*(1), 140-142.

Rankine, L., Long, J., Canseco, K., & Harris, G. F. (2008). Multisegmental foot modeling: a review. *Critical Reviews in Biomedical Engineering, 36*(2-3), 127-181. doi: 19160f1c0f24e5ff,40023b6346865895 [pii]

Rathleff, M. S., Moelgaard, C., & Olesen, J. L. (2011). Intra- and Interobserver reliability of quantitative ultrasound measurement of the plantar fascia. *Journal of Clinical Ultrasound, 39*, 128-134.

Reeser, L.A., Susman, R.L., Stem, J.T., Jr. (1983). Electromyographic studies of the human foot: experimental approaches to hominid evolution. Foot Ankle, 3, 391-407.

Ribeiro, A. P., Trombini-Souza, F., Tessutti, V., Rodrigues Lima, F., Sacco Ide, C., & Joao, S. M. (2011). Rearfoot alignment and medial longitudinal arch configurations of runners with symptoms and histories of plantar fasciitis. [Evaluation Studies Research Support, Non-U.S. Gov't]. *Clinics (Sao Paulo), 66*(6), 1027-1033.

Riddle, D. L., Pulisic, M., Pidcoe, P., & Johnson, R. E. (2003). Risk factors for Plantar fasciitis: a matched case-control study. *J Bone Joint Surg Am, 85-A*(5), 872-877.

Riddle, D. L., & Schappert, S. M. (2004). Volume of ambulatory care visits and patterns of care for patients diagnosed with plantar fasciitis: a national study of medical doctors. *Foot Ankle Int, 25*(5), 303-310.

Rome, K., Howe, T., & Haslock, I. (2001). Risk factors associated with the development of plantar heel pain in athletes. *The Foot, 11*, 119-125.

Roos, E., Engstrom, M., & Soderberg, B. (2006). Foot orthoses for the treatment of plantar fasciitis. *Foot and Ankle International, 27*(8), 606-611.

Roxas, M. (2005). Plantar fasciitis: diagnosis and therapeutic considerations. *Altern.Med Rev., 10*(2), 83-93.

Roy, T. C. (2011). Diagnoses and mechanisms of musculoskeletal injuries in an infantry brigade combat team deployed to Afghanistan evaluated by the brigade physical therapist. [Research Support, U.S. Gov't, Non-P.H.S.]. *Mil Med, 176*(8), 903-908.

Rush, S.M., Christensen, J.C., Johnson, C.H. (2000). Biomechanics of the first ray, part 2: metatarsus primus varus as a cause of hypermobility – a three dimensional kinematic analysis in a cadaver model. J Foot Ankle Surg, 39, 68-75.

Sabir, N., Demirlenk, S., Yagci, B., Karabulut, N., & Cubukcu, S. (2005). Clinical utility of sonography in diagnosing plantar fasciitis. *J Ultrasound Med, 24*(8), 1041-1048.

Sahin, N., Ozturk, A., & Atici, T. (2010). Foot mobility and plantar fascia elasticity in patients with plantar fasciitis. [Comparative Study]. *Acta Orthop Traumatol Turc, 44*(5), 385-391. doi: 10.3944/AOTT.2010.2348

Salathe, E. P., & Arangio, G. A. (2002). A biomechanical model of the foot: the role of muscles, tendons, and ligaments. [Comparative Study]. *J Biomech Eng, 124*(3), 281-287.

Salathe, E. P., Jr., Arangio, G. A., & Salathe, E. P. (1986). A biomechanical model of the foot. *J Biomech, 19*(12), 989-1001.

Sarrafian, S. K. (1987). Functional characteristics of the foot and plantar aponeurosis under tibiotalar loading. *Foot and Ankle, 8*(1), 4-18.

Schepsis, A. A., Leach, R. E., & Gorzyca, J. (1991). Plantar fasciitis. Etiology, treatment, surgical results, and review of the literature. *Clin Orthop Relat Res*(266), 185-196.

Scher, D. L., Belmont, P. J., Jr., Bear, R., Mountcastle, S. B., Orr, J. D., & Owens, B. D. (2009). The incidence of plantar fasciitis in the United States military. *J Bone Joint Surg Am, 91*(12), 2867-2872. doi: 10.2106/JBJS.I.00257

Shama, S. S., Kominsky, S. J., & Lemont, H. (1983). Prevalence of non-painful heel spur and its relation to postural foot position. *J Am Podiatry Assoc, 73*(3), 122-123.

Sharkey, N. A., Ferris, L., & Donahue, S. W. (1998). Biomechanical consequences of plantar fascial release or rupture during gait: part I--disruptions in longitudinal arch conformation. *Foot Ankle Int, 19*(12), 812-820.

Singh, D., Angel, J., Bentley, G., & Trevino, S. G. (1997). Fortnightly review. Plantar fasciitis. [Review]. *BMJ, 315*(7101), 172-175.

Subotnick, S. I. (1985). The biomechanics of running: implications for the prevention of foot injuries. *Sports Medicine, 2*, 144-153.

Tansey, P.A., Briggs, P.J. (2001). Active and passive mechanisms in the control of heel supination. J Foot Ankle Surg, 7, 131-136.

Taunton, J. E., Clement, D. B., & McNicol, K. (1982). Plantar fasciitis in runners. *Can J Appl Sport Sci, 7*(1), 41-44.

Taunton, J. E., Ryan, M. B., Clement, D. B., McKenzie, D. C., & Lloyd-Smith, D. R. (2002). Plantar fasciitis: a retrospective analysis of 267 cases. *Physical Therapy in Sport, 3* 57-65.

Taunton, J. E., Ryan, M. B., Clement, D. B., McKenzie, D. C., Lloyd-Smith, D. R., & Zumbo, B. D. (2002). A retrospective case-control analysis of 2002 running injuries. *British Journal of Sports Medicine, 36*(2), 95-101.

Tountas, A. A., & Fornasier, V. L. (1996). Operative treatment of subcalcaneal pain. *Clin Orthop Relat Res*(332), 170-178.

Tweed, J. L., Barnes, M. R., & Allen, M. J. (2009). An evaluation of the long-term effects of total plantar fasciotomy--a preliminary study. *Foot (Edinb), 19*(2), 75-79. doi: 10.1016/j.foot.2008.11.014

Uden, H., Boesch, E., & Kumar, S. (2011). Plantar fasciitis - to jab or to support? A systematic review of the current best evidence. *J Multidiscip Healthc, 4*, 155-164. doi: 10.2147/JMDH.S20053

Vedi, V., Williams, A., Singh, D., et al. (1999). Hick's revisited: a weightbearing in vivo study of the biomechanics of the plantar

Vogler, H. W., & Bojsen-Moller, F. (2000). Tarsal functions, movement, and stabilization mechanisms in foot, ankle, and leg performance. *Journal of the American Podiatric Medical Association, 90* (3), 112-125.

Warren, B. L. (1984). Anatomical factors associated with predicting plantar fasciitis in long-distance runners. *Med Sci Sports Exerc, 16*(1), 60-63.

Warren, B. L., & Jones, C. J. (1987). Predicting plantar fasciitis in runners. *Medicine and Science in Sports and Exercise, 19*(1), 71-73.

Wearing, S. C., Smeathers, J. E., Sullivan, P. M., Yates, B., Urry, S. R., & Dubois, P. (2007). Plantar fasciitis: are pain and fascial thickness associated with arch shape and loading? *Phys Ther, 87*(8), 1002-1008. doi: ptj.20060136 [pii] 10.2522/ptj.20060136

Wearing, S. C., Smeathers, J. E., Urry, S. R., Hennig, E. M., & Hills, A. P. (2006). The pathomechanics of plantar fasciitis. *Sports Med, 36*(7), 585-611. doi: 3674 [pii]

Wearing, S. C., Smeathers, J. E., Yates, B., Sullivan, P. M., Urry, S. R., & Dubois, P. (2004). Sagittal movement of the medial longitudinal arch is unchanged in plantar fasciitis. *Med Sci Sports Exerc, 36*(10), 1761-1767.

Williams, A., Vedi, V., Singh, D., et al. (1999). Hick's revisited: a weightbearing in vivo study of the biomechanics of the plantar fascia employing dynamic M.R.I. J Bone Joint Surg Br, 81, 381.

Williams, D. S., McClay, I. S., & Hamill, J. (2001). Arch structure and injury pattern in runners. *Clinical Biomechanics, 16*, 341-347.

Wolf, P., Stacoff, A., Liu, A., Nester, C., Arndt, A., Lundberg, A., & Stuessi, E. (2008). Functional units of the human foot. *Gait and Posture, 28*(3), 434-441.

Wolgin, M., Cook, C., Graham, C., & Mauldin, D. (1994). Conservative treatment of plantar heel pain: long-term follow-up. *Foot and Ankle, 15*(3), 97-102.

Wright, D. G., & Rennels, D. C. (1964). A Study of the Elastic Properties of Plantar Fascia. *J Bone Joint Surg Am, 46*, 482-492.

Wu, C. H., Chang, K. V., Mio, S., Chen, W. S., & Wang, T. G. (2011). Sonoelastography of the plantar fascia. *Radiology, 259*(2), 502-507. doi: radiol.11101665 [pii] 10.1148/radiol.11101665

Young, C. C., Rutherford, D. S., & Niedfeldt, M. W. (2001). Treatment of plantar fasciitis. [Review]. *Am Fam Physician, 63*(3), 467-474, 477-468.

Appendix A

DO YOU HAVE HEEL PAIN?

Do you run?

University of Wisconsin-Milwaukee
Musculoskeletal Injury Biomechanics Laboratory, Enderis 132

Purpose: **To investigate foot kinematics in runners with plantar fasciitis versus injury-free runners.**

Who can participate?
1. Runners (minimum of 10 miles/week)
2. Ages 18-45
3. No major surgery to the lower extremity
4. Must not be pregnant
5. Currently have heel pain

What will I do?

- *Initial Phone Screening*: Questions regarding study qualifications (~10 min)

- *Visit 1*: Flexibility measurements, Running gait analysis (~1.5 hrs)

- *Visit 2*: Foot structure assessment, Ultrasound images of the foot (~45 min)

Do I get paid?
☐ **YES!** You will receive **$40.00** in gift cards**!**

Interested? Contact:
Principal Investigator
Robin Bauer, BA
heel-pain@uwm.edu
414-229-5147

Co-Investigator
Stephen Cobb, PhD, ATC, CSCS

This research project has been approved by the University of Wisconsin-Milwaukee Institutional Review Board for the Protection of Human Subjects (IRB Protocol Number 12-229, approved on February 16,2012).

Appendix B

DO YOU RUN?

University of Wisconsin-Milwaukee
Musculoskeletal Injury Biomechanics Laboratory, Enderis 132

Purpose: To investigate foot kinematics in runners with plantar fasciitis versus injury-free runners.

<u>Who can participate?</u>
6. Runners (minimum of 10 miles/week)
7. Ages 18-45
8. No major surgery to the lower extremity
9. Must not be pregnant
10. Currently injury-free

<u>What will I do?</u>
- *Initial Phone Screening*: Questions regarding study qualifications (10 min)
- *Visit 1*: Flexibility measurements, Running gait analysis (~1.5 hrs)
- *Visit 2*: Foot structure assessment, Ultrasound images of the foot (~45 min)

Do I get paid?
☐ **YES!** You will receive **$40.00 in gift cards!**

Interested? <u>Contact:</u>
Principal Investigator   *Co-Investigator*
Robin Bauer, BA   Stephen Cobb, PhD, ATC, CSCS
heel-pain@uwm.edu
414-229-5147

This research project has been approved by the University of Wisconsin-Milwaukee Institutional Review Board for the Protection of Human Subjects (IRB Protocol Number <u>12-229</u>, approved on <u>February 16,2012</u>).

Reliability Code: _____   IRB Protocol Number: 12-229

Date: _____   IRB Approval Date: 02/16/2012

Appendix C

Overread For Plantar Fascia Imaging

Study Title: "The Effects of Plantar Fasciitis on Multi-segment Foot Running Gait Kinematics"

Right Foot

Parameter	Yes	No
1. Correct study subject ID entered		
2. Correct Preset Selected		
3. Gain settings adjusted appropriately		
4. Depth setting at 2.5 cm		
5. Plantar Fascia is demonstrate longitudinally		
6. Plantar Fascia is imaged perpendicular to ultrasound beam		
7. Plantar Fascia borders demonstrated clearly		
8. DICOM file saved and backed up per study guidelines		
9. Measurement made in triplicate		
10. All measures are with one digital pixel		
Quality Score: _____/10 = _____%		
Pass		

Left Foot

Parameter	Yes	No
1. Correct study subject ID entered		
2. Correct Preset Selected		
3. Gain settings adjusted appropriately		
4. Depth setting at 2.5 cm		
5. Plantar Fasica is demonstrate longitudinally		
6. Plantar Fasica is imaged perpendicular to ultrasound beam		
7. Plantar Facia borders demonstrated clearly		
8. Dicom file saved and backed up per study guidelines		
9. Measurement made in triplicate		
10. All measures are with one digital pixel		
Quality Score: _____/10 = _____%		
Pass		

16-March-2012

Informed Consent

Version:

Appendix D

UNIVERSITY OF WISCONSIN – MILWAUKEE
CONSENT TO PARTICIPATE IN RESEARCH

THIS CONSENT FORM HAS BEEN APPROVED BY THE IRB FOR A ONE YEAR PERIOD

1. General Information

Study title: The effects of plantar fasciitis on multi-segment foot running gait kinematics

Person in Charge of Study (Principal Investigator): The student Principle Investigator (SPI) for this study is Robin Bauer, B.A. Robin Bauer is a graduate student in the Department of Kinesiology, with an emphasis in Biomechanics, working on her Thesis project. She is under the supervision of Stephen Cobb, PhD, ATC, CSCS, the Principle Investigator (PI) of the study. Dr. Cobb is a faculty member in the Department of Kinesiology.

2. Study Description

You are being asked to participate in a research study. Your participation is completely voluntary. You do not have to participate if you do not want to.

Study description:
The primary purpose of this study is to compare running kinematics in runners with plantar fasciitis and injury-free runners using a multi-segment foot model. The secondary purpose is to investigate the relationship between plantar fascia thickness and the clinical signs and symptoms of plantar fasciitis in runners.

The results of the proposed study will enhance understanding of the mechanical effects of plantar fasciitis on running gait mechanics. Specifically use of the multi-segment foot model will allow identification of potential changes in the running mechanics of the different segments of the foot in individuals with plantar fasciitis. This study will also expand upon the proposed relationship between ultrasound assessment of plantar fascia thickness and the clinical signs and symptoms of plantar fasciitis in runners. Moreover, it may contribute to the understanding of the etiology of plantar fasciitis and to the development of more effective treatment protocols.

Initial participant screening will occur over the phone. Following the initial phone screen, the testing sessions and data collection will be conducted at the University of Wisconsin-Milwaukee in the Musculoskeletal Injury Biomechanics Laboratory (Enderis 132) and the Physical Activity and Health Research Lab Enderis 434). 30 individuals (age 18 – 45 years) will participate in this study, and will be recruited from the University, surrounding community, and local fitness clubs and medical clinics (i.e. podiatry clinics).

As a participant in this study, you will be asked to first complete an initial phone screen (~ 10 min). If you qualify, you will be asked to attend 2 testing sessions: the first testing session will be approximately 45 minutes and the second session will last about 1.5 hours.

3. Study Procedures

What will I be asked to do if I participate in the study?

If you agree to participate you will be asked to provide a telephone number at which you can be reached to complete a 10 minute initial screening. If you qualify, you will be asked to report to the Musculoskeletal Injury Biomechanics Laboratory (Enderis 132) for testing. All procedures and measurements involved in the testing session will be performed by the SPI or PI.

INITIAL SCREENING: PHONE ASSESSMENT (All potential participants)(~10 min)

- The Screening and Medical History Questionnaire for potential plantar fasciitis group participants will include questions pertaining to clinical signs and symptoms plantar fasciitis. Additionally, all participants will be asked questions pertaining to your physical activity level and previous lower body injury(ies) and surgeries, pregnancy, and presence of diseases/illness that may exclude participation.

VISIT ONE: (~ 45 minutes)

1. Informed Consent Process (All participants)

☐ If you agree to participate in the study, you will be asked to complete the following.

2. Physical Exam (~5 minutes) (Only plantar fasciitis group participants)

☐ This exam will be performed if you complain of heel pain.

☐ A Certified Athletic Trainer will perform a physical examination that includes palpating the proximal insertion of the plantar fascia (located on the inside part of your heel), as well as extending the first metatarsophalangeal joint (joint of big toe). Other potential sources of heel pain will be ruled out through muscle testing and palpation of relevant structures in the foot. Both of these exams may elicit moderate pain.

☐ You will be asked to rate your pain level on a scale from 0 to 10, with 0 indicating "no pain" and 10 indicating "worst pain ever."

☐ If you qualify as having plantar fasciitis, you will be asked to continue to the testing session.

3. Weight and height measurements (All participants)(~1 minute)

4. Foot Structure Assessment (All participants) (~10 minutes)

☐ This includes measurements of your feet. These will require small marks to be placed on your skin over specific bony landmarks with a pen or washable marker. The marks will be used to obtain measurements of your foot that will be used to

determine your foot structure (i.e. flat foot, high arch). A digital photograph will be taken of both feet with 10% of your body weight on the foot to be measured. Measurements will be taken from the digital pictures to determine your foot structure. This photograph will not have any of your identifiers attached to it, so individuals who see it will not know that it is your foot. If you choose not to have your foot photographed, you may not participate in the study.

5. *Ultrasound Assessment (All participants) (~15 minutes)*

   ☐ Ultrasound assessment will take place in the Physical Activity and Health Research Lab, in Enderis Hall, Room 434. Three successive ultrasound images of both feet will be captured. You will lie in a prone position with your ankle in a neutral position and toes extended to near end range. The thickness of the proximal attachment of your right and left plantar fascia will be imaged using an ultrasound machine. The SPI will apply ultrasonic gel to the transducer and to your skin and capture images of your feet. The procedure will be repeated until three successful images have been collected. A successful image will be based on the measurement of the thickness of the plantar fascia. If you choose not to have your foot imaged, you will not be able to participate in the study.

VISIT TWO: (~ 1 hour and 15 minutes)

1. Ultrasound Assessment *(All participants)* (~15 minutes)
    1. You may be randomly selected to have your foot imaged a second time. 20% of the participants will be randomly selected in order to test the reliability of the examiner.
    2. The ultrasound protocol will consist of the same procedures as Visit One.

2. *Ankle and Toe Flexibility Assessment (All participants) (~15 minutes)*

    ☐Ankle range of motion will be assessed first. You will be seated on a bench, with your leg hanging over the table. Your knee will be flexed at 90º and the ankle will be in a neutral position. The PI will stabilize your tibia and fibula, then use one hand to move the foot into end range dorsiflexion by pushing on the bottom of your foot. The SPI will then use a goniometer to measure your ankle dorsiflexion range of motion. This will be repeated 3 times.

    ☐First metatarsophalangeal (MTP) joint (big toe) range of motion for each foot will be measured next. Your knee and ankle will be positioned in the same starting position as that for ankle range of motion. The SPI will position your first MTP joint in a neutral position, stabilize your foot, and then push your big toe toward the top of your foot until the end range of motion is felt. The SPI will use a goniometer to measure the joint. This will be repeated 3 times.

    ☐Gastrocnemius (calf) muscle length will be measured next. You will lay supine with your knee fully extended and foot in a neutral position. The SPI will place pressure on the front portion of your leg to maintain an extended knee position, and then flex your ankle to the end of the range of motion by pushing upward across the bottom surface of the foot. The SPI will then measure the ankle position with a goniometer. This will be repeated 3 times.

3. *Gait Analysis (All participants) (~45 minutes)*
    • The running gait analysis session will consist of 10 successful running gait trials along a runway with a force plate (a device used to measure the forces between the ground and your foot) mounted in the middle of the runway. You will perform the

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running trials at a speed of 4.0 m/s ± 10% (8.96 mph ± 10%) wearing a sandal with no support.

- During the gait trials, you will have groups of small reflective markers located on your legs and feet. The markers will be placed directly on your skin or on custom built wands that will be placed on your skin. The markers and wands will be secured to your skin using double sided adhesive tape and a liquid adhesive. The position of the reflective markers during the gait trials will be recorded using a 10 camera Motion Analysis system. The Motion Analysis System will record the position of the reflective markers on your legs and feet, but will not record any images of your person. If you choose not to be recorded during the gait trials, you will not be eligible to complete the study.

- Prior to performing the gait trials, additional reflective markers will be located on specific bony landmarks on your legs and feet while you are in a seated position. The position of the additional markers will be recorded and then the markers will be removed before you complete the gait trials.

- If you are in the plantar fasciitis group, you will be asked to report your pain level after each trial based on the scale previously described.

4. Risks and Minimizing Risks

What risks will I face by participating in this study?
The potential risks other than muscle soreness or tightness for your participation in this research study are minimal.

Physical Risks:

Likely:
☐ Minor muscle soreness and/or tightness (< 30% of participants).

Less Likely:
☐ Musculoskeletal injury such as muscle strain (< 2%)
☐ Allergic reaction to the liquid adhesive used to secure the reflective markers (< 2%)

Protection of Physical Risks:

To reduce the above risks, appropriate warm-up has been incorporated before the running gait trials. If you feel any soreness or irritation while participating in this study, please tell the investigators as soon as possible. If you are injured, experience allergic reaction to the liquid adhesive used to secure the reflective markers, or experience shortness of breath while participating in this research study, initial first aid and/or appropriate emergency measures will be provided/initiated by the Principal Investigator, who is a Licensed Athletic Trainer. If you are a UWM student you will be referred to the Norris Health Center for follow-up care. Non-students will be referred to their primary care physician and will be responsible for all expenses incurred.

Risks to Privacy and Confidentiality:

Less Likely:

- Since a photograph and an ultrasound image will be taken of your foot, this might increase risks to your privacy (less than 1%).
- Since your private information will be collected for this study, there is always a risk of breach of confidentiality (less than 1%)

<u>Protection of Risks to Privacy and Confidentiality</u>:

All data will be stored in a locked filing cabinet in a locked room. All data will be given a letter and number that is uniquely associated with you. This code will not contain any partial identifiers (i.e. last four digits of your SSN) and will be stored in a separate locked office in a locked filing cabinet. No identifiers will be stored with the research data. Only those individuals with an active role in this study will have access to the research data and only the SPI and PI will have access to identifying information. When all participants' have completed active participation in the study and data collection is completed, the code will be destroyed. All appropriate measures to protect your private information will be taken.

In the event of an incidental finding (i.e. tumor) while imaging the foot, you have the option to be notified by phone and encouraged to follow-up with your physician. The researchers are not health care providers who have appropriate expertise to make a diagnosis based on ultrasound imaging. You also have the option to include your primary care physician name and contact information on the HIPAA form to allow us to follow-up with your primary care physician.

5. Benefits

Will I receive any benefit from my participation in this study?
  ☐ There are no benefits to you other than to further research.

6. Study Costs and Compensation

Will I be charged anything for participating in this study?
  1. You will not be responsible for any of the costs associated with this research study.

Are subjects paid or given anything for being in the study?
  1. You will not receive payment for participating in either the phone screen and/or Visit One.
  2. If following the phone screen and Visit One you qualify for participation in the study, you will receive $40.00 in gift cards upon successful completion of Visit Two.

7. Confidentiality

What happens to the information collected?
All information collected about you during the course of this study will be kept confidential to the extent permitted by law. We may decide to present what we find to others, or publish our results in scientific journals or at scientific conferences. Information that identifies you personally will not be released without your written permission. Only the SPI, PI, and limited Musculoskeletal Injury Biomechanics Laboratory personnel will have access to the information. However, the Institutional Review Board at UW-Milwaukee or appropriate federal agencies like the Office for Human Research Protections may review this study's records.

The confidentiality of your data and information will be safeguarded as outlined in "Risks & Minimizing Risks" section under the "Protection of Risks to Privacy and Confidentiality" header.

8. Alternatives

Are there alternatives to participating in the study?

There are no alternatives to participating in this research study. You may choose not to participate.

9. Voluntary Participation and Withdrawal

What happens if I decide not to be in this study?
Your participation in this study is entirely voluntary. You may choose not to take part in this study. If you decide to take part, you can change your mind later and withdraw from the study. You are free to not answer any questions or withdraw at any time. Your decision will not change any present or future relationships with the University of Wisconsin-Milwaukee.

If you withdraw from this study before completing the second testing session, we will destroy all information we collect about you. Your decision not to participate or to withdraw early will not result in penalty or harm, nor will it affect your grade or class standing.

10. Questions

Who do I contact for questions about this study?
For more information about the study or the study procedures or treatments, or to withdraw from the study, contact:

> Robin Bauer, BA, Student Principal Investigator
> Department of Kinesiology
> PO Box 413
> Milwaukee, WI 53201

Informed Consent

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(414) 229-5147

Stephen Cobb, PhD, LAT, CSCS, Principal Investigator
Athletic Training Education Program
Department of Kinesiology
PO Box 413
Milwaukee, WI 53201
(414) 229-3369

Who do I contact for questions about my rights or complaints towards my treatment as a research subject?
The Institutional Review Board may ask your name, but all complaints are kept in confidence.

Institutional Review Board
Human Research Protection Program
Department of University Safety and Assurances
University of Wisconsin – Milwaukee
P.O. Box 413
Milwaukee, WI 53201
(414) 229-3173

11. Signatures

Research Subject's Consent to Participate in Research:
To voluntarily agree to take part in this study, you must sign on the line below. If you choose to take part in this study, you may withdraw at any time. You are not giving up any of your legal rights by signing this form. Your signature below indicates that you have read or had read to you this entire consent form, including the risks and benefits, and have had all of your questions answered, and that you are 18 years of age or older.

_____
Printed Name of Subject/ Legally Authorized Representative

_____   _____
Signature of Subject/Legally Authorized Representative   Date

Research Subject's Consent to Audio/Video/Photo Recording:

It is okay to photograph my foot while I am in this study and use my photographed data in the research.
Please initial: _____Yes   _____No

It is okay to ultrasound my feet while I am in this study and use my photographed data in the research.
Please initial: _____Yes   _____No

Informed Consent

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It is possible the ultrasound may have incidental findings such as a tumor. In the case of an incidental finding, we will contact you by phone. However, since the researchers are not health care providers who have appropriate expertise, you will be encouraged to follow up with your physician.

Please initial:

_____ Please do not notify me of any incidental findings obtained from this research.

_____ Please ask me at the time of notification whether or not I want to receive incidental findings information.

Principal Investigator (or Designee)

I have given this research subject information on the study that is accurate and sufficient for the subject to fully understand the nature, risks and benefits of the study.

_____   _____

Printed Name of Person Obtaining Consent   Study Role

_____   _____

Signature of Person Obtaining Consent   Date

Appendix E

Demographic Information

Gender: _____

Age: _____

Height: _____

Weight: _____

BMI: _____

Screening & Medical History Questionnaire

Screening Criteria

Please answer the following questions to the best of your ability:

Yes   No   Are you between the ages of 18 and 45 years old?

Medical History Questionnaire

For your safety, a list of conditions that would make you unable to participate in this study has been prepared. Please read this list carefully and consider whether any of the conditions apply to you. If any of these conditions are true for you, you will not be able to participate in this study. For each condition, please indicate "yes" or "no" if this is true or not for you.

1. How many miles/week do you run?

   0-9
   10-20   How long have you been running this mileage? _____
   20-30   How long have you been running this mileage? _____
   ≥30   How long have you been running this mileage? _____

2.   Yes   No   Do you suffer from heel pain?
                       If no, **continue to question 3.**
                       If yes, **continue with the following questions then skip to question 6**.

     How long have you had heel pain? _____

          Yes   No   Do you have heel pain with your first few steps in the morning?

                                  On a scale from 0 to 10, with 0 indicating "no pain" and 10 indicating "worst pain ever", rate your pain level ____

          Yes   No   Do you have heel pain with the first few steps after prolonged periods of inactivity?

                                  On a scale from 0 to 10, with 0 indicating "no pain" and 10 indicating "worst pain ever", rate your pain level ____

Yes   No   Is the area where you have heel pain tender to touch?

Yes   No   Does your heel pain decrease with normal walking?

Yes   No   Does your heel pain increase during periods of
                        prolonged physical activity?

Yes   No   Have you received or are you currently receiving
                        treatment for your heel pain?

                        If yes, please indicate the *type* and *length* of treatment

Yes   No   Plantar fascia stretching   Length of treatment_____
Yes   No   Calf stretching   Length of treatment_____
Yes   No   Low-Dye taping   Length of treatment_____
Yes   No   Orthotics   Length of treatment_____
Yes   No   Extracorporeal shockwave therapy
                                            Length of treatment_____
Yes   No   Iontophoresis   Length of treatment_____
Yes   No   Surgery   Length of treatment_____
Yes   No   Other treatment. Please describe:
_____
_____

3.   Yes   No   Have you ever been diagnosed with plantar fasciitis? If so, when?
                     _____

4.   Yes   No   Do you have any current lower extremity injuries? If yes, please
                     describe: _____

5.   Yes   No   Have you had any lower extremity injuries in the last 6 months? If yes,
                     please describe: _____

6.   Yes   No   Have you ever had lower extremity surgery? If yes, please describe:
                     _____

7.   Yes   No   N/A Are you pregnant?

8.   Yes   No   Do you have a history of inflammatory or connective tissue disease (i.e.,
                     osteoarthritis, rheumatoid arthritis, Marfan Syndrome)? If yes, please
                     describe: _____

9.   Yes   No   Do you have a history any previous trauma or injury to the foot/heel? If
                     yes, please describe:_____

10. Yes   No   Do you have any diagnosed foot deformity (i.e. hallux valgus)?

11. Yes   No   Do you have a history of a neurologic systemic disorder?

11. Yes   No   Do you have a history of diabetes?

Participant Code: _____   IRB Protocol Number: <u>12-229</u>
Date: _____   IRB Approval Date: <u>02/16/2012</u>

12.   Yes   No   Do you have a buddy who you run with and can refer to us for
                        participation in this study?

Comments/Notes:

Appendix F

Patient Hx

☐ Primary c/o:

☐ Sx location

☐ Pain characteristics

SYMPTOMS

☐ VAS score (0-10)

☐ Functional disability

☐ Sx duration

☐ Sx progression

☐ Mechanism of injury

☐ Sx constancy

☐ Affecting factors

☐ Neurological Sxs

☐ Joint locking/catching/instability

☐ Current/previous treatment

☐ Current/previous medications

☐ Night pain (if indicated)

Adapted from: Magee DJ. *Orthopedic Physical Assessment*. 5th ed. Philadelphia: Saunders; 2008.
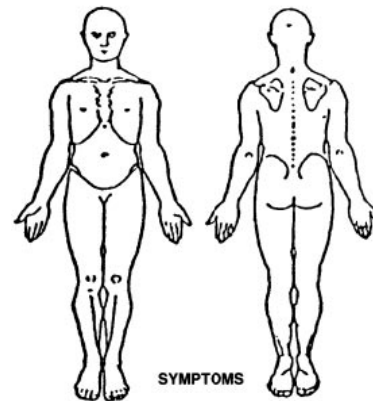
Participant Code: _____   **Ankle & Foot Physical Exam**   IRB Protocol Number: <u>12-229</u>

Date: _____   IRB Approval Date: <u>02/16/2012</u>

Objective
    ☐Observation (Signs of inflammation, symmetry, posture)

Assessment
☐   Scanning exam (*Lumbar spine if indicated)*

☐   Examination of movement
    ☐ AROM

    ☐ PROM

    ☐ Resisted Isometric movements

☐   Special tests *(as indicated)*

☐   Neurological evaluation *(if indicated)*

☐   Palpation*

Impression:

Adapted from: Magee DJ. *Orthopedic Physical Assessment*. 5th ed. Philadelphia: Saunders; 2008.

Appendix G

Case Report Form (CRF)

Date: ▭▭▭▭▭-▭▭▭▭▭-▭▭▭▭▭▭▭
       DD   MM   YYYY

Subject ID Number: _____-_____-00000000

1.	MSK Preset Selected	Y	N
2.	Right Plantar Fascia Imaged	Y	N
3.	Left Plantar Fascia Imaged	Y	N

Comments

Measurements:

Side	Measure 1 (cm)	Measure 2 (cm)	Measure 3 (cm)
Right Plantar Fascia			
Left Plantar Fascia			

Comments

Appendix H

Table 3. Phase 1 Mean (SD) of functional articulations.

Functional articulation	Plane	Motion	Group Mean (SD)	
			Plantar Fasciitis	**Control**
Rearfoot Complex	Sagittal	Dorsiflexion	3.38 (3.21)	2.37 (4.08)
		Plantarflexion	4.30 (2.86)	3.73 (3.51)
	Frontal	Inversion	0.02 (0.06)	0.18 (0.70)
		Eversion	7.39 (3.12)	5.92 (2.52)
	Transverse	Adduction	4.56 (2.84)	3.93 (1.73)
		Abduction	0.15 (0.27)	0.27 (0.90)
Calcaneocuboid Complex	Sagittal	Dorsiflexion	4.44 (3.74)	4.91 (2.73)
		Plantarflexion	0.23 (0.59)	0.00 (0.00)
	Frontal	Inversion	0.72 (0.84)	0.80 (0.95)
		Eversion	3.63 (2.73)	1.16 (1.14)
	Transverse	Adduction	3.63 (1.89)	3.28 (2.14)
		Abduction	0.53 (0.65)	0.17 (0.63)
Calcaneonavicular Complex	Sagittal	Dorsiflexion	2.44 (1.98)	2.86 (1.83)
		Plantarflexion	0.48 (1.00)	0.39 (1.26)
	Frontal	Inversion	2.55 (2.09)	1.93 (1.81)
		Eversion	0.66 (1.19)	0.53 (1.05)
	Transverse	Adduction	2.45 (2.57)	1.82 (1.39)
		Abduction	1.11 (1.82)	0.26 (0.48)
Medial Forefoot	Sagittal	Dorsiflexion	5.82 (5.76)	4.75 (3.15)
		Plantarflexion	0.76 (1.28)	0.38 (0.80)
Lateral Forefoot	Sagittal	Dorsiflexion	3.87 (2.45)	2.56 (2.50)
		Plantarflexion	0.37 (0.60)	0.64 (1.26)
First Metatarsophalangeal Complex	Sagittal	Extension	1.29 (1.55)	0.52 (1.07)
		Flexion	10.27 (7.04)	9.25 (5.62)

Table 4. Phase 2 Mean (SD) of functional articulations.

Functional Articulation	Plane	Motion	Group Mean (SD)	
			Plantar Fasciitis	Control
Rearfoot complex	Sagittal	Dorsiflexion	10.35 (2.56)	10.82 (2.63)
		Plantarflexion	0.00 (0.02)	0.00 (0.00)
	Frontal	Inversion	1.41 (1.32)	1.36 (0.78)
		Eversion	1.53 (1.39)	1.34 (0.75)
	Transverse	Adduction	3.35 (2.13)	3.22 (3.44)
		Abduction	0.49 (0.59)	0.65 (0.66)
Calcaneocuboid Complex	Sagittal	Dorsiflexion	2.90 (2.26)	2.46 (1.64)
		Plantarflexion	0.49 (0.76)	0.37 (0.58)
	Frontal	Inversion	1.94 (1.77)	1.82 (1.07)
		Eversion	1.24 (1.57)	0.40 (0.72)
	Transverse	Adduction	2.03 (1.15)	1.15 (1.43)
		Abduction	0.98 (1.13)	1.55 (1.47)
Calcaneonavicular Complex	Sagittal	Dorsiflexion	1.78 (1.52)	1.38 (1.18)
		Plantarflexion	0.68 (0.88)	0.57 (0.72)
	Frontal	Inversion	1.47 (2.53)	0.45 (0.68)
		Eversion	1.62 (1.76)	1.60 (0.99)
	Transverse	Adduction	1.65 (1.40)	1.41 (0.87)
		Abduction	0.74 (0.88)	0.45 (0.54)
Medial Forefoot	Sagittal	Dorsiflexion	6.54 (2.88)	6.00 (2.06)
		Plantarflexion	0.08 (0.12)	0.64 (0.18)
Lateral Forefoot	Sagittal	Dorsiflexion	3.50 (1.74)	2.99 (1.33)
		Plantarflexion	0.22 (0.67)	0.13 (0.20)
First Metatarsophalangeal Complex	Sagittal	Extension	1.76 (1.72)	1.91 (1.56)
		Flexion	7.04 (6.31)	5.56 (4.28)

Table 5. Phase 3 Mean (SD) of functional articulations.

Functional Articulation	Plane	Motion	Group Mean (SD)	
			Plantar Fasciitis	Control
Rearfoot complex	Sagittal	Dorsiflexion	0.48 (0.85)	0.45 (0.41)
		Plantarflexion	5.94 (2.81)	5.75 (2.09)
	Frontal	Inversion	4.16 (1.81)	4.77 (1.41)
		Eversion	0.19 (0.70)	0.00 (0.00)
	Transverse	Adduction	0.03 (0.05)	0.06 (0.17)
		Abduction	5.66 (2.74)	5.32 (1.76)
Calcaneocuboid Complex	Sagittal	Dorsiflexion	0.43 (0.44)	0.42 (0.68)
		Plantarflexion	2.66 (2.38)	2.79 (2.35)
	Frontal	Inversion	1.15 (1.01)	0.51 (0.75)
		Eversion	0.95 (0.94)	1.46 (1.16)
	Transverse	Adduction	0.20 (0.38)	0.18 (0.37)
		Abduction	2.65 (2.04)	2.12 (1.98)
Calcaneonavicular Complex	Sagittal	Dorsiflexion	1.26 (1.57)	0.94 (1.42)
		Plantarflexion	1.56 (1.90)	1.51 (1.94)
	Frontal	Inversion	0.02 (0.08)	0.00 (0.00)
		Eversion	3.58 (1.60)	3.03 (1.66)
	Transverse	Adduction	1.45 (1.16)	1.63 (1.70)
		Abduction	0.89 (1.26)	0.52 (0.87)
Medial Forefoot	Sagittal	Dorsiflexion	0.05 (0.07)	0.03 (0.09)
		Plantarflexion	7.84 (2.35)	7.95 (2.71)
Lateral Forefoot	Sagittal	Dorsiflexion	0.35 (0.61)	0.05 (0.13)
		Plantarflexion	3.56 (2.52)	3.14 (2.17)
First Metatarsophalangeal Complex	Sagittal	Extension	18.36 (3.34)	19.49 (4.22)
		Flexion	0.00 (0.00)	0.00 (0.00)

Table 6. Phase 4 Mean (SD) of functional articulations.

| Functional articulation	Plane	Motion	Group Mean (SD)	
			Plantar Fasciitis	**Control**
Rearfoot Complex	Sagittal	Dorsiflexion	0.00 (0.00)	0.13 (0.49)
		Plantarflexion	19.62 (4.26)	19.28 (5.46)
	Frontal	Inversion	2.77 (2.85)	3.47 (3.39)
		Eversion	0.97 (2.02)	0.81 (1.48)
	Transverse	Adduction	0.88 (1.28)	1.01 (1.91)
		Abduction	5.90 (4.27)	5.78 (4.27)
Calcaneocuboid Complex	Sagittal	Dorsiflexion	0.66 (1.18)	0.33 (0.96)
		Plantarflexion	4.92 (3.30)	4.71 (2.19)
	Frontal	Inversion	1.60 (1.96)	0.77 (1.13)
		Eversion	1.61 (2.14)	1.19 (1.09)
	Transverse	Adduction	1.18 (1.47)	1.02 (1.02)
		Abduction	2.13 (1.50)	1.64 (2.36)
Calcaneonavicular Complex	Sagittal	Dorsiflexion	2.50 (1.91)	1.21 (1.22)
		Plantarflexion	1.19 (1.64)	1.28 (1.33)
	Frontal	Inversion	0.48 (0.74)	0.67 (1.43)
		Eversion	3.42 (3.31)	1.60 (1.17)
	Transverse	Adduction	1.99 (2.15)	1.57 (1.70)
		Abduction	0.84 (1.38)	0.87 (1.337)
Medial Forefoot	Sagittal	Dorsiflexion	0.00 (0.00)	0.00 (0.01)
		Plantarflexion	16.98 (4.17)	15.58 (3.24)
Lateral Forefoot	Sagittal	Dorsiflexion	0.31 (0.98)	0.03 (0.13)
		Plantarflexion	7.67 (3.33)	6.68 (2.11)
First Metatarsophalangeal Complex	Sagittal	Extension	15.32 (6.19)	13.63 (3.45)
		Flexion	4.61 (3.67)	3.25 (3.29)

Appendix I
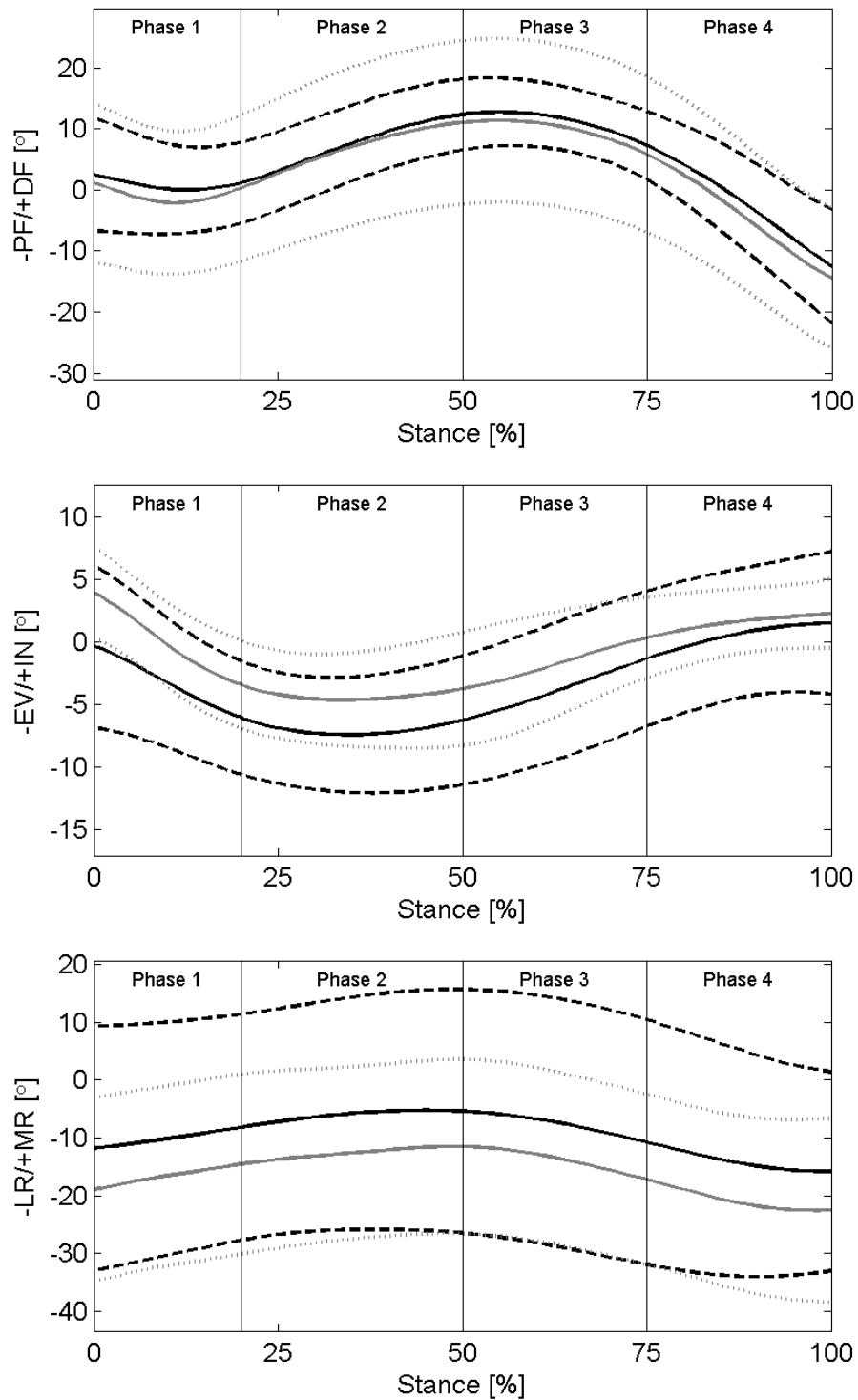
Figure 8. RC stance phase kinematics (mean ± 1 SD) for the control (black lines) and plantar fasciitis (gray lines) group participants. From top to bottom: sagittal, frontal, and transverse planes ROM
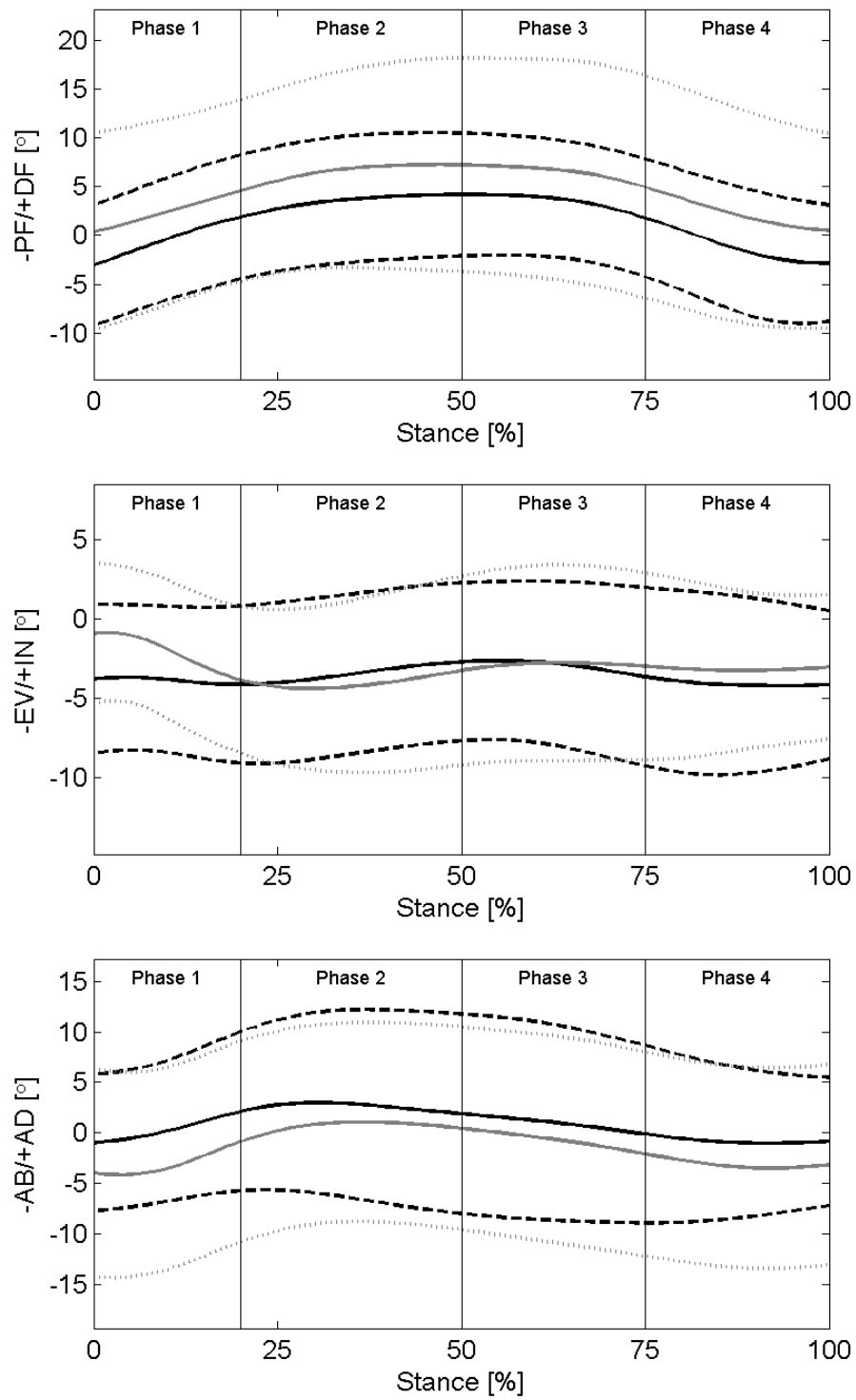
Figure 9. CC stance phase kinematics (mean ± 1 SD) for the control (black lines) and plantar fasciitis (gray lines) group participants. From top to bottom: sagittal, frontal, and transverse planes ROM

Figure 10. CNC stance phases kinematics (mean ± 1 SD) for the control (black lines) and plantar fasciitis (gray lines) group participants. From top to bottom: sagittal, frontal, and transverse planes ROM
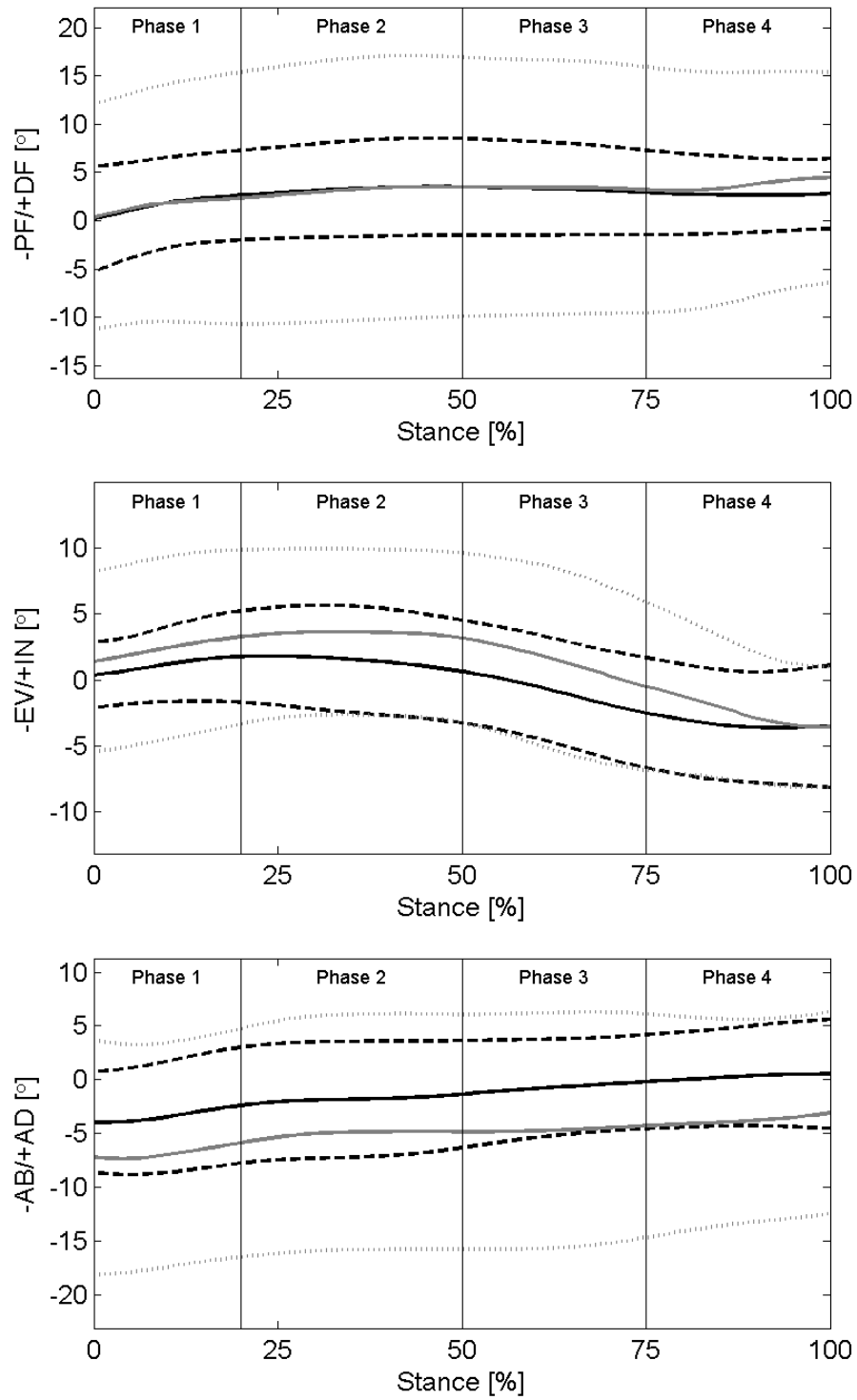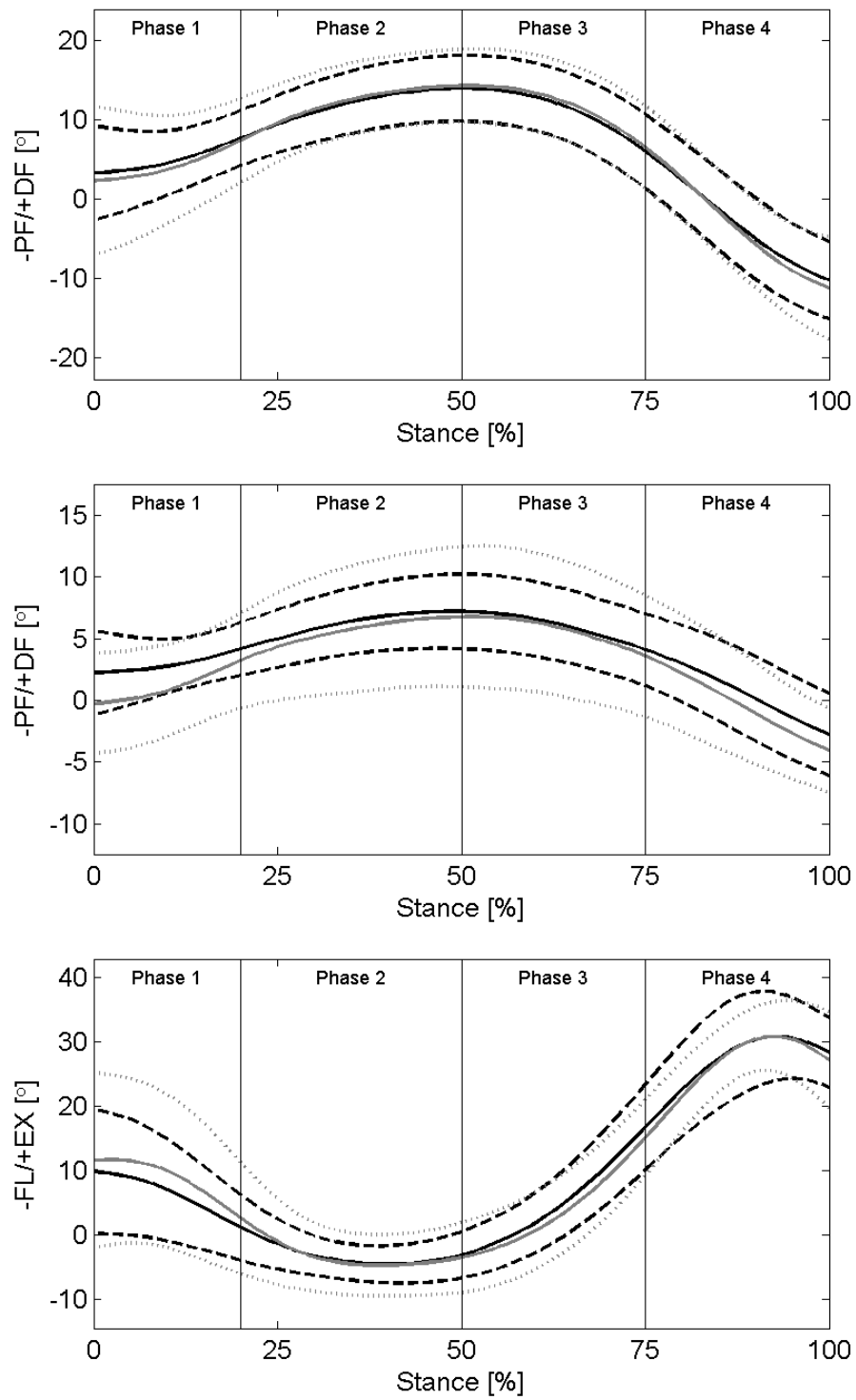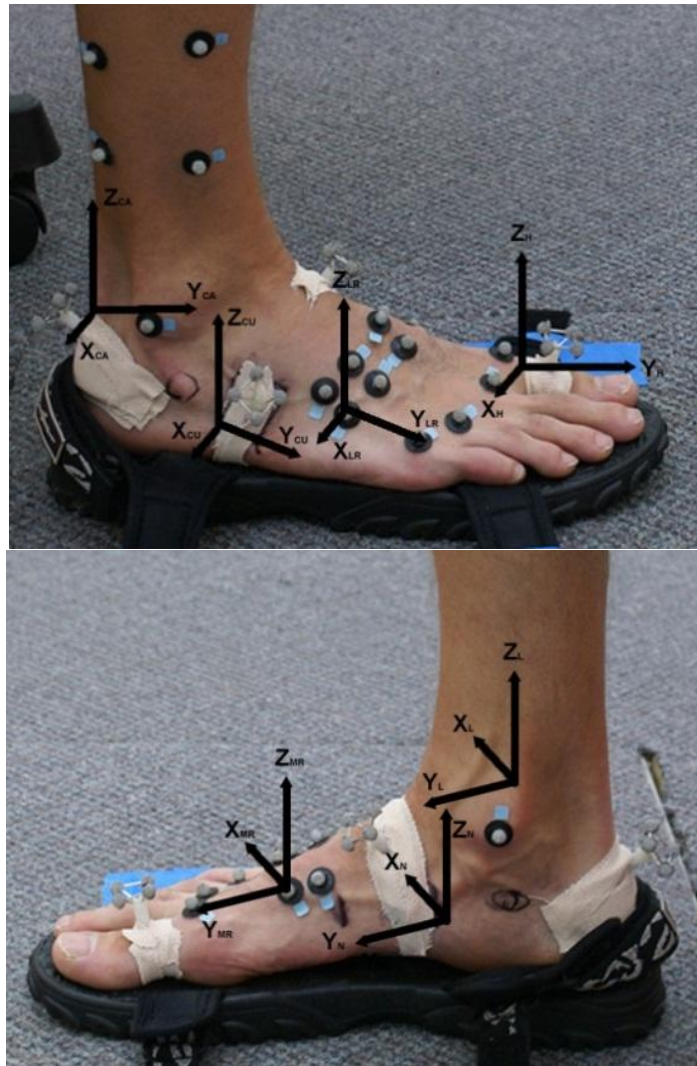
Figure 11. Stance phase kinematics (mean ± 1 SD) for the control (black lines) and plantar fasciitis (gray lines) group participants. From top to bottom: MFF sagittal plane ROM, LFF sagittal plane ROM, MTP sagittal plane ROM

Appendix J

Anatomical Reference Systems of Right Leg

(Same for the left leg)

Leg

$\bar{O}_L$: Midpoint between the medial and lateral malleoli anatomical markers.

$\hat{Z}_L$: Unit vector directed from $\bar{O}_L$ to the tibial tubercle anatomical marker, directed

    cranially.

$\hat{Y}_L$: Unit vector formed by the cross product of the $\hat{Z}_L$ unit vector and the position vector

directed from $\bar{O}_L$ to the lateral malleolus anatomical marker and the, directed

anteriorly.

X_L: Unit vector formed by the cross product of the $\hat{Z}_L$ and $\hat{Y}_L$ unit vectors, directed from

left to right.

Calcaneus

$\bar{O}_{CA}$: Located at the posterior-proximal calcaneus. $\hat{Y}_{CA}$: Unit vector directed from $\bar{O}_{CA}$ to

the midpoint between the sustentaculum tali and peroneal tubercle anatomical

markers, directed anteriorly.

$\hat{Z}_{CA}$: Unit vector formed by the cross product of the $\hat{Y}_{CA}$ unit vector and a position vector

directed from $\bar{O}_{CA}$ to the sustentaculum tali anatomical marker, directed cranially

X_{CA}: Unit vector formed by the cross product of the $\hat{Y}_{CA}$ and $\hat{Z}_{CA}$ unit vectors, directed

from left to right

Navicular

$\bar{O}_N$: Located at the plantar proximal navicular

$\hat{Y}_N$: Unit vector directed from $\bar{O}_N$ to the distal plantar navicular anatomical marker

directed anteriorly

X_N: Unit vector formed by the cross product of $\hat{Y}_N$ *and* a position vector directed from $\bar{O}_N$

to the dorsal proximal navicular anatomical marker , directed from left to right

$\hat{Z}_N$: Unit vector formed by the cross product of the X_N and $\hat{Y}_N$ unit vectors, directed

cranially

Cuboid

$\bar{O}_{CU}$: Located at the plantar proximal cuboid

$\hat{Y}_{CU}$: Unit vector directed from $\bar{O}_{CU}$ to the plantar distal cuboid anatomical marker,

    directed anteriorly

X_{CU}: Unit vector formed by the cross product of the $\hat{Y}_{CU}$ unit vector and a position vector

    directed from $\bar{O}_{CU}$ to the dorsal proximal cuboid anatomical marker directed from

    left to right

$\hat{Z}_{CU}$: Unit vector formed by the cross product of the X_{CU} and $\hat{Y}_{CU}$ unit vectors, directed

    cranially

Medial Rays

$\bar{O}_{MR}$: Located at the base of the first metatarsal

$\hat{Y}_{MR}$: A unit vector directed from $\bar{O}_{MR}$ to the head of the first metatarsal anatomical marker

    directed anteriorly

$\hat{Z}_{MR}$: The cross product of the position vector directed from $\bar{O}_{MR}$ to the head of the second

    metatarsal anatomical marker and the Y_{MR} unit vector, directed cranially

X_{MR}: The cross product of the $\hat{Z}_{MR}$ and $\hat{Y}_{MR}$ unit vectors, directed from left to right

Lateral Rays

$\bar{O}_{LR}$: Located at the base of the fifth metatarsal

$\hat{Y}_{LR}$: Unit vector directed from $\bar{O}_{LR}$ to the head of the fifth metatarsal directed anteriorly

$\hat{Z}_{LR}$: Unit vector formed by the cross product of the $\hat{Y}_{LR}$ unit vector and a position vector directed from $\bar{O}_{LR}$ to the head of the fourth metatarsal anatomical marker , directed cranially

X_{LR}: Unit vector formed by the cross product of the $\hat{Y}_{LR}$ and $\hat{Z}_{LR}$ unit vectors, directed from left to right

Hallux

$\bar{O}_H$: Located at the proximal end of the hallux

$\hat{Y}_H$: A unit vector directed from $\bar{O}_H$ to the head of the first distal phalanx anatomical marker, directed anteriorly

$\hat{Z}_H$: Unit vector formed by the cross product of the $\hat{Y}_H$ unit vector and a position vector directed from $\bar{O}_H$ to the medial surface of the first distal phalanx anatomical marker, directed cranially

X_H: Unit vector formed by the cross product of the $\hat{Y}_H$ and $\hat{Z}_H$ unit vectors, directed from left to right

Appendix K

Anatomical landmarks

Segment	
Leg	Medial malleolus*
	Lateral malleolus*
	Tibial tubercle*
Calcaneus	Sustentaculum tali†
	Peroneal tubercle†
	Dorsal posterior calcaneus*
Navicular	Dorsal proximal†
	Plantar proximal†
	Distal plantar†
Cuboid	Dorsal proximal†
	Plantar proximal†
	Plantar distal†
Medial rays	Head of 1st metatarsal†
	Head of 2nd metatarsal†
	Base of 1st metatarsal†
Lateral rays	Head of 5th metatarsal†
	Head of 4th metatarsal†
	Base of 5th metatarsal†
Hallux	Base of 1st proximal phalanx†
	Head of 1st distal phalanx
	Medial surface of 1st distal phalanx†

*Identified using a 6.4 mm retroreflective marker
†Identified using a digitizing pointer