# Changes in Lower Extremity Movement Patterns Following Exercise-induced Fatigue and Verbal Feedback

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Abstract

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The present study investigated how exercise-induced fatigue and verbal feedback altered lower extremity coordination, variability, and kinetic variables in male and female athletes. Sixty-one healthy, club level athletes were divided into two groups: one that received a verbal feedback intervention post-fatigue, and one that did not. All subjects performed an unanticipated side-step cut, agility task, and vertical jump pre-fatigue, then completed an intense, intermittent, multi-directional fatigue protocol. Subjects in the feedback group received a quick verbal feedback intervention, focusing on landing technique. All subjects then repeated the pre-fatigue testing. The results indicated that fatigue caused subjects in the non-feedback group to change their coordination pattern in the sagittal plane, while subjects in the feedback group maintained their pre-fatigue pattern in all but one segment pairing (thigh-trunk frontal plane decreased post-fatigue in the feedback group). Fatigue caused all subjects to decrease their variability in the foot-shank and shank-thigh pairings in both the frontal and sagittal plane. Subjects in the non-feedback group also increased their anterior tibial shear force and vertical ground reaction force (VGRF), while the feedback group decreased their VGRF and knee extension moment. Fatigue also decreased vertical jump, and increased the score in the agility task, in both groups post-fatigue. These results suggest that an intermittent, multi-directional fatigue protocol causes a more in-phase, stiffer, less variable movement pattern, but that a simple verbal feedback intervention can prevent many of these changes from occurring. While the implications of these results on actual injury risk are unknown, these changes do suggest that verbal feedback may be an effective method for acutely altering some proposed risk factors for lower extremity injury, particularly anterior cruciate ligament injury. These results also suggest that muscular fatigue may be an important factor to consider when addressing potential injury risk in athletes.

# Dedication

To Scott, the love of my life, and the reason I was able to pursue this dream. Equal credit for this project goes to you, for your patience, love, and the occasional kick in the rear.

To my parents Richard and Diane, who provided me with a true passion for learning and academia. Your love and support have carried me my entire life.

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List of Abbreviations and Symbols

ACL: Anterior Cruciate Ligament

ANOVA: Analysis of Variance

ASIS: Anterior Superior Iliac Spine

ATSF: Anterior Tibial Shear Force

DP: Deviation Phase

DST: Dynamic Systems Theory

IRB: Institutional Review Board

KEM: Knee Extension Moment

KVM: Knee Valgus Moment

L5-S1: 5$^{th}$ lumbar vertebral spinous process-1$^{st}$ sacral vertebral spinous process

MARP: Mean Absolute Relative Phase

OA: Osteoarthritis

PAR-Q: Physical Activity Readiness Questionnaire

RPE: Rating of Perceived Exertion

VGRF: Vertical Ground Reaction Force

# CHAPTER 1

## Introduction

Injuries to the Anterior Cruciate Ligament (ACL) are one of the most costly and debilitating injuries suffered by athletes, both recreational and competitive. Researchers estimate that approximately 112,500 ACL injuries occur in the United States per year, leading to over 500,000 physician visits and an estimated cost of over $2 billion. [1-3] While the majority of athletes are able to return to their respective sport activities within 6-9 months following injury or surgery, follow-up studies suggest that up to 70% of ACL injured athletes no longer participate in the high-risk activity that lead to the initial injury in as few as three years post-injury. [4, 5] Of increasing concern are the recent studies that suggest that the risk of osteoarthritis (OA) increases dramatically within 10-15 years post-injury. Several authors report that a history of knee injury is one of the strongest predictors of knee OA, and that radiographic changes that suggest the development of OA are present as early as 10 years post-injury. [6-9] Most researchers and clinicians agree that, in light of the current body of research, finding ways of preventing ACL injury is vitally important for both the short-term performance and long-term health of athletes and other active individuals.

One factor that has been associated with increased injury risk, but has been inadequately studied, is muscular fatigue. Epidemiologic studies have demonstrated that injuries are more likely to occur during the second half of matches in rugby as well as in the latter stages of rugby practices. [10, 11] Hawkins reported that noncontact knee injuries occur most often during the final 15 minutes of the first half, and final 30 min of the second half, of soccer matches. [12, 13] It seems feasible that player fatigue may play a role in these statistics, but this conclusion is far from definitive. Several other factors, in addition to fatigue, may explain these results (including playing intensity, psychological factors, or training changes). However, most researchers agree that player fatigue is one of the major factors contributing to the increased incidence of injury during the later stages of games or practices.

A significant body of research has been developed in the last 20 years by researchers hoping to pinpoint the risk factors associated with ACL injury. Initial studies, using cadaver models, demonstrated that the most direct method of inducing strain on the ACL is to apply a linear shear force at the proximal tibia, causing translation of the tibial plateau anteriorly relative to the femur. [14, 15] The application of either a valgus moment, or an internal rotation moment, further increases the strain on the ACL and decreases the shear load necessary to cause failure of the ligament. [14-16] The researchers associated with these studies all concluded that combination loading, specifically an anterior shear force at the tibial plateau combined with a valgus and/or internal rotation moment, represent a high-risk joint loading situation for the ACL.

The identification of these high-risk joint loads led to further research to identify other factors that may mediate or exacerbate these joint loads. Many studies have compared female athletes, particularly those participating in soccer, basketball, and volleyball, to sport- or activity-matched males, as they demonstrate a 1.5-4.6 higher risk of ACL injury. [3] These between-gender studies suggest that a combination of excessive frontal plane movement at the knee and hip (valgus and adduction), decreased sagittal plane motion at the knee, increased ground reaction forces along the vertical, medial-lateral, and anterior-posterior axes, excessive quadriceps activation compared to the hamstrings, and poor neuromuscular control over the lower extremity and trunk, may be responsible for non-contact ACL injuries. [17-30]

However, most of these gender-related factors may only indirectly cause non-contact ACL injury. For instance, many researchers have demonstrated that contraction of the quadriceps musculature, particularly with the knee flexed less than 30 degrees, created a significant amount of anterior tibial shear force (ATSF) at the tibial plateau, which may be offset by increased force production by the hamstring musculature. [31-33] Thus, the proposed relationship of sagittal plane knee position, quadriceps muscle activity, and/or hamstring muscle strength to non-contact ACL injury may be largely due to their influence on the forces responsible for increasing ACL strain. This is further supported by recent regression analyses that found posterior ground reaction forces, vertical ground reaction forces, knee flexion angle, and quadriceps EMG activity, are all correlated and predictive of ATSF. [28, 32] A recent prospective cohort study found that knee valgus moment was significantly related to non-contact ACL injury in female athletes. [34] While the precise mechanism of ACL injury still remains elusive, the current research seems to strongly suggest that multi-planar loading of the knee joint is the most direct cause of non-contact ACL injuries.

Of the many studies that examine possible risk factors for ACL injury in athletes, very few examine how the neuromuscular system functions as a whole. The majority of research attempting to identify risk factors for ACL injury has looked at discrete joint angles at specific points in time. There have been far fewer that attempt to examine the forces that directly influence ACL strain, and how these forces may be related to the function of the entire lower extremity over a full cycle of movement. However, researchers have begun using tools from Dynamic Systems Theory (DST), which allow for the study of the behavior of the neuromuscular system in simpler variables.[35-37] Using the relative phasing of the segments of the lower extremity, single variables can be calculated to represent the coordination and variability present during a movement cycle.[36] These variables, mean absolute relative phase (MARP) and deviation phase (DP) have been used in multiple studies examining coordination and variability in diseased or injured populations.[38-40] Different relative phase patterns have been observed during gait between ACL-reconstructed patients and controls, with ACL-reconstructed subjects demonstrating more out-of-phase patterns during walking between the foot and shank, but more in-phase patterns between the shank and thigh during walking and between the foot and shank during running.[39] Decreased variability has been observed in injured runners with patellofemoral pain syndrome.[38] Despite the growing body of literature in motor control with regard to phase dynamics, coordination, and variability, these methods have not been utilized as part of the current ACL injury literature. Using these variables may provide insight into the organization of the movement patterns in the lower extremity, and how these patterns may relate to both the forces that cause ACL injury, as well as ACL injury itself.

One area of research that has received increased interest in the past 5 years is the effects of fatigue on the proposed biomechanical risk factors for ACL injury. Many researchers have suggested that the neuromuscular fatigue that accompanies physical activity may change the biomechanics and motor control of the lower extremity in a negative fashion, leading to an increased risk of ACL injury. Recent studies have confirmed that frontal plane motion and moments at the knee increase after a full-body fatigue protocol.[41-43] Several researchers have also found that sagittal plane angles at the knee and hip decrease post-fatigue, or have found that women do not change knee flexion angle post-fatigue while men increase their flexion angle.[41, 42, 44, 45] This suggests that the development of ATSF may be enhanced post-fatigue. Accordingly, two studies have demonstrated that anterior tibial shear force (ATSF) changes post-fatigue and suggest that females do not attenuate landing forces effectively pre- or post-fatigue, leading to higher ATSF compared to males pre- and post-fatigue.[42, 44] Together, these studies suggest that whole-body, or functional, fatigue alters lower extremity biomechanics in a potentially negative fashion, and may be partially responsible for the higher rate of injury observed in the latter stages of games.[10-13]

Despite the extensive research into the possible risk factors for ACL injury, there has been little research on how to quickly and acutely modify these risk factors. Promising injury prevention protocols have been developed and researched at various institutions across the country that a combination of plyometric, strength, balance, and flexibility training, occasionally combined with visual and/or verbal feedback. Many studies have demonstrated a reduction in ACL injury rates following intervention;[46, 47] however, these programs typically last 6-8 weeks and require adherence to the protocol and education about how and when exercises should be progressed. Quick, simple interventions that may be implemented in a few minutes, and that have an immediate impact on movement, have yet to be studied extensively as a method for injury prevention. This may be particularly important when an athlete is experiencing fatigue. Of the multiple components of injury prevention protocols previously studied, verbal feedback appears to be the best suited for this "quick, immediate" intervention. Prior studies have demonstrated that verbal feedback on proper landing technique can decrease ground reaction forces and increase knee flexion angle upon landing.

[48-50] In addition, augmented feedback (both visual and verbal) is effective for decreasing landing forces and increasing peak knee flexion angle. [51] These studies suggest that feedback can immediately change both lower extremity motion and landing forces in a manner that is associated with decreased risk of ACL injury.

The existing literature on ACL injuries is extensive, but several significant gaps remain with regard to the coordination and variability profiles of men and women that may represent groups at different risk of ACL injury, the effect of fatigue on these movement patterns as well as lower extremity kinetics, and how feedback may change movement coordination, variability, and kinetics. The overall goal of this study is to examine how lower extremity coordination, variability, and kinetics associated with increased ACL strain, change after a functional fatigue protocol, to see if differences exist between men and women in these variables, and to examine the ability of a quick verbal feedback protocol to alter coordination, variability and kinetics post-fatigue. A group of 61 subjects was recruited in order to better understand the organization of the neuromuscular system both pre- and post-fatigue during a dynamic, athletic task, and to examine whether a quick feedback protocol can change these variables in a favorable manner.

**Operational Definitions**

1. Muscular fatigue: Reduction in the maximum force that a muscle can produce as the result of physical exertion.
2. Functional fatigue protocol: An agility protocol consisting of forward running, backward running, side-step shuffling, side-step cutting, and 5 standing broad jumps. This protocol is repeated until the subject cannot complete the running portion in

under 150% of the first time recorded three times in a row. This protocol has been validated and shown to produce substantial muscular fatigue that is both central and peripheral in nature.

3. Feedback: Augmented externally-provided information about the kinematics of the side-step cutting task, combined with auditory information about how the foot should sound when contacting the ground. This type of feedback is considered “knowledge of performance”, or KP.
4. Unanticipated Sidestep Cutting Task: Subjects stand a distance equal to 50% of their body height from the front edge of a forceplate. A 17cm high hurdle was placed a distance equal to 25% of their body height from the front of the forceplate. The subject jumped with both legs over the hurdle. As they jumped, they cued the unanticipated task cueing system to provide the direction of the cut (sidestep=cut to contralateral side of dominant leg, crossover=cut to ipsilateral side of dominant leg). They land on their dominant leg (defined as the leg used to kick a ball for maximum distance) and cut in the cued direction.
5. Initial Contact: The first time point during each trial where the vertical ground reaction force recorded by the forceplate registers over 10N.
6. Toe-off: The first time point after Initial Contact where the vertical ground reaction force recorded by the forceplate registers less than 10N.
7. Stance Phase: The period of time between Initial Contact and Toe-off, representing the period of time where the subject’s foot is in contact with the forceplate during the side-step cut.

8. Segment Angular Position: The instantaneous angular position of the segment of interest relative to the world horizontal axis, expressed in degrees (°).
9. Segment Angular Velocity: The instantaneous angular velocity of the segment of interest, calculated using the change in angular position across 5 points divided by the change in time:

$$V_i = \frac{[X_{(i+y)} - X_{(i-y)}]}{2y}$$

where X= segment angular position, and y= time (in seconds). Expressed as degrees per second ($° \cdot s^{-1}$). Five data points were used as pilot testing revealed that a longer window (10 points) over-smoothed the velocity data. Stance phases are typically less than 100 data points in length, thus the 5 second window provided accurate data.
10. Phase Portrait: A graphical representation of the current state of a dynamic system, produced by plotting a segment's angular position (x-axis) versus its angular velocity (y-axis) [36].
11. Phase Angle: The angle (Θ) formed between the radius and the horizontal (x) axis, when the Cartesian (x,y) coordinates of the phase portrait are transformed into polar coordinates (radius, Θ) [36].
12. Relative Phase: Representation of the interaction and coordination between two segments. Calculated as $\theta_{\text{relative phase}} = \varphi_{\text{distal segment}} - \varphi_{\text{proximal segment}}$, where $\theta_{\text{relative phase}}$ is the relative phase angle, $\varphi_{\text{distal segment}}$ is the phase angle of the distal segment, and $\varphi_{\text{proximal segment}}$ is the phase angle of the proximal segment. Calculated for each time point during the stance phase [36].
13. Mean Absolute Relative Phase: A method of quantifying the in-phase or out-of-phase relationship between two segments. High values indicate a more out-of-phase pattern,

while lower values indicate a more in-phase relationship. Calculated as the average of the sum of the absolute values of each relative phase angle, [36]

$$MARP = \sum_{i=1}^{N} \frac{|\varphi \; relative \; phase|}{N}$$

14. Deviation Phase: A single term to quantify the stability of the organization of the neuromuscular system. High values indicate a highly variable and unstable coordination pattern, while low values indicate a stable and low-variability pattern. Calculated as the average of the standard deviations of the ensemble relative phase curve points [36],

$$DP = \frac{\sum_{i=1}^{N} |SD_i|}{N}$$

15. Knee extension moment: The combined contribution of the soft tissue surrounding the knee joint producing a moment in the direction of knee extension.
16. Knee valgus moment: The combined contribution of the soft tissue surrounding the knee joint producing a moment in the direction of knee valgus.
17. Anterior tibial shear force: The net force applied in the anterior direction at the Tibiofemoral joint, causing translation of the tibia anteriorly relative to the femur.

**Limitations/assumptions**

1. Segment kinematics calculated from the motion analysis system and biomechanical software were accurate and reliable.
2. Subjects performed the unanticipated sidestep cutting task in the lab in the same way they would perform the task on the field of play.
3. Subjects provided maximal effort during the fatigue protocol.

4. The effects of the fatigue protocol lasted long enough to complete the post-fatigue and post-feedback testing.
5. Subjects accurately followed directions in the feedback group.

**Delimitations**

1. 61 subjects (31 men and 30 women) were recruited from the local university population.
2. All subjects were between 18-30 years of age.
3. All subjects were healthy with no history of lower extremity or lumbar spine surgery in the past year, no history of knee surgery, and no history of lower extremity injury in the past 6 months.
4. All subjects were current participants in the University of North Carolina club sports teams (soccer, lacrosse, basketball, volleyball, or handball).
5. Segment kinematic data were collected from the trunk, thigh, shank and foot using an infrared video camera motion capture system.
6. Ground reaction force data were collected using a conductive forceplate.

**Statement of Problem**

Non-contact injuries to the ACL are debilitating, and likely increase the risk of developing OA within the knee joint. OA is one of the leading health problems in the United States, costing billions of healthcare dollars and creating disability in a large segment of the population. Understanding the factors that may lead to the development of non-contact ACL injuries, and finding ways to change these factors in a positive way, is an important area of research in sports medicine. This research project provides critical insights on how neuromuscular fatigue, a condition commonly associated with athletic performance, impacts some of the accepted risk factors for ACL injury. The additional information provided by the use of a verbal feedback intervention may give clinicians a new method of correcting movement patterns, and potentially preventing injury, in a quick and simple manner. Ultimately, this research project aims to further the understanding of how the lower extremity responds to fatigue and feedback, and how these changes may be used to prevent injury and protect the long-term joint health of active individuals.

**Independent Variables**

1. Fatigue (pre-fatigue vs. post-fatigue)
2. Gender (men vs. women)
3. Feedback (feedback vs. non-feedback)

**Dependent Variables**

1. Mean Absolute Relative Phase (MARP) for the following segment pairs:
    a. Foot-shank sagittal plane
    b. Foot-shank frontal plane
    c. Shank-thigh sagittal plane
    d. Shank-thigh frontal plane
    e. Thigh-trunk sagittal plane
    f. Thigh-trunk frontal plane
2. Deviation Phase (DP) for the following segment pairs:

    a. Foot-shank sagittal plane
    b. Foot-shank frontal plane
    c. Shank-thigh sagittal plane
    d. Shank-thigh frontal plane
    e. Thigh-trunk sagittal plane
    f. Thigh-trunk frontal plane
3. Selected knee kinetic measures
    a. Peak knee extension moment
    b. Peak knee valgus moment
    c. Peak Anterior Tibial Shear Force
    d. Peak Vertical Ground Reaction Force

**Research Questions**

1. How does a functional fatigue protocol alter the coordination, variability, and kinetics of the lower extremity during the stance phase of an unanticipated sidestep cutting task in a healthy, athletic population?
    a. Compare the Mean Absolute Relative Phase (MARP) values pre- and post-fatigue for the following segment pairs: foot-shank sagittal plane, foot-shank frontal plane, shank-thigh sagittal plane, shank-thigh frontal plane, thigh-trunk sagittal plane, thigh-trunk frontal plane.
    b. Compare the Deviation Phase (DP) values pre- and post-fatigue for the following segment pairs: foot-shank sagittal plane, foot-shank frontal plane,

shank-thigh sagittal plane, shank-thigh frontal plane, thigh-trunk sagittal plane, thigh-trunk frontal plane.

    c. Compare the peak knee extension moment, knee valgus moment, peak anterior tibial shear force, and peak vertical ground reaction force, during the first 40% of the stance phase pre- and post-fatigue.

2. How does an acute intervention (verbal feedback) affect the post-fatigue coordination, variability, and kinetics of the lower extremity during the stance phase of an unanticipated sidestep cutting task in a healthy, athletic population?
    a. Compare the pre- and post-feedback Mean Absolute Relative Phase (MARP) values between the feedback and non-feedback groups for the following segment pairs: foot-shank sagittal plane, foot-shank frontal plane, shank-thigh sagittal plane, shank-thigh frontal plane, thigh-trunk sagittal plane, thigh-trunk frontal plane.
    b. Compare the pre- and post-feedback Deviation Phase (DP) values between the feedback and non-feedback groups for the following segment pairs: foot-shank sagittal plane, foot-shank frontal plane, shank-thigh sagittal plane, shank-thigh frontal plane, thigh-trunk sagittal plane, thigh-trunk frontal plane.
    c. Compare the pre- and post-feedback peak knee extension moment, knee valgus moment, peak anterior tibial shear force, and peak vertical ground reaction force, during the first 40% of the stance phase between the feedback and non-feedback groups.

3. Do men and women exhibit different lower extremity coordination, variability, and kinetics pre- and post-fatigue during the stance phase of an unanticipated sidestep cutting task?
    a. Compare the pre-fatigue Mean Absolute Relative Phase (MARP) and Deviation Phase (DP) values between men and women for the following segment pairs: foot-shank sagittal plane, foot-shank frontal plane, shank-thigh sagittal plane, shank-thigh frontal plane, thigh-trunk sagittal plane, and thigh-trunk frontal plane.
    b. Compare the pre-fatigue peak knee extension moment, knee valgus moment, peak anterior tibial shear force, and peak vertical ground reaction force, during the first 40% of the stance phase between men and women.
    c. Compare the post-fatigue Mean Absolute Relative Phase (MARP) and Deviation Phase (DP) values between men and women for the following segment pairs: foot-shank sagittal plane, foot-shank frontal plane, shank-thigh sagittal plane, shank-thigh frontal plane, thigh-trunk sagittal plane, and thigh-trunk frontal plane.
    d. Compare the post-fatigue peak knee extension moment, knee valgus moment, peak anterior tibial shear force, and peak vertical ground reaction force, during the first 40% of the stance phase between men and women.

**Hypotheses**

1. A functional fatigue protocol will alter the coordination, variability, and kinetics of the lower extremity during the stance phase of a sidestep cutting task in the following ways:

   a. Mean Absolute Relative Phase (MARP) values will become more in-phase (decrease in value) in the sagittal plane, and more out-of-phase (increase in value) in the frontal plane, post-fatigue.
   b. Deviation Phase (DP) values will increase post-fatigue.
   c. Peak knee extension moment, knee valgus moment, peak anterior tibial shear force, and peak vertical ground reaction force during the first 40% of the stance will increase post-fatigue.
2. An acute intervention (verbal feedback) will affect the post-fatigue coordination, variability, and kinetics of the lower extremity during the stance phase of a sidestep cutting task in the following ways:
   a. Post-feedback Mean Absolute Relative Phase (MARP) values will return to pre-fatigue values in the feedback group, but will remain at post-fatigue values in the non-feedback group.
   b. Post-feedback Deviation Phase (DP) values will return to pre-fatigue values in the feedback group, but will remain at post-fatigue values in the non-feedback group.
   c. Post-feedback peak knee extension moment, knee valgus moment, peak anterior tibial shear force, and peak vertical ground reaction force during the first 40% of the stance phase will return to pre-fatigue values in the feedback group, but will remain at post-fatigue values in the non-feedback group.
3. Men and women will exhibit different lower extremity coordination, variability, and kinetics pre- and post-fatigue in the following ways:

a. Pre-fatigue Mean Absolute Relative Phase (MARP) values in women will be more in-phase (decrease in value) in the sagittal plane, and more out-of-phase (increase in value) in the frontal plane, when compared to men.
b. Pre-fatigue Deviation Phase (DP) values will be higher in women than in men.
c. Pre-fatigue peak knee extension moment, knee valgus moment, peak anterior tibial shear force, and peak vertical ground reaction force during the first 40% of the stance phase will be higher in women than in men.
d. Post-fatigue Mean Absolute Relative Phase (MARP) and Deviation Phase (DP) values will change more in women than in men.
e. Post-fatigue peak knee extension moment, knee valgus moment, peak anterior tibial shear force, and peak vertical ground reaction force during the first 40% of the stance phase will increase more in women than in men.

# CHAPTER 2

Injuries to the ACL are a costly and debilitating injury. The mechanisms and potential risk factors for this injury have been extensively studied, yet we still do not have enough understanding about these factors to effectively prevent injury. One of the primary limitations of most ACL-related research is the performance of movements in controlled, laboratory environments that may not mimic the competitive environments where injuries occur. There has been little research that examines how perturbations in the environment, such as muscular fatigue or inability to anticipate movement direction, may alter proposed risk factors for injury. Additionally, there has been no research that has examined the potential role for movement coordination and variability to explain the differences between genders, or how different external constraints will change movement patterns. The overarching purpose of this study was to examine these factors in a collegiate athletic population, in order to better understand how proposed risk factors may change due to fatigue or anticipation.

This review will focus on four primary areas. First, an introduction to the epidemiology of ACL injuries is given, leading to a discussion of the proposed kinetic risk factors for injury. Second, the impact of muscular fatigue on proposed risk factors is discussed. Third, the concept of movement coordination and variability is discussed, with particular emphasis on how alterations in these variables may relate to lower extremity injury. Finally, verbal feedback is discussed as a potential intervention to quickly and acutely change movement patterns and kinetics in order to prevent injury. The current concepts in each area are discussed, as well as the gaps in the literature that this research addresses. The final paragraph will summarize the information in this review, and serve as the rationale behind the methods described in chapter 3.

**Section One: Injury Epidemiology and Kinetic Risk Factors**

Injuries to the Anterior Cruciate Ligament (ACL) are a costly and debilitating injury to both competitive and recreational athletes. Approximately 112,500 ACL injuries occur every year in the United States, which places a substantial burden on the healthcare system. These athletes can expect to lose 6-9 months of competition, and many athletes decide to stop participation in their sport altogether. However, the greatest concern may be the long-term joint health for these individuals. Several studies have confirmed that joint injury, and specifically ACL injury, may be a leading cause of osteoarthritis (OA). These concerns have led researchers and clinicians to study the factors that may increase the risk of suffering ACL injury, as well as methods of preventing this injury in high-risk groups. This section will focus on the factors that are associated with ACL injury, specifically the kinetic variables that have been suggested to be the most critical in the development of non-contact ACL injuries.

***Epidemiology of ACL Injury***

Injuries to the ACL are one of the most common athletic injuries that result in surgical repair and significant time lost from competition. Recent epidemiological studies have shown that the rate of ACL injury in the United States is approximately 1 in 2500 individuals.[2] The rate of ACL injury in younger athletes is considerably higher; as high as 1 in 1100 in men and women age 15-24.[2] The majority of ACL reconstructions are performed in persons aged 15-25.[52] Typical recovery time from surgery is 6-9 months, dependent on the presence of other joint injury (meniscal damage, concomitant damage to other ligaments, etc) and the type of rehabilitation program employed.[53] While surgical procedures have improved significantly in the past 10 years, many athletes decide to cease participation in the sport where the injury occurred. Myklebust and Bahr report that a significant majority of athletes can return to sport within the first year of the injury (65-88%).[5] However, in their review of the literature, Myklebust and Bahr also found that athletes who have suffered an ACL injury tend to retire from sports participation, participate in lower-risk activities, or play at lower competitive levels that prior to the injury at a higher rate than non-injured athletes.[5] In a separate study, Roos found that 70% of ACL injured athletes had stopped participation in soccer within 3 years of the injury, compared to 20% of a control group. [4] While research is still largely retrospective, it does appear that ACL injury may lead to a significant decline in athletic participation.

Injuries to the ACL are commonly classified as non-contact or contact. Non-contact injuries are typically defined as "forces applied to the knee at the time of injury resulted from the athlete's own movements and did not involve contact with another athlete or object". Contact injuries occur when another athlete or object impacts the knee joint, causing injury.[3] Marshall also advocates for a third category for ACL injury: indirect contact. These injuries would be classified as forces applied to the knee at the time of injury that result from a perturbation caused by another athlete or object, but that does not directly impact the knee.[3] Approximately 70% of ACL injuries can be classified as non-contact in nature, making them far more prevalent than contact-related injuries.[54] This is of particular interest to clinicians, because non-contact injuries are theoretically 100% preventable. Thus, the majority of research in this field has focused on non-contact ACL injuries, and the factors that are associated with this injury.

Participation in certain sports or activities can increase the risk of suffering non-contact ACL injury. Recent studies published from the National Collegiate Athletic Association's (NCAA) Injury Surveillance System (ISS) have found that women's gymnastics, soccer, basketball, and lacrosse, and men's football, are the sports associated with the highest risk of ACL injury.[55-58] All of these sports involve quick changes of direction, cutting and/or pivoting movements, and landing from jumps, motions that have been associated with non-contact ACL injury in the literature.[54, 59-61] While the specific mechanism of injury for ACL injury is still not clearly understood, it is apparent that performing cutting, pivoting, or landing activities places an athlete at higher risk of suffering ACL injury.

Perhaps the factor that has received the greatest amount of attention from researchers and clinicians is gender. Numerous studies have demonstrated that females in certain sports are at higher risk of non-contact ACL injury than their male counterparts.[55, 56, 58, 62, 63] A systematic review published in 2007 found that females were at 1.5-4.3 times the risk of non-contact ACL injury than their male counterparts.[3] Typically, researchers have found that soccer and basketball demonstrate the greatest gender disparity in ACL injury rates.[3, 55, 63] These statistics have lead to a considerable body of literature comparing proposed risk factors between genders, which has formed the theoretical basis for many injury prevention programs.

While ACL injury creates considerable short-term disability, recent literature has examined the long-term sequelae of joint injury, particularly regarding the development of osteoarthritis (OA). Studies by several researchers have demonstrated that injury to the knee joint is one of the most significant predictors of knee OA. [6, 7, 9, 64] Specifically, the study by Thelin found that a history of knee injury explained why participation in sport has been linked to the development of knee OA. A significant relationship existed for sport participation (specifically, soccer and ice hockey) prior to any adjustment for other confounding variables, but when adjusted for history of knee injury the relationship disappeared. [9] The study by Gelber demonstrated prospectively that knee injury during adolescence and young adulthood substantially increased the risk of OA (relative risk = 5.17, 95% CI: 3.07, 8.71). [6] This trend towards an increased risk of knee OA after knee injury is also apparent when ACL injury is considered. Salmon found that 79% of patients had radiographic changes within 13 years of surgical reconstruction, and that 50% of patients with an isolated ACL injury (no meniscus damage) had signs of the development of OA.[8] These results are similar to other reports using different surgical techniques to repair the ACL.[65, 66] These studies all demonstrate that a history of knee injury, and specifically ACL injury, lead to a substantial increase in the risk of OA development. Thus, prevention of these injuries is paramount in the prevention of future knee joint pathology, particularly for young individuals.

***Suggested Risk Factors for Non-Contact ACL Injury***

In the past 20 years a number of researchers have attempted to determine what factors may lead a non-contact ACL injury. The majority of these studies have relied on the distinct gender disparity in ACL injury rates in certain sports, particularly soccer, basketball and volleyball. In general, these proposed risk factors have fallen into one of four categories: environmental, anatomical, hormonal, and biomechanical/neuromuscular.[21, 67, 68] More recently, prospective and computer modeling studies have been used to confirm some of these proposed risk factors, independent of gender. However, despite the substantial body of research that has been accumulated, the precise mechanism and most predictive risk factors for ACL injury remain elusive. Researchers are continuing to develop research methodologies and study designs that can more definitively study the factors that lead to ACL injury in athletes.

***Environmental Risk Factors***

The study of risk factors for ACL injury began with footwear design in the late 1960's and 1970's. The development of artificial playing surfaces and new cleat designs coincided with a marked increase in the number of non-contact injuries, particularly ACL injuries. Researchers began to study how the external environment, specifically the playing surface and the footwear design, influenced injury rates. Torg and Quendenfeld provided the first evidence that footwear may influence injury rates.[69, 70] In these studies, American football players who wore footwear with a lower release coefficient had substantially lower knee injury rates than players who wore traditional football cleats. This lower release coefficient essentially prevented excessive fixation of the foot with the ground, which is associated with increased torques and forces transmitted to the lower extremity of the athlete.[71-73] Higher ACL injury rates have also been documented on surfaces that increase the fixation of the foot, via higher coefficient of friction values. Studies by Olsen and Pope all demonstrate higher injury rates certain surfaces that have higher frictional characteristics.[74, 75] These studies suggest that the interaction of the shoe and the playing surface significantly affect non-contact ACL injury risk.

The influence of playing surface and other environmental factors (precipitation, ground hardness, temperature) has also received attention from researchers in the past. Orchard and colleagues in Australia have published several studies demonstrating higher injury rates in general, and ACL injury rates in particular, when athletes play on hard and dry surfaces.[76-78] Orchard has also found that knee injury rates in American football players decrease when the ambient temperature was colder.[78] The authors all conclude that the external playing environment can significantly alter the traction developed between the shoe and the playing surface, and that fields with lower traction development are beneficial for the prevention of injuries.

***Anatomic Risk Factors***

Anatomical features of the lower extremity are also believed to play a role in the development of non-contact ACL injuries. In one of the first prospective studies on ACL injuries, Uhorchak and colleagues found that a narrow intercondylar notch was predictive of ACL injury in a cohort of United States Military Academy cadets.[79] The authors hypothesized that the ACL would more easily impinge on the walls of a narrow intercondylar notch, making it more likely to rupture. Despite this early prospective result, later studies failed to confirm intercondylar notch width as a predictive factor for ACL injury, casting doubt on its role as a risk factor for all athletes.[80-82] More recent studies have examined how other anatomic features of the knee joint may influence injury risk. Several researchers have demonstrated a potential link between ACL size or volume and injury risk. Females generally have smaller ACLs, both in cross-sectional size and volume. This may reduce the overall mechanical properties of the ligament, making it easier to load to failure.[83, 84] Additional research has also found a potential link between tibial slope (anterior to posterior slope of the tibial plateau) and increased risk of ACL injury.[85, 86] Both of these factors require additional study and prospective confirmation, but show promise as possible risk factors for injury.

Ligamentous laxity and anatomic alignment of the lower extremity has been extensively studied as a potential risk factor for ACL injury. Uhorchak and colleagues found that generalized joint laxity was predictive of noncontact ACL injury in their prospective study of military cadets.[79] Laxity of the knee joint, specifically in the anterior direction, has also been suggested as a factor for ACL injury. Uhorchak and colleagues found that anterior knee laxity predicted ACL injury in his prospective study, while Woodford-Rogers retrospectively found that anterior knee laxity could predict status as ACL injured in high school athletes.[79, 87] However, both general ligament laxity and anterior knee laxity require substantial further study to confirm their role as risk factors for injury. Additionally, anatomic alignment variables such as genu recurvatum, genu valgum, pronation at the subtalar joint, excessive Q-angle, pelvic tilt, tibial torsion, and femoral anteversion have all been suggested as possible reasons for the female predisposition towards noncontact ACL injuries.[87-90] While some studies have shown a possible link between these alignment variables and injury risk, the nature of the relationship is still unclear. To this point no prospective studies have found a significant link between lower extremity alignment and ACL injury risk.

***Hormonal Risk Factors***

Hormonal factors are believed to play a role in the material properties of the ACL, perhaps leading to changes that would lower the threshold for injury. Several studies have found receptors for female sex hormones on the ACL, which led researchers to hypothesize a link between female hormones and ACL injury risk.[91-94] However, more recent case-control studies have not found a definitive link between a specific phase of the menstrual cycle (which is correlated to certain hormone concentrations) and increased likelihood of ACL injury.[74, 95-98] It does appear that fluctuations in the concentration of female hormones may play a role in the laxity of the knee joint, and that this relationship is highly variable between female subjects.[99-101] Researchers have also suggested that the changes in serum hormone concentrations may have a delayed action on ligamentous properties, which may help explain the equivocal results from prior studies.[102] Currently, while most researchers agree that hormones likely play a role in ACL injury in female athletes, the precise mechanism behind this effect is unclear.

***Biomechanical & Neuromuscular Risk Factors***

The influence of lower extremity biomechanics and neuromuscular control on noncontact ACL injury risk is one of the most widely studied phenomena in sports medicine. A substantial body of literature has been published, examining which movements, forces, moments, and neuromuscular functions are related to ACL injury. Most of these studies have used a comparison of male and female athletes in order to draw conclusions about noncontact ACL injury risk. In general, females have been found to display less knee and hip flexion, more knee valgus and transverse plane movement, greater knee extension moments, greater knee valgus (or adduction) moments, higher anterior tibial shear forces, greater quadriceps muscle activation (particularly when compared to hamstring activation) during landing or cutting maneuvers, and lower hamstring muscular strength compared to males.[17-27, 103-111] While there is a general consensus that these factors likely play a role in the development of ACL injury, it is unclear from these gender-comparison studies exactly how each biomechanical factor may influence injury risk.

Sagittal plane biomechanical and neuromuscular factors may provide the most direct loading on the ACL. A great deal of cadaveric research has confirmed that the most direct way of inducing strain on the ACL is by applying a linear force in the anterior direction, causing anterior translation of the tibia on the femur.[14, 15, 31, 75, 112] This force, often referred to as anterior tibial shear force (ATSF), is believed to be caused in vivo by a combination of external ground reaction forces as well as quadriceps muscle force, particularly when the knee is near full extension.[28, 31, 33, 113] When the knee is between 0-20° of flexion, the angle formed between the patellar tendon and the shaft of the tibia is at its highest value. This angle, called the patellar tendon-tibial shaft angle, is a critical determinant of the amount of quadriceps muscle force that is directed anteriorly.[33, 113] At high patellar tendon-tibial shaft angles, the force the quadriceps produces will create a significant anteriorly-directed force at the tibia which will draw the tibia forward relative to the distal femur. Recent studies have demonstrated that the quadriceps is capable of inducing significant ATSF upon the knee joint, and that aggressive contraction of the quadriceps is capable of rupturing the ACL in cadaver models.[31, 33] Sell and colleagues examined the impact of various biomechanical and neuromuscular factors on the development of ATSF, and determined that quadriceps EMG activity was a significant predictor of ATSF during a landing task,[32] These studies all support the theory that ATSF is strongly influenced by quadriceps muscle force, specifically when the knee is near full extension.

The development of ATSF at the knee joint is also strongly related to the application of external forces upon the joint, specifically ground reaction forces that are transmitted up the kinetic chain. In most biomechanical studies, the process of inverse dynamics is used to calculate the forces and moments experienced at joints. This process relies upon using the ground reaction forces in the 3 cardinal directions (vertical, anterior-posterior, and medial-lateral) to calculate the transfer of moment and force to the most proximal joint to the floor.[114] These moments are then used to calculate the moments and forces in the next proximal joint, continuing in this fashion up the kinetic chain. Thus, the application of ground reaction forces will have a strong influence upon the kinetics of the joints in the lower extremity. Recent studies by Sell and colleagues, as well as Yu and colleagues, have demonstrated strong correlations between posterior ground reaction force and ATSF.[28, 32] The study by Yu demonstrated a very high correlation (r=0.85) between peak posterior ground reaction force and ATSF, and a smaller but significant correlation (r=0.51) between peak vertical ground reaction force and ATSF during jump landing maneuvers.[28] Sell examined several kinematic and kinetic variables as predictors of ATSF, and found that peak posterior ground reaction force was a significant predictor of ATSF and had a significant correlation to ATSF (r=-0.276). These studies highlight the importance of externally applied ground reaction forces when studying the kinetics of the lower extremity during athletic movements.

While ground reaction forces have a strong relationship to ATSF on their own, they may exert greater influence on the development of moment at the knee joint, which has the greatest influence on ATSF found in the literature. The study by Yu reported that both peak posterior ground reaction force (r=0.86) and peak vertical ground reaction force (r=0.57) had significant correlations to peak knee extension moment, which represents the internal moment requirement at the knee joint in order to maintain equilibrium.[28] While both posterior and vertical ground reaction forces also correlated with ATSF, the highest correlation in this study was seen between peak knee extension moment and ATSF (r=0.90).[28] Additionally, both peak knee extension moment and peak ATSF occurred simultaneously during the landing phase of the jump-landing task.[28] Sell found that the knee flexion moment (measured at the time of peak posterior ground reaction force) was the strongest predictor of ATSF and had the highest pairwise correlation (r=-0.8986) of any variable tested.[32] The use of knee flexion moment reflects an external moment applied to the knee joint. Researchers often report either external or internal moments, as both represent the demands placed upon a joint (internal moments represent the needed soft tissue requirements in order to respond to external moments).[114] Thus, while the type of moment represented in these two studies is different, the conclusion is the same. The sagittal plane moment at the knee joint is strongly related to the development of ATSF, and it appears that this moment is the result of posterior ground reaction force and quadriceps muscle force acting at the tibiofemoral joint.

While ATSF may represent the most direct method of inducing strain upon the ACL, several studies hypothesize that the ATSF generated in vivo during athletic movements is insufficient to cause injury to the ACL. McLean and colleagues conducted a computer simulation using forward dynamics, and found that the ATSF generated during the cutting task was insufficient to rupture the ACL. The values elicited during computer modeling were primarily negative (indicating a posterior drawer-type force), and the maximum values obtained were less than 900N.[115] Simonsen and colleagues, using a simulated side-step cut, reported an average of 520N of force transmitted to the ACL.[116] Their results were generally higher in magnitude than the study by McLean. Another computer simulation by Pflum, using a drop-landing, demonstrated higher anterior shear forces transmitted to the ACL (approximately 0.4 multiples of body weight), but still below the theoretical injury threshold of 2000N.[117] These studies suggest that it would be difficult to generate ACL injury via

ATSF alone. Considerable debate exists regarding this conclusion. Several studies have demonstrated differences in ATSF between genders, particularly during tasks that generate a high level of posterior ground reaction force, such as a jump-landing.[18, 28, 42, 118] These researchers believe that this gender disparity clearly highlights the importance of ATSF as a risk factor for ACL injury. However, the highest amount of ATSF measured in these inverse dynamics studies was 0.79 multiples of body weight, which is still below the theoretical ACL injury threshold of 2000N.[117] Thus, most researchers have examined how other motions in the frontal and/or transverse plane, may influence ACL strain and potentially lead to ACL injury.

Cadaver studies provided the first evidence that combined, multi-planar loading led to higher ACL strain than any single direction alone. Combining ATSF with valgus or varus moment dramatically increased ACL strain in several cadaver studies.[14, 15, 119] Researchers also demonstrated that a combination of ATSF and internal rotation moment elevated ACL strain.[14, 15, 120] Using computer modeling, Chaudhari and Andriacchi demonstrated that the threshold for injury to the ACL decreases as the knee moves into either valgus or varus, indicating that lower levels of ATSF would be needed to cause injury to the ACL.[16] The results of these studies demonstrated that forces and moments applied in the frontal and transverse planes may play a critical role in the development of ACL injury. Between-gender research, using females to represent a high-risk group of subjects, concluded that females display greater knee valgus moments during athletic movements than males.[17, 18, 21, 24, 26, 27, 41, 105, 108] These researchers believe that this gender difference in frontal plane kinetics can largely explain why females are more likely to injure their ACL than their male counterparts.

However, the extent to which valgus moment influences ACL injury risk is still unclear from these cross-sectional studies.

Perhaps the best evidence that frontal plane moments are associated with increased risk of ACL injury comes from a prospective study by Hewett and colleagues. This study prospectively collected biomechanical data from 205 female basketball, soccer, and volleyball athletes. These athletes were then followed by study personnel over the course of two competitive seasons. Females that suffered a non-contact ACL injury were then compared to the remaining healthy athletes to determine if any biomechanical measures were different between groups. Females that injured their ACL had greater knee valgus angles at both contact with the ground (8.4° more than uninjured) and maximum value (7.6° more than uninjured). They also demonstrated less maximum knee flexion (10.5° less) than uninjured athletes. However, the greatest difference was detected in knee abduction moment. Injured females had 2.5 times the knee abduction moment than their uninjured counterparts. When a logistic regression analysis was performed, knee abduction moment was a significant predictor of ACL injury status, and demonstrated a sensitivity of 78% and a specificity of 73%.[34] The findings from this prospective study provide the strongest evidence to date that knee valgus (or abduction) moment is significantly related to ACL injury, and may be a critical risk factor for female athletes.

While the influence of moments at the knee has received considerable attention from researchers, the relative contribution of other joints and body segments, particularly the ankle, hip and trunk, remains unclear. Between-gender studies have found some differences in hip joint motion during athletic tasks between men and women, generally reporting less hip flexion and more frontal plane movement (typically an increase in hip adduction, or less hip abduction) in females.[28, 108, 121-123] However, other studies have shown no difference in hip joint kinematics or kinetics.[19, 24] While evidence is building that the hip may play a role in ACL injury, the precise mechanism is unclear. Several researchers believe that a stiff landing style, which is associated with both decreased knee and hip flexion angles, is responsible for generating large joint resultant forces that may be responsible for ACL injury.[28, 108, 122, 123] Associations between frontal plane hip movement, specifically increased hip adduction angle, and knee valgus angle have been found in previous literature.[34, 121] Thus, hip motion may influence both knee valgus angle and moment, both of which are associated with increased risk of injury. Research is on-going on how hip motion may influence the risk of injury to the ACL.

Less research is available about how motion at the ankle may influence knee joint loading. Between-gender research has demonstrated that ankle excursions are greater in females than males, in both the frontal and sagittal plane.[19, 20, 121] Decker demonstrated that females have a greater reliance on an "ankle" energy absorption strategy, utilizing ankle motion and musculature to a greater degree than males.[19] This may not be the most advantageous strategy to utilize, as the ankle musculature has limited ability to absorb energy when compared to the larger musculature of the knee and hip joints.[19] While little research has been performed on how ankle and foot mechanics relate to ACL injury risk, it does appear to influence the development of motion and forces at the knee.

The trunk has received recent attention as another body segment that may have an influence on knee joint motion and moment. Several studies have demonstrated that movement at the trunk in all three planes influences the forces and moments at the knee joint. Dempsey and colleagues have examined how trunk movement influences knee moments.

Knee valgus moment increases significantly when the trunk leans to the opposite side of a sidestep cut. In addition, rotating the trunk during the cutting maneuver increases the internal rotation moment at the knee.[124] A study by Gupta also demonstrated an correlation between trunk obliquity and knee valgus moment, where increased frontal plane trunk movement was associated with higher knee valgus moment.[125] Thus, isolated changes in trunk movement can have a significant impact on moments at the knee that are associated with increased risk of ACL injury. Blackburn and Padua studied how isolated trunk flexion impacts knee kinematics, and found that increasing trunk flexion during a landing task creates a greater peak knee flexion angle when compared to a normal, preferred landing style.[126] While only a few studies exist that examine how trunk movement influence knee joint loading, these studies demonstrate that a link does exist between trunk position and lower extremity forces and moments.

Two recent studies published by Zazulak and colleagues concluded that poor neuromuscular control over the trunk is associated with knee injury. In one study, proprioception was measured prospectively via active and passive trunk repositioning in the transverse plane. Two hundred seventy-seven athletes were tested, and 25 suffered knee injury during the 3 year follow-up. ANOVA testing revealed that female athletes who suffered a knee injury had significantly higher active repositioning errors (indicating worse proprioception) than females who were uninjured.[30] The odds ratio for knee injury increased 2.9 times for every degree of repositioning error; the odds ratio for ligamentous or meniscal injury increased 3.3 times for every degree of repositioning error.[30] In a separate publication, Zazulak and colleagues reported that a lack of neuromuscular control over the trunk was associated with increased risk of knee injury. Neuromuscular control was measured by analyzing trunk angular displacement after a sudden force release into flexion, extension, and lateral bending. Logistic regression revealed that increased displacement was predictive of all knee injuries, as well as ligamentous or meniscal injuries, and ACL injuries specifically.[29] Lateral displacement was the strongest predictor of injury in athletes, with odds ratios of 1.9 (all knee injuries), 2.0 (ligamentous/meniscal injuries), and 2.2 (ACL injuries). These two studies suggest that the ability to control the movement of the trunk, particularly in the lateral direction, may be critical in the prevention of knee injuries. While this study was performed under relatively artificial conditions (highly controlled laboratory testing of trunk proprioception and control), the nature of the results do suggest that researchers should incorporate an analysis of trunk movement into injury risk factor studies.

The research on the biomechanical and neuromuscular risk factors for ACL injury has been substantial, and has led to several important conclusions. First, between-gender studies have established a group of risk factors that most researchers believe contribute to the gender disparity in ACL injury rates. Of these factors, excessive knee valgus moment at the knee has been demonstrated to be greater in female in athletes in most studies, and was the strongest predictor of ACL injury in the prospective study by Hewett.[34] Gender studies in combination with cadaver and computer modeling studies also suggest that knee extension moment and ATSF are important, as they apply strain to the ACL. When combined with knee valgus moment, these two kinetic variables likely play an important role in the development of ACL injury. While specific kinematic variables have also been associated with ACL injury risk (small knee flexion angle, high knee valgus angle), the evidence supporting the kinetic variables at the knee has been more consistent. Additionally, many researchers advocate further investigation of the other segments and joints of the lower extremity and trunk, as the research in these areas is still progressing. Thus, future research that adds to the knowledge base on the kinetics of the lower extremity during athletic tasks, as well as how movement of other body segments influences the potential for ACL injury, is warranted.

This research project was designed to address these key kinetic variables that have been suggested to directly produce strain on the ACL, thus leading to injury. This project was designed to investigate how these variables respond to different external conditions which likely occur during game-play (fatigue and unanticipated movements). The sidestep cutting task is commonly performed in athletics and is also associated with a high risk of injury to the ACL, which makes it well-suited to study how different perturbations affect joint kinetics during athletic movements associated with ACL injury. The results of this study provide needed insight on how these selected kinetic variables may change, and potentially increase strain on the ACL, during an athletic task under various external constraints designed to mimic actual game-play.

**Section Two: Muscular Fatigue**

Muscular fatigue is a common occurrence during athletic activities. Sports that involve a high risk of injury to the ACL (soccer, basketball, volleyball) regularly perform high-intensity exercise over a period of several hours, which may produce substantial fatigue. However, the mechanisms behind the development of fatigue during athletic activities are poorly understood. This has broad implications, as many researchers believe that fatigue may be a risk factor for lower extremity injury. Many different types of “fatigue protocols” have been used to study how fatigue alters movement, but whether those protocols effectively reproduce the type of fatigue that occurs during athletics is unclear. Thus, this research project aims to answer some of these questions, and provide a more clearly researched fatigue protocol to elicit fatigue.

***Muscular Fatigue***

Fatigue is a general term used to describe the experience of muscular weakness, loss of strength, or psychological exhaustion. Because this term can be interpreted in several ways, many researchers prefer that the term "fatigue" be more precisely defined. Experimentally, "muscular fatigue" refers to the loss of maximum force applied by a muscle.[127] Researchers also prefer that the perceived endpoint of performance, or the point of maximal exertion during physical activity, be referred to as "exhaustion" and not fatigue.[127] The term "muscular fatigue" does not apply to the loss of muscle force capability or perceived weakness due to disease or another medical condition. Thus, the definition of "muscular fatigue" should refer to a loss of peak force production by a muscle as a result of physical exertion.[128]

One important consideration when studying muscular fatigue is the location of the impairment of muscular force production. Several aspects of the neuromuscular system can be impaired during a fatiguing bout of exercise, including activation of the motor cortex, central nervous system drive to motor neurons, activation of particular motor units, propagation of the neural signal across the motor unit, excitation-contraction coupling, availability of substrates needed for metabolism, intracellular environmental factors, the contractile apparatus, and blood flow to muscular tissue.[127] Generally speaking, the above factors are grouped into two categories: Central fatigue and peripheral fatigue. Central fatigue includes any impairment in the activation of the motor unit from the central nervous system (a loss of voluntary activation), while peripheral fatigue incorporates any impairment in muscular function distal to the neuromuscular junction.[128] Different types of activities will produce different amounts of both central and peripheral fatigue. Understanding which "location" is most affected by a given exercise or type of physical activity can help researchers understand not only the physiological processes that may be involved in the development of muscular fatigue, but also how to target interventions to reduce fatigue-related impairments in the neuromuscular system.

Several authors have suggested that central fatigue is unlikely to occur during most types of athletic activity.[129-131] Kirkendall hypothesized that under normal conditions, when a subject is sufficiently motivated, central fatigue is unlikely to occur.[130] Several more recent studies have found that fatigue-related loss of muscular strength could be largely explained by peripheral changes in the muscle cell, specifically in the excitation-contraction coupling.[129, 131] However, the Lattier study examined uphill running on a treadmill, which eliminates the eccentric contribution of the leg musculature during activity. While this prevents muscular damage from contaminating the results, it also fails to simulate athletic activity which will always employ some eccentric muscle activity. The Skof study used a run at a set distance and intensity (6k at anaerobic threshold) in highly trained athletes. This may have prevented the athlete from reaching a true "exhaustion", which may have prevented the authors from seeing central contributions to the muscular fatigue observed from this protocol. Finally, both of these studies utilized continuous activities, which do not reflect the intermittent activity that is seen in many athletic events (i.e. soccer, basketball, volleyball).[132, 133] Thus, while peripheral fatigue likely occurs during athletics, central contributions to overall muscular fatigue are also likely to limit performance.

Evidence of central fatigue during full-body functional fatigue protocols has been found by several authors. A recent study by Theurel compared variable intensity exercise to a continuous bout of exercise.[134] The authors found that exercising at a variable intensity induces significantly greater central fatigue, due to a substantial decrease in voluntary activation level post-fatigue, when compared to continuous cycling. This result may help explain why some prior research has failed to find significant central fatigue during continuous running. Additionally, this study found that perceived exertion, blood lactate, and heart rate, were all higher in the variable exercise protocol, despite controlling for the total average power output. Thus, this fatigue protocol appears to produce greater physiological responses, as well as a higher reported experience of fatigue. This study highlights not only the presence of central fatigue during intermittent exercise, but that physiological and perceptual changes are also different following this type of exercise.

The physiological changes that occur during fatiguing bouts of exercise may play a crucial role in the development of central fatigue. Thomas and colleagues [135] found that a progressive exhaustive cycling protocol produced evidence of peripheral fatigue, demonstrated by a loss of peak torque during an evoked muscle twitch without a loss of voluntary peak torque. The authors concluded that this loss of twitch torque was the result of impairment in the excitation-contraction apparatus, which agrees with prior literature that found peripheral fatigue after exercise.[129, 131, 135] However, Thomas also found a decrease in oxygenation in the prefrontal cortex, an indicator that central fatigue did occur during the exercise protocol. The authors hypothesized that peripheral fatigue may influence the motor drive to nerves, creating central fatigue.[135]

The prospect that peripheral fatigue may influence the development of central fatigue has been suggested by other researchers as well. Amann [136-138] and his colleagues have published several studies investigating how respiratory factors (oxygenation, fatigue of respiratory muscles) influence the development of fatigue during cycling. Amann concludes that feedback to the central nervous system from muscles experiencing peripheral fatigue is the major determinant of central motor drive. Both Amann and Dempsey [139, 140] suggest that the body tightly regulates the amount of peripheral fatigue that can occur during exercise by changing central drive in response to sensory feedback from muscles. When peripheral fatigue reaches a critical threshold, then the body responds by decreasing central motor drive to prevent muscles from continuing to full exhaustion. Thus, exhaustive exercise likely results in both peripheral and central fatigue, due to the interaction of these two mechanisms.

An additional way that athletic activities likely produce both peripheral and central fatigue is via modulation of spinal reflexes. In the Theurel study, the authors postulate that some of the central fatigue was due to inhibition of alpha motor neurons.[134] This hypothesis is in accordance with conclusions drawn from fatigue studies using stretch-shortening cycle (SSC) exercise. SSC exercise involves the use of eccentric muscle contractions to augment the development of force during concentric contraction.[141] Many researchers believe that most human activities involve SSC muscular activity, and these researchers advocate studying this type of contraction to simulate actual exercise. Nicol and colleagues discuss the possible mechanisms for the fatigue-related changes in SSC contractions in their review. One of the most intriguing hypotheses was that small-diameter muscle afferents would inhibit reflex activity of alpha motor neurons. The authors discuss several studies that support this hypothesis, and suggest that some of the metabolic accumulation within the fatiguing muscle may be responsible for this mechanism.[141] Again, these discussions suggest that peripheral factors may play a role in the central fatigue developed during exercise, and support the notion that both central and peripheral fatigue occur during normal athletic activities.

Since the current research supports the hypothesis that central fatigue does occur during exercise, specifically exercise that simulates intermittent athletic activity, some important questions have been raised about how fatigue affects other tasks performed by the central nervous system. Of these, the possible role of fatiguing exercise on the ability to perform cognitively demanding tasks has received the most attention. Several studies have been performed that examine how fatiguing exercise influences reaction time, cognitive function, attentional focus, and other features of cognition. Tomporowski [142] performed an extensive review on how acute bouts of exercise may influence these factors, and concluded that there is no consensus on how exhaustive, high-intensity anaerobic-type exercise affects cognition. In this review, the author suggests that the studies that did show decreased cognitive functioning only found small, transitory effects. The only type of physical fatigue that produced consistent degradation in cognitive function were long, continuous bouts of physical activity that lead to dehydration or energy depletion (typically 2 hours or longer).[142] This review suggests that further research that specifically defines "fatigue" (a definition that is often missing in these studies) and uses measures that are sensitive to smaller changes in function, may produce more consistent results.

Several conclusions can be drawn from the multiple studies on central and peripheral contributions to muscular fatigue. First, it does appear that exhaustive physical activity produces both peripheral and central fatigue. Exercise that simulates the intermittent activity of several sports also appears to produce significant central fatigue. However, the interaction of these two types of fatigue, and whether the central fatigue is due to spinal or supraspinal changes, remains unknown. Second, exhaustive high-intensity fatigue does not produce consistent changes in cognitive function, despite the evidence that central fatigue exists. Methodological constraints may play a large role in these findings, but there is currently no evidence that cognitive function is significantly altered after this type of fatigue. Thus, it appears that the central mechanisms that are affected by fatigue may be independent of cognitive function, so long as energy supply is not adversely affected. However, this area remains an intriguing area for further research, particularly in regard to decision-making during athletic activities (such as deciding a movement direction), which is a necessary cognitive skill for athletes.

***Fatigue as a risk factor***

Anecdotally, many clinical sports medicine staff will report a higher incidence of injury when a player is experiencing muscular fatigue. However, there is still no good scientific evidence to support this point. Most researchers will use recent epidemiologic studies to support the hypothesis that player fatigue plays a role in injury incidence. Gabbett, using injury surveillance in South African Rugby matches and practices, found that injuries are more likely to occur during the second half of matches, or in the latter states of practices.[10, 11] Hawkins and colleagues reported that noncontact knee injuries occur most often during the final 15 minutes of the first half, and final 30 min of the second half, of soccer matches.[12, 13] While player fatigue may play a role in these findings, there are several confounding variables that were not controlled. Playing intensity and player aggression may increase in the latter stages of close matches or games. Environmental factors (heat, rain, field condition) that deteriorate during the course of games may also lead to this increased incidence of injury. Psychological factors may also play a significant role in injury occurrence. Additionally, fatigue was not defined or measured in any of these studies, so it is unknown how fatigued these players were, or if the injured players had greater fatigue than non-injured players. Thus, while muscular fatigue may be an important risk factor for knee injuries, these initial studies can make only broad suggestions about this point.

***Quantifying Muscle Fatigue during Athletic Activity***

More recent studies have attempted to quantify the level of muscular fatigue that may occur during athletic activity. Two studies [132, 133] have used treadmill protocols that are based on the relative "average" workload and activity level of soccer field players during a game, while one study simulated soccer activity in a gymnasium.[143] These protocols were both based on prior research that had quantified the type and intensity of activity performed by soccer players during a match.[144, 145] Rahnama [133] used 13 male soccer players from the university setting to perform a 90-minute fatigue protocol with a "halftime" given to more accurately simulate an actual game. An isokinetic dynamometer was used to quantify concentric and eccentric hamstring and quadriceps peak torque pre-fatigue, at halftime, and at the conclusion of the fatigue protocol. The authors found that, at all testing speeds, both eccentric and concentric hamstring and quadriceps peak torque declined between the pre-test and the post-test at the end of the full protocol. Most of the tests also declined between the pre-test and halftime, and between halftime and the end of the protocol. Concentric quadriceps torque dropped between 8.5-15.5% after the full fatigue protocol, while hamstrings torque dropped 15-17%. Eccentric torque in both the hamstrings and quadriceps fell by about 10%. Additionally, the hamstring:quadriceps ratio was calculated, and found to significantly decline pre-test to the end of the protocol, indicating that the hamstrings fatigued more than the quadriceps.[133] The authors concluded that both the quadriceps and hamstrings exhibited significant fatigue after a simulated soccer game.

Greig [132] also used a soccer-game simulation on a treadmill to study the effects of fatigue on muscular strength in ten male professional soccer players. Subjects performed 6-15 minute periods of activity (with a 15 minute halftime), with muscle strength tested after each 15 minute session. Isokinetic strength was tested concentrically for the knee extensors, and concentrically and eccentrically for the knee flexors at three testing speeds. The authors reported that concentric quadriceps and hamstrings strength were maintained during the simulated game, whereas eccentric hamstrings torque decreased significantly at 180 and 300 °/s during the fatigue protocol. Peak torque decreased 18.8% from pre-test to the end of the protocol at 180 °/s, and 24% at 300 °/s. Dynamic hamstring:quadriceps strength ratio (eccentric Hamstrings: concentric Quadriceps) also tended to decrease as the fatigue protocol continued.[132] The authors concluded that eccentric hamstring strength was particularly affected by a soccer simulation fatigue protocol, which may present a risk of injury to the ACL.

In a unique study that used several different types of fatigue protocols, Gleeson compared the effects of intermittent running simulating soccer activity, shuttle runs, or continuous treadmill runs on isokinetic concentric knee flexor and extensor torque. All protocols were performed over the same distance. Gleeson found that knee extensor and flexor torque dropped by 20.3% and 18.1% respectively following the full protocol. The shuttle run resulted in declines of 11.9% and 18.0%, while the continuous treadmill run only resulted in 4% and 5% decreases in torque.[143] This study agrees with the Rahnama study, and the relative decreases in torque were approximately equivalent. These three studies together suggest that most intermittent, athletic-type fatigue protocols that simulate actual game play intensity result in significant muscular fatigue.

***Impact of Fatigue on Knee Laxity***

Since authors have reported increased risk of injury, and specifically knee ligament injury, during the latter stage of games, several researchers have attempted to study how fatigue may impact factors that are hypothesized to increase the risk of injury to the ACL. A prospective study by Uhorchak demonstrated that increased knee laxity, particularly in the anterior direction, is a risk factor for subsequent ACL injury.[79] Thus, several studies have studied how muscular fatigue affects knee joint laxity. Both Wojtys and Rozzi [146, 147] used isokinetic protocols to induce quadriceps and hamstring muscular fatigue, performed until subjects were unable to sustain torque output of 50% (Wojtys) and 25% (Rozzi) of the peak pre-testing torque achieved. In the Wojtys study, anterior knee laxity increased significantly post-fatigue, an average of 32.5%. However, Rozzi failed to find any significant increase in anterior knee joint laxity, which may be due to the difference in cutoff torque output used to define fatigue.

More functional, full-body fatigue protocols have also demonstrated significant changes in knee laxity. Skinner [148] had highly trained subjects perform repeated distance sprint protocols, which resulted in an 8-10% increase in anterior knee laxity. Gleeson used several different types of full-body fatigue protocols: a soccer-simulation similar to the ones reported by Greig and Rahnama, a shuttle run, and a continuous treadmill run (with total distance controlled across protocols). Anterior knee displacement increased 24% after the treadmill run, but 44% in the shuttle run and 48% in the soccer simulation run.[143] Stoller, after 3.5 mile run, found that rotational knee laxity increased 14%.[149] Steiner [150] demonstrated increased anterior knee laxity following participation in sports practices, while Weisman [151] found increased medial knee laxity following sports practices. These studies suggest that activities that more accurately simulate athletic activity result in substantial knee laxity, which may place the ACL at greater risk of injury.

***Impact of Fatigue on Lower Extremity Biomechanics***

While changes in muscular strength and knee laxity have been relatively consistent following functional fatigue protocols, the impact of muscular fatigue on lower extremity biomechanics has been more mixed. One of the primary reasons for these discrepancies is the different "functional" fatigue protocols used by each author. These authors have used repetitive closed kinetic chain exercises, such as squatting [44], squatting and jumping [41, 152-154], step-ups [155] or step-ups and bounding [43]; running protocols such as modified treadmill tests similar to the Astrand protocol [45, 156, 157]; repetitive jumping and sprinting exercises [42, 158]; or intermittent shuttle runs [159], in order to produce this functional, full-body fatigue. However, prior research has demonstrated that different types of fatigue protocols, even when controlled for distance, produce different changes in muscular strength.[143] Thus, drawing conclusions across studies is difficult at best. Additionally, these studies examine biomechanical changes in different tasks. Some examine fatigue-related changes in sidestep cutting [41, 159], others study double-leg landing [42, 43, 156] or single-leg landing [44, 45, 152-155, 158], while Nyland used a run and rapid stop.[157] However, despite all of these differences, some basic conclusions may be drawn from the results of these studies, and may help guide future research in the area of muscular fatigue.

*Repetitive CKC Exercises*

Coventry and colleagues [152] studied the effects of repetitive single-leg squatting on landing biomechanics. Eight male subjects performed a single-leg drop-landing with immediate countermovement jump (CMJ) from 80% of their maximal vertical jump height. After pre-testing was complete, subjects performed 5 single-leg squats, followed by 2 drop-landings and CMJ's. This cycle was repeated until the subject reported they could no longer perform the landings. The landing mechanics during the final 2 landings completed were used for post-test analysis. Muscular fatigue resulted in greater knee and hip angles at initial contact, and greater peak hip flexion, compared to pre-fatigue landings. The ankle exhibited less ROM during the post-fatigue landings. However, there were no changes in extension moments at any joint. The authors concluded that this fatigue protocol caused an adaptation by the subject to land in a more flexed position, and rely on greater contributions from proximal joints during landing. This would allow for greater absorption of forces, particularly if the muscular system was compromised.

Orishimo et al. [155] used a repetitive stepping protocol to induce fatigue in thirteen male subjects. Single-leg hops were performed pre- and post-fatigue from a distance equal to 80% of the maximum single-leg hop value. Fatigue was induced by performing multiple sets of 50 step-ups and downs, where the dominant leg (used for hop test) performed both positive and negative work. Fifty repetitions were done per set, with the sets repeated until the subject was unable to achieve 80% of their maximum hop distance. The authors reported that knee flexion ROM and peak knee flexion angle during the stance phase of the landing increased significantly, as did peak ankle plantarflexion moment. No other sagittal plane kinematics or kinetics changed as a result of fatigue. The authors concluded that the increased knee flexion was an attempt to change joint motions and increase the ability of the lower extremity to absorb forces, to compensate for a loss of muscle force.

Magidan and colleagues [154] used single-leg squats and landings to induce fatigue in twelve male subjects. Subjects performed a single-leg landing onto a forceplate from 25cm, then performed a series of 3 single-leg squats and 2 landings until they reported they would collapse if they attempted to land again. Hip, knee, and ankle joint kinematics and forceplate data were calculated during each landing and compared to pre-fatigue values. The authors found that peak knee and ankle sagittal plane flexion angles increased between the final set of landings and the pre-fatigue landings, while peak VGRF decreased. The authors concluded that the subjects used increased joint excursions to compensate for a loss of muscular force production to attenuate ground forces during landings.

McLean and colleagues [43] examined how repetitive athletic-type activities may influence specific kinematics and kinetics in both males and females. Ten male and ten female NCAA Division I athletes performed 20 step-up and step-down movements as quickly as possible, followed by bounding strides (covering 12m) where they were instructed to move into a deep-knee flexion position upon landing. This protocol was repeated as many times as possible in 4 minutes. Ankle, knee and hip kinematics were calculated during a drop vertical jump, pre- and post-fatigue, at initial contact with the forceplate and at their peak value during their landing on the forceplate. Peak ankle, knee, and hip joint moments were also calculated during the landing. The authors found that peak knee abduction (valgus) and knee internal rotation angles were higher post-fatigue in both genders. Peak knee internal rotation moment was also higher post-fatigue. Peak knee abduction moment increased more post-fatigue in females than in males. The authors concluded that muscular fatigue induced changes in frontal and transverse plane knee biomechanics, and that knee abduction moment was most adversely affected in female athletes. These findings suggest that fatigue may increase ACL loading, as these joint kinematics and kinetics have been associated with increased ACL strain [15] and prospectively have been linked to noncontact ACL injury.[34]

Kernozek and colleagues [44] examined how fatigue, induced by repetitive squatting, may influence lower extremity biomechanics in males and females. Fourteen female and 16 male recreational athletes performed a single-leg drop-landing from a 50cm height pre- and post-fatigue. Muscular fatigue was induced by repetitive squats to failure, using a load equal to 60% of the subject's one repetition maximum (1RM). A minimum of 4 sets to failure were completed by each subject. Maximum and minimum joint angles and moments were calculated between initial contact with the ground and maximum knee flexion. This fatigue protocol resulted in increased peak hip flexion and ankle dorsiflexion in both genders. Males exhibited greater knee flexion post-fatigue, whereas females did not change. With regard to kinetics, both genders displayed decreased peak hip extensor moment, peak knee extensor moment, and peak knee abduction moment. Both males and females also displayed decreased peak shear force at the knee, but females did not decrease as much as males. The authors concluded that, while the fatigue protocol appeared to produce beneficial changes in landing mechanics, males responded in a "safer" manner than females by increasing knee flexion angle and decreasing shear forces at the knee. Thus, the authors conclude that females respond less favorably post-fatigue than males. However, the authors did not report if there was any change in muscular strength and performance (for instance, decreased vertical jump) during the post-fatigue trials. Thus, it is difficult to verify the level of fatigue experienced by these subjects.

Borotikar et al.[41] recently studied how repetitive double-leg squats, as well as double- and single-leg landings, influence performance on cutting maneuvers in female NCAA athletes. Twenty-four subjects performed both anticipated and unanticipated single-leg cutting maneuvers prior to the fatigue protocol, and then at set intervals during the fatigue protocol. Unanticipated cutting was cued by a light system that indicated both the direction to move, and the foot to plant on the forceplate. The fatigue protocol consisted of five double-leg squats at a set cadence, followed by a random type (anticipated vs. unanticipated) and direction of sidestep cut. This was repeated until the subjects could no longer complete three squats. Ankle, knee, and hip kinematics were calculated at initial contact during the cut, as well as the peak value during foot contact on the ground. The authors found that muscular fatigue produced decreased hip flexion and increased hip internal rotation angles at initial contact, and that these changes were exacerbated in the unanticipated conditions. Peak knee abduction and internal rotation angles, as well as peak ankle supination angle, also increased post-fatigue. The changes in peak knee abduction were also enhanced during unanticipated conditions. The authors concluded that the combination of muscular fatigue and decision-making produced significant challenges to the neuromuscular system, causing changes that have been associated with increased ACL strain[15] and noncontact ACL injury.[34]

The six studies outlined above display some disparate results. Both McLean and Borotikar found significant changes in frontal and transverse plane biomechanics, where both joint angles and moments increased in potentially harmful ways.[41,43] However, the changes displayed by the subjects in the Kernozek study were primarily in the sagittal plane, and could be considered protective.[44] In the Madigan, Orishimo, and Coventry studies, subjects seemed to compensate for the induced muscular fatigue by increasing joint ROM to absorb forces.[152, 154, 155] Kernozek and his co-authors also argued that these changes may be a conscious decision by the body to protect the joints from injury when the muscular system is compromised, and that females were less able to produce these changes may be why they are more prone to ACL injury. The unanticipated cutting in the Borotikar study may have compromised the ability of the central nervous system to "protect" the joints during fatigue, allowing more detrimental effects to be observed. Thus, these studies appear to support the conclusion that muscular fatigue results in movement compensations designed to increase joint absorption following fatigue, but that increasing cognitive demands may prevent subjects from exhibiting these changes and place the lower extremity at risk of injury.

The primary limitation of these studies is that the CKC activities may not induce a similar state of fatigue as normal athletic participation. None of these studies attempted to validate the type of muscular fatigue produced. Additionally, only one study (Borotikar) used an unanticipated task, which many believe will better simulate game-related decision making. While the results of the Borotikar study were unique, the authors only presented the kinematic changes, with no information on coordination or kinetics. The results of these studies help provide some rationale for this proposed study, and also demonstrate where the current gaps in the literature are in this area.

*Repetitive Jumping and/or Sprinting*

Willson et al. [153] studied the effects of fatigue on lower extremity mechanics in females with or without patellofemoral pain syndrome (PFPS). Twenty females with PFPS and 20 without knee pain performed single-leg hops. Isometric trunk lateral flexion, hip abduction, and hip external rotation strength was assessed pre- and post-fatigue, as were three-dimensional hip and knee kinematics and impulses. Joint angles were calculated at peak knee extension moment. Fatigue was induced via a series of 10 single-leg squats and 5 single-leg jumps. When subjects reported a RPE of 17 or greater, the protocol was terminated and post-testing began. The authors reported that all subjects exhibited decreased hip and knee flexion angles, as well as decreased hip internal rotation, following the fatigue protocol. They conclude that, despite the PFPS group having less hip strength than the healthy group, biomechanical changes were similar as a result of fatigue. The stiffer landing strategy appears to be a general compensation following this type of fatigue protocol.

Chappell and his colleagues [42] studied landing biomechanics in ten male and 10 female recreational athletes. Subjects performed a series of 5 maximum vertical jumps, followed by a maximal 30m sprint. This was repeated continuously until volitional exhaustion. Anterior tibial shear force (ATSF) was calculated as the peak value during a two-foot landing on a forceplate. Three-dimensional knee kinematics and sagittal and frontal plane knee kinetics were calculated at the peak ATSF. The authors reported that knee flexion decreased as a result of fatigue in both genders. While females displayed greater knee extension moments, there was no change as a result of fatigue. Knee valgus moment increased for both genders post-fatigue, as did ATSF. Both of these variables were higher in females than males, regardless of fatigue state. The authors concluded that muscular fatigue caused significant changes in knee biomechanics, and that the increased ATSF and knee valgus moment, combined with the decreased knee flexion angle, created a high-risk situation for the ACL. All three of these factors have been associated with increased ACL strain in previous literature.[15, 33, 119]

Wikstrom [158] studied the effects of different fatigue protocols on time to stabilization and selected ankle and knee kinematics. Twenty subjects (8 male, 12 female) performed a standing broad jump, landing on their dominant leg on a forceplate. They were instructed to stabilize their body as quickly as possible following landing. Fatigue was induced at two separate sessions separated by several days: one session used an isokinetic protocol to induce ankle plantarflexor/dorsiflexor fatigue, while the second session used a series of stations involving a sprinting agility drill, box jumps, bounds, and hops. A timed run was completed pre-fatigue, and once subject's completion time increased by 50% they were deemed to be fatigued. The authors reported that vertical and anterior-posterior stabilization time changed post-fatigue, and VGRF increased post-fatigue. However, there were no changes in peak joint angles (ankle dorsiflexion, knee flexion, or knee valgus). There were also no differences between the two fatigue protocols. The authors concluded that isolated isokinetic fatigue produces similar changes in biomechanics as a more functional fatigue protocol, but that neither protocol demonstrated high levels of compromise to the mechanics of the lower extremity. One area of concern in this study was the use of 2 video cameras collecting at 60Hz to calculate joint kinematics. Under-sampling is a concern with this data, as is the accuracy of this method to calculate joint movement in a landing task. Additionally, his study may have been underpowered to find interaction effects.

The three studies above offer a different set of conclusions than the studies that utilized only CKC exercises. Both Chappell and Willson found that a combination of jumping and either squatting or sprinting resulted in a stiffer landing strategy, while Wikstrom found no changes after his functional protocol.[42, 153, 158] Perhaps the increased demands placed upon the lower extremity as a result of impact following repetitive landing activities resulted in a different response from the neuromuscular system. Of interest is that both Chappell and Willson likely used similar points in the landing cycle, points which are different than those used in most studies. Both peak ATSF and peak knee extension moment occur at almost the same point in the landing cycle.[28] Neither author reported peak joint angles, which is what the studies using exclusively CKC exercise generally reported. Perhaps subjects have decreased sagittal plane joint angles when the knee experiences the greatest anterior-posterior stress, but is able to undergo a greater ROM during an entire movement cycle to attenuate landing forces. This is one advantage of examining the coordination profile of an entire landing cycle, which our study proposes to do. Looking at entire curves provides a greater understanding of how movement changes in response to external constraints, like fatigue.

*Intermittent Shuttle Runs*

Sanna and colleagues [159] studied the effect of intermittent shuttle runs, at speeds and durations that represent typical game play during soccer, on lower extremity biomechanics. Twelve female soccer players performed anticipated sidestep cuts before and after a fatigue protocol, designed to simulate soccer activity. The authors reported that knee internal rotation range of motion during the stance of the cut increased post-fatigue, but no other kinematic or kinetic variables changed in the hip, knee, or ankle. The authors concluded that, although this fatigue protocol had been used in prior literature and found to change physiological parameters, it did not significantly alter lower extremity biomechanics. The authors do point out that the cutting task (anticipated at 45°) may not have been very challenging, and that other tasks may have been more sensitive to fatigue-related changes. In pilot work performed for the proposed study, we found that few differences exist between genders during an anticipated, 45° cutting task. Thus, we propose looking at a more externally-valid, demanding unanticipated cutting task that should elicit more differences due to gender and fatigue state.

*Graded Treadmill Tests*

Nyland et al. [157] performed a study on the effects of a graded walking treadmill protocol on female athletes. Nineteen female basketball or volleyball players had lower extremity kinematic and EMG activity measured during a "run-and-stop", where the subject ran towards a forceplate and then landed with their dominant leg first, followed by the opposite leg, on the forceplate. Fatigue was induced via a graded uphill walking protocol on a treadmill, until the subject volitionally stopped. The authors found that the fatigue protocol did not change any sagittal plane joint angles, measured at peak posterior GRF, but that the quadriceps and hamstrings exhibited delayed onsets post-fatigue. The authors concluded that joint movements may not change post-fatigue, but that the neuromuscular strategy employed by the body to absorb forces may change to accommodate the loss of muscular force production.

Moran and Marshall [156] examined how fatigue impacts landing forces and mechanics during drop-landings from two different heights. Fifteen male students were asked to land from two different heights: 30cm and 50cm. They were then instructed to complete a graded treadmill running protocol until they reached a rating of perceived exertion of 17 (on the Borg scale). The subjects then repeated the drop landings from the two heights. Sagittal lane knee joint kinematics and tibial accelerations were analyzed pre- and post-fatigue. The authors found that fatigue did not change knee flexion at either initial contact with the ground, or peak flexion value. However, knee flexion velocity increased post-fatigue, as did peak tibial accelerations at the 30cm height. While fatigue did not impact knee joint kinematics, it did increase velocity and tibial accelerations in such a manner that may predispose an athlete to lower extremity injury. The authors concluded that performing landings while fatigued may be detrimental to lower extremity joint movement and health.

Benjaminse and colleagues [45] recruited 30 physically-active subjects (15 male, 15 female) to perform single-leg stop-jumps pre- and post-fatigue. Each subject completed a modified Astrand treadmill protocol, where speed was kept constant and grade was gradually increased until the subject could no longer continue running. Frontal and transverse plane hip kinematics, as well as sagittal and frontal plane knee kinematics, were calculated during the single-leg landings. The authors found that hip kinematics at initial contact and peak value did not change post-fatigue, but both peak knee valgus and knee flexion at initial contact decreased after fatigue. There were no gender differences pre- or post-fatigue. From these results, the authors concluded that both genders employ a stiffer landing style post-fatigue. The joint mechanics may reflect a greater reliance on static joint structures to provide stability, due to the decreased ability of muscular tissue to provide restraints to movement. However, there was no assessment of muscular fatigue or changes in kinetics, thus conclusions from this study may be viewed with some caution.

Generally, the fatigue protocols that likely induced significant cardiovascular fatigue (via graded treadmill tests and intermittent shuttle runs) did not display many changes to lower extremity kinematics. One of the major limitations to these studies is that none actually measured muscular fatigue as a result of the fatigue protocol. Sanna and Benjaminse utilized changes in vertical jump height to assess loss of muscular strength. The effect size for the change in vertical jump in the Benjaminse study was relatively small, and a possible outlier prevented significant changes in power output to be found in the Sanna study from pre to the first post-test measurement.[45, 159] The other two studies relied on RPE or volitional exhaustion, which may not accurately reflect a loss of muscle force production. Additionally, these studied used protocols that relied solely on straight-ahead running, which would not be expected to produce as much fatigue in frontal or transverse-plane stabilizing muscles as activities that require lateral movements. The results from these studies suggest that cardiovascular fatigue protocols do not display the same changes in biomechanics as more controlled, squatting and/or landing movements.

***General Conclusions***

The studies outlined above appear to give a complex, disparate picture of the effects of muscular fatigue on lower extremity biomechanics. Indeed, the proposed task specificity of muscular fatigue is broadly confirmed as a result of these studies.[127] However, some interesting questions arise from these studies. First, which "fatigue protocol" best simulates actual athletic activity? There have been no studies that attempt to compare the strength changes after a typical practice or game to those seen after an imposed fatigue protocol. This is an important area to address in future research. The results of the Wikstrom study suggest that isokinetic and functional protocols produce similar changes, but that conclusion may be limited to the ankle musculature and is hampered by both the methodology and sample size of the study.[158] While more pronounced fatigue effects occur during CKC, or CKC plus jumping, activities, it is unclear whether this fatigue reflects on-field experiences.

Another important area to address is the contributions of central and peripheral fatigue during athletic activity. The studies outlined earlier support the notion that variable-intensity exercise produces different levels of central fatigue than constant-state exercise, and that SSC activity also exhibits significant central fatigue compared to isolated concentric muscle action. The activities that stressed the SSC mechanism of lower extremity muscles (repeated squats or jumps) tended to display greater changes in joint biomechanics than simple running protocols. While the treadmill protocols likely induced significant "fatigue", the fact that two studies used volitional exhaustion to indicate fatigue may have hampered the development of central fatigue. As Amann and Dempsey hypothesized, the central nervous system may tightly control the level of peripheral fatigue that occurs during activity.[139, 140] In the Moran and Nyland studies the subjects may have simply elected to stop activity to prevent further peripheral fatigue. This may have prevented central fatigue from occurring to a significant degree. The notion that central fatigue may be a critical determinant of biomechanical changes is illustrated by the Borotikar study, where cognitive demands significantly changed the biomechanical response to fatigue.[41] Thus, examining the level of central and peripheral fatigue during fatigue protocols, as well as actual athletic activity, will allow for more definitive conclusions to be drawn from these studies.

So what is likely to occur during athletic activities in sports that are high in ACL injury? These sports typically display high levels of intermittent activity that require high levels of both aerobic and anaerobic performance.[144, 145] It is likely that high levels of muscular fatigue are elicited during these activities.[132, 133] The muscles likely perform SSC-type contractions, where both eccentric and concentric contractions must occur for adequate power and performance. However, a complete loss of muscular function is unlikely. Anecdotally, most clinicians and coaches would suggest that an athlete would be removed from a contest prior to a severe decrement in performance level. Thus, the fatigue exhibited by athletes during competition would likely include mechanisms tested by all of the reviewed studies, but in different combinations that would produce a combination of central and peripheral fatigue. Thus, while the reviewed studies are somewhat contradictory, they do provide an excellent platform for continued research in this area.

The research study for this dissertation addresses some of these concerns and gaps in the literature. The fatigue protocol being used has been previously studied, and has demonstrated significant levels of both central and peripheral fatigue (see appendix 1). This should better simulate on-field fatigue experiences. This fatigue protocol is multi-directional in nature, which also better simulates game-play. This fatigue protocol has significant cardiovascular and landing components, which makes it a hybrid of previous fatigue protocols. Based on the results of the Borotikar study, we have chosen an unanticipated task that will better simulate decision-making conditions, but should also elicit greater responses between genders and conditions. We feel that this protocol provides a greater understanding of the mechanisms responsible for the movement changes that occur post-fatigue, as well as more closely simulate actual athletic-type fatigue.

**Section Three: Movement Coordination/Variability**

The study of movement coordination and variability during lower extremity motions has progressed in the past 20 years, moving from the simple study of joint angles and timing of events to the integration of multiple movement patterns into single variables. Dynamic Systems Theory (DST) has provided a novel framework for this research, allowing researchers to explore how multiple body segments orchestrate movement in both healthy and pathologic populations. The majority of this research has examined gait parameters in a healthy group of subjects, comparing how these patterns change with the presence of environmental constraints or movement pathologies.[38-40, 160-163] However, little research has attempted to examine how these coordination and variability profiles change during more discrete athletic tasks that are associated with increased risk of acute injury, specifically ACL rupture. Knowing how the body effectively coordinates the movement of the lower extremity during athletic tasks, and how these patterns are influenced by different constraints (for example, fatigue or unanticipated directional changes), may provide a new and critical insight on potential injury risk factors in the lower extremity.

***Methods of Quantifying Coordination and Variability***

There are several methods available to measure and analyze the coordination and variability of a limb. One of the most basic techniques is to plot the position of one joint or segment to the position of another at the same point in time. These angle-angle plots provide a visual representation of the movement of two joints in a single plane, but by itself the angle-angle plot does not provide a quantifiable measure of coordination.[164] In order to analyze the angle-angle plot, vector-coding techniques are often utilized.[165] This technique has allowed for the examination of joint coupling, or how two joints move relative to one another. This technique has been used in the sports medicine literature to examine the potential for injury in runners due to changes in coupling parameters, as well as examining movement variability during unanticipated cutting.[166, 167] Principle component analysis, as well as cross-correlation techniques, have also been used to quantify coordination using variable-variable plots.[168, 169] However, these measures have little meaning without a theoretical framework to guide the interpretation of the resultant data. Additionally, many of these measures look at joint movement, which itself is a hybrid measure of the relative position of two adjacent segments. It may be difficult to interpret these data since the motion of each segment cannot be inferred simply from joint angle.

The advent of DST has provided a theoretical framework for the examination of multi-segment coordination in the human body. DST is based on the work of Bernstein, who stated that human movement is based upon the ability to organize the multiple “degrees of freedom” in the neuromuscular system.[170] Each degree of freedom represents a possible joint motion in a single plane, a possible muscular contraction, or a possible force or moment produced at a joint. Thus, there are a tremendous number of factors that the neuromuscular system must control and organize in order to produce a fluid, synchronous movement. DST states that the neuromuscular system will coordinate these various degrees of freedom based upon the constraints within and outside of the system.[36, 37, 164, 171] Thus, no two motions are identical, but will share similar patterns when the constraints within and outside the system are similar. This framework is a drastic change from the traditional views of motor control and programming theory, which believed that motor patterns were generated from the central nervous system. DST advocates for a more “softly” organized system, where movements self-organize based upon the environment and the desired outcome.

One of the critical aspects of movement coordination and the ability to produce any given motion is the presence of variability within the system. Under traditional views of movement and motor control, the variability seen during human movement was considered “noise”, due to error in measurement or error in the production of motion. However, under the framework of DST, variability is a necessary component of the neuromuscular system. The presence of a certain amount of variability from movement cycle to movement cycle indicates that the neuromuscular system can adapt to small changes (or perturbations) within the system. These changes can be due to new constraints within the environment or small changes to the internal environment of the human body. Without some variability within the system, the human body would be unable to respond to any change without completely changing the pattern of movement. Work in both cardiology and neurology has demonstrated the importance of variability within EKG and postural stability measures.[37, 172, 173] Researchers now believe that an inherent amount of variability within the neuromuscular system is critical to normal, healthy movement.

The theoretical framework provided by DST has spawned several new approaches to examining motor coordination and variability during human locomotion. The analyses used most often when DST is used as the theoretical construct are relative phase measures. The relative phase measures utilize both the position and velocity of a segment when calculating coordination (typically presented in a phase portrait). This allows for both the current position, as well as the rate of change in position, to be represented in a single measure. Many researchers believe that measures that can capture both position and rate of change give a more comprehensive picture of the current organization of a dynamic system.[174]

The use of relative phase measures was developed after researchers determined that lower extremity segments can be modeled as inverted pendulums that act as limit cycle oscillators.[175] This indicates that the pendulums move in a quasi-sinusoidal pattern, that there is a cycle of energy exchange, and that during gait-like activities these segments have a closed, periodic orbit.[36, 175] Since the segments of the lower extremity display these characteristics, then the coupling (coordination) between two segments can be expressed via relative phase measures.

Creating phase portraits for each segment allows for phase angles to be calculated for each point during an event (such as a gait cycle). In order to view the coordination between two segments, the phase angle from the proximal segment is subtracted from the corresponding phase angle in the distal segment. These values can be graphed for an entire movement cycle to visualize how two segments interact with each other to produce movement.[36] While these graphs provide a visual representation of coordination, it is difficult to statistically compare them. Calculating the mean absolute relative phase (MARP, the average value on the relative phase plot) gives a single value that can be used to compare two groups.[36] Additionally, the variability in the coordination pattern observed during each movement cycle can also be calculated as the average standard deviation of the relative phase plot. This variable (deviation phase, or DP) gives a measure of the variability in the organization of the neuromuscular system.[36]

The use of relative phase measures, when compared to other methods of quantifying movement coordination, has several distinct advantages. First, the relative phase measures are a low-dimensional parameter, meaning that a single variable can capture the dynamic state of a system. Prior research has shown that this measure represents an "order" parameter that can define the movement of the lower extremity during activities such as gait.[36, 175] As stated earlier, this parameter incorporates both position and velocity, which may better represent the entire organization of movement, and not simply angular position changes. Finally, relative phase measures have been demonstrated to be sensitive to changes in "control" parameters (variables which change the phasing and coordination between two segments). These control parameters have included neuromuscular dysfunction, gait velocity, and clearance of obstacles during gait.[38, 160, 163, 175] Thus, alterations in movement due to fatigue or feedback (two control parameters used in the present study) are likely to be reflected in relative phase measures if they cause any appreciable alteration in movement organization.

***Movement Coordination and Variability in the Lower Extremity***

The study of relative phase coordination and variability in the lower extremity has been primarily conducted during gait. Two studies by Stergiou and colleagues examined the influence of obstacles during running gait.[161, 162] These two studies demonstrated that coordination parameters (MARP values) change when obstacles of different heights must be cleared prior to foot strike. In particular, the obstacle caused a more in-phase relationship in the sagittal plane between the shank and thigh, particularly during the first period of the stance phase of gait.[161, 162] The relationship between the foot and shank in the frontal plane also became more in-phase.[161] In both studies, the obstacle generally increased variability in the coordination patterns, although in many instances these changes were small and non-significant. These studies suggest that introducing a perturbation to a normal gait pattern will cause changes, particularly in the phasing of segment action. In particular, during the initial "impact" phase of running, an increase in ground reaction force is related to a decreased MARP value. This may indicate a compensation to the higher forces, or potentially a movement profile that may place runners at risk for injury.[161] However, these studies did not use a patient population, so these suggestions require additional study.

Some novel studies have attempted to fill this gap in the literature by comparing normal populations to patient samples, in order to understand how changes in phase dynamics may relate to injury. Kurz and colleagues studied coordination changes between ACL-reconstructed patients and healthy controls.[39] These subjects were tested during both walking and running gait. Sagittal plane relative phase profiles for the foot-shank, and shank-thigh, were calculated, and revealed that ACL-reconstructed patients had a more out-of-phase relationship between the foot and shank during walking gait, but a more in-phase relationship during running. Also, a more in-phase relationship was seen between the shank and thigh during walking. The relative phase portraits also showed dramatic differences in pattern and timing of local minima during gait cycles.[39] The authors conclude that these changes are due to a loss of proprioceptive ability due to surgical reconstruction of the ligament, and may be one reason that ACL-reconstruction leads to lower extremity joint pain and disability. The results of this study do generally agree with the previous literature that suggested that a more in-phase relationship during gait is associated with higher ground impact forces. Thus, these studies may suggest that a more in-phase relationship between segments (particularly in the sagittal plane) may be related to injury.

Hamill and colleagues, as well as Heiderscheit and colleagues, examined how coordination and variability are related to structural abnormalities and the presence of anterior knee pain in runners.[38, 163] Relative phase portraits as well as DP were calculated, comparing runners with high Q-angle (representing the angle of pull of the quadriceps on the patella), a condition associated with a higher risk of developing anterior knee pain or patello-femoral pain syndrome (PFPS), to runners with lower Q-angles. The authors of both studies found no changes in phase dynamics, patterns, or variability between these groups, suggesting that this particular structural abnormality does not substantially change movement coordination.[38, 163] However, Hamill and his co-authors also studied subjects who were currently experiencing PFPS to those who were healthy, and did find that variability was generally lower in symptomatic individuals, particularly during the terminal part of the stance phase.[38] The authors suggest that, while the presence of a high or low Q-angle is not reflected in the coordination profiles of runners, those who are symptomatic with PFPS do have altered stability in their profiles. The lower variability may be an attempt to constrain knee motion to prevent pain during activity, or it may be a pre-injury profile that caused repetitive overload of structures within the knee, leading to pain and injury.[38] Again, these results are preliminary, but do suggest a relationship between coordination, variability, and the presence of injury in the lower extremity.

The measurement of movement variability has been performed between genders who participate in soccer, hypothesizing that females will exhibit differences in variability that may be related to their higher incidence of ACL injury.[26, 108, 167] However, these authors have used different methods to quantify variability. McLean and colleagues used the standard deviation, or coefficient of variation, of various kinematic joint measures during the stance phase of sidestep cutting maneuvers. In his studies, females demonstrated higher variability in knee valgus position and knee internal rotation angles.[26, 108] His conclusion was that higher variability may increase the likelihood of performing a cutting task with excessive valgus and tibial rotation, which both increase load on the ACL and are associated with potential injury. However, use of this single variable does not represent the organization of movement, just the resultant joint motion. Thus, these results may not directly compare to other investigations that use relative phase variability.

Pollard and colleagues examined how coupling variability is influenced by gender during an unanticipated cutting task.[167] Using vector coding techniques on angle-angle plots, she quantified the variability in these measured between the thigh and leg in multiple planes of motion. Her results suggested that female soccer players had significantly less variability in the frontal and transverse planes between the thigh and leg, as well as in a knee flexion/extension-knee rotation couple, and knee flexion/extension-hip rotation couple.[167] She concluded that this decreased variability limits the ability of the female soccer player to respond to perturbations, which may put them at higher risk of injuring the ACL during cutting maneuvers. However, since this study utilized healthy athletes in a generally well-controlled environment, it remains to be seen whether these variability patterns would hold true on the field.

The current literature in sports medicine is unclear about the role coordination or variability may play on injury development. The above studies suggest that there are discernable differences in coordination and variability in injured runners during cyclic gait patterns. Additionally, there are changes in variability during more discrete athletic tasks between male and female soccer players. However, there have been no studies that have attempted to quantify the relative phase coordination and variability profiles of athletes during discrete athletic tasks (such as cutting). Quantifying these measures will allow researchers to examine how healthy athletes organize their neuromuscular system in order to perform these athletic tasks. Researchers can then compare these values by gender (to see if the 'higher risk' females are different than males), or by changing task and environmental constraints (via fatigue or unanticipation) to see how coordination profiles and variability change. Using DST as the theoretical framework will allow for some conclusions to be drawn about how the lower extremity coordinates movement under a variety of conditions, and will guide future research on how these profiles may be related to injury risk.

**Section Four: Verbal Feedback**

The use of feedback is an important part of learning motor skills, and improving technique of previously-learned skills. Feedback involves the use of either sensory information, or external information given by an instructor, to learn new motor skills or behaviors.[50] The type of feedback given to learners can be classified in two ways- knowledge of results (KR) or knowledge of performance (KP). Knowledge of results refers to feedback that strictly addresses the end result of a skill (such as making a basket in basketball). However, KP addresses the movement or components of the skill being performed (such as flexing the wrist and "following through" with the arm during a basketball shot). The way in which feedback is delivered has been the subject of numerous investigations in the motor learning and psychology literature. However, few investigations have been conducted on how feedback can be used to prevent injury. The researchers who have utilized feedback as a method to change lower extremity movement patterns have focused on reducing forces while landing from a jump, a movement associated with increased risk of ACL injury.[48-51, 176] These studies have helped provide the theoretical framework for inclusion of technique training in ACL injury prevention programs, but have not addressed how feedback can be used to change unanticipated cutting technique, or whether feedback is effective under fatigued conditions. These gaps in the literature should be addressed in order to fully understand the impact of feedback on injury prevention methods.

***Feedback and Decreased Landing Forces***

The work of Prapavessis and McNair provided the early evidence that feedback could be used to alter lower extremity landing forces. In three studies published from 1999-2003, these authors and their colleagues examined how verbal feedback, focusing primarily on decreasing landing forces, could influence how children and adults perform jump landings. Prapavessis and McNair (1999) initially examined if sensory feedback or augmented feedback produced significant changes in landing forces. Subjects either received information to "land as softly as possible" which required they use their own sensory information as feedback, or to bend their knees and land on the balls of their feet, which provided external (augmented) cues on how to physically perform the movement. The authors found that augmented feedback resulted in a greater decrease in vertical ground reaction force compared to sensory feedback.[50] A second study published in 2000 by McNair expanding on these data, by incorporating an imagery condition where subjects were given feedback in metaphorical terms (for example, to imagine landing like leaves floating down towards the ground). Two other feedback groups received either technical instruction or were told to focus on auditory cues to reduce landing force. The authors found that technical instruction (similar to the instruction provided in the Prapavessis study) and auditory cues resulted in a significant decrease in landing forces, whereas imagery feedback did not. In a final study, Prapavessis (2003) studied the effect of augmented verbal feedback on landing forces in a pediatric population. Again, information on landing technique was provided, and again this feedback resulted in lowered landing forces.[176] These three studies all demonstrated that simple, technical instruction can positively influence landing forces in a variety of populations.

The work of Onate (2001, 2005) further examined how feedback can be used to alter landing forces, as well as joint kinematics. In his studies, verbal feedback was combined with video modeling, so that subjects had several sources of KP between trials. In his 2001 study, the effects of both sensory and augmented feedback were studied. The results demonstrated a decrease in landing forces after augmented feedback, but not in the sensory feedback condition.[177] In his second study, a variety of lower extremity kinematics and kinetics were studied following augmented feedback. Onate found that augmented feedback increased maximum knee flexion angle and decreased ground reaction forces compared to control.[51]

This study was one of the first to demonstrate that feedback could be used not only to decrease landing forces, but also to influence landing kinematics in a way that is believed to decrease risk of ACL injury. However, the ability to use video modeling on the field to change technique is not always feasible, which limits the ability to use this feedback technique for large numbers of athletes.

A study by Cowling and colleagues (2003) used a strictly verbal feedback model to attempt to change landing technique and forces in athletes. Subjects were given instruction to either “land with their knee bending” or to “turn on the muscles at the back of your thigh”. These two types of feedback were designed to instruct athletes on ways to perform a landing maneuver in way associated with lower risk of ACL injury (by increasing knee flexion or increasing hamstring activity). The authors found that the “knee flexing” instruction decreased both ground reaction forces and knee flexion angles at initial contact and peak flexion angle, while the “hamstring” group did not have any significant changes.[48] The authors concluded that verbal instruction should be simple and focus on gross motor patterns in order to effectively elicit changes in technique.

The above studies have demonstrated how feedback can be used to change lower extremity landing forces and movements during jump landings. These authors have all concluded that feedback should be used to instruct athletes how to perform jump landings in a safer manner, one that reduces landing forces and may serve to protect the knee from injury. In most of these studies, the use of simple technical verbal feedback was enough to elicit the desired changes.[48-50, 176] These results have important implications for injury prevention. If coaches and athletic trainers can provide feedback on movement performance, and athletes can acutely change their performance in a favorable manner, then feedback may be a simple and effective way to prevent injury.

***Feedback as an Injury Prevention Modality***

Since feedback and technical instruction have been shown to favorably influence lower extremity movement in athletes, several injury prevention programs have incorporated feedback as a method for ACL injury prevention. The late Charles Henning was the first to specifically investigate how intensive technique instruction could influence ACL injury rates. In his unpublished research, athletes who received feedback on how to perform landing and cutting maneuvers reduced their risk of ACL injury by 89%.[178] While promising, this work was conducted as a case-series, and lacked a control group for comparison. However, this work is still the only research that used technique instruction as the sole modality for injury prevention. Several other injury prevention programs have used technique instruction and feedback as part of a multi-component training series.[74, 179-181] All of these studies found a decrease in injury rates following a prevention program. However, it is unclear what the relative impact of feedback was in these programs, and whether feedback itself was beneficial outside of the other modalities.

The use of verbal feedback as a method for changing movement patterns and influencing injury risk has been generally positive. Multiple studies have found that feedback can significantly reduce landing forces while landing.[50, 51, 176, 177] Two studies have also found that knee flexion angle will increase while landing after feedback.[48, 51] The use of verbal feedback and technique instruction are commonly used as components of injury prevention programs that have been successful at decreasing ACL injury.[74, 178-181] However, there are several questions that require further research. First, can feedback be used to change movement patterns and landing forces during more complex movements, such as sidestep cutting? Will feedback still be effective when a movement is unanticipated? And finally, will feedback still produce changes when it is given under fatigued conditions? All of these situations are critical, as they are more relevant and externally valid conditions that are experienced by athletes during play. The current research project aims to understand just how feedback can be used as a method for injury prevention in athletes in high-risk sports.

# CHAPTER 3

## Rationale

The primary purposes of this study were to examine how fatigue and verbal feedback alter coordination, movement variability, and selected kinetics in healthy college-aged athletes. The selected tasks and dependent variables allow the researchers to draw meaningful conclusions based upon the results of this study. Utilizing an unanticipated cutting task removes one limitation of most laboratory-based studies on athletic movement: the anticipatory knowledge of the precise direction of movement. Prior literature has established that single-leg cutting is a movement associated with many non-contact ACL injuries.[54, 59-61] These authors also suggest that many injuries occur during unplanned, or perturbed, motions. In most field athletics, participants are unaware of the direction they will need to move in order to respond to the play of other athletes as well as the movement of the ball (or other apparatus). Several authors have studied the effects of anticipation on cutting movements, and have found that both kinematics and kinetics of the lower extremity change in unanticipated conditions.[41, 182] In this study, we wanted to simulate actual athletic movements as closely as possible in the laboratory setting, thus we felt that using an unanticipated task was most appropriate.

The fatigue protocol used for this study has been found to produce significant levels of both central and peripheral muscular fatigue (see Appendix 1). Several authors believe that typical athletic play produces both forms of muscle fatigue, thus we felt that this fatigue protocol more closely approximated the type of fatigue experienced by athletes during practices or games.[41, 134] Pilot testing revealed that the current methodology for this study allows researchers to see fatigue-related changes in movement and performance for 5-10 minutes, within the time-frame for the post-fatigue testing proposed in this section.

Finally, the dependent variables chosen for this study are thought to directly relate to ACL injury risk. The peak values for the kinetic variables (knee extension moment, knee valgus moment, and ATSF) were measured at their peak value during the first 40% of the stance phase. Most researchers suggest that ACL injury occurs immediately following landing, when ground reaction forces and moments are high.[60, 61] Vertical ground reaction forces peak early following ground contact during landing, typically during the first 20% of the stance phase. Thus, measuring kinetics during the initial landing phase when ground reaction forces have peaked, and the body is attempting to decelerate, provides a more appropriate measurement of factors possibly linked to injury risk. The use of coordination and variability analyses is novel in the ACL body of research. However, these measures provide a way of examining the movement of multiple body segments, as well as studying how the neuromuscular system organizes and changes movement under different circumstances. These variables give a new, unique insight into the movement and control of the lower extremity, as well as provide additional information on how these variables may relate to ACL injury risk.

## Population

### *Subjects*

A total of 61 students (31 males, 30 females) from the club sport population at the University of North Carolina at Chapel Hill were recruited for this study. Club sports athletes were chosen due to their relatively high level of fitness, regular competitive athletic activity, and their exposure to both cutting motions and situations that place them at higher risk of ACL injury. We believed that this population would provide a more homogenous study sample and reduce some of the variability in the results, so that significant changes could be more easily seen. Students who participated in soccer, lacrosse, basketball, volleyball, or handball were eligible for participation. All subjects were between the ages of 18-30, currently participated in their respective club sport's practices and/or competitions, performed a minimum of 30 minutes of physical activity 4 days a week, and in good health. The following exclusion criteria applied to all subjects:

- No history of lower extremity surgery in the past year
- No history of any knee surgery
- No history of prior ACL injury
- No history of lower extremity injury in the previous 6 months that prevented participation in practices, games, or conditioning for greater than 3 consecutive days
- No history of cardiac/respiratory conditions that would prevent participation in strenuous physical activity

Subjects were recruited via informational flyers given to club team members by the primary investigator. The primary investigator also attended club team practices to recruit interested subjects.

All subjects read and signed an informed consent form approved by the University of North Carolina at Chapel Hill Biomedical IRB, completed a basic health questionnaire, and completed a Physical Activity Readiness Questionnaire (PAR-Q, based on American College of Sports Medicine guidelines).

***Group Assignment***

Following consent, all subjects were assigned to either a feedback group (n=31) or non-feedback group (n=30), balanced with respect to gender and sport. A master group assignment list was created prior to data collection, stratified by gender and sport. An equal number of "Feedback" and "Non-feedback" assignments were randomly created for each sport and gender combination (for instance, for women's basketball, the list of assignments was FB, NFB, FB, FB, FB, NFB, NFB, FB, NFB, NFB). As each subject came in, they were assigned sequentially to the feedback or non-feedback groups based on their sport and gender list. All subjects performed the same pre-testing procedures and fatigue protocol, and the group assignment was used to determine whether the subject received the verbal feedback protocol following the fatigue protocol.

***Power Analyses***

An a priori power analysis, using pilot data and previously published data, revealed that a sample size of 30 per feedback comparison group would allow the investigators to detect a minimum 20% change in all dependent variables with a power of 0.70 and an $\alpha$=0.05 (see table 1). Prior research using verbal feedback has shown a 22-27% decline in VGRF after feedback.[50, 51, 176] Onate et al. also found a 20% decrease in peak ATSF following augmented feedback.[51] Additional studies have also found increased knee flexion angles of greater than 20% post-feedback.[48, 51] Thus, the sample size should allow for the detection of changes post-feedback in our study population.

Power analysis also revealed that the proposed repeated measures research design would allow researchers to detect a minimum 15% change in all dependent variables post-fatigue with a power of at least 0.75 and a sample size of 30 (see table 2). Previous literature suggested that fatigue-related changes in knee joint kinetics are between 15-20%.[42-44] Thus, the study design should provide adequate power to detect differences due to fatigue.

A change in the dependent variables of 15-20% represents a clinically-relevant change in coordination, variability, and knee joint kinetics. Previously published literature suggests that a minimum 20% difference has been found in MARP in the sagittal plane between ACL-reconstructed patients compared to healthy controls during gait (ranging from 20.1-25.2%) [39]. Variability (using the DP measure) in the sagittal plane has been shown to increase 29.2-60.7% between young and old patients during gait.[36] McLean and colleagues demonstrated kinematic variability differences (measured by coefficient of variation and standard deviations) of 20-80% between genders.[26, 108] Finally, differences of over 20% have been demonstrated in knee extension moment, knee valgus moment, and ATSF, both between sexes and prospectively between ACL-injured and healthy groups.[18, 34] Thus, this study design was adequately powered to detect changes due to fatigue and/or feedback that have been suggested to be clinically relevant.

## Data Collection

### *Instrumentation*

A Vicon MX-40 infrared camera motion analysis system (Vicon Systems, Centennial, CO) was used to collect segment kinematic data during the unanticipated sidestep cutting task. Seven cameras were positioned in the laboratory with a capture volume of approximately 2x2x2m that was calibrated according to manufacturer guidelines. Data was collected at 150 Hz. Pilot testing revealed that 150Hz provided accurate marker data using the camera set-up in our laboratory. A global axis system was defined for the laboratory capture volume, and local coordinate systems for each body segment were aligned such that the positive Y-axis was aligned to the subject's left along the medial-lateral axis, the positive X-axis was pointing in the direction that the subject was facing along the anterior-posterior axis, and the positive Z-axis was pointing vertically along the superior-inferior axis. Digital camera data was imported into Vicon Nexus version 1.3 data collection software package (Vicon Systems, Centennial, CO) for integration with forceplate data and marker identification.

One Bertec forceplate (Type 4060-08, Bertec Corporation, Worthington, OH) was used to collect ground reaction force data during the unanticipated sidestep cutting task. The axis system of this forceplate was aligned to the global coordinate system of the laboratory defined by the motion capture system. Analog data were collected at 1500Hz and passed through an A/D board prior to being imported into the Vicon Nexus software. These data was synchronized with the segment kinematic data within the Vicon Nexus software.

During the unanticipated sidestep cutting task, a custom device was used to cue the direction of the cut for each trial. A gate using an infrared-laser emitter and reflector was placed 65cm in front of the forceplate and was wired into an A/D board interfaced with a personal computer. When the subject broke the laser beam, the signal from the device provided a trigger on a custom LabVIEW program (National Instruments, Austin, TX) to illuminate either a "left" or "right" arrow. This image was projected onto a video screen placed in front of the subject within their direct visual field. The direction of the arrow directed the subject to cut off of their dominant leg to either the left or right.

A Vertec Jump Training System (Questtek Corp, Northridge, CA), consisting of 49 color-coded moveable plastic vanes, was used to assess jump height. The device was set for each participant according to manufacturer's guidelines. Participants started with both feet shoulder-width apart standing just below the device. They reached as high as they could, without rising on their toes, with both arms stretched overhead. The bottom vane of the Vertec device was placed such that the subject's fingertips just touched the bottom of the vane. The participant then performed a maximum standing vertical jump while reaching for the tallest vane on the device. The participants were not allowed to take a step prior to the jump.

***Procedure***

*Warm-up:* Subjects reported to the Sports Medicine Research Laboratory for a single testing session that lasted approximately 120 minutes. All subjects initially read and signed the Informed Consent form, as well as a brief medical history, Physical Activity Readiness Questionnaire (PAR-Q), and a sport participation and current activity history questionnaire. The subject's height (cm) and mass (kg) were recorded using a stadiometer and digital scale, respectively. The subject then changed into a pair of black spandex bike shorts and a black spandex compression-style tank. Subjects provided their own athletic shoes for testing. Once the subjects changed, a 5-minute warm-up was performed on a bicycle ergometer at an estimated rating of perceived exertion (RPE) of 12, followed by a standardized 5-minute dynamic warm-up protocol designed to actively prepare the muscles of the lower extremity for physical activity.

*Testing battery:* Following the warm-up period, the pre-testing battery was performed. This testing battery involved testing maximum vertical jump height, motor skill and agility, and performance of an unanticipated sidestep cutting task. Vertical jump height was assessed using the Vertec device. Subjects were given 1-3 practice trials to become accustomed to the use of the device. Each subject then performed 3 maximum vertical jumps. The height reached for all three jumps was recorded, and the highest one was used as the subject's maximum vertical jump. One minute of rest was given between each jump to reduce the likelihood of fatigue.

The Motor Skill Test (MST) was developed by Welsh and colleagues.[183] This task involves single-leg hopping and split-jumps on a pre-marked course. Twelve 12-inch by 12-inch squares (six black, six white) were arranged in a grid 6 squares wide and 2 squares deep (see figure 1). The subjects were instructed to begin to the left side of the grid, and hop on their right leg in the black squares (from left to right). Once they reached the right side of the grid, they were instructed to hop on their left leg in the white squares (from right to left). Once they reached the left side of the grid, they were instructed to perform split jumps (jump with one leg in front of the other) to the right side of the grid and back. They were instructed to always jump with the right foot in the black squares, and the left foot in the white squares, resulting in switching the front foot on each jump. Once they had completed the jumps, they were finished with the test. During the entire test, the subjects were instructed to keep their foot completely within the square they were supposed to land in. If more than ½ of their foot was outside the confines of the square, they were given an "error".

The MST is scored in three ways. The total time (in seconds) to complete the course (from the first hop to the last jump) is the MST-time score. The number of errors were also counted, and represent the MST-error score. The MST-total score is the MST-time added to the (number of errors multiplied by 0.5). Thus, each error represented a 0.5 second penalty for the MST-total score. A higher score on any component indicates a poorer ability to control full-body movement. Welsh and colleagues [183] found that the MST had an intraclass correlation coefficient of 0.91, indicating that changes in this variable are not likely due to measurement error.

The unanticipated sidestep cutting task was performed in the calibration volume of the infrared camera system. A custom 25-point markerset was used for all data collection (see figure 2). Fourteen millimeter diameter retroreflective markers were placed bilaterally on each subject in the following locations: tip of the acromion, anterior superior iliac spine (ASIS), greater trochanter, anterior thigh, medial femoral epicondyle, lateral femoral epicondyle, anterior tibia, medial malleolus, lateral malleolus, calcaneus, head of the 5$^{th}$ metatarsal, and head of the 1$^{st}$ metatarsal. One additional marker was placed over the L5-S1 joint space. The medial epicondyle and medial malleolus markers were used for calibration purposes only, in order to calculate joint centers during data processing. The location of each retroreflective marker was marked using permanent marker on the skin, and the markers were affixed using double-sided tape, prewrap, and white athletic tape as necessary. The subject then stood with their feet shoulder-width apart, feet pointed straight ahead, with one foot on each forceplate, and their arms abducted 90 degrees. This position was held for 1-2 seconds while marker positions were recorded, and represented the calibration/static trial for data processing. The calibration markers were then removed.

Performance of the unanticipated sidestep cutting task involved a single-leg cut off the dominant leg (see figure 3). Leg dominance was determined as the leg the subject would use to kick a ball for maximum distance. A hurdle (17cm) was placed 25% of the subject's height from the front edge of the forceplate. The laser gate was placed 65cm from the front of the forceplate, so that the time from the directional cue to landing on the forceplate is constant for all subjects and between 300-400ms. A piece of tape was placed 50% of the subject's height from the front edge of the forceplates and represented the starting point for the cut. Subjects performed a double-leg jump over the hurdle. When they passed over the hurdle and broke the laser beam, they cued the direction of the cut to appear on the video screen. The arrow on the screen directed them to cut in one of two directions: to the contralateral side of their dominant leg (sidestep cut), or to the ipsilateral side of their dominant leg (crossover cut). After the direction of the cut had been cued, the subject landed with his/her dominant foot on the forceplate, and cut 60 degrees in the direction indicated on the screen. A total of 16 unanticipated cuts were performed, 10 sidestep and 6 crossover, with the directions of the cut randomized. Any trial where the subject failed to make the 60 degree cut or did not land with the foot completely on the forceplate were discarded and repeated.

*Fatigue protocol:* Following this pre-fatigue testing battery, the participants performed a bout of exercise to induce fatigue (fatigue protocol). This fatigue protocol has been found to produce significant central and peripheral fatigue in preliminary testing, which likely simulates the fatigue experienced during athletic participation (see appendix 1). First, the participant performed a standing broad jump for maximum horizontal distance. The investigator measured this distance and placed a mark on the floor which represents 75% of the maximum distance. This was the target for the broad jumps during the fatigue protocol. The primary investigator then instructed the participant on the modified agility course and had the participant practice once before beginning the full protocol. The agility course was setup on a gymnasium floor in the same building as the testing laboratory. The course consisted of a forward sprint around the 3-point line of the court, a backward run on the baseline, a side-shuffle to the top of the “key”, a forward run and sidecut to one corner of the baseline, a side-shuffle back to the top of the “key”, and a final sprint and cut back to the baseline and the start area. A diagram is provided in figure 4. Once the participant finished the agility course, they performed 5 standing broad jumps where they jumped to 75% of his/her maximum jump distance.

Participants performed the agility course once at full-speed while they were timed. The time it took for them to complete the agility course was their target. Participants then performed the agility course and the 5 broad jumps (with 5 seconds of rest between each sequence) until their completion time for the agility course was 150% of the target 3 successive times or they completed 30 trials. During pilot testing, subjects performed 20.5 (range: 7-30) repetitions of the fatigue protocol, resulting in an average of 20-25 minutes of physical exertion. At this time the fatigue protocol ended, and subjects jogged back down to the laboratory.

*Feedback delivery:* Once subjects returned to the laboratory (maximum 45 seconds), those in the feedback group received augmented verbal feedback on the performance of the unanticipated sidestep cutting task. This augmented verbal feedback consisted of 30 seconds of verbal coaching on landing technique. This feedback was based on prior augmented feedback instructions and studies on minimizing joint moments during cutting tasks.[48-50, 124] The other half of the subjects were allowed to rest for 30 seconds (equivalent time that it took to deliver the feedback). The feedback script stated:

*"For these next cuts, I want you to focus on three things. First, land softly on the ground. Second, keep your body centered over your foot as you cut. Third, try to make this movement as smooth and coordinated as possible."*

*Post-test:* After the feedback or rest, the subjects completed the testing battery described above a second time. All of the same procedures were followed as stated in the pre-fatigue testing battery (see figure 5 for a diagram of the entire testing procedure).

## Data Processing and Reduction

### *Marker Identification and Processing*

Kinematic and ground reaction force data were collected by the Vicon Nexus software program. All retroreflective markers (25 for calibration/static trial, 21 for movement trials) were identified and labeled using a pre-defined labeling template. Any missing marker trajectories were filled using a Woltring spline function provided by the Vicon Nexus software. Extra reflections in the field of view were deleted following marker identification and trajectory processing. These data, along with the synchronized ground reaction force data, were then saved as a .c3d file.

Marker and ground reaction force data were imported into The Motion Monitor biomechanical data analysis software package (Innsport Inc, Chicago, IL). The calibration/static trial was used to build the biomechanical segment model of the subject. The segments of each subject's lower extremity and trunk were defined as rigid bodies with a minimum of 3 non-collinear markers per segment. The following markers were used for each segment:

- Left foot: Left Calcaneus, left lateral malleolus, left 5$^{th}$ metatarsal, left 1$^{st}$ metatarsal

- Right foot: Right Calcaneus, right lateral malleolus, right 5th metatarsal, right 1st metatarsal
- Left shank: Left lateral malleolus, left lateral epicondyle, left tibia
- Right shank: Right lateral malleolus, right lateral epicondyle, right tibia
- Left thigh: Left lateral epicondyle, left greater trochanter, left thigh
- Right thigh: Right lateral epicondyle, right greater trochanter, right thigh
- Sacrum (pelvis): Left ASIS, right ASIS, L5-S1 marker
- Trunk: Left acromion, right acromion, L5-S1 marker

***Joint Center Calculation***

Following segment definition, joint centers were calculated. Ankle and knee joint centers were defined as the centroid between the lateral and medial malleolus markers, and lateral and medial epicondyle markers, respectively. Hip joint center was defined using the location of the right and left ASIS markers, as described by Bell.[184] The joint center between the trunk and the pelvis was defined at the location of the L5-S1 marker.

***Importing and Aligning Files***

Once joint centers have been defined, segment coordinate systems were aligned to the global coordinate system defined during data collection. Ground reaction force data were then imported into The Motion Monitor synchronized to kinematic data. Once this process had finished, all movement and static/calibration trials for an individual subject were imported using the above parameters. Segment kinematic data were calculated as the angular movement of the rigid segment, with the distal joint center as the axis of rotation. Angles were calculated from the longitudinal axis of the segment to the positive horizontal axis of the global axis system. For sagittal plane angles, this horizontal axis was represented by the positive X-axis, and for frontal plane angles the horizontal axis was represented by the positive Y-axis.

***Kinetic Calculations***

Ground reaction force data was used to calculate segment kinetics using standard inverse dynamic procedures as described by Gagnon and Gagnon.[185] The net knee extension moment was calculated as the resultant soft tissue moment required at the knee joint center. The net knee valgus moment was calculated as the net moment applied by the muscles crossing the tibiofemoral joint in the frontal plane. Anterior tibial shear force was calculated as the maximum value of the net shear force directed anteriorly at the tibiofemoral joint causing the tibia to translate anteriorly relative to the femur.

***Data Reduction***

Once segment kinematics and kinetics had been calculated, these data and the ground reaction force data were exported into a custom MatLab program (version 7, Mathworks, Natick, MA) for further data processing and reduction. Segment kinematic data were exported at 150Hz in order to control for any variability that may be introduced if motion data were time synchronized to kinetic data at 1500Hz. However, kinetic data were exported at 1500Hz for analysis. Exporting the kinetic data at a separate sampling frequency allowed for the important aspects of the respective data to be retained. Sampling kinetic data at a lower frequency (150Hz) may cause a substantial loss of data points and power spectrum. All segment kinematic and kinetic data were filtered using a Butterworth $4^{th}$-order zero-phase lag filter with an estimated cut-off frequency at 15 Hz, to optimize the integrity of the passed data. The 15Hz lowpass cutoff was determined by performing a residual frequency density analysis on the first 10 subjects' data, which demonstrated that over 99% of the signal could be retained using 15Hz as the cutoff (see Appendix 2). Segment angular velocities were calculated using the methods described by Winter [114], using a 5 data point (0.0267 second) moving window to calculate average instantaneous velocity for each data point. Following data filtering and velocity calculation, the stance phase of the sidestep cut was calculated from the ground reaction force data. Initial contact with the forceplate was defined as the first timeframe where vertical ground reaction force (VGRF) exceeded 10N. Toe-off was defined as the first timeframe following initial contact where the VGRF fell below 10N. The stance phase was defined as IC-toe-off, and all segment kinematic and kinetic variables were calculated during this phase.

***Dependent Variable Calculation***

All dependent variables were calculated from the first 9 successful trials (pre- and post-fatigue). Analysis of the data post-hoc revealed that a majority of subjects did not have 10 acceptable trials during each phase of data collection, due to markers not being visible or the subject "hopping" during the cut, causing the VGRF to drop to zero during the stance phase. Thus, we chose to use the first 9 acceptable trials for all subjects. Exceptions to this are noted in the results section.

*Coordination and Variability:* Segment angular position and velocity values during the stance phase were used to calculate the coordination and variability variables used for this study, using the methods outlined by Kurz and Stergiou.[36] Initially, each segment's position and velocity were normalized to 101 data points. Each point was then plotted on a phase portrait, with angular position on the x-axis and angular velocity on the y-axis. From these Cartesian coordinates, polar coordinates were calculated (r, Θ), and the phase angle Θ calculated as the inverse tangent of the velocity/position for each data point. These phase angles were then used to calculate the relative phasing of one segment relative to another. For these analyses, the relative phase angle θ is calculated as

$$\theta_{\text{relative phase}} = \varphi_{\text{distal segment}} - \varphi_{\text{proximal segment}},$$

where $\varphi_{\text{distal segment}}$ was the phase angle of the distal segment, and $\varphi_{\text{proximal segment}}$ was the phase angle of the proximal segment. Thus, for each segment pair (foot-shank, shank-thigh, thigh-trunk) in each plane (sagittal, frontal), a total of 101 relative phase angles were generated for each movement trial. These angles were then plotted for visual inspection.

Following the calculation of the relative phase angles for each segment pair in each plane of motion, the Mean Absolute Relative Phase (MARP) and Deviation Phase (DP) were calculated. Pilot testing revealed that within-day infraclass correlation for both the MARP and DP to be fair to excellent, ranging from 0.48-0.97 (see table 3). The MARP represents a single value that can be used to statistically compare the phasing coordination between adjacent segments. For this study, an ensemble relative phase curve was calculated from the 9 pre-test unanticipated sidestep cuts, and a second curve for the 9 post-test unanticipated sidestep cuts. MARP is then calculated as:

$$MARP = \sum_{i=1}^{N} \frac{|\varphi \ relative \ phase|}{N}$$

A large MARP value indicated that the two segments demonstrated a largely out-of-phase relationship, whereas a small MARP value indicated the two segments were more in-phase. MARP was calculated for the foot-shank, shank-thigh, and thigh-trunk pairs in the sagittal plane, and the foot-shank, shank-thigh, and thigh-trunk pairs in the frontal plane.

Deviation Phase (DP) represented the variability in the coordination between two segments. The standard deviations from each point on the ensemble relative phase plots were used to calculate DP as:

$$DP = \frac{\sum_{i=1}^{N} |SD_i|}{N}$$

A high DP value indicated a high level of variability in the coordination between two segments, indicating instability in the organization of the system. A low DP indicated low variability, and a high degree of stability in the organization of the system.

*Peak Kinetics:* Lower-extremity kinetics (knee extension moment (KEM), knee valgus moment (KVM), ATSF) and vertical ground reaction force were analyzed during the stance phase on the dominant leg as determined above. Peak KEM, KVM, and ATSF during the first 40% of the stance phase were calculated for each trial of the unanticipated sidestep cut. The peak VGRF was also calculated for each trial. All moment data were normalized to the product of subject height x subject mass (Nm), and all force data were normalized to subject mass (N). These values were averaged across the first 5 pre-test and 5 post-test trials to assess changes in knee joint kinetics. Additionally, the time-to-peak VGRF, ATSF, KEM, and KVM were calculated as the number of milliseconds (ms) from IC to the peak value calculated above. These values allow for an investigation into the timing of peak forces and moments relative to landing during the sidestep cut.

*Physical Testing:* The maximum height reached during the 3 vertical jump trials pre-test, and 3 trials post-test, were used for statistical analyses. The MST-time, MST-errors, and MST-total were calculated both pre- and post-fatigue, and used for statistical analyses.

**Statistical Analysis**

Means, standard deviations, and 95% confidence intervals were calculated for each dependent variable, as well as all demographic data. T-tests were used to compare all pre-fatigue testing dependent variables between the feedback and non-feedback groups to ensure that randomization was effective. Twenty-four mixed model 2x2x2 ANOVAs (group x fatigue x gender) were performed to determine changes in segment coordination (6 variables), variability (6 variables), kinetics (4 variables), time-to-peak kinetics (4 variables), and physical testing (4 variables) due to the testing performed. Post-hoc testing using Bonferroni post-hoc adjusted t-tests were performed for significant findings from the ANOVA's. Alpha was set a priori at $\alpha \leq 0.05$.

RQ 1: How does a functional fatigue protocol alter the coordination, variability, and kinetics of the lower extremity during the stance phase of an unanticipated sidestep cutting task in a healthy, athletic population?

Statistical method: We expected a significant fatigue x group interaction in the ANOVA model, with post-hoc testing indicating that the non-feedback group changed significantly from pre-test to post-test for all coordination, variability, and kinetic variables.

RQ 2: How does an acute intervention (verbal feedback) affect the post-fatigue coordination, variability, and kinetics of the lower extremity during the stance phase of an unanticipated sidestep cutting task in a healthy, athletic population?

Statistical method: We expected a significant group x fatigue interaction in the ANOVA model, with post-hoc testing indicating that the feedback group did not change any coordination, variability, and kinetic variables post-fatigue, or improved significantly compared to pre-fatigue values.

RQ 3: Do men and women exhibit lower extremity coordination, variability, and kinetics pre- and post-fatigue during the stance phase of an unanticipated sidestep cutting task?

Statistical method: We expected a significant main effect for gender in the ANOVA model, with females displaying lower MARP values in the sagittal plane, higher MARP values in the frontal plane, higher DP in all planes, and higher kinetic variables. We also expected a significant fatigue x gender interaction, with post-hoc testing revealing that women exhibit greater differences in coordination, variability, and kinetic values post-fatigue when compared to males post-fatigue.

# CHAPTER 4

## Results

Results for this study are given in tables 4-21. Demographic information is provided in table 4. Independent-samples t-tests ($\alpha$=0.05) revealed that there were no significant differences between the feedback and non-feedback groups in gender, sport, age, height, and mass (table 5). Information regarding the fatigue protocol is provided in table 6 including the initial time taken to complete the run (which was used to determine failure), the total number of fatigue trials successfully completed, total number failed, and total number performed, the RPE (rating of perceived exertion) prior to, maximum during, and at three points during the post-testing period, and the total time elapsed from the end of the fatigue protocol to the end of post-testing. Independent-samples t-tests ($\alpha$=0.05) revealed one difference between groups: the feedback group had a significantly lower RPE during the first post-fatigue assessment when compared to the non-feedback group (t=2.598, p=0.012). However, there were no other significant differences in any variable related to the fatigue protocol (table 5). Additionally, the post-testing times for both groups were equivalent, indicating that there should be no systematic difference in the level of fatigue between both groups during post-testing.

Pre-fatigue independent t-tests to compare the feedback and non-feedback groups on all dependent variables were also performed. There were only two variables where the groups were significantly different: thigh-trunk MARP in the sagittal plane (t=2.092, p=0.041) and thigh-trunk MARP in the frontal plane (t=2.098, p=0.040). No other dependent variables were significantly different during pre-fatigue testing. Thus, with the exception of the thigh-trunk MARP variables, any differences post-fatigue should be the result of fatigue.

A total of 61 subjects were tested for this study (31 Feedback, 30 Non-feedback). One subject (female feedback) was excluded because she did not complete the fatigue protocol (the primary investigator terminated the protocol early due to subject difficulty performing the tasks). One subject (female feedback) was excluded due to a mistake in marker placement during post-fatigue testing. This left 59 subjects (29 Feedback, 30 Non-feedback) that were used for data analysis. Two subjects' (female non-feedback and female feedback) joint moment and ATSF data could not be used due to a mistake in data collection procedures (AP GRF was not collected, thus any joint moments or forces would be incorrect).

All variables were calculated from the first 9 acceptable unanticipated sidestep cutting trials performed pre- and post-fatigue. There were a few exceptions where fewer trials needed to be used, due to missing markers or subject's failing to perform the cut task correctly. These were: subject 104 post-fatigue (8 trials), subject 201 post-fatigue (8 trials), subject 204 pre-fatigue (7 trials), subject 204 post-fatigue (7 trials), subject 206 pre-fatigue (8 trials), subject 207 pre-fatigue (8 trials), subject 304 pre-fatigue (8 trials), subject 308 post-fatigue (8 trials), subject 401 post-fatigue (7 trials), and subject 505 post-fatigue (8 trials).

**Coordination**

The fatigue protocol caused several changes in intersegmental coordination, as did the feedback protocol delivered to half of the subjects (see tables 7 and 8, figure 6). The ANOVAs revealed significant fatigue x group interactions for foot-shank sagittal plane MARP ($F_{1,55}$=4.641, p=0.036) (figure 7), shank-thigh sagittal plane MARP ($F_{1,55}$=4.719,

$p=0.034$) (figure 8), shank-thigh frontal plane MARP ($F_{1,55}=4.464$, $p=0.039$) (figure 9), thigh-trunk sagittal plane MARP ($F_{1,55}=4.967$, $p=0.030$) (figure 10), and thigh-trunk frontal plane MARP ($F_{1,55}=7.708$, $p=0.008$) (figure 11) (also see table 9). Bonferroni post-hoc testing (adjusted $\alpha=0.025$) revealed that fatigue resulted in a decreased MARP for the foot-shank sagittal plane ($t=7.958$, $p<0.001$), shank-thigh sagittal plane ($t=5.779$, $p<0.001$), and thigh-trunk sagittal plane ($t=4.065$, $p<0.001$) in the non-feedback group. This decrease in MARP value indicates a more in-phase pattern between the two segments. However, the feedback group only demonstrated a decrease in thigh-trunk frontal plane MARP ($t=3.087$, $p=0.004$). Thus, the results suggest that the feedback protocol was largely successful at correcting the fatigue-related phasing changes in coordination demonstrated by the non-feedback group.

Gender main effects were demonstrated in two variables: foot-shank sagittal plane MARP ($F_{1,55}=7.776$, $p=0.007$), and shank-thigh sagittal plane MARP ($F_{1,55}=7.954$, $p=0.007$). In both cases, females had higher MARP values than males. This indicates that movement patterns were more out-of-phase for females. However, there were no gender interactions with either fatigue or group.

**Variability**

The fatigue protocol caused changes in movement variability, as did the feedback protocol (see tables 10 and 11, and figure 12). A fatigue main effect was seen for the foot-shank sagittal plane DP ($F_{1,55}=13.634$, $p=0.001$), foot-shank frontal plane DP ($F_{1,55}=41.262$, $p<0.001$), shank-thigh sagittal plane DP ($F_{1,55}=4.078$, $p=0.048$), and shank-thigh frontal plane DP ($F_{1,55}=21.093$, $p<0.001$) (see table 12). In all cases, fatigue decreased movement variability (as indicated by a decrease in DP) in both the non-feedback and feedback groups.

A fatigue x group x gender interaction was seen in the thigh-trunk frontal plane DP ($F_{1,55}$=6.386, p=0.014) (figure 13). However, Bonferroni post-hoc testing (adjusted $\alpha$=0.0125) found no significant differences by gender within the two groups (feedback and non-feedback) as a result of fatigue. The main effect for gender was significant ($F_{1,55}$=4.402, p=0.041), where females had lower DP values than males.

**Kinetics**

The fatigue protocol caused changes in two kinetic variables, with respect to the feedback protocol (see table 13 and figures 14-16). A significant fatigue x group interaction was present for ATSF ($F_{1,53}$=6.783, p=0.012) (figure 17), VGRF ($F_{1,55}$=14.259, $p<0.001$) (figure 18), and KEM ($F_{1,53}$=4.367, p=0.041) (figure 19) (see table 14). Bonferroni post-hoc testing (adjusted $\alpha$=0.025) revealed that the non-feedback group experienced increased ATSF (t=-2.731, p=0.011) and VGRF (t=-2.485, p=0.019) post-fatigue, whereas the feedback group had no change in ATSF and a decrease in VGRF (t=3.145, p=0.004) and KEM (t=-2.485, p=0.019) post-fatigue. Thus, it appears that the feedback protocol was effective at preventing the fatigue-related changes in VGRF and ATSF, and even resulted in an improvement in VGRF and KEM, compared to the non-feedback group.

A gender main effect was demonstrated in ATSF ($F_{1,53}$=9.206, p=0.004). Contrary to previous research, males demonstrated higher ATSF than females, despite normalizing to body mass.

**Time-to-peak Kinetics**

The fatigue protocol caused significant changes in both the length of the stance phase, as well as the time-to-peak values (see table 15). A main effect for fatigue was evident in total stance time ($F_{1,55}$=13.135, p=0.001). Fatigue resulted in a significant increase in stance time in both groups. A significant fatigue x group interaction was present for both time-to-peak VGRF ($F_{1,55}$=8.535, p=0.005) and time-to-peak KEM ($F_{1,53}$=5.126, p=0.028). Bonferroni post-hoc testing (adjusted α=0.025) revealed that the feedback group had significantly lower time-to-peak KEM post-fatigue (=-2.870, p=0.008), whereas the non-feedback group had significantly lower time-to-peak VGRF post-fatigue (t=4.193, p<0.001). These results indicate that fatigue increases stance time, but decreased the time-to-peak VGRF in the non-feedback group. The feedback protocol was able to increase the time-to-peak KEM, although there was no change post-fatigue in the non-feedback group.

There was a significant fatigue x gender interaction in time-to-peak ATSF ($F_{1,53}$=4.871, p=0.032). Bonferroni post-hoc testing (adjusted α=0.025) revealed that males had a significant decrease in time-to-peak ATSF post-fatigue (t=2.912, p=0.007), whereas females did not change post-fatigue. There was also a significant main effect for gender in stance phase length ($F_{1,55}$=8.439, p=0.005), where males had a significantly longer stance phase than females. These results suggest that males and females differ in their response to fatigue in regards to time-to-peak ATSF, and that in general males prolong their stance phase when compared to females, regardless of fatigue status.

**Vertical Jump and Motor Skill Test**

The fatigue protocol caused significant changes in vertical jump height and motor skill test performance (see table 16). A fatigue main effect was seen in maximum vertical jump height ($F_{1,55}$=9.064, p=0.004), motor skill test time ($F_{1,55}$=17.607, p<0.001), motor skill test errors ($F_{1,54}$=64.074, p<0.001), and overall motor skill test score ($F_{1,54}$=81.760, p<0.001).

The fatigue protocol caused a decrease in vertical jump height, and an increase in MST time, errors, and overall score.

A gender main effect was also seen for vertical jump height ($F_{1,55}$=92.752, p<0.001). Males had significantly higher vertical jump heights compared to females. A group main effect was also seen for MST errors ($F_{1,54}$=4.592, p=0.037). In this case, the feedback group had significantly lower errors compared to the non-feedback, both pre- and post-fatigue. Thus, the groups did not appear to be equal prior to testing, which indicates the results of this comparison should be viewed with caution.

**Overall**

The results indicate that fatigue creates several significant changes in movement patterns and kinetics, but that the application of a simple feedback protocol can modify many of the changes during an unanticipated side-step cut. Fatigue generally caused more in-phase movement patterns in the sagittal plane, lower variability in movement patterns in the frontal and sagittal plane for the foot-shank and shank-thigh pairs, and increases in ATSF and VGRF. However, application of the feedback protocol resulted in no changes in sagittal plane MARP values, a more in-phase coordination pattern in the thigh-trunk frontal plane pair, no change in ATSF and a decrease in VGRF. Only variability values appeared to be resistant to the feedback protocol. The fatigue protocol also significantly changed physical performance of vertical jump height and agility, indicating a small but significant decrease in performance post-fatigue. However, it is interesting to note that there was only one significant fatigue and gender interaction, which indicates that both genders respond similarly to this fatigue protocol.

## CHAPTER 5

### Discussion

The primary purpose of this study was to examine the effect of a functional fatigue protocol and verbal feedback on movement coordination, variability, and knee kinetics. Using the results of this study, we can draw several conclusions. First, physical fatigue does change movement coordination profiles between the foot and shank, shank and thigh, and thigh and trunk, in both the frontal and sagittal plane. The coordination profiles generally become more in-phase post-fatigue. Second, physical fatigue also decreases movement variability between the foot and shank, and shank and thigh, in the sagittal and frontal planes. Generally subjects exhibited a loss of variability post-fatigue. Third, fatigue caused an increase in anterior tibial shear force (ATSF) and vertical ground reaction force (VGRF). Fourth, the verbal feedback protocol was largely effective at correcting the fatigue-related changes in sagittal plane movement coordination and knee joint kinetics. Additionally, the feedback protocol was also able to change thigh-trunk frontal plane coordination, and decrease knee extension moment (KEM), even though fatigue did not alter these variables. The feedback protocol was not able to modify any of the changes in movement variability. Finally, there were very few gender effects within the data. Main effects were seen for few coordination and variability variables, and there were no differences between genders in the response to fatigue. Thus, gender does not appear to play a role in altering the effects of either fatigue or feedback on movement profiles or kinetics.

This discussion is divided into five sections. The first discusses the effects of fatigue on coordination and variability. The second discusses the effects of fatigue on knee joint kinetics. The third discusses how feedback mediates the effects of fatigue on coordination and variability, and the fourth discusses how feedback mediates the effects of fatigue on knee joint kinetics. The fifth section discusses some of the ancillary data collected for this dissertation (time-to-peak kinetic data, and physical testing data), that was not a part of the formal research questions. Finally, the limitations of the current research design and suggestions for future research are given.

**Fatigue and Coordination and Variability**

Of all of the changes seen in this study, the most powerful and consistent were demonstrated in the movement coordination measure (MARP) used to quantify intersegmental phasing coordination. In the sagittal plane, all three segment pairs (foot-shank, shank-thigh, and thigh-trunk) decreased in value from pre- to post-fatigue in the non-feedback group. This indicated that the fatigue protocol caused the subjects to change the organization of their movement patterns post-fatigue, to cause the segments to move in a more in-phase pattern. This was most evident at the most distal pair (foot-shank), where effect sizes in the non-feedback group were greater than 0.69 and the mean differences from pre- to post-fatigue were over three times greater than the SEM calculated during the prior reliability analysis (see tables 17 and 18). These data suggest that the change in foot-shank sagittal plane MARP is not likely due to measurement error or variability within the subject pool. The effect sizes for the shank-thigh sagittal plane MARP were above 0.46 and the mean differences were all greater than three times the SEM calculated during the reliability analysis, again suggesting that these changes were strong and not due to error or chance. For the thigh-trunk sagittal plane pair, effect sizes for the non-feedback group were greater than 0.47 and at least twice the SEM. This all suggests that the more in-phase coordination profiles seen in the sagittal plane are strongest distally, and all are most likely due to actual alterations of movement organization due to fatigue.

This change to a more in-phase pattern likely indicates that the subjects needed to change the way they organized their movement in order to complete the cutting task with a new constraint to the system. This functional fatigue protocol produces significant loss of voluntary torque production in at least one major muscle of the lower extremity (quadriceps), as well as losses in muscular power (as measured by vertical jump) and central drive to produce force. These changes should constitute a significant new constraint to the neuromuscular system. Dynamic systems theory suggests that this change in constraints should alter the way the subjects organize and create their movement, reflecting this change in muscular functioning.[36, 37, 164] The results of this study suggest that fatiguing the neuromuscular system causes movements between segments to become more in-phase, indicating a new pattern for this select movement.

The change to a more in-phase pattern may indicate a general “stiffening” of the lower extremity in response to the muscular fatigue. Prior studies have demonstrated that after certain fatigue protocols, joint flexion decreases and ground reaction forces increase.[42, 153, 158] This likely indicates that the neuromuscular system responds to the loss of muscular strength and power by “freezing” the movement of the lower extremity, limiting the available motion of the joints in order to preserve movement within a set range of motion that can still be controlled by the compromised neuromuscular system. A more in-phase movement indicates that the two segments are moving in generally the same direction at the same velocity. However, under normal circumstances (pre-fatigue), the movement is more out-of-phase (segments moving in opposite directions). If the neuromuscular system attempts to limit the motion of one or more segments in response to fatigue, the segments would flex less and move together in a stiffer pattern, one that would be more in-phase.

The hypothesis that fatigue would cause the system to move more stiffly is also supported by the variability data. The movement variability of the foot-shank and shank-thigh pairs decreased post-fatigue in all subjects. This loss of variability indicates that the pattern of movement is more stable, but also indicates that the system has lost some ability to respond to external perturbations.[36, 167] This is analogous to "freezing" the available degrees of freedom in the system in order to produce the required movement. If there is a new constraint, the subject may respond by limiting movement, and by limiting the amount of variability in that movement, so that the subject can produce the required action. This may explain both the change to a more in-phase pattern and the loss of variability.

The loss of movement variability is also most pronounced in the distal segments, as was the change in coordination. Effect sizes were primarily moderate, and generally the 95% CI's did not overlap for the foot-shank segment pairings, and overlapped very little for the shank-thigh (see tables 19 and 20). Thus, the effects seemed to be strongest between the foot-shank. However, there were no significant changes at the most proximal pairing (thigh-trunk). This may indicate that this movement organizes itself, and thus attempts to control itself, from the "bottom-up". In response to fatigue and the increase in VGRF, the neuromuscular system limits the most distal segment first, in an attempt to prevent the forces from being transmitted further up the kinetic chain. Changes to a more distal-dominant strategy have been demonstrated in prior research.[186] However, the more pronounced changes in distal segments may be due to higher levels of muscular fatigue in distal musculature, or the proximal segments may have additional musculature available to compensate for fatigued muscles. The amount of fatigue experienced by different muscles in the lower extremity is an area of future research, as is the neuromuscular strategy to compensate for fatigued musculature in the lower extremity.

The alterations in frontal plane coordination, as well as variability, are not as pronounced as the changes in sagittal plane pairings, but exhibit many of the same changes. Foot-shank frontal plane MARP values decrease post-fatigue, indicating a more in-phase movement pattern. However, there were no other significant changes due to fatigue in frontal plane movement patterns. Frontal plane shank-thigh coordination exhibited a significant interaction, but this change was not great enough to elicit differences during post-hoc testing. This may have been due to some under-powering of the comparison, but the change seen was relatively small and there was significant overlap in all of the 95% CI's, indicating that this change may not be clinically relevant. Frontal plane variability decreased between the foot-shank and shank-thigh, and effect sizes were moderate to large. Thus, although the coordination patterns were largely unchanged, the neuromuscular system still increased the stability of the patterns in response to fatigue.

These alterations in coordination and variability are generally similar to prior research examining different movement strategies and control in different populations and situations. Stergiou's work examining obstacle height found that increasing the height of an obstacle to be cleared during gait caused sagittal plane segment coordination to become more in-phase, similar to the effect seen in this study.[161, 162] The authors had suggested that increasing the obstacle height also increased VGRF upon landing, which may have driven the phasing changes observed. This also agrees with this study, where increased VGRF was seen post-fatigue along with the alterations in MARP. Similar conclusions regarding increased VGRF and in-phase segment coordination were suggested in the study of ACL-reconstructed patients by Kurz.[39] ACL-reconstructed patients had a more in-phase relationship during walking between the shank and thigh, and between the foot and shank during running. Again, this coincided with higher VGRF. These results all seem to agree that changes in segment coordination patterns occur in the presence of increased VGRF. However, it is still unclear if this is a cause-effect relationship, or merely a correlation.

The decrease in variability seen in this study is also in line with prior research. Work by Pollard et al. suggested that women exhibited lower variability than men during an unanticipated cutting task.[167] Since women are at greater risk of ACL injury than men, the authors concluded that lower variability may be a potential risk factor. Hamill and colleagues also found that individuals with PFPS displayed lower variability than healthy controls.[38] In both studies, the authors felt that lower variability may play a role in the development of injury. This study found that fatigue, a condition that has been associated with increased risk of ACL injury, decreases variability in club athletes. Thus, this study furthers the evidence that decreased variability may be associated with the development of lower extremity injury.

**Fatigue and Kinetics**

The fatigue protocol caused significant changes in two kinetic variables associated with ACL injury: ATSF and VGRF. Subjects in the non-feedback group experienced a significant increase in both forces with moderate effect sizes (0.38-0.78) (see table 21). There was some overlap in the 95% CI's, but this overlap was small between the means with significant changes. Thus, the changes due to fatigue appear to be clinically relevant and not due simply to variability within the subject pool.

The results of this study agree with some of the prior research on fatigue's effects on lower extremity kinetics. Chappell found that his fatigue protocol caused a significant increase in ATSF, and Wikstrom found an increase in VGRF.[42, 158] These two studies employed an intermittent fatigue protocol that utilized intense activity and stretch-shortening cycle activity, which closely matches the types of activity used in this protocol. The values obtained in this study are within the ranges expressed in the Chappell study, and the VGRF is slightly lower than the Wikstrom study. The differences may be due to the different landings employed by these two studies and the current research, but there do not appear to be major differences between our results and theirs.

The increase in VGRF and ATSF likely represents a stiffer landing strategy and an inability for the neuromuscular system to absorb the force of landing post-fatigue. This is also supported by the effects on time-to-peak VGRF and ATSF, which both indicate that peak forces are reached much sooner during the stance phase post-fatigue. Since this fatigue protocol does produce a significant loss of volitional muscular force, it is reasonable to believe that this loss of force-producing capability would have a negative impact upon the ability to eccentrically control the movement of the joints during landing, as well as allow the limb to flex in response to an apparent increase in rate-of-loading post-fatigue. The results of this study suggest that this type of fatigue increase two forces that have been associated with loading of the ACL, and prospectively VGRF is associated with ACL injury, and that the rate of loading for these two forces is likely higher as well.[15, 34]

However, this fatigue protocol did not alter KEM or KVM, two variables that are also associated with ACL strain. Prior fatigue research has demonstrated increased KVM post-fatigue.[42, 43] However, the male subjects in this study appear to have higher knee valgus moments than males in prior studies.[27, 32, 42, 43] This may have influenced the results of this study. If the non-feedback group was already at a high level of KVM pre-fatigue, the subjects may not have been able to tolerate even higher levels of KVM without injury. This is especially true given the higher levels of ATSF in our male subjects compared to females, which may have hampered the response of the male subjects. There was a nearly significant ($p=0.052$) fatigue by gender interaction for KVM, although this appears to be driven more by differences between the feedback and non-feedback groups, and less by the actual fatigue changes between gender. Thus, differences in the subject pool's demographics may have played a role in this non-significant result.

A second reason that KVM may not have been altered in this study, when compared to prior studies, is the task used to evaluate lower extremity movement. This study employed a difficult single-leg landing, as well as an unanticipated change in direction, which may have "maximized" the loads in the frontal plane pre-fatigue. This may explain why the KVM measured in this study appears to be somewhat higher than in previous studies. If this task was highly challenging, then the subjects may not have been able to control the movement sufficiently pre-fatigue. Again, if the values were particularly high prior to fatigue, the body may have compensated by attempting to prevent any further strain in the frontal plane post-fatigue. This compensation may have been to stiffen the extremity, a strategy that may prevent changes in the frontal plane but would create changes in the sagittal plane. This matches the results of our study, further supporting the hypothesis that the neuromuscular response to fatigue during a challenging task may be to increase the stiffness of the system.

The lack of effect on KEM, despite changes in VGRF and ATSF, is unexpected. Although prior research has not shown a significant effect of fatigue on KEM,[42, 43] it has been strongly associated with both VGRF and ATSF.[28, 32] However, in this study KEM remained statistically unchanged post-fatigue. Part of this may have been due to relatively large 95% CI's and standard deviations, indicating a rather large range of values in all subjects. Effect sizes for fatigue were also very low, although this is likely due to the high variability as well. Finally, the KEM seen in this study does appear to be higher in males than in previous research.[32, 42, 44] These all may have played a role in this non-significant result.

**Feedback and Coordination and Variability**

While fatigue created several changes in movement coordination in the lower extremity, the application of a verbal feedback protocol post-fatigue altered these changes in all of the sagittal plane segment pairings. While subjects in the non-feedback group (whom did not receive the feedback post-fatigue) exhibited a more in-phase movement pattern, the subjects in the feedback group maintained their pre-fatigue coordination profile. This suggests that, although fatigue is a powerful constraint to the neuromuscular system, verbal feedback may be a simple way to counteract this constraint and allow athletes to maintain their preferred, pre-fatigue movement strategies.

The fact that a cognitive intervention was effective at correcting many of the coordination changes seen post-fatigue is intriguing, especially given the level of central fatigue exhibited as a result of this fatigue protocol. The pilot work associated with this study suggested a 10% loss of central drive post-fatigue in the quadriceps. However, that data cannot help determine where this central fatigue exists in the neuromuscular system (is it supraspinal, or reflex inhibition?). The results of this study may indicate that, while significant central fatigue exists, this fatigue does not alter the cognitive ability of the subjects or their ability to consciously control movement. This does agree with prior research that has not shown any definitive decline in cognitive ability after fatigue.[142] Thus, utilizing cognitive interventions after this type of fatiguing exercise may be an important method for preventing fatigue-related changes in movement.

The ability for feedback to correct fatigue-related changes in movement coordination was not as pronounced in the frontal plane segment pairings. Foot-shank frontal plane MARP did not exhibit an interaction between the two groups, indicating that both the non-feedback and feedback groups became more in-phase post-fatigue. This may be a limitation of the current feedback protocol, which may have prompted subjects to be most concerned with sagittal plane motions and not frontal plane movement. Additionally, the feedback group demonstrated a more in-phase coordination pattern between the thigh and trunk in the frontal plane, a pairing that was unaffected by fatigue in the non-feedback group. This suggests that this feedback protocol is not only effective at correcting fatigue-related changes, but can also alter movement patterns that are not affected by this fatigue protocol. Since no subject received this feedback protocol pre-fatigue, it is unclear how this feedback protocol could affect movement patterns alone. However, this finding between the thigh and trunk does suggest that this feedback protocol may also be effective at changing some movement coordination patterns without the presence of fatigue.

The more in-phase relationship between the thigh and trunk has some potential implications on injury risk. The change to a more in-phase pattern indicates that these two segments are moving in a similar direction compared to pre-feedback. This may help prevent high levels of frontal plane excursion, where the trunk and thigh are moving in opposite directions producing a high amount of trunk lateral flexion and/or thigh abduction. Both of these postures are associated with higher levels of joint loading compared to a more neutral movement.[124] Keeping the thigh and trunk moving in similar directions in the frontal plane may promote a more neutral alignment, which may help protect the ACL during lateral movement by keeping the body's center of mass closer to the knee joint.[126, 187]

Despite the ability of the feedback protocol to alter changes in coordination post-fatigue, movement variability was resistant to the feedback. Both the feedback and non-feedback groups exhibited the same decrease in variability post-fatigue in the foot-shank and shank-thigh pairs, although the shank-thigh sagittal plane pair approached a significant interaction ($p=0.072$). Although this suggests that the changes in variability are more robust to feedback post-fatigue, this may also be a consequence of the feedback protocol. The subjects were told to land in a specific way and to "focus" on landing in a certain manner. This may have constrained the subjects, and caused them to reduce variability in order to stabilize the movement in the requested manner. Since the results of the feedback protocol confirm that subjects were able to change movement patterns short-term, another short-term consequence may be a reduction in variability in order to control the change. Future examinations of the effects of pre-fatigue feedback on variability should be conducted.

**Feedback and Kinetics**

The effects of feedback post-fatigue were relatively pronounced in the kinetics of the lower extremity. VGRF was actually decreased from pre-fatigue levels in the feedback group, compared to a significant increase in the non-feedback group. Of all of the variables studied in this research project, this is the only one where the non-feedback group demonstrated a significant impairment, while the feedback group improved, from pre-fatigue levels. This suggests that VGRF is likely the easiest variable changed via feedback. This agrees with most prior research that has also shown that feedback can dramatically improve VGRF.[48-50, 176, 177] This study adds to this knowledge by illustrating that this effect is robust in the face of muscular fatigue. This may partially be due to the instructions given to the subjects, as “land softly” was the first instruction given and directly impacts VGRF. However, since VGRF is one of the factors prospectively confirmed as a characteristic of ACL-injured athletes, this result is potentially important for injury prevention.[34] It is also interesting to note that the feedback protocol retained the same time-to-peak VGRF from pre- to post-fatigue, while the non-feedback group decreased the time-to-peak VGRF substantially. This indicates that not only can the feedback protocol decrease force, but also protect against the change in rate of loading that is suggested by the change in time-to-peak values.

While prior studies have shown that VGRF is affected by the application of verbal feedback, the change in KEM and the preservation of ATSF at pre-fatigue levels is a new finding. Subjects in the feedback group did not demonstrate the increase in ATSF that the non-feedback group did, indicating that the feedback protocol was effective at ameliorating this change acutely. This result may be partially the result of the decrease in VGRF, given their strong relationship.[28, 32] The lack of change in ATSF may also be due to the improvement in KEM seen only in the feedback group. The decrease in KEM and VGRF seen post-feedback may have allowed ATSF to remain at pre-fatigue levels.

The lack of increase in ATSF has direct implications on ACL injury prevention. ATSF is the most direct loading mechanism on the ACL.[14, 15] Any method that can either reduce ATSF, or prevent its increase during high-risk situations, could be an important method for preventing injury. This is the first study that has demonstrated that feedback could influence ATSF, and thus further suggests that verbal feedback should be a part of ACL injury prevention programs, particularly as an acute method for altering joint forces. The only kinetic variable that was seemingly unaffected by feedback was KVM. This knee joint moment has been prospectively shown to predict ACL injury in female athletes,[34] but was unaffected by both fatigue and feedback in this study. There may be a few reasons for this lack of effect. First, as previously mentioned, the KVM seen in this study was somewhat higher than in previous studies. The task used in this study may have influenced this, as well as the non-significant gender effect. Prior studies that have examined KVM as a prospective risk factor have utilized a double-leg jump landing, which is likely less challenging than the single-leg unanticipated cut used in this study. A second reason for this non-significant result could have been the point at which KVM was calculated. The peak moment measured during the first 40% of the stance phase may have missed a reduction in KVM at the very onset of the landing phase, where KEM, VGRF, and ATSF are highest. Perhaps the KVM measured at this point is more important than the peak measured later in the stance phase. The importance of peak KVM vs. the peak measured very early on during the peak forces at the knee should be studied further.

**Ancillary Data**

In addition to the coordination, variability, and kinetic data collected to answer the research questions, some additional data was collected in order to study other effects of fatigue and feedback on movement and athletic performance. Time variables (stance length and time-to-peak) were calculated to examine how fatigue and feedback influence both the time available to respond to peak forces, but also the potential effect on the rate of loading (the combination of both peak force and time to reach that force, or time-to-peak). This study found that fatigue causes subjects to increase the amount of time spend in the stance phase of the cut, but that did not correspond to increased times-to-peak force and moment. Instead, time-to-peak VGRF and ATSF both decreased significantly as a result of fatigue. This suggests that subjects experience peak loads which increase strain on the ACL much faster post-fatigue. This has important implications for injury risk, as a shorter time-to-peak force may prevent the subject from contracting antagonist muscles (such as the hamstrings) to protect against increased ACL strain. Studies have shown that muscle latency in response to perturbation is at least 57ms, which is longer than the time-to-peak VGRF, and over twice as long as the post-fatigue time-to-peak ATSF..[146, 188, 189] These reflex latencies also appear to increase post-fatigue.[146] This means that these forces, particularly ATSF, increase substantially faster and can become much higher post-fatigue. This also means that the entire load is transmitted to the ACL, and cannot be absorbed by the musculature around the knee. Finally, studies have demonstrated that the rate of strain dramatically increases the stress upon the ACL, which likely places the ligament at greater risk of rupture.[190] Thus, a combination of higher load plus faster rate-of-loading may indicate a worst-case scenario for the ACL.

Feedback appears to be able to maintain time-to-peak VGRF post-fatigue, and can actually increase time-to-peak KEM although there was no change due to fatigue. This may help maintain (or prevent a major increase in) the rate-of-loading in the limb due to higher

VGRF, and when taken in context with the substantial decrease in actual VGRF, appears to help protect the lower extremity from experiencing this force more rapidly. The increase in time-to-peak KEM likely indicates maintenance of the rate-of-moment development (in context with the longer stance period). This may indicate lower reliance on extensor musculature to control the deceleration of the body post-fatigue, or at the very least a change in neuromuscular strategy prolonging the peak in extensor moment in the feedback group.

There are some significant gender effects with this data, particularly with ATSF. Men significantly decreased their post-fatigue time-to-peak ATSF, while women did not. This may be due to the substantial difference in their pre-fatigue values, where men had a longer time-to-peak ATSF, and thus more "room" to drop this value post-fatigue. Females, whom already had a very short time-to-peak ATSF pre-fatigue, simply may not have been able to decrease this without sacrificing the ability to control the movement or the force. However, the fact that men had a longer stance phase means that this change in time-to-peak ATSF was particularly dramatic for males, as it represented a substantial change in the percent of stance where the peak ATSF took place.

These results lend additional support to the conclusion that this fatigue protocol increases the stiffness of landing. Decker suggested that softer landing styles may be associated with an increased time-to-peak knee extensor moment associated with a softer landing in females.[19] Other researchers have demonstrated that stiffer landings produce a more rapid time-to-peak VGRF, which may decrease the ability of the body to absorb and dissipate landing forces.[191, 192] A more rapid time-to-peak forces and moments will decrease the ability of the neuromuscular system to respond to strain on the lower extremity, and may prevent the effective attenuation of injurious forces and moments at the knee.

The changes in both vertical jump and MST suggest that overall athletic performance may be somewhat inhibited post-fatigue, but these changes may not lead to a complete loss of athletic capability. The decrease in vertical jump height was less than ½ inch (just over 1cm), which is less than what has been reported in previous studies.[42, 43] While this change was significant, it is debatable whether it is clinically or functionally relevant. However, the changes in MST were more significant, particularly in the number of errors committed post-fatigue. This may suggest a decline in the ability of the body to control quick, repetitive changes in direction. This may be partially due to the decreased variability in the system exhibited post-fatigue, which may limit the ability of the subject to adapt to the movement changes needed during this task. Thus, the number of errors (relative lack of accuracy) increases, as does the time it takes to successfully complete the task. This finding is somewhat at odds with the Welsh study, which found that subjects maintained the number of errors in the MST post-fatigue but significantly increased the time to complete the task.[183] The authors hypothesized that it was due to their emphasis on foot placement accuracy, which may have led the subjects to focus heavily on that aspect. In this study, both time and accuracy were equally emphasized in the instructions, which may have allowed a larger increase in errors in this study.

These two tasks suggest that some aspects of overall body performance and control are impaired post-fatigue, but that these changes may not signal a complete loss of athletic ability. We believe that this is a strength of this study, as the likelihood of an athlete reaching full fatigue and a complete loss of function during sports is unlikely. Anecdotally, athletes and coaches will report that if an athlete begins to show substantial degradation in performance during athletics, he/she will be removed from the contest. Thus, a level of fatigue that is high, but does not result in significant performance loss, is likely to simulate actual athletic participation better than a fatigue protocol that results in drastic vertical jump height changes, or greater losses in motor skill. This study also suggests that a substantial loss of performance is not necessary to see significant alterations in motor control, movement organization, or joint loading. This matches the conclusion of the Borotikar study, which found significant changes in joint kinematics at 50% fatigue levels.[41] This lends further support that fatigue-related changes are likely to occur rapidly during intermittent, high-intensity exercises, and thus potential injury risk may be elevated relatively quickly during exercise.

**General Conclusions**

The results of this study shed new light on several aspects of ACL injury development, as well as the potential ways to prevent ACL injury. Muscular fatigue that results from an intermittent, functional fatigue protocol does have potentially detrimental effects on movement coordination and variability. This fatigue also causes increases in joint loading forces. However, a simple verbal feedback protocol can offset most of these changes, allowing subjects to maintain pre-injury movement patterns and joint loads. We believe that the fatigue protocol used in this study is a valid method for inducing the types of fatigue experienced by athletes during intermittent sports, such as soccer and basketball. This is the only study that has examined such a large number of athletes pre- and post-fatigue, which may have allowed for changes in movement to be more clearly determined. And this study is the first to quantify the organization and control of movement in the lower extremity during athletic tasks, both after fatigue and after feedback.

We can use the results of this study to begin to construct a theoretical model describing how intermittent muscular fatigue influences the movement and kinetics of the lower extremity during an unanticipated cutting task, and how feedback influences these results (see figure 20). The intermittent fatigue protocol induces both peripheral and central fatigue within the musculature of the lower extremity. Both forms of fatigue will influence the ability of the muscular system to control movement at lower extremity joints. The loss of volitional muscle force may limit the ability to eccentrically control motion, leading to a decision to stiffen the extremity to allow for successful completion of the cutting movement. Stiffening the joint can be achieved by constraining sagittal plane motion, creating a more in-phase coordination pattern. This also leads to increased VGRF and ATSF. In order to exert control over the movement under fatigued conditions, the body limits the variability in the system. This allows for a more tightly controlled coordination pattern, but also limits the ability to respond to perturbations. The increase in joint loads, the increased stiffness of the system, and the lack of variability may predispose the limb to injury, particularly the ACL.

The feedback intervention likely intervenes at a central processing level. While it is unlikely that peripheral fatigue can be altered via feedback, the mechanisms that produce central fatigue may be more easily altered, particularly if supraspinal mechanisms can be utilized to overcome either the supraspinal centers involved in muscular fatigue, or to overcome reflex inhibition at the spinal cord. The feedback likely guides the subjects to focus attention on two or three specific movements that can prevent a stiffening of the extremity and an increase in joint loading. The specific guidelines provided in the feedback will likely influence the compensations made by the subject, thus future studies should attempt to determine what variables are most important to control, and what feedback is most effective at producing the desired results.

**Limitations**

Any laboratory-based research study has limitations that require consideration when interpreting results. Although every attempt was made to enhance the external validity of the current study (a well-researched fatigue protocol that incorporates many aspects of athletic play, a cutting task that involves an unanticipated change of direction on a single foot), this design may not be completely applicable to on-field situations or injurious movements. Additionally, since healthy athletes were studied, it is inappropriate to make definitive conclusions about actual injury risk with this study. Instead, these results point to areas that may be associated with situations that may place athletes at higher risk. In order to determine actual risk factors, prospective studies must be performed.

Another limitation is the consistent testing order. Every subject performed the pre-testing and the post-testing in the same order (pre-test: Vertical jump, then unanticipated cuts, then motor skill test. Post-test: Motor skill test, unanticipated cuts, then vertical jump). The consistency of this order, particularly during the post-fatigue testing, may have influenced the results of this study. The motor skill test was completed within 90 seconds of the end of the fatigue protocol, the cutting task within 12-15 minutes, and the vertical jump during the final minute. This may have made the changes in the MST more apparent, as less recovery took place prior to this test than either the cutting or the vertical jump. However, during pilot testing the mean decrease in vertical jump was one inch, when vertical jump was tested within 60 seconds of the termination of the fatigue protocol. During this study, where more than 10 minutes elapsed, the mean decrease was ½ inch. Thus, although testing order may influence the results, the actual changes in some variables are not very different than what has been observed with less recovery time.

A third limitation is the pre-fatigue differences in the thigh-trunk MARP data. The independent t-tests indicated that the feedback group was more out-of-phase prior to fatigue than the non-feedback group (see table 5). This does call into question the results for these two variables, as the two groups were not equivalent prior to the fatigue protocol or the delivery of feedback. However, if we view the results simply as change scores (as in table 17), we can see that the two changes due to fatigue and feedback were very different between groups. Although this may be partially due to the difference pre-fatigue, in the sagittal plane the group that theoretically has less "room" to change (the non-feedback group, which had a lower MARP pre-fatigue) exhibited the greater change. Thus, although these differences should lead to some caution in the interpretation of the results, it does not appear to be a major limiting factor in the overall study design.

One additional limitation is the demographics of the subject pool. As stated earlier, this group of subjects did not display many of the between-gender differences in variability or joint kinetics seen in prior studies. This may have been due to differences in training practices by each team, or in differences in sport experience. Fitness was not assessed during this study, thus the male subjects may not have had the same resistance to fatigue as the females (although there was no difference in the number of repetitions completed by either sex). However, the difficulty of the task may have caused both males and females to display similar movement patterns. Many of the between-sex difference studies have used double-leg landings or anticipated cutting. The relative difficulty of this task may have caused both sexes enough difficulty that the differences normally present were ameliorated.

A final limitation concerned the elapsed time between the end of the fatigue protocol and the delivery of the feedback. After the fatigue protocol ended, approximately 5-7 minutes elapsed before the feedback was delivered. This time may have allowed for substantial recovery to occur. An analysis of the RPE values between the end of the fatigue protocol and the first post-fatigue measurement (which is approximately the same time that the feedback was delivered) indicates that a significant amount of recovery did occur (from almost 20 RPE to just over 12.5). Thus, the delivery of feedback immediately following exercise, when RPE values are still high, may not produce the same results as this study. Due to the design of the study and limitations of the data collection apparatus, testing the immediate effects of feedback was not possible. However, future research should attempt to see if feedback can be effective at altering movement patterns during fatigue, and immediately after fatiguing exercise.

## Future Research Directions

There are a variety of areas that warrant future research as a result of this (and other) fatigue and feedback studies. First, attempts should be made to study the actual level and type of muscular fatigue experienced during athletic play, particularly in sports at high risk of injury. Once the fatigue of athletic play is quantified, lab-based protocols can be validated to simulate this, so that future studies have more validity. Second, the duration of the effects of fatigue need to be studied in-depth. If the negative changes as a result of fatigue are relatively short-lived, then shorter interventions may be appropriate. However, if effects last a prolonged time, or compound during repeated activity, then interventions and training should be tailored to these changes. Greater understanding of muscular fatigue will lead to more effective studies on possible injury risk factors and ways to intervene during fatigue.

The ability for coordination and variability to assess for possible injury risk factors is an area for considerable research. This is one of the first studies to quantify coordination and variability during a discrete task associated with ACL injury. Thus, additional research should quantify normative values, and prospectively be used to evaluate injury risk in several different types of athletes. Future research should also examine the development of coordination and variability during the lifespan, from childhood to adulthood, during athletic tasks, as well as in injured and uninjured cohorts. These studies will help define how coordination and variability analyses can be used to assess for injury risk, as well as their respective use as potential targets for injury prevention.

Finally, the ability for feedback to be used as an effective prevention program requires some further study. The acute effects of feedback are relatively well-studied and supported, but it is unknown how long these effects last. It is also unknown how a chronic feedback-only intervention program would fare in regards to preventing ACL (or other) injuries. Is technique change an effective way to prevent actual injury? And are the short-term changes due to feedback the same as what may be seen chronically? The dose-response relationship for feedback, as well as the chronic changes possible with technique-only training, need significant attention from researchers before the most effective injury prevention programs can be created.

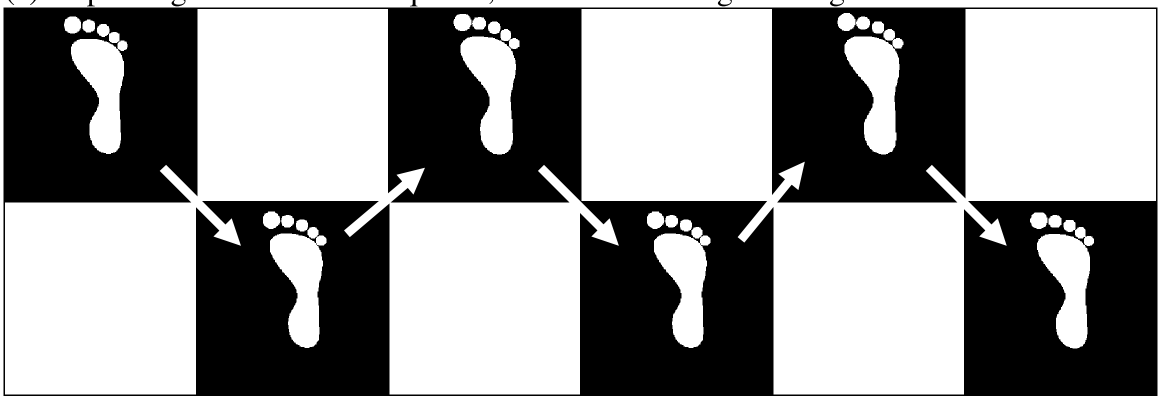
## Conclusion

This study examined how fatigue and verbal feedback alter the coordination, variability, and kinetics of the lower extremity during an unanticipated sidestep cutting task in healthy club sport athletes. The results indicate that fatigue does significantly alter the way movement is organized and controlled, and lead to higher joint loading forces upon landing.

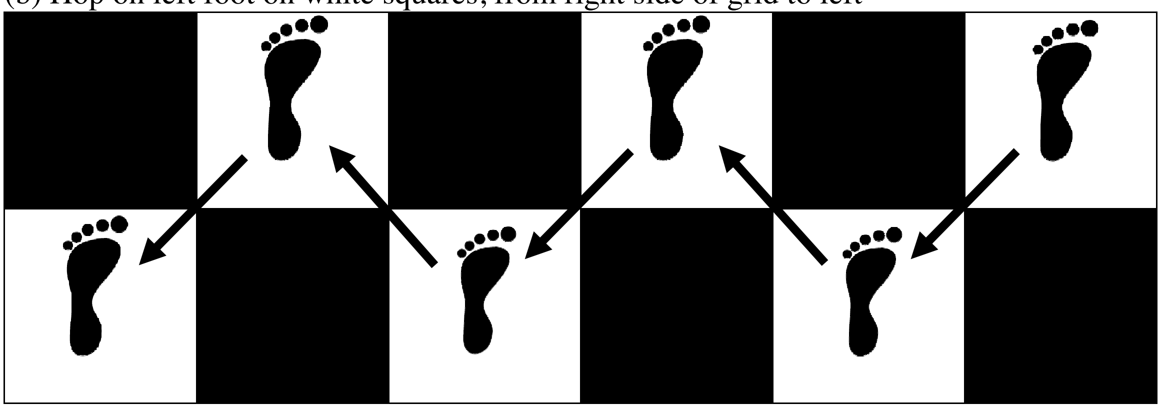
However, verbal feedback appears to acutely correct many of the changes when administered post-fatigue. If ACL injury risk is higher after fatigue, then the differences in coordination, variability, and joint kinetics may be partially responsible. In addition, the verbal feedback protocol may be able to acutely correct these changes, thus possibly be a useful addition to the injury prevention protocols currently in use. Although ACL injury remains a major health problem for athletes, studies such as this help provide further evidence that may be used to prevent this injury, and the long-term sequelae, in our physically active population.

Figure 1. Diagram of the Motor Skill Test (MST)

(a) Hop on right foot in black squares, from left side of grid to right



(b) Hop on left foot on white squares, from right side of grid to left



(c) Split jump with right foot on black squares and left foot on white squares, from left to right side of grid. Switch directions and repeat from right to left side of grid.

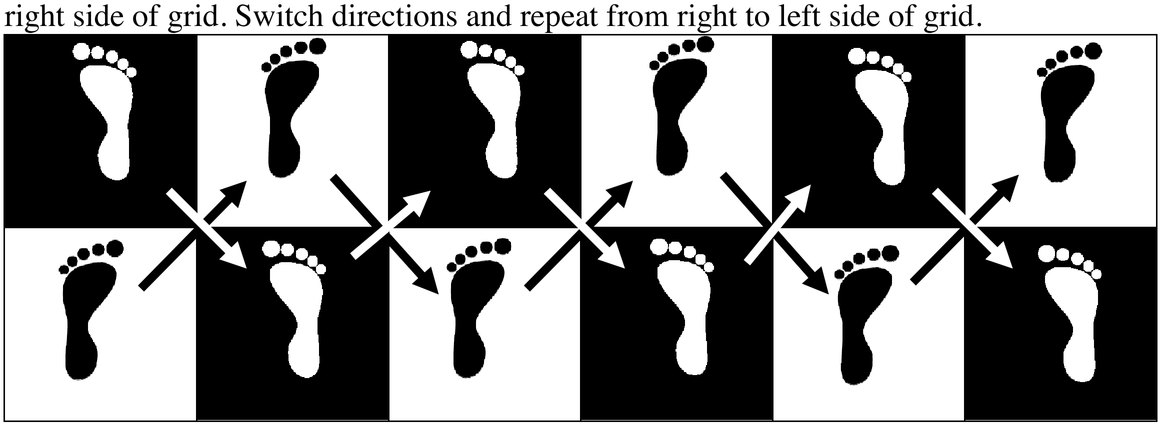


Figure 2. Illustration of the 25-marker markerset used for data collection with the VICON camera system. Lighter circles represent posterior markers (placed on the Calcaneus bilaterally and the L5-S1 joint space).

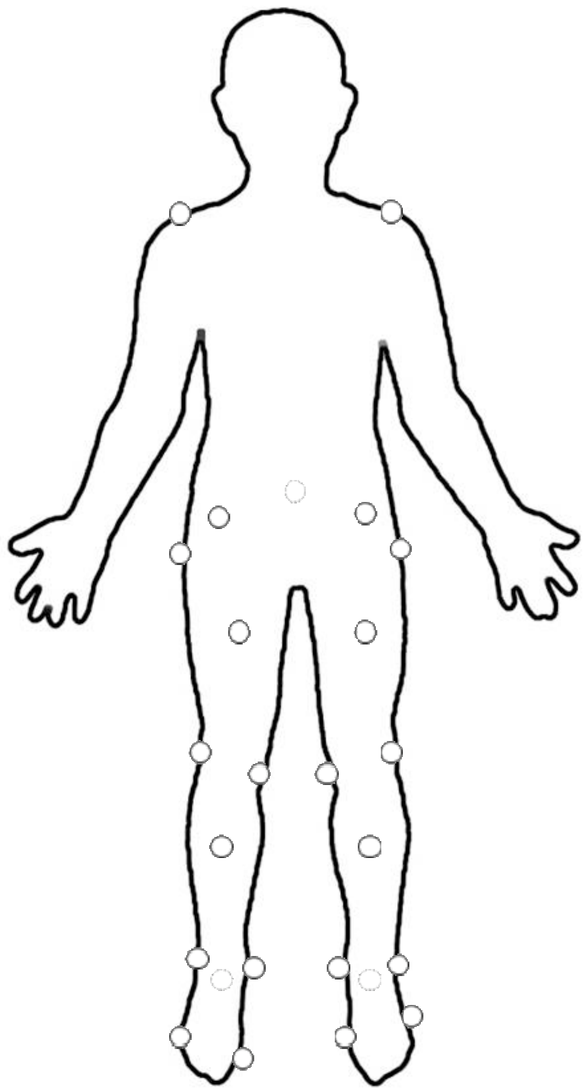


Figure 3. Pictures of the sidestep cutting task. Subjects stand on a line 50% of their body height behind a forceplate (a). They then jump forward over a short hurdle, landing on their dominant leg (b). They then “cut”, or quickly change direction, in the direction indicated on the video screen (screen is behind the photographer) (c).

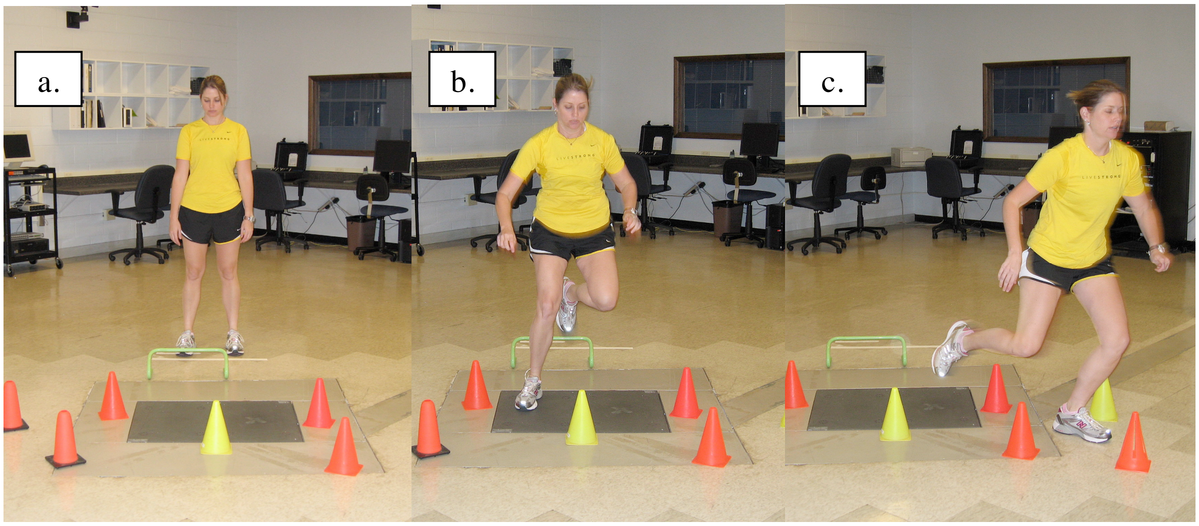


Figure 4. Fatigue protocol course diagram. Cones will be placed on a basketball court marking the course. Subjects will start at cone 1, and run forward around cones 2, 3, and 4 to cone 5. At cone 5, subjects will run backwards to cone 1 via cone 6. Next subjects will side-shuffle around cone 2 to cone 3 and then run forward around cone 6 to cone 5. They will then side-shuffle around cone 4 to cone 3 and then run around cone 6 to finish at cone 1. Five seconds of rest will be given, then the subjects will perform 5 standing broad jumps from cone 1.

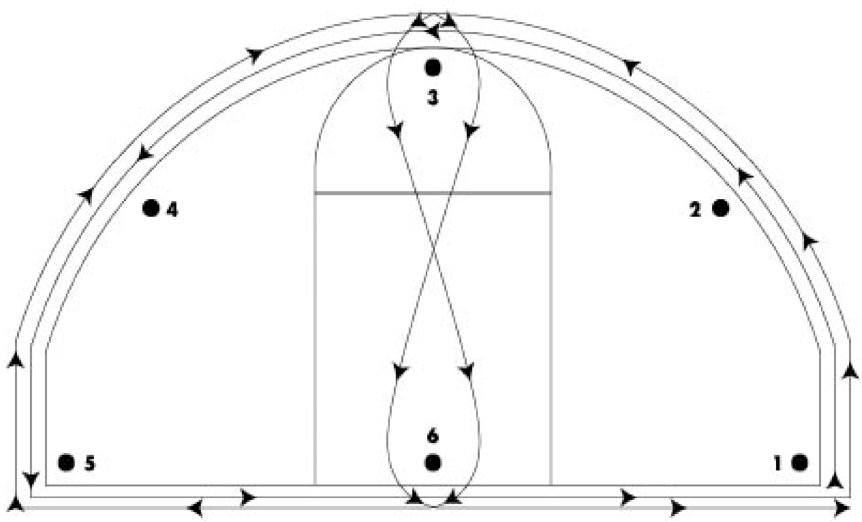


Figure 5. Diagram of the testing protocol.

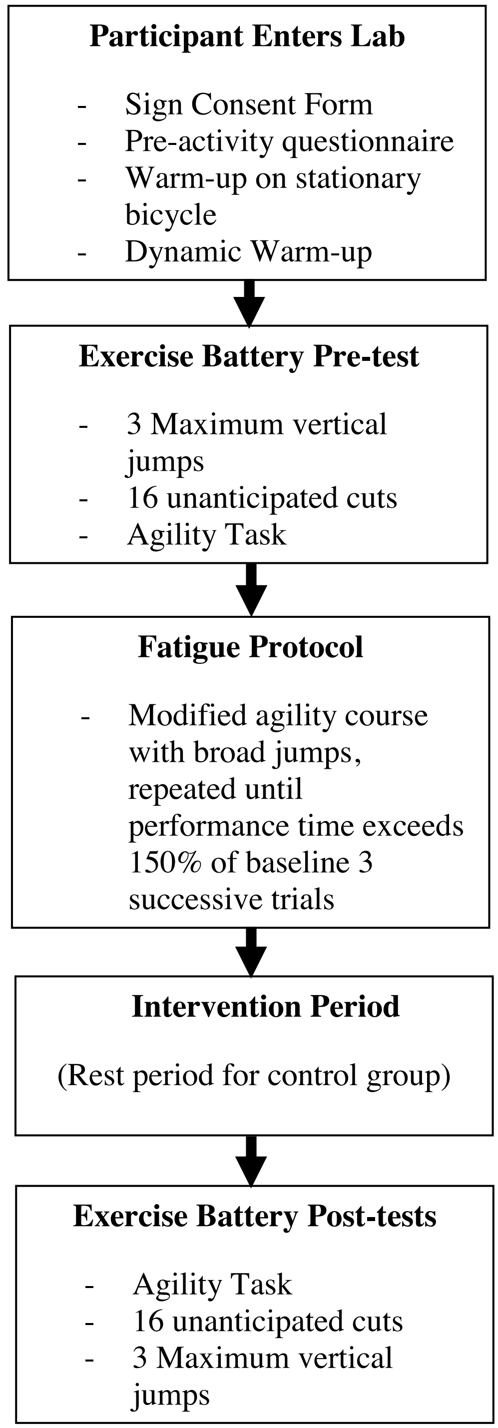
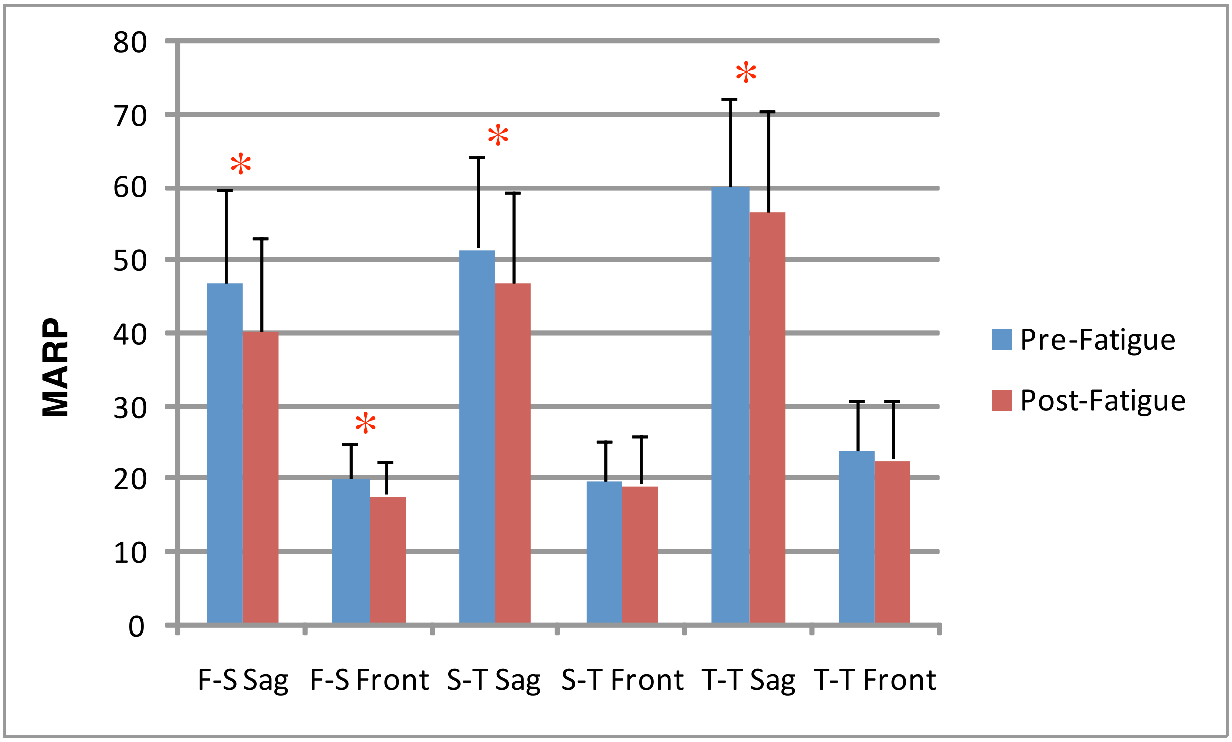


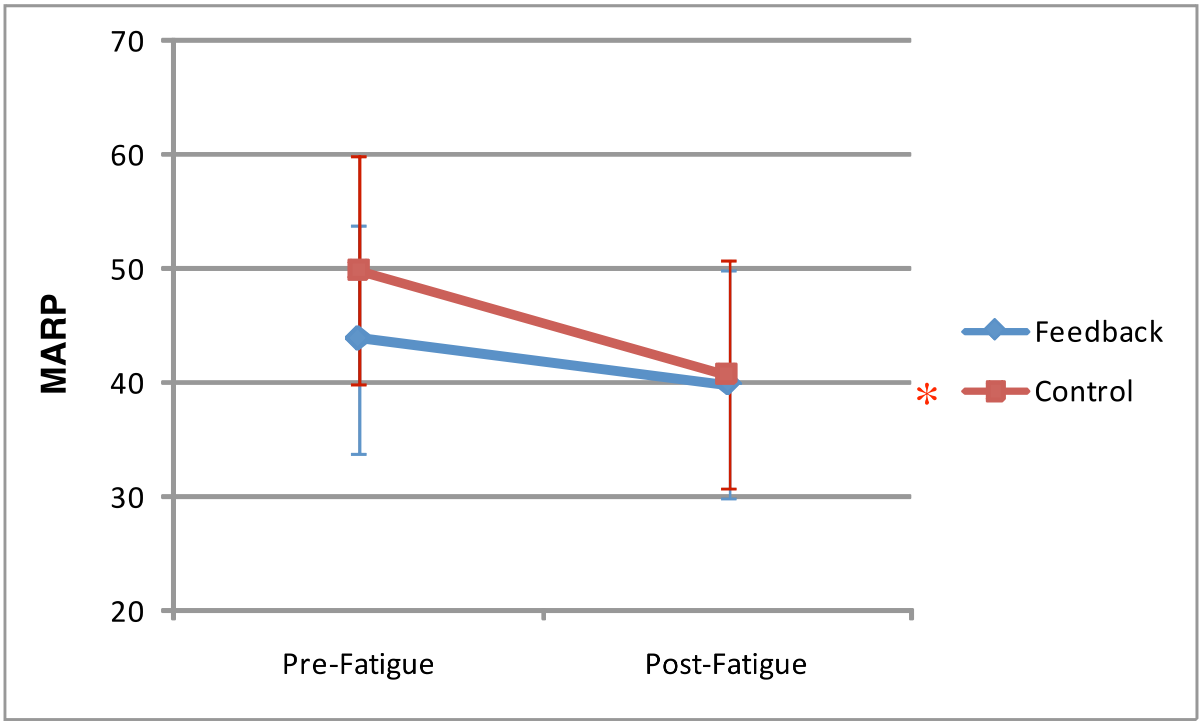
Figure 6. Main Effects for Fatigue on Coordination (MARP) values. Data are presented as means and standard deviation bars.




* Main effect for fatigue ($p<0.05$)

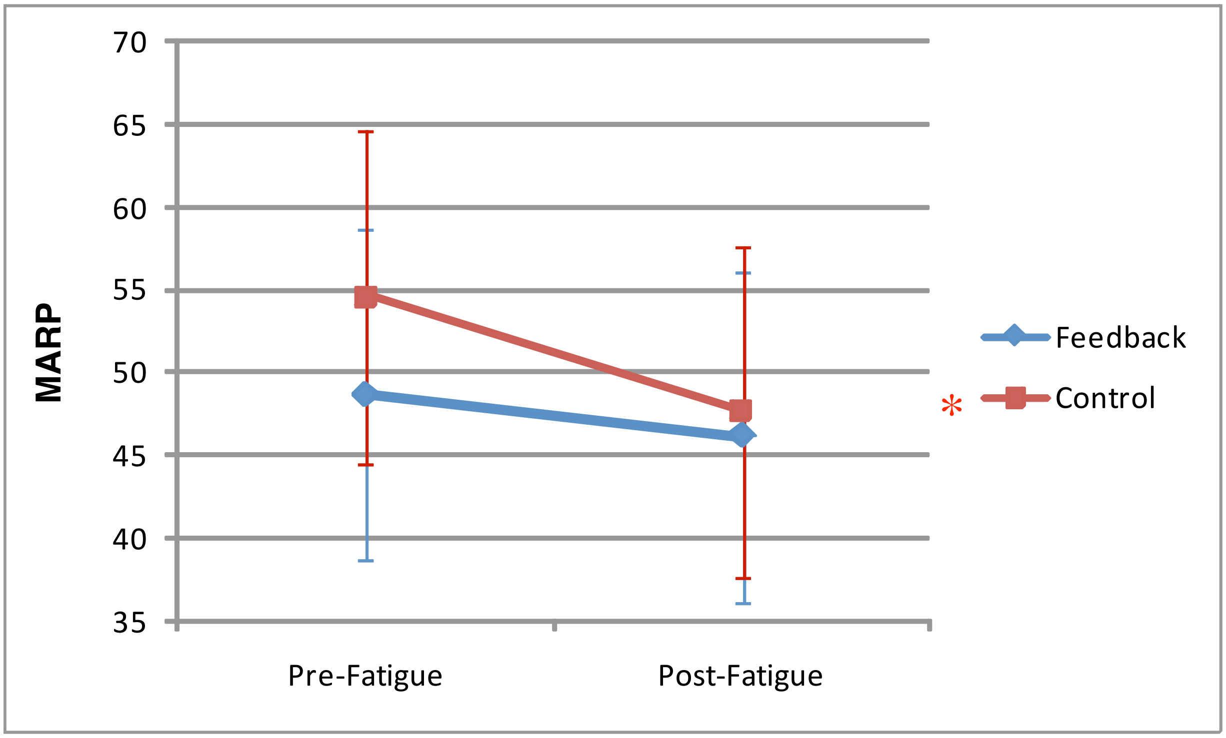
F-S Sag= foot-shank sagittal plane, F-S Front = foot-shank frontal plane, S-T Sag= shank-thigh sagittal plane, S-T Front= shank-thigh frontal plane, T-T Sag= thigh-trunk sagittal plane, T-T Front= thigh-trunk frontal plane

Figure 7. Interaction of fatigue and feedback group on foot-shank sagittal plane coordination (MARP). Data are presented as means and standard deviation bars.



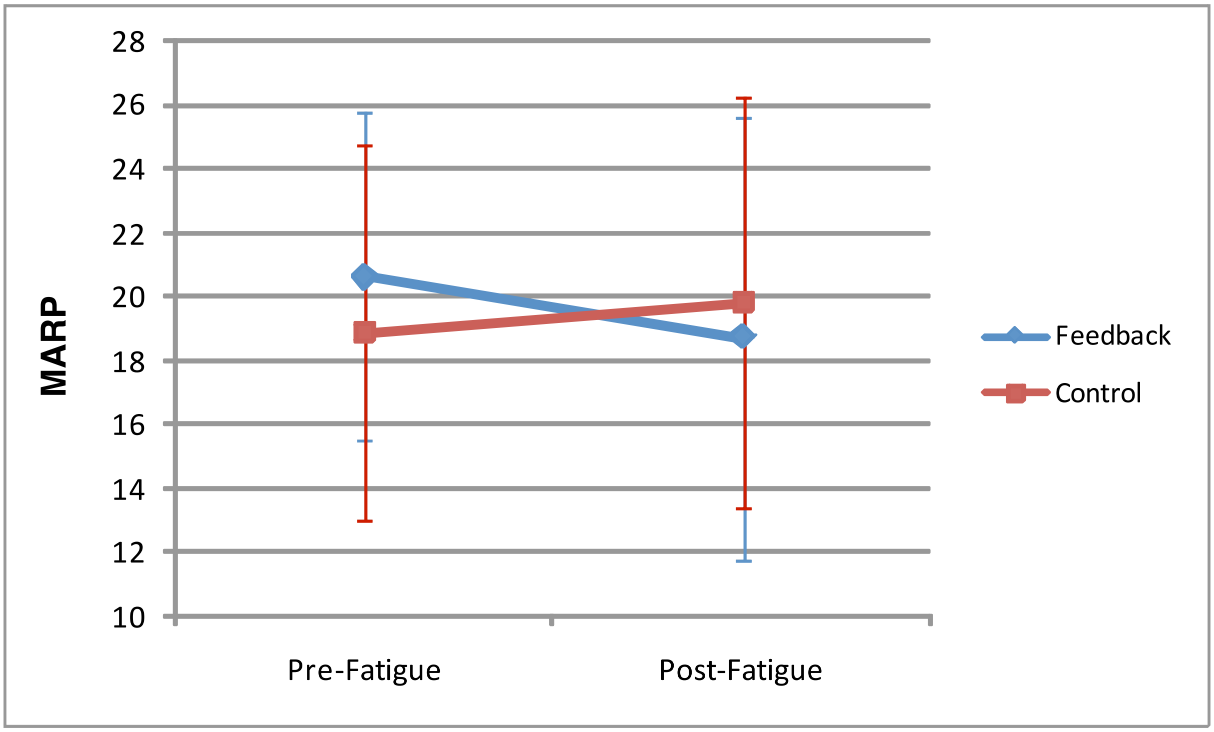

* Interaction effect for fatigue*feedback group (p<0.05). Non-feedback group changed significantly pre- to post-fatigue, but feedback group did not.

Figure 8. Interaction of fatigue and feedback group on shank-thigh sagittal plane coordination (MARP). Data are presented as means and standard deviation bars.



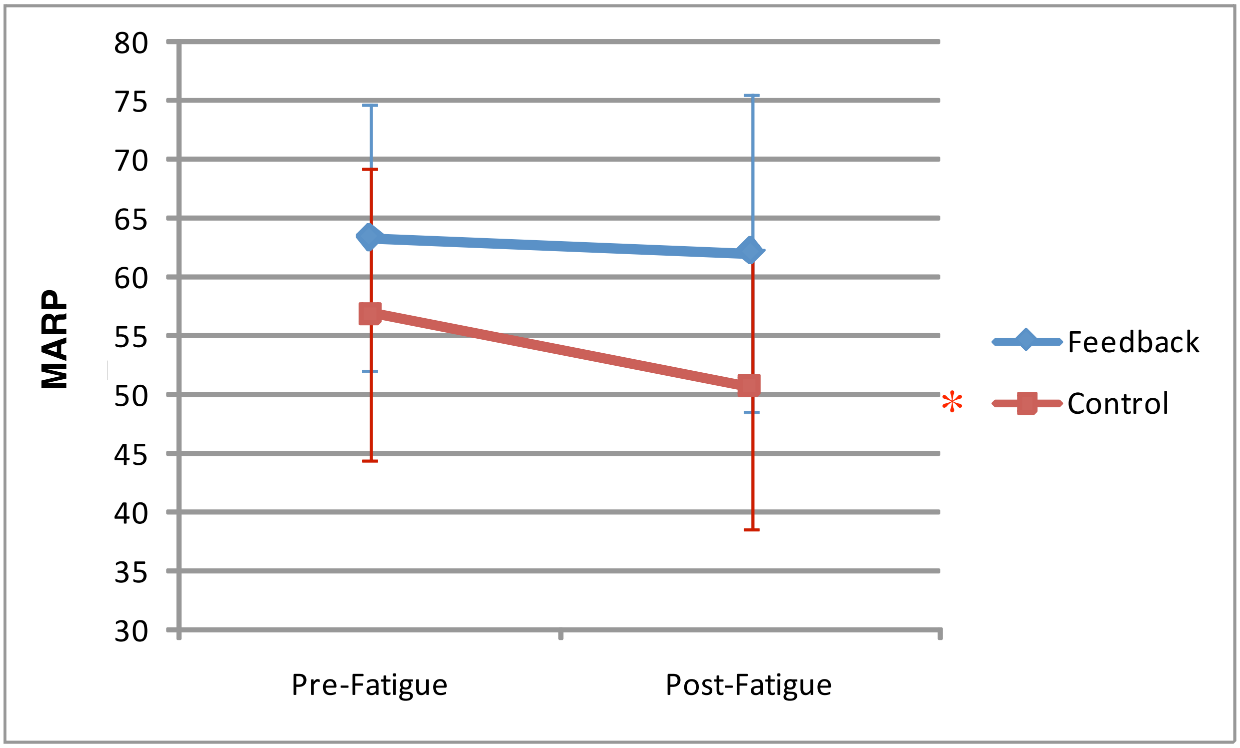

* Interaction effect for fatigue*feedback group ($p<0.05$). Non-feedback group changed significantly pre- to post-fatigue, but feedback group did not.

Figure 9. Interaction of fatigue and feedback group on shank-thigh frontal plane coordination (MARP). Data are presented as means and standard deviation bars.



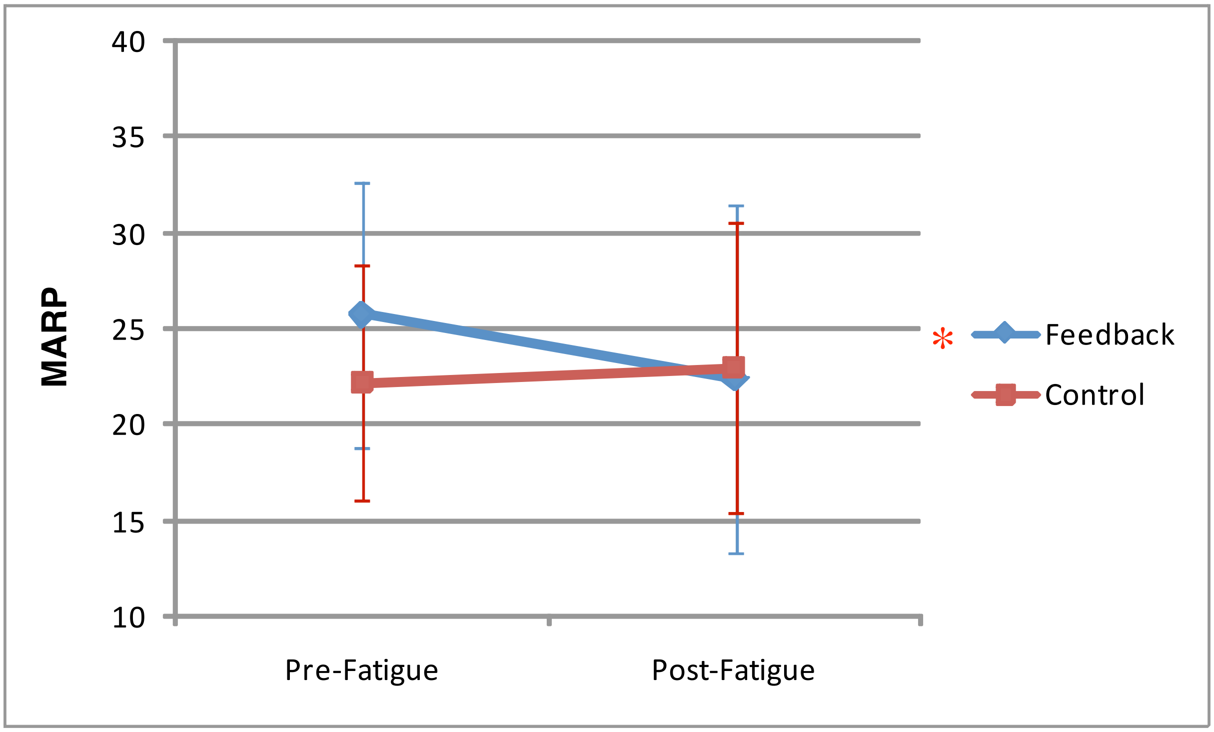

* Interaction effect for fatigue*feedback group (p<0.05). Post-hoc testing did not reveal any significant differences in either feedback or non-feedback groups pre- to post-fatigue.

Figure 10. Interaction of fatigue and feedback group on thigh-trunk sagittal plane coordination (MARP). Data are presented as means and standard deviation bars.



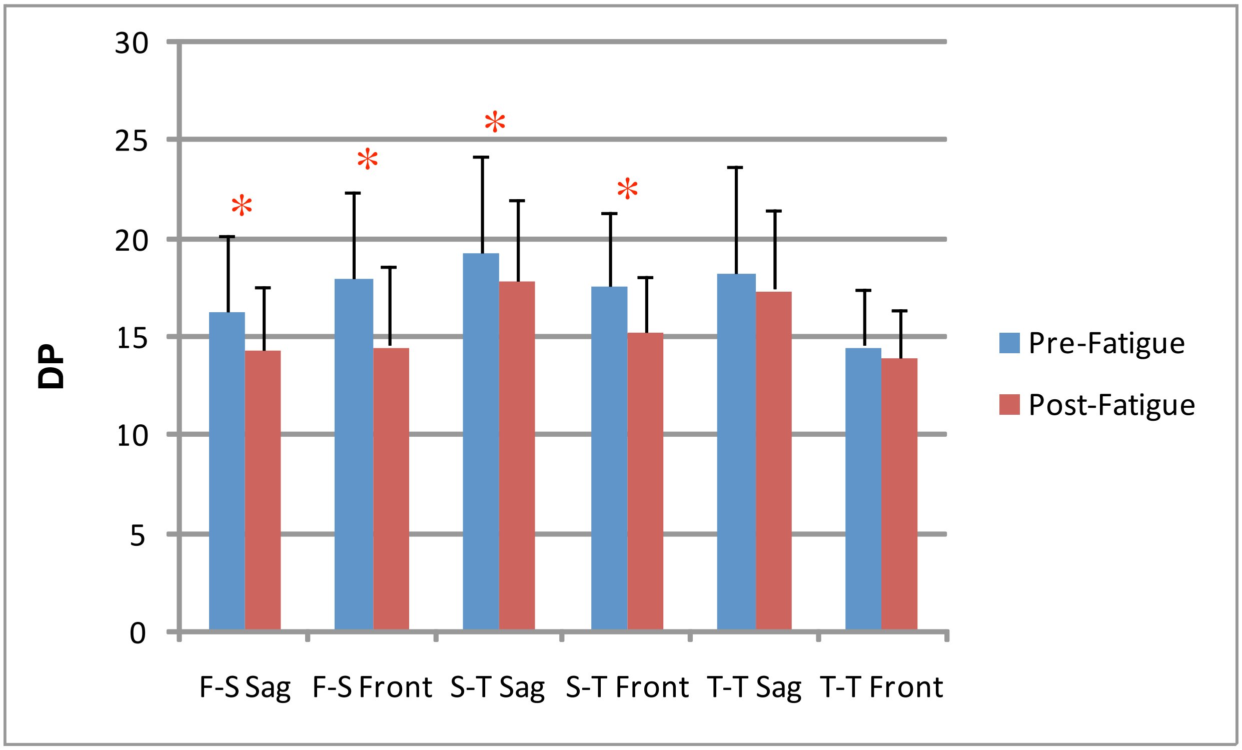

* Interaction effect for fatigue*feedback group (p<0.05). Non-feedback group changed significantly pre- to post-fatigue, but feedback group did not.

Figure 11. Interaction of fatigue and feedback group on thigh-trunk frontal plane coordination (MARP). Data are presented as means and standard deviation bars.




* Interaction effect for fatigue*feedback group (p<0.05). Feedback group changed significantly pre- to post-fatigue, but non-feedback group did not.

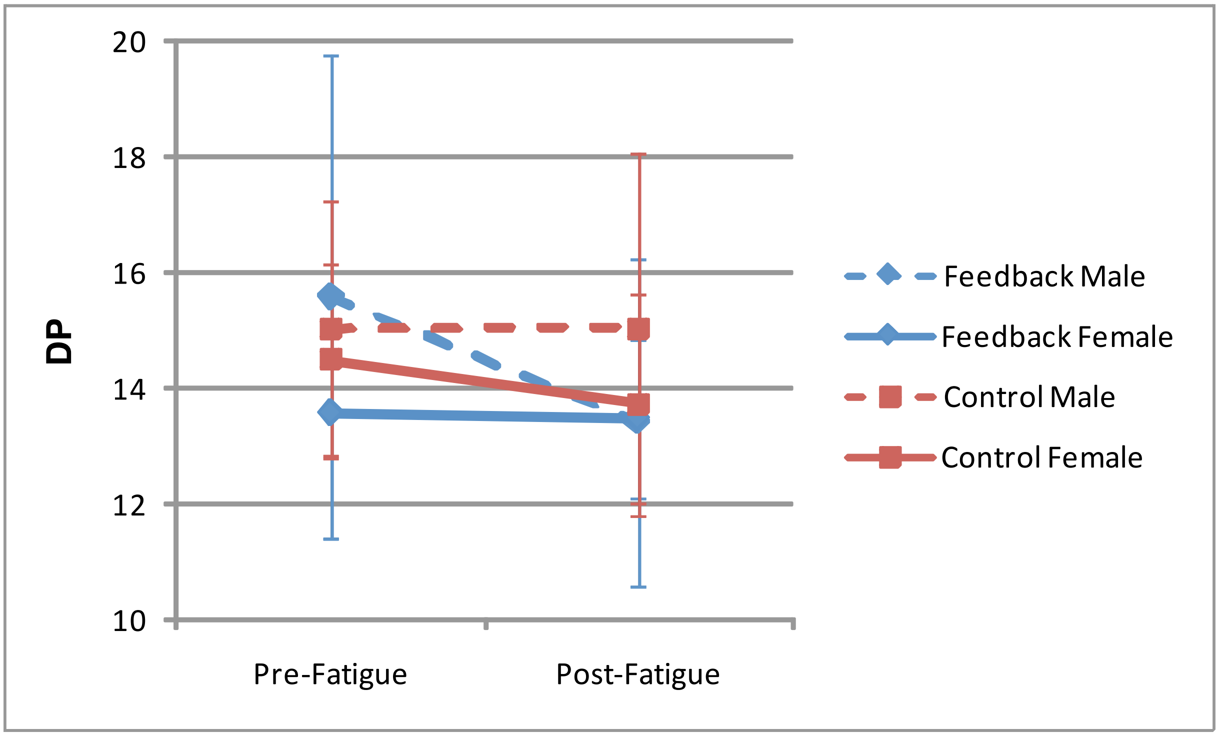
Figure 12. Main Effects for Fatigue on Variability (DP) values. Data are presented as means and standard deviation bars.




* Main effect for fatigue ($p<0.05$)

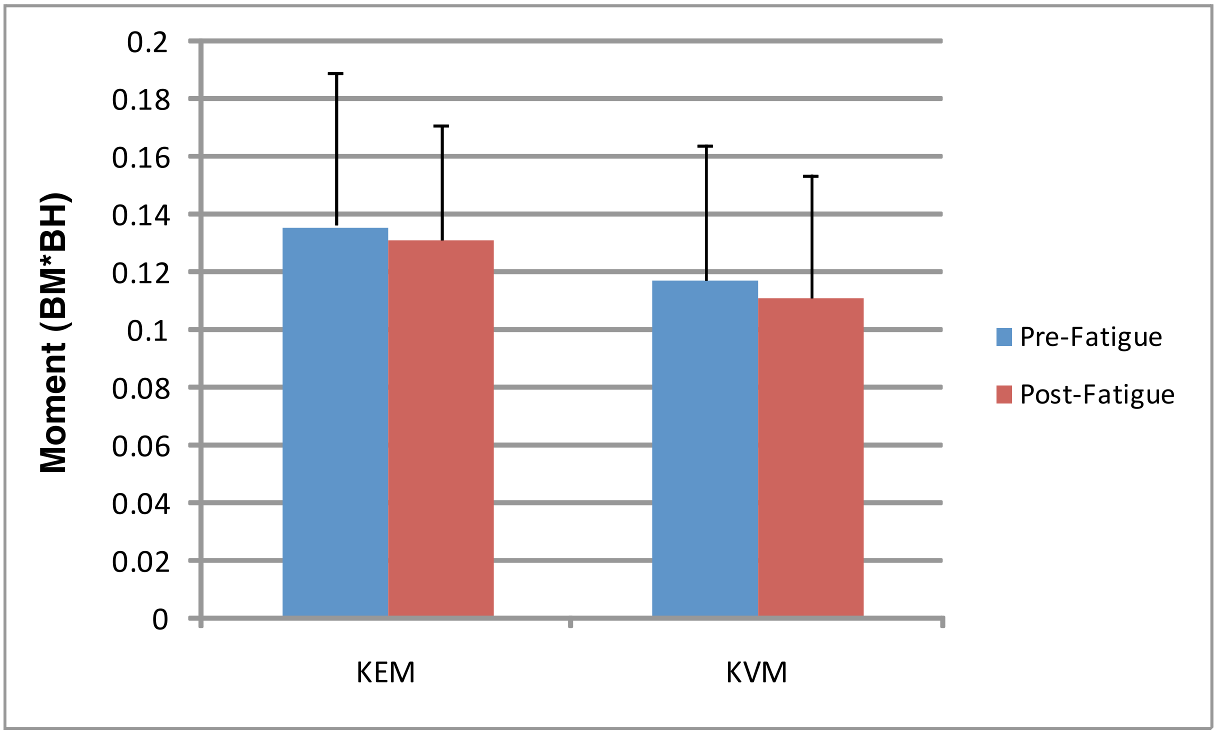
F-S Sag= foot-shank sagittal plane, F-S Front = foot-shank frontal plane, S-T Sag= shank-thigh sagittal plane, S-T Front= shank-thigh frontal plane, T-T Sag= thigh-trunk sagittal plane, T-T Front= thigh-trunk frontal plane

Figure 13. Interaction of fatigue, gender, and feedback group on shank-thigh frontal plane variability (DP). Data are presented as means and standard deviation bars.



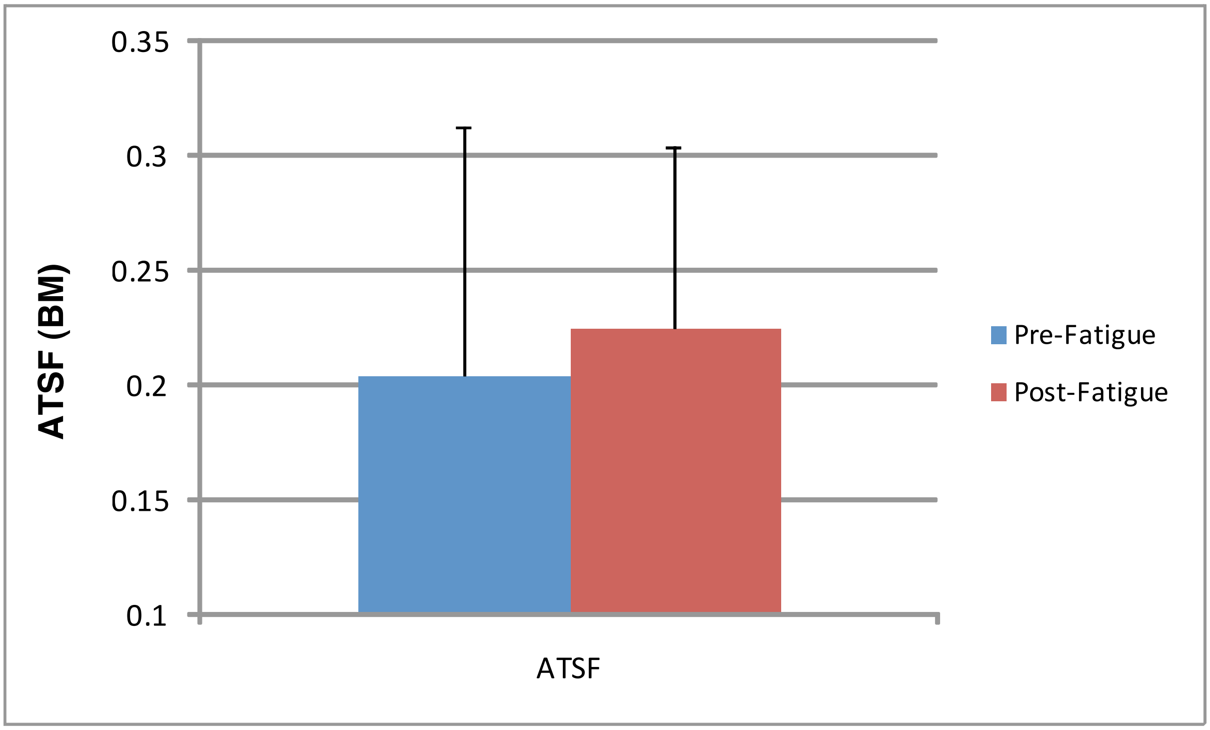

* Interaction effect for fatigue*gender*feedback group (p<0.05). Post-hoc testing revealed no significant differences between any mean.

Figure 14. Main Effects for Fatigue on Moments. Data are presented as means and standard deviation bars.



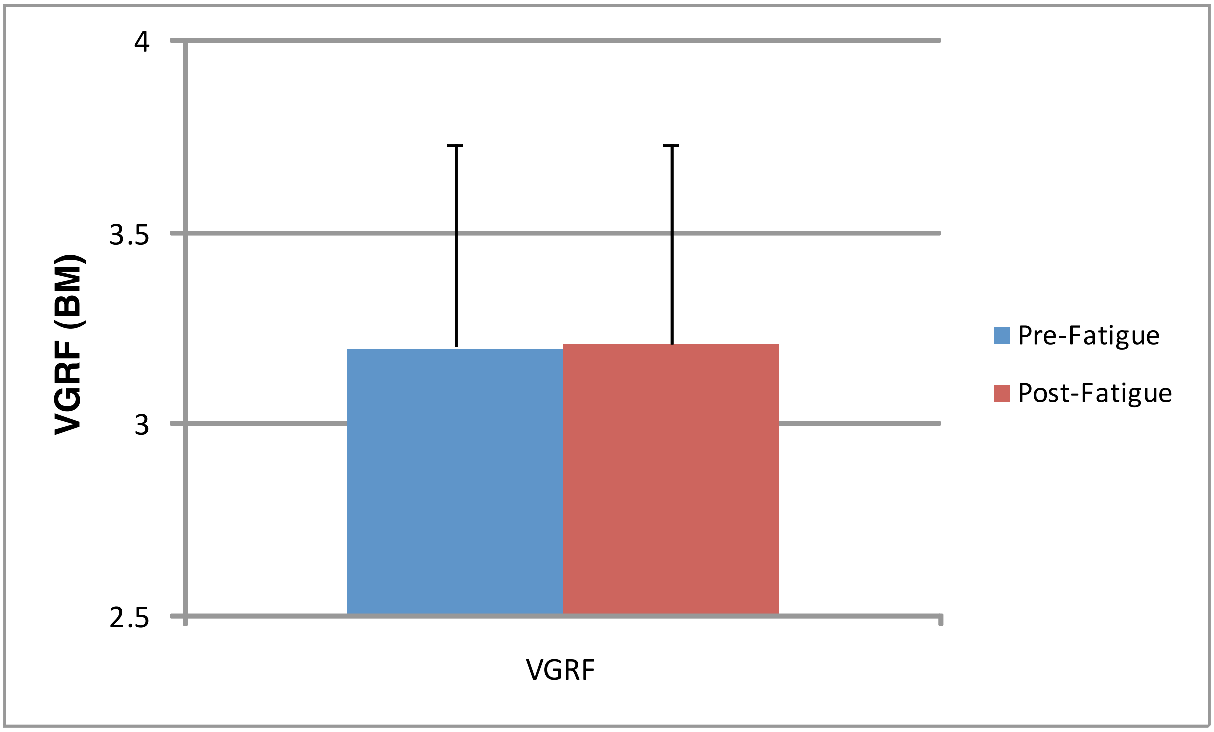

KEM= Knee extension moment, KVM= Knee valgus moment

Figure 15. Main effects for fatigue on ATSF. Data are presented as means and standard deviation bars.



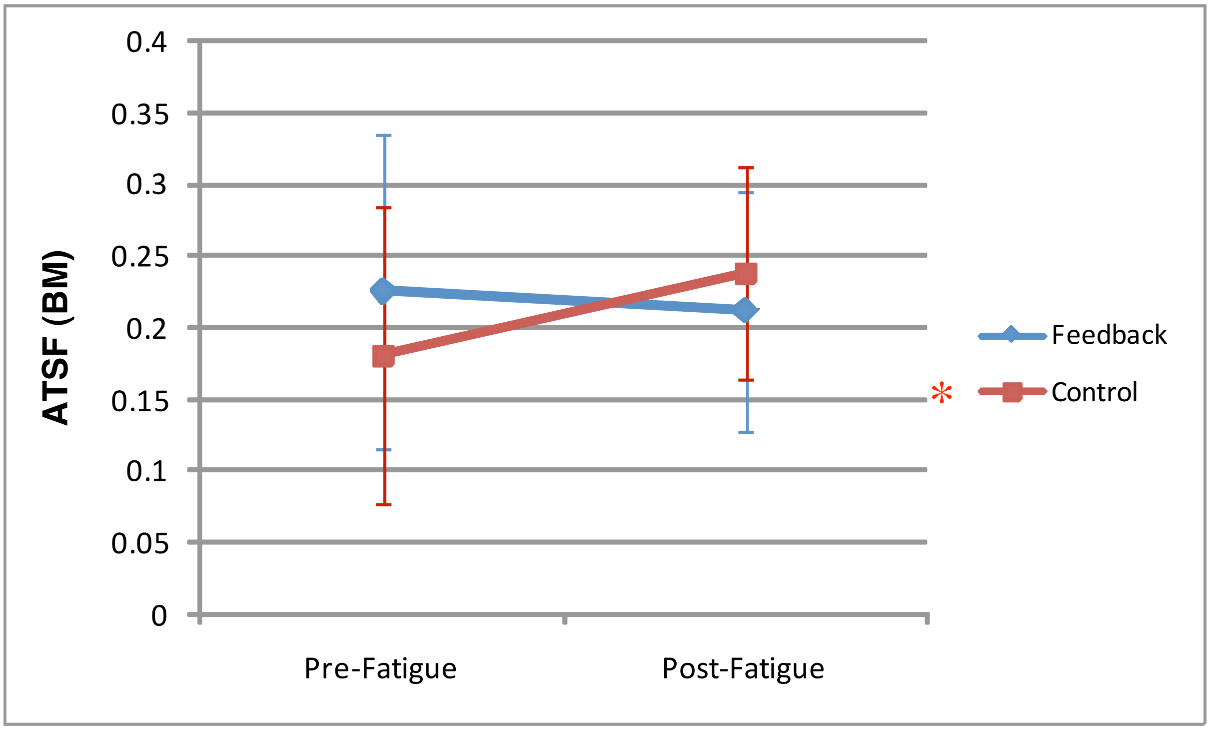

ATSF= Anterior tibial shear force, BM= multiple of body mass

Figure 16. Main effects for fatigue on VGRF. Data are presented as means and standard deviation bars.




VGRF= Vertical ground reaction force, BM= multiple of body mass

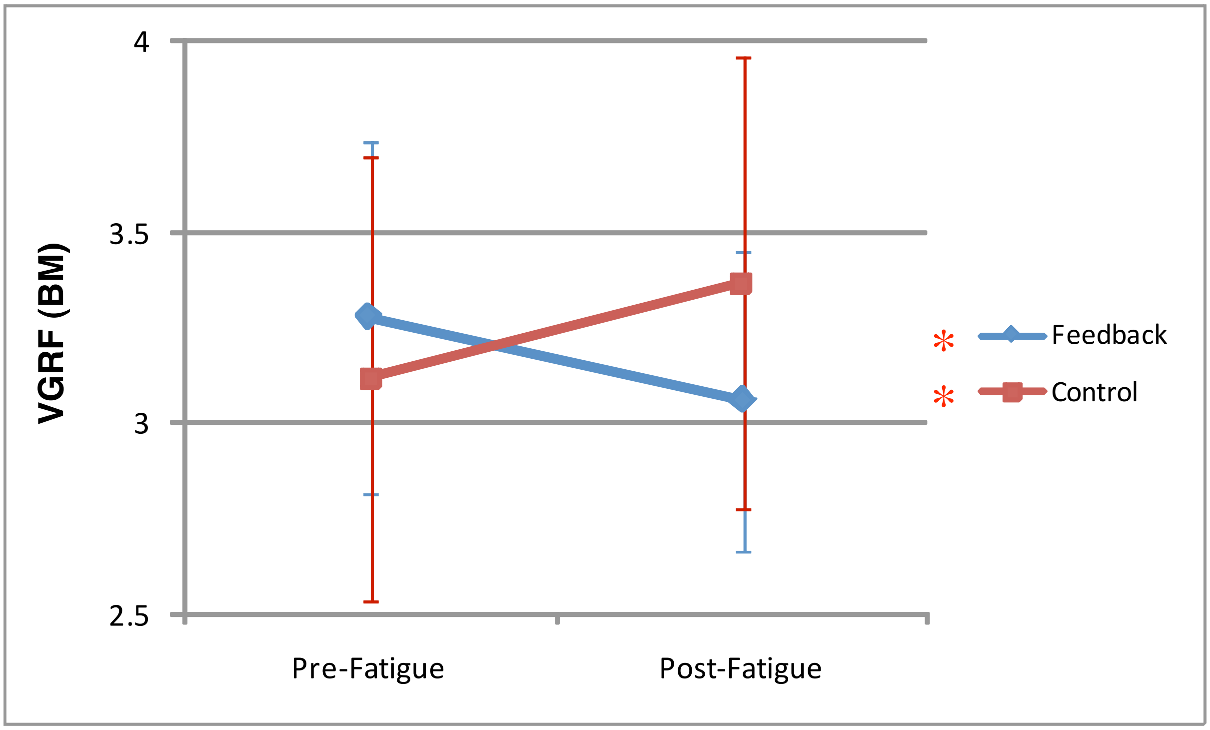
Figure 17. Interaction of fatigue and feedback group on ATSF. Data are presented as means and standard deviation bars.




* Interaction effect for fatigue*feedback group (p<0.05). Non-feedback group changed significantly pre- to post-fatigue, but feedback group did not.

ATSF= Anterior tibial shear force, BM= multiple of body mass

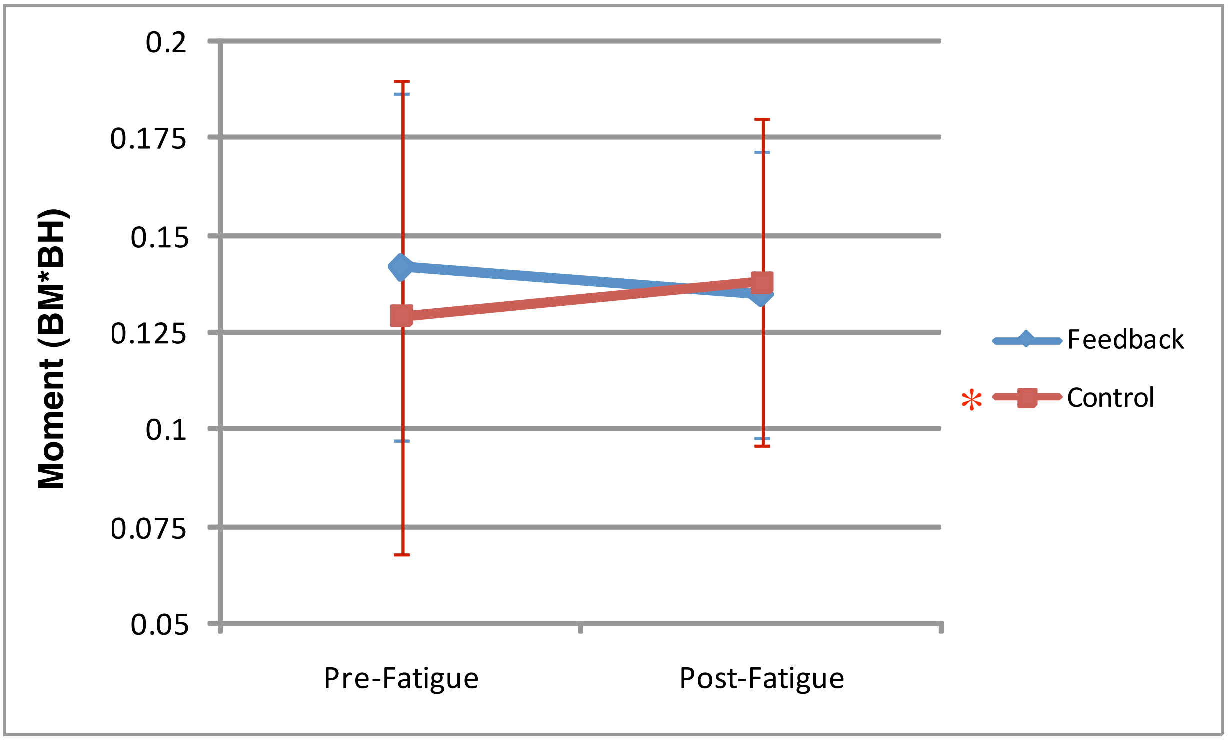
Figure 18. Interaction of fatigue and feedback group on VGRF. Data are presented as means and standard deviation bars.




* Interaction effect for fatigue*feedback group ($p<0.05$). Non-feedback group increased significantly pre- to post-fatigue, while feedback group decreased significantly.

VGRF= Vertical ground reaction force, BM= multiple of body mass

Figure 19. Interaction of fatigue and feedback group on KEM. Data are presented as means and standard deviation bars.




* Interaction effect for fatigue*feedback group ($p<0.05$). Feedback group changed significantly pre- to post-fatigue, but non-feedback group did not.

KEM= Knee extension moment, BM*BH= multiple of the product of body mass and body height

Figure 20. Theoretical Model of the Effects of Fatigue on Movement Patterns and Forces

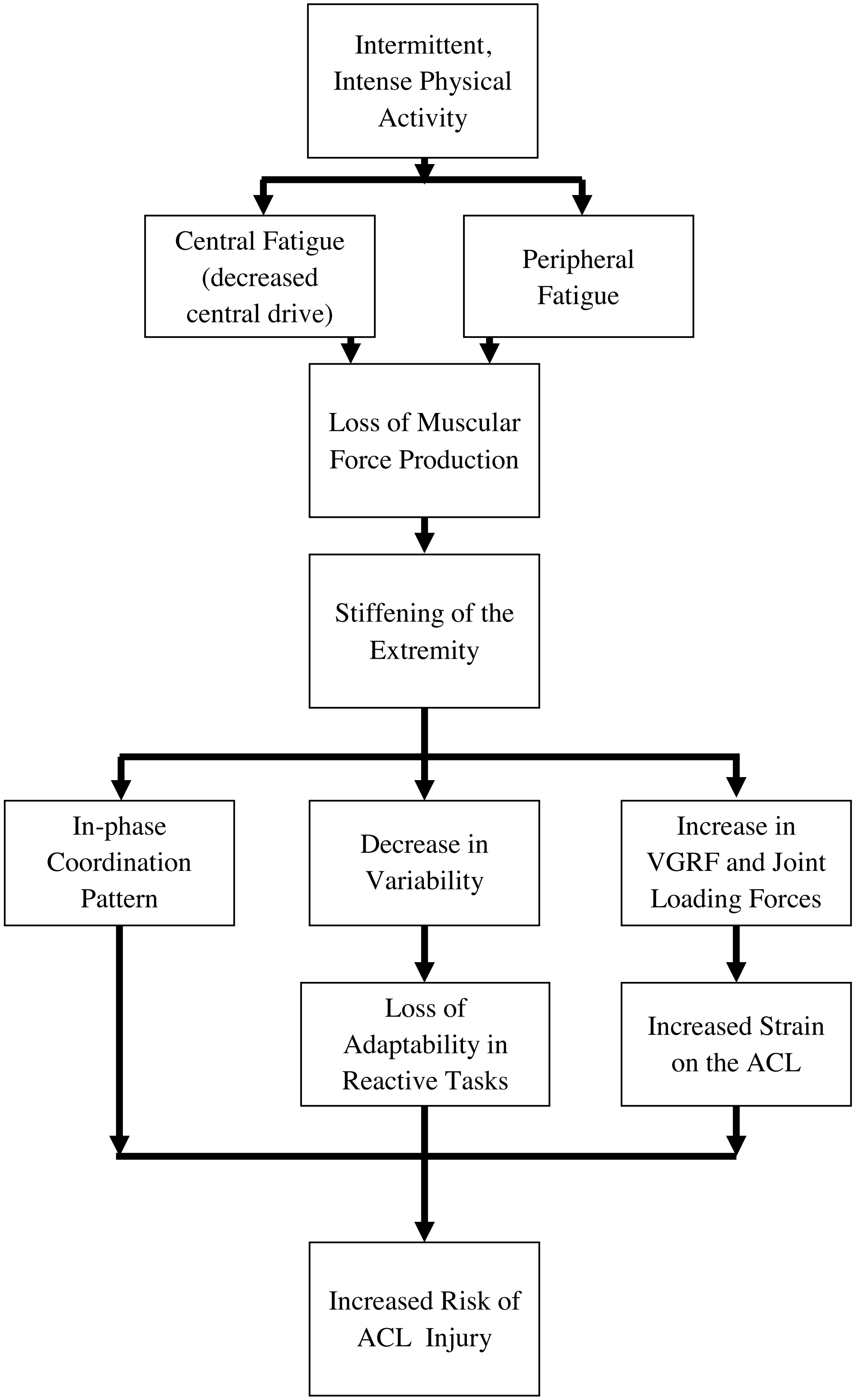


Table 1. Feedback Power Analyses for all Dependent Variables

| Variable | Grand Mean | Standard Deviation | Proposed n (per group) | % Change | Numeric Change | Power | % Change | Numeric Change | Power | % Change | Numeric Change | Power |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Peak Knee Extension Moment | 0.16 | 0.05 | 30 | 20 | 0.03 | 0.69 | 15 | 0.02 | 0.45 | 10 | 0.02 | 0.23 |
| Peak Knee Valgus Moment | 0.18 | 0.02 | 30 | 20 | 0.04 | 0.99 | 15 | 0.03 | 0.99 | 10 | 0.02 | 0.91 |
| Anterior Tibial Shear Force | 0.21 | 0.06 | 30 | 20 | 0.04 | 0.76 | 15 | 0.03 | 0.47 | 10 | 0.02 | 0.25 |
| Vertical Ground Reaction Force | 2.92 | 0.54 | 30 | 20 | 0.58 | 0.99 | 15 | 0.44 | 0.88 | 10 | 0.29 | 0.51 |
| Foot-shank Sagittal Plane MARP | 47.16 | 9.41 | 30 | 20 | 9.44 | 0.97 | 15 | 7.08 | 0.81 | 10 | 4.72 | 0.49 |
| Foot-shank Frontal Plane MARP | 33.80 | 9.01 | 30 | 20 | 6.76 | 0.81 | 15 | 5.07 | 0.55 | 10 | 3.38 | 0.30 |
| Shank-thigh Sagittal Plane MARP | 48.56 | 7.91 | 30 | 20 | 9.72 | 0.99 | 15 | 7.29 | 0.95 | 10 | 4.86 | 0.64 |
| Shank-thigh Frontal Plane MARP | 38.01 | 9.51 | 30 | 20 | 7.60 | 0.86 | 15 | 5.70 | 0.63 | 10 | 3.80 | 0.34 |
| Thigh-trunk Sagittal Plane MARP | 71.82 | 9.21 | 30 | 20 | 14.36 | 0.99 | 15 | 10.77 | 0.99 | 10 | 7.18 | 0.84 |
| Thigh-trunk Frontal Plane MARP | 34.15 | 10.35 | 30 | 20 | 6.84 | 0.70 | 15 | 5.13 | 0.47 | 10 | 3.42 | 0.25 |
| Foot-shank Sagittal Plane DP | 11.56 | 3.37 | 30 | 20 | 2.32 | 0.75 | 15 | 1.74 | 0.49 | 10 | 1.16 | 0.27 |
| Foot-shank Frontal Plane DP | 16.49 | 4.89 | 30 | 20 | 3.30 | 0.71 | 15 | 2.48 | 0.47 | 10 | 1.65 | 0.25 |
| Shank-thigh Sagittal Plane DP | 12.46 | 2.32 | 30 | 20 | 2.50 | 0.98 | 15 | 1.88 | 0.86 | 10 | 1.25 | 0.50 |
| Shank-thigh Frontal Plane DP | 15.28 | 3.86 | 30 | 20 | 3.06 | 0.85 | 15 | 2.30 | 0.63 | 10 | 1.53 | 0.33 |
| Thigh-trunk Sagittal Plane DP | 11.96 | 2.72 | 30 | 20 | 2.40 | 0.94 | 15 | 1.80 | 0.70 | 10 | 1.20 | 0.39 |
| Thigh-trunk Frontal Plane DP | 14.06 | 4.02 | 30 | 20 | 2.82 | 0.76 | 15 | 2.12 | 0.53 | 10 | 1.41 | 0.27 |

Table 2. Fatigue Power Analyses for all Dependent Variables

| **Variable** | **Grand Mean** | **Standard Deviation** | **Proposed n (per group)** | **% Change** | **Numeric Change** | **Power** | **% Change** | **Numeric Change** | **Power** | **% Change** | **Numeric Change** | **Power** |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Peak Knee Extension Moment | 0.16 | 0.05 | 30 | 20 | 0.03 | 0.94 | 15 | 0.02 | 0.75 | 10 | 0.02 | 0.42 |
| Peak Knee Valgus Moment | 0.18 | 0.02 | 30 | 20 | 0.04 | 0.99 | 15 | 0.03 | 0.99 | 10 | 0.02 | 0.99 |
| Anterior Tibial Shear Force | 0.21 | 0.06 | 30 | 20 | 0.04 | 0.97 | 15 | 0.03 | 0.83 | 10 | 0.02 | 0.48 |
| Vertical Ground Reaction Force | 2.92 | 0.54 | 30 | 20 | 0.58 | 0.99 | 15 | 0.44 | 0.99 | 10 | 0.29 | 0.84 |
| Foot-shank Sagittal Plane MARP | 47.16 | 9.41 | 30 | 20 | 9.44 | 0.99 | 15 | 7.08 | 0.98 | 10 | 4.72 | 0.78 |
| Foot-shank Frontal Plane MARP | 33.80 | 9.01 | 30 | 20 | 6.76 | 0.98 | 15 | 5.07 | 0.87 | 10 | 3.38 | 0.54 |
| Shank-thigh Sagittal Plane MARP | 48.56 | 7.91 | 30 | 20 | 9.72 | 0.99 | 15 | 7.29 | 0.99 | 10 | 4.86 | 0.92 |
| Shank-thigh Frontal Plane MARP | 38.01 | 9.51 | 30 | 20 | 7.60 | 0.99 | 15 | 5.70 | 0.91 | 10 | 3.80 | 0.59 |
| Thigh-trunk Sagittal Plane MARP | 71.82 | 9.21 | 30 | 20 | 14.36 | 0.99 | 15 | 10.77 | 0.99 | 10 | 7.18 | 0.99 |
| Thigh-trunk Frontal Plane MARP | 34.15 | 10.35 | 30 | 20 | 6.84 | 0.95 | 15 | 5.13 | 0.78 | 10 | 3.42 | 0.44 |
| Foot-shank Sagittal Plane DP | 11.56 | 3.37 | 30 | 20 | 2.32 | 0.97 | 15 | 1.74 | 0.81 | 10 | 1.16 | 0.47 |
| Foot-shank Frontal Plane DP | 16.49 | 4.89 | 30 | 20 | 3.30 | 0.96 | 15 | 2.48 | 0.79 | 10 | 1.65 | 0.46 |
| Shank-thigh Sagittal Plane DP | 12.46 | 2.32 | 30 | 20 | 2.50 | 0.99 | 15 | 1.88 | 0.99 | 10 | 1.25 | 0.84 |
| Shank-thigh Frontal Plane DP | 15.28 | 3.86 | 30 | 20 | 3.06 | 0.99 | 15 | 2.30 | 0.90 | 10 | 1.53 | 0.58 |
| Thigh-trunk Sagittal Plane DP | 11.96 | 2.72 | 30 | 20 | 2.40 | 0.99 | 15 | 1.80 | 0.95 | 10 | 1.20 | 0.68 |
| Thigh-trunk Frontal Plane DP | 14.06 | 4.02 | 30 | 20 | 2.82 | 0.97 | 15 | 2.12 | 0.82 | 10 | 1.41 | 0.49 |

Table 3. Within-day Intraclass Correlation Coefficients ($ICC_{2,k}$) for MARP and DP. For these analyses, 16 subjects were brought in for one testing session where markers were applied, data was collected for 10 trials of an anticipated sidestep cut, then markers were removed. The subject rested for 5 minutes before markers were re-applied, and 10 more trials completed. Similar data was presented at the 2008 National Athletic Trainers Association Annual Meeting (McGrath ML, Padua DA, Thigpen CA (2008). Reliability of lower-extremity coordination and variability analyses. *Journal of Athletic Training 43*(3), S-27.)

| **Variable** | **$ICC_{2,k}$** | **SEM** |
|---|---|---|
| Foot-shank Sagittal Plane MARP | 0.96 | 2.31 |
| Foot-shank Frontal Plane MARP | 0.97 | 2.36 |
| Shank-thigh Sagittal Plane MARP | 0.97 | 1.57 |
| Shank-thigh Frontal Plane MARP | 0.88 | 2.81 |
| Thigh-trunk Sagittal Plane MARP | 0.94 | 1.89 |
| Thigh-trunk Frontal Plane MARP | 0.95 | 2.17 |
| Foot-shank Sagittal Plane DP | 0.76 | 1.37 |
| Foot-shank Frontal Plane DP | 0.48 | 2.44 |
| Shank-thigh Sagittal Plane DP | 0.78 | 1.04 |
| Shank-thigh Frontal Plane DP | 0.67 | 1.52 |
| Thigh-trunk Sagittal Plane DP | 0.69 | 1.82 |
| Thigh-trunk Frontal Plane DP | 0.68 | 1.53 |

Table 4. Participant Demographics (Mean (sd))

| | | Feedback | Non-feedback | Total |
|---|---|---|---|---|
| Age (years) | | 19.90 (1.47) | 19.59 (1.74) | 19.75 (1.60) |
| Height (cm) | | 177.97 (9.24) | 175.32 (9.11) | 176.67 (9.19) |
| Mass (kg) | | 72.83 (9.79) | 69.58 (10.10) | 71.23 (9.99) |
| Gender | Male | 17 | 14 | 31 |
| | Female | 13 | 15 | 28 |
| Sport | Basketball | 3 | 2 | 5 |
| | Volleyball | 7 | 7 | 14 |
| | Soccer | 10 | 11 | 21 |
| | Lacrosse | 7 | 7 | 14 |
| | Handball | 2 | 3 | 5 |

Table 5. Pre-fatigue t-tests to determine equivalency of groups (Mean(sd))

| | Feedback | Non-feedback | t-value | p-value |
|---|---|---|---|---|
| Age (years) | 19.90 (1.47) | 19.59 (1.74) | 0.748 | 0.457 |
| Height (cm) | 177.97 (9.24) | 175.32 (9.11) | 1.110 | 0.272 |
| Mass (kg) | 72.83 (9.79) | 69.58 (10.10) | 1.254 | 0.215 |
| Initial Completion Time (s) | 22.74 (2.26) | 23.11 (1.95) | -0.672 | 0.504 |
| Number Successful | 21.80 (8.62) | 21.76 (9.19) | 0.018 | 0.986 |
| Number Failed | 2.47 (2.16) | 2.21 (2.11) | 0.467 | 0.642 |
| Total Completed | 24.27 (7.25) | 23.97 (7.67) | 0.155 | 0.877 |
| Initial RPE | 8.37 (1.65) | 8.80 (1.72) | -0.932 | 0.356 |
| Maximum RPE | 19.77 (0.77) | 19.83 (0.38) | -0.381 | 0.705 |
| Recovery RPE 1 | 12.66 (3.14) | 14.52 (3.03) | -2.598 | 0.012 |
| Recovery RPE 2 | 11.38 (2.42) | 12.60 (2.59) | -1.339 | 0.187 |
| Recovery RPE 3 | 10.36 (2.30) | 11.74 (2.92) | -1.430 | 0.159 |
| Time to post-test (min) | 14.90 (2.36) | 15.37 (2.01) | -0.765 | 0.448 |
| Foot-shank Sagittal Plane MARP | 43.93 (12.42) | 49.93 (13.00) | -1.814 | 0.075 |
| Foot-shank Frontal Plane MARP | 20.33 (3.58) | 20.08 (5.82) | 0.195 | 0.846 |
| Shank-thigh Sagittal Plane MARP | 48.69 (11.49) | 54.61 (13.33) | -1.830 | 0.073 |
| Shank-thigh Frontal Plane MARP | 20.64 (5.14) | 18.90 (5.85) | 1.214 | 0.230 |
| Thigh-trunk Sagittal Plane MARP | 63.39 (11.29) | 56.92 (12.45) | 2.092 | 0.041 |
| Thigh-trunk Frontal Plane MARP | 25.77 (6.89) | 22.19 (6.15) | 2.098 | 0.040 |
| Foot-shank Sagittal Plane DP | 15.82 (3.70) | 16.74 (4.07) | -0.900 | 0.372 |
| Foot-shank Frontal Plane DP | 17.37 (5.02) | 18.55 (3.73) | -1.018 | 0.313 |
| Shank-thigh Sagittal Plane DP | 19.01 (4.09) | 19.49 (5.82) | -0.367 | 0.715 |
| Shank-thigh Frontal Plane DP | 17.10 (4.55) | 18.11 (2.86) | -1.013 | 0.315 |
| Thigh-trunk Sagittal Plane DP | 18.66 (5.99) | 17.85 (4.82) | 0.573 | 0.569 |
| Thigh-trunk Frontal Plane DP | 14.29 (3.72) | 14.76 (1.93) | -0.612 | 0.543 |
| VGRF (BM) | 3.28 (0.46) | 3.12 (0.58) | 1.161 | 0.250 |
| KEM (BM*BH) | 0.142 (0.045) | 0.129 (0.061) | -0.899 | 0.372 |
| KVM (BM*BH) | 0.112 (0.052) | 0.122 (0.042) | 0.840 | 0.405 |
| ATSF (BM) | 0.226 (0.110) | 0.181 (0.104) | 1.598 | 0.116 |

Initial Completion Time= number of seconds to complete the running portion of the fatigue protocol during the first trial; Number Successful= the total number of fatigue protocol trials completed under the time limit (<150% of initial completion time); Number Failed= the total number of fatigue protocol trials completed over the time limit (>150% of initial completion time); Total Completed= the total number of fatigue protocol trials completed, both successful and failed;RPE= Rating of Perceived Exertion; RPE 1= RPE taken immediately prior to beginning of post-testing; RPE 2=RPE taken after 8 cutting trials (approximately halfway through post-testing); RPE 3=RPE taken following the final post-test trial; Sag=sagittal plane, Front=frontal plane

Table 6. Fatigue protocol statistics (mean(sd))

| | Feedback | Non-feedback | Total |
|---|---|---|---|
| Initial Completion Time (s) | 22.74 (2.26) | 23.11 (1.95) | 23.02 (2.19) |
| Number Successful | 21.80 (8.62) | 21.76 (9.19) | 21.69 (8.65) |
| Number Failed | 2.47 (2.16) | 2.21 (2.11) | 2.47 (2.20) |
| Total Completed | 24.27 (7.25) | 23.97 (7.67) | 24.17 (7.20) |
| Initial RPE | 8.37 (1.65) | 8.80 (1.72) | 8.56 (1.64) |
| Maximum RPE | 19.77 (0.77) | 19.83 (0.38) | 19.86 (0.35) |
| Recovery RPE 1 | 12.66 (3.14) | 14.52 (3.03) | 13.83 (2.83) |
| Recovery RPE 2 | 11.38 (2.42) | 12.60 (2.59) | 11.81 (2.42) |
| Recovery RPE 3 | 10.36 (2.30) | 11.74 (2.92) | 10.46 (2.32) |
| Time to post-test (min) | 14.90 (2.36) | 15.37 (2.01) | 15.55 (2.20) |

Initial Completion Time= number of seconds to complete the running portion of the fatigue protocol during the first trial; Number Successful= the total number of fatigue protocol trials completed under the time limit (<150% of initial completion time); Number Failed= the total number of fatigue protocol trials completed over the time limit (>150% of initial completion time); Total Completed= the total number of fatigue protocol trials completed, both successful and failed;RPE= Rating of Perceived Exertion; RPE 1= RPE taken immediately prior to beginning of post-testing; RPE 2=RPE taken after 8 cutting trials (approximately halfway through post-testing); RPE 3=RPE taken following the final post-test trial;

Table 7. Pre- and post-fatigue results for sagittal-plane coordination variables (MARP), by feedback group and gender

| | | | Pre-Fatigue | | Post-Fatigue | |
|---|---|---|---|---|---|---|
| | | | Mean (sd) | 95% CI | Mean (sd) | 95% CI |
| Foot-shank Sag *§‡ | FB | F | 46.56 (15.65) | 37.11, 56.01 | 41.27 (16.18) | 31.50, 51.05 |
| | | M | 41.91 (9.29) | 37.14, 46.59 | 38.89 (9.65) | 33.92, 43, 85 |
| | | Total | 43.93 (12.42) | 39.29, 48.57 | 39.92 (12.70) | 35.18, 44.66 |
| | NFB | F | 56.33 (11.40) | 50.02, 62.64 | 46.92 (13.49) | 39.45, 54.39 |
| | | M | 43.08 (11.23) | 36.59, 49.56 | 34.26 (9.54) | 28.75, 39.77 |
| | | Total | 49.93 (13.00) | 44.99, 54.87 | 40.81 (13.22) | 35.78, 45.84 |
| | Total | | 46.88 (12.96) | 43.50, 50.25 | 40.36 (12.85) | 37.01, 43.71 |
| Shank-thigh Sag*§‡ | FB | F | 51.26 (14.39) | 42.56, 59.95 | 47.82 (14.62) | 38.98, 56.66 |
| | | M | 46.73 (8.64) | 42.29, 51.17 | 44.93 (6.93) | 41.37, 48.49 |
| | | Total | 48.69 (11.49) | 44.40, 52.98 | 46.18 (10.82) | 42.14, 50.22 |
| | NFB | F | 60.73 (13.11) | 53.47, 67.99 | 53.84 (15.05) | 45.50, 62.17 |
| | | M | 48.06 (10.40) | 42.05, 54.07 | 41.09 (9.60) | 35.55, 46.64 |
| | | Total | 54.61 (13.33) | 49.54, 59.68 | 47.68 (14.07) | 42.33, 53.04 |
| | Total | | 51.60 (12.68) | 48.30, 54.91 | 46.92 (12.44) | 43.68, 50.16 |
| Thigh-trunk Sag*†‡ | FB | F | 61.29 (13.24) | 53.29, 69.29 | 60.10 (14.23) | 51.50, 68.70 |
| | | M | 64.99 (9.65) | 60.03, 69.95 | 63.67 (13.07) | 56.95, 70.38 |
| | | Total | 63.39 (11.29) | 59.17, 67.60 | 62.12 (13.46) | 57.09, 67.15 |
| | NFB | F | 59.95 (11.02) | 53.85, 66.06 | 54.73 (10.65) | 48.84, 60.63 |
| | | M | 53.67 (13.44) | 45.90, 61.43 | 46.55 (12.48) | 39.34, 53.76 |
| | | Total | 56.92 (12.45) | 52.18. 61.65 | 50.78 (12.10) | 46.18, 55.39 |
| | Total | | 60.21 (12.21) | 57.02, 63.39 | 56.55 (13.93) | 52.92, 60.18 |

* Significant Main Effect for fatigue (α<0.05)
† Significant Main Effect for group (α<0.05)
§ Significant Main Effect for gender (α<0.05)
‡ Significant Interaction (Group x Fatigue) (α<0.05)
¶ Significant Interaction (Group x Fatigue x Gender) (α<0.05)

FB=Feedback group, NFB=Non-feedback group, F=females, M=males, Sag=sagittal plane

Table 8. Pre- and post-fatigue results for frontal-plane coordination variables (MARP), by feedback group and gender

| | | | Pre-Fatigue | | Post-Fatigue | |
|---|---|---|---|---|---|---|
| | | | Mean (sd) | 95% CI | Mean (sd) | 95% CI |
| Foot-shank Front* | FB | F | 21.10 (4.08) | 18.64, 23.57 | 17.09 (3.01) | 15.27, 18.91 |
| | | M | 19.74 (3.15) | 18.12, 21.36 | 17.08 (4.35) | 14.84, 19.32 |
| | | Total | 20.33 (3.58) | 18.99, 21.67 | 17.08 (3.77) | 15.68, 18.49 |
| | NFB | F | 20.72 (4.80) | 18.06, 23.37 | 20.25 (4.80) | 17.59, 22.90 |
| | | M | 19.41 (6.86) | 15.45, 23.37 | 17.07 (5.20) | 14.07, 20.07 |
| | | Total | 20.08 (5.82) | 17.87, 22.30 | 18.71 (5.16) | 16.75, 20.68 |
| | Total | | 20.21 (4.77) | 18.97, 21.45 | 17.88 (4.54) | 16.70, 19.07 |
| Shank-thigh Front‡ | FB | F | 20.53 (5.11) | 17.44, 23.62 | 17.58 (4.92) | 14.61, 20.55 |
| | | M | 20.72 (5.31) | 17.99, 23.46 | 19.61 (8.18) | 15.41, 23.82 |
| | | Total | 20.64 (5.14) | 18.72, 22.56 | 18.73 (6.93) | 16.15, 21.32 |
| | NFB | F | 19.80 (6.16) | 16.38, 23.21 | 21.45 (5.55) | 18.38, 24.52 |
| | | M | 17.95 (5.56) | 14.74, 21.15 | 18.09 (7.04) | 14.03, 22.16 |
| | | Total | 18.90 (5.85) | 16.68, 18.71 | 19.83 (6.43) | 17.39, 22.27 |
| | Total | | 19.79 (5.52) | 18.35, 21.22 | 19.27 (6.65) | 17.54, 21.01 |
| Thigh-trunk Front‡ | FB | F | 23.04 (5.27) | 19.85, 26.22 | 20.95 (6.43) | 17.06, 24.84 |
| | | M | 27.86 (7.39) | 24.05, 31.65 | 23.51 (10.68) | 18.02, 29.00 |
| | | Total | 25.77 (6.89) | 23.19, 28.34 | 22.40 (9.04) | 19.03, 25.78 |
| | NFB | F | 21.40 (5.51) | 18.35, 24.45 | 23.67 (5.97) | 20.36, 26.97 |
| | | M | 23.05 (6.88) | 19.07, 27.02 | 22.26 (9.22) | 16.94, 27.59 |
| | | Total | 22.19 (6.15) | 19.85, 24.53 | 22.99 (7.60) | 20.10, 25.88 |
| | Total | | 24.01 (6.73) | 22.26, 25.76 | 22.69 (8.30) | 20.53, 24.85 |

* Significant Main Effect for fatigue ($\alpha<0.05$)
† Significant Main Effect for group ($\alpha<0.05$)
§ Significant Main Effect for gender ($\alpha<0.05$)
‡ Significant Interaction (Group x Fatigue) ($\alpha<0.05$)
¶ Significant Interaction (Group x Fatigue x Gender) ($\alpha<0.05$)

FB=Feedback group, NFB=Non-feedback group, F=females, M=males, Front=frontal plane

Table 9. Summary of coordination (MARP) ANOVA analyses. F-values, p-values, partial eta-squared ($\eta^2$), and observed power for all analyses.

| Variable | Comparison | F-Value | p-value | $\eta_p^2$ | Observed Power |
|---|---|---|---|---|---|
| Foot-shank Sag | Fatigue | 33.252 | <0.001 | 0.377 | 1.000 |
| | Gender | 7.776 | 0.007 | 0.124 | 0.782 |
| | Feedback group | 1.023 | 0.316 | 0.018 | 0.169 |
| | Fatigue*Gender | 0.386 | 0.537 | 0.007 | 0.094 |
| | Fatigue*Feedback group | 4.641 | 0.036 | 0.078 | 0.562 |
| | Fatigue*Gender*Feedback group | 0.131 | 0.719 | 0.002 | 0.065 |
| Foot-shank Front | Fatigue | 18.565 | <0.001 | 0.252 | 0.988 |
| | Gender | 1.824 | 0.182 | 0.032 | 0.264 |
| | Feedback group | 0.314 | 0.578 | 0.006 | 0.085 |
| | Fatigue*Gender | 0.055 | 0.815 | 0.001 | 0.056 |
| | Fatigue*Feedback group | 3.088 | 0.084 | 0.053 | 0.408 |
| | Fatigue*Gender*Feedback group | 2.137 | 0.149 | 0.037 | 0.301 |
| Shank-thigh Sag | Fatigue | 23.131 | <0.001 | 0.296 | 0.997 |
| | Gender | 7.954 | 0.007 | 0.126 | 0.791 |
| | Feedback group | 1.243 | 0.270 | 0.022 | 0.195 |
| | Fatigue*Gender | 0.156 | 0.694 | 0.003 | 0.067 |
| | Fatigue*Feedback group | 4.719 | 0.034 | 0.079 | 0.569 |
| | Fatigue*Gender*Feedback group | 0.185 | 0.669 | 0.003 | 0.071 |
| Shank-thigh Front | Fatigue | 0.662 | 0.419 | 0.012 | 0.126 |
| | Gender | 0.266 | 0.608 | 0.005 | 0.080 |
| | Feedback group | 0.041 | 0.841 | 0.001 | 0.054 |
| | Fatigue*Gender | 0.014 | 0.907 | 0.000 | 0.052 |
| | Fatigue*Feedback group | 4.464 | 0.039 | 0.075 | 0.546 |
| | Fatigue*Gender*Feedback group | 1.453 | 0.233 | 0.026 | 0.220 |
| Thigh-trunk Sag | Fatigue | 11.352 | 0.001 | 0.171 | 0.911 |
| | Gender | 0.360 | 0.551 | 0.006 | 0.091 |
| | Feedback group | 8.569 | 0.005 | 0.135 | 0.820 |
| | Fatigue*Gender | 0.213 | 0.646 | 0.004 | 0.074 |
| | Fatigue*Feedback group | 4.967 | 0.030 | 0.083 | 0.591 |
| | Fatigue*Gender*Feedback group | 0.160 | 0.691 | 0.003 | 0.068 |
| Thigh-trunk Front | Fatigue | 3.004 | 0.089 | 0.052 | 0.399 |
| | Gender | 1.090 | 0.301 | 0.019 | 0.177 |
| | Feedback group | 0.46 | 0.498 | 0.008 | 0.103 |
| | Fatigue*Gender | 3.467 | 0.068 | 0.059 | 0.448 |
| | Fatigue*Feedback group | 7.708 | 0.008 | 0.123 | 0.779 |
| | Fatigue*Gender*Feedback group | 0.079 | 0.780 | 0.001 | 0.059 |

Table 10. Pre- and post-fatigue results for sagittal-plane variability (DP), by feedback group and gender

| | | | Pre-Fatigue | | Post-Fatigue | |
|---|---|---|---|---|---|---|
| | | | Mean (sd) | 95% CI | Mean (sd) | 95% CI |
| Foot-shank Sag* | FB | F | 15.54 (4.10) | 13.07, 18.02 | 14.03 (3.35) | 12.00, 16.05 |
| | | M | 16.04 (3.48) | 14.25, 17.83 | 13.46 (3.42) | 11.70, 15.22 |
| | | Total | 15.82 (3.70) | 14.44, 17.21 | 13.71 (3.35) | 12.46, 14.96 |
| | NFB | F | 16.96 (4.09) | 14.69, 19.23 | 14.66 (2.60) | 13.22, 16.10 |
| | | M | 16.50 (4.19) | 14.08, 18.91 | 15.35 (3.25) | 13.47, 17,23 |
| | | Total | 16.74 (4.07) | 15.19, 18.29 | 15.00 (2.90) | 13.89, 16.10 |
| | Total | | 16.27 (3.88) | 15.26, 17.28 | 14.34 (3.18) | 13.51, 15.17 |
| Shank-thigh Sag* | FB | F | 19.39 (4.53) | 16.65, 22.13 | 17.87 (4.53) | 15.13, 20.60 |
| | | M | 18.72 (3.83) | 16.75, 20.69 | 15.25 (3.71) | 13.34, 17.16 |
| | | Total | 19.01 (4.09) | 17.49, 20.54 | 16.83 (4.22) | 14.81, 17.96 |
| | NFB | F | 19.95 (5.92) | 16.67, 23.22 | 19.09 (4.36) | 16.67, 21.50 |
| | | M | 19.00 (5.90) | 15.60, 22.41 | 19.62 (2.96) | 17.92, 21.33 |
| | | Total | 19.49 (5.82) | 17.28, 21.71 | 19.35 (3.70) | 17.94, 20.75 |
| | Total | | 19.25 (4.98) | 17.95, 20.55 | 17.84 (4.21) | 16.74, 18.94 |
| Thigh-trunk Sag | FB | F | 17.31 (4.27) | 14.73, 19.89 | 16.97 (3.71) | 14.73, 19.22 |
| | | M | 19.69 (6.98) | 16.10, 23.28 | 16.05 (4.27) | 13.86, 18.25 |
| | | Total | 18.66 (5.99) | 16.42, 20.89 | 16.45 (4.00) | 14.96, 17.94 |
| | NFB | F | 17.33 (5.20) | 14.45, 20.21 | 16.84 (4.12) | 14.56, 19.12 |
| | | M | 18.40 (4.51) | 15.79, 21.00 | 20.04 (3.45) | 18.05, 22.03 |
| | | Total | 17.85 (4.82) | 16.01, 19.68 | 18.38 (4.08) | 16.83, 19.94 |
| | Total | | 18.26 (5.42) | 16.85, 19.67 | 17.40 (4.12) | 16.33, 18.48 |

* Significant Main Effect for fatigue (α<0.05)
† Significant Main Effect for group (α<0.05)
§ Significant Main Effect for gender (α<0.05)
‡ Significant Interaction (Group x Fatigue) (α<0.05)
¶ Significant Interaction (Group x Fatigue x Gender) (α<0.05)

FB=Feedback group, NFB=Non-feedback group, F=females, M=males, Sag=sagittal plane

Table 11. Pre- and post-fatigue results for frontal-plane variability (DP), by feedback group and gender

| | | | Pre-Fatigue | | Post-Fatigue | |
|---|---|---|---|---|---|---|
| | | | Mean (sd) | 95% CI | Mean (sd) | 95% CI |
| Foot-shank Front* | FB | F | 16.48 (5.17) | 13.36, 19.61 | 13.90 (3.84) | 11.58, 16.22 |
| | | M | 18.05 (4.95) | 15.51, 20.59 | 13.89 (4.32) | 11.67, 16.11 |
| | | Total | 17.37 (5.02) | 15.50, 19.24 | 13.90 (4.05) | 12.38, 15.41 |
| | NFB | F | 18.36 (3.65) | 16.34, 20.37 | 14.41 (2.62) | 12.95, 15.86 |
| | | M | 18.75 (3.95) | 16.47, 21.03 | 16.03 (5.17) | 13.04, 19.01 |
| | | Total | 18.55 (3.73) | 17.13, 19.96 | 15.19 (4.07) | 13.64, 16.73 |
| | Total | | 17.95 (4.43) | 16.79, 19.10 | 14.53 (4.08) | 13.47, 15.59 |
| Shank-thigh Front* | FB | F | 15.53 (3.90) | 13.17, 17.88 | 15.27 (2.78) | 13.59, 16.96 |
| | | M | 18.31 (4.76) | 15.86, 20.75 | 14.75 (2.89) | 13.26, 16.24 |
| | | Total | 17.10 (4.55) | 15.40, 18.80 | 14.98 (2.81) | 13.93, 16.03 |
| | NFB | F | 17.81 (2.71) | 16.31, 19.31 | 15.15 (2.15) | 13.96, 16.34 |
| | | M | 18.43 (3.08) | 16.65, 20.20 | 16.10 (3.61) | 14.02, 18.18 |
| | | Total | 18.11 (2.86) | 17.02, 19.20 | 15.61 (2.93) | 14.49, 16.72 |
| | Total | | 17.60 (3.82) | 16.60, 18.59 | 15.29 (2.86) | 14.54, 16.03 |
| Thigh-trunk Front§¶ | FB | F | 13.58 (2.16) | 11.28, 13.89 | 13.49 (1.36) | 12.67, 14.31 |
| | | M | 15.59 (4.17) | 13.45, 17.74 | 13.42 (2.84) | 11.96, 14.88 |
| | | Total | 14.29 (3.72) | 12.90, 15.68 | 13.45 (2.28) | 12.60, 14.30 |
| | NFB | F | 14.51 (1.66) | 13.59, 15.44 | 13.73 (1.92) | 12.66, 14.79 |
| | | M | 15.03 (2.21) | 13.75, 16.31 | 15.05 (3.02) | 13.30, 16.79 |
| | | Total | 14.76 (1.93) | 14.03, 15.50 | 14.36 (2.55) | 13.39, 15.34 |
| | Total | | 14.52 (2.96) | 13.75, 15.29 | 13.90 (2.44) | 13.26, 14.54 |

* Significant Main Effect for fatigue ($\alpha<0.05$)
† Significant Main Effect for group ($\alpha<0.05$)
§ Significant Main Effect for gender ($\alpha<0.05$)
‡ Significant Interaction (Group x Fatigue) ($\alpha<0.05$)
¶ Significant Interaction (Group x Fatigue x Gender) ($\alpha<0.05$)

FB=Feedback group, NFB=Non-feedback group, F=females, M=males, Front=frontal plane

Table 12. Summary of variability (DP) ANOVA analyses. F-values, p-values, partial eta-squared ($\eta^2$), and observed power for all analyses.

| Variable | Comparison | F-Value | p-value | $\eta_p^2$ | Observed Power |
|---|---|---|---|---|---|
| Foot-shank Sag | Fatigue | 13.634 | 0.001 | 0.199 | 0.952 |
| | Gender | 0.003 | 0.959 | 0.000 | 0.050 |
| | Feedback group | 1.957 | 0.167 | 0.034 | 0.280 |
| | Fatigue*Gender | 0.002 | 0.964 | 0.000 | 0.050 |
| | Fatigue*Feedback group | 0.103 | 0.749 | 0.002 | 0.061 |
| | Fatigue*Gender*Feedback group | 1.177 | 0.283 | 0.021 | 0.187 |
| Foot-shank Front | Fatigue | 41.262 | <0.001 | 0.429 | 1.000 |
| | Gender | 0.807 | 0.373 | 0.014 | 0.143 |
| | Feedback group | 1.720 | 0.195 | 0.030 | 0.252 |
| | Fatigue*Gender | 0.029 | 0.866 | 0.001 | 0.053 |
| | Fatigue*Feedback group | 0.001 | 0.976 | 0.000 | 0.050 |
| | Fatigue*Gender*Feedback group | 1.808 | 0.184 | 0.032 | 0.262 |
| Shank-thigh Sag | Fatigue | 4.078 | 0.048 | 0.069 | 0.510 |
| | Gender | 0.858 | 0.358 | 0.015 | 0.149 |
| | Feedback group | 2.603 | 0.112 | 0.045 | 0.354 |
| | Fatigue*Gender | 0.033 | 0.856 | 0.001 | 0.054 |
| | Fatigue*Feedback group | 3.366 | 0.072 | 0.058 | 0.438 |
| | Fatigue*Gender*Feedback group | 1.752 | 0.191 | 0.031 | 0.255 |
| Shank-thigh Front | Fatigue | 21.093 | <0.001 | 0.277 | 0.995 |
| | Gender | 1.700 | 0.198 | 0.030 | 0.249 |
| | Feedback group | 1.539 | 0.220 | 0.027 | 0.230 |
| | Fatigue*Gender | 2.410 | 0.126 | 0.042 | 0.332 |
| | Fatigue*Feedback group | 0.377 | 0.542 | 0.007 | 0.093 |
| | Fatigue*Gender*Feedback group | 3.610 | 0.063 | 0.062 | 0.463 |
| Thigh-trunk Sag | Fatigue | 0.831 | 0.366 | 0.015 | 0.146 |
| | Gender | 2.157 | 0.148 | 0.038 | 0.303 |
| | Feedback group | 0.439 | 0.510 | 0.008 | 0.100 |
| | Fatigue*Gender | 0.140 | 0.710 | 0.003 | 0.066 |
| | Fatigue*Feedback group | 2.737 | 0.104 | 0.047 | 0.369 |
| | Fatigue*Gender*Feedback group | 3.073 | 0.085 | 0.053 | 0.406 |
| Thigh-trunk Front | Fatigue | 1.757 | 0.191 | 0.031 | 0.256 |
| | Gender | 4.402 | 0.041 | 0.074 | 0.540 |
| | Feedback group | 2.007 | 0.162 | 0.035 | 0.285 |
| | Fatigue*Gender | 2.186 | 0.145 | 0.038 | 0.306 |
| | Fatigue*Feedback group | 0.103 | 0.749 | 0.002 | 0.061 |
| | Fatigue*Gender*Feedback group | 6.386 | 0.014 | 0.104 | 0.699 |

Table 13. Pre- and post-fatigue results for kinetic variables, by feedback group and gender

| | | | Pre-Fatigue | | Post-Fatigue | |
|---|---|---|---|---|---|---|
| | | | Mean (sd) | 95% CI | Mean (sd) | 95% CI |
| VGRF (BM) ‡ | FB | F | 3.22 (0.45) | 2.95, 3.49 | 2.99 (0.26) | 2.83, 3.14 |
| | | M | 3.32 (0.49) | 3.07, 3.57 | 3.11 (0.48) | 2.87, 3.35 |
| | | Total | 3.28 (0.46) | 3.10, 3.45 | 3.06 (0.39) | 2.91, 3.20 |
| | NFB | F | 2.97 (0.62) | 2.63, 3.31 | 3.23 (0.65) | 2.87, 3.60 |
| | | M | 3.28 (0.51) | 2.99, 3.57 | 3.52 (0.49) | 3.23, 3.80 |
| | | Total | 3.12 (0.58) | 2.90, 3.34 | 3.37 (0.59) | 3.15, 3.59 |
| | Total | | 3.20 (0.53) | 3.06, 3.33 | 3.21 (0.52) | 3.08, 3.35 |
| KEM (BM*BH) ‡ | FB | F | 0.127 (0.043) | 0.110, 0.155 | 0.120 (0.041) | 0.094, 0.146 |
| | | M | 0.152 (0.045) | 0.129, 0.175 | 0.128 (0.035) | 0.110, 0.146 |
| | | Total | 0.142 (0.045) | 0.125, 0.159 | 0.135 (0.037) | 0.111, 0.139 |
| | NFB | F | 0.128 (0.060) | 0.093, 0.162 | 0.142 (0.045) | 0.116, 0.169 |
| | | M | 0.131 (0.064) | 0.094, 0.167 | 0.133 (0.039) | 0.110, 0.133 |
| | | Total | 0.129 (0.061) | 0.106, 0.153 | 0.138 (0.042) | 0.121, 0.154 |
| | Total | | 0.136 (0.053) | 0.122, 0.150 | 0.131 (0.040) | 0.121, 0.142 |
| KVM (BM*BH) | FB | F | 0.115 (0.053) | 0.081, 0.149 | 0.084 (0.030) | 0.065, 0.103 |
| | | M | 0.109 (0.053) | 0.082, 0.136 | 0.112 (0.039) | 0.092, 0.132 |
| | | Total | 0.112 (0.052) | 0.092, 0.131 | 0.100 (0.039) | 0.086, 0.115 |
| | NFB | F | 0.126 (0.052) | 0.106, 0.166 | 0.131 (0.053) | 0.100, 0.162 |
| | | M | 0.108 (0.023) | 0.095, 0.122 | 0.114 (0.036) | 0.093, 0.135 |
| | | Total | 0.122 (0.042) | 0.106, 0.138 | 0.123 (0.045) | 0.105, 0.140 |
| | Total | | 0.117 (0.047) | 0.104, 0.129 | 0.111 (0.043) | 0.100, 0.123 |
| ATSF (BM) §‡ | FB | F | 0.165 (0.100) | 0.101, 0.228 | 0.165 (0.088) | 0.109, 0.221 |
| | | M | 0.270 (0.098) | 0.220, 0.320 | 0.244 (0.063) | 0.212, 0.277 |
| | | Total | 0.226 (0.110) | 0.184, 0.268 | 0.212 (0.083) | 0.180, 0.243 |
| | NFB | F | 0.172 (0.128) | 0.098, 0.245 | 0.221 (0.059) | 0.187, 0.256 |
| | | M | 0.190 (0.076) | 0.147, 0.234 | 0.256 (0.085) | 0.207, 0.305 |
| | | Total | 0.181 (0.104) | 0.141, 0.221 | 0.239 (0.074) | 0.210, 0.267 |
| | Total | | 0.204 (0.109) | 0.175, 0.233 | 0.225 (0.079) | 0.204, 0.246 |

* Significant Main Effect for fatigue (α<0.05)
† Significant Main Effect for group (α<0.05)
§ Significant Main Effect for gender (α<0.05)
‡ Significant Interaction (Group x Fatigue) (α<0.05)
¶ Significant Interaction (Group x Fatigue x Gender) (α<0.05)

FB=Feedback group, NFB=Non-feedback group, F=females, M=males, VGRF=vertical ground reaction force, KEM=knee extension moment, KVM=knee valgus moment, ATSF=anterior tibial shear force, BM=multiple of body mass, BM*BH=multiple of the product of body mass and height

Table 14. Summary of kinetic ANOVA analyses. F-values, p-values, partial eta-squared ($\eta^2$), and observed power for all analyses.

| Variable | Comparison | F-Value | p-value | $\eta_p^2$ | Observed Power |
|---|---|---|---|---|---|
| VGRF (BM) | Fatigue | 0.063 | 0.803 | 0.001 | 0.057 |
| | Gender | 3.018 | 0.088 | 0.052 | 0.400 |
| | Feedback group | 0.595 | 0.444 | 0.011 | 0.118 |
| | Fatigue*Gender | 0.000 | 0.989 | 0.000 | 0.050 |
| | Fatigue*Feedback group | 14.259 | <0.001 | 0.206 | 0.960 |
| | Fatigue*Gender*Feedback group | 0.052 | 0.820 | 0.001 | 0.056 |
| KEM (BM*BH) | Fatigue | 0.396 | 0.532 | 0.007 | 0.095 |
| | Gender | 0.343 | 0.561 | 0.006 | 0.089 |
| | Feedback group | 0.016 | 0.899 | 0.000 | 0.052 |
| | Fatigue*Gender | 1.610 | 0.210 | 0.029 | 0.238 |
| | Fatigue*Feedback group | 4.367 | 0.041 | 0.076 | 0.537 |
| | Fatigue*Gender*Feedback group | 0.022 | 0.881 | 0.000 | 0.052 |
| KVM (BM*BH) | Fatigue | 1492 | 0.227 | 0.027 | 0.224 |
| | Gender | 0.286 | 0.595 | 0.005 | 0.082 |
| | Feedback group | 2.776 | 0.102 | 0.050 | 0.373 |
| | Fatigue*Gender | 3.954 | 0.052 | 0.069 | 0.497 |
| | Fatigue*Feedback group | 1.676 | 0.201 | 0.031 | 0.246 |
| | Fatigue*Gender*Feedback group | 1.112 | 0.296 | 0.021 | 0.179 |
| ATSF (BM) | Fatigue | 2.837 | 0.098 | 0.051 | 0.380 |
| | Gender | 9.206 | 0.004 | 0.148 | 0.846 |
| | Feedback group | 0.004 | 0.949 | 0.000 | 0.050 |
| | Fatigue*Gender | 0.040 | 0.843 | 0.001 | 0.054 |
| | Fatigue*Feedback group | 6.783 | 0.012 | 0/113 | 0.725 |
| | Fatigue*Gender*Feedback group | 0.613 | 0.437 | 0.011 | 0.120 |

Table 15. Stance Time and Time-to-peak Forces and Moments (Mean (sd))

| | | Pre-Fatigue | Post-Fatigue |
|---|---|---|---|
| Stance Time (ms) *§ | FB | 703 (185) | 774 (217) |
| | NFB | 636 (192) | 712 (236) |
| Time-to-peak VGRF (ms) *‡ | FB | 41 (14) | 41 (11) |
| | NFB | 53 (21) | 42 (15) |
| Time-to-peak KEM (ms) ‡ | FB | 85 (57) | 101 (65) |
| | NFB | 93 (41) | 86 (24) |
| Time-to-peak KVM (ms) | FB | 76 (42) | 68 (47) |
| | NFB | 73 (35) | 76 (59) |
| Time-to-peak ATSF (ms) *# | FB | 43 (61) | 27 (39) |
| | NFB | 56 (71) | 24 (30) |

* Significant Main Effect for fatigue ($\alpha<0.05$)
† Significant Main Effect for group ($\alpha<0.05$)
§ Significant Main Effect for gender ($\alpha<0.05$)
‡ Significant Interaction (Group x Fatigue) ($\alpha<0.05$)
# Significant Interaction (Gender x Fatigue) ($\alpha<0.05$)

Table 16. Physical testing results pre and post-fatigue

| | Pre-fatigue | | Post-fatigue | |
|---|---|---|---|---|
| | Mean (sd) | 95% CI | Mean (sd) | 95% CI |
| Vertical Jump (cm)*§ | 54.33 (12.03) | 51.20, 57.46 | 53.12 (11.68) | 50.08, 56.17 |
| Motor Skill Test Time (s)* | 9.10 (1.10) | 8.81, 9.38 | 9.56 (1.34) | 9.21, 9.91 |
| Motor Skill Test Errors*† | 1.93 (0.95) | 1.67, 2.16 | 3.50 (1.54) | 3.10, 3.90 |
| Motor Skill Test Score* | 10.04 (1.25) | 9.73, 10.38 | 11.31 (1.49) | 10.92, 11.70 |

* Significant Main Effect for fatigue ($\alpha<0.05$)
† Significant Main Effect for group ($\alpha<0.05$)
§ Significant Main Effect for gender ($\alpha<0.05$)
‡ Significant Interaction (Group x Fatigue) ($\alpha<0.05$)
¶ Significant Interaction (Group x Fatigue x Gender) ($\alpha<0.05$)

Table 17. Summary of Mean Changes and Effect Sizes (Cohen's d) for sagittal plane MARP values

| | | | Mean Difference (pre-fatigue – post-fatigue) | Cohen's d |
|---|---|---|---|---|
| Foot-shank Sag | FB | F | 5.29 | 0.33 |
| | | M | 3.02 | 0.31 |
| | | Total | 4.01 | 0.32 |
| | NFB | F | 9.41 | 0.70 |
| | | M | 8.82 | 0.79 |
| | | Total | 9.12 | 0.69 |
| | Total | | 6.52 | 0.50 |
| Shank-thigh Sag | FB | F | 3.44 | 0.24 |
| | | M | 1.80 | 0.21 |
| | | Total | 2.51 | 0.22 |
| | NFB | F | 6.89 | 0.46 |
| | | M | 6.97 | 0.67 |
| | | Total | 6.93 | 0.49 |
| | Total | | 4.68 | 0.37 |
| Thigh-trunk Sag | FB | F | 1.19 | 0.08 |
| | | M | 1.32 | 0.10 |
| | | Total | 1.27 | 0.09 |
| | NFB | F | 5.22 | 0.47 |
| | | M | 7.12 | 0.53 |
| | | Total | 6.14 | 0.49 |
| | Total | | 3.66 | 0.26 |

FB=Feedback group, NFB=Non-feedback group, F=females, M=males, Sag=sagittal plane

Table 18. Summary of Mean Changes and Effect Sizes (Cohen's d) for frontal plane MARP values

| | | | Mean Difference (pre-fatigue – post-fatigue) | Cohen's d |
|---|---|---|---|---|
| Foot-shank Front | FB | F | 4.01 | 0.98 |
| | | M | 2.66 | 0.61 |
| | | Total | 3.25 | 0.86 |
| | NFB | F | 0.47 | 0.10 |
| | | M | 2.34 | 0.34 |
| | | Total | 1.37 | 0.24 |
| | Total | | 2.33 | 0.49 |
| Shank-thigh Front | FB | F | 2.95 | 0.58 |
| | | M | 1.11 | 0.14 |
| | | Total | 1.91 | 0.28 |
| | NFB | F | -1.65 | 0.27 |
| | | M | -0.14 | 0.02 |
| | | Total | -0.93 | 0.14 |
| | Total | | 0.52 | 0.08 |
| Thigh-trunk Front | FB | F | 2.09 | 0.33 |
| | | M | 4.35 | 0.41 |
| | | Total | 3.37 | 0.37 |
| | NFB | F | -2.27 | 0.38 |
| | | M | 0.79 | 0.09 |
| | | Total | -0.80 | 0.11 |
| | Total | | 1.32 | 0.16 |

FB=Feedback group, NFB=Non-feedback group, F=females, M=males, Front=frontal plane

Table 19. Summary of Mean Changes and Effect Sizes (Cohen's d) for sagittal plane DP values

| | | | Mean Difference (pre-fatigue – post-fatigue) | Cohen's d |
|---|---|---|---|---|
| Foot-shank Sag | FB | F | 1.51 | 0.37 |
| | | M | 2.58 | 0.74 |
| | | Total | 2.11 | 0.57 |
| | NFB | F | 2.30 | 0.56 |
| | | M | 1.15 | 0.27 |
| | | Total | 1.74 | 0.43 |
| | Total | | 1.93 | 0.50 |
| Shank-thigh Sag | FB | F | 1.52 | 0.34 |
| | | M | 3.47 | 0.91 |
| | | Total | 2.18 | 0.52 |
| | NFB | F | 0.86 | 0.15 |
| | | M | -0.62 | 0.11 |
| | | Total | 0.14 | 0.02 |
| | Total | | 1.41 | 0.28 |
| Thigh-trunk Sag | FB | F | 0.34 | 0.08 |
| | | M | 3.64 | 0.52 |
| | | Total | 2.21 | 0.37 |
| | NFB | F | 0.49 | 0.09 |
| | | M | -1.64 | 0.36 |
| | | Total | -0.53 | 0.11 |
| | Total | | 0.86 | 0.16 |

FB=Feedback group, NFB=Non-feedback group, F=females, M=males, Sag=sagittal plane

Table 20. Summary of Mean Changes and Effect Sizes (Cohen's d) for frontal plane DP values

| | | | Mean Difference (pre-fatigue – post-fatigue) | Cohen's d |
|---|---|---|---|---|
| Foot-shank Front | FB | F | 2.58 | 0.50 |
| | | M | 4.16 | 0.84 |
| | | Total | 3.47 | 0.69 |
| | NFB | F | 3.95 | 1.08 |
| | | M | 2.72 | 0.53 |
| | | Total | 3.36 | 0.83 |
| | Total | | 3.42 | 0.77 |
| Shank-thigh Front | FB | F | 0.26 | 0.07 |
| | | M | 3.56 | 0.75 |
| | | Total | 2.12 | 0.47 |
| | NFB | F | 2.66 | 0.98 |
| | | M | 2.33 | 0.65 |
| | | Total | 2.50 | 0.85 |
| | Total | | 2.31 | 0.60 |
| Thigh-trunk Front | FB | F | 0.09 | 0.04 |
| | | M | 2.17 | 0.52 |
| | | Total | 0.84 | 0.23 |
| | NFB | F | 0.78 | 0.41 |
| | | M | -0.02 | 0.01 |
| | | Total | 0.40 | 0.16 |
| | Total | | 0.62 | 0.21 |

FB=Feedback group, NFB=Non-feedback group, F=females, M=males, Front=frontal plane

Table 21. Summary of Mean Changes and Effect Sizes (Cohen's d) for kinetic values

| | | | Mean Difference (pre-fatigue – post-fatigue) | Cohen's d |
|---|---|---|---|---|
| VGRF (BM) | FB | F | 0.23 | 0.51 |
| | | M | 0.21 | 0.43 |
| | | Total | 0.22 | 0.48 |
| | NFB | F | -0.26 | 0.40 |
| | | M | -0.24 | 0.47 |
| | | Total | -0.25 | 0.42 |
| | Total | | -0.01 | 0.02 |
| KEM (BM*BH) | FB | F | 0.01 | 0.16 |
| | | M | 0.02 | 0.53 |
| | | Total | 0.01 | 0.16 |
| | NFB | F | -0.01 | 0.23 |
| | | M | 0.00 | 0.03 |
| | | Total | -0.01 | 0.15 |
| | Total | | 0.01 | 0.09 |
| KVM (BM*BH) | FB | F | 0.03 | 0.58 |
| | | M | 0.00 | 0.06 |
| | | Total | 0.01 | 0.23 |
| | NFB | F | -0.01 | 0.09 |
| | | M | -0.01 | 0.17 |
| | | Total | 0.00 | 0.02 |
| | Total | | 0.01 | 0.13 |
| ATSF (BM) | FB | F | 0.00 | 0.00 |
| | | M | 0.03 | 0.27 |
| | | Total | 0.01 | 0.13 |
| | NFB | F | -0.05 | 0.38 |
| | | M | -0.07 | 0.78 |
| | | Total | -0.06 | 0.56 |
| | Total | | -0.02 | 0.19 |

FB=Feedback group, NFB=Non-feedback group, F=females, M=males, VGRF=vertical ground reaction force, KEM=knee extension moment, KVM=knee valgus moment, ATSF=anterior tibial shear force, BM=multiple of body mass, BM*BH=multiple of the product of body mass and height

APPENDIX 1

**Evidence of Central and Peripheral Fatigue After a Functional Fatigue Protocol**

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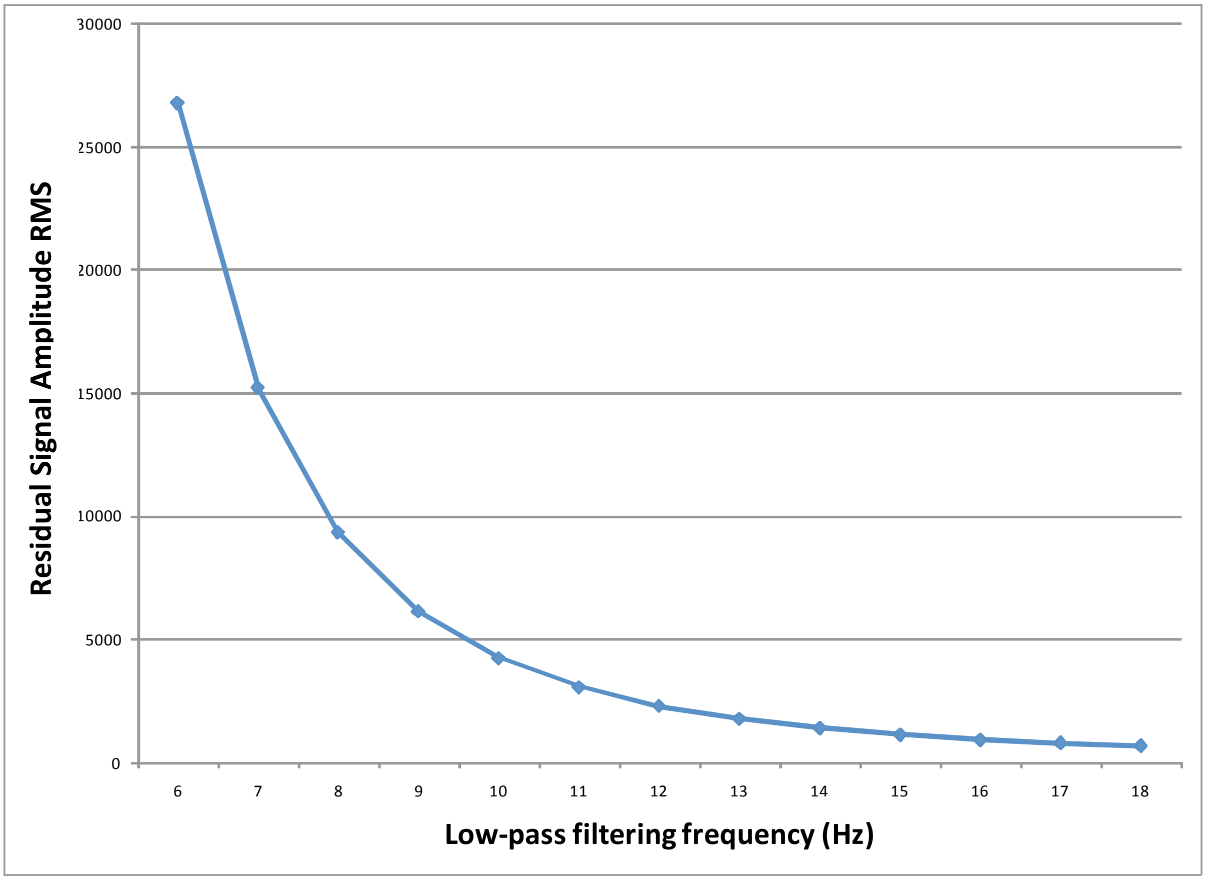
University of North Carolina, Chapel Hill, NC

Muscle fatigue often occurs during athletic participation, and may lead to injury. The mechanisms that produce muscular fatigue during intermittent, multi-directional exercise (e.g., soccer or basketball) remain unknown. We propose that central activation failure (CAF) will account for a major portion of the decrease in muscle force production that occurs during a functional fatigue protocol (FFP) designed to simulate high-level activities. **PURPOSE**: To determine the extent of central and peripheral fatigue during a FFP in unimpaired individuals. **METHODS**: Fourteen (7 M, 7 F) recreationally-active participants volunteered for this study (age: 21±2years, height: 175±9cm, mass: 68±8kg). The FFP consisted of a maximum-effort timed 90m agility course and 5 standing broad jumps for maximum distance. The FFP was repeated (with a 5-sec rest between each repetition) until the time to complete the agility course exceeded 150% of the initial repetition. Voluntary (MVC) and electrically-elicited (EEC) peak isometric quadriceps torque, and maximum vertical jump height (VJ), were measured pre- and post-FFP. Central fatigue was assessed by using the twitch superimposition technique to measure CAF in the quadriceps. A 130V, 10 pulse train of electrical impulses (lasting 100ms) were delivered via two electrodes placed over the quadriceps during a 3s MVC. Peak torque was calculated 100ms prior to, and immediately after the initiation of the electrical pulses. CAF was calculated as the change in peak torque (EEC–MVC) divided by the EEC torque. Peripheral fatigue was assessed by

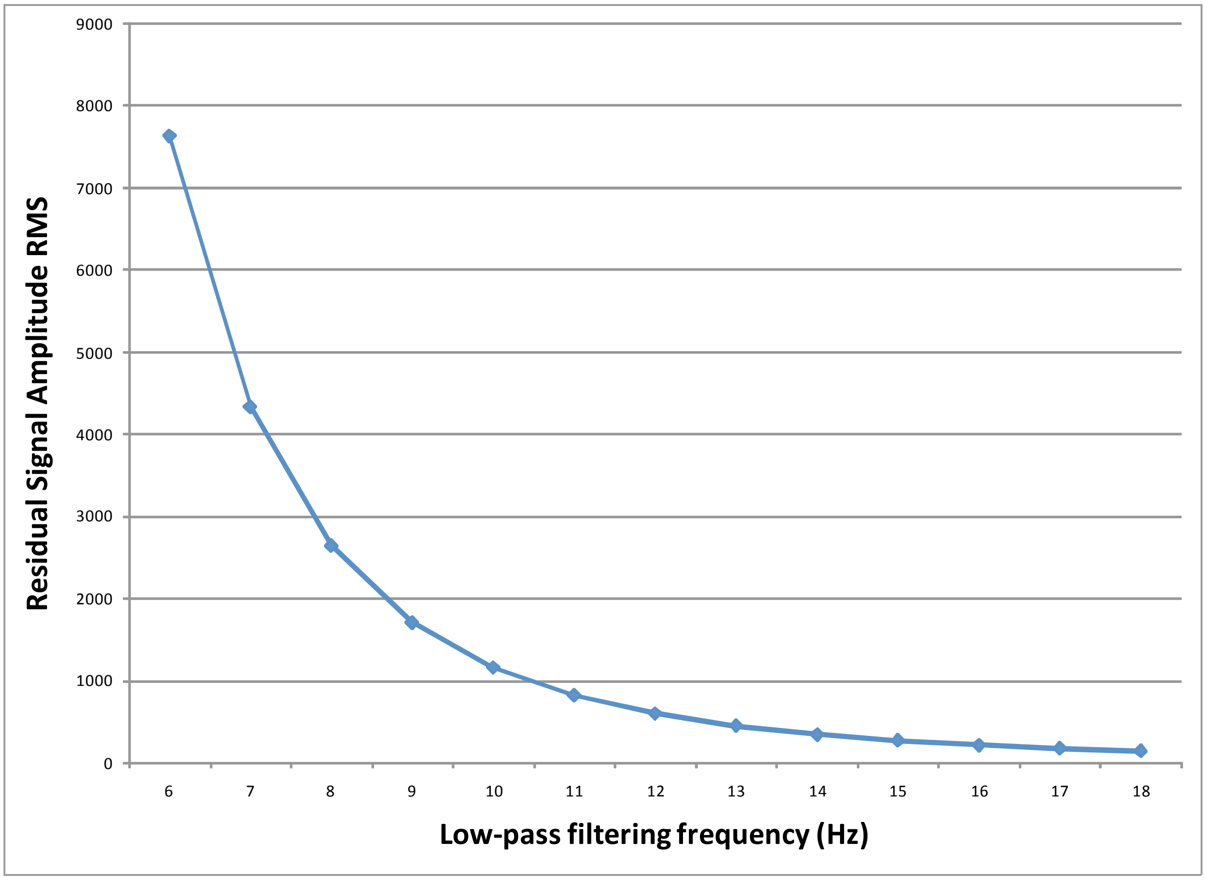
examining the change in EEC torque. Paired t-tests and bivariate correlations were performed to analyze the results between conditions. **RESULTS**: CAF increased post-fatigue (pre:3.5±2.8%; post:10.2±6.2%, $p=0.004$). Both MVC (166.4±40.1Nm to 139.1±42.3Nm, $p=0.010$) and EEC (172.7±41.8Nm to 153.7±40.7Nm, $p=0.018$) decreased post-fatigue. The change in CAF was highly correlated to the loss of MVC torque ($r=0.797$), explaining 59.4% of the variance ($p=0.002$). VJ also decreased post-fatigue (48.5±8.3cm to 45.4±11.8cm, $p=0.034$). **CONCLUSION**: This FFP produces significant levels of central and peripheral fatigue and decreases isometric quadriceps force and VJ height. The loss of voluntary muscle force is significantly related to the change in CAF.

APPENDIX 2. Residual Analysis Performed to Determine Low-pass Cut-off Frequency

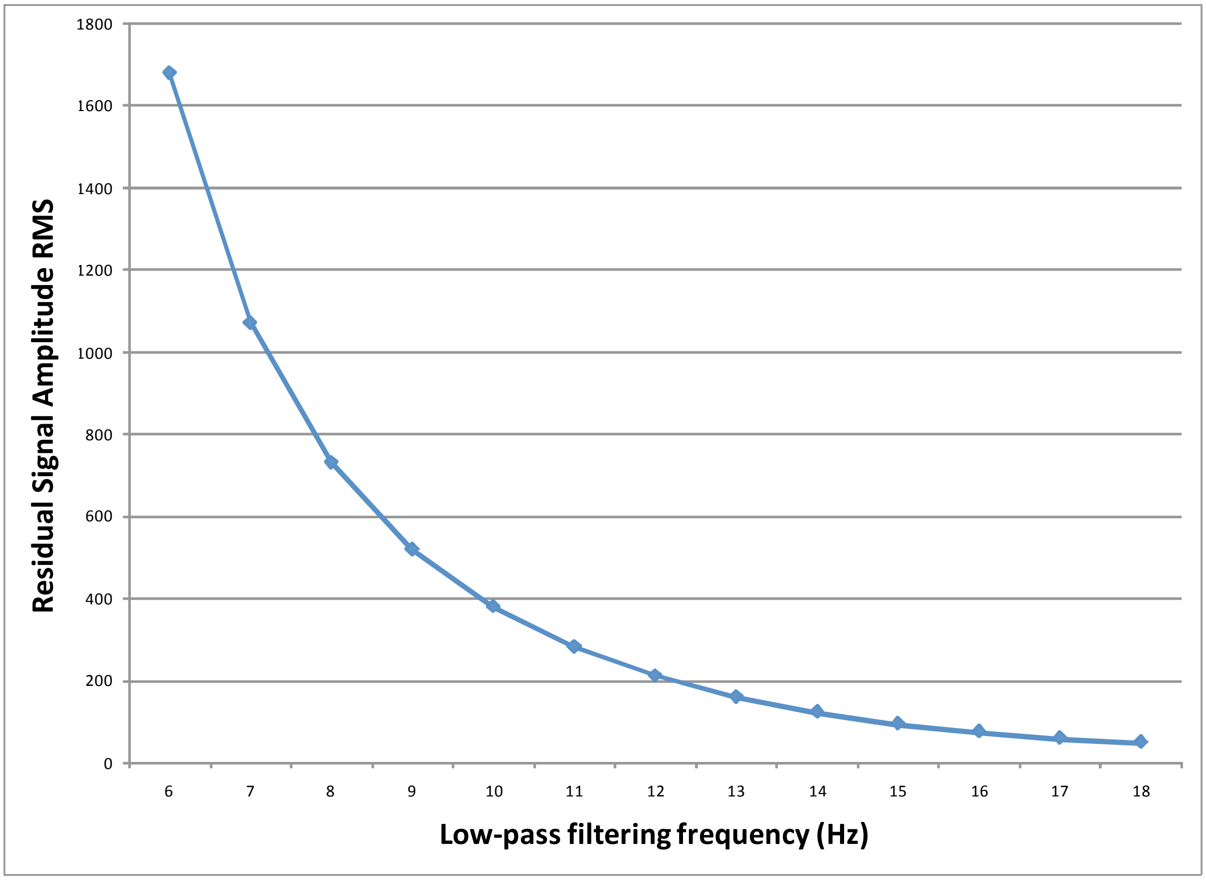
Shank Segment, Sagittal plane



Thigh Segment, Sagittal plane

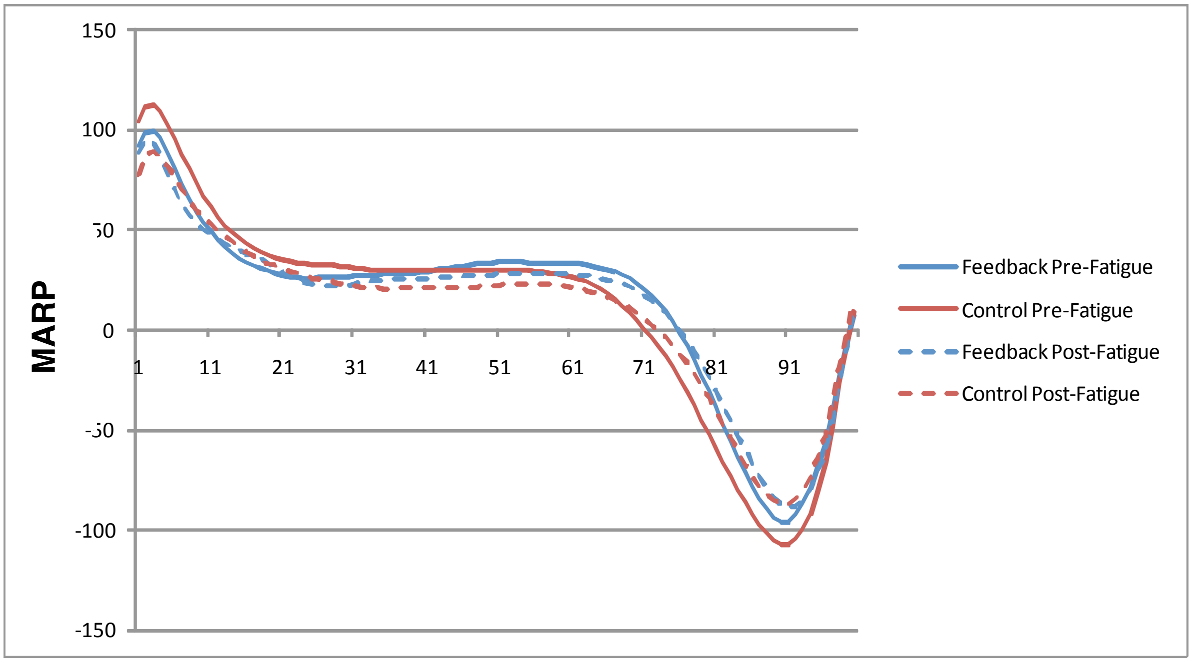


Trunk Segment, Sagittal plane

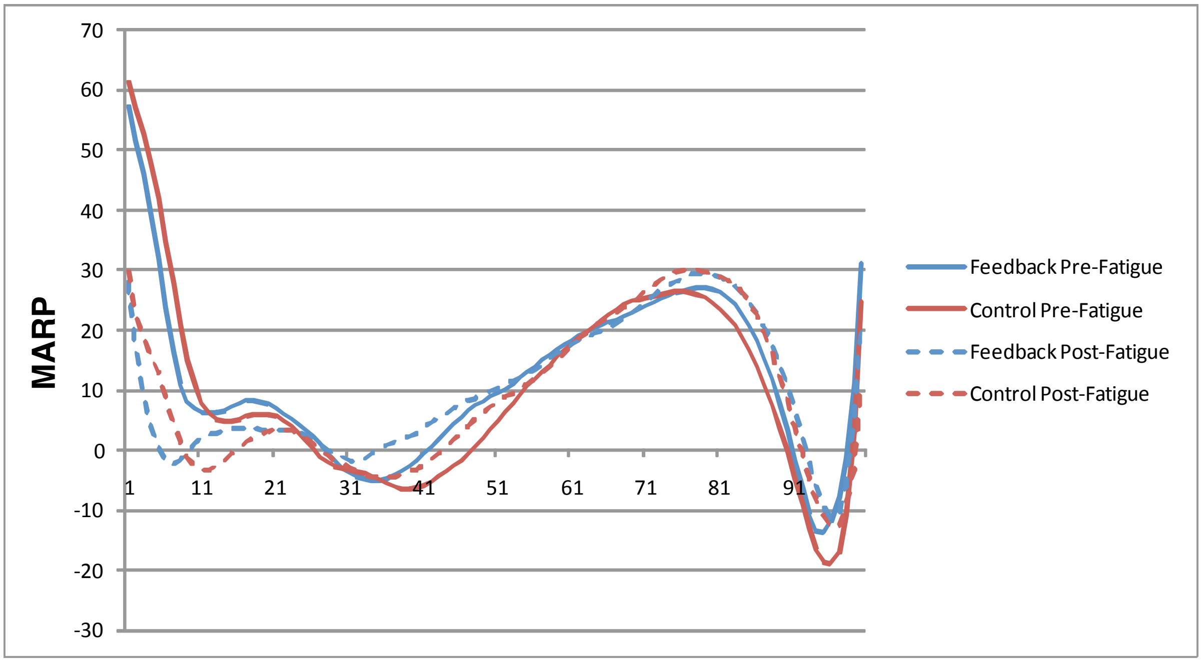


APPENDIX 3. Mean Ensemble Curves for all Dependent Variables, by Group and Fatigue

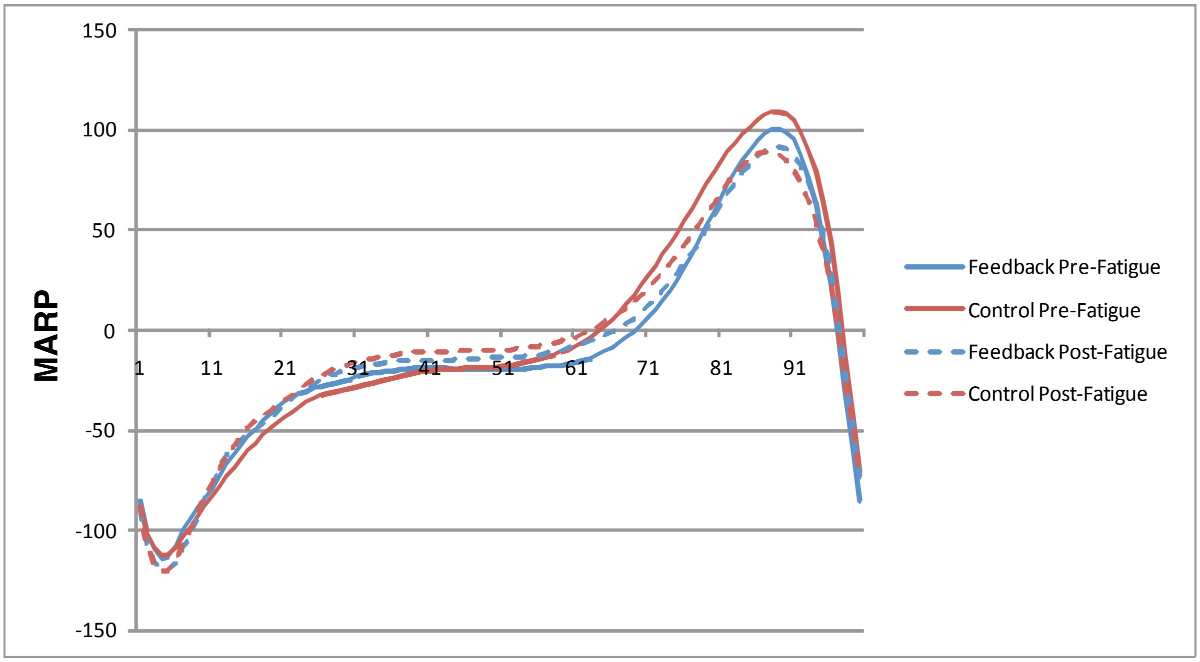
Foot-Shank Sagittal Plane RPA Curves, by Group and Fatigue



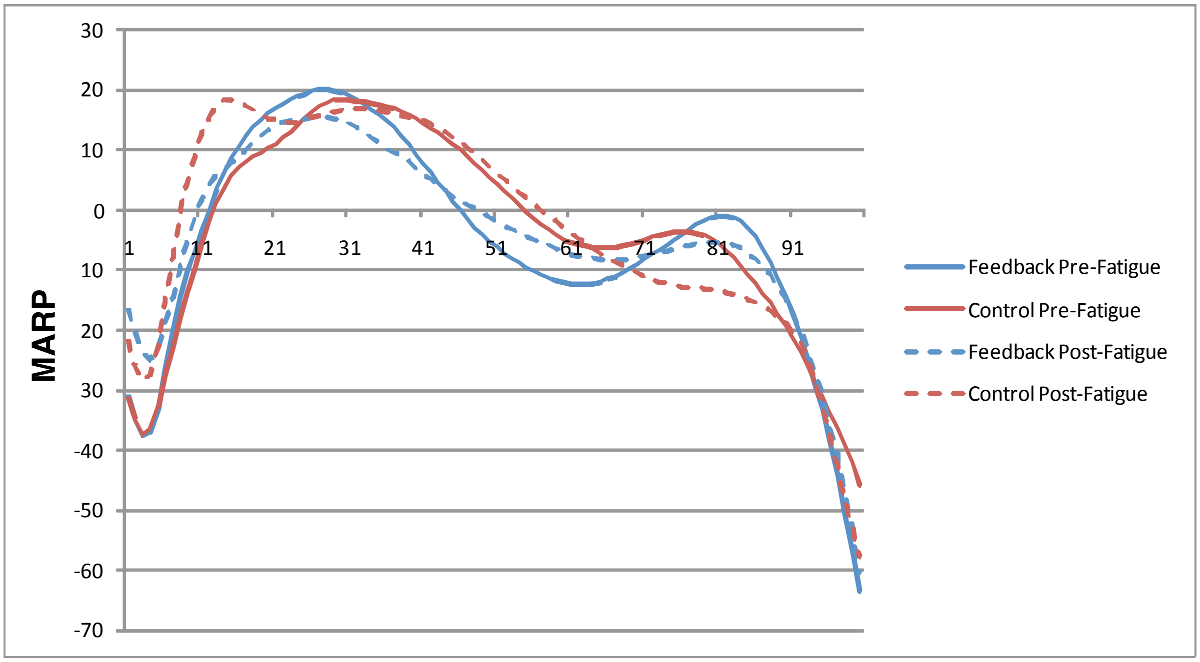
Foot-shank Frontal Plane RPA Curves, by Group and Fatigue



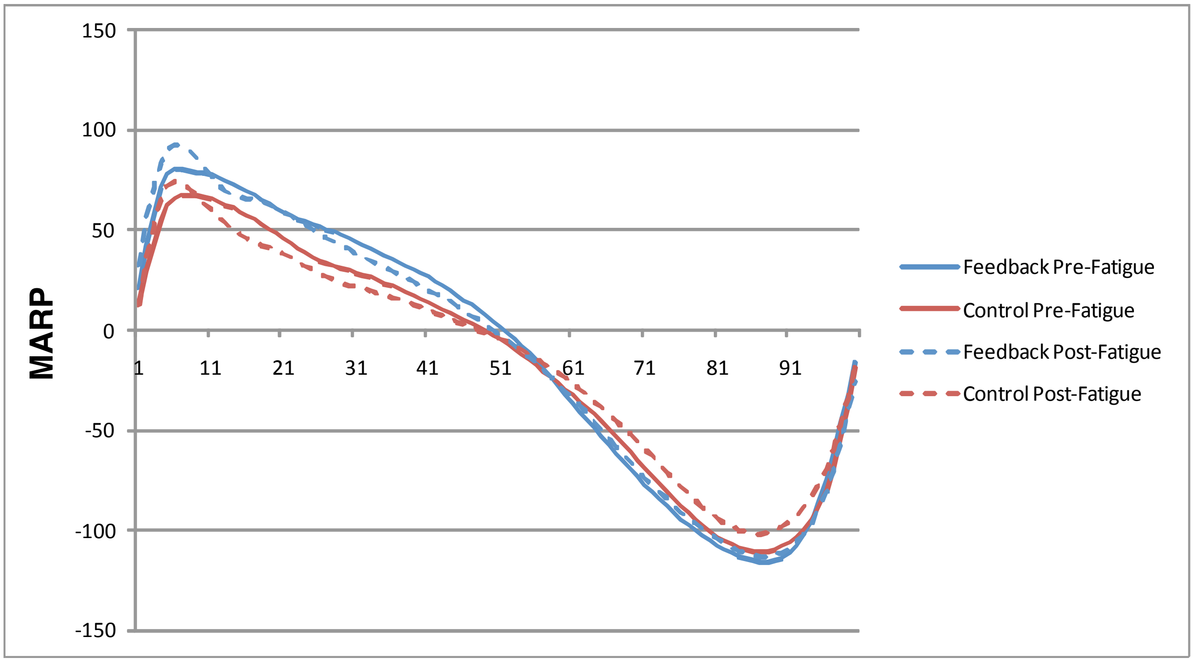
Shank-thigh Sagittal Plane RPA Curves, by Group and Fatigue



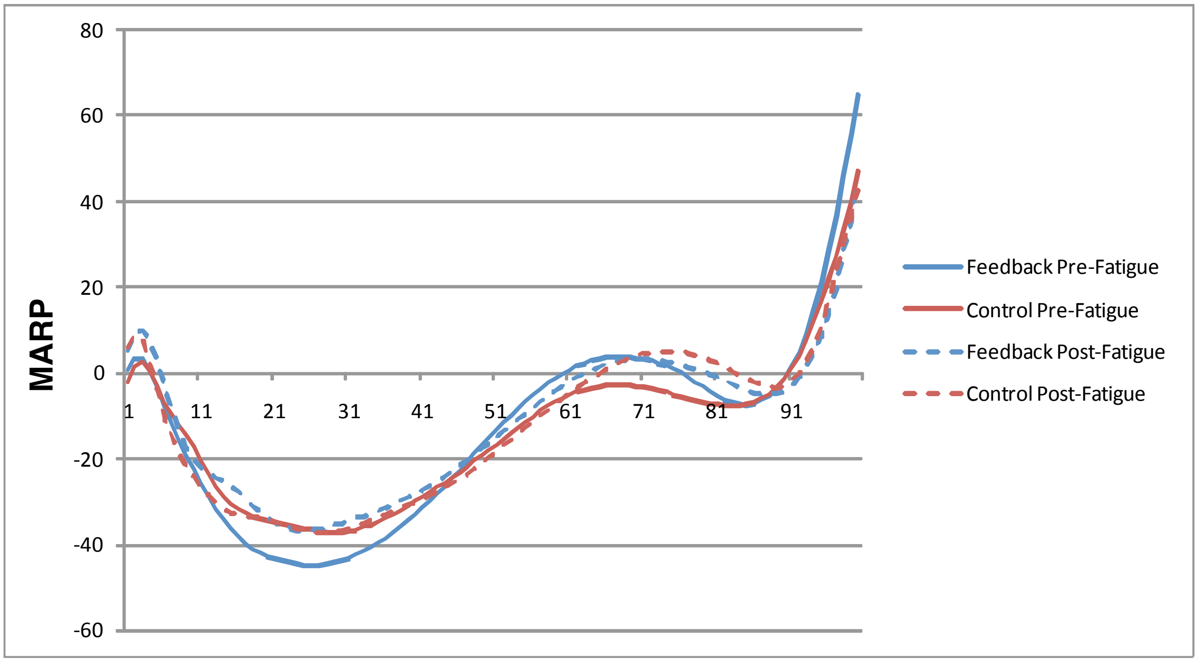
Shank-thigh Frontal Plane RPA Curves, by Group and Fatigue



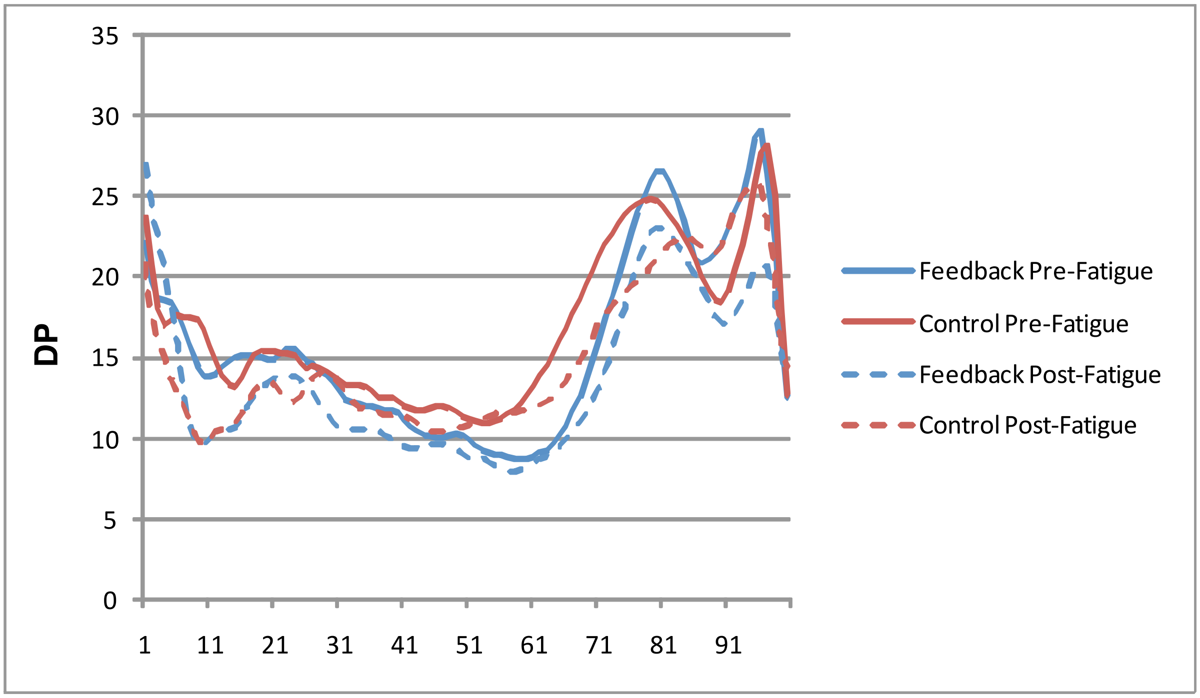
Thigh-trunk Sagittal Plane RPA Curves, by Group and Fatigue



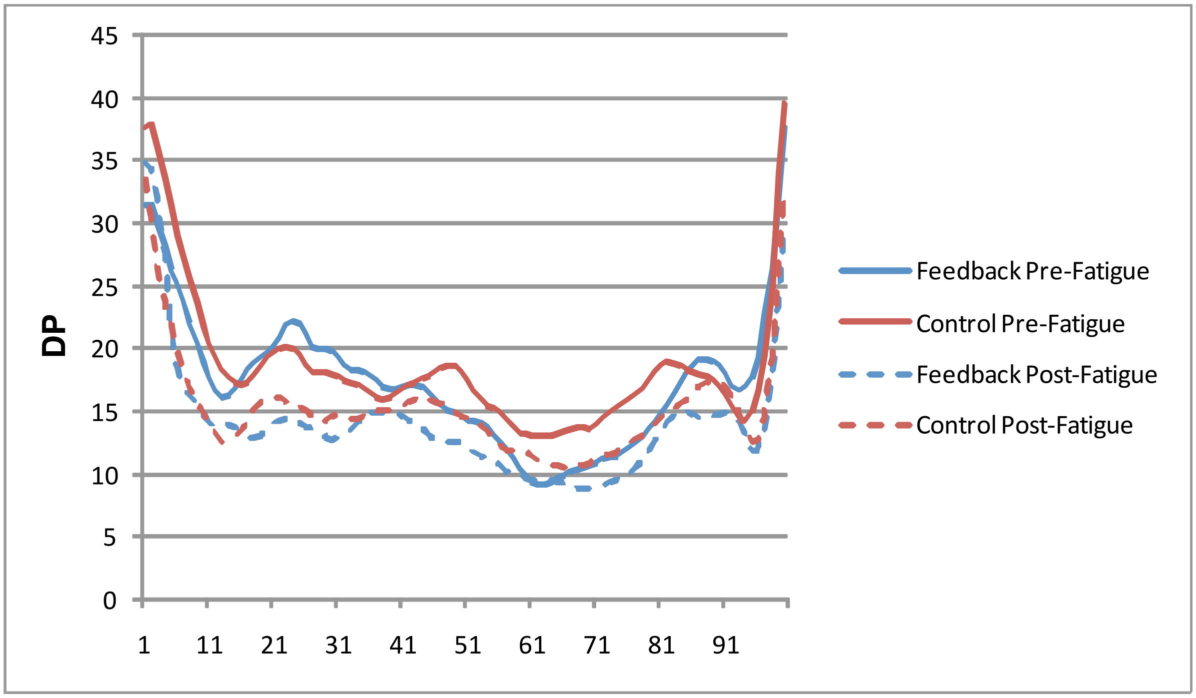
Thigh-trunk Frontal Plane RPA Curves, by Group and Fatigue



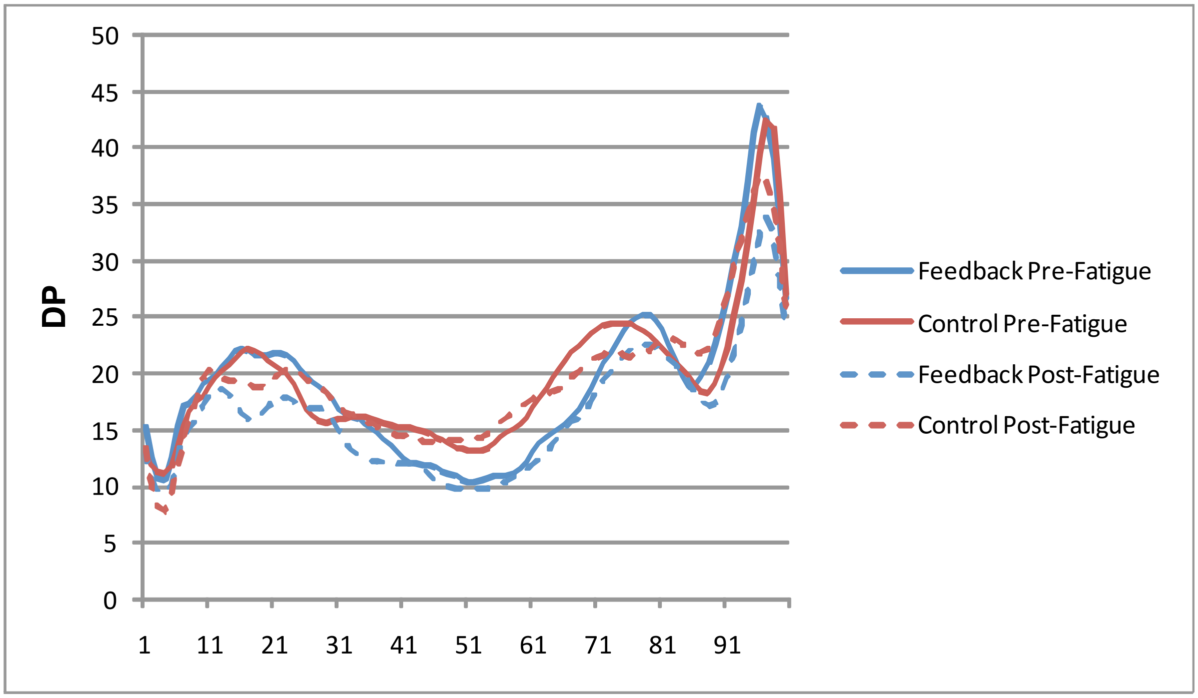
Foot-Shank Sagittal Plane DP Curves, by Group and Fatigue



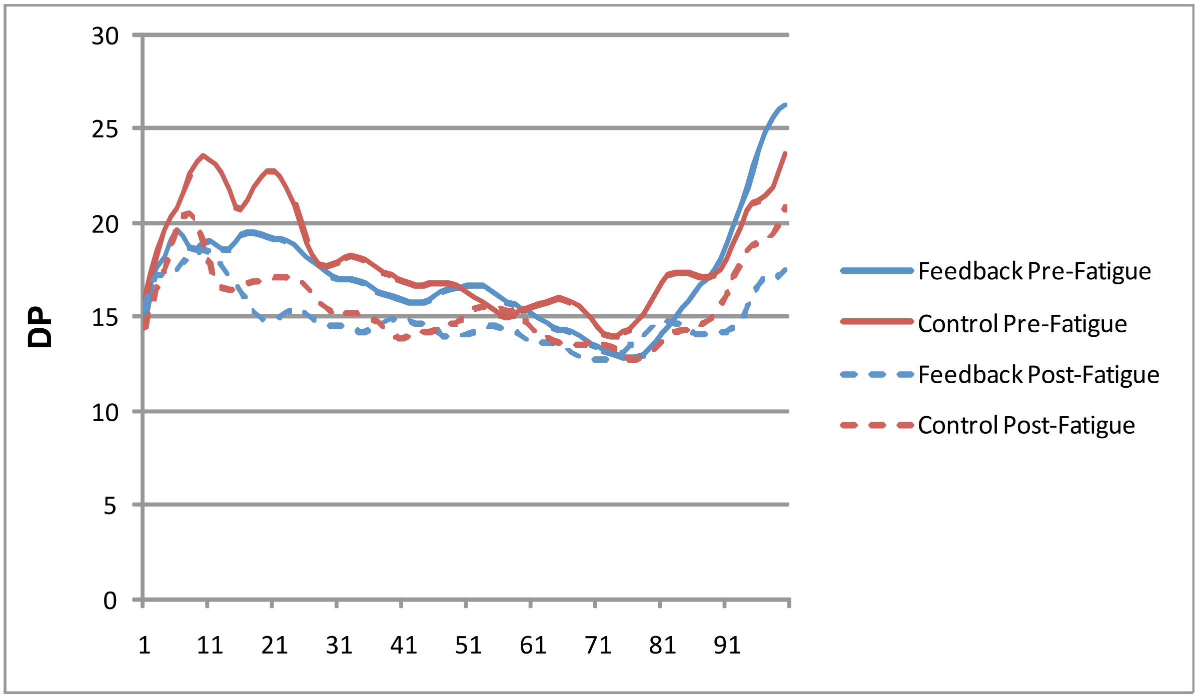
Foot-Shank Frontal Plane DP Curves, by Group and Fatigue



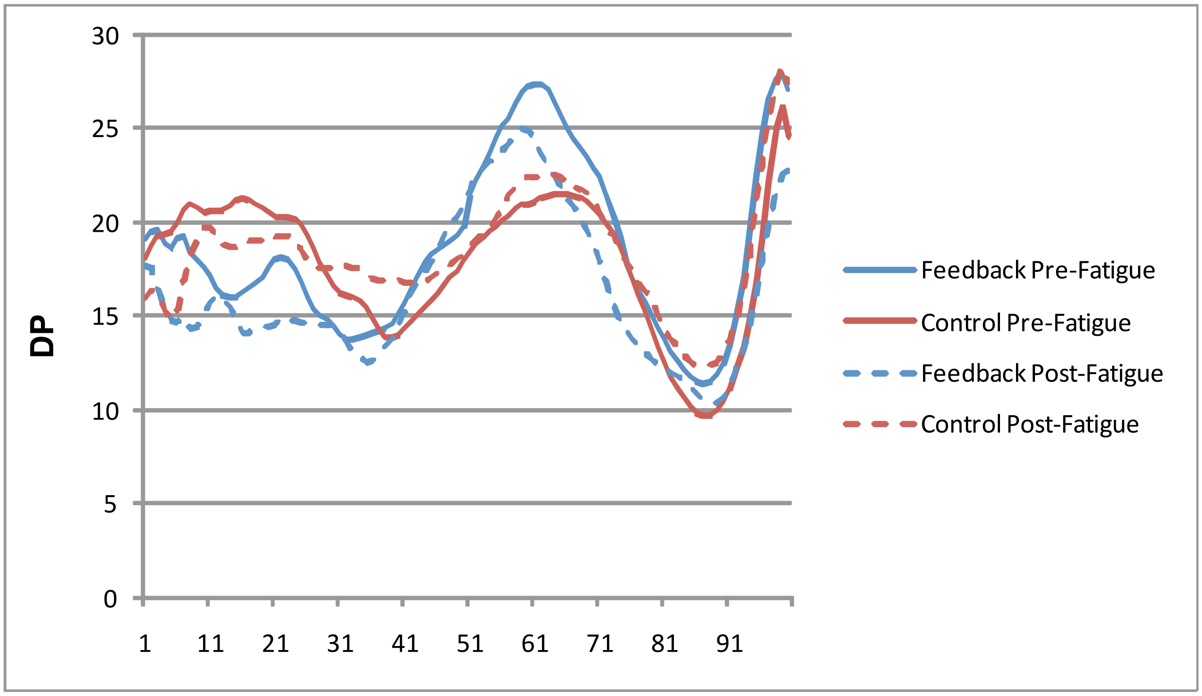
Shank-Thigh Sagittal Plane DP Curves, by Group and Fatigue



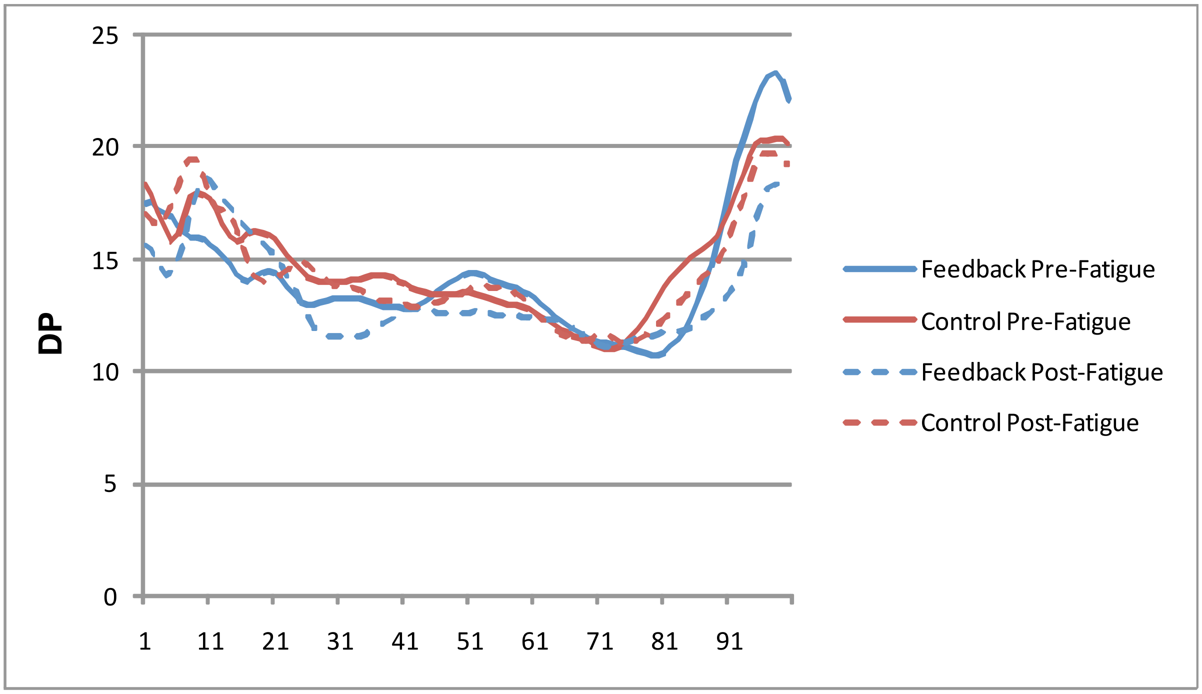
Shank-Thigh Frontal Plane DP Curves, by Group and Fatigue



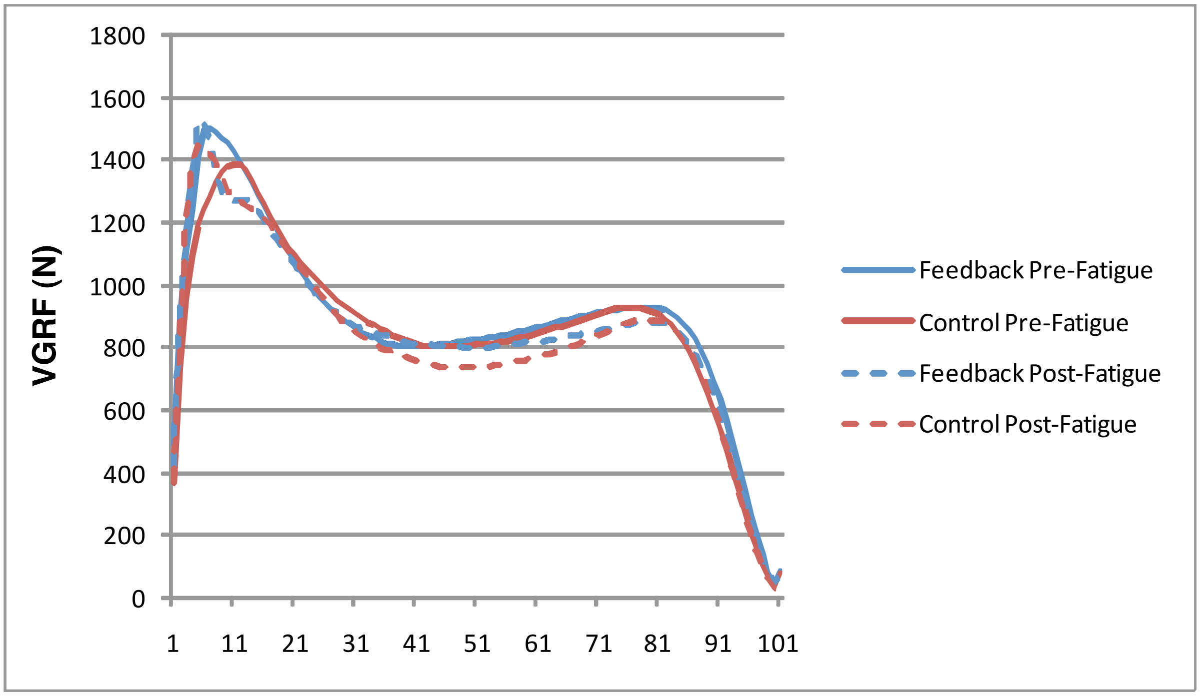
Thigh-Trunk Sagittal Plane DP Curves, by Group and Fatigue



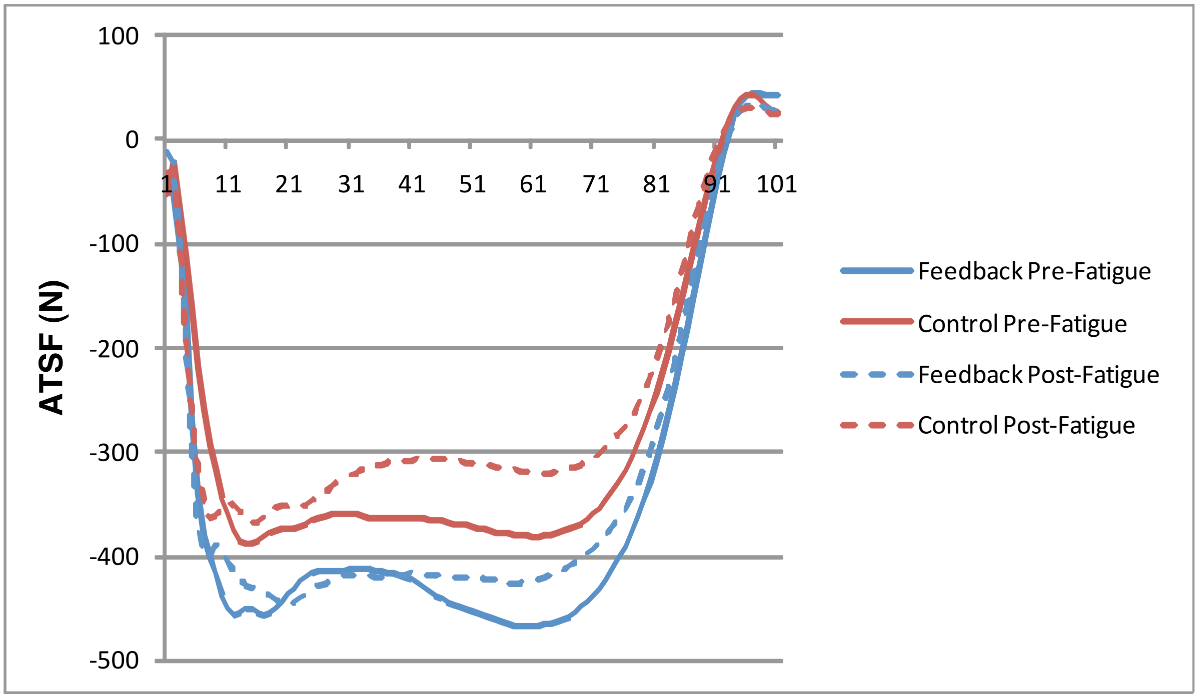
Thigh-Trunk Frontal Plane DP Curves, by Group and Fatigue



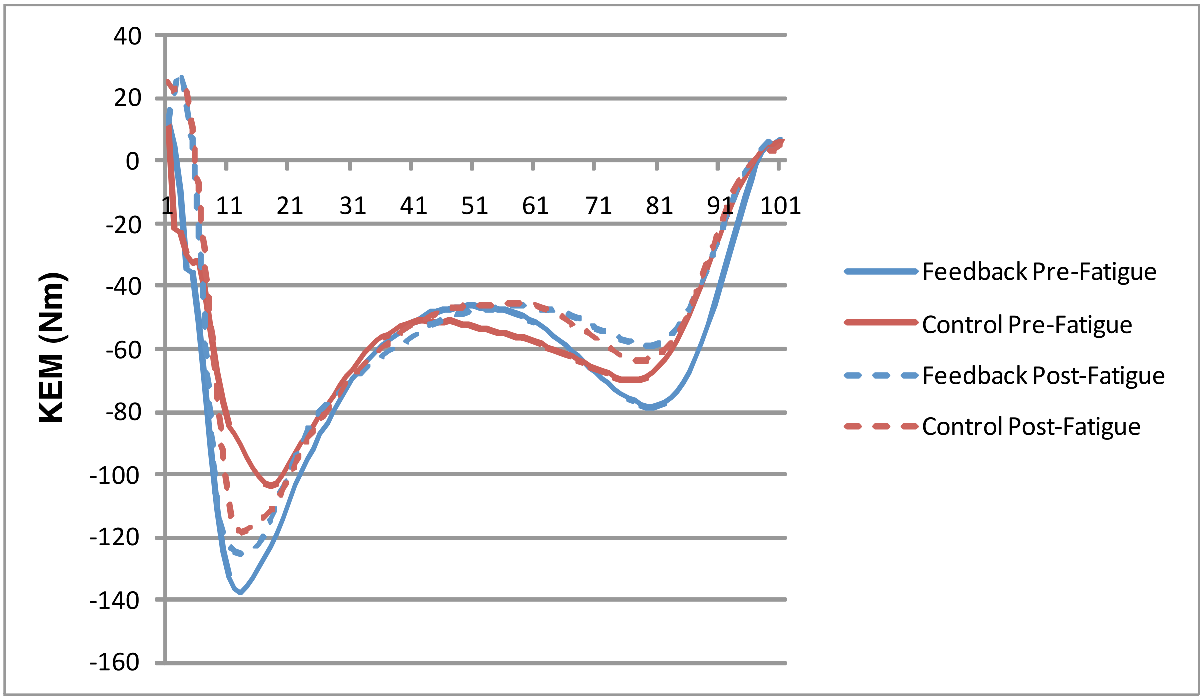
Vertical Ground Reaction Force Curves, by Group and Fatigue



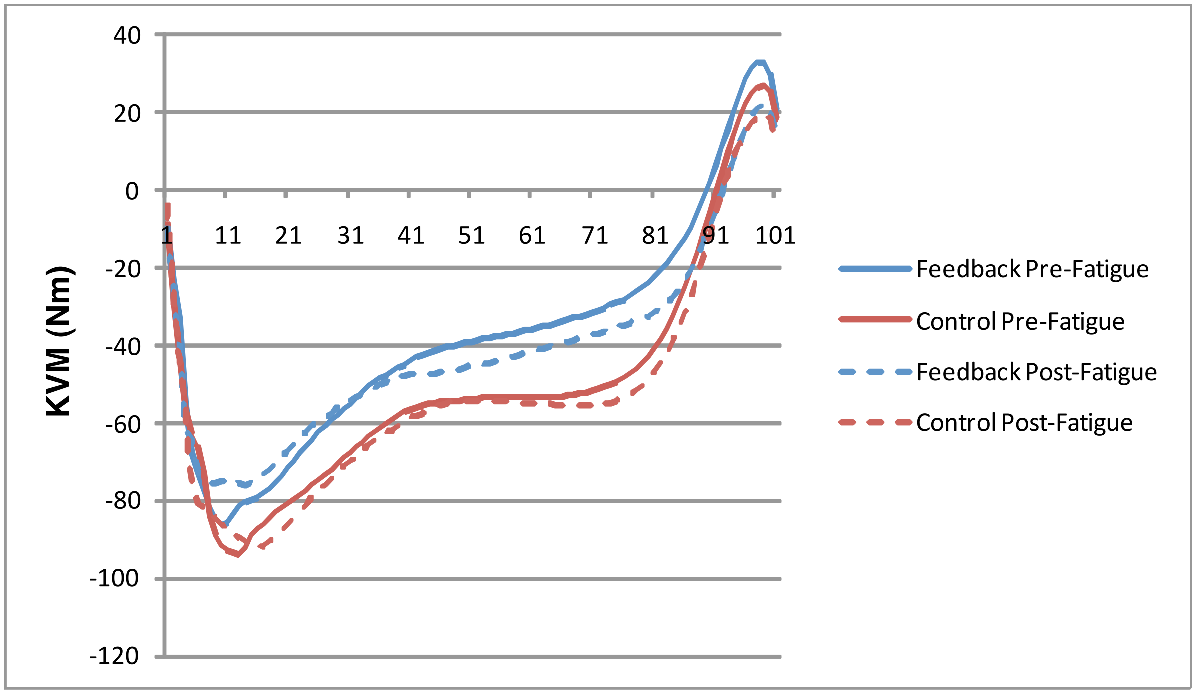
Anterior Tibial Shear Force Curves, by Group and Fatigue



Knee Extension Moment Curves, by Group and Fatigue



Knee Valgus Moment Curves, by Group and Fatigue



## REFERENCES


1. Gottlob CA, Baker CL, Jr., Pellissier JM, Colvin L. Cost effectiveness of anterior cruciate ligament reconstruction in young adults. *Clin Orthop Relat Res*. 1999:272-282.

2. Marshall SW, Padua DA, McGrath ML. Incidence of cruciate ligament (CL) injury in the United States, 1997-2004. *J Athl Train*. 2007;42:S-54.

3. Marshall SW, Padua DA, McGrath ML. Incidence of ACL injury. In: Hewett TE, Shultz SJ, Griffin LY, ed. *Understanding and Preventing Noncontact ACL Injuries*. Champaign, IL: Human Kinetics; 2007.

4. Roos H, Ornell M, Gardsell P, Lohmander LS, Lindstrand A. Soccer after anterior cruciate ligament injury--an incompatible combination? A national survey of incidence and risk factors and a 7-year follow-up of 310 players. *Acta Orthop Scand*. 1995;66:107-112.

5. Myklebust G, Bahr R. Return to play guidelines after anterior cruciate ligament surgery. *Br J Sports Med*. 2005;39:127-131.

6. Gelber AC, Hochberg MC, Mead LA, Wang NY, Wigley FM, Klag MJ. Joint injury in young adults and risk for subsequent knee and hip osteoarthritis. *Ann Intern Med*. 2000;133:321-328.

7. Roos EM. Joint injury causes knee osteoarthritis in young adults. *Curr Opin Rheumatol*. 2005;17:195-200.

8. Salmon LJ, Russell VJ, Refshauge K, Kader D, Connolly C, Linklater J, Pinczewski LA. Long-term outcome of endoscopic anterior cruciate ligament reconstruction with patellar tendon autograft: minimum 13-year review. *Am J Sports Med*. 2006;34:721-732.

9. Thelin N, Holmberg S, Thelin A. Knee injuries account for the sports-related increased risk of knee osteoarthritis. *Scand J Med Sci Sports*. 2006;16:329-333.

10. Gabbett TJ. Incidence, site, and nature of injuries in amateur rugby league over three consecutive seasons. *Br J Sports Med*. 2000;34:98-103.

11. Gabbett TJ. Incidence of injury in junior and senior rugby league players. *Sports Med*. 2004;34:849-859.

12. Hawkins RD, Fuller CW. A prospective epidemiological study of injuries in four English professional football clubs. *Br J Sports Med*. 1999;33:196-203.

13. Hawkins RD, Hulse MA, Wilkinson C, Hodson A, Gibson M. The association football medical research programme: an audit of injuries in professional football. *Br J Sports Med*. 2001;35:43-47.

14. Berns GS, Hull ML, Patterson HA. Strain in the anteromedial bundle of the anterior cruciate ligament under combination loading. *J Orthop Res*. 1992;10:167-176.

15. Markolf KL, Burchfield DM, Shapiro MM, Shepard MF, Finerman GA, Slauterbeck JL. Combined knee loading states that generate high anterior cruciate ligament forces. *J Orthop Res*. 1995;13:930-935.

16. Chaudhari AM, Andriacchi TP. The mechanical consequences of dynamic frontal plane limb alignment for non-contact ACL injury. *J Biomech*. 2006;39:330-338.

17. Besier TF, Lloyd DG, Cochrane JL, Ackland TR. External loading of the knee joint during running and cutting maneuvers. *Med Sci Sports Exerc*. 2001;33:1168-1175.

18. Chappell JD, Yu B, Kirkendall DT, Garrett WE. A comparison of knee kinetics between male and female recreational athletes in stop-jump tasks. *Am J Sports Med*. 2002;30:261-267.

19. Decker MJ, Torry MR, Wyland DJ, Sterett WI, Richard Steadman J. Gender differences in lower extremity kinematics, kinetics and energy absorption during landing. *Clin Biomech (Bristol, Avon)*. 2003;18:662-669.

20. Ford KR, Myer GD, Toms HE, Hewett TE. Gender differences in the kinematics of unanticipated cutting in young athletes. *Med Sci Sports Exerc*. 2005;37:124-129.

21. Hewett TE, Myer GD, Ford KR. Anterior cruciate ligament injuries in female athletes: Part 1, mechanisms and risk factors. *Am J Sports Med*. 2006;34:299-311.

22. Hewett TE, Zazulak BT, Myer GD, Ford KR. A review of electromyographic activation levels, timing differences, and increased anterior cruciate ligament injury incidence in female athletes. *Br J Sports Med*. 2005;39:347-350.

23. Huston LJ, Vibert B, Ashton-Miller JA, Wojtys EM. Gender differences in knee angle when landing from a drop-jump. *Am J Knee Surg*. 2001;14:215-219; discussion 219-220.

24. Kernozek TW, Torry MR, H VANH, Cowley H, Tanner S. Gender differences in frontal and sagittal plane biomechanics during drop landings. *Med Sci Sports Exerc*. 2005;37:1003-1012; discussion 1013.

25. Malinzak RA, Colby SM, Kirkendall DT, Yu B, Garrett WE. A comparison of knee joint motion patterns between men and women in selected athletic tasks. *Clin Biomech (Bristol, Avon)*. 2001;16:438-445.

26. McLean SG, Neal RJ, Myers PT, Walters MR. Knee joint kinematics during the sidestep cutting maneuver: potential for injury in women. *Med Sci Sports Exerc*. 1999;31:959-968.

27. Sigward SM, Powers CM. The influence of gender on knee kinematics, kinetics and muscle activation patterns during side-step cutting. *Clin Biomech (Bristol, Avon)*. 2006;21:41-48.

28. Yu B, Lin CF, Garrett WE. Lower extremity biomechanics during the landing of a stop-jump task. *Clin Biomech (Bristol, Avon)*. 2006;21:297-305.

29. Zazulak BT, Hewett TE, Reeves NP, Goldberg B, Cholewicki J. Deficits in neuromuscular control of the trunk predict knee injury risk: a prospective biomechanical-epidemiologic study. *Am J Sports Med*. 2007;35:1123-1130.

30. Zazulak BT, Hewett TE, Reeves NP, Goldberg B, Cholewicki J. The effects of core proprioception on knee injury: a prospective biomechanical-epidemiological study. *Am J Sports Med*. 2007;35:368-373.

31. DeMorat G, Weinhold P, Blackburn T, Chudik S, Garrett W. Aggressive quadriceps loading can induce noncontact anterior cruciate ligament injury. *Am J Sports Med*. 2004;32:477-483.

32. Sell TC, Ferris CM, Abt JP, Tsai YS, Myers JB, Fu FH, Lephart SM. Predictors of proximal tibia anterior shear force during a vertical stop-jump. *J Orthop Res*. 2007;25:1589-1597.

33. Withrow TJ, Huston LJ, Wojtys EM, Ashton-Miller JA. The relationship between quadriceps muscle force, knee flexion, and anterior cruciate ligament strain in an in vitro simulated jump landing. *Am J Sports Med*. 2006;34:269-274.

34. Hewett TE, Myer GD, Ford KR, Heidt RS, Jr., Colosimo AJ, McLean SG, van den Bogert AJ, Paterno MV, Succop P. Biomechanical measures of neuromuscular control and valgus loading of the knee predict anterior cruciate ligament injury risk in female athletes: a prospective study. *Am J Sports Med*. 2005;33:492-501.

35. Davids K, Glazier P, Araujo D, Bartlett R. Movement systems as dynamical systems: the functional role of variability and its implications for sports medicine. *Sports Med*. 2003;33:245-260.

36. Kurz MJ, & Stergiou, N. Applied Dynamic Systems Theory for the analysis of movement. In: Stergiou N, ed. *Innovative Analyses of Human Movement*. Champaign, IL: Human Kinetics; 2004:93-120.

37. van Emmerik REA, & van Wegen, E. E. H. On variability and stability in human movement. *Journal of Applied Biomechanics*. 2000;16:394-406.

38. Hamill J, van Emmerik RE, Heiderscheit BC, Li L. A dynamical systems approach to lower extremity running injuries. *Clin Biomech (Bristol, Avon)*. 1999;14:297-308.

39. Kurz MJ, Stergiou N, Buzzi UH, Georgoulis AD. The effect of anterior cruciate ligament reconstruction on lower extremity relative phase dynamics during walking and running. *Knee Surg Sports Traumatol Arthrosc*. 2005;13:107-115.

40. van Uden CJ, Bloo JK, Kooloos JG, van Kampen A, de Witte J, Wagenaar RC. Coordination and stability of one-legged hopping patterns in patients with anterior cruciate ligament reconstruction: preliminary results. *Clin Biomech (Bristol, Avon)*. 2003;18:84-87.

41. Borotikar BS, Newcomer R, Koppes R, McLean SG. Combined effects of fatigue and decision making on female lower limb landing postures: central and peripheral contributions to ACL injury risk. *Clin Biomech (Bristol, Avon)*. 2008;23:81-92.

42. Chappell JD, Herman DC, Knight BS, Kirkendall DT, Garrett WE, Yu B. Effect of fatigue on knee kinetics and kinematics in stop-jump tasks. *Am J Sports Med*. 2005;33:1022-1029.

43. McLean SG, Felin RE, Suedekum N, Calabrese G, Passerallo A, Joy S. Impact of fatigue on gender-based high-risk landing strategies. *Med Sci Sports Exerc*. 2007;39:502-514.

44. Kernozek TW, Torry MR, Iwasaki M. Gender differences in lower extremity landing mechanics caused by neuromuscular fatigue. *Am J Sports Med*. 2008;36:554-565.

45. Benjaminse A, Habu A, Sell TC, Abt JP, Fu FH, Myers JB, Lephart SM. Fatigue alters lower extremity kinematics during a single-leg stop-jump task. *Knee Surg Sports Traumatol Arthrosc*. 2008;16:400-407.

46. Hewett TE, Ford KR, Myer GD. Anterior cruciate ligament injuries in female athletes: Part 2, a meta-analysis of neuromuscular interventions aimed at injury prevention. *Am J Sports Med*. 2006;34:490-498.

47. Padua DA, Marshall, S.M. Evidence supporting ACL-injury-prevention exercise programs: A review of the literature. *Athletic Therapy Today*. 2006;11:11-23.

48. Cowling EJ, Steele JR, McNair PJ. Effect of verbal instructions on muscle activity and risk of injury to the anterior cruciate ligament during landing. *Br J Sports Med*. 2003;37:126-130.

49. McNair PJ, Prapavessis H, Callender K. Decreasing landing forces: effect of instruction. *Br J Sports Med*. 2000;34:293-296.

50. Prapavessis H, McNair PJ. Effects of instruction in jumping technique and experience jumping on ground reaction forces. *J Orthop Sports Phys Ther*. 1999;29:352-356.

51. Onate JA, Guskiewicz KM, Marshall SW, Giuliani C, Yu B, Garrett WE. Instruction of jump-landing technique using videotape feedback: altering lower extremity motion patterns. *Am J Sports Med*. 2005;33:831-842.

52. Yu B, Kirkendall, D.T., Taft, T.N., & Garrett, W.E. Lower extremity motor control-related and other risk factors for noncontact anterior cruciate ligament injuries. In: Beaty JH, ed. *Instructional Course Lectures*. Vol. 51. Rosemont, IL: American Academy of Orthopaedic Surgeons; 2002:315-324.

53. Cascio BM, Culp L, Cosgarea AJ. Return to play after anterior cruciate ligament reconstruction. *Clin Sports Med*. 2004;23:395-408, ix.

54. Boden BP, Dean GS, Feagin JA, Jr., Garrett WE, Jr. Mechanisms of anterior cruciate ligament injury. *Orthopedics*. 2000;23:573-578.

55. Agel J, Arendt EA, Bershadsky B. Anterior cruciate ligament injury in national collegiate athletic association basketball and soccer: a 13-year review. *Am J Sports Med*. 2005;33:524-530.

56. Arendt E, Dick R. Knee injury patterns among men and women in collegiate basketball and soccer. NCAA data and review of literature. *Am J Sports Med*. 1995;23:694-701.

57. Arendt EA, Agel J, Dick R. Anterior Cruciate Ligament Injury Patterns Among Collegiate Men and Women. *J Athl Train*. 1999;34:86-92.

58. Hootman JM, Dick R, Agel J. Epidemiology of collegiate injuries for 15 sports: summary and recommendations for injury prevention initiatives. *J Athl Train*. 2007;42:311-319.

59. Myklebust G, Maehlum S, Engebretsen L, Strand T, Solheim E. Registration of cruciate ligament injuries in Norwegian top level team handball. A prospective study covering two seasons. *Scand J Med Sci Sports*. 1997;7:289-292.

60. Krosshaug T, Bahr, R. Biomechanics Associated with Injury. In: Hewett TE, Shultz, S.J., Griffin L.Y., ed. *Understanding and Preventing Noncontact ACL Injuries*. Champaign, IL: Human Kinetics; 2007:131-140.

61. Olsen OE, Myklebust G, Engebretsen L, Bahr R. Injury mechanisms for anterior cruciate ligament injuries in team handball: a systematic video analysis. *Am J Sports Med*. 2004;32:1002-1012.

62. de Loes M, Dahlstedt LJ, Thomee R. A 7-year study on risks and costs of knee injuries in male and female youth participants in 12 sports. *Scand J Med Sci Sports*. 2000;10:90-97.

63. Mihata LC, Beutler AI, Boden BP. Comparing the incidence of anterior cruciate ligament injury in collegiate lacrosse, soccer, and basketball players: implications for

anterior cruciate ligament mechanism and prevention. *Am J Sports Med*. 2006;34:899-904.

64. Conaghan PG. Update on osteoarthritis part 1: current concepts and the relation to exercise. *Br J Sports Med*. 2002;36:330-333.

65. Maletius W, Messner K. Eighteen- to twenty-four-year follow-up after complete rupture of the anterior cruciate ligament. *Am J Sports Med*. 1999;27:711-717.

66. Shelbourne KD, Gray T. Results of anterior cruciate ligament reconstruction based on meniscus and articular cartilage status at the time of surgery. Five- to fifteen-year evaluations. *Am J Sports Med*. 2000;28:446-452.

67. Ireland ML. The female ACL: why is it more prone to injury? *Orthop Clin North Am*. 2002;33:637-651.

68. Griffin LY, Albohm MJ, Arendt EA, Bahr R, Beynnon BD, Demaio M, Dick RW, Engebretsen L, Garrett WE, Jr., Hannafin JA, Hewett TE, Huston LJ, Ireland ML, Johnson RJ, Lephart S, Mandelbaum BR, Mann BJ, Marks PH, Marshall SW, Myklebust G, Noyes FR, Powers C, Shields C, Jr., Shultz SJ, Silvers H, Slauterbeck J, Taylor DC, Teitz CC, Wojtys EM, Yu B. Understanding and preventing noncontact anterior cruciate ligament injuries: a review of the Hunt Valley II meeting, January 2005. *Am J Sports Med*. 2006;34:1512-1532.

69. Torg JS, Quedenfeld TC, Landau S. The shoe-surface interface and its relationship to football knee injuries. *J Sports Med*. 1974;2:261-269.

70. Torg JS, Quedenfeld T. Effect of shoe type and cleat length on incidence and severity of knee injuries among high school football players. *Res Q*. 1971;42:203-211.

71. Bonstingl RW, Morehouse CA, Niebel BW. Torques developed by different types of shoes on various playing surfaces. *Med Sci Sports*. 1975;7:127-131.

72. Livesay GA, Reda DR, Nauman EA. Peak torque and rotational stiffness developed at the shoe-surface interface: the effect of shoe type and playing surface. *Am J Sports Med*. 2006;34:415-422.

73. Nigg BM, Segesser B. The influence of playing surfaces on the load on the locomotor system and on football and tennis injuries. *Sports Med*. 1988;5:375-385.

74. Myklebust G, Engebretsen L, Braekken IH, Skjolberg A, Olsen OE, Bahr R. Prevention of anterior cruciate ligament injuries in female team handball players: a prospective intervention study over three seasons. *Clin J Sport Med*. 2003;13:71-78.

75. Renstrom P, Arms SW, Stanwyck TS, Johnson RJ, Pope MH. Strain within the anterior cruciate ligament during hamstring and quadriceps activity. *Am J Sports Med*. 1986;14:83-87.

76. Orchard J. Is there a relationship between ground and climatic conditions and injuries in football? *Sports Med*. 2002;32:419-432.

77. Orchard J, Seward H, McGivern J, Hood S. Intrinsic and extrinsic risk factors for anterior cruciate ligament injury in Australian footballers. *Am J Sports Med*. 2001;29:196-200.

78. Orchard JW, Powell JW. Risk of knee and ankle sprains under various weather conditions in American football. *Med Sci Sports Exerc*. 2003;35:1118-1123.

79. Uhorchak JM, Scoville CR, Williams GN, Arciero RA, St Pierre P, Taylor DC. Risk factors associated with noncontact injury of the anterior cruciate ligament: a prospective four-year evaluation of 859 West Point cadets. *Am J Sports Med*. 2003;31:831-842.

80. Lombardo S, Sethi PM, Starkey C. Intercondylar notch stenosis is not a risk factor for anterior cruciate ligament tears in professional male basketball players: an 11-year prospective study. *Am J Sports Med*. 2005;33:29-34.

81. Schickendantz MS, Weiker GG. The predictive value of radiographs in the evaluation of unilateral and bilateral anterior cruciate ligament injuries. *Am J Sports Med*. 1993;21:110-113.

82. Teitz CC, Lind BK, Sacks BM. Symmetry of the femoral notch width index. *Am J Sports Med*. 1997;25:687-690.

83. Chandrashekar N, Mansouri H, Slauterbeck J, Hashemi J. Sex-based differences in the tensile properties of the human anterior cruciate ligament. *J Biomech*. 2006;39:2943-2950.

84. Chandrashekar N, Slauterbeck J, Hashemi J. Sex-based differences in the anthropometric characteristics of the anterior cruciate ligament and its relation to intercondylar notch geometry: a cadaveric study. *Am J Sports Med*. 2005;33:1492-1498.

85. Brandon ML, Haynes PT, Bonamo JR, Flynn MI, Barrett GR, Sherman MF. The association between posterior-inferior tibial slope and anterior cruciate ligament insufficiency. *Arthroscopy*. 2006;22:894-899.

86. Stijak L, Herzog RF, Schai P. Is there an influence of the tibial slope of the lateral condyle on the ACL lesion? A case-control study. *Knee Surg Sports Traumatol Arthrosc*. 2008;16:112-117.

87. Woodford-Rogers B, Cyphert L, Denegar CR. Risk Factors for Anterior Cruciate Ligament Injury in High School and College Athletes. *J Athl Train*. 1994;29:343-346.

88. Beckett ME, Massie DL, Bowers KD, Stoll DA. Incidence of Hyperpronation in the ACL Injured Knee: A Clinical Perspective. *J Athl Train*. 1992;27:58-62.

89. Hertel J, Dorfman JH, Braham RA. Lower extremity malalignments and anterior cruciate ligament injury history. *Journal of Sports Science and Medicine*. 2004;3:220-225.

90. Loudon JK, Jenkins W, Loudon KL. The relationship between static posture and ACL injury in female athletes. *J Orthop Sports Phys Ther*. 1996;24:91-97.

91. Liu SH, al-Shaikh R, Panossian V, Yang RS, Nelson SD, Soleiman N, Finerman GA, Lane JM. Primary immunolocalization of estrogen and progesterone target cells in the human anterior cruciate ligament. *J Orthop Res*. 1996;14:526-533.

92. Liu SH, Al-Shaikh RA, Panossian V, Finerman GA, Lane JM. Estrogen affects the cellular metabolism of the anterior cruciate ligament. A potential explanation for female athletic injury. *Am J Sports Med*. 1997;25:704-709.

93. Hamlet WP, Liu SH, Panossian V, Finerman GA. Primary immunolocalization of androgen target cells in the human anterior cruciate ligament. *J Orthop Res*. 1997;15:657-663.

94. Dragoo JL, Lee RS, Benhaim P, Finerman GA, Hame SL. Relaxin receptors in the human female anterior cruciate ligament. *Am J Sports Med*. 2003;31:577-584.

95. Arendt EA, Bershadsky B, Agel J. Periodicity of noncontact anterior cruciate ligament injuries during the menstrual cycle. *J Gend Specif Med*. 2002;5:19-26.

96. Beynnon BD, Johnson RJ, Braun S, Sargent M, Bernstein IM, Skelly JM, Vacek PM. The relationship between menstrual cycle phase and anterior cruciate ligament injury: a case-control study of recreational alpine skiers. *Am J Sports Med*. 2006;34:757-764.

97. Myklebust G, Maehlum S, Holm I, Bahr R. A prospective cohort study of anterior cruciate ligament injuries in elite Norwegian team handball. *Scand J Med Sci Sports*. 1998;8:149-153.

98. Wojtys EM, Huston LJ, Boynton MD, Spindler KP, Lindenfeld TN. The effect of the menstrual cycle on anterior cruciate ligament injuries in women as determined by hormone levels. *Am J Sports Med*. 2002;30:182-188.

99. Shultz SJ, Sander TC, Kirk SE, Perrin DH. Sex differences in knee joint laxity change across the female menstrual cycle. *J Sports Med Phys Fitness*. 2005;45:594-603.

100. Shultz SJ, Gansneder BM, Sander TC, Kirk SE, Perrin DH. Absolute serum hormone levels predict the magnitude of change in anterior knee laxity across the menstrual cycle. *J Orthop Res*. 2006;24:124-131.

101. Heitz NA, Eisenman PA, Beck CL, Walker JA. Hormonal Changes Throughout the Menstrual Cycle and Increased Anterior Cruciate Ligament Laxity in Females. *J Athl Train*. 1999;34:144-149.

102. Shultz SJ, Kirk SE, Johnson ML, Sander TC, Perrin DH. Relationship between sex hormones and anterior knee laxity across the menstrual cycle. *Med Sci Sports Exerc*. 2004;36:1165-1174.

103. Barber-Westin SD, Noyes FR, Galloway M. Jump-land characteristics and muscle strength development in young athletes: a gender comparison of 1140 athletes 9 to 17 years of age. *Am J Sports Med*. 2006;34:375-384.

104. Chappell JD, Creighton RA, Giuliani C, Yu B, Garrett WE. Kinematics and Electromyography of Landing Preparation in Vertical Stop-Jump: Risks for Noncontact Anterior Cruciate Ligament Injury. *Am J Sports Med*. 2006.

105. Ford KR, Myer GD, Hewett TE. Valgus knee motion during landing in high school female and male basketball players. *Med Sci Sports Exerc*. 2003;35:1745-1750.

106. Huston LJ. Clinical Biomechanical Studies on ACL Injury Risk Factors. In: Hewett TE, Shultz, S.J., Griffin L.Y., ed. *Understanding and Preventing Noncontact ACL Injuries*. Champaign, IL: Human Kinetics; 2007:141-154.

107. Lephart SM, Ferris CM, Riemann BL, Myers JB, Fu FH. Gender differences in strength and lower extremity kinematics during landing. *Clin Orthop Relat Res*. 2002:162-169.

108. McLean SG, Lipfert SW, van den Bogert AJ. Effect of gender and defensive opponent on the biomechanics of sidestep cutting. *Med Sci Sports Exerc*. 2004;36:1008-1016.

109. Noyes FR, Barber-Westin SD, Fleckenstein C, Walsh C, West J. The drop-jump screening test: difference in lower limb control by gender and effect of neuromuscular training in female athletes. *Am J Sports Med*. 2005;33:197-207.

110. Pollard CD, Davis IM, Hamill J. Influence of gender on hip and knee mechanics during a randomly cued cutting maneuver. *Clin Biomech (Bristol, Avon)*. 2004;19:1022-1031.

111. Salci Y, Kentel BB, Heycan C, Akin S, Korkusuz F. Comparison of landing maneuvers between male and female college volleyball players. *Clin Biomech (Bristol, Avon)*. 2004;19:622-628.

112. Fleming BC, Renstrom PA, Beynnon BD, Engstrom B, Peura GD, Badger GJ, Johnson RJ. The effect of weightbearing and external loading on anterior cruciate ligament strain. *J Biomech*. 2001;34:163-170.

113. Nunley RM, Wright D, Renner JB, Yu B, Garrett WE. Gender Comparison of Patellar Tendon Tibial Shaft Angle with Weight Bearing. *Res Sports Med*. 2003;11:173 - 185.

114. Winter DA. *Biomechanics and Motor Control of Human Movement* 3rd ed. Hoboken, NJ: John Wiley & Sons; 2005.

115. McLean SG, Huang X, Su A, Van Den Bogert AJ. Sagittal plane biomechanics cannot injure the ACL during sidestep cutting. *Clin Biomech (Bristol, Avon)*. 2004;19:828-838.

116. Simonsen EB, Magnusson SP, Bencke J, Naesborg H, Havkrog M, Ebstrup JF, Sorensen H. Can the hamstring muscles protect the anterior cruciate ligament during a side-cutting maneuver? *Scand J Med Sci Sports*. 2000;10:78-84.

117. Woo SL, Hollis JM, Adams DJ, Lyon RM, Takai S. Tensile properties of the human femur-anterior cruciate ligament-tibia complex. The effects of specimen age and orientation. *Am J Sports Med*. 1991;19:217-225.

118. Sell TC, Ferris CM, Abt JP, Tsai YS, Myers JB, Fu FH, Lephart SM. The effect of direction and reaction on the neuromuscular and biomechanical characteristics of the knee during tasks that simulate the noncontact anterior cruciate ligament injury mechanism. *Am J Sports Med*. 2006;34:43-54.

119. Withrow TJ, Huston LJ, Wojtys EM, Ashton-Miller JA. The effect of an impulsive knee valgus moment on in vitro relative ACL strain during a simulated jump landing. *Clin Biomech (Bristol, Avon)*. 2006;21:977-983.

120. Weinhold PS, Stewart JD, Liu HY, Lin CF, Garrett WE, Yu B. The influence of gender-specific loading patterns of the stop-jump task on anterior cruciate ligament strain. *Injury*. 2007.

121. Ford KR, Myer GD, Smith RL, Vianello RM, Seiwert SL, Hewett TE. A comparison of dynamic coronal plane excursion between matched male and female athletes when performing single leg landings. *Clin Biomech (Bristol, Avon)*. 2006;21:33-40.

122. McLean SG, Walker KB, van den Bogert AJ. Effect of gender on lower extremity kinematics during rapid direction changes: an integrated analysis of three sports movements. *J Sci Med Sport*. 2005;8:411-422.

123. Pollard CD, Sigward SM, Powers CM. Gender differences in hip joint kinematics and kinetics during side-step cutting maneuver. *Clin J Sport Med*. 2007;17:38-42.

124. Dempsey AR, Lloyd DG, Elliott BC, Steele JR, Munro BJ, Russo KA. The effect of technique change on knee loads during sidestep cutting. *Med Sci Sports Exerc*. 2007;39:1765-1773.

125. Gupta RT, Vankoski S, Novak RA, Dias LS. Trunk kinematics and the influence on valgus knee stress in persons with high sacral level myelomeningocele. *J Pediatr Orthop*. 2005;25:89-94.

126. Blackburn JT, Padua DA. Influence of trunk flexion on hip and knee joint kinematics during a controlled drop landing. *Clin Biomech (Bristol, Avon)*. 2008;23:313-319.

127. Enoka RM. *Neuromechanics of Human Movement*. 3rd ed. Champaign, IL: Human Kinetics; 2002.

128. Gandevia SC. Spinal and supraspinal factors in human muscle fatigue. *Physiol Rev*. 2001;81:1725-1789.

129. Skof B, & Strojnik, V. Neuromuscular fatigue and recovery dynamics following prolonged continuous run at anaerobic threshold. *Br J Sports Med*. 2006;40:219-222.

130. Kirkendall DT. Mechanisms of peripheral fatigue. *Med Sci Sports Exerc*. 1990;22:444-449.

131. Lattier G, Millet GY, Martin A, Martin V. Fatigue and recovery after high-intensity exercise part I: neuromuscular fatigue. *Int J Sports Med*. 2004;25:450-456.

132. Greig M. The influence of soccer-specific fatigue on peak isokinetic torque production of the knee flexors and extensors. *Am J Sports Med*. 2008;36:1403-1409.

133. Rahnama N, Reilly T, Lees A, Graham-Smith P. Muscle fatigue induced by exercise simulating the work rate of competitive soccer. *J Sports Sci*. 2003;21:933-942.

134. Theurel J, Lepers R. Neuromuscular fatigue is greater following highly variable versus constant intensity endurance cycling. *Eur J Appl Physiol*. 2008;103:461-468.

135. Thomas R, Stephane P. Prefrontal cortex oxygenation and neuromuscular responses to exhaustive exercise. *Eur J Appl Physiol*. 2008;102:153-163.

136. Amann M, Dempsey JA. Locomotor muscle fatigue modifies central motor drive in healthy humans and imposes a limitation to exercise performance. *J Physiol*. 2008;586:161-173.

137. Amann M, Eldridge MW, Lovering AT, Stickland MK, Pegelow DF, Dempsey JA. Arterial oxygenation influences central motor output and exercise performance via effects on peripheral locomotor muscle fatigue in humans. *J Physiol*. 2006;575:937-952.

138. Amann M, Romer LM, Subudhi AW, Pegelow DF, Dempsey JA. Severity of arterial hypoxaemia affects the relative contributions of peripheral muscle fatigue to exercise performance in healthy humans. *J Physiol*. 2007;581:389-403.

139. Amann M, Dempsey JA. The concept of peripheral locomotor muscle fatigue as a regulated variable. *J Physiol*. 2008.

140. Dempsey JA, Amann M, Romer LM, Miller JD. Respiratory system determinants of peripheral fatigue and endurance performance. *Med Sci Sports Exerc*. 2008;40:457-461.

141. Nicol C, Avela J, Komi PV. The stretch-shortening cycle : a model to study naturally occurring neuromuscular fatigue. *Sports Med*. 2006;36:977-999.

142. Tomporowski PD. Effects of acute bouts of exercise on cognition. *Acta Psychol (Amst)*. 2003;112:297-324.

143. Gleeson NP, Reilly T, Mercer TH, Rakowski S, Rees D. Influence of acute endurance activity on leg neuromuscular and musculoskeletal performance. *Med Sci Sports Exerc*. 1998;30:596-608.

144. Bangsbo J. Energy demands in competitive soccer. *J Sports Sci*. 1994;12 Spec No:S5-12.

145. Bangsbo J, Norregaard L, Thorso F. Activity profile of competition soccer. *Can J Sport Sci*. 1991;16:110-116.

146. Wojtys EM, Wylie BB, Huston LJ. The effects of muscle fatigue on neuromuscular function and anterior tibial translation in healthy knees. *Am J Sports Med*. 1996;24:615-621.

147. Rozzi SL, Lephart SM, Fu FH. Effects of Muscular Fatigue on Knee Joint Laxity and Neuromuscular Characteristics of Male and Female Athletes. *J Athl Train*. 1999;34:106-114.

148. Skinner HB, Wyatt MP, Stone ML, Hodgdon JA, Barrack RL. Exercise-related knee joint laxity. *Am J Sports Med*. 1986;14:30-34.

149. Stoller DW, Markolf KL, Zager SA, Shoemaker SC. The effects of exercise, ice, and ultrasonography on torsional laxity of the knee. *Clin Orthop Relat Res*. 1983:172-180.

150. Steiner ME, Grana WA, Chillag K, Schelberg-Karnes E. The effect of exercise on anterior-posterior knee laxity. *Am J Sports Med*. 1986;14:24-29.

151. Weisman G, Pope MH, Johnson RJ. Cyclic loading in knee ligament injuries. *Am J Sports Med*. 1980;8:24-30.

152. Coventry E, O'Connor KM, Hart BA, Earl JE, Ebersole KT. The effect of lower extremity fatigue on shock attenuation during single-leg landing. *Clin Biomech (Bristol, Avon)*. 2006;21:1090-1097.

153. Willson JD, Binder-Macleod S, Davis IS. Lower extremity jumping mechanics of female athletes with and without patellofemoral pain before and after exertion. *Am J Sports Med*. 2008;36:1587-1596.

154. Madigan ML, Pidcoe PE. Changes in landing biomechanics during a fatiguing landing activity. *J Electromyogr Kinesiol*. 2003;13:491-498.

155. Orishimo KF, Kremenic IJ. Effect of fatigue on single-leg hop landing biomechanics. *J Appl Biomech*. 2006;22:245-254.

156. Moran KA, Marshall BM. Effect of fatigue on tibial impact accelerations and knee kinematics in drop jumps. *Med Sci Sports Exerc*. 2006;38:1836-1842.

157. Nyland JA, Shapiro R, Stine RL, Horn TS, Ireland ML. Relationship of fatigued run and rapid stop to ground reaction forces, lower extremity kinematics, and muscle activation. *J Orthop Sports Phys Ther*. 1994;20:132-137.

158. Wikstrom EA, Powers ME, Tillman MD. Dynamic Stabilization Time After Isokinetic and Functional Fatigue. *J Athl Train*. 2004;39:247-253.

159. Sanna G, O'Connor KM. Fatigue-related changes in stance leg mechanics during sidestep cutting maneuvers. *Clin Biomech (Bristol, Avon)*. 2008;23:946-954.

160. Van Emmerik RE, Wagenaar RC, Winogrodzka A, Wolters EC. Identification of axial rigidity during locomotion in Parkinson disease. *Arch Phys Med Rehabil*. 1999;80:186-191.

161. Stergiou N, Jensen JL, Bates BT, Scholten SD, Tzetzis G. A dynamical systems investigation of lower extremity coordination during running over obstacles. *Clin Biomech (Bristol, Avon)*. 2001;16:213-221.

162. Stergiou N, Scholten SD, Jensen JL, Blanke D. Intralimb coordination following obstacle clearance during running: the effect of obstacle height. *Gait Posture*. 2001;13:210-220.

163. Heiderscheit BC, Hamill J, Van Emmerik RE. Q-angle influences on the variability of lower extremity coordination during running. *Med Sci Sports Exerc*. 1999;31:1313-1319.

164. Glazier PS, Davids K, Bartlett RM. Dynamical systems theory: a relevant framework for performance-oriented sports biomechanics research. *Sportscience*. 2003;7:sportsci.org/jour/03/psg.htm (4063 words).

165. Sparrow WA, Donovan E, Van Emmerik R, Barry EB. Using relative motion plots to measure changes in intra-limb and inter-limb coordination. *Journal of motor behavior*. 1987;19:115-129.

166. McClay I, Manal K. Coupling Parameters in Runners With Normal and Excessive Pronation. *JOURNAL OF APPLIED BIOMECHANICS*. 1997;13:109-124.

167. Pollard CD, Heiderscheit BC, van Emmerik RE, Hamill J. Gender differences in lower extremity coupling variability during an unanticipated cutting maneuver. *J Appl Biomech*. 2005;21:143-152.

168. Stergiou N, Bates BT, Kurz MJ. Subtalar and knee joint interaction during running at various stride lengths. *J Sports Med Phys Fitness*. 2003;43:319-326.

169. St-Onge N, Duval N, Yahia L, Feldman AG. Interjoint coordination in lower limbs in patients with a rupture of the anterior cruciate ligament of the knee joint. *Knee Surg Sports Traumatol Arthrosc*. 2004;12:203-216.

170. Bernstein N. *Coordination and Regulation of Movement*. New York: Pergamon Press; 1967.

171. Thelen E. The(re) discovery of motor development: learning new things from an old field. *Developmental psychology*. 1989;25:946-949.

172. Malik M, Camm AJ. Heart rate variability. *Clin Cardiol*. 1990;13:570-576.

173. van Wegen EE, van Emmerik RE, Wagenaar RC, Ellis T. Stability boundaries and lateral postural control in parkinson's disease. *Motor Control*. 2001;5:254-269.

174. Winstein CJ, Garfinkel A. Qualitative dynamics of disordered human locomotion: a preliminary investigation. *J Mot Behav*. 1989;21:373-391.

175. Clark JE, Phillips SJ. A longitudinal study of intralimb coordination in the first year of independent walking: a dynamical systems analysis. *Child Dev*. 1993;64:1143-1157.

176. Prapavessis H, McNair PJ, Anderson K, Hohepa M. Decreasing landing forces in children: the effect of instructions. *J Orthop Sports Phys Ther*. 2003;33:204-207.

177. Onate JA, Guskiewicz KM, Sullivan RJ. Augmented feedback reduces jump landing forces. *J Orthop Sports Phys Ther*. 2001;31:511-517.

178. Griffin LY. The Henning Program. In: Griffin LY, ed. *Prevention of Noncontact ACL Injuries*. Rosemont, IL: American Academy of Orthopaedic Surgeons; 2001:93-96.

179. Heidt RS, Jr., Sweeterman LM, Carlonas RL, Traub JA, Tekulve FX. Avoidance of soccer injuries with preseason conditioning. *Am J Sports Med*. 2000;28:659-662.

180. Hewett TE, Lindenfeld TN, Riccobene JV, Noyes FR. The effect of neuromuscular training on the incidence of knee injury in female athletes. A prospective study. *Am J Sports Med*. 1999;27:699-706.

181. Olsen OE, Myklebust G, Engebretsen L, Holme I, Bahr R. Exercises to prevent lower limb injuries in youth sports: cluster randomised controlled trial. *British Medical Journal*. 2005;330:449.

182. Besier TF, Lloyd DG, Ackland TR, Cochrane JL. Anticipatory effects on knee joint loading during running and cutting maneuvers. *Med Sci Sports Exerc*. 2001;33:1176-1181.

183. Welsh RS, Davis, J. M., Burke, J. R., & Williams, H. G. . Carbohydrates and physical/mental performance during intermittent exercise to fatigue. *Medicine and Science in Sports and Exercise*. 2002;34:723-731.

184. Bell AL, Pedersen DR, Brand RA. A comparison of the accuracy of several hip center location prediction methods. *J Biomech*. 1990;23:617-621.

185. Gagnon D, Gagnon M. The influence of dynamic factors on triaxial net muscular moments at the L5/S1 joint during asymmetrical lifting and lowering. *J Biomech*. 1992;25:891-901.

186. Padua DA, Arnold BL, Perrin DH, Gansneder BM, Carcia CR, Granata KP. Fatigue, vertical leg stiffness, and stiffness control strategies in males and females. *J Athl Train*. 2006;41:294-304.

187. Kulas A, Zalewski P, Hortobagyi T, DeVita P. Effects of added trunk load and corresponding trunk position adaptations on lower extremity biomechanics during drop-landings. *J Biomech*. 2008;41:180-185.

188. Shultz SJ, Perrin DH, Adams JM, Arnold BL, Gansneder BM, Granata KP. Assessment of neuromuscular response characteristics at the knee following a functional perturbation. *J Electromyogr Kinesiol*. 2000;10:159-170.

189. Huston LJ, Wojtys EM. Neuromuscular performance characteristics in elite female athletes. *Am J Sports Med*. 1996;24:427-436.

190. Pioletti DP, Rakotomanana LR, Leyvraz PF. Strain rate effect on the mechanical behavior of the anterior cruciate ligament-bone complex. *Med Eng Phys*. 1999;21:95-100.

191. Dufek JS, Bates BT. The evaluation and prediction of impact forces during landings. *Med Sci Sports Exerc*. 1990;22:370-377.

192. Kovacs I, Tihanyi J, Devita P, Racz L, Barrier J, Hortobagyi T. Foot placement modifies kinematics and kinetics during drop jumping. *Med Sci Sports Exerc*. 1999;31:708-716.