Gait Changes During Exhaustive Running

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GAIT CHANGES DURING EXHAUSTIVE RUNNING

A Thesis Presented

by

NATHANIEL SMITH

Submitted to the Graduate School of the University of Massachusetts Amherst in partial fulfillment of the requirements for the degree of

MASTER OF SCIENCE

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Department of Kinesiology
GAIT CHANGES DURING EXHAUSTIVE RUNNING

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ABSTRACT

GAIT CHANGES DURING EXHAUSTIVE RUNNING

FEBRUARY 2016

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Runners adopt altered gait patterns as they fatigue, and literature indicates that these fatigued gait patterns may increase energy expenditure and susceptibility to certain overuse injuries (Derrick et al., 2002; van Gheluwe & Madsen, 1997). Investigations conducted to date have described changes in muscle performance (Avela & Komi, 1998) and kinematic gait variables (Siler & Martin, 1991) resulting from running fatigue, but the kinetic gait variables that span these realms have received little attention. The purpose of this investigation was to characterize changes in joint moment patterns that develop as runners fatigue in order to better understand the kinetic bases for previously reported kinematic fatigue effects. It was hypothesized that as fatigue accumulates over the course of a submaximal run, runners would demonstrate: increased stance time, increased peak stance phase knee flexion angle, increased peak knee extension moment, increased peak swing phase hip flexion moment, and increased hip extension and plantarflexion angular impulse generated during the stance phase. Eight recreational rearfoot-striking runners (height = 170.0 ± 9.8 cm; mass = 69.6 ± 15.1 kg) ran on a force-instrumented treadmill at a velocity 5% slower than that of lactate threshold (determined on a previous visit) until volitional exhaustion. Force and motion data
were collected for ten seconds each minute. Data from the first minute, 20%, 40%, 60%, 80% of run duration and the final minute were analyzed. Repeated measures ANOVA were used to test for significant differences in dependent variable values, and regression analyses were used to test for significant trends in the data over the course of the run. Of the six hypotheses, only stance time changed significantly (p = 0.004), increasing in a linear manner over the course of the run ($r^2 = 0.946$). The significance of changes in joint moment variables was limited by high variability among participant responses to fatigue. Of the remaining hypotheses, regression analyses indicated that peak knee flexion ($r^2 = 0.899$) and hip extension angular impulse ($r^2 = 0.608$) followed increasing trends over the course of the run. Changes in the magnitude and timing of joint moments offer potential explanations for two kinematic trends which may have contributed to an increase in vertical loading rate (p = 0.04). Individual participants often demonstrated joint moment trends which explained joint angle trends and reflect changes reported in the literature, despite inconclusive group results. As fatigue accumulates during a submaximal run, runners demonstrate altered timing of joint moments during swing phase, which result in changes in segment orientation at initial contact. These kinematic changes have the potential to alter ground reaction force patterns and place the runner in a position of deeper joint flexion which may necessitate higher magnitude extensor joint moments to counter and may reduce running economy as fatigue builds.
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CHAPTER 1

INTRODUCTION

The accumulated fatigue induced by a run of sufficient intensity and duration causes notable changes in running form. Fatigue is a failure to maintain muscular force during sustained or repeated contractions (Gibson & Edwards, 1985). In the context of running, this impaired force-producing capacity of muscle underlies the changes in running form observed in gait analyses of fatigued runners. Past researchers have documented changes in several mechanical variables in response to running-induced fatigue, including lower-extremity kinematics, limb stiffness parameters, and the dynamics of muscular force development (Avela & Komi, 1998; Avela, Kyröläinen, Komi, & Rama, 1999; Clansey, Hanlon, Wallace, & Lake, 2012; Derrick, Dereu, & McLean, 2002; Dutto & Smith, 2002; Farley & Gonzalez, 1996; Nicol, Komi, & Marconnet, 1991; Siler & Martin, 1991).

In addition to providing descriptions of these mechanical changes, prior research has shown that certain fatigue-induced changes may constitute risk factors for the development of overuse injuries (Clansey et al., 2012; Mizrahi, Verbitsky, & Isakov, 2000; Zadpoor & Nikooyan, 2011). Previous investigations have also demonstrated that when fatigued, runners alter their stride frequency and assume a posture of increased lower limb flexion. Each of these changes has been shown to independently reduce running economy (Hamill, Derrick, & Holt, 1995; McMahon, Valiant, & Frederick, 1987). Thus, improving our understanding of the mechanisms of fatigue in running is important, as it has implications for both performance and
injury risk. The kinetic and kinematic manifestations of fatigue are the end result of the cascading effects of accumulated metabolites, damage to muscle fibers, and reduced neural drive (Kyröläinen et al., 2000; Millet, Martin, Lattier, & Ballay, 2003; Skof & Strojnik, 2006). While changes in mechanical variables help describe the manifestations of running-induced fatigue, the relationships among these outcomes and the impaired muscle function that characterizes fatigue remain unclear.

Joint moments represent the non-invasive measure most directly related to the forces generated by muscles during movement, but surprisingly have not been studied during fatigued running. Joint moments are defined as the net angular effect of the forces exerted across a joint, primarily by muscles, and thus may be considered to be the mechanical cause of the movements comprising gait (Vaughan, 1996). If coordination of leg muscles is affected by fatigue, then changes in the muscular forces used to complete a fatiguing run should be readily apparent as changes to joint moment profiles. Past literature indicates that strenuous running decreases the isometric (Nicol et al., 1991) and isokinetic (Denadai, Greco, Tufik, & de Mello, 2007) force-producing capacity of muscle, and that this impairment is present after runs as short as twenty minutes (Skof & Strojnik, 2006). Changes in joint kinematic and limb stiffness variables, described below, also suggest that joint moment patterns may differ substantially in the fatigued state; however, at present we can only speculate as to the nature of these differences.

The most recognizable gross-motor indicators of fatigue are changes in kinematics. Studies investigating kinematic changes typically employ a treadmill run of constant velocity, ending when participants become too exhausted to
continue. At the conclusion of one such fatiguing run, Derrick and colleagues (2002) observed significant increases in knee flexion angle at heelstrike and at midstance as well as increased ankle range of motion in the frontal plane. Siler and Martin (1991) also reported this trend towards increased lower extremity flexion as a result of fatigue, along with increased stride length and increased hip joint range of motion in the sagittal plane. The increased limb flexion and range of motion generally reported in kinematic studies imply an increased compliance of the body as a result of fatigue. Taken alone, increased limb flexion implies lengthening of the moment arm of the ground reaction force vector about the knee joint. While the magnitude of the ground reaction force vector will also affect joint moments, this implied change in moment arm length would tend to increase the magnitude of extensor moments at the knee joint (Derrick, Hamill, & Caldwell, 1998).

The stiffness of a runner’s leg can be calculated by dividing the peak vertical ground reaction force magnitude by the change in length of the leg between the instants of heelstrike and midstance. As each of these values can independently affect joint moments, reported changes in stiffness resulting from fatigue are only useful in predicting shifts in joint moments if authors report that the stiffness changes were caused by changes in peak ground reaction forces, leg length, or some combination of the two. Dutto and Smith (2002) have reported significant reductions in the leg stiffness of trained runners after a fatiguing run protocol, and that these reductions in stiffness were the result of increased flexion of the leg and not of changes in the peak ground reaction force. The means by which runners alter
their hip, knee and ankle ranges of motion without significantly affecting their force applied to the ground would be greatly clarified by joint moment analysis.

The fatigue-induced decreased stiffness and increased stride length reported by Dutto and Smith (2002) mirror the effects of manipulating stride length in non-fatigued runners, reported by Farley and Gonzalez (1996). These authors observed a nearly linear inverse relationship between stride length and leg stiffness as participants ran at a constant velocity. In explaining the decreased stiffness with increasing stride length, Farley and Gonzalez emphasize that the 23% increase in peak vertical ground reaction force was overshadowed by a 186% increase in leg length change between heelstrike and midstance. In one of the few studies to date documenting fatigue-induced changes in ground reaction force profiles, Gerlach and colleagues (2005) report only slightly reduced impact peak magnitudes and loading rates along with unchanged active force peaks in a large cohort of recreational runners.

If there is a lesson to be learned from the aforementioned kinematic and limb stiffness studies, it is that runners tend to respond to fatigue by assuming an increasingly flexed leg posture with little to no change in ground reaction force profiles. While this trend is worth mentioning, the kinetic mechanism runners used to achieve increased compliance and range of motion while maintaining a constant running velocity remains unknown. Joint moment analysis could help fill this gap in our understanding of the way runners alter the stiffness of their legs in response to fatigue.
Past research may offer insight into fatigue-induced changes in joint moments at specific parts of the stride cycle. Analysis of the landing phase of heel-toe running (Bobbert, Yeadon, & Nigg, 1992) suggests that the rapid shift from net flexor moment to net extensor moment about the knee shortly after impact is only possible with co-contraction of knee flexors and extensors late in the flight phase. This co-contraction has been verified by electromyography (Mann, Moran, & Dougherty, 1986) and is believed to aid in joint stabilization (Elliott & Blanksby, 1979). By this proposed mechanism, pre-activation of knee extensors would have the effect of removing slack in the patellar tendon, thus enhancing the ability of the quadriceps to rapidly develop force during early stance. Knee extensors perform work to absorb energy and control the descent of the body during the first half of stance (Winter, 1983). Any benefit granted by co-contraction prior to ground contact would clearly be compromised by a reduction in force produced by the muscles on either side of the joint.

Results of fatigue studies demonstrate that a reduction in pre-activation of leg muscles does in fact occur. Running at high intensity for 30 minutes has been shown to cause an imbalance in activation of the dorsiflexors and plantar flexors (Mizrahi et al., 2000a). Fatigue induced by brisk uphill walking delays activation of leg muscles prior to impact in a rapid stop maneuver (Nyland, Shapiro, Stine, Horn, & Ireland, 1994). Following a marathon run, activation of the soleus and quadriceps is reduced prior to ground contact (Avela & Komi, 1998). These results indicate that the rapid rise in net extensor moment about the knee reported by Bobbert et al. (1992) could be impaired in the fatigued state. If the net extensor moment
necessary to slow the decent of the body arises more gradually, the joints could be expected to flex more and the body to descend to a greater degree, in a manner consistent with the results of previous kinematic and stiffness studies. Previous findings indicate that reaching a deeper position of flexion would also require a runner to generate a greater knee extension moment at midstance (Derrick et al., 1998). This evidence implies that the fatigue induced by a strenuous run may result in a reduced net knee extension moment during early stance and a corresponding increase in net knee extension moment at midstance.

Changes in joint moment about the knee resulting from fatigue can be predicted based on previously documented changes in moment arm length as well as ground reaction force magnitudes. However, predicting joint moment changes about the hip and ankle is more difficult, because the distance between these joint centers and the ground reaction force vector is not as clearly influenced by joint angle as it is at the knee. Moreover, the hip, knee, and ankle joint moments during locomotion are not completely independent, and changes in one moment will likely require adaptations in one or both of the other moments to ensure support and progression of the body.

Joint moments are generated primarily by muscular forces, and as such, a gait variable that reflects both the magnitude of joint moments and the time over which they are exerted could provide a measure of the cumulative demand placed on muscles during cyclical movements. Angular impulse, the time integral of the net joint moment, is one such variable. Prior research indicates that the increased compliance of the limbs resulting from fatigue has the effect of increasing total
contact time, thus decreasing the average magnitude of external forces (Avela & Komi, 1998; Gollhofer, Komi, Miyashita, & Aura, 1987; Nicol et al., 1991). While these two factors have opposing implications for changes in angular impulse exerted by extensor muscles, the increasing degree of joint flexion observed in past studies of running fatigue (Derrick et al., 2002; Siler & Martin, 1991) leads to predictions of increasing angular impulse generated by extensor muscle groups at the hip and ankle joints. While the same line of reasoning can be applied to the knee joint, an increasing degree of knee flexion would tend to increase the net extensor moment, thus complicating predictions of changes in extensor angular impulse at the knee.

While stance phase biomechanics is a natural focus in the study of running, past research has also identified fatigue effects that may imply changes in joint moments during the swing phase. An increase in the angle swept by the thigh during swing (Siler & Martin, 1991), together with less time spent in swing (Elliott & Roberts, 1980) could indicate an increase in the magnitude of the hip flexion moment needed to swing the leg forward as the foot leaves the ground.

The mechanical effects of fatigue on running form have historically been characterized as changes in kinematic patterns, stiffness parameters, and muscle function before and after a fatiguing run. While direct measurements of fatigue-induced reduction in isokinetic strength and the gross-motor end results of fatigue have been described, the joint moments which bridge the gap between these realms have not been addressed. The purpose of the proposed study was to thoroughly describe the effects of progressively accumulating fatigue induced by a treadmill run
of constant velocity, with a focus on changes in the joint moments generated by the runner. It was hypothesized that over the course of the exhaustive run, participants would demonstrate:

i) increased contact time,

ii) increased peak stance phase knee flexion,

iii) increased peak swing phase hip flexion moment,

iv) increased stance phase hip extension angular impulse,

v) increased peak stance phase knee extension moment, and

vi) increased stance phase plantar flexion angular impulse.

Hypotheses were supported or rejected based upon the direction and significance of changes in variable values occurring over the course of the run. Specifically, analyses of variance were used to determine whether time had a significant effect on variable values over the course of the run. Orthogonal polynomial comparisons were then used to describe trends in the data and hypotheses were accepted if significant trends existed, indicating that variable values increased or decreased as predicted.

Distance running is comprised of rhythmic, submaximal compound movements, utilizing multiple energy pathways. Fatigue in this dynamic context is a poorly understood phenomenon. Thus, many factors combine to prevent a complete understanding of fatigue-induced changes in running performance. Because joint moments are the result of muscular forces, changes in the muscular forces used by a fatigued runner will necessarily cause changes in joint moments. While kinematic variables describe the final manifestations of muscular fatigue,
joint moments represent the best non-invasive means for characterizing how the changes in muscular force patterns that arise with fatigue affect the way people run. The gait alterations brought about by fatigue increase the metabolic cost of running and put runners at increased risk of injury. Therefore, knowledge of the joint moment changes underlying these gait alterations should prove useful to both therapists and coaches seeking to minimize injury risk and maximize performance in runners.
CHAPTER 2
REVIEW OF LITERATURE

2.1 Introduction

Since the jogging boom of the 1970s, running has become one of the most common forms of exercise in America. A considerable body of research has demonstrated a variety of health benefits associated with regular running, including increased bone mineral density (Lane et al., 1986), improved psychiatric health (Taylor, Sallis, & Needle, 1985), and decreased risk of coronary heart disease (Paffenbarger et al., 1993). Despite these benefits, injury rates among recreational and competitive runners remain startlingly high, with annual incidence of lower-extremity injury ranging from 20% and 79% (van Gent, van Middelkoop, van Os, Bierma-Zeinstra, & Koes, 2007). For this reason, a considerable portion of running research has sought to improve our understanding of the mechanisms underlying common injuries. Many investigations of running injuries are retrospective in nature, attempting to determine whether previously injured runners differ from uninjured controls in selected mechanical variables (Miller, Lowry, Meardon, & Gillette, 2007; Opar, Williams, Timmins, Dear, & Shield, 2013; Zifchock, Davis, & Hamill, 2006). When the situation allows, prospective studies can identify mechanical differences between runners who go on to develop injuries and those who do not (Hamill, Miller, Noehren, & Davis, 2008).

Investigations of running injuries utilizing retrospective and prospective designs rely on elegantly simple logic: If a group of injured runners differs from a
control group in a mechanical variable that is thought to be associated with injury, then significant differences between the groups may represent an injury mechanism. Despite the validity of this argument, approaching running research from this angle fails to replicate actual running conditions by neglecting an inevitable complicating variable: Fatigue.

Among studies of running-related injuries, the vast majority are either cross-sectional in nature or analyze gait during a single laboratory session at or near participants’ preferred pace, lasting just long enough to reach a steady state. Fatigue increases the likelihood of acute injury (Mair, Seaber, Glisson, & Garrett, 1996; Whiting, 1998), causes progressive mechanical changes with implications for overuse injuries (Derrick et al., 2002; van Gheluwe & Madsen, 1997), and most importantly, is an inevitable consequence of running. An understanding of the way fatigue affects gait variables is therefore an essential piece of the puzzle of running-induced injuries.

The etiology of running injuries is but one area of research, which cannot be considered complete without accounting for fatigue effects. Running humans optimize their gait in order to minimize metabolic cost by tightly regulating such variables as stride frequency (Cavanagh & Williams, 1982) and stance leg flexion (McMahon, Valiant, & Frederick, 1987). If any variable affecting the metabolic cost of running is altered in the fatigued state, advancing our knowledge of these mechanical changes could provide valuable knowledge to anyone interested in improving running performance.
Attempts to characterize the changes that accompany running fatigue date back nearly sixty years (Ehrhart, 1957). Despite this, results of these studies are often inconclusive or contradictory. Running is a complex motor task and as such, it must be understood that any kinematic changes induced by fatigue are underlain by altered muscular performance and, potentially, altered control by the nervous system. Despite the abundance of research aimed at kinematic changes induced by running fatigue, the muscular bases for these changes has received scant attention in the biomechanics literature. Reductions in muscular force capacity have been reported after runs as short as 20 minutes (Skof & Strojnik, 2006), yet direct measurement of muscular forces during locomotion is highly invasive and currently limited to animal studies (Roberts, Marsh, Weyand, & Taylor, 1997).

In the absence of direct force measurements, joint moment values can provide a useful surrogate method for analyzing the changing role of muscles, resulting from running fatigue. As the resultant effect of the forces exerted by muscles crossing a joint, joint moments can be thought of as the mechanical cause of the movements comprising gait (Vaughan, 1996). Despite the non-invasive methods of collecting joint moment data and considering the knowledge that we stand to gain from such an analysis, it may come as a surprise that joint moments have not been characterized over the course of a fatigue-inducing run. It is hoped that this review of literature will provide a clear yet concise background on the manifestations of fatigue, the concept of joint moments, and previously reported fatigue induced mechanical changes, which allow predictions to be made regarding joint moment changes during an exhaustive run.
2.2 Background on Fatigue

Fatigue is generally defined as any reduction in the force-generating capacity of the neuromuscular system, regardless of the ability to maintain a submaximal target force (Bigland-Ritchie & Woods, 1984). Defined this way, fatigue is the inevitable result of muscle activation, regardless of duration or intensity. Fatigue also differs from exhaustion, defined as an inability to sustain exercise at the target intensity (Vøllestad, Sejersted, Bahr, Woods, & Bigland-Ritchie, 1988). In applying these working definitions to running, fatigue is responsible for the gait alterations that the runner uses to continue running, despite his muscles’ reduced force-generating capacity. Exhaustion occurs when this capacity declines to a point at which running cannot continue at the target velocity.

When individuals attempt to maintain a maximum intensity contraction against a fixed resistance, the fatigue that develops can be measured as the decline in force from the recruited muscles. This phenomenon has been extensively studied in single joint movements in controlled laboratory conditions. Though similar methods have been used to characterize fatigue development in a multitude of muscles, an investigation of fatigue in the knee extensors neatly demonstrates these methods and results (Bigland-Ritchie, Jones, Hosking, & Edwards, 1978).

In order to ensure that the knee extensors were not assisted by the hip flexors, Bigland-Ritchie and colleagues (1978) had participants sit in an adjustable chair with the thigh strapped to the seat. Force was recorded by a transducer linking a strap around the ankle to a leg of the chair. Upon the investigator’s instruction, participants attempted to extend the knee with maximal effort for sixty
seconds. In the nine subjects in the present investigation, the force produced had fallen to an average of 30% of the initial value by the end of the test (Bigland-Ritchie et al., 1978).

Fatigue can result from impairment in normal functioning of the neuromuscular system at any one of many points between the motor cortex and the contractile filaments. The specific impairments present in a given case allow the categorization of the fatigue as either central or peripheral in nature. Central fatigue refers to impairment within the central nervous system, while peripheral fatigue denotes impairment distal to the neuromuscular junction, within the muscle fibers themselves (Bigland-Ritchie & Woods, 1984).

The relative contribution of central and peripheral mechanisms to any observed reduction in muscular force can be determined using the setup described above (Bigland-Ritchie et al., 1978). The ability of the muscle to produce force can be separated from the central command by means of direct electrical stimulation of the muscle's motor neuron, or by percutaneous electrical stimulation of the muscle. The results of the study in question revealed that in some subjects, the force produced by a maximum voluntary effort fell more rapidly than the force resulting from direct electrical stimulation, such that by the end of the sixty second effort, voluntary effort resulted in forces 10-30% lower than the actual force-producing capacity of the muscle (Bigland-Ritchie et al., 1978).

Central fatigue reflects a failure of the central nervous system to fully activate all motor units within a muscle, and is thought to result partly from a reduction in conscious effort or motivation of the individual (Bigland-Ritchie,
Cafarelli, & Vollestad, 1986; Kent-Braun, 1997). This reduced neural drive is likely the result of increasing discomfort, and its role in reducing the force of a muscular contraction can be minimized with practice (Bigland-Ritchie & Woods, 1984). Similar studies have found that the contribution of central fatigue to overall reduced force capacity also varies depending on the muscle in question (Bigland-Ritchie et al., 1986a).

Experimental protocols utilizing sustained contractions to induce fatigue have the advantage of simplicity, but lack similarity to muscular demands in sport and everyday life. Realizing this shortfall, many researchers have employed methods similar to those used to characterize sustained contraction fatigue in order to examine force reduction in intermittent, submaximal contractions (Bigland-Ritchie, Furbush, & Woods, 1986). In one such experiment, participants were asked to perform quadriceps contractions representing 50% of their maximal force for periods of six seconds, separated by four-second rest periods. The ratio of contraction time to cycle time is referred to as the duty cycle, and was equal to 0.6 for this particular study. Every minute, participants performed a maximal voluntary contraction to assess fatigue accumulation, as the decline in maximum force-generating capacity. These intermittent contractions continued until the so-called "limit of endurance," when maximal voluntary contractions were equal to the target force, and participants could not maintain the target force for the full six seconds. The soleus muscle was tested in the same manner on a separate day (Bigland-Ritchie et al., 1986b). Results of these experiments indicate that given the same duty cycle and submaximal force level, endurance times differ greatly between the
quadriceps (4.4 ± 0.6 minutes) and the soleus (35.1 ± 15.7 minutes). Despite varying endurance times, the maximal voluntary strength of the leg muscles tested declined linearly with time.

Regardless of relative force required and whether contractions are constant or intermittent, fatigue of isolated muscle groups increases linearly with time (Bigland-Ritchie et al., 1986a; Bigland-Ritchie et al., 1986b; Bigland-Ritchie et al., 1978; Bigland-Ritchie & Woods, 1984). However, the means by which a muscle continues to produce force as its maximal capacity decreases is another question entirely, and one which may have implications for more dynamic, compound movement patterns, such as locomotion. This question has been addressed using serial muscle biopsies during an intermittent submaximal contraction protocol similar to that described above (Vøllestad et al., 1988). Examination of quadriceps muscle samples obtained at several points during an exhaustive protocol lasting roughly 45 minutes revealed that no significant changes in intramuscular metabolite concentrations occurred during the first 30 minutes of exercise. However, significant changes in the concentrations of ATP, creatine phosphate, glycogen, and lactate occurred during the final minutes preceding exhaustion. The non-linear nature of metabolic changes within the muscle lies in sharp contrast to the linear decline in maximal force-generating capacity of the muscle, and implies a shift in metabolism towards anaerobic energy sources in the final minutes of exercise (Vøllestad et al., 1988).

In addition to changes in metabolite concentrations, alterations in activation patterns offer another lens with which to analyze muscular adaptations during
prolonged contractions. Examination of motor neuron firing rates during intermittent submaximal contractions provides evidence that fast-twitch fibers are recruited to a greater degree as muscle nears the limit of endurance (Vollestad, Vaage, & Hermansen, 1984). These results provide partial explanation of the observed non-linear changes in metabolite concentration, as fast-twitch fibers deplete glycogen and produce lactate at a greater rate than the slow-twitch fibers preferentially recruited early in exercise (Powers & Howley, 2009).

Recent work examining the interaction of individual myosin and actin filaments *in vitro* also provides evidence that the byproducts of an increased rate of glycolysis may themselves have deleterious effects on the force and velocity of crossbridge cycling, thus accelerating the fatigue process (Debold, 2012). Without beginning a separate discussion, it bears mentioning at this juncture that an acceleration of compensatory mechanisms in the final stages of an exhaustive effort is also a frequent observation in studies of high-intensity running (Clansey et al., 2012; Derrick et al., 2002; Elliott & Ackland, 1981; Elliott & Roberts, 1980; Nicol et al., 1991; Verbitsky, Mizrahi, Voloshin, Treiger, & Isakov, 1998).

Several authors have noted large increases in the integrated surface EMG of muscles fatigued using submaximal methods similar to those described above (Edwards & Lippold, 1956; Lind & Petrofsky, 1979). Because this increase in integrated EMG is of far greater magnitude than the changes in firing rates, these results strongly suggest that recruitment of additional motor units plays a larger part in maintaining a submaximal force than increasing motor unit firing rate, as muscles approach exhaustion.
Despite the abundance of factors contributing to the accumulation of muscular fatigue and the various mechanisms available to maintain submaximal muscular force, higher force demands unfailingly result on shorter endurance times. When represented mathematically, this relationship between relative intensity and endurance time differs slightly between muscle groups, but always takes the form of an exponential decrease in endurance time with increasing relative intensity (Law & Avin, 2010). Similarly, when a trained runner puts forth a maximal effort in running a variety of distances, the relationship between running velocity and duration exhibits an exponential decrease in duration as velocity increases (Londeree, 1986). While it would be foolish to propose that running performance hinges solely on muscular endurance, the parallels in response to prolonged exertion of muscle and of the whole athlete should not be overlooked. Therefore, an understanding of fatigue mechanisms at the muscular level provides a necessary foundation for the study of fatigue effects in the whole organism during complex tasks, such as locomotion.

2.3 Fatigue effects in Running

The purpose of running is to translate the body through space with alternating periods of flight and single-leg support. This linear motion of the whole body is accomplished by angular motion of the body segments about the joints, which is in turn brought about by linear lengthening and shortening of muscles. Thus, any impairment in muscle function will necessarily have consequences for whole-body translation. The means by which muscular fatigue affects running
performance can be examined separately from the way that the general fatigue
induced by running effects performance of specific muscle groups. Studies of these
interactions between muscle and running performance can be divided into three
categories: Running-induced fatigue effects on muscular force; muscular fatigue
effects on running performance; and running-induced fatigue effects on continued
running performance. This last category will be discussed in detail in sections 2.6
through 2.8, while the former two are described below.

When people are asked to run at a given velocity until exhaustion, the
generalized fatigue that ultimately causes the runner to stop is partly caused by
fatigue of individual muscles. Several investigators have used isometric testing of
knee extensors and ankle plantar flexors before and after strenuous runs to quantify
these muscular effects. Skof and Strojnik (2006) have reported decreases in twitch
torque and electrically-stimulated torque from the knee extensors after a twenty
minute run at the intensity of lactate threshold. Interestingly, considering the
participants’ conditioning levels and the intensity and duration of the run, the
protocol was likely not especially strenuous, indicating that these neuromuscular
changes began to accumulate long before the point of volitional exhaustion.
Following a thirty-kilometer trail race, Millet and colleagues (2003) observed a 23%
decrease in knee extensor maximum voluntary torque. Marathon runs have been
shown to reduce plantar flexor rate of torque development and maximum voluntary
torque (Avela et al., 1999), as well as reduced stiffness of the soleus muscle during
the deceleration phase of ground contact (Avela & Komi, 1998).
Notably, torque was measured isometrically in all of the above studies, which differs from the dynamic role muscles play during the majority of the stride cycle. Reports of concentric and eccentric strength changes resulting from running-induced fatigue are rare, but a study of trained runners has found significant reductions in dynamic strength after a 45 minute run at the velocity of lactate threshold (Denadai et al., 2007). Strength loss was greater in the eccentric condition than concentric, and greater at lower angular velocity (60 °/s) than high velocity (180 °/s). All strength changes represented deviations from the unfatigued condition of less than 10%, though this value may underestimate the true strength loss due to a fifteen-minute delay between the run and the isokinetic testing.

The underlying causes of running-induced strength loss should not be overlooked, as central and peripheral fatigue differ greatly in their implications. The degree of central and peripheral fatigue induced by strenuous running can be evaluated using the same methods utilized by the muscular fatigue studies described in section 2.1. Central fatigue is said to have occurred when the force voluntarily produced by a muscle group decreases after a strenuous effort to a greater extent than the force elicited by direct electrical stimulation. Of the studies referenced above, the fatigue resulting from the twenty minute run employed by Skof and Strojnik (2006) was entirely peripheral in nature, while conversely, central fatigue did contribute to the 23% strength loss observed after the 30-kilometer trail run utilized by Millet and colleagues (2003). A thorough review of the muscular effects of various endurance sports supports the notion that the degree of central fatigue elicited increases with the duration of the activity (Millet & Lepers, 2004).
These authors also propose that the eccentric muscle action inherent to running underlies the observation that running results in greater damage to muscle fibers than bicycling or cross-country skiing of similar intensity and duration. Muscle damage can be detected by changes in the concentrations of blood markers, and has been observed by several researchers following marathon runs (Avela et al., 1999; Kyröläinen et al., 2000; Ostrowski, Rohde, Zacho, Asp, & Pedersen, 1998).

Inducing fatigue in specific muscle groups before analyzing a runner's gait may offer insight into the role of those muscles in running. In order to parse out differences in the roles of individual muscle groups, such studies must employ complete protocols for each muscle group of interest on separate days. Christina, White, and Gilchrist (2001) used repeated concentric and eccentric contractions to induce fatigue of the dorsiflexors and inverters prior to recording kinematics and ground reaction forces during a brief run. Unsurprisingly, fatigue of the dorsiflexors resulted in a decreased degree of dorsiflexion at the instant of heel contact. This flatter foot position was accompanied by an increased vertical ground reaction force loading rate. The authors suggest that together, these results indicate that when the dorsiflexors are fatigued, their role in properly positioning the foot prior to ground contact is compromised. An increasingly plantar flexed position indicates a longer muscle length of the tibialis anterior at contact, and therefore a reduced capacity for this muscle to eccentrically absorb energy in early stance. If the dorsiflexors normally act to control the passive plantar flexion experienced by rearfoot striking runners at heel contact (Elliott & Blanksby, 1979; Novacheck, 1998), then their fatigue may result in reduced impact attenuation and increased potential for
overuse injuries (Mizrahi et al., 2000a). Fatigue of the inverters did not cause significant changes in kinematics, though the resulting subtle changes in ground reaction forces occurred at points in the running stride during which these muscles are thought to control pronation (Christina et al, 2001).

Kellis and Liassou (2009) used a similar protocol to investigate the effects of specific muscular fatigue on kinematics and muscle activation patterns. On two separate days, the knees and ankles were fatigued with an isokinetic dynamometer using cyclic flexion and extension contractions at 120 °/s until the torque generated fell to 30% of the initial maximum value. Kinematic and electromyographic data were collected during a ten second run immediately following the isokinetic fatigue protocol. Mirroring the results of Christina and colleagues (2001), ankle fatigue resulted in increased plantar flexion during the swing phase and at initial contact. Ankle fatigue also caused decreased knee extension at toe-off, supposedly due to reduced plantar flexor torque during propulsion. Knee fatigue induced kinematic changes reflecting a lower carriage of the body, including increased knee flexion at initial contact and at toe-off and increased hip extension at toe-off. This position of greater limb flexion was accompanied by increases in activation of knee extensors. Interestingly, fatigue of muscles controlling knee motion induced kinematic gait alterations similar to those observed after fatigue induced by prolonged, strenuous running (Derrick et al., 2002; Dutto & Smith, 2002).
2.4 Variable responses to Running Fatigue

Despite the unquestionable applicability of the subject, the literature devoted to running fatigue is fraught with inconsistency. An investigator’s choice of protocol and participants each independently dictate the nature of the fatigue induced, and therefore the real life scenarios to which the results apply. Of all causes of inconsistency among studies, whether or not the speed of the run is controlled is perhaps the most rudimentary. Strenuous running often results in a gradual reduction in velocity. While slowing down is a natural reaction to accumulating fatigue, allowing velocity to decrease creates a scenario in which changes in gait due to accumulating fatigue occur simultaneously with those simply due to slowing down. The presence of both effects makes isolation of fatigue-induced changes impossible. While many early studies in running fatigue observed athletes gradually slowing during competition (Bates, Osternig, & James, 1977; Ehrhart, 1957; Elliott & Ackland, 1981), the complication of speed effects has pushed researchers towards constant speed protocols in recent years.

Just as in studies of isolated muscle groups, fatigue induced by strenuous running can be attributed to both central and peripheral mechanisms. When levels of voluntary muscle activation are evaluated before and after fatiguing runs, a trend emerges relating the degree of central fatigue and the run’s duration. Specifically, no central fatigue of leg extensor muscles has been detected after a twenty-minute (Skof & Strojnik, 2006) or one hour (Davies & White, 1982) run, but has been observed after a three-hour mountain trail run (Millet et al., 2003). These results may imply that reduced neural drive or impairments of central nervous system
function do not contribute significantly to reduced muscle function in running until run duration exceeds a threshold between one and three hours.

Following this observation, temporospatial changes resulting from fatigue tend to differ between marathons and shorter runs. Due to logistical challenges, mechanical changes in running form after fatigue induced by marathon runs are rarely reported, but one such study has found a decrease in stride length during controlled-speed treadmill runs before and after the race. Interestingly, this gait alteration following a 2.5 to 3.5 hour run is opposite in direction to stride length changes observed after many fatiguing runs of shorter duration (Dutto & Smith, 2002; Mizrahi, Verbitsky, Isakov, & Daily, 2000; Siler & Martin, 1991; Verbitsky et al., 1998).

In instances when the intensity of a fatigue-inducing run approaches the upper limits of participants' abilities, researchers must be aware of the possibility of cardiovascular fatigue. Mechanical changes in running gait are highly variable between studies utilizing runs approaching participants' maximal oxygen uptake. Using the example of stride length, running above participants' lactate threshold has caused increases (Cavanagh et al., 1985), decreases (Elliott & Roberts, 1980), or no change (Abt et al., 2011) in this variable. In explaining this variability, it has been suggested that cardiovascular stress may cause participants to terminate these runs before fatigue of the neuromuscular system induces gait alterations (Abt et al., 2011). If muscular fatigue is not a factor in terminating such high intensity efforts, other factors must be responsible for observed gait changes.
Aside from systematic differences rooted in duration and relative intensity between studies, the specific protocol utilized by researchers plays a role in the nature of results, as well as their validity. In order for findings of running fatigue studies to be applicable, changes in gait characteristics must be evaluated during the fatiguing effort. Clansey and colleagues (2012) observed increased hip, knee, and ankle extension at initial contact following two separate bouts of twenty minutes of running at the velocity of lactate threshold. The two fatiguing bouts were performed on a treadmill, and mechanical data were collected immediately after each bout by having participants dismount the treadmill and perform five runs through the motion capture space. The running velocity used for data collection was 4.5 m/s, leaving participants very little time to accelerate and subsequently decelerate on a runway only fifteen meters in length. These collection conditions may well have caused a discrepancy between the gait used by these runners during the twenty-minute bouts and during the collection trials. The kinematic changes reported by these authors are at odds with those reported by Siler and Martin (1991) resulting from a fatiguing run of similar intensity and duration in participants of similar training status, but who collected data during the fatiguing run, itself.

If there is a single lesson to be learned from past studies of running fatigue, it is that the specific fatigue-induced changes in gait mechanics elicited depend on the relative intensity and duration of the run. Fatiguing protocols of any intensity will have application to real world running in similar conditions, though gait alterations
become less predictable as velocity approaches the limits of participants’ capabilities.

2.5 Background on Joint Moments

If the goal of running-related research is to maximize performance while minimizing injury, then an understanding of the way muscles bring about the movements of gait would be a valuable piece of knowledge. However, as mentioned above, measuring the forces produced by muscles within the living athlete is extremely difficult and invasive, requiring surgical intervention that would almost inevitably change the movement of interest. Joint moments are the best means available to non-invasively estimate muscle forces, due to their functional situation between muscle forces and joint rotation.

When a muscle is activated, the force it produces takes the form of tension transmitted to the bones to which its tendons are attached. With few exceptions, skeletal muscles span joints, meaning that the bones to which a muscle is anchored rotate relative to one another about an axis coincident with the joint. The force developed by a muscle will thus produce a torque of some magnitude about its joint, dependent on the tension developed and the length of the muscle’s moment arm. The moment developed by muscles results in a tendency to produce angular acceleration of the body segments, as well as resistance to external loads such as ground reaction forces. As such, joint torques may be considered the cause of the movements comprising complex activities such as running (Vaughan, 1996).
Joint moments at any joint can be calculated based on motion and force data collected while the participant performs the movement of interest. In most cases, these data will consist of the linear motion of individual segments as well as any external forces between the participant and the environment. What follows is a cursory description of the inverse dynamics method of calculating joint moments, using the lower limb in gait as an example scenario (Selbie, Hamill & Kepple, 2014).

As a first step, local coordinate systems for each segment are established. These segment coordinate systems are often centered at the segments' centers of mass, and determined using the position of skin-mounted markers corresponding to known skeletal landmarks. Local coordinate systems enable the expression of joint rotation and torque as that of the segment distal to the joint in the local coordinate system of the segment proximal to the joint - i.e. the ankle joint moment is reported as the torque on the foot segment within the leg segment's coordinate system.

An inverse dynamics analysis of gait begins at the most distal joint, normally the ankle. At each joint, we begin by determining the joint reaction forces. For the ankle, joint reaction forces are calculated as follows:

\[ \text{jrf}_{\text{Ankle}} = m_{\text{CM}} \cdot a_{\text{CM}} - mg - f_{\text{GRF}} \quad (1) \]

where \( \text{jrf}_{\text{Ankle}} \) is the vector form of the ankle joint reaction force, \( m \) is the foot's mass, \( a_{\text{CM}} \) is the linear acceleration of the foot's center of mass, \( mg \) is the weight vector, and \( f_{\text{GRF}} \) is the ground reaction force vector. Depending on whether the analyses are conducted in two or three dimensions, this equilibrium equation will be solved along each of two or three axes, respectively. Once joint reaction forces are computed, joint moments can be calculated in a similar manner, this time
subtracting rotational components of motion from the intrinsic motion of the segment, as follows:

\[ jm_{\text{Ankle}} = I \alpha - (d_1 \times jrf_{\text{Ankle}}) - (d_2 \times f_{\text{GRF}}) - t \]  

(2)

where \( jm_{\text{Ankle}} \) is the vector describing the ankle joint moment, \( I \) is the moment of inertia of the foot, \( \alpha \) is the angular acceleration of the foot, the \( d_1 \times jrf_{\text{Ankle}} \) term is the vector describing the moment caused by the ground reaction force, the \( d_2 \times f_{\text{GRF}} \) term is the vector describing the moment caused by the ground reaction force, and \( t \) is the ground reaction torque, used in three-dimensional analyses.

This sequence of calculations is repeated for each segment in a distal to proximal order. In calculating the moment of all segments proximal to the foot, the term representing the moment caused by ground reaction forces is replaced by the moment resulting from joint reaction forces at the distal joint. The mass, moment of inertia, and center of mass location for each segment are estimated based on the participant’s anthropometrics and values found in the literature (de Leva, 1996; Dempster, 1955).

Figure 2.1: Joint moment curves for the stance leg of a healthy adult running at four speeds: \( C_1 = 3.06 \) m/s; \( C_2 = 3.57 \) m/s; \( C_3 = 4.09 \) m/s; and \( C_4 = 4.60 \) m/s. Adopted from Simpson & Bates, 1990.

Several authors have characterized joint moments in running, both from a purely descriptive perspective (Winter, 1983) and to enable comparisons between
speeds (Simpson & Bates, 1990; see Figure 2.1) and modes of gait (Novacheck, 1998). As a general principle, the joints of the lower extremity tend to flex and absorb energy during the first half of ground contact and subsequently extend as the body is propelled into flight. Joint moment analysis has allowed a deeper understanding of the mechanical cause of joint kinematics. For example, Winter (1983) was the first to note the consistent sequence of extensor moment peaks during stance: The hip reaches its extensor peak before midstance, followed by the knee, and finally the ankle during the second half of stance. Energy generated and absorbed by a given joint is equal to the time integral of joint power, which is calculated simply as the product of instantaneous joint moment and angular velocity. Mechanical energy calculations have revealed that the ankle acts primarily as an energy generator, while the knee serves primarily to absorb energy (Winter, 1983) and is the most responsive of these three joints to changes in stride length (Derrick et al., 1998). The hip musculature serves to stabilize the trunk during the stance phase and acts to actively reposition the limb during swing (Novacheck, 1998). Because joint moment magnitudes are much higher while the foot is in contact with the ground, it is not uncommon for authors to report joint moment values during only the stance phase of running (Simpson & Bates, 1990; Winter, 1983).

In the continuing endeavor to understand the mechanics of movement, joint moments have helped to fill the void between muscle activation and observable kinematics. However, calculated moments are subject to several limitations and as
Vaughan (1996) has said, our search for the perfect gait analysis tool has not yet yielded the Grail.

Chief among these limitations is the very aspect of joint moments, which makes their implementation so convenient that they represent a simplified solution to the problem of indeterminacy. As illustrated in Figure 2.2, many bones and muscles often cross a single joint, each of which exerts a force between the adjacent segments. Even in an idealized hinge joint spanned by a single flexor and a single extensor muscle, the single joint moment value calculated will reflect only the net torque produced by these components.

At instances when all muscles active at a given moment are those tending to cause the same motion about the joint, then all of the calculated joint moment value can be ascribed to these active muscles. However, this is rarely the case in running, as co-contraction has been detected at the hip, knee, and ankle during the majority of the running gait cycle (Elliott & Blanksby, 1979; Mann et al., 1986).

Figure 2.2: A) The propulsive role of the ankle during late stance, simple at first glance, belies the complexity of the structures beneath. B) Many musculoskeletal components independently act in compression or tension, thereby influencing the calculated net joint moment. Adopted from Vaughan, 1996.
As described above, joint moment values are calculated based upon measured kinematics as well as external forces. Moment values are therefore subject to errors occurring in the collection and processing of both varieties of data. Special care must be taken in any gait analysis using force-instrumented treadmills, as vibration from the treadmill motor can add noise to the mediolateral forces of magnitudes approaching those between the foot and ground during gait (Kram, Griffin, Donelan, & Chang, 1998).

Last but not least of the pitfalls of joint moments are errors in estimations of participants' anthropometry. Values such as fractional mass of each segment, segment center of mass location, and inertial parameters are difficult to measure on an individual subject basis and are often estimated based on literature values (Clauser, McConville, & Young, 1969; de Leva, 1996; Dempster, 1955). Discrepancy between estimations and actual participant anthropometry can cause large errors in calculated joint reaction forces and moments (Pearsall & Costigan, 1999).

2.6 Determinants of Joint Moments in Gait

Mathematically speaking, moment (τ) is the cross product of a force vector (F) and a lever arm length (r). Despite the rigorous calculations involved in an inverse dynamics analysis,

\[ \tau = r \times F \quad (3) \]

can be used as a first approximation of joint moment magnitude and direction in gait (Vaughan, 1996). Estimating joint moments in this manner does not account for segment inertial parameters, but this simplification proves useful for providing
approximations of joint moments, which are the product only of force vector magnitude and lever arm length as long as the inertial terms (products of I and α for each segment) are small. Therefore, differences within subjects and between conditions in joint moments, approximated as such, can be attributed to changes in the force vector, lever arm length, or both.

In gait analysis, the lever arm length for a given joint can be thought of as the perpendicular distance between the ground reaction force vector's line of action and the joint center (Vaughan, 1996). In the lower extremity, the lever arm length about the knee tends to increase with knee flexion (Derrick et al., 1998).

Altering running velocity (Simpson & Bates, 1990) or stride length while running at a constant velocity (Derrick et al., 1998) each have significant effects on the magnitude and shape of joint moment profiles in non-fatigued individuals. Neither velocity nor stride length, however, give insight into whether such effects result from changes in ground reaction forces or lever arm lengths. Predicting changes in joint moments induced by fatigue requires insight into how these two values are individually affected by fatigue.

Past studies have reported altered ground reaction forces after running-induced fatigue (Gerlach et al., 2005) as well as isokinetically-induced fatigue (Christina et al., 2001). Additionally, a sizeable body of prior research indicates that kinematic adaptations to fatigue include increased lower-extremity flexion, which may suggest changes in lever arm length of ground reaction forces (Derrick et al., 2002; Dutto & Smith, 2002; Kellis & Liassou, 2009; Mizrahi et al., 2000b; Siler & Martin, 1991).
While no studies yet exist characterizing fatigue-induced joint moment changes, clues as to the nature of these changes can be found in various sub-disciplines of the running fatigue literature. The following sections describe how three of these sub-disciplines offer evidence of changing joint moments.

2.7 Fatigue effects: Kinematics

Of all manifestations of running fatigue, kinematic changes are the most readily apparent. Spectators and coaches are often able to visually diagnose fatigue in a variety of running situations, from the locked-up, almost robotic gait of the middle distance racer approaching the tape to the downcast gaze of the marathon runner, trudging past the twenty-mile marker. Despite the abundance of subjective evidence, the magnitude of kinematic changes induced is greatly reduced when runners are made to run at a constant speed. Though not always subjectively profound, changes in kinematics offer a first glimpse into underlying changes in muscle function.

Past research has reported fatigue-induced kinematic changes during constant-velocity runs spanning a wide range of durations and intensities, from ten minutes at a velocity exceeding participants’ 5 km race pace (Williams, Snow, & Agruss, 1991) to a marathon (Kyröläinen et al., 2000). As discussed previously, fatiguing runs of near-maximal intensity often yield inconsistent gait alterations, and run protocols approaching marathon duration induce considerable central fatigue and are beyond the scope of the present study. Therefore, the literature summarized below encompasses studies utilizing run durations long enough to
minimize cardiovascular fatigue, yet short enough to avoid central fatigue. These relative intensity bounds constrain reported results to a range that applies to the vast majority of running scenarios encountered by recreational runners and, coincidently, also yields somewhat consistent kinematic findings.

Derrick and colleagues (2002) induced fatigue in ten recreational runners at a speed corresponding to participants' best effort in a 3200 meter run, lasting around fifteen minutes. Data collected during the first minute, middle, and final minute of the run demonstrated progressive changes in kinematics with accumulating fatigue. At the knee joint, the degree of flexion at the instant of initial ground contact increased by 4.4°, while maximum flexion at midstance increased by 3.8° over the course of the run, indicating a stable range of motion despite increasing flexion throughout the stance phase. Rearfoot motion, collected with an electrogoniometer, showed an increasingly supinated position at initial contact, which developed concurrently with increased pronation at midstance, resulting in a larger range of motion. These changes in joint angles were accompanied by increased angular velocity of these two joints between the instants of initial contact and midstance. These fatigue-induced changes in ankle joint motion echoed results from Van Gheluwe and Madsen (1997), who found significantly increased peak pronation angle and rearfoot eversion angle following a brief exhaustive run at 4.5 m/s.

Exhaustive running of similarly high intensity (5% above the velocity of ventilatory threshold) has demonstrated similar kinematic changes in a group of non-runners (Mizrahi et al., 2000b). Kinematic data collected every five minutes
over the thirty-minute effort indicated a 3.8° increase in knee flexion at the instant of initial contact. Additionally, the vertical displacement of the body during each stride was found to increase towards the end of the run.

Dutto and Smith (2002) have also reported an increase in magnitude of vertical oscillations of the body as a result of accumulating fatigue. The authors chose to quantify "leg length" as the linear distance between the hip joint and ankle joint, in order to express a "leg stiffness" value of peak ground reaction force divided by leg length. Kinematic measures were not explicitly reported, but because knee angle is the only variable that can influence the authors' definition of leg length, the increase in change in leg length between initial contact and midstance as the run progressed evidences an increase in range of motion at the knee. While these kinematic results mirror those of the high intensity runs described above, the fatiguing run employed by Dutto and Smith (2002) was of much longer duration (57 ±19 minutes).

Training status or experience level of a runner has been suggested to influence the mechanical changes brought on by fatigue (Clansey et al., 2012). In a well-designed study comparing fast and slower runners, Siler and Martin (1991) addressed this issue by inducing fatigue with a treadmill run simulating participants' recent 10-kilometer race performances. These runs lasted an average of 29.5 and 44.2 minutes for the fast and slower runners, respectively. Interestingly, fast and slower runners differed neither in the nature nor the timing of fatigue-induced mechanical changes. Both groups of runners demonstrated increased knee flexion throughout the gait cycle, increased hip range of motion in the sagittal plane,
and increased forward lean of the trunk. The trend of increasing forward lean with fatigue has also been observed following runs of much shorter duration (3 kilometers in 9.7 minutes) in well-trained runners (Elliott & Roberts, 1980).

Reports of kinematic changes resulting from accumulating fatigue generally indicate that runners assume a position of greater knee flexion throughout the gait cycle. This knee flexion is sometimes accompanied by an increased degree of forward lean and a larger frontal plane ankle range of motion. As a greater degree of knee flexion is associated with a longer lever arm of the ground reaction force vector about the knee joint (Derrick et al., 1998), kinematic results, taken alone, could indicate larger joint moments at the knee.

### 2.8 Fatigue effects: Temporospatial

For the purposes of this review, temporospatial variables will refer to the timing of gait events and to the displacement of the whole body between and at the instants of these events. Specifically, the gait events of interest are initial contact, midstance, and toe-off, with midstance defined as the instant when the vertical velocity of the athlete's center of mass changes from negative to positive. At this point, a few definitions must be established. A stride can be thought of as one complete cycle of running gait, beginning and ending with subsequent initial contacts of the same foot. Following this logic, a step begins and ends with subsequent ground contacts of opposite feet.

Horizontal velocity is equal to the product of stride length (meters per stride) and stride frequency (strides per second). Stride frequency is also referred to as
cadence. Therefore, when a runner’s horizontal velocity is held constant, stride length and stride frequency are inversely related, and knowledge of one variable’s value allows the calculation of the other. This inverse relationship is important to keep in mind, as it is common in gait studies to find one value reported with no mention of the other. Horizontal velocity is often controlled using a treadmill, though it can be measured in a motion capture laboratory during overground running (Clansey et al., 2012) or controlled by other means in outdoor running conditions (Elliott & Blanksby, 1979; Nicol et al., 1991).

The mathematical relationship between temporal and spatial variables also allows insight into the components of a single running stride. If horizontal velocity is known, then the timing of initial contact, toe-off, and subsequent contact of the opposite foot can be used to calculate the horizontal displacement of the body during stance and during flight.

The discussion of temporospatial variables becomes relevant to questions of joint moments when considering the role played by muscles in supporting the weight of the body against gravity. Continuous running on level surfaces requires that the vertical position and velocity of a runner’s center of mass be approximately the same at the start and end of each step. By employing Newton’s law of acceleration, it can be shown that in order for these position and velocity conditions to be met, the time integral of vertical force between the runner and the ground must be the same during every step, with a value equal to the individual’s body weight multiplied by the time elapsed during the step. The time integral of force is impulse, and during a given step this impulse can naturally only be applied during
stance. Thus, the magnitude of vertical impulse during a given step divided by stance duration yields the average vertical force between the ground and the runner. If stance time is expressed as a fraction of step time (i.e. if stance time = 0.18s and step time = 0.36s, the decimal representation of stance time is 0.5), the exact value of average vertical force during stance can be determined by dividing the individual’s body weight by fractional stance time.

The strength of applying the vertical impulse concept to running gait analysis lies in its simplicity. As long as vertical position, velocity and stride time are consistent, the vertical impulse value will remain constant, regardless of any changes in stance time, flight time, or vertical motion of the body during each stride. When variables such as fatigue alter the stance time used by a runner, relative changes in average vertical ground reaction forces can be quickly determined.

Flight time, or the time elapsed between toe-off and initial contact of opposite feet, also proves useful in gait analysis. If gravity is the only external force acting on an athlete’s body in the vertical direction, then the rules governing projectile motion dictate that flight time is a function of vertical velocity at toe-off and the relative vertical position of the body at toe-off and subsequent initial contact of the opposite foot. If the relative height of the body at toe-off and initial contact remains constant across steps, then changes in flight time indicate changes in vertical velocity at toe-off in the same direction. With these definitions and concepts established, let us turn to the literature.

Exhaustive running for durations shorter than the marathon frequently cause a gradual increase in stride length used by the runner. In one such study,
recreationally active participants ran for thirty minutes on a treadmill at a velocity corresponding to their individual anaerobic thresholds. Respiratory data indicated which of the subjects became fatigued during the run, and these subjects increased the length of their strides by an average of 5% (Verbitsky et al., 1998). The same group of authors has similarly found 5% increases in stride length in runners who were fatigued over the course of thirty minutes at a velocity 5% faster than their anaerobic thresholds (Mizrahi et al., 2000b). Other work has demonstrated similar increases in stride length between competitive and recreational runners during a treadmill run simulating 10-kilometer race conditions, each of around 2% (Siler & Martin, 1991).

The two previously mentioned studies, like many in the running literature, report changes in stride length or stride frequency without reporting detailed timing of gait events. Without information concerning changes in stance or flight time, it is unclear whether the increased step time (implied by increased step length at a constant velocity) is a result of increased flight time, stance time, or some combination of the two. However, literature that reports changes in these components does exist. Dutto and Smith (2002) have reported increases in stride length in ten of fifteen highly trained runners after an exhaustive run at 80% of VO₂ max. Nine of the fifteen participants used significantly different contact times as a result of fatigue, six of these nine being longer than in the un-fatigued state.

Elliott and Roberts (1980) have reported more consistent temporospatial adjustments in the latter stages of a high-intensity 3-kilometer run. Though the small sample size (n=8) likely kept results from reaching statistical significance,
stance time increased by 5% while flight time decreased by 14%. This may indicate that despite increased time spent in contact with the ground, fatigue caused these athletes to leave the ground with reduced vertical velocity. Nicol and colleagues (1991) asked runners to perform brief maximal sprints over a series of force platforms at several points during a marathon run. Compared to those collected at the beginning of the race, data from the 30-kilometer mark demonstrated 16% reduced velocity, increased contact time and reduced average ground reaction forces. Fatigue induced by cyclical flexion and extension movements of submaximal intensity also tends to prolong the period of contact in upper extremity exercise (Gollhofer et al., 1987).

Regardless of whether or not a run induces fatigue, increasing stride length while maintaining running velocity results in reduced leg stiffness (Farley & Gonzalez, 1996). When stiffness is calculated as peak vertical ground reaction force divided by change in distance between the hip and ankle joints between initial contact and midstance, the decreased stiffness accompanying increased stride length is caused by greatly increased leg length change, in spite of slightly increased peak forces. While the non-fatigued runners studied by Farley and Gonzalez (1996) demonstrated greater knee flexion in response to increasing stride length, similarly to fatigue-induced changes, the increased peak forces they report lie in contrast to results from fatigued runners. Gerlach et al. (2005) have reported increased stride length accompanied by reduced ground reaction force impact peak and loading rate values in fatigued runners.
2.9 Fatigue effects: Neuromuscular

As the nervous system is responsible for activating and coordinating muscles, changes in movement patterns likely reflect alterations in nervous control signals. The observed changes in kinematics and temporospatial variables resulting from running fatigue are ultimately rooted in altered control by the nervous system and impaired muscle function. The latter culprit has been discussed at length in section 2.2, while the former is addressed below.

For the purposes of this review, neuromuscular performance refers to the timing of muscle activation in cyclical movements, the magnitude of muscle activation, and stretch-induced reflexes. While an ideal experimental design would permit the observation of gradual changes in these measures over the course of a fatiguing effort, logistical challenges typically constrain such measurements to before and immediately after fatiguing performances.

A number of researchers have evaluated changes in neuromuscular performance induced by marathon runs (Avela & Komi, 1998; Avela et al., 1999; Nicol et al., 1991). Maximal countermovement jumping exercise after marathon runs consistently demonstrates increased contact time and decreased takeoff velocity, compared to unfatigued performance (Avela & Komi, 1998; Nicol et al., 1991). Average vertical force during the eccentric phase of these maximal jumps tends to decrease significantly, which may be related to reduced stiffness of extensor muscles as they act eccentrically to control the descent of the body (Avela & Komi, 1998). This reduced stiffness may, in turn, be due to reduced activation of these extensor muscles prior to ground contact and during the eccentric phase.
(Avela & Komi, 1998; Avela et al., 1999), as well as to reduced stretch reflex response from these muscles (Avela & Komi, 1998). Runs as short as twenty minutes in duration can reduce the torque resulting from individual muscle twitches, thereby magnifying the force-reducing effects of impaired muscle activation (Skof & Strojnik, 2006).

These results imply a reduced ability of extensor muscles to act eccentrically to control the descent of the body, at both the knee and the ankle. Kuitunen, Avela, Kyröläinen, Nicol, and Komi (2002) have used repeated countermovement sled jumps to induce fatigue, and have calculated joint stiffness as the change in joint moment divided by the change in joint angle. An exhaustive battery of continuous jumping yields results similar to those resulting from marathon running, with greatly reduced stiffness of the knee and ankle during the braking phase of the movement.

The reduced stiffness of extensor muscles following ground contact may be partially due to changes in the timing of muscle activation, in addition to reduced magnitude of activation. Fatigue induced with maximal isometric contractions has been shown to increase the electromechanical delay of the knee extensors (Chan, Lee, Wong, Wong, & Yeung, 2001). Additionally, fatigue induced by brisk uphill walking has been associated with delayed activation of knee flexor and extensor muscles during a jump stop maneuver (Nyland et al., 1994). These results indicate that central as well as peripheral nervous impairments may underlie fatigue-induced alterations in muscle activation timing.
If co-contraction of muscles about the lower extremity joints occurs during normal, unimpaired running, then fatigue of flexor muscles may also prove detrimental to performance. A thorough analysis of the landing phase of heel-toe running (Bobbert et al., 1992) has revealed a rapid increase in net knee extensor torque following impact (5,400 Nm/s), which far exceeds the ability of an average person to develop torque in isometric conditions (1,000 Nm/s). The authors theorized that activation of muscles on both sides of the knee joint prior to ground contact could explain this phenomenon. Upon ground contact, the knee flexes rapidly. This motion allows shortening of the knee flexors, reducing their force contribution, while simultaneously lengthening the pre-activated knee extensors (Bobbert et al., 1992). In this scenario, co-contraction both facilitates a rapid increase in the net extensor moment and removes slack from the patellar tendon, such that the lengthening of the quadriceps musculotendon unit following impact occurs primarily in the contractile component, thus promoting stretch reflex activity.

Electromyographic data collected during running provide evidence that co-contraction prior to ground contact does in fact occur at both the knee and the ankle (Elliott & Blanksby, 1979; Mann et al., 1986), and that this mechanism helps to establish a stable base of support at initial contact (Elliott & Blanksby, 1979). If co-contraction is necessary both to provide stability and to assist in slowing the descent of the body, fatigue of muscles on either side of these joints could impair this mechanism. The knee extensors in particular perform work to absorb energy and control descent during the first half of stance (Winter, 1983). It seems a cruel
irony that a fatigued runner’s inability to produce adequate extensor moments in early stance puts him in a position of deeper knee flexion, requiring greater knee extensor moments at midstance (Derrick et al., 1998).

2.10 Implications for Injury and Efficiency

When fatigue causes mechanical changes in running form, it is important to identify the consequences of these changes. While the basic research reviewed above describes the nature of some of these gait alterations, it is the implications of fatigued mechanics that motivates much of the running fatigue literature. It is possible that continuous running in the fatigued state places a runner at increased risk of injury. Likewise, the idea that certain aspects of unfatigued running form maximize efficiency implies that deviations from this form may increase the metabolic cost of running. These two concepts are discussed below.

As a general rule, mental and physical fatigue increase the likelihood of injury, and athletes tend to show a greater risk of injury in the latter stages of an exercise bout (Whiting, 1998). Muscular fatigue decreases the force required to incur a strain injury when the muscle is eccentrically loaded, and reduces the amount of energy a muscle can absorb while acting eccentrically (Mair et al., 1996). While muscle strains are acute injuries, most research devoted to the etiology of running injuries focuses on overuse injury mechanisms.

It is possible that the altered running form accompanying fatigue puts athletes at greater risk of developing tibial stress fractures, and efforts have been made to quantify tibial stress using accelerometers attached to the lower leg.
(Clansey et al., 2012; Derrick et al., 2002; Mercer, Bates, Dufek, & Hreljac, 2003; Mizrahi et al., 2000a; Mizrahi et al., 2000b; Verbitsky et al., 1998). However, acceleration of the leg is not necessarily proportional to force transmitted through the tibia, due to the concept of effective mass. When a runner's foot contacts the ground, the mass that is immediately accelerated by the ground reaction force is less than the mass of the runner's body. This "effective mass" is determined by such factors as impact velocity, knee angle at the instant of contact, and rate of knee flexion following contact (Valiant, 1990). Increases in the degree of knee flexion at contact, as often occurs in response to fatigue, tends to increase tibial acceleration while actually decreasing the magnitude of impact ground reaction forces (Derrick, 2004).

In contrast to acceleration profiles, a recent review (Zadpoor & Nikooyan, 2011) has found that the loading rate of ground reaction forces following impact is associated with the development of tibial stress fractures. Fatigue studies have been inconsistent in this area, reporting impact loading rate to either increase (Clansey et al., 2012) or decrease (Gerlach et al., 2005) after exhaustive running. However, as discussed above, the protocol employed by Clansey and colleagues (2012) may have resulted in differences between the gait mechanics reported, and the mechanics used during the fatiguing effort. Research into this particular injury has not consistently demonstrated any increased risk associated with fatigue, though it has been suggested that in the fatigued state, the role of dorsiflexor muscles in protecting the anterior tibia from tensile strain is compromised (Mizrahi et al., 2000a).
While most gait analysis techniques employed by running fatigue studies are limited to the sagittal plane, many injury mechanisms are associated with lower extremity movements in the frontal or transverse planes. Patellofemoral pain syndrome (PFPS) is one such injury, and is aggravated by excessive hip adduction. A recent study from Dierks, Manal, Hamill, and Davis (2008) has demonstrated that runners suffering from PFPS experience greater midstance hip adduction at the end of a fatiguing run, and that this increased range of motion is associated with strength loss of the abductor muscles.

Ankle motion in the frontal plane may also constitute a risk factor for overuse injury. Meta-analysis has revealed that athletes demonstrating a greater degree of navicular drop, a common measure of standing pronation, develop medial tibial stress syndrome at significantly higher rates than those with less subtalar motion (Newman, Witchalls, Waddington, & Adams, 2013). Rearfoot eversion is a component of pronation, and as such, the increasing peak values of eversion that develop with accumulating fatigue (Derrick et al., 2002; van Gheluwe & Madsen, 1997) may constitute a fatigue-induced injury mechanism.

Prolonged running tends to have the effect of reducing athletes’ running economy, defined as the aerobic demand (VO₂) of running at a given speed. Defined this way, increased oxygen consumption is considered a reduction in running economy. Kyröläinen and colleagues (2000) have reported a 16% increase in oxygen consumption between the beginning and end of a marathon, run at a constant velocity. Xu and Montgomery (1995) have found increases in oxygen consumption of 4.9% and 6.6% after running for 1.5 hours at 65% and 80% of
participants’ VO₂ max, respectively. If one assumes that higher intensity running for the same duration incurs more fatigue, then these results suggest that a greater degree of fatigue is associated with greater increases in aerobic demand.

Explaining decreases in running economy brought about by strenuous running is difficult without knowledge of concurrently developing mechanical changes used by the runner. Potential explanations may be proposed, however, by reviewing the literature correlating specific mechanical variables with oxygen consumption. In their classic study of Groucho running, McMahon, Valiant, and Frederick (1987) performed a myriad of analyses on the effects of altering knee joint flexion angle at midstance. In positions of extreme flexion, oxygen consumption was found to increase by up to 50% with the knee flexed 20° beyond the preferred angle. This dramatic decrease in running economy was accompanied by decreases in vertical stiffness of the body, similar to results of fatigue studies (Dutto & Smith, 2002). Similarly, Valiant (1990) has estimated that for every 5° increase in midstance knee angle, oxygen consumption should increase by roughly 25%. This does not bode well for the runners tested by Derrick and colleagues (2002), whose 4° increase in knee angle induced by fatigue may have increased aerobic demand by up to 20%.

Prior research has also demonstrated that the metabolic cost of continuous contermovement jumping is related to the degree of knee flexion during ground contact (Bosco et al., 1982). High flexion jumps and low flexion jumps of the same height for one minute resulted in calculated mechanical efficiency values of 30% and 39%, respectively. High flexion jumping also demonstrated lower flexion
velocity and smaller average vertical forces than jumps using a low degree of knee flexion.

Heise and Martin (1998) have evaluated differences in vertical stiffness as a potential factor underlying interindividual variability in running economy. An inverse relationship was found between oxygen consumption and vertical stiffness, indicating that runners who use a more "compliant" style may be less efficient. However, because the stiffness reported was calculated as peak ground reaction force divided by vertical displacement of the athlete's center of mass during the stride, it is therefore unclear whether reduced stiffness resulted from a higher center of mass position in flight, a lower position in stance, or some combination of the two.

In addition to the effects of such specific mechanical variables as degree of knee flexion, past research has found that simply deviating from a runner's preferred stride frequency causes notable increases in metabolic cost. Cavanagh and Williams (1982) have reported that experienced recreational runners tend to freely choose a stride length that minimizes metabolic cost compared to longer or shorter strides. Hamill and colleagues (1995) recruited a cohort of runners of various fitness levels and found that more experienced runners used a freely chosen stride rate that was significantly closer to their optimal stride rate than did the inexperienced runners. Following, other research has demonstrated that recreational runners who, initially, are not especially adept at optimizing stride rate to minimize metabolic cost can be trained to do so, if given visual and audio feedback (Morgan et al., 1994).
The mechanical variables described above are by no means an exhaustive list of factors that may reduce a fatigued runner's economy. In fact, the increased aerobic cost associated with deviations from optimal stride frequency are no doubt due to more subtle mechanical changes that, together, determine stride frequency. It is possible that fatigue during a run that initially requires recruitment of only slow-twitch oxidative fibers gradually causes more recruitment of less economical, fast-twitch glycolytic fibers as slow fibers fatigue and become unable to produce the required force. While prior work has demonstrated a gradual increase in recruitment of fast-twitch fibers during exhaustive cycling exercise (Vøllestad et al., 1984), no study to date exists, demonstrating this shift in response to running-induced fatigue.

Despite the abundance of variables yet unstudied, it is likely that decreased running economy caused by fatigue is due in part to the implied changes in joint moment requirements evidenced by kinematic, temporospatial, and neuromuscular studies such as those described above. Increases in joint moments, at the knee in particular, imply increases in muscle forces. Finally, increases in muscle force can only be accomplished by recruitment of additional fibers or increases in the discharge rate of recruited fibers, each of which would incur increases in metabolic cost (Bigland-Ritchie & Woods, 1974).

**2.11 Conclusion**

The ramifications of running while fatigued include reduced efficiency and increased risk of musculoskeletal injury. While the former may not come as a
surprise, an understanding of the specific mechanisms by which running becomes more taxing with accumulating fatigue allows for the design of specific training interventions which could delay fatigue effects. The latter consequence is perhaps more relevant to recreational runners, who represent the majority of the running population and tend to become injured at greater rates than their competitive counterparts.

The evidence presented in this review strongly suggests a mechanism by which fatigue at the muscular level ultimately causes the changes in gait associated with reduced running economy and increased injury risk. Prior research in physiology and biomechanics establishes the following chain of events: Neuromuscular changes, such as delayed and reduced activation of extensor muscles, diminish a runner's ability to generate forces early in the stance phase. Increased joint flexion at midstance requires additional time to propel the body upwards against gravity, resulting in a prolonged stance phase. This prolonged stance phase likely underlies lengthening of the stride cycle, and thus the resulting increase in stride length commonly reported by fatigue studies. Reaching a position of deeper joint flexion requires greater muscle activation for an increased duration and thus, reduces running economy. Simultaneously, reduced ability of the ankle musculature to control foot motion after ground contact results in an increase in the degree and rate of rearfoot motion and fatigued hip abductors are unable to prevent excessive hip adduction at midstance, each of which may constitute separate injury mechanisms.
The missing link in this chain of events is the joint moment required to control the descent of the body and subsequently propel it upwards during stance. This is but one component of the stride cycle in which joint moment analysis could, and arguably should, be applied. In the absence of direct muscle force measurements, joint moments can fill this void in our knowledge and provide crucial information in the continuing endeavor to understand and mitigate the consequences of fatigue in running.
CHAPTER 3
METHODS

The protocol of this study involved the collection of temporospatial, kinematic and kinetic data during a high-intensity run to volitional exhaustion. Several variables were calculated from the data collected. Hypotheses were supported or rejected based on the statistical significance of changes in these variables observed between the initial and final collection periods.

3.1 Participants

Eight recreational runners were recruited to participate in this study from the University of Massachusetts and surrounding communities. The eight participants were four males and four females, and had the following characteristics (format: mean ± 1 standard deviation): Age of 24.6 ± 5.8 years, height of 170.0 ± 9.8 cm, body mass of 69.6 ± 15.1 kg, and weekly training volume of 41.6 ± 14.2 km at the time of testing.

Because of the inherent risks of sustained vigorous running, all participants were required to sign a physical activity readiness questionnaire (PARQ) before beginning the experimental protocol. This questionnaire aimed to identify any risks of participation based on past exercise-induced cardiac discomfort. Participants also read and signed an informed consent document in accordance with the policies of the University of Massachusetts.
3.2 Protocol

The proposed study involved two visits to room 28 of Totman Gymnasium at the University of Massachusetts, Amherst. The first visit served to describe participants’ fitness levels and determine the appropriate running speed for the fatiguing run that was conducted on the second visit.

Participants reported to the laboratory for their first visit after having abstained from vigorous physical activity during the previous forty-eight hours (Spurway & Jones, 2007). Participants were also asked to abstain from caffeine during the four hours prior to the test and from alcohol during the previous day. Effort was made to ensure that participants completed both visits at or near the same time of day, as circadian rhythm can have an effect on maximal exercise performance (Reilly, 2007).

After filling out the PARQ and signing the informed consent document, body mass was determined using a standard scale to facilitate normalization of oxygen consumption. Participants wore their own running shoes during both visits in order to eliminate any effects of adjusting to unfamiliar shoes. Participants then completed a multistage incremental treadmill test according to the guidelines presented by Spurway and Jones (2007). This test was used to determine the velocity and oxygen consumption rate associated with lactate threshold (LT). The incremental test was conducted on a Bertec FIT treadmill (Bertec Corporation, Columbus, OH). Blood lactate concentration was determined using a handheld lactate meter (Lactate Pro 2, Arkray, Inc., Kyoto, Japan). A metabolic cart (TrueOne,
Parvo Medics, Sandy, UT) was calibrated before the test according to the manufacturer specifications. Ventilation and respiratory data were recorded and expressed over 5-second epochs, and inclination of the treadmill belt remained at 0% throughout the test.

The running velocity of the initial stage was set 0.5 m/s slower than each participant’s predicted 5-kilometer race pace (Spurway & Jones, 2007). Each stage lasted three minutes, and ended with brief periods of walking at 1.3 m/s while 0.3 μL of blood was collected from a fingertip for lactate content determination before beginning the next stage at a 0.14 m/s faster velocity. Brief periods spent walking between stages do not significantly affect the LT value determined (Gullstrand, Sjödin, & Svedenhag, 1994). The test continued using three-minute stages until end-stage blood lactate concentration exceeded 4 mmol/L, at which point the test was terminated.

Running velocity associated with LT was determined by two independent evaluators not otherwise associated with the study. These evaluators used plots of blood lactate concentration against running velocity to identify the velocity just preceding a sudden and sustained increase in blood lactate (Figure 3.1). The mean velocity selected by the reviewers was taken as the lactate threshold (Jones & Doust, 1998). Problems associated with subjective identification of threshold values are minimized with small speed increments between stages, such as those utilized in the present protocol. Additionally, prior work has demonstrated high reliability associated with subjective interpretation of physiological thresholds (Caiozzo et al., 1982; Davis, Vodak, Wilmore, Vodak, & Kurtz, 1976). In the event that the reviewers
were not able to identify an obvious threshold, the fastest velocity eliciting a blood lactate concentration less than 4 mmol/L was used, instead. This blood lactate concentration is often referred to as the onset of blood lactate accumulation (OBLA). Once determined, the running velocity at LT or OBLA was applied to a plot of oxygen consumption ($\dot{VO}_2$) against running speed to determine the rate of oxygen consumption at the threshold.

Figure 3.1. Typical blood lactate (squares) and heart rate (diamonds) responses to a multi-stage incremental treadmill test in an endurance athlete. In this example, lactate threshold occurs at 15 km/h. Adopted from Spurway and Jones (2007).

The primary objective of the first laboratory visit was the establishment of an appropriate running velocity for the exhaustive run, during which kinematic and kinetic data collection occurred. The desired running velocity gradually induced muscular fatigue without accumulation of lactate within the working muscles. The maximum exercise intensity that can be maintained indefinitely without lactate
accumulation in the blood is the maximal lactate steady state, or MLSS (Faude, Kindermann, & Meyer, 2009). While the exercise intensity of MLSS is best determined using discrete constant-velocity tests (Faude et al., 2009), the running velocities at LT and at OBLA are not significantly different from and are highly correlated with (r = 0.94 and r = 0.93, respectively) the velocity of MLSS among trained runners (Jones & Doust, 1998). In order to ensure that the fatiguing run did not result in lactate accumulation, the exhaustive run was performed at a velocity eliciting a value that is 5% lower than the associated with LT. It is important to control for VO2 accumulation because lactate converts to lactic acid and causes acidosis within muscle fibers (McArdle, Katch, & Katch, 2010), which in turn decreases contractile velocity and calcium sensitivity of muscle (Debold, 2012). While it is difficult to say how these cellular effects of lactate accumulation impact running performance, controlling the relative intensity of an exhaustive effort helped to ensure that the fatigue experienced by all participants resulted from the same underlying mechanisms. Additional details regarding the determination of exhaustive run speeds are given in Appendix D.

The second laboratory visit followed at least forty-eight hours after the first, with participants having observed the same dietary and activity restrictions. Participants changed into tight-fitting bicycle shorts and warmed up at a self-selected recreational pace for ten minutes. After warming up, retro-reflective markers were placed on anatomical landmarks to identify four body segments for motion capture (right foot, leg, thigh, and pelvis). These markers were placed on the toe tip, head of the first and fifth metatarsals, medial and lateral malleolus, medial
and lateral femoral epicondyle, greater trochanter, anterior superior iliac spines, iliac crests, posterior superior iliac spines, and on the sacrum. Additionally, plastic plates mounted with four non-collinear markers were secured to the lateral aspect of the thigh and leg segment, and plates with three markers each were taped to the back of the right shoe, just above the sole material. Markers served to establish local coordinate systems for each of the defined segments (Hamill, Selbie & Kepple, 2014).

A global coordinate system was established with orthogonal medio-lateral, fore-aft, and vertical components designated X, Y, and Z, respectively. The exhaustive run was performed on the same Bertec FIT treadmill used during the first visit. Ground reaction force and torque signals from the right side embedded force plate passed through an amplifier (AM6501, Bertec Corporation, Columbus, OH) and an analog-to-digital converter (Analog Interface, Qualysis, Gothenburg, Sweden) before being stored on a personal computer (Precision 3610, Dell, Round Rock, TX). Three-dimensional kinematic data were collected using five Oqus 500+ cameras (Qualysis, Gothenburg, Sweden). The motion capture system was calibrated to a volume of roughly 4 m³ above the treadmill belt. Prior to the exhaustive run, the right side force plate was zeroed and the participant stood on the treadmill in anatomical position for the collection of a calibration trial. This calibration trial served to relate the positions of the markers to specific limb segments. Due to drift in the force signal, it was necessary to zero the force plate approximately every fifteen minutes throughout the run. This was accomplished by stopping the treadmill belt and zeroing the right side plate while participants stood
on the left plate. These interruptions lasted no more than twenty seconds. The run was performed at the constant velocity previously determined until the participant reported being unable to continue. A fan was placed directly in front of participants to facilitate convective cooling. Verbal encouragement was given throughout the run. Upon completion of the test, participants were provided with sports drinks and encouraged to stay in the laboratory until they felt recovered.

Data were collected at regular intervals throughout the run. Every two minutes, participants’ ratings of perceived exertion were recorded (Borg, 1970). Every minute, kinematic and kinetic data were collected for ten seconds. Kinematic data were collected at a sampling rate of 60 Hz, while ground reaction force data were collected from the treadmill force transducers at a rate of 120 Hz. Data collection was coordinated within Qualysis Track Manager (QTM) (Qualysis, Gothenburg, Sweden). For the purposes of this investigation, fatigue is defined as an increase in rating of perceived exertion developing without an increase in exercise intensity (belt speed). Exhaustion is defined as an inability to continue running at the target speed.

3.3 Data Analysis

Because of the unique duration of each participant’s exhaustive run, six collections from each participant, as evenly spaced as possible over the duration of the run, were selected for further analysis. These collections corresponded to elapsed times of one minute, 20% of total duration, 40% of total, 60% of total, 80% of total, and the final collection. In cases when the total number of collections did
not result in data at the exact desired percentage of total duration, the collection
closest to the desired percent duration was used.

Individual marker trajectories were tracked and labeled within QTM. Marker-trajectories and force signals from the right side force plate for each participant were exported from QTM to the biomechanical modeling software Visual 3-D (C-Motion Inc., Germantown, MD). Raw data were also exported to text files, and a custom MATLAB (R2009b, The MathWorks, Natick, MA) program was used to determine optimal cutoff frequencies for low-pass filtering, based on the analysis of residuals (Winter, 2005). Within Visual 3-D, a 6 degree of freedom method was used to track modeled segments at each recorded time step. Using this approach, segment positions were described by the displacement and rotation of local segment coordinate systems within the laboratory coordinate system, as determined by an optimization algorithm (Hamill, Selbie & Kepple, 2014). The location of the hip joint center was estimated based on the positions of the anterior and posterior superior iliac spines (Seidel, Marchinda, Dijkers, & Soutas-Little, 1995). Raw marker position data were fourth-order low-pass filtered at the frequencies determined by residual analysis. Specifically, the low-pass filter cutoff frequency used to filter all markers for a participant was the average of the optimal frequencies indicated by residual analysis of a marker on the right heel and a marker on the pelvis. This method resulted in a group mean cutoff frequency of 15 Hz for marker position data. A customized pipeline within Visual 3-D was employed to identify the gait events of heel strike and toe-off from the position of foot markers and the ground reaction force signal.
The same pipeline was then used to calculate joint angles at each recorded time step, resolved using a joint coordinate system approach (Grood & Suntay, 1983), and expressed as the distal segment rotating in the joint coordinate system. Intersegmental forces and torques were calculated using an inverse dynamics approach and the post-processing methods described by Edwards, Troy and Derrick (2011). These methods involve combining unfiltered segmental kinematics, unfiltered ground reaction forces, and unfiltered centers of pressure of the ground reaction force vector with literature values of segment masses, centers of mass locations, and moments of inertia, based on individual subject whole body mass and segment dimensions. Segment masses reflected fractions of the whole body mass reported by Dempster (1955), and individual segments were modeled as geometric shapes to determine center of mass locations and moments of inertia (Hanavan, 1964). Using this approach, the inverse dynamics calculations resulted in "raw" joint moments. Net joint moments were expressed in the local reference frame of the proximal segment forming the joint, with positive values indicating extension/plantar flexion and negative values indicating flexion / dorsiflexion.

Joint moment and angular impulse values were scaled to dimensionless quantities to facilitate comparisons between participants, as described by Hof (1996). Specifically, original joint moment values in units of newton meters were divided by the product of participant body weight in newtons and leg length in meters. Leg length was defined as the length of a vector between the greater trochanter and lateral malleolus markers during the standing calibration trial.
Angular impulse values were scaled by dividing original values in units of newton meter seconds by the quantity:

\[
[ \text{body mass} \times (\text{leg length})^2 \times \sqrt[3]{\frac{g}{\text{leg length}}} ]
\]

where \( g \) is the gravitational constant (9.81).

The temporospatial, joint angle, and joint moment data resulting from these analyses were exported in text format for calculation of the desired dependent variables using custom-written MATLAB software. Before the calculation of joint moment dependent variable values, unfiltered ground reaction forces and unfiltered joint reaction forces were used to determine low-pass filter frequency cutoffs for the "raw" joint moment signals. Joint moment data were filtered on a joint-by-joint basis according to the frequency content of the external force acting on the distal end of the relevant segment (Edwards et al., 2011). Low-pass filters applied to the joint moment data used cutoff frequencies of 10 Hz, 10 Hz and 20 Hz for the hip, knee and ankle, respectively, averaged across all participants.

Previous research indicates that eight to ten ipsilateral footfalls are necessary to adequately capture variability in ground reaction force profiles in running (Bates, Osternig, & Sawhill, 1983). Therefore, ten stride cycles of the right leg were selected from each ten-second collection for further analysis. For each stride, contact time was defined as the time elapsed between initial contact and toe-off. Peak magnitudes of joint angles and joint moments were taken directly from the exported text files. Hip extension angular impulse was calculated as the time integral of the sagittal hip joint moment signal between initial contact and the point
at which the signal crosses from net extension to net flexion moment. Similarly, plantar flexion angular impulse was calculated as the time integral of the sagittal ankle moment signal between the first and last frames of net plantar flexion moment during the stance phase. Once these values were determined for each of the ten strides analyzed per collection, the mean across ten strides for each variable was considered representative of one of the six collection periods, described previously.

Several additional dependent variables were analyzed as part of a secondary analysis. Secondary dependent variables describing discrete joint angle values from each stride included peak hip flexion angle relative to the pelvis and the laboratory, peak hip adduction angle, peak knee flexion angle during swing phase, hip and knee angles at initial contact relative to their proximal segments, peak ankle eversion angle, and sagittal plane angles of all four modeled segments at initial contact relative to the laboratory. Secondary dependent variables generated from joint moment data included knee extension angular impulse generated during stance phase, peak hip extension moment, peak hip abduction moment, peak plantar flexion moment, and peak dorsiflexion moment. Stride time was defined as the time elapsed between subsequent right heelstrikes, and cycle as the quotient of stance time divided by stride time, expressed as a percentage. Vertical loading rate was defined as the change in vertical ground reaction force divided by the time elapsed between 20% and 80% of the time window between initial contact and the impact peak of the vertical ground reaction force profile. An additional external force
metric described the magnitude of the vertical ground reaction force impact peak as a percentage of the active peak.

Another secondary variable described the runner’ vertical limb stiffness. Stiffness was defined as the peak vertical ground reaction force recorded during a stride divided by the vertical displacement of the runner’s center of mass between the instants of heelstrike and midstance. Vertical stiffness values were rendered dimensionless by scaling vertical ground reaction force values by body weight and vertical displacement values by leg length.

3.4 Statistical Analysis

Separate repeated-measured ANOVA (RMANOVA) for contact time, peak knee flexion angle, hip extension and ankle plantar flexion angular impulse, peak hip flexion moment, and peak knee extension moment were used to determine whether changes occurred over the course of the exhaustive run (SAS version 9.1.3, SAS Institute Inc., Cary, NC). Values used in RMANOVA testing were the mean of dependent variable values over the ten strides within each collection period. Orthogonal polynomial comparisons were then employed to determine whether the relationship between variable values and time could be significantly captured by a trend line or curve. Regression lines were then fit to the data and $r^2$ or $R^2$ values calculated for linear or quadratic trends, respectively. The same statistical methods were applied to test for time effects on secondary outcome variables. These methods helped describe the manner in which mechanical changes developed over the course of the exhaustive run.
CHAPTER 4

RESULTS

4.1 Participant Characteristics and Incremental Test Results

The incremental test revealed that participants reached lactate threshold at a running velocity of $3.81 \pm 0.50$ m/s, and consumed $48.4 \pm 5.5$ ml/kg/min of oxygen at this pace. Based on these data, running speeds utilized for the exhaustive runs were $3.63 \pm 0.46$ m/s (range: 3.06 - 4.29 m/s). Exhaustive run durations were $34.5 \pm 14.8$ minutes (range 14 - 60). Participants reported their ratings of perceived exertion every two minutes during the exhaustive run. RPE values increased from $10.25 \pm 2.19$ after the second minute of running to $19.13 \pm 0.83$ in the final two minutes (Figure 4.1).

Figure 4.1. Ratings of perceived exertion. Colored lines represent data from individual participants. Error bars represent the standard error about the mean among participants.
4.2 Temporal Changes

Participants did not significantly change their stride length or stride time during the exhaustive run (RMANOVA p = 0.809). Time spent in stance phase increased (p = 0.004) from 0.247 ± 0.015 to 0.256 ± 0.020 seconds between the beginning and end of the run, and the slope of this linear trend was significantly greater than zero (p < 0.001) (Figure 4.2A). This increase in stance time was mirrored by a decrease in swing time that did not reach statistical significance (p = 0.881). These two trends contributed to an increase in duty cycle from 36.1 ± 3.2 to 37.2 ± 4.2 %. RMANOVA indicated that this change approached significance (p = 0.07) and regression analysis revealed that the slope of this trend was significantly greater than zero (p = 0.004) (Figure 4.2B).

![Figure 4.2](image)

Figure 4.2. Stance time (A) and duty cycle (B) over the course of the exhaustive run. Error bars represent standard error about the mean across participants.
4.3 Kinematic Changes

Plots of the hip, knee and ankle joint angles in the sagittal, frontal and transverse planes at each of the six time points analyzed are displayed in Figure 4.3. Plots of joint moments at the hip, knee and ankle in three planes are displayed in Figure 4.4. Participants generally reached greater levels of peak joint flexion as the exhaustive run progressed. Peak hip flexion angle increased significantly ($p < 0.001$) from $49.3 \pm 13.6$ to $52.6 \pm 13.7$ degrees between the beginning and end of the run (Figure 4.5A). Further analysis indicated that this increase in flexion angle was due to changes in global thigh angle, rather than an increased degree of anterior pelvic tilt.

In the frontal plane, an increase in peak hip adduction angle from $16.4 \pm 4.5$ to $17.3 \pm 4.9$ degrees was described by a trend line with a significant positive slope ($p = 0.012$), but RMANOVA results indicated that differences between time points were not significant ($p = 0.244$) (Figure 4.5B). The peak knee flexion achieved in the stance phase increased from $43.2 \pm 4.9$ to $43.9 \pm 3.9$ degrees over the course of the run (Figure 4.5D). This increase was described by a trend line with a slope significantly greater than zero ($p = 0.047$) though RMANOVA time effects did not reach statistical significance ($p = 0.467$). Participants also adopted a greater degree of knee flexion at the instant of initial ground contact ($9.5 \pm 5.8$ vs. $11.3 \pm 5.2$ degrees). Similarly, this linear trend possessed a significant positive slope ($p = 0.007$) without registering significant RMANOVA time effects ($p = 0.141$) (Figure 4.5C). Kinematic changes at the instant of ground contact also included a
significant decrease in the degree of ankle dorsiflexion (p = 0.002) (Figure 4.5E). Participants tended to reach a greater amount of rearfoot eversion as the run progressed (12.1 ± 6.5 vs. 13.6 ± 5.4 degrees), though this change was not significant (p = 0.12) (Figure 4.5F). An increase in the maximum knee flexion angle in the swing phase from 102.3 ± 18.3 to 105.7 ± 17.6 degrees was described by a linear trend with a slope significantly greater than zero (p = 0.014), though RMANOVA indicated that time effects were not significant (p = 0.194).

4.4 Ground Reaction Force Changes

Vertical and anterior-posterior ground reaction force curves are displayed in Figure 4.6. All force values are scaled by body weight. Repeated measures analysis of variance revealed that a steady increase in vertical loading rate from 55.1 ± 13.6 to 61.5 ± 18.4 body weights per second was significant (p = 0.04), and regression analysis indicated that the slope of this linear trend was significantly greater than zero (p = 0.001) (Figure 4.7). The magnitude of the vertical ground reaction force active peak decreased from 2.43 ± 0.21 to 2.39 ± 0.24 body weights. The slope of this linear trend was significantly less than zero (p = 0.015) but RMANOVA time effects were not significant (p = 0.144). A final ground reaction force variable described the magnitude of the impact peak as a percentage of the magnitude of the active peak. This fraction increased significantly over the course of the run (p = 0.005) from 67.7 ± 11.6 to 72.5 ± 14.2% following a linear trend with a slope significantly greater than zero (p < 0.001).
Figure 4.3. Hip, knee and ankle joint angles in the sagittal, frontal and transverse planes at six time points during the exhaustive run. Vertical lines indicate toe-off.
Figure 4.4. Hip, knee and ankle joint moments in the sagittal, frontal and transverse planes at six time points during the exhaustive run. Vertical lines indicate toe-off.
Figure 4.5. Joint angle trends over the course of the exhaustive run. Variables in the left column describe flight phase kinematics and the resulting orientation of segments at heelstrike. Variables in the right column describe peak joint excursions at or near midstance. Error bars represent standard error about the mean across participants.

4.5 Vertical Stiffness Changes

Plots of vertical displacement of participants' centers of mass and vertical stiffness are displayed in Figure 4.8. As the run progressed, the center of mass displacement increased then decreased in a manner significantly captured by a quadratic trend (p = 0.025). Repeated measures analysis of variance indicated that
Figure 4.6. Vertical (A) and anterior-posterior (B) ground reaction forces at six points during the exhaustive run.

Figure 4.7. Vertical ground reaction force loading rate. Error bars represent standard error about the mean across participants.

This displacement did not change significantly during the run (p = 0.249). Vertical limb stiffness did not change in a significant manner (RMANOVA p = 0.560), nor did it follow a trend with a slope significantly different from zero.
Figure 4.8. Center of mass vertical displacement between initial contact and midstance (A) and vertical limb stiffness in stance (B). Error bars represent standard error about the mean across participants.

4.6 Joint Moment Changes

Plots of the hip, knee and ankle joint moments in the sagittal, frontal and transverse planes at each of the six time points analyzed are displayed in Figure 4.4. Joint moment and angular impulse values are reported in dimensionless units.

At the hip, the peak extension moment increased by 5.4% over the course of the run (Figure 4.9A). Regression analysis indicated that this change was described by a linear trend with a significant positive slope (p = 0.017), though RMANOVA time effects were not significant (p = 0.151). Angular impulse generated by the hip extensors during stance also increased along a trend with a significant positive slope (p = 0.041), though variability among participants prevented this 7.4% change from reaching significance (RMANOVA p = 0.219) (Figure 4.9B). In the frontal plane, the
peak hip abduction moment utilized by participants increased \( (p = 0.151) \) by 5.0% between the beginning and 80% of the duration of the run, before decreasing in magnitude by 0.7% of the initial value at the end of the run (Figure 4.9C).

Regression analysis indicated that the slope of the data between 0% and 80% of the

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Figure 4.9. Selected joint moment trends over the course of the exhaustive run. Peak hip extensor, hip abductor and knee extensor moments describe the kinetics of controlling the descent of the body. Hip extensor impulse gives an indication of exertion from the hip extensors during stance. Peak plantarflexor and dorsiflexor moments illustrate the role of muscles in propelling the body into flight and controlling foot motion after impact, respectively. Error bars represent standard error about the mean across participants.
run’s duration was significantly greater than zero \( (p = 0.04) \), though the trend was not significant when the final time point was included. The peak adduction angle described above followed a similar pattern, increasing for the majority of the run’s duration and reversing direction in the final minute of the run.

At the knee, the peak extension moment utilized by participants varied over the course of the run \( (p = 0.452) \), with no significant trend in the data (Figure 4.9D). During the final 20% of the run’s duration a trend was more consistent, with six participants demonstrating a decrease in this peak moment and one showing no change. The angular impulse exerted by the knee extensors did not change during the run \( (p = 0.657) \) and the data did not follow any significant trends.

Peak plantar flexion moment decreased by 1.4% over the course of the run, but this change was not significant \( (p = 0.578) \) and regression procedures indicated that the slope of this increase was not significantly greater than zero \( (p = 0.066) \) (Figure 4.9E). The peak dorsiflexion moment utilized by participants decreased to a greater degree over the course of the run, by 22.7%, though time effects were similarly not significant \( (p = 0.384) \) and the data were not significantly described by a trend line \( (p = 0.204) \) (Figure 4.9F). Angular impulse exerted by the plantar flexors was greater at the end of the run than the start by 1.3% but RMANOVA time effects were not significant \( (p = 0.617) \) and the slope of this trend was not significantly greater than zero \( (p = 0.243) \).

Changes in joint moment patterns over the course of the run varied between participants, as exemplified by the peak hip extension moment (Figure 4.10A). While the pooled group data follow a significant linear increasing trend, only two of
eight participants saw steady increases in this value between each sequential time point, and only four of eight reached their highest peak extension moment in the final minute of the run. Trends were more consistent for other joint moment variables. Hip extension angular impulse increased in the last 20% of the run for all but one participant (Figure 4.10B), when RPE values indicate that fatigue effects were at their strongest.

![Graphs showing peak hip extensor moment and hip extensor angular impulse](image)

**Figure 4.10.** Peak hip extensor moment (A) and hip extensor angular impulse (B). Colored lines belong to the same participants in both plots. Error bars represent the standard error about the mean across participants.

In several cases, trends in joint moment patterns that developed during the run mirrored trends in joint angle patterns at the same joint. This is exemplified at the knee, where changes in the degree of flexion reached at midstance closely resemble changes in knee extensor impulse generated during the stance phase for
three participants (Figure 4.11). At the hip, four participants who demonstrated steady increases in the peak flexion angle late in the swing phase also demonstrated regular increases in either peak hip extensor moment or extensor angular impulse generated in the stance phase.

Figure 4.11. Peak knee flexion angle reached in stance (A) and knee extensor angular impulse generated during stance (B). Colored lines indicate data for the same three participants in both plots.
CHAPTER 5
DISCUSSION

This investigation aimed to use a joint moment-based analysis to offer a new perspective on changes in running form that accompany fatigue. Joint moment patterns followed significant linear trends over the course of the exhaustive run, most notably at the hip. Changes in joint moment patterns were accompanied by alterations in joint angles and external forces, which corroborate results of previous fatigue studies. In some cases, altered joint moments indicated shifts in potential motor control strategies and injury mechanisms that would have been overlooked in a purely kinematic analysis.

5.1 Primary Outcomes

It was hypothesized that as runners became fatigued, stance time would increase due to a reduced ability to generate forces against the ground. The results supported this prediction, as group mean results reached statistical significance and all eight participants utilized a longer stance time at the end of the run than they had at the start. This finding is consistent with previous literature, which indicates that fatigue tends to elongate periods of force generation in running (Dutto & Smith, 2002) as well as in upper extremity countermovement exercise (Gollhofer et al., 1987). Contrary to the results of many previous investigations, participants did not change their stride length or stride time as the run progressed, and decreases in swing time were not significant. An increase in duty cycle, which approached
statistical significance, indicates that these athletes spent more time in contact with the ground in both an absolute and a relative sense.

It was predicted that as runners became fatigued, altered or reduced activation of knee extensor muscles would result in a greater degree of knee flexion at mid-stance. This hypothesis was not supported, likely due to the variable fatigue effects exhibited across participants (Figure 4.5). One athlete increased peak knee flexion angle by five degrees between the start and end of the run, while another decreased this angle by three degrees. Runners who do tend to utilize an increase in knee flexion with fatigue (including the majority of the present participants) may be increasing their rate of energy expenditure, as running with greater knee flexion can lead to substantially increased metabolic cost (McMahon et al., 1987; Valiant, 1990).

A second hypothesis describing sagittal plane knee mechanics was likewise not supported by the results. Trends in the peak knee extension moment were variable among the participants, with only two of eight runners displaying consistent increases in this peak moment (Figure 4.9). It was predicted that the peak extension moment would follow a pattern similar to that of the peak flexion angle at the knee, with the joint moment change necessitated by the increases in knee flexion angle commonly reported in the running fatigue literature (Derrick et al., 2002; Dutty & Smith, 2002; Mizrahi et al., 2000b). While the group results did not support this relationship between kinetic and kinematic fatigue effects, three participants demonstrated consistent changes in peak knee flexion angle that were accompanied by consistent changes in knee extensor angular impulse. As angular impulse values reflect both the magnitude and duration of exertion of the net joint
moment, it may be the case that angular impulse, more so than the peak joint moment, must change in order to accommodate changes in the degree of flexion at the knee.

Participants did not increase the peak hip flexion moment required to swing the leg forward. This kinetic change was expected to develop as a compensation for a fatigue-induced increased stride length and reduced swing time. However, neither of these temporospatial predictions were supported by the results in any significant way. It is worth noting that despite a lack of increase in the peak hip flexion moment in swing, all but one participant reached a greater hip flexion angle in late swing phase as the run progressed (Figure 4.3). This significant kinematic change implies a shift in hip flexor activation pattern away from peak moment generation, a reduction in eccentric control from the hip extensors, or some combination.

Two final hypotheses predicted a decrease in the angular impulse generated by the hip extensor and plantar flexor muscle groups during stance. The plantarflexor angular impulse hypothesis was rejected as this value increased by roughly 2% over the course of the run. The peak plantarflexor moment exerted during stance decreased steadily, though the first and final values differed by only 1.4%. Though these changes are small in magnitude, a decrease in the maximum moment exerted by the plantar flexors without a concurrent decrease in the angular impulse generated by these muscles may suggest a fatigue-induced change in activation patterns.

The hypothesis predicting decreased angular impulse exerted by the hip extensors was also rejected. This value increased by 7.4% and was described by a
linear trend with a slope significantly greater than zero, though RMANOVA time effects were not significant.

5.2 External Forces

Changes in external forces are ultimately rooted in muscular forces generated during both the swing and stance phases of running. A consistent and significant increase in vertical loading rate may have been the most surprising finding of this investigation. Group mean results indicate that loading rate increased by 11.1% over the course of the run (Figure 4.7), while the magnitude of the impact peak increased by 5.4%. This shift accompanying fatigue is potentially injurious, as a recent review has reported an association between increased loading rates and the development of tibial stress fractures (Zadpoor & Nikooyan, 2011).

Altered kinematics at heelstrike may partially explain the observed increase in vertical loading rate. Ankle angle at initial contact can influence impact forces, and runners in the present study adopted a progressively less dorsiflexed ankle position at contact over time. The modeling results of Gerritsen, van den Bogert and Nigg (1995) suggest that the observed 2.4° shift could have increased the impact peak magnitude by as much as 0.31 body weights. This estimation overshoots the observed increase in impact magnitude of 0.09 body weights, likely due to the simultaneous development of increased knee flexion at contact. Previous research by Derrick (2004) among others indicates that the sagittal knee angle at contact has a substantial influence on both impact forces and shock attenuation, and the 1.5°
increase in knee flexion angle at contact exhibited by the present participants would have tended to decrease the vertical loading rate.

In what may be the only previous investigation reporting ground reaction force profiles before and after running-induced fatigue, Gerlach and colleagues (2005) found that impact peak magnitude decreased by 7% while vertical loading rate decreased by 12% in a large group of recreational runners. These results are of similar magnitude but opposite sign from results of the present study. However, Gerlach et al. induced fatigue in their runners using a discontinuous VO₂ max test, and this difference in fatiguing protocols may underlie the discrepancy in results. It is possible that the fatigue responsible for the altered force profiles observed by Gerlach and colleagues differed from the gradually accumulating fatigue induced by the present protocol, which more closely replicates training and racing conditions.

Participants’ vertical limb stiffness did change significantly over the course of the exhaustive run (Figure 4.8B). This variable was investigated because its components, ground reaction force and center of mass vertical displacement, have each demonstrated significant changes in response to fatigue in prior studies (Dutto & Smith, 2002; Gerlach et al., 2005) and because running with lower vertical stiffness is associated with higher aerobic demand (Heise & Martin, 1998). The lack of change observed in the present results was likely the result of a quadratic trend in the center of mass displacement data (Figure 4.8A) combined with steadily decreasing peak ground reaction forces over the course of the run.
5.3 Utility of Joint Moments

Results of this investigation indicate that the utility of joint moment analysis as a lens through which to examine fatigue effects in running is mixed. Analyses of variance revealed no significant effects of time on joint moment values, likely due to high variability among participant responses to fatigue. However, in certain cases trends in joint moment patterns offer tentative explanations for observed changes in kinematic patterns, some of which may be associated with injury development and reduced running economy. Investigation of data from individual participants indicated that some runners displayed changes in kinetic and kinematic variables which closely resembled the fatigue mechanisms predicted at the hip and knee in the sagittal plane.

Changes in kinematics at heelstrike are naturally brought about by changes in kinetics during flight. As described above, changes observed in the magnitude of impact forces likely reflect the competing effects of increased knee flexion and decreased dorsiflexion at contact.

It is important to note that the increase in knee flexion at contact that developed during the exhaustive run was the result of changes in the global angle of the thigh, without concurrent changes in the ankle of the shank. A closer inspection of the sagittal hip joint moment (Figure 4.4) reveals that, starting at 80% of the gait cycle, a temporal shift is evident between the moments used at the beginning and end of the run. In the final minute of the run the extension moment, used to slow the forward swing of the leg (Novacheck, 1998), lags behind the moment from the first minute by 1-2% of the gait cycle. Activation of the hip extensors late in flight is
essential in preparing the limb for contact with the ground (Elliott & Blanksby, 1979), and this delay in the development of hip extension torque may have brought about the observed increase in knee flexion at contact in these runners. Previous literature has demonstrated that fatigue causes a delay in extensor muscle activation prior to ground contact in jump stop maneuvers, as well (Nyland et al., 1994), lending support to this proposed mechanism. Four participants demonstrated fatigue-induced changes in hip mechanics which may indicate a kinetic consequence of making contact with the ground in a position of deeper hip flexion. These four runners demonstrated consistent increases in peak hip flexion angle over the course of the run (as much as 6.2°) which developed concurrently with consistent increases in hip extensor angular impulse and the peak hip extensor moment generated in early stance.

The observed flatter foot position at contact is consistent with fatigue of the dorsiflexor muscles. Christina and colleagues (2001) have found that applying isokinetic fatigue to the dorsiflexor muscles prior to a run caused a decreased degree of dorsiflexion at contact as well as an increase in vertical loading rate, similar to results of the current investigation. The decrease in peak dorsiflexion moment observed in the present study may serve as evidence that runners experienced localized fatigue of this muscle group (Figure 4.7). Fatigue of these muscles may be responsible for the decreased degree of dorsiflexion at initial contact that developed with fatigue, described above. Strengthening of this muscle could potentially help in reducing risk of fatigue fracture.
When runners increase their degree of knee flexion, oxygen consumption increases (McMahon et al., 1987; Valiant et al., 1990). This effect can be substantial, evidenced by the 16% increase reported to occur in athletes running an entire marathon at a constant speed (Kyröläinen et al., 2000). Considering this upward drift in oxygen consumption, the hypothesis that an exhaustive run would result in increased knee extension moments was expected to provide a kinetic explanation for the link between knee flexion and oxygen consumption. Surprisingly, group mean data indicated that the peak knee extensor moment and the angular impulse generated by these muscles did not change during the run. However, while trends in individual participant data were often inconsistent, three runners demonstrated consistent trends over the course of the run which provide support for a kinetic link between kinematics and running economy (Figure 4.11). Specifically, the peak knee flexion angle reached in stance increased consistently and in parallel with increases in knee extensor angular impulse in two runners. A third runner demonstrated decreased knee flexion and angular impulse as the run progressed. The changes observed in this latter runner, lying in stark contrast to predictions, illustrate the myriad of adaptations employed by fatigued runners and highlight the importance of examining fatigue effects at all joints.

Increases in the magnitude of peak extensor moments and extensor angular impulse values were generally larger at the hip than at the knee. While literature relating hip mechanics to whole body oxygen demand in running is scarce, it should be noted that in the absence of changes in pelvic tilt, increasing knee flexion implies increasing hip flexion. Increases in the peak extensor moment and angular impulse
at the hip of 5.4% and 7.4%, respectively, may suggest that mechanical changes at the hip may play as large a role as those at the knee in offering partial explanation for fatigue-induced upward VO₂ drift. These kinetic findings should be interpreted with caution, however, as hip and knee moment variables showed no significant RMANOVA time effects. It is also possible that increases in oxygen consumption accompanying fatigue have as much to do with joint moments and muscular forces as they do with shifts in recruitment patterns in the active muscles toward less efficient, fast-twitch motor units as slow-twitch motor units cycle out of recruitment.

In contrast to the fatigue effects observed in selected runners at the sagittal knee and hip, changes in frontal hip mechanics demonstrate that joint moment trends did not consistently offer explanations for joint angle trends. The hip abductor muscles work to eccentrically control pelvic list during stance (Novacheck, 1998). Over the course of the exhaustive run, trends toward increasing peak adduction angle (1.4° from the first minute to 80% of the run’s duration) closely resembled trends toward increasing peak abduction moment (4.6% over the same period). Further analysis revealed that the hip adduction angle at initial contact increased by 0.5° over the same duration, indicating that the frontal hip range of motion increased in early stance, despite an increase in the joint moment controlling this motion. Unlike the kinematic changes in sagittal hip angle, this increase in joint excursion following impact is not readily explained by a delay in generating an eccentric joint moment.
5.4 Strengths and Limitations

Exhaustive running on treadmills presents many psychological and physiological challenges for participants. Some potential stressors were anticipated and addressed (i.e. using a fan to cool runners, allowing participants to wear their own shoes), but several others could not be addressed without altering the test protocol.

The marker clusters strapped to the thigh and shank were carefully attached at a comfortable level of tightness before the run. However as the run progressed, vasodilation in the active leg musculature (Armstrong & Laughlin, 1983) could have increased the perceived tightness of the straps and acted as a subtle stressor. Drift in the treadmill force signal necessitated brief "breaks" in the run to allow the force plate to be zeroed. The split-belt feature of the treadmill may have presented another mild stressor, as it restricted participants to running either entirely on the right side belt or just right of center in order to ensure all right footfalls landed entirely on the right belt.

A final potential stressor takes the form of the constant-speed protocol, itself. The fixed speed of a treadmill prevents any subtle changes in speed that would be available to runners in overground conditions. This inability to modulate speed in response to subtle internal factors could be perceived as a stressor. To expect exhaustive efforts in steady speed conditions could even be regarded as futile, given that exhaustive endurance efforts so often involve considerable variation in running speed or cycling power output (Tucker et al., 2006a; Tucker et al., 2006b). In addressing this issue of speed modulation, future research utilizing exhaustive
treadmill running could consider prescribing a treadmill speed pattern that emulates common overground pacing strategies, or even giving participants full speed control during the run. In such a hypothetical protocol, comparing participants’ running form at intervals throughout the run could be accomplished by requiring participants to briefly run at a prescribed speed at intervals throughout the run.

An important limitation of any joint moment analysis is the inability to determine the role of co-contraction in producing movement. This limitation is especially poignant in a study of gradually-accumulating fatigue, as reduced activation of antagonist muscles could carry energetic benefits, while also exacting a cost in the form of reduced joint stability.

Despite a sound physiological motivation, attempts to tailor the speed of the exhaustive run according to each participant’s running speed at lactate threshold may have introduced additional variability to the protocol. The stage of the incremental test at which lactate threshold occurred was determined by reviewers, who visually inspected plots of blood lactate level against running speed. While this is the recommended method of deciding an athlete’s lactate threshold (Jones & Doust, 1998), technical difficulties resulted in recorded blood lactate values that often did not follow the expected exponential trend (Figure 3.1) and were likely erroneous. The difference in running speed between successive stages of the incremental test (0.14 m/s) represents an increase in pace from 7:30 to 7:13 per mile. Misidentifying the speed at which a runner reached lactate threshold could easily result in an exhaustive run speed that exceeded a runner’s threshold or did
not provide sufficient challenge. This potential discrepancy between desired and actual relative intensity of the exhaustive runs may underlie the wide range of exhaustive run durations (14 to 60 minutes). Despite this wide range of times to exhaustion, it is noteworthy that all participants reported an RPE score of 19 or 20 in the final minutes of the run. This serves as an indication that levels of somatic stress were nearly identical among participants at the end of the run, despite potential differences in the physiological underpinnings of this distress.

This study has several strengths. Despite the aforementioned drawbacks of steady speed protocols, the enforcement of a constant running speed does ensure that changes in gait due to accumulating fatigue are not masked by changes in gait simply due to gradual slowing. This logic is generally accepted, and it has been many years since researchers last allowed their fatigued runners to slow down (Elliott & Ackland, 1981).

Another strength concerns the six regularly-spaced collection periods at which data were analyzed. Prior investigations describing the effects of running-induced fatigue have used two collection periods (early and late in the fatiguing run) (Abt et al., 2011) or three, including a collection midway through the run (Clansey et al., 2012; Derrick et al., 2002). Tibial acceleration and metabolic data have been analyzed at up to six points in prior studies of fatiguing running, but joint angle and moment data have not been examined at this frequency (Mizrahi et al., 1997). Using differences between two or three time points to describe fatigue effects does not provide adequate resolution to examine the time course of mounting fatigue effects. Unexpected trends in gait variables, exemplified by the frontal hip kinematics and
kinetics in the current study, are all but invisible when analyzing data at so few points throughout a fatiguing run.

That only one sixth of the run duration elapsed between each successive collection period made certain trends visible that would have otherwise gone unnoticed. In contrast to the varying patterns in joint moment variables demonstrated by participants during the first 80% of the exhaustive run (Figure 4.9A), runners demonstrated greater consistency in changes during the final minutes, when fatigue likely had the greatest effect on joint mechanics. This is exemplified by the hip extensor angular impulse and the peak knee extensor moment, for which all but one participant followed the same trends (increasing and decreasing, respectively) in the final 20% of the run.

5.5 Conclusion

This investigation marks the first effort to describe changes in joint moment patterns over the course of an exhaustive run. Hypotheses regarding joint moment changes were based on well-documented kinematic, temporal and neuromuscular effects of fatigue. While external forces and certain joint angle variables changed significantly over the course of the run, the variety of kinetic adaptations to mounting fatigue utilized by the participants limited significant time effects for joint moment-related variables. However, several joint moment variables displayed significant linear trends over the course of the run and individual participants often demonstrated intuitive congruency between kinetic and kinematic fatigue effects, despite inconclusive group results.
The main findings of this investigation can be summarized as follows: i) Despite increasing levels of fatigue, some runners adopt mechanics that include increased joint moments generated by large muscle groups. This is exemplified by the peak hip extension moment and extensor angular impulse, which increased steadily over the course of the run for four participants. ii) Changes in joint moment patterns provide partial explanations for two independent kinematic contributors to the observed increase in vertical loading rate. The reduced dorsiflexion moment may explain a reduction in dorsiflexion angle at initial contact, while a delay in the generation of hip extension moment could underlie the increased knee flexion at contact. iii) Kinetic changes accompanying fatigue induced by submaximal running are more pronounced at the hip than at either the knee or ankle, evidenced by the absence of any significant trends in the joint moment data at either of the latter two joints.

Despite the aforementioned limitations, investigating fatigue effects using joint moment analyses is not a fruitless endeavor, and such future research employing a larger number of runners and allowing participants to use more natural pacing strategies could lead to greater confidence in understanding the kinetic compensations used by fatigued runners in conditions more similar to running in the real world.
APPENDIX A

PHYSICAL ACTIVITY READINESS QUESTIONNAIRE

Participant Number____________

MODIFIED PHYSICAL ACTIVITY READINESS QUESTIONNAIRE

Date (MM/DD/YY): _____/_____/

Please answer the following questions to the best of your knowledge (circle YES or NO).

1. Yes No Has your doctor ever said you had heart trouble or a heart murmur?
2. Yes No Do you ever suffer pains or discomfort in your chest?
3. Yes No Has the doctor ever told you that your blood pressure was too high? (systolic > 160 mm Hg or diastolic > 90 mm Hg on at least 2 separate occasions)
4. Yes No Do you smoke cigarettes or have you quit in the past 6 months?
5. Yes No Do you have diabetes or prediabetes?
6. Yes No Have any men under 55 or women under 65 in your immediate family suffered a heart attack or sudden cardiac death?
7. Yes No Has your serum cholesterol ever been elevated?
8. Yes No Is there any physical reason not mentioned here why you should not follow an activity program even if you wanted to?

Below please provide an explanation for any of the questions to which you answered YES.
___________________________________________________________________________
___________________________________________________________________________
___________________________________________________________________________
___________________________________________________________________________

____________________________________________________________
APPENDIX B
RUNNING QUESTIONNAIRE

Participant Number__________

RUNNING QUESTIONNAIRE

Date (MM/DD/YY): ______/_____/______

Age (in years) ________________

Gender: (check one) Female ________ Male ________

Height: _____ Feet, _____ inches or ______ cm

Weight: ________________ lbs or __________ kg

Weekly running mileage: _________ miles or __________ kilometers

Please Circle One:

How would you describe your footstrike pattern? Rearfoot / Midfoot / Forefoot

Please Check One:

Do you use specialized insoles or foot orthotics? YES ________ NO ________

Do you have any injuries that may affect the way you walk or run: YES_______ NO ______

If YES, please describe the injury, and when it happened:
________________________________________________________________________
________________________________________________________________________
________________________________________________________________________

Have you injured your lower extremities in the last six months? YES ______ NO ________

If YES, please describe the injury, and when it happened:
________________________________________________________________________
________________________________________________________________________
APPENDIX C

INFORMED CONSENT DOCUMENT

Consent Form for Participation in a Research Study
University of Massachusetts Amherst

Researcher(s): Nathaniel Smith, B.S. and Brian Umberger, PhD
Study Title: Gait Changes during Exhaustive Running

1. WHAT IS THIS FORM?

This form is called a Consent Form. It will give you information about the study so you can make an informed decision about participation in this research.

This consent form will give you the information you will need to understand why this study is being done and why you are being invited to participate. It will also describe what you will need to do to participate and any known risks, inconveniences or discomforts that you may have while participating. We encourage you to take some time to think this over and ask questions now and at any other time. If you decide to participate, you will be asked to sign this form and you will be given a copy for your records.

2. WHO IS ELIGIBLE TO PARTICIPATE?

Subjects must be recreational runners, and must be free from major injury or surgery to the legs or back during the previous six months. Subjects must be rearfoot (heelstrike) runners and be currently training at a volume of 20 to 80 kilometers (12 to 50 miles) per week. Subjects must be at least 18 and at most 45 years old to participate, and can be women or men of any ethnic background.

3. WHAT IS THE PURPOSE OF THIS STUDY?

We are conducting this research study in order to thoroughly describe the changes in running form that develop as runners become tired. This information will help to fill gaps in our understanding of the way fatigue affects runners.

4. WHERE WILL THE STUDY TAKE PLACE AND HOW LONG WILL IT LAST?

The study will involve two visits to room 28 in Totman hall on the University of Massachusetts Amherst campus. The first visit will last approximately one hour, and the second will last between 1.5 and two hours. Therefore the total time commitment is between 2.5 and three hours. Subjects will not be contacted after the study ends.
5. WHAT WILL I BE ASKED TO DO?

If you agree to take part in this study, you will be asked to complete two visits:

Visit #1

Preparation:
You will be asked to observe certain diet and exercise restrictions before the first visit, in order to control for factors that may affect your running performance. These restrictions include:
• Abstain from vigorous physical activity for the forty-eight hours prior to the visit
• Abstain from alcohol for the twenty-four hours prior to the visit
• Abstain from caffeine for four hours prior to the visit

Upon Arriving at the Lab:
• You will be asked to sign this Informed Consent document
• You will fill out a Physical Activity Readiness Questionnaire. This questionnaire is designed to identify any health factors which may make it unsafe for you to participate.
• You will also be asked to complete a running questionnaire, containing questions about your current training volume, footstrike pattern, and history of recent injuries or surgeries.
• Note that these questionnaires contain items that address your eligibility to participate in this study, and that it is therefore possible that your answers may disqualify you.
• You will then change into your own running shoes, shirt, and shorts.
• You will weigh yourself with a standard scale.

Performing the Incremental Treadmill test:
The primary aim of this first visit is to determine the value of variables that describe your aerobic fitness, including your lactate threshold and maximal oxygen uptake (VO₂ max). These values will be determined using a standard incremental treadmill test. This test will involve running for brief stages of progressively increasing speed. The test uses equipment and follows a protocol described below:
• During the test, your heart rate will be monitored using a wireless heart rate monitor.
• Your oxygen consumption will also be monitored, using a metabolic cart. The equipment on the cart will determine the gas content of your inspired and expired air by means of a flexible plastic tube connecting the device to a rubber mouthpiece.
• The test will begin with you running on the treadmill at a speed slightly slower than your 5-kilometer race pace.
• Every three minutes, you will be asked to briefly stand on the sides of the treadmill belt. One of your fingers will be cleaned with alcohol and pierced with a spring-loaded lancet. Two to three drops of blood will be collected using a handheld measurement device, and your finger will be bandaged. Before resuming running, the speed of the treadmill will be increased slightly. Your finger will be lanced and blood collected between five and seven times total, each time using a different finger.
• These three-minute stages will continue until the concentration of lactate in your blood (as determined using the handheld device) reaches a threshold value, at which point the time between stages will decrease to two minutes, and the investigators will stop collecting blood between stages.
• These two-minute stages will continue until you reach the point of maximal oxygen uptake, as determined from your breathing and heart rate.
Visit #2

Prior to the Fatiguing run:

- You will be asked to observe the same dietary and activity restrictions as for the first visit, with the exception of the vigorous activity restriction increased to seventy-two hours.
- You will then be asked to change into your own running shoes and shirt, as well as the shorts provided.
- You will warm up by running on the treadmill for ten minutes at a recreational pace.
- After the warm-up, small reflective spherical markers and plastic clusters of markers will be taped to your right foot, leg, and pelvis. These markers allow the investigators to track your motion during the run.
- Once all markers are in place, you will be asked to stand still on the treadmill for ten seconds while the camera system is calibrated to your unique body dimensions.

Performing the Fatiguing run:

- The Fatiguing run will use a constant speed, which will be tailored to your unique physiology based on the information collected during the first visit. The pace will be slightly slower than your lactate threshold, and should fall between the paces you would use for a 10-kilometer and a half marathon race.
- The run will continue at this speed until you report being unable to continue running or you wish to stop for any reason.

6. WHAT ARE MY BENEFITS OF BEING IN THIS STUDY?

If you complete this study, you will personally benefit from knowledge of your physiology. Specifically, the first visit will be used to determine your maximal oxygen uptake (VO$_2$ max), running speed at VO$_2$ max, and your running speed at lactate threshold. The value of maximal oxygen uptake is a strong indicator of cardiorespiratory fitness. Your running speeds at lactate threshold and VO$_2$ max are helpful reference values for training and racing.

In addition to these personal benefits, the information gathered in this study will hold potential benefit for all runners. Results may indicate that specific training may be beneficial to runners in maintaining running efficiency and reducing risk of injuries when runners perform in fatiguing conditions.

7. WHAT ARE MY RISKS OF BEING THIS STUDY?

During the treadmill run completed on the first visit, capillary blood will be drawn from your fingers. You will experience slight pain when the lancet first breaks the skin, and the site may be sore to the touch for a few hours following. There is a roughly one in ten chance that bruising will develop at the site. The risk of local infection at the puncture site is less than one in one thousand. Risk of infection will be minimized by use of a new needle, cleaning the site with alcohol prior to puncturing, gloves worn by the investigator, and bandaging the finger immediately after blood is collected.

Both visits will involve strenuous treadmill runs. Running at high speed as in the first visit, or for a long duration as in the second visit, will be physically uncomfortable in a manner similar to the experience of participating in a race. It is likely that you will experience delayed-onset muscle soreness (DOMS) in your leg muscles during the two to three days following each visit. Although
you have been asked to participate based on your familiarity with running, it is foreseeable that you may feel faint or light-headed during the treadmill runs. If you do begin to feel faint or light-headed, simply inform the investigator and the test will be terminated immediately. There is also a very small chance that you may suffer a cardiac episode during or after the treadmill runs. If you lose consciousness at any point, the investigators will immediately stop the treadmill and give emergency care. One investigator will stay with you to give care while another dials emergency services. It should be emphasized again that you have been asked to participate in this study based on your familiarity with running, thus minimizing the chances of such an episode. The physical activity readiness questionnaire is also being employed to help reveal cardiac risk factors associated with exercise.

This study will involve two visits, each of which must be preceded by two to three days of abstention from vigorous physical activity, including running. Therefore including the visits themselves, you will be required to take six days off of your regular training. It should be noted, however, that the visits themselves will involve runs strenuous enough to minimize the effects of missing your regular training. Aside from these physical inconveniences, you are being asked to abstain from alcohol during the twenty-four hours prior to each visit and caffeine during the four hours prior to each visit.

8. HOW WILL MY PERSONAL INFORMATION BE PROTECTED?

The following procedures will be used to protect the confidentiality of your study records. Study records will include several values recorded on paper, including your height, weight, age, and predicted 5-kilometer race pace. Records will also include information relating your oxygen consumption, heart rate and blood lactate level to your running speed, which will be processed on a laptop computer and subsequently printed. Study records will also include several electronic files containing data describing your motion during the run completed on the second visit. Research records will be labeled with a code instead of using identifying information. A master key that links names and codes will be maintained in a secure location, specifically in a locked cabinet which is only accessible to the investigators. The master key will be destroyed 3 years after the close of the study. All electronic files, including data files and results spreadsheets containing identifiable information will be labeled with a code instead of identifying information. Any computer hosting such files will also have password protection to prevent access by unauthorized users. Only the members of the research staff will have access to the passwords. At the conclusion of this study, the researchers may publish their findings. Information will be presented in summary format and you will not be identified in any publications or presentations.

9. WHAT IF I HAVE QUESTIONS?

Take as long as you like before you make a decision. We will be happy to answer any question you have about this study. If you have further questions about this project or if you have a research-related problem, you may contact the researchers, Nathan Smith (425-770-6434) or Brian Umberger (413-545-1436). If you have any questions concerning your rights as a research subject, you may contact the University of Massachusetts Amherst Human Research Protection Office (HRPO) at (413) 545-3428 or humansubjects@ora.umass.edu.
10. CAN I STOP BEING IN THE STUDY?

You do not have to be in this study if you do not want to. If you agree to be in the study, but later change your mind, you may drop out at any time. There are no penalties or consequences of any kind if you decide that you do not want to participate.

11. WHAT IF I AM INJURED?

The University of Massachusetts does not have a program for compensating subjects for injury or complications related to human subjects research, but the study personnel will assist you in getting treatment.

12. SUBJECT STATEMENT OF VOLUNTARY CONSENT

When signing this form I am agreeing to voluntarily enter this study. I have had a chance to read this consent form, and it was explained to me in a language which I use and understand. I have had the opportunity to ask questions and have received satisfactory answers. I understand that I can withdraw at any time. A copy of this signed Informed Consent Form has been given to me.

Participant Signature: __________________________  Print Name: __________________________  Date: __________________________

By signing below I indicate that the participant has read and, to the best of my knowledge, understands the details contained in this document and has been given a copy.

Signature of Person Obtaining Consent: __________________________  Print Name: __________________________  Date: __________________________
APPENDIX D

DETERMINING EXHAUSTIVE RUN SPEED

Running speeds utilized for the exhaustive runs were set based on blood lactate and oxygen consumption data, collected during an incremental test conducted on the initial visit. The steps involved in this process are outlined below.

Step 1: Identification of Lactate Threshold speed

Two independent reviewers examined plots of blood lactate level against running speed at each stage. These reviewers were instructed to identify the speed of lactate threshold (LT) as the speed immediately preceding a sudden and sustained increase in blood lactate level with increasing speed. When this point was unclear, the final point preceding 4.0 mmol/L blood lactate was selected. In cases of disagreement between reviewers, the average of the selected LT speeds was used.

In the plot below, LT was identified by both reviewers at 3.78 m/s.
Step 2: Determination of Exhaustive Run speed

Once the reviewers had decided upon the LT speed for a runner, a custom MATLAB program was used to establish a relationship between rate of oxygen consumption and running speed at each stage. This was accomplished by means of linear regression, with running speed as the independent variable and rate of oxygen consumption as the dependent variable. The number of data points used to establish this relationship varied between five and eight. An example of the oxygen consumption data and resulting regression line are illustrated in the figure below.

The speed utilized for the exhaustive run was set such as to elicit 95% of the oxygen consumption rate as that observed at the speed of LT. This speed is indicated by the red "plus" in the plot above.
APPENDIX E

A PRIORI SAMPLE SIZE ESTIMATION

Estimating the sample size required to achieve statistical significance at a
given power and significance level is ideally performed based on values of expected
differences and variability attained from pilot data or similar prior investigations.
In the case of the present investigation, no pilot data were available and changes in
joint moment patterns induced by fatigue had not been previously described.
Therefore, sample size necessary was estimated from effect sizes and variability
measures from kinematic variables observed to change during fatiguing running
(Derrick et al., 2002). Required sample size was estimated using methods described
by the National Research Council (2003). The sample size estimates resulting from
analysis of four documented fatigue effects were collected, and the mean of these
values was taken as the necessary sample size for the present investigation, with a
significance level (α) equal to 0.05 and power (1-β) equal to 0.8. This mean value
was 15. The table below lists the four variables examined by Derrick and colleagues
(2002) used to calculate sample size for the present study.

Table A.1. Sample size estimation from literature data. See text for details.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Value at beginning of run (mean ± SD)</th>
<th>Value at end of run (mean ± SD)</th>
<th>Resulting sample size estimation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Knee angle at contact (°)</td>
<td>164.9 ± 2.3</td>
<td>160.5 ± 2.9</td>
<td>7</td>
</tr>
<tr>
<td>Knee angle maximum (°)</td>
<td>127.7 ± 1.4</td>
<td>123.8 ± 1.5</td>
<td>4</td>
</tr>
<tr>
<td>Rearfoot angle at contact (°)</td>
<td>12.2 ± 1.6</td>
<td>13.6 ± 1.9</td>
<td>26</td>
</tr>
<tr>
<td>Rearfoot angle maximum (°)</td>
<td>-6.5 ± 1.4</td>
<td>-7.8 ± 1.4</td>
<td>20</td>
</tr>
</tbody>
</table>
Observed powers for dependent variables were calculated in order to assess the confidence with which the alternate hypothesis could be rejected. Power values were calculated using methods described by Fitzmaurice, Laird & Ware (2004). The method considers sample size, effect size, within-participant variance, and an alpha (\(\alpha\)) level of 0.05. The table on the following page lists observed powers for dependent variables.
Table A.2. Significance level and observed power for all dependent variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>RMANOVA p-value</th>
<th>Power (1 - β)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Joint Moment variables:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hip Extensor Impulse</td>
<td>0.219</td>
<td>0.484</td>
</tr>
<tr>
<td>Knee Extensor Impulse</td>
<td>0.657</td>
<td>0.230</td>
</tr>
<tr>
<td>Plantarflexor Impulse</td>
<td>0.617</td>
<td>0.268</td>
</tr>
<tr>
<td>Peak Hip Flexor</td>
<td>0.929</td>
<td>0.145</td>
</tr>
<tr>
<td>Peak Hip Extensor</td>
<td>0.151</td>
<td>0.552</td>
</tr>
<tr>
<td>Peak Hip Abductor</td>
<td>0.151</td>
<td>0.568</td>
</tr>
<tr>
<td>Peak Knee Extensor</td>
<td>0.452</td>
<td>0.417</td>
</tr>
<tr>
<td>Peak Ankle Plantarflexor</td>
<td>0.578</td>
<td>0.271</td>
</tr>
<tr>
<td>Peak Ankle Dorsiflexor</td>
<td>0.384</td>
<td>0.421</td>
</tr>
<tr>
<td><strong>Joint Angle variables:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peak Hip Flexion</td>
<td>&lt;0.001</td>
<td>0.773</td>
</tr>
<tr>
<td>Peak Hip Adduction</td>
<td>0.244</td>
<td>0.666</td>
</tr>
<tr>
<td>Knee Angle at Heelstrike</td>
<td>0.141</td>
<td>0.484</td>
</tr>
<tr>
<td>Knee Flexion in Stance</td>
<td>0.467</td>
<td>0.281</td>
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<tr>
<td>Knee Flexion in Swing</td>
<td>0.194</td>
<td>0.496</td>
</tr>
<tr>
<td>Peak Ankle Eversion</td>
<td>0.120</td>
<td>0.599</td>
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<tr>
<td>Dorsiflexion at Heelstrike</td>
<td>0.002</td>
<td>0.903</td>
</tr>
<tr>
<td><strong>External Force variables:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Impact / Active Peak Ratio</td>
<td>0.005</td>
<td>0.736</td>
</tr>
<tr>
<td>Vertical Loading Rate</td>
<td>0.040</td>
<td>0.618</td>
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<tr>
<td>Active Peak Magnitude</td>
<td>0.144</td>
<td>0.677</td>
</tr>
<tr>
<td><strong>Stiffness variables:</strong></td>
<td></td>
<td></td>
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<tr>
<td>Center of Mass Vert. Disp.</td>
<td>0.249</td>
<td>0.433</td>
</tr>
<tr>
<td>Vertical Stiffness</td>
<td>0.560</td>
<td>0.305</td>
</tr>
<tr>
<td><strong>Temporal Variables:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stride Time</td>
<td>0.809</td>
<td>0.236</td>
</tr>
<tr>
<td>Stance Time</td>
<td>0.004</td>
<td>0.839</td>
</tr>
<tr>
<td>Swing Time</td>
<td>0.881</td>
<td>0.171</td>
</tr>
<tr>
<td>Duty Cycle</td>
<td>0.070</td>
<td>0.655</td>
</tr>
</tbody>
</table>


