Internal structural loading of the lower extremity during running:
Implications for skeletal injury

by

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ABSTRACT

Running is a popular activity of choice for many, and a necessity for athletes and military personnel. The positive physiological adaptations associated with running are well established, and these adaptations can only be exploited if runners remain free from overuse injury. This dissertation utilized a combination of experimentation, musculoskeletal modeling, and a probabilistic model of bone damage, repair, and adaptation to investigate internal structural loading of the lower extremity during running. Specific emphasis was placed on stress fracture development, a common overuse injury that results, in part, from the mechanical fatigue of bone. A series of studies were conducted that addressed the influence of speed on lower-extremity contact forces during running, the relationship between internal femoral loads and stress fracture development, and changes in the probability of tibial stress fracture with practical alterations in kinematics and running mileage. The findings of these studies can be summarized as follows: 1) musculoskeletal models provide meaningful non-invasive estimations of internal structural loads in healthy young adults; 2) joint contact forces increase with speed, 3) stress fractures tend to occur at femoral locations experiencing the largest mechanical loads; 4) the probability of tibial stress fracture increases with stride length and running mileage for a given speed; and 5) the probability of tibial stress fracture increases with running speed for a given mileage. Ultimately this information can be used to develop running regimens that maximize the positive adaptations associated with running and minimize the potential for overuse injury and stress fracture development.
CHAPTER 1. INTRODUCTION

The human form is such that some scientists believe endurance running was instrumental in the evolution of modern-day man (Bramble & Lieberman, 2004). In fact, no primates other than humans are capable of running a sustained long-distance. But what was once presumably a necessity for life has today become a recreational sport for many. Cardiovascular fitness (Morgan et al., 1995), muscular endurance (Costill et al., 1976), and skeletal strength gains (Kemmler et al., 2006; Stewart & Hannan, 2000) are believed to occur with running. The number of recreational runners is likely to increase as the physiological benefits of exercise continue to gain acceptance. Occasionally, however, running regimens are interrupted by acute (Fitch et al., 1989; Heiderscheit et al., 2005; Luchini et al., 1983) or chronic bouts (Messier et al., 1991; Messier et al., 1995; Nichols, 1989; Pohl et al., 2008; Warren, 1990) of musculoskeletal injuries. Research aimed at identifying methods to minimize the occurrence of injury is essential to exploit the positive benefits of running.

Overuse running injury has a diverse and multifactorial etiology, making the identification of obvious risk factors complicated. One thing is clear, however, it is the interaction of these risk factors with the mechanical loading environment that lends itself to injury. The fact that biological tissues respond and adapt to the mechanical loads placed upon them has been established for centuries (Wolff, 1892). Bone, for example, will respond in a site-specific manner by improving its strength and resilience to withstand local strain gradients (Gross et al., 1997; Judex et al., 1997). This process requires recovery time between loading bouts in order to allow the bone to sufficiently remodel itself at the adaptation site. If adequate remodeling time is not allowed (Mashiba et al., 2000) and the resulting strain continuously threatens the physiological boundaries of the tissue,
microdamage will manifest in the bony matrix and a stress fracture may occur (Burr et al., 1990; Li et al., 1985). As tissue integrity is dependent on the mechanical loading environment, the ability to quantify and monitor loads acting on the musculoskeletal system during running may aid in the prevention of overuse injury.

Ideally, mechanical loads are quantified in vivo at site-specific locations for overuse injury. Buckle transducers mounted to the Achilles tendon have been used to quantify plantarflexor force development during locomotion (Komi, 1990; Komi et al., 1992). Bone strains can also be directly measured using strain gages adhered to the tibia (Burr et al., 1996; Lanyon et al., 1975) and femur (Aamodt et al., 1997). However, due to the invasiveness of these procedures, human subject review boards are hesitant to grant approval of such techniques in the United States. For this reason, it is common practice to use surrogate measures such as external ground reaction forces as estimates of musculoskeletal loading (Whalen et al., 1988).

During running, peak external ground reaction forces in the vertical direction range between 2 and 3 bodyweights (BW) of force (Cavanagh & Lafortune, 1980; Keller et al., 1996; Munro et al., 1987). Research investigating the influence of ground reaction forces on injury development has been ambiguous. Some studies have found peak ground reaction forces to be positively correlated with injury (Ferber et al., 2002; Hreljac et al., 2000; Milner et al. 2006), while others have found no relationship (Bennell et al., 2004; Crossley et al., 1999; Nigg, 1997). In part, this may be due to the fact that reaction forces by themselves make up only a small portion of the musculoskeletal loading environment. Muscle forces may account for an additional 12.5 BW of compressive axial force during running (Komi,
1990). In addition, the stress caused by these axial forces is minimal when compared to the stress arising on the periphery of the bone due to bending (Biewener et al., 1983).

In order to obtain a more accurate understanding of the mechanical loading environment at common sites of musculoskeletal injury, researchers have turned to mathematical modeling and simulation techniques. These models range on a continuum scale from the whole body level, down to the cellular level. The possibilities are endless, and researchers are able to ask questions related to joint contact forces during locomotion (Glitsch & Baumann, 1997), ligament and tendon loading during rehabilitation exercises (Zheng et al., 1998), and the ability of bone tissue to sustain microdamage (Taylor & Lee, 2003). The following dissertation presents a series of studies that utilize a combination of experimental and musculoskeletal modeling techniques to investigate internal structural loading of the lower extremity during running. Specific focus is given to stress fracture development, a common injury among long-distance runners and military recruits that results from the cyclical fatigue of bone.

**Purpose**

The purpose of the first study was to determine the influence of running speed on lower-extremity joint contact forces. It was hypothesized that joint contact forces would increase with speed. A combination of experimental and modeling techniques were used to determine three-dimensional bone-on-bone contact forces at the ankle, knee, and hip. Three running speeds were investigated including 2.5, 3.5 and 4.5 m/s. To verify that our modeling procedures provided reasonable estimates of joint contact force, we also collected data on the same subject pool walking at 1.25 m/s. Calculated hip contact forces during walking were compared to *in vivo* hip contact force profiles, directly measured with an instrumented
prosthetic, in a 61 year old male walking at an identical speed (file KWR0602 from the database OrthoLoad, Bergmann, 2008). This provided information regarding the validity and limitations of the modeling procedures utilized for all subsequent studies presented in this dissertation.

The purpose of the second study was to determine internal femoral forces and moments during running. It was hypothesized that internal loads would be greatest at femoral locations most commonly cited to experience a stress fracture. These locations are the femoral neck, the medial-proximal shaft, and the distal condylar area. To this end, a musculoskeletal modeling approach was developed to determine three orthogonal forces and moments along a centroid path through the femur. No attempt was made to incorporate bone cross sectional geometry or material properties into the model, leaving the calculation of femoral stress and strain to a potential future analysis.

The third study in this dissertation utilizes a stress fracture model to investigate tibial fatigue failure during running. The specific purpose of this study was to determine the influence of stride length reduction and running mileage on the probability of tibial stress fracture. It was hypothesized that reducing stride length would decrease the likelihood for stress fracture despite the increased number of loading cycles required for a given running mileage. It was further hypothesized that increasing running mileage would increase the likelihood for stress fracture. The finite element method was used to estimate tibial stress and strain. These data in combination with stride frequency were incorporated into a probabilistic stress fracture model (Taylor et al., 2004). This model incorporates bone damage, repair, and adaptation, and allows for the investigation of practical kinematic alterations on stress fracture probability.
The fourth and final study builds upon study three. The purpose of this study was to determine if decreasing running speed reduces the likelihood for tibial stress fracture. It was hypothesized that reducing running speed would decrease the probability of stress fracture despite the increased number of loading cycles required for a given mileage. This study utilizes the finite element method to obtain tibial stress and strain. These data in combination with stride frequency were incorporated into the probabilistic model of Taylor et al. (2004).

Significance of Research

Running is a popular leisure activity, as well as a necessity for athletes and military personnel. Unfortunately, the occurrence of stress fracture is well documented in the running literature. Mechanical loading experienced by the skeletal system during physical activity has the potential to increase bone integrity, but the threshold distinguishing positive from negative bone adaptation remains to be established. This is primarily due to a lack of information regarding the mechanical loading environment of the skeletal system during physical activity. This dissertation focuses on methods to estimate internal structural loads during running as well as theoretical models to predict training patterns that decrease stress fracture probability. This information can be used by recreational runners, athletes, and military personnel to maximize the osteogenic benefits of exercise. It is hoped that the information gained from these studies will lead to a better understanding of stress fracture development, and in turn, lessen the medical costs and reduced training time associated with this injury.

References


CHAPTER 2. REVIEW OF LITERATURE

In the mid-eighties, Jacobs and Berson (1986) estimated that approximately 30 million Americans participated in running on some level. Today, running remains a recreational sport for many, but it is estimated that 26% to 65% of runners, both recreational and competitive, will sustain some form of overuse injury during any given year (Caspersen et al., 1984; Lysholm & Wiklander, 1987; Macera et al., 1989; Marti et al., 1988). Stress fractures account for approximately 15% to 20% of overuse injuries (Bennell et al., 1996; Brubaker & James, 1974). The most frequently cited location for stress fracture is the tibia (Korpelainen et al., 2001; Milgrom et al., 1985), but the metatarsals (Korpelainen et al., 2001; Milgrom et al., 1985) and femur (Finestone et al., 1991; McBryde, 1985; Rauh et al., 2006; Sullivan et al., 1984) account for a large percentage of stress fractures as well.

Stress Fracture

Stress fractures are monetarily, physiologically, and psychologically detrimental. The U.S. Department of Defense estimated that stress fractures cost them in excess of $10 million dollars a year in medical costs and lost training time (USAMRMC, 1999). In fact, several months of reduced weight-bearing or non-ambulatory activities may be necessary before training can be resumed (Ivkovic et al., 2006; Pihlajamäki et al., 2006). Early detection is critical, as certain stress fractures have a tendency to displace and require surgical fixation (Lee et al., 2003; Visuri et al., 1988). Any tissue damage resulting from the fracture itself, as well as perioperative trauma can lead to avascular necrosis, osteoarthritis, and in some instances permanent handicap (Lee et al., 2003; Pihlajamäki et al., 2006; Visuri et al., 1988).

The etiology of stress fractures is multifactorial and contributing risk factors can be categorized as being either extrinsic (e.g., surface, athletic footwear, training regime) or...
intrinsic (e.g., bone strength, bone fatigability, bone turnover rate) (Bennell et al., 1999). The combination of risk factors has the potential to vary between each stress fracture occurrence, but it is the interaction of these risk factors with the mechanical loading environment that ultimately leads to injury.

*Bone Fatigue and Microdamage*

Stress fractures result from repetitive cyclical loading of the skeletal system. Over time the bone fatigues and microdamage manifests as small cracks in the bony matrix (Burr et al., 1985). Microcracks in the cortices of long bones were first detected and classified by Frost approximately half a century ago (Frost, 1960). The major axis of a typical microcrack spans approximately 400 μm and is aligned with the longitudinal axis of the bone; the minor axis approximates 100 μm (Taylor & Lee, 1998). If the energy release rate, or “crack driving force”, is greater than the microstructure’s crack resistance, these microcracks can propagate into macrocracks, or stress fractures. However, because bone is a living tissue, it has two fundamental abilities that basic materials do not: 1) bone can repair itself, and 2) bone can adapt to the mechanical loads placed upon it.

*Bone Repair*

Stress fractures will only occur if the accumulation of microdamage exceeds the rate of bone repair (Burr et al., 1990). Although the exact mechanism by which bone detects microdamage and signals for repair remains unclear, the process of crack removal, or bone remodeling, is carried out by basic multicellular units (BMUs) (Frost, 1973). The coupled actions of bone resorption cells, osteoclasts, and bone building cells, osteoblasts, make up a BMU. Approximately three days are required for a BMU to be activated, and a typical BMU moves at a speed of 40 μm/day (Martin et al., 1998). Osteoclasts arrive at the site of the
crack first to resorb the damaged bone. Osteoblasts follow suit and lay down new bone such that the microcrack is completely eliminated or reduced in size. It is through this continuous process that bone maintains its integrity despite being exposed to cyclical mechanical fatigue on a daily basis.

**Bone Adaptation**

Bone can adapt to its mechanical loading environment in a site-specific manner in order to prevent microdamage at heavily loaded areas. The bone adaptation response is proportional to the number of cycles (Rubin & Lanyon, 1984), magnitude (Rubin & Lanyon, 1985) and rate of strain (O’Conner & Lanyon, 1982). It is believed that mechanical loading produces fluid forces in the extracellular matrix of bone. These fluid forces cause shear stress on mechanosensing osteocytes, which lead to the deposition of new bone and an overall increase in bone strength (Turner et al., 1995). If the bone is sufficiently adapted to the mechanical loads placed upon it, the nucleation of microcracks will be minimized. By measuring and monitoring the mechanical loading environment of bone we may elucidate loading patterns that maximize the osteogenic response and minimize the potential for bone injury.

**In Vivo Measures of Loading**

Indeed, methods exist that allow for the *in vivo* assessment of musculoskeletal loads during human movement. Strain gages adhered directly to the bone, Achilles tendon mounted force transducers, and instrumented prosthetics have all been used to directly measure internal structural loads. These techniques are considered the gold standards for mechanical loading quantification and have been extremely influential in our understanding of bone, muscle, and joint mechanics.
Bone Mounted Strain Gages

The first direct bone strain assessment in a human was performed in the mid-seventies (Lanyon et al., 1975). Lanyon et al. reported principal strains from a strain gage rosette adhered to the distal anteriomedial tibia of a 35 year old male walking at 1.4 m/s and jogging at 2.2 m/s. Magnitudes were low, peaking at 400 με during walking and 850 με during jogging. It is unclear if these values are indicative of the global strain environment of the tibia during locomotion as rosette information was limited to a single site and human long bones are known to bend under load. In addition, the walk-to-run gait transition speed has consistently been reported around 2.0 m/s (Diedrich & Warren, 1995; Hreljac, 1993), and therefore, the jogging results of Lanyon et al. may be more closely related to a brisk walk.

Similar methods have been used to characterize the strain environment of the proximal lateral aspect of the human femur (greater trochanter) during walking (Aamodt et al., 1997). This procedure was slightly more invasive than adhering a strain gage to the anteriomedial face of the tibia, because it required the splitting of the vastus lateralis fascia and the fascia lata in the longitudinal direction. The work of Aamodt et al. was instrumental in answering the age old question of whether or not the lateral aspect of the femur was under tension or compression during gait. Axial strain was tensile (1,133 με) during the stance phase of walking, leading the authors to conclude that the lateral femur is indeed under tension; no walking speed was reported.

Numerous studies have since been carried out to enhance our understanding of the in vivo strain environment in human long bones during running, primarily at the tibia. The relative invasiveness of this technique has been reduced with the advent of the bone strain “staple”, a device requiring only two small drilled bone holes for strain gage application.
rather than complete adhesion of the gage itself to the bony surface. This method has provided consistent results, with the literature reporting principal strains ranging between 1000 to 2000 με at the anteriomedial tibia during running (Burr et al., 1996; Milgrom et al., 2000; Milgrom et al., 2003; Milgrom et al., 2007). Unfortunately, these studies are limited to small sample sizes, require costly surgical procedures, and still only provide information about the strain environment at a single location.

Achilles Tendon Force Transducers

In vivo Achilles tendon forces have been directly measured with the use of a “buckle”-type transducer (Komi, 1990; Komi et al., 1992; Komi et al., 1987). The buckle is surgically implanted around the Achilles tendon and a subject-specific calibration is performed. The transducer senses changes in resistance that is proportional to Achilles tendon force. Achilles tendon force development can reach 9 kN, or 12.5 BW, during fast running at 6.0 m/s (Komi, 1990). For normal running velocities around 4 m/s Achilles tendon force reaches approximately 8.5 BW. Komi’s work has been instrumental in the field of muscle mechanics, allowing for the development of force-length and force-velocity relationships for the triceps surae muscle. Unfortunately, it would be extremely difficult to obtain similar measurements from other muscles in the lower extremity.

Instrumented Prosthetics

Whereas ankle joint contact forces can be estimated from the in vivo Achilles tendon force literature, no method exists with which to directly measure the in vivo forces of muscles crossing the knee and hip joints. Instrumented prosthetics are considered the current gold standard for knee and hip joint contact force calculation. Loads are transmitted from strain gages or load cells embedded within the prosthetic (Bergmann et al., 2001; Bergmann et al.,
1993; D'Lima et al., 2005, 2006). The problem is that instrumented prosthetic loads are limited to elderly subjects that have undergone total joint replacements. Losses in muscle function from operative procedures occur (Lu et al., 1998; Lu et al., 1997; Taylor & Walker, 2001), and typical gait speeds used to investigate instrumented prosthetic loads are usually less than the walk-to-run gait transition. These limitations make the validation of modeled joint contact forces in young healthy adults difficult. A study is needed in which the instrumented prosthetic literature is used to validate a musculoskeletal model in young healthy adults walking at slow speeds, which can then be used to estimate joint contact forces at faster running speeds.

It would be impractical to implant an instrumented prosthetic device into a young healthy subject, and due to the potential complications associated bone mounted strain gages and Achilles tendon force transducers, human subject review boards are hesitant to grant approval of such techniques in the United States. For this reason, it is common practice to use external measurements, such as ground reaction forces, for an indirect or surrogate measure of mechanical loading (Whalen et al., 1988).

Surrogate Measures of Loading

During the stance phase of running, a ground reaction force that is indicative of the acceleration of the body’s center of mass can be measured. A typical vertical ground reaction force profile shows the presence of two distinct peaks (Figure 1). The first of these peaks is preceded by a rapid increase in force that is associated with the change in velocity during ground contact. This initial peak, referred to as the “impact peak”, occurs within 5 to 30 ms following heel-strike (Nigg, 1986) and ranges in magnitude from 1.6 to 2.3 BW (Cavanagh & Lafortune, 1980; Munro et al., 1987). Following the impact peak, the ground reaction
force more slowly increases to a second peak that occurs at mid-stance. This second peak is referred to as the “active peak” because it is associated with the actual movement of running. The active peak ranges in magnitude from 2.5 to 2.8 BW (Cavanagh & Lafontune, 1980; Keller et al., 1996; Munro et al., 1987).

![Graph of ground reaction force during running.](image)

**Figure 1.** Vertical ground reaction force during running.

Ground reaction forces have traditionally been used to experimentally investigate the relationship between mechanical loading parameters and subjects with retrospective stress fracture. Results have been ambiguous, with some studies reporting larger external loads in subjects with a previous history of stress fracture (Milner et al., 2006a; Milner et al., 2006b; Pohl et al., 2008), and other studies reporting no relationship (Bennell et al., 2004; Crossley et al., 1999). When a relationship was observed, the biomechanical factors separating controls from subjects with retrospective stress fracture were peak instantaneous loading rate...
during the impact phase, peak absolute free moment during stance, or both. The latter variable, peak absolute free moment, is a vertically oriented torque experienced between the foot and ground. Therefore, if a relationship exists between external forces and stress fracture development, it may be related to impulsive loading during impact or torsional loading during stance.

Musculoskeletal Modeling

External measures of loading may have failed to consistently distinguish between subjects with and without a history of stress fracture, because external loads by themselves comprise only a small portion of the skeletal loading environment. Scott and Winter (1990) recognized the limitations with surrogate measures of mechanical loading, and as such, used a lower-extremity modeling procedure to obtain internal forces at common running injury sites. What was gained from this venerable study was an appreciation for how large these internal forces can be, and how much information goes unrealized using traditional surrogate measures of loading. Scott and Winter estimated ankle compressive joint contact forces between 10.3 to 14.1 BW. Contact forces were quantified by summing the ankle joint reaction force with the net ankle joint moment divided by the Achilles tendon moment arm. At first glance these magnitudes may seem unreasonably large. However, if one were to add a vertical reaction force of 2.5 BW with the 8.5 BW of Achilles tendon force measured by Komi (1990) the predicted ankle joint contact force would fall directly within the range of values predicted by Scott and Winter (1990). Several other modeling studies have estimated similar values for ankle joint contact forces during running (Burdett, 1982; Glitsch & Baumann, 1997; Sasimontonkul et al., 2007).
Methods for Muscle Force Estimation

A fundamental problem in movement science is the determination of force patterns in individual muscles. The redundancy of the musculoskeletal system allows for an infinite number of muscle force combinations capable of producing the resultant joint moments observed during a movement. The only current means with which to estimate these muscle forces is through mathematical modeling techniques. Several techniques are available including the reduction, electromyographical (EMG) driven, forward dynamics simulation, and static optimization method.

Reduction and EMG method. The reduction method reduces the system to a limited number of muscles equal to the number of available dynamic equations (Collins, 1995). This is a relatively simplified approach requiring a number of functional and anatomical assumptions. The EMG method is much more elegant, transforming muscle activation patterns from surface mounted EMG electrodes into force using a Hill-type muscle model (Besier et al., 2003; Lloyd & Besier, 2003). Although considered to be an accurate method, the EMG driven models require individual subject calibration techniques for EMG-to-force relationships. The number of muscles in the model is usually reduced to reflect only the surface muscles from which EMG information can be obtained.

Forward dynamics method. Forward dynamic simulation is becoming a widely used procedure to obtain individual muscle forces. This procedure can account for most if not all of the musculature in the lower extremity and traditionally uses optimization techniques in order to find a particular set of muscle activation patterns that minimize the differences between experimental kinematic data and the model (McLean et al., 2004; McLean et al., 2003). The problem is that forward dynamic simulations are mathematically intensive and
computationally expensive. A more computationally efficient method that allows for the prediction of forces for all muscles in the lower extremity is static optimization.

*Static optimization method.* Static optimization solves the “force sharing” problem by determining a set of muscle forces that minimize a specified cost function. The optimization is constrained so that when the estimated muscle forces are multiplied by their respective moment arms the resultant moments are equal to the net internal joint moments calculated from inverse dynamic solutions of experimentally derived data. The choice of cost function is left to the researcher, but Crowninshield and Brand (1981) suggest that the cost function should be theoretically supported by physiologically based criteria. A cost function that maximizes muscular endurance by minimizing the summation of muscle stresses to some exponent, $n$, appears to work well for human gait. Glitsch and Baumann (1997) found best agreement between EMG profiles and estimated muscle forces from static optimization with an exponent of 2. Static optimization solutions have also been shown to agree well with dynamic optimization solutions (Anderson & Pandy, 2001).

*Joint Contact Forces*

Several researchers have estimated joint contact forces during locomotion using a static optimization approach (Heller *et al.*, 2001a; Pedersen *et al.*, 1997; Stansfield *et al.*, 2003; Taylor *et al.*, 2004b). Joint contact forces are calculated by summing the reaction force and muscle forces that cross a particular joint (Figure 2). Brand *et al.* (1994) suggested that the optimization approach overestimated joint contact forces due to a lack of realistic muscle wrapping points in the musculoskeletal model that essentially underestimated muscle moment arms. Recent improvements in anatomical and physiological assumptions may have adequately addressed this matter. Heller *et al.* (2001b) observed close agreement, within
subjects, between hip joint contact forces measured with an instrumented prosthetic and those estimated with the static optimization procedure. Peak hip contact forces differed by less that 14%. Sasimontonkul et al. (2007) used the static optimization technique to estimate joint contact forces at the ankle during running. The estimated muscle forces showed close agreement to the *in vivo* Achilles tendon literature of Komi (1990).

![Figure 2](image)

**Figure 2.** Ankle joint reaction force, muscle force, and contact force during running.

Despite the improvements in joint contact force estimation over recent years, contact forces by themselves give little information about the loaded skeletal tissue throughout lower-extremity long bones. For example, a hip contact force of 5 BW does not represent the load experienced by the midshaft of the femur. In order to get a true appreciation for the loads experienced by specific regions of the skeletal system, one can calculate the local loads acting along a centroid path through the long bone. In this instance is it also useful to
determine the local moments throughout the bone because the stresses arising on the periphery of bone due to bending are considerable (Biewener et al., 1983).

Internal Bone Forces

Duda et al. (1997) compiled kinematic and muscle force data from the literature and determined the internal force and moments acting along a centroid path through the femur during the stance phase of walking. The authors assumed that the femur was in a state of equilibrium characterized by the summation of joint contact, muscle, and gravitational forces. Three orthogonal forces and moments were determined at specific points within the femur by assuring equilibrium with all loads acting on the section of the femur above. Duda et al. demonstrated that the proximal and distal regions of the femur experienced the largest loads due the hip and knee musculature spanning the joints in these regions. It was also observed that muscle forces play a critical role in reducing bending moments throughout the femur. No study has yet to estimate these internal loads during running. Knowledge of the site specific loads within the femur during running may increase our knowledge of stress fracture development and prevention.

Beam Theory

It is surely the intensity of the resulting loads, rather than the loads themselves that are so crucial in the development of stress fracture. Large loads will not be detrimental if they are distributed over a large area or the bone’s resistance to deformation is high. As such, the quantification of stress and strain is essential for predicting the adverse reactions to mechanical overload. The traditional approach to solve for stresses and strains in long bones was to use beam theory.
Just under a century ago, Koch (1917) applied beam theory to the human femur in an attempt to show that bone structure and internal architecture were dependent upon its mechanical loading environment. Koch approximated the femur as a two-dimensional prismatic beam under symmetrical bending. Unfortunately, Koch was limited by the technology of the era. He was unable to account for the forces created by muscles and therefore wrongly concluded that muscular activity had little influence on femoral loading.

More recent studies that rely on beam theory assumptions to estimate skeletal stresses do account for muscular loading (Biewener et al., 1983; Pollock et al., 2008a; Pollock et al., 2008b), three-dimensionality (Pollock et al., 2008a; Pollock et al., 2008b), non-symmetrical bending (Pollock et al., 2008a; Pollock et al., 2008b), and radius of curvature (Beck et al., 1998; Mourtada et al., 1996). The fundamental problem with beam theory arises when applying Hooke’s law to convert stresses into strains. Human long bones are not made of homogeneous material throughout. To conserve mass the epiphyseal regions of long bones are dominated by trabecular bone, a low density material with high surface area that is highly resistant to compressive load. The diaphyseal region is made up of cortical bone, a solid and high density material that varies in thickness depending on its mechanical requirements. The strength of cortical bone makes it highly resistant to bending. Due to the inherent inhomogeneity of long bones, strains can only be approximated in homogeneous locations (i.e., the diaphysis), or a technique known as composite beam theory must be used.

In the composite beam theory approach, a bone made up of different materials is reduced to an equivalent bone having only one material by changing the cross sectional dimensions accordingly (Raftopoulos & Qassem, 1987). Raftopoulos and Qassem wrote a theoretical review on the topic with applications to the femur. This technique has not been
utilized extensively in the human movement sciences due to recent advances in computer power and subsequently the finite element method.

**Finite Element Modeling**

The finite element method has only been around since the mid-1950’s. Finite element analysis uses computational numerical techniques to obtain approximate solutions for differential equations. An object of complex geometry is essentially divided into many small simplified geometries (e.g., pyramids, cubes, etc.) called elements. The inhomogeneity of the structure can be captured by assigning different material properties to each element. The governing equations for all elements are integrated and summed across the problem domain. Linear algebra techniques can then be used to solve for the displacements, strains, and stresses over the entire geometry.

Finite element modeling has been instrumental in the field of orthopedic biomechanics. A majority of the research has been focused towards improving artificial joint replacement designs and fixation techniques (Huiskes & Chao, 1983). Finite element models have also been used to investigate the effects of muscle forces on bone loading (Duda et al., 1998; Polgár et al., 2003), the influence of daily stresses on functional bone adaptation (Beaupre et al., 1990; Jacobs et al., 1997), and the ultimate strength and fracture location of bone (Lotz et al., 1991; Schileo et al., 2008). Inputs to these models are based on walking and falling, and only a small number of studies have investigated the influence of running type loads on skeletal stress and strain. The few studies that have investigated running, utilized joint reaction forces rather than joint contact forces as inputs to their models (Voo et al., 2004). It is evident that this would drastically underestime the resulting skeletal
deformation. Running investigations that utilize appropriate loading conditions are necessary if researchers use this method as a means to investigate skeletal injury.

**Beyond Peak Instantaneous Loads**

The majority of biomechanical research dealing with musculoskeletal injury has utilized peak instantaneous loads to quantify injury potential. This approach is appropriate for many types of experimental designs. However, because stress fracture results from the cyclical fatigue of bone it is important to account for the number of loading cycles. Many loading cycles at a low magnitude can be more detrimental than few cycles at a high magnitude. Potential mechanisms of load reduction include reducing stride length and running velocity. Both these mechanisms, however, increase the number of loading cycles for a given running mileage. The probabilistic stress fracture model developed by David Taylor (Taylor & Kuiper, 2001; Taylor *et al.* 2004a) takes load amplitude and loading cycles into consideration. In doing so, Taylor’s model allows for a better assessment of stress fracture potential than the more traditional approach of peak instantaneous load measurement.

**The Probabilistic Stress Fracture Model**

The traditional means to describe the fatigue life of a material is with a stressed-life plot, or “S-N curve”. A typical S-N curve can be expressed by an inverse power-law relationship:

\[ N_f = C\Delta\sigma^{-n} \]

where \( N_f \) is the number of cycles to failure, \( \Delta\sigma \) is the stress range, \( n \) is the slope of the S-N curve, and \( C \) is a constant. This is also known as the standard fatigue equation. Constants \( n \)
and $C$ are experimentally derived. Carter and Caler (1985) observed a slope of $n = 6.6$ for fatigue damage of cortical bone at large $N_f$ values relevant to human locomotion.

**The Weibull Equation**

Due to inherent differences between experimental testing samples, considerable variability in $N_f$ at a given $\Delta \sigma$ can be obtained for similar specimens. In the field of engineering this variability is termed scatter. Weibull (1951) developed a statistical procedure to deal with the scatter associated with the fatigue of materials. Utilizing the Weibull equation one can predict the probability that a material will fail within a given amount of cycles $N$ at a particular $\Delta \sigma$. The Weibull equation takes the form:

$$P_f = 1 - \exp \left( -\left( \frac{\Delta \sigma}{\Delta \sigma^*} \right)^m \right)$$

where $P_f$ is the cumulative probability that the material will fail at the stress ranges up to $\Delta \sigma$. The reference stress range $\Delta \sigma^*$ is a measure of the materials fatigue strength. It represents the stress range at which the probability for failure is 0.63 for a given $N$. In bone, $\Delta \sigma^*$ will depend on things like bone age and the type of cyclical loading (e.g., zero-tension, zero-compression, or tension-compression). The Weibull modulus $m$ expresses the degree of inherent scatter in the material’s fatigue behavior. Both $\Delta \sigma^*$ and $m$ are experimentally derived constants. For cortical bone, Taylor (1998) found $\Delta \sigma^*/\Delta \sigma_{\text{mean}}$ to be 1.067 and $m$ to be 8. Here, $\Delta \sigma_{\text{mean}}$ is the mean stress range for failure at a given $N$.

**Stressed Volume**

Traditionally, fatigue tests of materials are performed on small volume specimens. However, small specimens are inherently stronger than large specimens because they have
fewer weak points, or in the case of bone, less microdamage. In other words the probability of finding a microcrack in a large volume specimen is greater than that of a small volume specimen. This is an important concept when utilizing experimental data to predict the failure of whole long bones. For this reason, Taylor (1998) extended the Weibull equation to account for the effect of “stressed volume”:

\[ P_f = 1 - \exp\left[-\left(\frac{V_s}{V_{so}}\right)^n\left(\frac{\Delta\sigma}{\Delta\sigma^*}\right)^m\right] \]

where \( P_f \) in this case is the cumulative probability that a specimen having stressed volume \( V_s \) will fail at the stress ranges up to \( \Delta\sigma \), given a reference stressed volume \( V_{so} \) and reference stress range \( \Delta\sigma^* \). For a 96 mm\(^3\) specimen of cortical bone from a relatively young (27 yrs) individual, \( \Delta\sigma^* = 86 \text{ MPa} \) for an endurance test of \( 10^5 \) cycles to failure (Zioupos et al., 1996).

**Variable Loading**

Like most materials bone is subjected to loads that vary in amplitude. For example, bone stress during walking will be less than bone stress during running or jumping. To account for variable loading, Taylor and Kuiper (2001) recommended using the concept of equivalent stress, in which a variable amplitude is transformed to an equivalent constant amplitude based on a weighted average procedure:

\[ \Delta\sigma_{eq} = \left(\frac{1}{N_T} \sum_{i=1}^{k} N_i \Delta\sigma_i^n\right)^{1/n} \]

where \( \Delta\sigma_{eq} \) is the equivalent stress, \( N_i \) is the number of cycles at stress range \( \Delta\sigma_i \), \( N_T \) is the total number of cycles, and \( n \) is the slope of the S-N curve. This procedure assumes that the order in which the variable stress amplitudes are applied makes no difference on \( N_f \).
When dealing with whole bone a second problem arises that deals with variable loading. The $\Delta\sigma$ and therefore $P_f$ is not constant throughout the entire bone. Using the finite element method one can obtain a separate $P_f$ for each element: call this $P_i$. If there are $k$ elements, then $P_f$ for the whole bone is the probability that any one element will fail. This is analogous to saying an entire chain will fail if a single chain link fails. The probability that a single element will fail is:

$$P_f = 1-(1-P_1)(1-P_2)(1-P_3)\ldots(1-P_k).$$

Taylor and Kuiper (2001) found, through trial and error, that all elements can be separated into a minimum of eight groups experiencing similar $\Delta\sigma$ for an accurate estimation of the whole bone $P_f$. In practice, the researcher determines an $\Delta\sigma_{eq}$ and corresponding $V_s$ for each of these eight groups prior to $P_f$ calculation.

**Incorporating Bone Remodeling and Adaptation**

Indeed, bone will remodel itself and adapt to the mechanical loads placed upon it. The process of bone remodeling and adaption are time dependent and for this reason Taylor (2004a) rewrites the Weibull equation in terms of $t$, the time in days:

$$P_f = 1 - \exp\left[-\left(\frac{V_s}{V_{so}}\right)\left(\frac{t}{t_f}\right)^w\right]$$

where $t_f$ is calculated from the $N_f$ associated with a given $\Delta\sigma$. To obtain $t_f$ in days, $N_f$ is divided by the number of cycles/day at the given $\Delta\sigma$. In this equation the Weibull modulus, $w$, is dependent on both the scatter in the data and the slope of the S-N curve ($w = m/n = 1.2$).

**Bone remodeling.** The mean repair time for a BMU to be activated and tunnel through a microcrack is $18.50 \pm 12.95$ days (Taylor et al., 2004a). Because there is variability
associated with repair time, there is also a probability that the microdamage will be repaired on any given day. The equation for the probability of repair $P_r$ is:

$$P_r = 1 - \exp \left[ - \left( \frac{t}{t_r} \right)^v \right]$$

where the reference time for repair $t_r$ is 26 days and the Weibull modulus $v$ is 2.

Written in terms of $t$, both $P_f$ and $P_r$ are the cumulative probabilities that failure or repair will occur from time zero to $t$. In order to combine $P_f$ and $P_r$ it is necessary to calculate the differential of $P_f$ with respect to time, or the “probability density function”. The probability density function can be thought of as the instantaneous probability that failure will take place within a unit time period (e.g., one day). The bone will not fail if sufficient time has elapsed for repair to occur. Accordingly, the probability density function of failure with repair $Q_{fr}$ is:

$$Q_{fr} = Q_f(1 - P_r)$$

where $Q_f$ is the probability density function of failure. The cumulative probability of failure with repair $P_{fr}$ is then:

$$P_{fr} = \int_0^t Q_{fr} \, dt .$$

Incorporating bone repair into the model drastically reduces the $P_f$. The $P_f$ continuously increases over time without repair, but plateaus at a relatively low value with repair (Figure 3).
Figure 3. Estimated cumulative failure probabilities for a bone loaded at a stress range of 37 MPa and a frequency of 4100 cycles/day. Stressed volume was assumed to be equal to the reference volume.

**Bone adaptation.** New bone can take one of two forms: woven bone or lamellar bone. Woven bone is a highly disorganized, randomly oriented tissue. In the mature adult, woven bone is most commonly seen in the healing process of bone fracture, where it provides a quickly forming tissue to reinforce the damaged area. Lamellar bone is a highly organized tissue, and differs from the rapidly forming woven bone in that it requires a pre-existing hard tissue model to grow upon. Lamellar bone is laid down in response to gradual changes in activity and thus mechanical loading. The adaptation of bone in response to mechanical loads during running can be attributed primarily to the deposition of lamellar bone.

Lamellar bone can be deposited at a maximum rate of 4 μm/day (Taylor et al., 2004a). Deposition will occur on the endosteal and periosteal surface of the bone. In turn, the
cross sectional area will increase such that the stresses experienced by the bone are reduced. In order to account for bone adaption, the idea of equivalent stress $\Delta \sigma_{eq}$ is reintroduced. In the case of adaption, $\Delta \sigma_{eq}$ can be calculated in integral form:

$$\Delta \sigma_{eq} = \left( \frac{1}{t_T} \int_{0}^{t_T} \Delta \sigma^n \, dt \right)^{1/n}$$

Where $t_T$ is the total time over which adaptation takes place and $n$ is, again, the slope of the S-N curve. This is, in fact, the $\Delta \sigma_{eq}$ utilized within the model to determine the probability of failure with repair and adaptation $P_{fra}$. Technology permitting, lamellar bone is added to the most highly stressed locations of the bone within the finite element model and the stresses are recalculated for each time iteration. If, however, the stresses can be estimated using beam theory, the changes in cross sectional area and areal moment of inertia can be approximated, and $\Delta \sigma_{eq}$ can be determined analytically.

Summary

There is an abundance of literature pertaining to the mechanical loading environment of bone during walking. Extrapolation of these data to running is unrealistic because of the innate differences between these two styles of gait. Knowledge of the internal structural loading environment of the skeletal system during running will bring us one step closer to understanding why some athletes receive overuse injuries and others do not. This information can be used in conjunction with theoretical modeling techniques to determine training patterns that maximize the osteogenic effects of running and minimize the potential for injury. Although the following dissertation focuses primarily on long-distance running, this information can also be used by military personnel to lessen the occurrence of stress fractures during basic training.
References


CHAPTER 3. THE INFLUENCE OF RUNNING SPEED ON LOWER EXTREMITY JOINT CONTACT FORCES

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Abstract

Reducing speed is a potential mechanism of joint contact force reduction during running. Direct \textit{in vivo} measurements of joint contact forces during running are limited to older adults at slow speeds of locomotion. Previous estimations of joint contact forces using analytical techniques have been limited to a single running speed. The purpose of this study was to determine the influence of speed on lower-extremity joint contact forces during running. Ten males ran overground at speeds of 2.5, 3.5, and 4.5 m/s. Motion capture and force platform data were collected concurrently. A combination of experimentation and musculoskeletal modeling were used to determine joint contact forces at the hip, knee, and ankle. The influence of running speed on peak contact forces was examined using one-way repeated measures ANOVA’s with Bonferroni post-hoc comparisons. In general peak contact forces increased with running speed (p<0.01), however, no difference in peak knee lateral shear force was observed between 3.5 and 4.5 m/s (p=0.17), and no effect of speed was observed for peak medial ankle shear force (p=0.08). Across speeds, peak resultant contact forces at the hip, knee, and ankle ranged from 7.42-10.45, 11.98-14.72, and 12.03-15.46 bodyweights, respectively. The observed increase in joint contact force with speed suggests that overuse injury potential may increase with speed as well.

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\textsuperscript{3}Co-Investigator.
Introduction

Approximately 25% to 65% of recreational and competitive runners will sustain some form of overuse injury each year (Caspersen et al., 1984; Lysholm & Wiklander, 1987; Marti et al., 1988). Overuse injury is believed to occur when the frequency of repetitive loads reaches a critical threshold beyond the tissue’s fatigue strength (Hreljac et al., 2000). The fatigue strength of biological tissue is heavily dependent on the resulting stress from the applied mechanical load. For bone and cartilage, this relationship can be described using an inverse-power law, in which small changes in stress result in large changes in the number of cycles to failure (Carter & Beaupre, 2001).

Reducing speed is a potential mechanism of load reduction during running. A positive relationship between ground reaction force and speed has been well established (Keller et al., 1996; Munro et al., 1987). However, reaction forces account for a small portion of the overall mechanical loading environment. An accurate interpretation of the applied mechanical load requires knowledge of the load contribution provided by muscle forces (Scott & Winter, 1990). Arampatzis et al. (1999) observed no change in knee and ankle internal joint moments between typical long-distance running speeds of 3.5 and 4.5 m/s. As the individual muscle forces that create these moments compress the articulating joint surfaces, the relationship between joint contact force and running speed remains unclear.

There are two methods currently being used to quantify joint contact force. Instrumented prosthetics are considered the gold standard for joint contact force calculation. However, data from instrumented prosthetics are limited to older adults who have undergone total joint replacements (Bergmann et al., 1993; Taylor & Walker, 2001) and typical speeds used to investigate running are usually less than the walk-to-run gait transition speed (2.0
m/s; Diedrich & Warren, 1995; Hreljac, 1993). Several studies have estimated joint contact forces during running using musculoskeletal modeling techniques (Burdett, 1982; Glitsch & Baumann, 1997; Harrison et al., 1986; Sasimontonkul et al., 2007; Scott & Winter, 1990; van den Bogert et al., 1999). Unfortunately these studies limited their analyses to a single speed, or narrow range of speeds, between 3.5 and 5.3 m/s. No study has systematically manipulated running speed and estimated joint contact forces using analytical techniques.

Therefore, the purpose of this study was to determine the influence of running speed on lower-extremity joint contact forces using a combination of experimental and musculoskeletal modeling procedures. Ultimately, this information could be used in advanced numerical and theoretical models aimed at identifying running regimens that optimize the positive benefits of running and reduce the potential for overuse injury.

Methods

Subjects

Ten males were recruited for this study (age 24.9 ± 4.7 yrs; height 1.7 ± 0.1 m; mass 70.1 ± 8.9 kg). All subjects were free from lower-extremity injury at the time of data collection. The study was approved by the institutional review board, and subjects signed an informed consent document prior to testing.

Data Collection

Subjects wore commercially available running shoes and were outfitted with tight fitting athletic wear. A series of anthropometric measurements were taken including height, mass, thigh length, mid thigh circumference, calf length, calf circumference, foot length, foot breadth, malleolus height, and malleolus width. Seventeen retroreflective markers were then placed on anatomical landmarks of the trunk and right lower-extremity. Markers were
adhered to the dorsi-foot, fifth metatarsal, heel, medial and lateral malleolus, distal and proximal anterior calf, posterior calf, medial and lateral femoral epicondyle, anterior and lateral thigh, left and right greater trochanter, left and right anterior superior iliac spine, and the joint between the fifth lumbar and first sacrum (L5S1). A static motion capture trial was collected to determine joint center locations and segmental coordinate systems. The segmental coordinate systems were defined using a right handed rule, with the x-axes oriented in the anterior-posterior (AP) direction, the y-axes oriented in the axial direction, and the z-axes oriented in the medial-lateral (ML) direction (positive axes were directed anteriorly, proximally, and laterally).

Subjects were instructed to run overground at 2.5, 3.5, and 4.5 m/s. Motion capture (Vicon MX, Vicon, Centennial, CO) and force platform (AMTI, Watertown, MA) data were collected concurrently at sampling frequencies of 160 and 1600 Hz, respectively. Running speed was monitored with motion capture using the horizontal component of a marker adhered to the joint between the fifth lumbar and first sacrum (L5S1). Speed order was varied between subjects and ten trials were performed at each speed. Trials were deemed successful if running speed was ± 5% the target speed and there was no visually identified targeting of the force platform.

Data Processing

The synchronized raw motion capture and force platform data were processed using customized Matlab software (The Mathworks, Natick, MA). Cardan segment angles for the thigh, leg, and foot, and joint angles for the hip, knee, and ankle were calculated in a flexion-extension, abduction-adduction, internal-external rotation sequence (a “zxy” sequence using our reference convention). The subtalar joint angle was determined in accordance with
O’Conner and Hamill (2005). Inverse dynamics with rigid body assumptions were used to calculate net internal joint moments at the hip, knee, and ankle. Thigh, leg, and foot segment masses, center of mass locations, and moments of inertia were obtained from the equations of Vaughan et al. (1992). The subtalar moment was estimated by rotating the ankle joint moment into the subtalar coordinate system. When performing inverse dynamics analysis for impact activities such as running and landing, it has been established that filtering kinematic and kinetic data at different cutoff frequencies can create an impact-like artifact that is visible in the hip and knee moment (Bisseling & Hof, 2006; van den Bogert & de Koning, 1996; White & Podraza, 2007). White and Podraza (2007) recommended performing inverse dynamics on the raw data and then filtering the joint moments and reaction forces according to the frequency content of the vertical ground reaction force. For an objective measure, we chose to filter (4th order zero-lag Butterworth) joint moments and reaction forces at the 95th percentile frequency of the vertical ground reaction force. The 95th percentile frequency was calculated from the cumulative sum of an integrated power spectral density curve. The resulting mean cutoff frequencies were 20.3 ± 3.1, 26.6 ± 2.8, and 32.8 ± 3.9 Hz for the 2.5, 3.5, and 4.5 m/s conditions, respectively.

Processed data were interpolated to 101 points of stance using a cubic spline routine. Joint angles were imported into a SIMM musculoskeletal model (Musculo-Graphics Inc., Santa Rosa, CA) that was scaled to the individual’s segment lengths. The model consisted of 43 muscles (Delp et al., 1990) and was used to obtain maximal dynamic muscle forces adjusted for velocity and length, muscle moment arms, and muscle orientations in the segment coordinate systems (Delp & Loan, 1995). Individual muscle forces were then estimated using a static optimization routine with the objective of minimizing the sum of
squared muscle stresses (Glitsch & Baumann, 1997). Six experimentally determined joint moments were used to constrain the optimization including the three orthogonal moments at the hip, the flexion-extension moment at the knee and ankle, and the subtalar moment. This optimization procedure, apart from the utilization of the subtalar moment, has been previously described in detail (Edwards et al., 2008).

Joint contact forces were determined as the vector sum of reaction forces and muscle forces crossing the joint:

\[
F_{c_j}^h = \left[ RF_j^h + \sum_{i=1}^{27} f_{ij} \right] \quad j = x, y, z
\]

\[
F_{c_j}^k = \left[ RF_j^k + \sum_{i=21}^{33} f_{ij} \right] \quad j = x, y, z
\]

\[
F_{c_j}^a = \left[ RF_j^a + \sum_{i=31}^{43} f_{ij} \right] \quad j = x, y, z
\]

where \( F_{c_j}^h, F_{c_j}^k \) and \( F_{c_j}^a \) are the three components of the joint contact force at the hip, knee and ankle, respectively, \( RF_j^h, RF_j^k \) and \( RF_j^a \) the three components of the joint reaction forces at the hip, knee and ankle, respectively, and \( f_i \) are the three components of the \( i^{th} \) predicted muscle force crossing the respective joint. Joint contact forces were normalized to bodyweight (BW) and are reported below as the forces acting on the distal segment in the distal segment coordinate system (e.g., hip contact force acting on the femoral head in the thigh coordinate system).

Statistics

Peak instantaneous joint contact forces during running were determined for each trial and averaged within speed conditions for each subject. Nine one-way repeated measures (RM) ANOVA’s were used to determine significant differences in peak contact forces.
between running speeds (3 joints with 3 components of force each). A Bonferroni adjustment was made to avoid experimentwise error ($\alpha = 0.05/9 = 0.006$). In the event that assumptions of sphericity were not met for the univariate RM ANOVA (Huynh-Feldt $\varepsilon < 0.75$), we observed the Pillai’s Trace statistic for multivariate tests. Significant $F$ statistics from either the univariate or multivariate tests were followed up with Bonferroni adjusted pairwise comparisons between speeds ($\alpha = 0.05/3 = 0.017$). All statistical analyses were performed in SPSS 15.0 (SPSS Inc., Chicago, IL).

Results

Mean speeds during running were 2.53 ± 0.04, 3.51 ± 0.04, and 4.50 ± 0.05 m/s for the 2.5, 3.5, and 4.5 m/s conditions, respectively. The largest joint reaction forces were observed in the axial direction (Figure 1). The largest internal joint moments at the hip, knee, and ankle were observed about the ML axes (Figure 2); peak values were extensor. A relatively large abductor moment at the hip and supinator moment at the subtalar joint were also observed. The prominent extensor muscles followed the general trend of the internal joint moments (Figure 3). The largest muscle forces were created by the vasti and gastrocnemius muscles. A small amount of hamstring activity was observed during early and late stance. The tibialis anterior muscle was only active during early stance.

In general, joint contact forces increased with speed ($p < 0.01$; Table 1). However, no difference in peak ML shear force at the knee was observed between running speeds of 3.5 and 4.5 m/s ($p = 0.17$). In addition, no speed effect was observed for peak ML shear force at the ankle ($p = 0.08$). The axial components of the joint contact forces were dominant over the shear components (Figure 4). The largest axial force was observed at the knee, followed by
the ankle, then the hip. Peak shear joint contact forces were directed posterior and lateral at the ankle, anterior and lateral at the knee, and posterior and medial at the ankle.

Discussion

The purpose of this study was to determine the influence of speed on joint contact forces during running. In general joint contact forces increased with running speed; exceptions included the ML knee contact force between 3.5 and 4.5 m/s, and the ML ankle contact force among all speeds.

To verify that our modeling procedures provided reasonable estimates of joint contact force, we collected additional data on the same subject pool walking at 1.25 m/s. Joint contact forces were determined using identical procedures, and subject ensemble curves for hip contact force were compared to in vivo hip contact force profiles, directly measured with an instrumented prosthetic, in a 61 year old male walking at 1.25 m/s (file KWR0602 from the database OrthoLoad, Bergmann, 2008). Subject ensemble curves were similar in shape and form when compared to in vivo hip contact force profiles (Figure 5). Pearson’s r correlations ranged from 0.48-0.79, 0.93-0.99, and 0.88-0.97 for the AP, axial, and ML direction, respectively. The respective root mean squared errors ranged from 0.11-0.22, 0.29-0.85, and 0.13-0.24 BW.

The push-off peak in the axial direction during walking was noticeably lower for the subject with the instrumented prosthetic (Figure 5). The second peak hip contact force coincides with peak flexor moment during push-off. Devita and Hortobagyi (2000) found that older adults used 37% less hip flexor activity during the latter half of stance when compared to young adults walking at an identical speed. The mean percent difference for this peak between our subjects and the subject with the instrumented prosthetic was 36%. While
it cannot be stated definitively, we feel our observed differences stemmed more from inherent differences in locomotion kinetics between these two populations, rather than error in our modeling procedures.

We observed a positive relationship between most joint contact force components and running speed; likely meaning increased injury potential with a corresponding increase in speed. This argument is supported by epidemiological literature that found runners who sustain overuse injuries are more likely to run at faster speeds than non-injured runners (Jacobs & Berson, 1986). Of course one could argue that the observed increases in joint loads may provide a more positive tissue adaptation response with increases in running speed. Indeed, this may be the case if adequate rest time is practiced between running bouts and the frequency of repetitive loads does not threaten the tissue’s fatigue strength (Robling et al., 2002).

It is certainly the resulting stress and strain from the applied mechanical load that ultimately leads to overuse injury. However, the relative differences in joint contact forces among speeds would presumably be linearly related to the relative differences in tissue stress and strain assuming similar mixed-modes of loading and constant cross sectional geometry. Averaged across joints, the mean peak resultant force was reduced by 10% from 4.5 to 3.5 m/s and 15% from 3.5 to 2.5 m/s. Carter and Caler (1985) observed the fatigue life of cortical bone to be inversely proportional to stress by a power of 6.6. This value would predict an approximate twofold increase in the number of cycles to failure with a speed decrease from 4.5 to 3.5 m/s and an approximate threefold increase in the number of cycles to failure with a speed decrease from 3.5 to 2.5 m/s. It is clear that the benefits to bone, from a linear reduction in speed, become more pronounced at lower speeds of running. A similar argument
can be made for articular cartilage (Mansour, 2003). These data could serve as boundary conditions for finite element models to determine the influence these contact forces have on tissue stress and strains during running. This would allow for more accurate predictions of overuse injury potential in specific locations of the lower extremity.

Our peak resultant hip and knee contact forces during the 4.5 m/s condition (10.45 ± 1.53 BW and 14.72 ± 2.05 BW, respectively) were substantially lower than those previously reported by Glitch and Baumann (1997; 20 BW, hip) and Harrison et al. (1986; 33 BW, knee). These studies used similar musculoskeletal modeling techniques, but assumed straight muscle paths between origin and insertion, which would allow for muscles to pass through bones and deep muscles during larger ranges of motion. As a consequence, moment arms of various muscles may have been underestimated leading to potential overestimations in muscle and joint contact forces. The model we utilized consisted of several constraining and wrapping points for various muscles to minimize this problem (Delp et al., 1990). In order to compare our ankle contact forces to previous literature it was necessary to place them in the leg coordinate system. This provided values ranging from -0.73 to -0.89 BW for AP shear, -11.92 to -15.33 BW for axial compression, and 0.59 to 0.80 for ML shear. These values agree well with those previously reported (AP shear, -3.9 to 5.5 BW; Axial -8.0 to -14.1 BW; ML shear, -0.8 to 0.5 BW; Burdett, 1982; Glitsch & Baumann, 1997; Harrison et al., 1986; Sasimontonkul et al., 2007; Scott & Winter, 1990).

Until non-invasive methods are available to directly measure individual muscle forces, analytical techniques will be necessary for joint contact force estimation. The choice of cost function can influence muscle force magnitude and force distribution. We selected a cost function that has been shown to agree well with EMG profiles during sub-maximal
speeds of gait (Glitsch & Baumann, 1997), but the relationship between EMG and muscle force is non-linear. Nevertheless, individual muscle forces were not of primary interest in this study, and subtle changes in force sharing between agonistic muscles would not drastically affect the joint contact force calculation. The musculoskeletal geometry and muscle force-generating capacity of the generic model was scaled to each individual’s segments lengths. Knowledge of these respective parameters on a subject specific basis would greatly improve these types of studies. Assuming these scaled properties resulted in random variation from actual joint contact forces between subjects, merit should be given to the mean values and relative differences reported in this study.

To conclude, we calculated joint contact forces across three running speeds. In general, joint contact forces increased with speed suggesting that injury potential may increase with speed as well. These data could be used in advanced numerical and theoretical models aimed at identifying running regimens that optimize the positive benefits of running and reduce the potential for overuse injury.

References


Table 1. Mean (SD) peak joint contact forces across running speeds. Positive values represent anterior shear, axial tension, and lateral shear. † = significantly different from 2.5 m/s (p<0.01); ‡ = significantly different from 3.5 m/s (p<0.01). Statistics were not run on peak resultant forces.

<table>
<thead>
<tr>
<th>Joint Contact Force (BW)</th>
<th>Running Speed (m/s)</th>
<th>2.5</th>
<th>3.5</th>
<th>4.5</th>
</tr>
</thead>
<tbody>
<tr>
<td>AP Hip</td>
<td>-1.25(0.32)</td>
<td>-1.53(0.42)†</td>
<td>-1.82(0.50)‡</td>
<td></td>
</tr>
<tr>
<td>Axial Hip</td>
<td>-6.98(1.18)</td>
<td>-8.24(1.15)†</td>
<td>-9.34(1.19)‡</td>
<td></td>
</tr>
<tr>
<td>ML Hip</td>
<td>2.46(0.63)</td>
<td>3.66(0.96)†</td>
<td>4.63(1.03)‡</td>
<td></td>
</tr>
<tr>
<td>AP Knee</td>
<td>0.56(0.15)</td>
<td>0.70(0.18)†</td>
<td>0.75(0.19)‡</td>
<td></td>
</tr>
<tr>
<td>Axial Knee</td>
<td>-11.95(1.46)</td>
<td>-13.83(1.74)†</td>
<td>-14.69(2.04)‡</td>
<td></td>
</tr>
<tr>
<td>ML Knee</td>
<td>0.44(0.16)</td>
<td>0.52(0.16)†</td>
<td>0.55(0.17)†</td>
<td></td>
</tr>
<tr>
<td>AP Ankle</td>
<td>-4.87(0.74)</td>
<td>-5.91(1.05)†</td>
<td>-6.47(1.21)‡</td>
<td></td>
</tr>
<tr>
<td>Axial Ankle</td>
<td>-10.93(1.14)</td>
<td>-12.66(1.10)†</td>
<td>-13.94(1.50)‡</td>
<td></td>
</tr>
<tr>
<td>ML Ankle</td>
<td>-1.18(0.37)</td>
<td>-1.36(0.51)</td>
<td>-1.79(0.77)</td>
<td></td>
</tr>
<tr>
<td>Resultant Hip</td>
<td>7.42(1.29)</td>
<td>9.03(1.43)</td>
<td>10.45(1.53)</td>
<td></td>
</tr>
<tr>
<td>Resultant Knee</td>
<td>11.98(1.46)</td>
<td>13.86(1.75)</td>
<td>14.72(2.05)</td>
<td></td>
</tr>
<tr>
<td>Resultant Ankle</td>
<td>12.03(1.25)</td>
<td>14.04(1.36)</td>
<td>15.46(1.85)</td>
<td></td>
</tr>
</tbody>
</table>
Figure 1. Group ensemble joint reaction forces (BW) during running and their standard deviation bands (BW; dashed line = AP; solid line = axial; dotted line = ML). Positive reaction forces are directed anterior, proximal, and lateral.
Figure 2. Group ensemble net internal joint moments (BWm) during running and their standard deviation bands (for the hip, knee, and ankle dashed line = AP; dotted line = Torsion; solid line = ML). Positive moments correspond to adduction, internal rotation, extension, and supination. Note the different scaling for y-axes between joints.
Figure 3. Group ensemble muscle forces (BW) of representative muscle groups during running and their standard deviation bands (GMAX = gluteus maximus; HAM = hamstring muscles; RF = rectus femoris; VAS = vasti muscles; GAS = gastrocnemius; SOL = soleus; TA = tibialis anterior). Note the different scaling for y-axes between muscles.
Figure 4. Group ensemble joint contact forces (BW) at the hip, knee, and ankle during running and their standard deviation bands (dashed line = AP; solid line = axial; dotted line = ML). Positive contact forces correspond to anterior shear, axial tension, and lateral shear. Note the different scaling for y-axes between joints.
Figure 5. Subject ensemble curves for calculated hip contact force (grey) during walking (1.25 m/s) compared to *in vivo* hip contact force (black) from a 61 year old male walking at the same speed (file KWR0602 from the database OrthoLoad, Bergmann, 2008).
CHAPTER 4. INTERNAL FEMORAL FORCES AND MOMENTS DURING RUNNING: IMPLICATIONS FOR STRESS FRACTURE DEVELOPMENT

Modified from a paper published in *Clinical Biomechanics*

W. Brent Edwards\(^1,3\), Jason C. Gillette\(^1,4\), Joshua M. Thomas\(^2,4\), and Timothy R. Derrick\(^1,4\)

Abstract

Background: Femoral stress fractures tend to occur at the neck, medial proximal-shaft, and distal-shaft. The purpose of this study was to determine the internal femoral forces and moments during running. It was expected that larger loads would occur at these common sites of femoral stress fracture. Methods: Ten subjects ran at their preferred running speed over a force platform while motion capture data were collected. Static optimization in conjunction with a SIMM musculoskeletal model was used to determine individual muscle forces of the lower extremity. Joint contact forces were determined, and a quasi-static approach was used to calculate internal forces and moments along a centroid path through the femur. Findings: The largest mean peak loads were observed at the following regions: anterior-posterior shear, 7.47 bodyweights (BW) at the distal-shaft (posteriorly directed); axial force, 11.40 BW at the distal-shaft (compression); medial-lateral shear, 3.75 BW at the neck (medially directed); anterior-posterior moment, 0.42 BWm at the proximal-shaft (medial surface compression); torsional moment, 0.20 BWm at the distal-shaft (external rotation); medial-lateral moment, 0.44 BWm at the distal-shaft (anterior surface compression). Interpretation: The mechanical loading environment of the femur during running appears to explain well the redundancy in femoral stress fracture location. We observed the largest internal loads at the three femoral sites prone to stress fracture.

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\(^3\)Primary Investigator.  
\(^4\)Co-Investigator.
Introduction

Stress fractures result from repetitive cyclical loading of the skeletal system. Over time the bone fatigues and microdamage manifests as small cracks in the bony matrix (Burr et al., 1985). If the accumulation of microdamage exceeds the rate of bone repair, a stress fracture will result (Burr et al., 1990). Stress fractures commonly occur in the tibia and metatarsals (Korpelainen et al., 2001; Milgrom et al., 1985), but the femur accounts for approximately 10% to 33% of all stress fractures in military recruits (Finestone et al., 1991; Rauh et al., 2006) and approximately 4% to 14% in runners (McBryde, 1985; Sullivan et al., 1984).

In spite of the low relative occurrence of femoral stress fractures, they are among the most serious of overuse injuries. Several months of reduced weight-bearing or non-ambulatory activities may be necessary before training can be resumed (Ivkovic et al., 2006; Pihlajamäki et al., 2006). Early detection is critical, as certain femoral stress fractures have a tendency to displace and require surgical fixation (Lee et al., 2003; Visuri et al., 1988). Any tissue damage resulting from the fracture itself, as well as perioperative trauma can lead to avascular necrosis, osteoarthritis, and in some instances permanent handicap (Lee et al., 2003; Pihlajamäki et al., 2006; Visuri et al., 1988).

There are three femoral locations that are particularly susceptible to stress fracture. Approximately 50% of all femoral stress fractures occur at the neck (McBryde, 1985; Niva et al., 2005). This is followed by a relatively high incidence of proximal shaft fractures in runners that primarily occur on the medial surface (Butler et al., 1982; Hershman et al., 1990; Korpelainen et al., 2001; Lombardo & Benson, 1982), and distal shaft fractures in military personnel that occur between distal one-third and femoral condyles (Giladi et al.,
Previous models of femoral loading fall short of identifying why femoral stress fractures occur in these locations. In part, this may be due to the complex nature of bone loading and the simplifications used when modeling gait. However, even in the most complex of models (Duda et al., 1998; Polgár et al., 2003), extrapolation of the results to running is difficult because joint contact and muscle force inputs are often based on walking.

Traditionally, external reaction forces have been used to experimentally investigate the relationship between mechanical loading and stress fracture development (Bennell et al., 2004; Milner et al., 2006). Although running ground reaction forces typically range between 2 to 3 body weights (BW) (Munro et al., 1987), they comprise only a small portion of the skeletal loading environment. Results from instrumented hip prostheses suggest that muscle forces contribute an additional 3 BW of force during slow jogging (1.1-2.2 m/s) (Bergmann et al., 1993). A recent modeling study estimated that muscles crossing the ankle joint contribute an additional 7 BW of force during running (3.5-4.0 m/s) (Sasimontonkul et al., 2007). Furthermore, the stresses arising from axially oriented muscle loading is small when compared to the stresses on the periphery of the bone due to bending (Biewener et al., 1983).

The purpose of this study was to determine internal femoral loads during running. A combination of experimental and modeling techniques were used to calculate three orthogonal forces and three orthogonal moments acting at 11 equidistant points along the length of the femur. It was expected that the largest internal femoral forces and moments would occur at common sites of femoral stress fracture reported in the literature.
Methods

Subjects

Ten experienced male runners (6 rear-foot strikers, 4 fore-foot strikers) were recruited for this study (age 22.20 ± 3.16yrs, height 1.78 ± 0.05m, mass 69.15 ± 6.48kg). At the time of data collection subjects were free from lower-extremity injury and ran more than 20 miles/week. Prior to participation, subjects gave written informed consent and the study was approved by the Iowa State University Human Subjects Review Board.

Data Collection

Upon arrival, each subject was outfitted with a standardized commercially available running shoe, a black spandex shirt, and black spandex shorts. A series of anthropometric measurements were taken and thirteen retroreflective markers were placed on anatomical landmarks of the trunk and right lower-extremity. All anthropometric measurements and retroreflective marker placements were performed by the same researcher. Preferred running speed was determined over a series of practice trials, during which the subjects ran along the 28.5 m runway of the lab. The subjects were asked to aim for a speed they would select for an 8-10 mile recovery run. Running speed was monitored with motion capture, and the average value of 3-4 practice trials within a range of 5% was considered the preferred running speed.

Subjects ran at their preferred running speed (4.43 ± 0.48m/s) over a force platform (AMTI, Watertown, MA) until ten successful trials were completed. Trials were accepted if the speed was ± 5% of their preferred running speed and the subject’s right foot hit the force platform with no visually identified targeting. Motion-capture data were collected with a Peak Motus 3D optical capture system (Vicon Peak, Centennial, CO) at a sampling
frequency of 120 Hz. Force platform data were collected concurrently at a sampling frequency of 1200 Hz. The synchronized raw motion-capture and force platform data were then exported to Matlab (The Mathworks, Natick, MA) for processing.

Data Processing

The raw motion capture data were interpolated to 1200 Hz using a cubic spline technique and then smoothed using a low-pass zero-lag Butterworth filter with a cutoff frequency of 8 Hz. Ground reaction force data were also smoothed in the same manner with a cutoff frequency of 50 Hz. A static trial was used to estimate joint center locations which were assumed to be stationary in the segmental coordinate systems. Three-dimensional Cardan segment and joint angles were then calculated with a flexion/extension, abduction/adduction, internal/external rotation sequence.

Segment masses, center of mass locations, and moments of inertia were obtained according to Vaughan et al. (1992), using anthropometric measurements acquired prior to data collection. Joint moments and reaction forces were calculated using inverse dynamics and rigid body assumptions. Values were transformed to the distal segment coordinate system of each joint. The segment coordinate systems were described using a right handed rule, with the x-axis oriented in the anterior-posterior (AP) direction, the y-axis oriented in the axial direction, and the z-axis oriented in the medial-lateral (ML) direction.

The stance phase joint angles for each trial were interpolated to percentage of stance (1% increments) and imported into a scaled SIMM 4.0 musculoskeletal model (MusculoGraphics, Inc., Santa Rosa, CA). The reader is referred to Delp and Loan (1995) for a detailed overview of the musculoskeletal modeling software. The SIMM model was used to obtain dynamic maximal muscle forces, muscle moment arms, and muscle orientations for
43 lower-extremity muscles. The maximum dynamic muscle forces were adjusted for length and velocity. The information provided by SIMM was then re-imported into Matlab for the estimation of individual muscle forces.

Muscle forces were optimized using the \textit{fmincon} function in Matlab. The \textit{fmincon} function uses sequential quadratic programming, it begins with an initial guess and searches the solution space in the direction of ‘steepest descent’ such that the magnitude of the cost function is decreased. The cost function ($u$) to be minimized was the sum of squared muscle stresses (Glitsch & Baumann, 1997):

$$u = \sum_{i=1}^{43} \left( \frac{f_i}{PCSA_i} \right)^2$$

where $f_i$ is the force generated by the $i^{th}$ muscle, and $PCSA_i$ is the physiological cross-sectional area of the $i^{th}$ muscle. The optimization was constrained so that the resulting hip, knee, and ankle moments equaled those from inverse dynamics. Five moments were utilized in the optimization procedure including three orthogonal components ($j$) of the resultant moment at the hip, and one (flexion-extension) component at the knee and ankle:

$$M^h_j = \sum_{i=1}^{27} r^h_i \times f_i, \quad j = x, y, z$$

$$M^k_z = \sum_{i=21}^{33} r^k_i \times f_i$$

$$M^a_z = \sum_{i=31}^{43} r^a_i \times f_i$$

where $M^h$, $M^k$, and $M^a$ are the three components of the resultant moment at the hip, and one component each of the resultant moment at the knee and ankle, respectively, and $r^h$, $r^k$, and $r^a$ are the muscle moment arms about the hip, knee, and ankle, respectively. The lower bound
muscle forces were initially set to zero and the upper bound muscle forces were initially set to
the maximal dynamic muscle forces obtained from SIMM. The bounds were then adjusted
in subsequent frames to prevent non-physiological changes in muscle force (Pierrynowski &
Morrison, 1985):

\[
\begin{align*}
l_b_i &= q - \left(1 - e^{-dt/t_{down}}\right) \times q \\
u_b_i &= q + \left(1 - e^{-dt/t_{up}}\right) \times (1 - q)
\end{align*}
\]

where \( l_b_i \) and \( u_b_i \) are the lower and upper bounds for the \( i^{th} \) muscle, \( q = f_i \) divided by the respective maximal dynamic muscle force, \( dt \) is the time step, \( t_{down} = 0.034 \) s, and \( t_{up} = 0.003 \) s. If the predicted muscle force went beyond its bound it was penalized within the cost function such that:

\[
\begin{align*}
l_bpen_i &= 0 \quad \text{if} \quad (f_i \geq l_b_i) \\
l_bpen_i &= \left(\frac{(l_b_i - f_i)}{PCSA_i}\right)^2 \quad \text{if} \quad (f_i < l_b_i) \\
u_bpen_i &= 0 \quad \text{if} \quad (f_i \leq u_b_i) \\
u_bpen_i &= \left(\frac{(f_i - u_b_i)}{PCSA_i}\right)^2 \quad \text{if} \quad (f_i > u_b_i)
\end{align*}
\]

\[
u' = \sum_{i=1}^{43} \left(\frac{f_i}{PCSA_i}\right)^2 + lbpen_i + ubpen_i
\]

where \( lbpen_i \) and \( ubpen_i \) are the penalties for the \( i^{th} \) muscle force, and \( u' \) is the cost function adjusted for the penalty.
Three-dimensional joint contact forces were calculated as the sum of reaction force and muscle forces crossing the joint. Joint contact forces were referenced to the local coordinate system of the distal segment.

\[
F_{c_j}^h = \begin{bmatrix} R_{F_j}^h + \sum_{i=1}^{27} f_{ij} \end{bmatrix} \quad j = x, y, z
\]

\[
F_{c_j}^k = \begin{bmatrix} R_{F_j}^k + \sum_{i=21}^{33} f_{ij} \end{bmatrix} \quad j = x, y, z
\]

where \( F_{c_j}^h \) and \( F_{c_j}^k \) are the three components of the joint contact force at the hip and knee, respectively, \( R_{F_j}^h \) and \( R_{F_j}^k \) the three components of the joint reaction forces at the hip and knee, respectively, and \( f_{ij} \) are the three components of the \( i^{th} \) predicted muscle force crossing the respective joint. The patella-femoral contact force was calculated as the resultant of the quadriceps and patella ligament forces assuming a ratio of 1:1.

Internal forces and moments of the femur were calculated in a similar manner to Duda et al. (1997). The calculations were based on a quasi-static equilibrium of forces and moments at each 1% of the stance phase. The overall equilibrium of the femur was described as:

\[
\sum_{i=1}^{33} f_i + F_{c_j}^h + F_{c_j}^k + F_{c_j}^p + G = 0
\]

\[
\sum_{i=1}^{33} r_i \times f_i + r_{c_j}^h \times F_{c_j}^h + r_{c_j}^k \times F_{c_j}^k + r_{c_j}^p \times F_{c_j}^p + g \times G = 0
\]

where \( F_{c_j}^p \) is patella-femoral contact force, \( G \) is the weight of the thigh, and \( r, r_{c_j}^h, r_{c_j}^k, r_{c_j}^p \) and \( g \) are the corresponding moments arms to the muscle force, hip contact force, knee contact force, patella-femoral contact force and weight component, respectively.
Internal forces and moments were calculated along a centroid path at 11 equidistant points within the femur, beginning at the femoral neck (Fig 1). The vertices defining the femur of the musculoskeletal model were used as a basis for the calculation of centroid path. The loading environment at each point was determined by assuring equilibrium with all forces acting on the section above. Internal forces and corresponding moments were calculated in the thigh coordinate system and then rotated into their respective local-internal femoral coordinate systems. The axial component of the local-internal femoral coordinate systems were described by an axially oriented vector extending towards the point above. The AP axes were calculated as the cross product of the local-internal femoral axial axes and the thigh ML axis. The ML axes were calculated as the cross product of the local-internal femoral axial axes and the local-internal femoral AP axes. The thigh weight component was assumed to be linearly distributed extending from the center of mass towards the proximal and distal ends of the femur.

All data were normalized to BW and analyzed descriptively. Peak internal femoral forces and moments and their time of occurrence were calculated and the mean and standard deviation across subjects was determined. In this paper the first 30% of stance is referred to as the impact phase and the later 70% is referred to as the active phase.

Results

Joint Moments

The joint reaction forces and moments obtained from inverse dynamics are displayed in Figure 2. The axial oriented reaction forces were large in comparison to the AP and ML reaction forces. The sagittal plane moments were primarily extensor, with peak magnitudes occurring near midstance for the knee and ankle, and during the impact phase for the hip. In
addition, a relatively large abduction moment was observed at the hip and knee, and a relatively large inversion moment at the ankle.

**Muscle Forces**

Ensemble average muscle forces for representative muscle groups are displayed in Figure 3. The prominent hip extensors, knee extensors, and ankle extensors tended to follow the general trend of the joint moments. The largest muscle forces were created by the knee extensors. Similar to the joint moments, peak vasti (VAS), rectus femoris (RF), gastrocnemius (GAS), and soleus (SOL) occurred near midstance while peak gluteus maximus (GMAX) activity occurred earlier in stance. Some hamstring (HAM) and tibialis anterior (TA) muscle activity was observed at both early and late stance.

**Joint Contact Forces**

The axial joint contact forces were compressive and large in relation to the other components of the joint contact force (Fig 4). The peak AP joint contact force acted in the posterior direction at the hip (-1.60 ± 0.45 BW) and knee (-1.83 ± 0.08 BW). Peak axial forces were larger at the knee (-15.09 ± 0.59 BW) than at the hip (-11.89 ± 2.19 BW). The peak ML joint contact force acted in the lateral direction at the hip (6.25 ± 0.83 BW) and in the medial direction at the knee (-1.19 ± 0.07 BW). The mean peak resultant patella-femoral contact force was 7.09 ± 0.27 BW.

**Internal Femoral Forces**

The internal forces at 11 equidistant points along the femur are displayed in Figure 5. Point 1 corresponds to the most proximal point analyzed (at the femoral neck) and point 11 corresponds to the most distal point analyzed (at the femoral condyles). Prominent AP shear forces were observed at the proximal and distal ends of the femur. Mean peak AP forces
ranged from 3.57 BW at point 1 to -7.47 BW at point 11 (Table 1). The anteriorly oriented force at point 1 occurred during the impact phase, while the posteriorly oriented force at point 11 occurred during the active phase near midstance.

The axial oriented forces were larger than the AP and ML forces at each point within the femur. Mean peak axial forces ranged from -6.79 BW at point 1 to -11.40 BW at point 11. Peak axial forces at the proximal femur occurred during the impact phase, while those at the middle and distal femur occurred during the active phase closer to midstance.

A prominent ML shear force was observed at the proximal femur. Mean peak ML forces ranged from 1.06 BW at point 10 to -3.75 BW at point 1. The medially oriented force at point 1 occurred during the impact phase, while all other peak ML shear forces occurred during the beginning of the active phase.

**Internal Femoral Moments**

The internal moments at 11 equidistant points along the femur are displayed in Figure 5. Large bending moments about the AP axis were observed during the impact and active phase. The peak AP moments during impact were negative, signifying compression on the medial surface of the femur and tension on the lateral surface of the femur. The peak AP moments during the active phase were negative at the proximal end of the femur and positive at the distal end of the femur. The mean peak AP moments ranged from 0.31 BWm at point 11 to -0.42 BWm at point 2 (Table 2).

The torsional moments were small compared to the AP and ML bending moments. Apart from points 2 and 3, mean peak torsional moments occurred during the impact phase of running. The peak torsional moments would cause internal rotation at the proximal femur and
external rotation at the middle and distal femur. The mean peak torsional moments ranged from 0.15 BWm at point 2 to -0.20 BWm at point 11.

Large ML bending moments were observed during the impact phase and active phase. The peak ML bending moments were all negative signifying compression on the anterior surface of the femur and tension on the posterior surface of the femur. At the proximal femur the peak ML bending moments occurred during impact, but occurred during the active phase at the middle and distal femur. The mean peak ML moments ranged from -0.20 BWm at points 1, 5 and 6 to -0.44 BWm at point 11.

Discussion

The purpose of this study was to determine the internal femoral forces and moments during running, and to find out if larger loads occurred at common sites of femoral stress fracture. The results of our study suggest that frequently cited locations of femoral stress fracture do experience larger loads relative to the rest of the femur.

The etiology of stress fractures is multifactorial and contributing risk factors can be categorized as being either extrinsic (e.g., surface, athletic footwear, training regime) or intrinsic (e.g., bone strength, bone fatigability, bone turnover rate) (Bennell et al., 1999). The combination of risk factors has the potential to vary between each stress fracture occurrence, but it is the interaction of these risk factors with the mechanical loading environment that ultimately leads to injury. The subsequent paragraphs suggest a biomechanical relationship between the loading environment and frequently cited locations of femoral stress fracture.

The femoral neck is subjected to large anteriorly and medially oriented shear forces during running. These shear forces occur early on in stance and are likely related to the breaking force that occurs following heel-strike. Although axial forces and moments at the
neck were no larger than those experienced by the rest of the femur, peak loads always occurred during the impact phase of running. The impact phase of running is associated with a high rate of loading, and microdamage to cortical bone increases proportionally to loading rate (Schaffler et al., 1989). Over time, this type of loading combined with the small diameter of the femoral neck may pose a threat to skeletal integrity.

Femoral stress fractures at the proximal shaft tend to occur on the medial aspect of the femur (Butler et al., 1982; Hershman et al., 1990; Lombardo & Benson, 1982). We found the largest bending moments about the AP axes at the proximal femur during the impact phase of loading. The direction of this bending moment in conjunction with the axially oriented compressive force would place the largest normal stress on the medial aspect of the femur. As cortical bone is strongest in compression (Reilly & Burstein, 1975), the reason that proximal shaft fractures materialize on the medial surface is unclear. It is possible that in an in vivo situation the difference between medial surface compressive stress and lateral surface tensile stress outweighs the difference in anisotropic strength. These differences may become even more pronounced with training when the muscles that resist the AP bending moment (hip abductors) begin to fatigue.

The largest AP shear, axial forces, and ML bending moments were observed at the distal femur near the femoral condyle. Peak loads occurred during midstance and were associated with peak patella-femoral contact force and peak muscular force of the quadriceps and gastrocnemius. In addition, the largest torsional moment was observed at the distal femur, and in-phase combined torsion and axial loading has been shown to cause a seven-fold reduction in cortical bone fatigue life (George & Vashishth, 2005). Recently, the free moment, a vertically oriented torque experienced between the foot and ground, was found to
be a strong predictor of subjects with a history of tibial stress fracture (Milner *et al.*, 2006). It is unclear how influential the free moment is on the torsional moment experienced throughout the femur, but it appears that torsional type loading may play an important role in stress fracture development.

Published research on the internal loading environment of the femur during running is sparse. Internal femoral loads have been directly measured during slow jogging by way of an instrumented femoral replacement (Taylor & Walker, 2001). Forces and moments were telemetered from a location approximating the mid-femur. Peak loads averaging -3.3 BW, -0.08 BWm, -0.01 BWm, and -0.06 BWm were reported for axial force, the AP moment, torsion moment, and ML moment, respectively. Although the directions of these loads are in agreement with our analysis, the magnitudes are on average less than half the peak loads estimated at the mid-femur in this study. Several explanations can be given for this discrepancy. First, the slow jogging speed of 1.8 m/s reported by Taylor and Walker (2001), is below that consistently reported for the walk to run gait transition (2.0 m/s) (Diedrich & Warren, 1995; Hreljac, 1993), and may therefore be more closely related to a brisk walk. Our subjects’ preferred running speed averaged 4.4 m/s, and many loading characteristics during running are more than double those found during walking, such as ground reaction forces and net internal joint moments (Novacheck, 1998). Second, while Taylor and Walker (2001) mention that internal forces from an instrumented femoral replacement may be lower than normal due to reductions and losses in muscle function, the static optimization procedure we used to determine muscle forces has been criticized for over-predicting muscle force magnitudes (Prilutsky *et al.*, 1997). Therefore, it is possible that the internal loads calculated in this study are slightly overestimated. Nevertheless, static optimization shows close
agreement to EMG patterns during locomotion (Crowninshield & Brand, 1981; Heintz & Gutierrez-Farewik, 2007), and compares well with dynamic optimization solutions (Anderson & Pandy, 2001). In addition, the experimental joint moments used to constrain our optimized muscle forces agree well with previous running literature (Derrick et al., 1998; Pollard et al., 2004).

Equally important to the limitations of static optimization are the limitations associated with our generic musculoskeletal model. The model was linearly scaled to the length of each subject’s segments, but this scaling did not account for subject variation in bone morphology. Differences in femoral anteversion and neck-shaft angle could influence the internal loading environment by altering the centroid path as well as muscle moment arms. Knowledge of inter individual bone geometry and strength would also improve our study. These parameters would allow for the estimation of skeletal stresses and strains which have a more direct relationship with stress fracture.

In conclusion, the mechanical loading environment of the femur explains well the redundancy in femur stress fracture location cited in the literature, i.e. the neck, medial proximal-shaft, and distal-shaft. Each of these locations experiences a relatively unique loading environment and it is quite possible that several different mechanisms are responsible for the development of femoral stress fracture. As these internal loads cannot be realized through measurement of external reaction forces, it may be necessary for future research to utilize the technique presented herein in order to better explain the correlation between mechanical loading and stress fracture development.
References


### Tables

Table 1. Mean (SD) peak internal femoral force magnitudes and % stance of occurrence for all subjects and all trials.

<table>
<thead>
<tr>
<th>Femur Point</th>
<th>AP Force</th>
<th>Axial Force</th>
<th>ML Force</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Magnitude (BW)</td>
<td>% Stance</td>
<td>Magnitude (BW)</td>
</tr>
<tr>
<td>1</td>
<td>3.57 (0.14)</td>
<td>22.32 (2.50)</td>
<td>-6.79 (1.33)</td>
</tr>
<tr>
<td>2</td>
<td>-1.91 (0.83)</td>
<td>41.71 (12.13)</td>
<td>-8.40 (0.92)</td>
</tr>
<tr>
<td>3</td>
<td>-1.78 (0.43)</td>
<td>38.57 (10.76)</td>
<td>-8.12 (0.79)</td>
</tr>
<tr>
<td>4</td>
<td>-1.97 (0.13)</td>
<td>34.92 (8.79)</td>
<td>-8.01 (0.51)</td>
</tr>
<tr>
<td>5</td>
<td>-1.62 (0.08)</td>
<td>28.08 (6.02)</td>
<td>-8.66 (0.60)</td>
</tr>
<tr>
<td>6</td>
<td>-1.27 (0.09)</td>
<td>25.50 (5.35)</td>
<td>-9.98 (0.64)</td>
</tr>
<tr>
<td>7</td>
<td>-1.03 (0.09)</td>
<td>21.58 (3.21)</td>
<td>-9.84 (0.67)</td>
</tr>
<tr>
<td>8</td>
<td>1.15 (0.09)</td>
<td>53.24 (9.08)</td>
<td>-9.84 (0.67)</td>
</tr>
<tr>
<td>9</td>
<td>3.04 (0.17)</td>
<td>36.97 (5.26)</td>
<td>-9.37 (0.64)</td>
</tr>
<tr>
<td>10</td>
<td>2.77 (0.16)</td>
<td>36.67 (4.42)</td>
<td>-9.59 (0.66)</td>
</tr>
<tr>
<td>11</td>
<td>-7.47 (0.22)</td>
<td>48.95 (3.73)</td>
<td>-11.40 (0.64)</td>
</tr>
</tbody>
</table>
Table 2. Mean (SD) peak internal femoral moment magnitudes and % stance of occurrence for all subjects and all trials.

<table>
<thead>
<tr>
<th>Femur Point</th>
<th>AP Moment</th>
<th>Torsional Moment</th>
<th>ML Moment</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Magnitude (BWm)</td>
<td>% Stance</td>
<td>Magnitude (BWm)</td>
</tr>
<tr>
<td>1</td>
<td>-0.31 (0.06)</td>
<td>24.65 (6.32)</td>
<td>0.05 (0.01)</td>
</tr>
<tr>
<td>2</td>
<td>-0.42 (0.07)</td>
<td>22.00 (6.66)</td>
<td>0.15 (0.02)</td>
</tr>
<tr>
<td>3</td>
<td>-0.39 (0.04)</td>
<td>22.33 (4.00)</td>
<td>0.11 (0.02)</td>
</tr>
<tr>
<td>4</td>
<td>-0.34 (0.02)</td>
<td>22.97 (3.80)</td>
<td>-0.12 (0.01)</td>
</tr>
<tr>
<td>5</td>
<td>-0.33 (0.02)</td>
<td>24.03 (3.57)</td>
<td>-0.14 (0.01)</td>
</tr>
<tr>
<td>6</td>
<td>-0.32 (0.02)</td>
<td>24.56 (3.07)</td>
<td>-0.15 (0.01)</td>
</tr>
<tr>
<td>7</td>
<td>-0.31 (0.02)</td>
<td>25.94 (3.42)</td>
<td>-0.16 (0.01)</td>
</tr>
<tr>
<td>8</td>
<td>-0.30 (0.03)</td>
<td>27.43 (4.41)</td>
<td>-0.16 (0.01)</td>
</tr>
<tr>
<td>9</td>
<td>-0.29 (0.03)</td>
<td>28.47 (4.92)</td>
<td>-0.19 (0.01)</td>
</tr>
<tr>
<td>10</td>
<td>0.27 (0.03)</td>
<td>34.70 (8.01)</td>
<td>-0.18 (0.01)</td>
</tr>
<tr>
<td>11</td>
<td>0.31 (0.04)</td>
<td>34.00 (7.64)</td>
<td>-0.20 (0.01)</td>
</tr>
</tbody>
</table>
Figure 1: Local-internal femoral coordinate systems for eleven points along a centroid path of the femur. View is approximately 45° between frontal and sagittal plane.
Figure 2: Ensemble average joint reaction forces and moments calculated from inverse dynamics. Positive reaction forces are directed anterior, upward, and lateral. Positive moments correspond to adduction, internal rotation, and extension.
Figure 3: Ensemble average muscle forces for representative muscle groups calculated from computer optimization (GMAX = gluteus maximus; VAS = vasti muscles; RF = rectus femoris; TA = tibialis anterior; HAM = hamstring muscles; GAS = gastrocnemius; SOL = soleus).
Figure 4: Ensemble average joint contact forces at the hip and knee. Positive contact forces are directed anterior, upward, and lateral.
Figure 5: Ensemble average internal forces and moments at the femur. Positive internal forces correspond to anterior shear, tension, and lateral shear. Positive internal moments correspond to lateral-surface compression, internal-rotation torsion, and posterior-surface compression.
CHAPTER 5. EFFECTS OF STRIDE LENGTH AND RUNNING MILEAGE ON A PROBABILISTIC STRESS FRACTURE MODEL

Modified from a paper to be published in Medicine and Science in Sports and Exercise

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Abstract

The fatigue life of bone is inversely related to strain magnitude. Decreasing stride length is a potential mechanism of strain reduction during running. If stride length is decreased the number of loading cycles will increase for a given mileage. It is unclear if increased loading cycles are detrimental to skeletal health despite reductions in strain.

Purpose: To determine the effects of stride length and running mileage on the probability of tibial stress fracture. Methods: Ten male subjects ran overground at their preferred running velocity during two conditions: preferred stride length, and 10\% reduction in preferred stride length. Force platform and kinematic data were collected concurrently. A combination of experimental and musculoskeletal modeling techniques were used to determine joint contact forces acting on the distal tibia. Peak instantaneous joint contact forces served as inputs to a finite element model to estimate tibial strains during stance. Stress fracture probability for stride length conditions and three running mileages (3, 5, 7 miles/day) were determined using a probabilistic model of bone damage, repair, and adaptation. Differences in stress fracture probability were compared between conditions using a 2 x 3 repeated measures ANOVA.

Results: The main effects of stride length ($p = 0.017$) and running mileage ($p = 0.001$) were significant. Reducing stride length decreased the probability of stress fracture by 3\% to 6\%.

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\textsuperscript{6}Technical Consultant.
Increasing running mileage increased the probability of stress fracture by 4% to 10%.

Conclusions: Results suggest that strain magnitude plays a more important role in stress fracture development than the total number of loading cycles. Runners wishing to decrease their probability for tibial stress fracture may benefit from a 10% reduction in stride length.

Introduction

Cyclical loads, such as those experienced by the skeletal system during running, have the potential to cause bone fatigue. Over time, material property degradation takes place (Pattin et al., 1996; Zioupos et al., 1996b) as microdamage manifests as small cracks in the bony matrix (Burr et al., 1998; Schaffler et al., 1989). With sufficient time for bone remodeling, this process may improve bone integrity through adaptation (Burr et al., 1985; Chamay & Tschantz, 1972). However, if the accumulation of microdamage outweighs the rate of bone repair, microcracks may propagate into stress fractures (Burr et al., 1990; Li et al., 1985).

The fatigue life of bone is inversely related to the applied mechanical load (Carter & Caler, 1985; Carter et al., 1981). When strain magnitudes are low, it is believed that microdamage accumulation will be limited and the tissue will have sufficient time to repair microcracks. Conversely, high strain magnitudes increase the rate of microdamage and subsequently overwhelm the repair process (Frost, 1998). Accordingly, identifying loading patterns that minimize strain magnitudes may aid in the prevention of stress fracture.

Decreasing stride length is a potential mechanism for bone strain reduction during running. Surrogate measures of bone strain, such as external ground reaction force and tibial shock, display positive relationships with stride length (Derrick et al., 1998; Mercer et al., 2005). The problem is that overuse injuries, such as stress fracture, are dependent on both
loading magnitude and loading exposure. As stride length reduction results in an increase in loading cycles for a given amount of mileage, it is unclear if such a kinematic adjustment would decrease the likelihood for stress fracture.

The purpose of this study was to determine the effects of stride length and running mileage on the probability of stress fracture at the tibia, a major site for stress fracture development (Korpelainen et al., 2001; Milgrom et al., 1985b). To this end, we investigated two stride lengths (preferred and -10% preferred) and three running regimens (3, 5, and 7 miles/day; 4.83, 8.05, and 11.27 km/day). We hypothesized that decreasing stride length by 10% would reduce the likelihood for stress fracture at each running mileage. We further hypothesized that increasing running mileage would increase the likelihood for stress fracture at each stride length.

Methods

Stress Fracture Model

A probabilistic model of bone fatigue, repair, and adaptation was used to test our hypothesis. A step-by-step account of the equations and constants utilized within the model are introduced below. For a comprehensive review and theoretical development of the model the reader is referred to Taylor (1998), Taylor and Kuiper (2001), and Taylor et al. (2004)

Bone fatigue. The traditional means to describe the fatigue life of a material is with a stress-life plot, or “S-N curve”. A typical S-N curve can be expressed by the standard fatigue equation:

\[ N_f = C\Delta\sigma^{-n} \]  

[1]
where \( N_f \) is the number of cycles to failure, \( \Delta \sigma \) is the stress range, \( n \) is the slope of the S-N curve, and \( C \) is a constant. Carter and Caler (1985) observed a slope of \( n = 6.6 \) for fatigue damage of cortical bone at the large \( N_f \) values relevant to the present study.

Due to inherent differences between experimental testing samples, considerable scatter in the number of cycles to failure at a given stress range can be obtained for similar specimens. Weibull (1951) developed a statistical procedure to deal with the scatter associated with the fatigue of materials. Taylor’s stress fracture model begins with a modified Weibull equation that accounts for stressed volume (Taylor, 1998):

\[
P_f = 1 - \exp \left( - \left( \frac{V_s}{V_{so}} \right) \left( \frac{\Delta \sigma}{\Delta \sigma^*} \right)^m \right) \tag{2}
\]

where \( P_f \) is the cumulative probability that a volume of bone \( V_s \), will fail at stress ranges up to \( \Delta \sigma \). The reference stress range \( \Delta \sigma^* \) is a measure of the material strength defined as the stress range at which the probability for failure is 0.63 for a reference volume \( V_{so} \). For a 96 mm\(^3\) specimen of cortical bone from a relatively young (27 yrs) individual, \( \Delta \sigma^* = 86 \) MPa for an endurance test of \( 10^5 \) cycles to failure (Zioupos et al., 1996a). The Weibull modulus \( m \) expresses the degree of inherent scatter in the material’s fatigue behavior. For human cortical bone \( m = 8 \) (Taylor, 1998).

Equation 2 refers to \( P_f \) at a particular \( \Delta \sigma \) for a fixed number of loading cycles. For our purposes, it is more appropriate to obtain \( P_f \) for a given number of loading cycles at a fixed \( \Delta \sigma \). Different \( \Delta \sigma \) are related to \( N_f \) with equation 1. Dividing \( N_f \) by the number of loading cycles/day gives the time to failure \( t_f \) in days. Replacing \( \Delta \sigma \) with \( t \) gives a Weibull equation of the form:
\[ P_f = 1 - \exp \left[ -\left( \frac{V_s}{V_{so}} \right) \left( \frac{t}{t_f} \right)^w \right] \]  

where \( t_f \) is a function of both the material strength and applied stress range (Taylor et al., 2004). In this equation the Weibull modulus \( w \) is dependent on both the scatter in the data and the slope of the S-N curve (\( w = m/n = 1.2 \)).

Like most materials bone is subjected to loads that vary in amplitude. For example, bone stress during walking will be less than bone stress during running or jumping. To account for variable loading, Taylor and Kuiper (2001) recommended using the concept of equivalent stress, in which a variable amplitude is transformed to an equivalent constant amplitude based on a weighted average procedure:

\[
\Delta \sigma_{eq} = \left( \frac{1}{N_T} \sum_{i=1}^{j} \left( N_i \Delta \sigma_i^n \right)^{1/n} \right)
\]

where \( \Delta \sigma_{eq} \) is the equivalent stress, \( N_i \) is the number of cycles at stress range \( \Delta \sigma_i \), and \( N_T \) is the total number of cycles. This procedure assumes that the order in which the variable stress amplitudes are applied makes no difference on the fatigue life.

When dealing with whole bone \( \Delta \sigma \) and therefore \( P_f \) is not constant throughout the entire bone. Using the finite element method one can obtain a separate \( P_f \) for each element: call this \( P_{f_i} \). If there are \( k \) elements, then \( P_f \) for the whole bone is the probability that any one element will fail, thus (Taylor & Kuiper, 2001):

\[
P_f = 1 - (1 - P_{f_1})(1 - P_{f_2})(1 - P_{f_3}) \ldots (1 - P_{f_k}).
\]

For convenience, elements experiencing similar stresses can be grouped together; Taylor and Kuiper (2001) found that eight groups could be used without significant error. In practice, the
researcher determines a $\Delta \sigma_{eq}$ and corresponding $V_s$ for each of these eight groups prior to $P_f$ calculation.

*Bone repair.* Bone repair is incorporated into the model with the use of a second Weibull equation:

$$P_r = 1 - \exp \left[ - \left( \frac{t}{t_r} \right)^v \right]$$

where $P_r$ is the probability of repair, the reference time for repair $t_r$ is 26 days and the Weibull modulus $v$ is 2 (Taylor *et al.*, 2004). Written in terms of $t$, both $P_f$ and $P_r$ are the cumulative probabilities that failure or repair will occur from time zero to $t$. In order to combine $P_f$ and $P_r$ it is necessary to calculate the differential of $P_f$ with respect to time, or the “probability density function”. The probability density function can be thought of as the probability that failure will take place within a unit time period (e.g. one day). The bone will not fail if sufficient time has elapsed for repair to occur. Accordingly, the probability density function of failure with repair $Q_{fr}$ is:

$$Q_{fr} = Q_f (1 - P_r)$$

where $Q_f$ is the probability density function of failure. The cumulative probability of failure with repair $P_{fr}$ is then:

$$P_{fr} = \int_0^t Q_{fr} \, dt .$$

*Bone adaptation.* Deposition of new bone will occur on the perosteal surface in response to mechanical loading. In turn, the cross sectional area and areal moment of inertia will increase such that the stresses experienced by the bone are reduced over time. In order to
account for bone adaptation, the idea of \( \Delta \sigma_{eq} \) is reintroduced. In the case of adaptation, \( \Delta \sigma_{eq} \) can be calculated in integral form:

\[
\Delta \sigma_{eq} = \left( \frac{1}{t_T} \int_{t_0}^{t_T} \Delta \sigma^a \, dt \right)^{1/n}
\]  

[9]

where \( t_T \) is the total time over which adaptation takes place (Taylor et al., 2004). This is, in fact, the \( \Delta \sigma_{eq} \) utilized within the model to determine the probability of failure with repair and adaptation \( P_{fra} \).

**Experimental Data Collection**

**Subjects.** Ten experienced male runners were recruited for this study (age 22.2 ± 3.2 yrs, height 1.8 ± 0.1 m, mass 69.2 ± 6.5 kg), eight of which were former or current collegiate level cross-country runners. All subjects were free from lower-extremity injury at the time of data collection. Prior to participation, subjects gave written informed consent and the study was approved by the Institutional Human Subjects Review Board.

**Data Collection.** Each subject was outfitted with a commercially available running shoe. A single researcher took anthropometric measurements and placed thirteen retroreflective markers on anatomical landmarks of the subject’s trunk and right lower-extremity. Markers were adhered to the dorsi-foot, heel, medial and lateral malleolus, anterior calf, medial and lateral femoral epicondyle, anterior thigh, left and right greater trochanter, left and right anterior superior iliac spine, and the joint between the fifth lumbar and first sacrum (L5S1). Preferred running speed and preferred stride length were determined over a series of practice trials, during which the subjects ran along a 28.5 m runway. The subjects were asked to aim for a speed they would select for an 8-10 mile recovery run.
Subjects ran at their preferred running speed (4.4 ± 0.5 m/s) over a force platform (AMTI, Watertown, MA) during two conditions including preferred stride length (PSL; 3.1 ± 0.2 m) and 10% reduction in preferred stride length (-10%PSL; 2.8 ± 0.2 m). Speed was monitored with motion capture using the horizontal component of the L5S1 marker. Stride length manipulation was accomplished using tape adhered to the floor. Subjects were asked to land on the tape at a consistent location underneath their foot. Ten trials were performed at each condition and trials were accepted if the speed was ± 5% the preferred running speed and foot placement was visually reliable with tape location.

Motion-capture data were collected with a Peak Motus 3D optical capture system (Vicon Peak, Centennial, CO) at a sampling frequency of 120 Hz. Force platform data were collected at a sampling frequency of 1200 Hz. The synchronized raw motion-capture and force platform data were exported to Matlab (The Mathworks, Natick, MA) for processing.

Musculoskeletal Modeling

Raw motion capture data were smoothed using a 4th order zero-lag Butterworth filter with a low-pass cutoff frequency of 8 Hz. Ground reaction force data were smoothed in the same manner with a cutoff frequency of 50 Hz. A static trial was used to estimate joint center locations and these were assumed to be stationary in the segmental coordinate systems. Three-dimensional Cardan segment and joint angles were calculated with a flexion/extension, abduction/adduction, internal/external rotation sequence. Joint moments and reaction forces were calculated using inverse dynamics and rigid body assumptions. The equations of Vaughan et al. (1992) were used to obtain segment masses, center of mass locations, and moments of inertia.
The stance phase joint angles for each trial were interpolated to 1% increments and imported into a scaled SIMM 4.0 musculoskeletal model (MusculoGraphics, Inc., Santa Rosa, CA). For a detailed overview of the musculoskeletal modeling software see Delp and Loan (Delp & Loan, 1995). The SIMM model was used to obtain maximal dynamic muscle forces, muscle moment arms, and muscle orientations for 43 lower-extremity muscles. The maximum dynamic muscle forces were adjusted for length and velocity. The information provided by SIMM was used to estimate individual muscle forces with a static optimization routine previously described by Edwards et al. (2008).

Briefly, the static optimization routine used sequential quadratic programming. The cost function to be minimized was the sum of squared muscle stresses. Five joint moments determined from inverse dynamics were used to constrain the optimization. These included three orthogonal components of the resultant moment at the hip, and one (flexion-extension) component at the knee and ankle. The lower bound and upper bound muscle forces were initially set to zero and the maximal dynamic muscle forces, respectively. The bounds were adjusted in subsequent frames to prevent non-physiological changes in muscle force (Pierrynowski & Morrison, 1985).

FEBio software (Musculoskeletal Research Laboratories, Salt Lake City, UT; URL: http://mrl.sci.utah.edu/software.php?soft_id=7) was used to perform finite element analysis to calculate tibial strains. The tibia model consisted of 6,340 nodes and 5,391 hexahedral elements (VAKHUM data set; URL: http://www.ulb.ac.be/project/vakhum/); the fibula was not considered. Material properties were assumed to be linear elastic, with cortical bone having an elastic modulus of 17 GPa, trabecular bone an elastic modulus of 1 GPa, and Poisson’s ratio of 0.3. A separate model was created for each subject that was scaled to the
individual’s leg length. Each model was fully constrained at the tibial plateau and a
distributed joint contact force was applied to the distal tibia.

Based on previous research, Sasimontonkul et al. (2007) concluded that
approximately 10% of the ankle joint contact force is borne by the fibula. Therefore, the
tibial contact force for running was calculated as follows:

\[ F_{c_a} = 0.9 \left( F_{R_a} + \sum_{i=31}^{43} f_{ia} \right) \]  \[ [10] \]

\[ F_{c_s} = 0.9 \left( F_{R_s} + \sum_{i=31}^{43} f_{is} \right) \]  \[ [11] \]

where \( F_c \) is the tibial contact force, \( RF \) is the ankle joint reaction force, and \( f_i \) is the \( i^{th} \)
predicted ankle joint muscle force (muscles \( i = 1 \) to 30 do not cross the ankle joint).

Subscripts \( a \) and \( s \) denote axial and AP shear components, respectively. Written in this form
\( RF \) and \( f_i \) are the forces in the leg coordinate system acting on the tibia. The ML shear
component was not considered because subtalar moments were not utilized within the
optimization routine. The mean peak instantaneous tibial contact forces for the ten running
trials were applied to the individual subject-models.

Any cyclical loading, in addition to running, that takes place during daily activity will
contribute to stress fracture development. To account for variable loading due to normal daily
walking activity, tibial strains for normal and fast walking speeds were also determined. This
has the effect of increasing the absolute accuracy in stress fracture prediction with the model.

Walking tibial contact forces for fast walking speeds (0.9 * 4.6 BW) were obtained from
Glitsch and Baumann (1997). The resulting strains were multiplied by a factor of 0.88 to
obtain tibial strains for normal walking speeds, assuming a strain ratio equal to an external ground reaction force ratio between normal and fast walking speeds (Whalen et al., 1988).

**Applying the Stress Fracture Model**

We assume the bone is operating within its elastic range. Therefore, strain is proportional to stress and can be used in the stress fracture model equations provided constants such as \( C \) and \( \Delta \sigma^* \) are converted.

The maximum absolute principal strains for walking and running were obtained for each element. Equation 4 was then used to obtain the element \( \Delta \sigma_{eq} \). For each running condition, three mileages were investigated (3, 5, and 7 miles/day). Running cycles/day were determined by dividing mileage by stride length. Walking cycles for normal (12,240 cycles/day) and fast (16,320 cycles/day) walking in athletic populations were obtained from Whalen et al. (1988) and held constant across running conditions.

We found that we could accurately predict the ratio of compression to bending in our finite element model using beam theory. Assuming a maximum rate of lamellar bone deposition of 4 \( \mu \)m/day (Taylor et al., 2004), effectively changing the cross sectional area and area moment of inertia, we determined an adaptation “strain ratio” for each day. The strain ratio was defined as the ratio of strain after bone deposition to strain with original bone geometry. This ratio was multiplied by the element strains to determine changes in tibial strain over time due to bone adaptation. Equation 9 was then used to determine an equivalent strain for each element that accounted for adaptation.

All elements were separated into eight groups experiencing similar strain levels. The corresponding \( V_s \) for each group were obtained by summing individual element volumes within each group. Using the strain values from the mid-points of each group and the
corresponding $V_s$ (multiplied by 2 to account for both legs), equations 3 and 5 were used to obtain $P_f$. Equations 6-8 were then implemented to obtain $P_{fra}$.

**Statistics**

Peak $P_{fra}$ over the course of 100 days of training were determined for each running and mileage condition. Differences in $P_{fra}$ were compared using a 2 x 3 repeated measures ANOVA (2 stride lengths by 3 running mileages). Statistical analyses were performed in SPSS with the criterion alpha level set to 0.05. In the event of a significant main effect of running mileage we used Bonferroni adjustments to assess pairwise comparisons of the estimated marginal means (alpha = 0.05/3 = 0.017).

**Results**

A 10% reduction in stride length resulted in a corresponding reduction in the peak resultant tibial contact force. For PSL the peak $Fc_a$ and $Fc_s$ were 13.4 ± 1.2 BW and 0.1 ± 0.3 BW, respectively. Corresponding peak values for -10%PSL were 12.7 ± 1.0 BW and 0.1 ± 0.2 BW. Ensemble average tibial contact force profiles for both conditions are displayed in Figure 1.

For each subject the finite element model was primarily loaded in bending with compressive strain on the posterior surface and tensile strains on the anterior surface (Fig 2). Peak compressive principal strains ranged from 2800 to 4800 με during running conditions; peak compressive principal strains during walking were approximately 2.5 to 4.0 times lower.

The $P_{fra}$ peaked and leveled off after approximately 40 days of running (Fig 3). A significant interaction between stride length and running mileage was present ($p = 0.038$). Figure 4 shows that $P_{fra}$ increased with running mileage at a faster rate during PSL. Because
the resulting interaction was codirectional (i.e., the change in $P_{fra}$ with increased mileage was in the same direction for both stride length conditions), interpretation of main effects is both appropriate and meaningful (Hinkelmann, 2004). For peak $P_{fra}$, the main effects of stride length ($p = 0.017$) and running mileage ($p = 0.001$) were significant. Bonferroni-adjusted pairwise comparisons of the estimated marginal means revealed significant differences between all mileages (mileage 3 to 5, $p = 0.008$, mileage 3 to 7, $p = 0.004$; mileage 5 to 7, $p = 0.002$). A 10% reduction in preferred stride length decreased the likelihood for stress fracture by 3% to 6% (Table 1). Increasing running mileage from 3 to 5 miles/day resulted in an increase in stress fracture probability of 4% to 5%. Increasing running mileage from 3 to 7 miles/day resulted in an increase in stress fracture probability of 7% to 10%.

Discussion

The purpose of this study was to determine the effects of stride length and running mileage on the probability of stress fracture at the tibia. The results of this study suggest that a 10% reduction in preferred stride length effectively reduces the likelihood for tibial stress fracture. This is true for weekly running mileages of 21, 35, and 49 miles/week. Not surprisingly, the probability for stress fracture increased with running mileage regardless of stride length condition. However, the rate at which $P_{fra}$ increased with mileage was higher for PSL.

In general, stress fractures occur during the first 2 to 8 weeks of a new training regimen (Burr, 1997). Annual incidence rates of stress fracture for male track and field athletes have been reported and range from approximately 10% to 20% (Bennell et al., 1996; Johnson et al., 1994). These studies found the tibia to be the most common site for stress fracture development. Our mean peak probability for tibial stress fracture across all
conditions ranged from 6% to 20%, with \( P_{\text{fra}} \) peaking around day 40 (5.7 weeks). While this type of agreement between our modeling results and clinical findings is impressive, it would be unrealistic to try and predict actual stress fracture probability in individual runners with the current model. Table 1 illustrates the large between-subject variability found with this model; some explanations for this are presented in detail below. What is reliable with this type of modeling is the relative difference between conditions as indicated by the significant main effects of stride length and mileage.

The clinical implications for these results are clear. Those runners wanting to decrease their likelihood for stress fracture can do so by reducing their stride length by 10%. This reduction would also allow for runners to run an addition two miles/day and maintain the same \( P_{\text{fra}} \). The presence of the interaction further suggests that the benefits of reduced stride length are more pronounced at higher running mileages and this is most likely a direct effect of the nonlinear nature of the standard fatigue equation (equation 1). The difficulty for the clinician is in identifying those runners “at risk” for stress fracture that would benefit from a 10% stride length reduction. Presumably these would be inexperienced runners beginning a weekly running routine, or runners with a history of stress fracture. Poor physical fitness and low physical activity prior to physical training (Shaffer et al., 1999) and a previous history of stress fracture (Milgrom et al., 1985a) are both associated with a higher risk of stress fracture development.

The metabolic cost of locomotion is optimized at preferred stride frequency and therefore preferred stride length (Hamill et al., 1995). If a 10% reduction in stride length were to increase the metabolic cost of running it is likely that muscle fatigue would onset sooner in comparison to a preferred stride length run. It has been argued that tensile muscle
activity produces counteractive bending moments across long bones that help to lessen the peripheral stresses and strains. If the muscles fatigue, strains could potentially increase leading to a higher $P_{\text{fat}}$. Increased strains at the tibia following muscular fatigue have been empirically shown in both humans (Milgrom et al., 2007) and dogs (Yoshikawa et al., 1994). However, the work of Hamill et al. (1995) showed that a 10% reduction in stride length does not significantly change oxygen consumption and heart rate from a preferred stride length condition. We can thus conclude that the change in metabolic cost from a 10% reduction in stride length is negligible and that this type of kinematic adjustment would not accelerate the bone microdamage process through fatiguing muscles. However, if indeed strains were to increase over the course of the run due to muscular fatigue, the effects of running mileage within conditions would be exacerbated.

The basic motor pattern for locomotion is produced at the spinal level in mammals (Pearson & Gordon, 2000). Although these processes are somewhat “automatic” and subconscious, they can be overridden by higher level brain activity for adaptive control. There is recent evidence to suggest that runners can improve faulty kinematics in as little as eight 10 to 30 minute sessions of real-time visual feedback of kinematic data (Crowell III & Davis, 2006). These kinematic adjustments were maintained at a 1 month follow-up free from visual feedback. We feel that a 10% reduction in preferred stride length is a practical kinematic adjustment and one that could be successfully implemented using these types of gait retraining techniques.

There are several limitations that need to be borne in mind when interpreting the results of the current study. Of course most of these limitations are minimized by the study’s cross-over design (i.e., subjects serving as their own control). For example, we did not have
subject-specific musculoskeletal-model bone morphologies and material properties. Although the models were scaled to the subject’s individual segment lengths, subtle differences in muscle orientations and moment arms could have lead to an over- or under-estimation in individual muscle forces and subsequently tibial contact forces. Subject-specific material properties would have increased the accuracy in absolute stress fracture probability, and this may be one reason for the large running strains in certain subjects (i.e., 4800 με). In vivo research suggests that tibial strains rarely reach magnitudes higher than 2000 με at the anterior tibia however, Ekenmen et al. (1998) observed tibial strains of 4200 με at the posterior tibia during forward jumping. Inclusion of individual muscle forces as boundary conditions within the finite element model could have also reduced our strains in specific areas of the tibia. Unfortunately, there is still much work to be done on the most appropriate way to load and constrain the tibia when using the finite element method.

The probabilistic stress fracture model is designed for runners beginning a running regimen. As our subjects were mostly experienced level track and cross-country runners it is likely that the microdamage repair process was already underway when they came in for laboratory testing. The same argument applies to the method we used to incorporate adaptation into the model. Future work could utilize more detailed modeling of bone adaptation, such as those proposed by Beaupre et al. (1990) or Hazelwood and Castillo (2007). Again, these limitations would not be expected to affect the relative differences between conditions and therefore influence the overall outcomes of the study. While the study may have been improved by collecting walking data on our current subject pool, walking data were kept constant between running conditions, and a sensitivity analysis showed that manipulating number of walking cycles/day had little effect on $P_{fra}$. Specifically,
changing the time spent during normal and fast walking from 4 hours/day to 1 hour/day resulted in a percent change in stress fracture probability of less than 1%.

In conclusion, the results of our study suggest that a 10% reduction in preferred stride length is an effective means to reduce the likelihood for stress fracture. Thus, it appears that the benefits of reducing strain with stride length manipulation outweigh the detriments of increased loading cycles associated with a given mileage. These benefits become more pronounced at higher running mileages. Our future work will focus on other practical kinematic alterations that may also reduce the probability for stress fracture.

References


Table 1. Mean (SD) peak probability of failure ($P_{\text{fra}}$). Main effects of stride length ($p=0.017$) and running distance ($p=0.001$) were significant. A significant interaction was also observed ($p=0.038$).

<table>
<thead>
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<th>-10%PSL</th>
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</thead>
<tbody>
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</tr>
<tr>
<td></td>
<td>(0.11)</td>
<td>(0.07)</td>
</tr>
<tr>
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</tr>
<tr>
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<td>(0.11)</td>
</tr>
<tr>
<td>7</td>
<td>0.19</td>
<td>0.13</td>
</tr>
<tr>
<td></td>
<td>(0.18)</td>
<td>(0.15)</td>
</tr>
</tbody>
</table>
Figure 1. Ensemble average tibial contact forces for stride length conditions. Positive values represent axial compressive and anteriorly oriented shear force.
Figure 2. Sagittal views of a representative finite element model of the tibia displaying maximum principal strains (με) during running. The models were loaded in bending and axial compression with compression on the posterior surface (left) and tension on the anterior surface (right).
Figure 3. Ensemble average probabilities of failure ($P_{fr}$) across 100 days of training.
Figure 4. Mean peak probabilities of failure ($P_{fr}$). The likelihood for stress fracture increases with running distance at a faster rate during PSL.
CHAPTER 6. EFFECTS OF RUNNING SPEED ON A PROBABILISTIC STRESS FRACTURE MODEL

W. Brent Edwards¹,⁴, David Taylor²,⁶, Thomas J. Rudolphi³,⁶, Jason C. Gillette¹,⁵, and Timothy R. Derrick¹,⁵

Abstract

Stress fractures result, in part, from the mechanical fatigue of bone. Failure of materials subjected to mechanical fatigue is dependent on both loading magnitude and loading exposure. Decreasing speed is a potential mechanism of strain reduction during running. However, if running speed is decreased the number of loading cycles will increase for a given mileage. It is unclear if these increased loading cycles are detrimental despite reductions in bone strain. Purpose: To determine the effects of running speed on the probability of tibial stress fracture. Methods: Ten male subjects ran overground at running speeds of 2.5, 3.5 and 4.5 m/s. Force platform and kinematic data were collected synchronously. A combination of experimentation and musculoskeletal modeling was used to determine joint contact forces acting on the distal tibia. Peak tibial contact force served as input to a finite element model to estimate tibial strains during stance. Stress fracture probability for each running speed was determined using a probabilistic model of bone damage, repair, and adaptation. Differences in stress fracture probability, as a function of speed, were compared using a repeated measures ANOVA. Results: Decreasing running speed from 4.5 to 3.5 m/s reduced the likelihood for stress fracture by 4% (p=0.01). Decreasing running speed from 3.5 to 2.5 m/s reduced the likelihood for stress fracture by
12% (p=0.01). Conclusions: Runners wanting to reduce their probability for tibial stress fracture may benefit from a decrease in running speed. Because a reduction in running speed was also associated with an increase in the number of loading cycles for a given mileage, it appears that stress fracture development is more dependent on loading magnitude rather than loading exposure.

Introduction

Running is a leisure activity of choice for many. Unfortunately, it is estimated that 26% to 65% of runners, both recreational and competitive, will sustain some form of overuse injury during any given year (Caspersen et al., 1984; Lysholm & Wiklander, 1987; Marti et al., 1988). Stress fractures account for approximately 15% to 20% of overuse injuries (Bennell et al., 1996a; Brubaker & James, 1974). Although stress fractures can materialize within any load-bearing bone, they most frequently develop in the tibia (Korpelainen et al., 2001; Milgrom et al., 1985).

Stress fractures result, in part, from the mechanical fatigue of bone (Burr et al., 1990). Over time, cyclical loading results in material property degradation (Pattin et al., 1996; Zioupos et al., 1996) as microcracks are nucleated within the bony matrix (Burr et al., 1998; Schaffler et al., 1989). If the energy release rate, or “crack driving force”, is greater than the microstructure’s crack resistance, microcracks will propagate into macroracks, or stress fractures. The crack driving force increases with loading magnitude and crack length (Martin et al., 1998). When loading magnitude is low, it is believed that bone will have sufficient time for remodeling; the microcrack will be completely removed or reduced in size, and a stress fracture will not occur. Conversely, high magnitude loading increases the rate of microcrack nucleation and subsequently overwhelms the repair process (Frost, 1998).
Identifying loading patterns that reduce bone strain may therefore aid in the prevention of stress fracture.

The fatigue life of bone, as a function of applied load, can be described using an inverse power-law relationship (Carter & Caler, 1985). Many cycles of low magnitude loading may be as, or more, detrimental than fewer cycles of high magnitude loading. In a recent study, we showed that reducing stride length by 10% at preferred running speed decreased the likelihood for tibial stress fracture (Edwards et al., 2009). This was because the benefits of decreased strain with stride length reduction outweighed the detriments of increased loading cycles required for a given running mileage. Reducing running speed is an alternative kinematic adjustment that has the potential to decrease tibial strain, but also requires an increased number of loading cycles for a given mileage (assuming a positive relationship between running speed and stride length). Consequently, it remains to be seen if reducing running speed decreases the probability of stress fracture.

The purpose of this study was to determine the influence of running speed on the probability of tibial stress fracture. We hypothesized that reducing running speed would decrease tibial strain sufficiently enough to negate the detrimental increase in loading cycles associated with a given running mileage. This would lead to a reduction in the probability of tibial stress fracture with a corresponding decrease in running speed. To answer this question we used a probabilistic model of bone damage, repair, and adaptation. In addition to running, walking data were included within the model because any cyclical loading that takes place during daily activity will contribute to the development of stress fracture.
Methods

Subjects

Ten males were recruited for this study (age 24.9 ± 4.7 yrs; height 1.7 ± 0.1 m; mass 70.1 ± 8.9 kg). At the time of data collection all subjects were free from lower-extremity injury and participated in running, or athletic activity requiring running, on a weekly basis. Prior to data collection, the experimental protocol was approved by the institutional review board and subjects provided written informed consent.

Experimental Data Collection

Subjects arrived at the lab wearing their personal running shoes and were outfitted with tight fitting athletic clothing. A single researcher took anthropometric measurements and placed retroreflective markers on anatomical landmarks of the trunk and right lower-extremity. Markers were adhered to the dorsi-foot, fifth metatarsal, heel, medial and lateral malleolus, distal and proximal anterior calf, posterior calf, medial and lateral femoral epicondyle, anterior and lateral thigh, left and right greater trochanter, left and right anterior superior iliac spine, and the joint between the fifth lumbar and first sacrum (L5S1). A static motion capture trial was collected while the subject stood in the anatomical position to establish joint center locations and segmental coordinate systems.

Subjects were instructed to either walk (1.25 and 1.75 m/s) or run (2.5, 3.5, and 4.5 m/s) overground at five prescribed locomotion speeds; speed order was balanced across subjects. Motion capture (Vicon MX, Vicon, Centennial, CO) and force platform (AMTI, Watertown, MA) data were collected synchronously at sampling frequencies of 160 and 1600 Hz, respectively. Locomotion speed was monitored via motion capture using the horizontal component of the L5S1 marker. The length of the laboratory runway was 28.5 m, but was
only used in its entirety during faster running conditions. Ten trials were performed at each speed. Trials were accepted if the speed was ± 5% of the prescribed speed and the subject’s right foot hit the force platform with no visually identified targeting. During each trial a researcher used a stop watch to measure the time it took for the subject to take three strides. These data were used to determine the average stride frequency and stride length for each condition.

Data Processing

The raw motion capture and force platform data were exported to Matlab (The Mathworks, Natick, MA) for data processing. Using a 4th order zero-lag Butterworth filter, motion capture and force platform data were smoothed at identical cutoff frequencies (Bisseling & Hof, 2006; van den Bogert & de Koning, 1996). For an objective measure, the cutoff frequency corresponded to the 95th percentile frequency of the vertical ground reaction force. The 95th percentile frequency was calculated from the cumulative sum of an integrated power spectral density curve. Three-dimensional Cardan joint and segment angles were determined using a flexion-extension, abduction-adduction, internal-external rotation sequence. The subtalar joint angle was determined in accordance with O’Conner and Hamill (2005). Joint reaction forces and net internal joint moments were determined using standard inverse dynamics with rigid body assumptions. Segment masses, moments of inertia, and center of mass locations were calculated from the equations of Vaughan et al. (1992) using the anthropometrics measured prior to data collection. The subtalar joint moment was estimated by transforming the ankle joint moment into the subtalar coordinate system.
Musculoskeletal Modeling

The stance phase joint angles were interpolated to 101 points using a cubic-spine technique and imported to a SIMM musculoskeletal model (Musculo-Graphics Inc., Santa Rosa, CA) that was scaled to the individual’s segment lengths. The SIMM model (Delp et al., 1990) consisted of 43 lower-extremity muscles and was used to obtain maximum dynamic muscle forces (adjusted for muscle length and velocity), muscle moment arms, and muscle orientations in the segmental coordinate systems (Delp & Loan, 1995).

Individual muscle forces were estimated with static optimization using the `fmincon` function in Matlab. The `fmincon` function uses a gradient based search algorithm to identify optimal solutions. The cost function to be minimized was the sum of squared muscle stresses (Glitsch & Baumann, 1997). The optimization was constrained such that when the muscle forces were multiplied by their respective moment arms the solution equaled experimentally determined joint moments. Six moments were utilized as constraints including the three orthogonal components at the hip, the flexion-extension moment at the knee and ankle, and the subtalar moment. Lower and upper bound muscle forces were initially set to zero and the maximum dynamic muscle forces, respectively. The equations of Pierrynowski and Morrison (1985) were used in subsequent frames to prevent non-physiological changes in muscle force. This optimization routine, apart from the utilization of the subtalar moment, has been previously described by Edwards et al. (2008).

The ankle joint contact force was calculated as the vector sum of the reaction force and muscle forces crossing the joint. It was assumed that 10% of the ankle joint contact force was borne by the fibula (Sasimontonkul et al., 2007). Thus, the contact force acting on the tibia was calculated as:
\[ Fc_j^t = 0.9 \left[ RF_j^a + \sum_{i=31}^{43} f_{ij} \right] \quad j = x, y, z \]  

where \( Fc^t \) is the three components of the joint contact force acting on the tibia, \( RF^a \) is the three components of the joint reaction force acting on the ankle, and \( f_i \) is the three components of the \( i^{th} \) predicted muscle force crossing the ankle joint (muscles \( i = 1 \) through 30 do not cross the ankle).

Maximum tibial strains for each speed were determined using the finite element method. The model consisted of 6,340 nodes and 5,391 hexahedral elements (VAKHUM data set; URL: http://www.ulb.ac.be/project/vakhum/); the fibula was not considered. The peak instantaneous contact force, averaged across trials, was applied as a distributed load to the distal end of the tibia and the proximal end was fully constrained. A separate model was created for each subject that was scaled to the individual’s leg length. Material properties were assumed to be linear elastic, with cortical bone having a Young’s modulus of 18.6 GPa, and trabecular bone a Young’s modulus of 10.4 GPa (Rho, Ashman & Turner, 1993). Poisson’s ratio for both materials was 0.3. Finite element analysis was performed using FEBio software (Musculoskeletal Research Laboratories, Salt Lake City, UT; URL: http://mrl.sci.utah.edu/software.php?soft_id=7).

**Probabilistic Model of Stress Fracture**

The likelihood for tibial stress fracture was determined using a probabilistic model of bone damage, repair, and adaptation. A brief overview of the model and its equations are introduced below; equation constants for the application presented herein have been previously reported (Edwards et al., 2009). For a comprehensive review, theoretical
development, and sample demonstrations of the model the reader is referred to Taylor (1998), Taylor and Kuiper (2001), and Taylor et al. (2004).

The fatigue life of a bone can be described using the standard fatigue equation:

$$N_f = C\Delta\varepsilon^{-n}.$$  \[2\]

Therefore, if we know the strain range $\Delta\varepsilon$, and experimentally derived constants $n$ and $C$, we can determine the number of loading cycles to failure $N_f$. For in vivo applications $\Delta\varepsilon$ equals maximum strain because minimum strain during a stride cycle is close to zero.

Like most materials bone is subjected to loads that vary in amplitude (e.g., walking strain is lower than running strain). The maximum absolute principal strains for walking and running conditions were obtained for each finite element. We then accounted for variable loading due to normal daily activity by determining an equivalent strain $\Delta\varepsilon_{eq}$ for each element (Taylor & Kuiper, 2001):

$$\Delta\varepsilon_{eq} = \left( \frac{1}{N_T} \sum_{i=1}^{j} N_i \Delta\varepsilon_i^n \right)^{1/n}$$  \[3\]

where $j$ is the number of different strain levels $\Delta\varepsilon_i$, $N_i$ is the number of loading cycles at $\Delta\varepsilon_i$, $N_T$ is the total number of loading cycles, and $n$ is the slope of the standard fatigue equation. For each running condition we assumed subjects ran 3 miles/day. Running cycles/day were determined by dividing mileage by average stride length. Assuming 4 hrs of walking/day (Whalen et al., 1988) at both normal (1.25 m/s) and fast (1.75 m/s) walking speeds, we determined walking cycles/day by multiplying walking time by average stride frequency. Within the stress fracture model, the daily time spent walking was held constant across running speed simulations.
Bone adaptation will take place in response to mechanical loading such that an increase in cross sectional area and areal moment of inertia will reduce tibial strain over time. Assuming a maximum rate of lamellar bone deposition of 4 μm/day on the periosteal surface (Taylor et al., 2004), we obtained an adaptation “strain ratio” using equations of beam theory. The strain ratio was defined as the ratio of strain after bone deposition to strain with original bone geometry. This ratio was multiplied by the $\Delta \varepsilon_{\text{eq}}$ to determine changes in tibial strain over time due to bone adaptation. An equivalent strain for each element that accounted for adaptation was then determined (Taylor et al., 2004):

$$\Delta \varepsilon_{\text{eq}} = \left( \frac{1}{t_T} \int_0^{t_T} \Delta \varepsilon_{\text{eq}}^n \, dt \right)^{1/n}$$

[4]

where $t_T$ is the total time over which adaptation takes place. The total time of interest for our application was 100 days.

The $\Delta \varepsilon_{\text{eq}}$, accounting for variable loading and adaptation, can be used within the standard fatigue equation to obtain $N_f$. However, due to inherent microstructural differences in experimental testing specimens, considerable scatter in the fatigue life of bone is present. A common procedure in fatigue mechanics used to determine the probability of failure when there is considerable scatter in a materials fatigue behavior is the Weibull approach (Weibull, 1951). The model used a modified Weibull equation that accounted for stressed volume (Taylor et al., 2004):

$$P_{fa} = 1 - \exp \left[ - \left( \frac{V}{V_{so}} \right) \left( \frac{t}{t_f} \right)^w \right]$$

[5]

where $V_{so}$ is the reference stressed volume, $t_f$ is the reference time until failure at the applied strain level and number of loading cycles/day, and $w$ expresses the degree of scatter in the
material. These constants were derived from experimental fatigue testing literature and allowed us to predict the cumulative probability of failure with adaptation \( P_{fa} \) for a specimen having stressed volume \( V_s \) from time zero to \( t \). Accounting for stressed volume is important when considering whole bone fatigue because small experimental testing specimens are inherently stronger than large specimens (i.e., small specimens have fewer “weak” points) (Taylor, 1998).

As \( \Delta \varepsilon_{eq} \) varies across the entire bone, \( P_{fa} \) is not constant throughout the tibia. Using the finite element method we obtained a separate \( P_{fa} \) for each element: call this \( P_i \). Given \( k \) elements, then \( P_{fa} \) for the entire tibia was the probability that any one element would fail, thus (Taylor & Kuiper, 2001):

\[
P_{fa} = 1 - (1 - P_1)(1 - P_2)(1 - P_3) \ldots (1 - P_k).
\]  

For convenience, elements experiencing similar strains were grouped together; Taylor and Kuiper (2001) found that eight groups could be used without significant error. The corresponding \( V_s \) for each group were obtained by summing individual element volumes within the eight groups. Using the strain values from the mid-points of each group and the corresponding \( V_s \) (multiplied by 2 to account for both legs) we used equation 6 to determine a single \( P_{fa} \) for the entire tibia.

The process of bone repair is somewhat stochastic. For example, the mean time for a basic multicellular unit to sufficiently tunnel through and repair a microcrack is around 18.5 days with an associated standard deviation of 12.95 days. Thus, we described the cumulative probability of bone repair \( P_r \) with a second Weibull equation (Taylor et al., 2004):

\[
P_r = 1 - \exp \left[ - \left( \frac{t}{t_r} \right)^v \right]
\]  

[7]
where \( t_r \) is the reference time for repair and \( v \) expresses the degree of scatter in repair time. By determining the probability that bone will not repair itself \((1-P_r)\) and multiplying it by the instantaneous probability that failure with adaptation will take place (time differential of \( P_{fa} \)), we obtained an instantaneous probability that accounted for failure, repair and adaptation; integrating with respect to time gave the cumulative probability of failure with repair and adaptation \( P_{fra} \).

**Statistics**

Peak \( P_{fra} \) over the course of 100 days was determined for each subject and running speed. Differences in peak \( P_{fra} \), as a function of running speed, were compared using a one-way repeated measures ANOVA. Statistical analyses were performed in SPSS with the criterion alpha level set to 0.05. In the event of a significant main effect of running speed we used Bonferroni adjusted post-hoc comparisons (alpha = 0.05/3 = 0.017).

**Results**

In general, the joint contact force acting on the distal tibia increased with both walking and running speed (Figure 1). The axial component, directed along the longitudinal axis of the tibia, was the dominant force. The anterior-posterior (AP) component was only slightly larger than the medial-lateral (ML) component. The mean peak instantaneous tibial contact forces used as inputs to the finite element models are displayed in Table 1. At the instant of peak instantaneous resultant force the shear components were directed posteriorly and laterally.

During both walking and running, the tibia was loaded in bending and axial compression. This type of combined loading resulted in compressive principal strain on the posterior surface that was larger than the tensile principal strain on the anterior surface.
Peak principal strains increased with locomotion speed (Table 2). Mean peak walking strains ranged from 1330 to 1422 με, while mean peak running strains ranged from 3805 to 4864 με.

Assuming 4 hours/day of walking at 1.25 and 1.75 m/s, average stride frequency resulted in daily walking exposures of 12,075 and 13,922 cycles/day, respectively (Table 2). Due to the positive relationship between locomotion speed and stride length, the number of loading exposures decreased with running speed. Assuming a daily running regimen of 3 miles/day, the number of loading exposures were 2435, 1829, and 1549 cycles/day for 2.5, 3.5, and 4.5 m/s, respectively.

The probability of failure $P_{fra}$ peaked and leveled off after approximately 40 days of training (Figure 3). A linear reduction in running speed resulted in a corresponding non-linear reduction in $P_{fra}$. Specifically, decreasing running speed from 4.5 to 3.5 m/s reduced mean $P_{fra}$ by 4% ($p = 0.01$), while decreasing running speed from 3.5 to 2.5 m/s reduced mean $P_{fra}$ by 12% ($p=0.01$; Table 2). The large standard deviations shown in Table 2 indicate that $P_{fra}$, and therefore stress fracture probability, was highly variable between subjects. Despite the large between subject variability, the within subject response was consistent across running conditions with the exception of one case for subject 10 (Figure 4). For subject 10, changing running speed from 4.5 to 3.5 m/s resulted in a small increase in $P_{fra}$ of 0.5%.

Discussion

The purpose of this study was to determine if decreasing running speed reduced the probability of tibial stress fracture. Our hypothesis that reducing running speed would decrease tibial strain sufficiently enough to negate the detrimental increase in loading cycles
associated with a given running mileage was supported by the results of this study. More specifically, a linear reduction in running speed resulted in a corresponding non-linear reduction in the probability of tibial stress fracture.

Stress fractures tend to materialize during the first 2 to 8 weeks of a new training regimen (Burr, 1997). This time frame is consistent with our cumulative probability of failure $P_{fra}$ peaking and leveling off after approximately 5.7 weeks, or 40 days of training. Our mean peak probabilities of failure ranged from 17% to 33%. These values are only slightly higher than the 10% to 20% annual incidence rate of stress fracture in male track and field athletes (Bennell et al., 1996b; Johnson et al., 1994).

In our previous publication, analyzing the effects of stride length manipulation on stress fracture probability, we reported a 10% mean probability of tibial stress fracture for subjects running 3 miles/day at 4.4 m/s (Edwards et al., 2009). This value is considerably lower than the 33% mean probability of tibial stress fracture reported here for subjects running 3 miles/day at 4.5 m/s. Several explanations can be given for this discrepancy: First, our previous study did not include the subtalar moment within the muscle optimization and therefore the axial tibial contact force was slightly higher in this study leading to larger compressive strains (present mean value of 13.8 BW compared to a previous mean value of 13.4 BW). Second, we previously excluded the medial-lateral component of the tibial contact force as a finite element boundary condition. This extra shear component aligned the resultant contact force further away from the longitudinal axis of the tibia, creating a larger bending moment leading to increased flexural strain. Finally, viewing the individual subject responses in Figure 4, the reader will notice that 3 subjects (5, 7 and 10) had substantially large probabilities of failure. Although the tibial contact forces of these subjects were no
larger than the rest of the group when normalized to units of BW, the resulting strains were much larger. This suggests, perhaps, a problem with the allometric scaling of the finite element models for these subjects, as well as our assumption of generic bone material properties (this limitation will be addressed further below). Recalculation of the mean peak $P_{\text{fr}}$, with the exclusion of the three outlying subjects, resulted in a mean probability of failure of 15.6% for the 4.5 m/s condition. This is a much smaller discrepancy compared to our previous study which can be explained solely by the inclusion of the subtalar moment and the medial-lateral shear component in the present study.

Little concern should be given to our potential overestimations in probability magnitudes, as the model is not specific enough to predict absolute stress fracture occurrence for individual subjects. The absolute values in stress fracture probability are heavily dependent on the accuracy in tibial strains obtained from the finite element method, an analysis technique of some uncertainty that has not been extensively used to examine the tibia. Merit should therefore be given to the relative differences in stress fracture probability between conditions. The significant effect of running speed suggests a reliable response for stress fracture probability within subjects. The model can thus serve as a useful tool for predicting the influence of practical kinematic alterations on stress fracture probability, without placing particular emphasis on the absolute magnitudes of stress fracture prediction.

This statement is specific to the type of experimental design utilized herein where subjects served as their own control and does not apply to case-control designs aimed at examining differences between separate groups.

Subject 10’s probability for tibial stress fracture reduced with speed from 3.5 to 4.5 m/s and this outlying response warrants further discussion. Although this subject displayed
an increase in joint contact force, and therefore tibial strain, with increased running speed, his relative change in loading magnitude was lowest for the group, namely 2.3%. Additionally, subject 10 did not display a marked difference in loading cycles/day compared to the group mean. This response is directly in line with the impetus for this study, suggesting that a reduction in loading magnitude is not always synonymous with a reduction in stress fracture probability. Rather, there exists a required reduction in strain magnitude necessary to negate the detrimental increase in loading cycles for a given mileage. For the running speeds and mileage relevant to the current study, our statistical results suggest that a speed decrease of 1 m/s is sufficient to reach the required reduction in strain magnitude. It is unclear if similar conclusions would be obtained at faster running speeds due to the observed non-linear increase in stress fracture probability with speed.

Changes in stride length naturally occur with changes in running speed. There is evidence to suggest that the observed positive relationship between external ground reaction force and running speed is more related to resulting changes in stride length rather than changes in speed itself (Mercer et al., 2005). If this relationship were to hold true for joint contact forces, and runners reduced their speed while maintaining a fixed stride length, reductions in running speed would not be associated with corresponding reductions in tibial strain. The number of loading cycles for a given mileage would not change, and consequently, the probability of tibial stress fracture would stay the same. For this reason, future research should focus on which factor, running speed or stride length, is the critical determinant for joint contact force magnitude.

This study is limited by our inability to account for certain subject specific parameters in our modeling procedures. Even though the musculoskeletal model was scaled to the
individual’s segment lengths, an underestimation in muscle moment arms can lead to an overestimation in muscle forces when using static optimization. \textit{In vivo} Achilles tendon forces across a range of running speeds have been directly measured with the use a “buckle”-type transducer (Komi, 1990). A least-squares fit of these data would predict peak Achilles tendon forces of 7.5 to 9.5 BW between running speeds of 3.5 and 4.5 m/s. Adding a reaction force of 2.5 BW one can obtain a conservative ankle joint contact force estimate between 10 and 12 BW. These values are slightly less than the tibial contact forces reported for this study. These differences can be easily explained by the inclusion of the non-triceps surae muscles within our model, and thus, we feel our measure of tibial contact force to be valid.

Our largest error in absolute stress fracture probability most likely stems from potential error in the calculation of tibial strains. Our mean running strains ranged from 3805 to 4864 $\mu$e. These values are on the high side of reported \textit{in vivo} tibial strains, which can reach 4200 $\mu$e on the posteromedial surface of the tibia during forward jumping (Ekenman \textit{et al.}, 1998). Potential error in our calculated tibial strains may be attributed to our inability to account for subject specific bone size and morphology. Even though each individual finite element model was linearly scaled in all directions to leg length, there is evidence to suggest bone diameter should be allometrically scaled to body mass (van der Meulen & Carter, 1995). This could explain the large strains, and therefore $P_{fra}$ for subjects 5, 7, and 10, as these three subjects weighed on the heavy side of the group mean. Assignment of generic bone material properties could have lead to error in our estimation of tibial strain, as well as our neglecting to include individual muscle forces within the finite element model. Tensile forces in muscles can create counteractive bending moments along long bones (Lu \textit{et al.}, 1997) thereby reducing peripheral stresses and strains. All these limitations would only be
expected to change our absolute magnitudes in stress fracture prediction and not the relative differences between conditions. Thus, the generalizability and interpretation of results would not be expected to change by eliminating the aforementioned limitations.

To conclude, the probabilistic model of bone fatigue, repair, and adaptation is designed for individuals beginning a weekly running regimen. Our results suggest that reducing running speed is an effective kinematic adjustment that can be implemented during the regimen’s initial stages to reduce the probability for tibial stress fracture. Because a reduction in running speed is also associated with an increased number of loading cycles for a given mileage, it appears that stress fracture development is more dependent on loading magnitude rather than loading exposure.

References


Table 1. Mean (SD) tibial contact force across locomotion speeds used as inputs to the finite element models. Values represent the instant of peak resultant force. Positive numbers indicate anterior shear, axial compression, and lateral shear.

<table>
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<tr>
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<th>Anterior-Posterior</th>
<th>Axial</th>
<th>Medial-Lateral</th>
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<tbody>
<tr>
<td>Walking (m/s)</td>
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<td></td>
</tr>
<tr>
<td>1.25</td>
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<td>1.75</td>
<td>-0.08(0.04)</td>
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<tr>
<td>Running (m/s)</td>
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<tr>
<td>2.5</td>
<td>-0.53(0.06)</td>
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<td>0.51(0.18)</td>
</tr>
<tr>
<td>3.5</td>
<td>-0.62(0.07)</td>
<td>12.63(1.19)</td>
<td>0.61(0.21)</td>
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<tr>
<td>4.5</td>
<td>-0.66(0.09)</td>
<td>13.80(1.63)</td>
<td>0.68(0.22)</td>
</tr>
</tbody>
</table>
Table 2: Mean (SD) absolute principal strain, daily loading exposure, and probability of failure $P_{fra}$ across locomotion speeds. Walking data were held constant within the probabilistic stress fracture model for each running speed condition. * = significantly different from all other running speeds ($p<0.017$).

<table>
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<th>Walking (m/s)</th>
<th>Running (m/s)</th>
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<tr>
<td>Loading Exposure</td>
<td>12074.69</td>
<td>13921.86</td>
</tr>
<tr>
<td>(cycles/day)</td>
<td>(757.27)</td>
<td>(584.13)</td>
</tr>
<tr>
<td>Probability of</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>Failure ($P_{fra}$)</td>
<td>N/A</td>
<td>N/A</td>
</tr>
</tbody>
</table>
Figure 1. Ensemble average tibial contact forces across locomotion speeds. Positive numbers indicate anterior shear, axial compression, and lateral shear. Note that y-axes scaling is twice as large for running.
Figure 2. Sagittal views of a representative finite element model of the tibia displaying maximum principal strains (με) during running at 3.5 m/s. The models were loaded in bending and axial compression with compression on the posterior surface (right) and tension on the anterior surface (left).
Figure 3. Ensemble average probabilities of failure ($P_{fra}$) for 100 days of training.
Figure 4. Within subject response for peak probabilities of failure ($P_{fra}$).
CHAPTER 7. SUMMARY AND RECOMMENDATIONS

Summary

There is a dearth of information related to the internal mechanical loads experienced by the musculoskeletal system during running. This is an important area of research. Running is a popular activity of choice for many, and a necessity for athletes and military personnel. The positive physiological adaptations associated with running are well established, and these adaptations can only be exploited if runners remain free from overuse injury. This dissertation utilized a combination of experimental and musculoskeletal modeling techniques to investigate internal structural loading of the lower extremity during running. Specific emphasis was placed on stress fracture development, a common overuse injury that results, in part, from the mechanical fatigue of bone.

A series of studies were presented that addressed the influence of speed on lower-extremity contact forces during running, the relationship between internal femoral loads and stress fracture development, and changes in the probability of tibial stress fracture from practical alterations in kinematics and running mileage. The findings of these studies can be summarized as follows: 1) musculoskeletal models provide meaningful estimations of joint contact forces in healthy young adults; 2) lower-extremity contact forces increase with speed during running; 3) stress fractures tend to occur at femoral locations experiencing the largest mechanical loads; 4) the probability of tibial stress fracture increases with stride length and running mileage for a given speed; and 5) the probability of tibial stress fracture increases with running speed.

The purpose of the first study was to determine the influence of speed on lower-extremity joint contact forces during running. We observed a positive relationship between
running speed and joint contact forces. This suggests that injury potential increases with running speed as well. To verify that our modeling procedures provided reasonable estimates of joint contact forces during running, we also calculated contact forces during walking and compared them to those directly measured in vivo. Our calculated contact forces compared favorably to those directly measured with an instrumented prosthetic in a 61 year old male walking at an identical speed. Time series profiles were similar in shape and form, and discrepancies in peak magnitudes could be explained by inherent differences in neuromuscular strategies between older adults that have undergone total joint replacements and young healthy adults. While it cannot be stated definitively, it appears that musculoskeletal modeling provides accurate and meaningful estimations of joint contact forces. As overuse injury potential cannot be truly realized through surrogate measures of joint loading, such as external ground reaction force and net internal joint moments, future work on running injuries would benefit from the procedures discussed herein.

The purpose of the second study was to determine the internal femoral forces and moments during running, and to find out if larger loads occurred at common sites of femoral stress fracture. The results of this study illustrated that frequently cited locations of femoral stress fracture do indeed experience larger loads relative to the rest of the femur. This finding further emphasizes the precision and sensitivity of our modeling procedures. Despite the fact that the etiology of stress fracture is multifactorial, these results suggest that mechanical loading plays a fundamental role in their development. Thus, the ability to quantify and monitor the mechanical loading environment experienced by the skeletal system may aid the prevention of stress fracture.
Studies one and two dealt with peak instantaneous measures of loading. However, because stress fracture is caused by the mechanical fatigue of bone, it is dependent on both loading magnitude and loading exposure. Many cycles of low magnitude loading may be as or more detrimental than few cycles of high magnitude loading. Reducing stride length is a potential mechanism of load reduction, but it requires an increase in the number of loading cycles for a given mileage. The purpose of study three was to determine the influence of stride length and running mileage on the probability of tibial stress fracture. To answer this question we used a probabilistic model of bone damage, repair, and adaptation. The results of this study suggested that a 10% reduction in preferred stride length is an effective means to reduce the likelihood for tibial stress fracture. The probability for tibial stress fracture also increased with running mileage. Interestingly, however, the relative decrease in the probability of tibial stress fracture with stride length reduction became more pronounced with running mileage.

Reducing running speed is another potential mechanism of load reduction that requires an increase in the number of loading cycles for a given mileage. The purpose of study four was to determine the influence of running speed on the probability of tibial stress fracture. Similar to study three, we answered this question using a probabilistic model of bone damage, repair, and adaptation. The results of this study suggested that reducing running speed is an effective way to decrease the likelihood for tibial stress fracture. Taking into account the findings from both studies three and four, it would appear that stress fracture development is more dependent on loading magnitude rather than loading exposure.

The information gained from this dissertation has applications to long-distance runners, athletes, and military personnel wanting to lessen their chances for stress fracture
development. As the probability for stress fracture increases with stride length, running speed, and mileage, the inexperienced runner should begin their training with shorter distances at a slow pace. Breaking up the running regimen into several short bouts during the week would improve bone integrity through adaptation (Robling, Hinant, Burr & Turner, 2002). As the bone adapts to the mechanical loads placed upon it over the course of the first few months, training volume and mileage could be increased, with shorter stride lengths being utilized during longer distance runs. Similarly, experienced runners with already high training volumes could lessen their probability for stress fracture by taking shorter strides and running at a slower pace. For high level intensity training, experienced runners could run shorter distances at a faster pace. Reducing stride length during this type of training would also reduce the experienced runner’s probability of stress fracture, and any potential increase in oxygen uptake from the increase in cadence would allow for an improved cardiovascular workout. By definition, overuse injuries such as stress fracture result from cumulative tissue trauma through repetitive use. Stress fractures rarely occur without premonitory symptoms of injury (e.g., localized pain, “hot spots”, etc.). For this reason runners are encouraged to follow the words of van Mechelen (1992), and “listen and respect the language of their body”. When pain begins, training should be ceased; bone remodeling will take place, the fatigue damage will be repaired, and training can be resumed.

Recommendations for Future Research

This dissertation dealt with practical kinematic alterations with the potential to reduce the probability of stress fracture. Adjustments in running speed and stride length were of primary interest. Changes in stride length naturally occur with changes in running speed. There is evidence to suggest that the observed positive relationship between external ground
reaction force and running speed is more related to resulting changes in stride length rather than changes in speed itself (Mercer et al., 2005). It is unclear if a similar relationship exists for joint contact forces. For this reason, future research should focus on which factor, running speed or stride length, is the critical determinant for joint contact force magnitude.

The ultimate goal of this research is to be able to predict absolute stress fracture probability on a subject specific basis so that running regimens can be optimized to the individual. This would allow for runners to maximize the positive benefits of running and minimize the occurrence of injury. The main factor preventing absolute predictions of stress fracture occurrence is the accuracy in mechanical load estimation both in terms of loading magnitude and exposure. Subject specific musculoskeletal geometry, muscle force-generating capacity, bone material properties, and daily loading patterns would greatly increase the accuracy in stress fracture predication. Future work could utilize advanced imaging techniques, dynomometry, and activity monitoring to obtain this information.

The finite element method is an analysis technique of some uncertainty that has not been extensively used to examine the tibia. Work is needed to determine the most appropriate way to load and constrain the tibia so that accurate measures of tissue strains are obtained. In addition, the finite element approach used in this dissertation quantified tissue strains at a single instant in time corresponding to peak instantaneous resultant force. As a result, we were unable to account for strain rate (i.e., change in strain/time) within the probabilistic model, which can have a strong influence on bone microdamage accumulation (Schaffler et al., 1989). Future work could rely on time-dependent finite element modeling procedures that allow for strain rate calculation.
The methods we employed to account for bone adaptation were simplified. We assumed 4 μm/day of lamellar bone deposition on the periosteal surface regardless of the running condition. Bone adaptation is dependent upon loading magnitude (Rubin & Lanyon, 1985), loading frequency (Rubin & Lanyon, 1984), loading rate (O'Conner & Lanyon, 1982), and timing between loads (Robling et al., 2001) and loading bouts (Robling et al., 2000). The accuracy in stress fracture probability would be improved by accounting for these parameters within the probabilistic model. Perhaps, a more sophisticated finite element modeling procedure that includes time based functional adaptation of element material properties is warranted. This approach could utilize the principles of bone mechanosensitivity to determine an osteogenic index based on osteocyte activation, saturation, and recovery (Turner & Robling, 2003).

Finally, the probabilistic model used in this dissertation assumed that stress fracture development can be entirely explained by mechanical fatigue. In this circumstance bone remodeling is believed to prevent stress fracture. However, there is growing support for a new theory in which bone remodeling plays a role in both preventing and promoting stress fracture development (Burr, 1997; Martin, 1995). In this hypothesis, microcracks create areas of high localized strain, thereby signaling for bone repair. The amount of osteonal remodeling is therefore proportional to the accumulation of microdamage. High levels of microdamage cause accelerated remodeling, which results in increased bone porosity as the osteoclasts tunnel through the damaged area. This transient reduction in bone mass further increases localized strain and the positive feedback loop continues until stress fracture occurs. Further development of the probabilistic model could incorporate this theory by allowing bone remodeling to be mediated by microdamage accumulation. Perhaps a critical
threshold could be determined, at which point accelerated remodeling takes place thereby promoting, rather than preventing, stress fracture development.

References


