

**EFFECT OF CARBOHYDRATE-ELECTROLYTE FEEDINGS ON KNEE
BIOMECHANICS AND POSTURAL STABILITY DURING INTERMITTENT HIGH-
INTENSITY EXERCISE TO FATIGUE**

by

Matthew Everett Darnell

Bachelor of Science, University of Pittsburgh, 2008

Master of Science, University of Pittsburgh, 2010

Submitted to the Graduate Faculty of
The School of Health and Rehabilitation Sciences in partial fulfillment
of the requirements for the degree of
Doctor of Philosophy

University of Pittsburgh

2015

UNIVERSITY OF PITTSBURGH
SCHOOL OF HEALTH AND REAHABILITATION SCIENCES

This dissertation was presented

by

Matthew Everett Darnell

It was defended on

February 12, 2015

and approved by

John P. Abt, PhD, ATC, Assistant Professor

Scott M. Lephart, PhD, Distinguished Professor

Mita Lovalekar, PhD, MPH, MBBS, Assistant Professor

Elizabeth F. Nagle, PhD, Assistant Professor

Kim Beals, PhD, RD, CSSD, Assistant Professor

Dissertation Advisor: Timothy C. Sell, PhD, PT, Associate Professor

Copyright © by Matthew Everett Darnell

2015

EFFECT OF CARBOHYDRATE-ELECTROLYTE FEEDINGS ON KNEE BIOMECHANICS AND POSTURAL STABILITY DURING INTERMITTENT HIGH-INTENSITY EXERCISE TO FATIGUE

Matthew Everett Darnell, PhD

University of Pittsburgh, 2015

Fatiguing exercise alters neuromuscular and biomechanical characteristics that increase athlete's risk for non-contact lower extremity injury. Sports nutrition research has demonstrated the ability to delay fatigue and improve performance through carbohydrate feedings. However, the effect of nutrition on delaying neuromuscular risk factors affected by exercise and fatigue has not been examined. The purpose of this study was to evaluate the effects of a carbohydrate-electrolyte (CHO-E) beverage compared to a placebo (PLA) on landing kinematics, balance, and muscle activation patterns throughout an intermittent high-intensity exercise (IHE) protocol. The IHE protocol was designed to mimic sports competition and has demonstrated the ability to induce changes in landing mechanics during pilot testing. A total of 24 (12 male/12 female) athletes (age: 23.0 ± 4.0 yrs; height: 173.3 ± 7.1 cm; weight: 72.9 ± 11.5 kg; body fat: 18.0 ± 6.4 %) completed three days of testing. The first day of testing consisted of anthropometrics and familiarization with the IHE protocol. The next two testing sessions participants performed four quarters of IHE while consuming either a CHO-E or PLA beverage. Landing kinematics, muscle activation, and dynamic postural stability index (DPSI) scores were assessed immediately before, at break three, and break four of the IHE protocol. The DPSI scores were measured during a single-leg jump landing. Landing kinematics (knee flexion and valgus/varus at initial contact, and peak hip flexion) and muscle activity (surface electromyography pre-activity and re-activity of the hamstrings and quadriceps) were measured during a single-leg stop-jump. Separate two-way repeated measures analysis of variance was performed to determine the interaction between time and treatment for

landing kinematics and DPSI. Change in muscle activation was examined using a related samples Wilcoxon Signed Rank test. The CHO-E beverage had no effect on preventing changes in knee flexion at initial contact ($p=0.472$), peak hip flexion ($p=0.456$), and muscle activation ($p>0.05$). A significant interaction effect occurred for DPSI scores ($p=0.023$) and knee valgus/varus at initial contact ($p=0.007$) however, these changes were small and lack clinical significance. Consuming a 6% CHO-E beverage before, during, and after IHE had no effect on preventing neuromuscular/biomechanical changes occurring as a result of IHE. Future studies should continue to investigate nutritional approaches for injury prevention.

TABLE OF CONTENTS

PREFACE.....	XIII
1.0 INTRODUCTION.....	1
1.1 FATIGUE AND INJURY	2
1.1.1 Functional Joint Stability and the Sensorimotor System.....	3
1.2 FATIGUE	5
1.3 NUTRITION AND FATIGUE	6
1.4 DEFINITION OF THE PROBLEM.....	7
1.5 PURPOSE.....	8
1.6 SPECIFIC AIMS AND HYPOTHESES	8
1.7 STUDY SIGNIFICANCE	9
2.0 REVIEW OF LITERATURE.....	10
2.1 EPIDEMIOLOGY OF LOWER EXTREMITY SPORTS INJURY	10
2.1.1 Fatigue and Lower Extremity Injury	11
2.2 SENSORIMOTOR SYSTEM AND FUNCTIONAL JOINT STABILITY .	13
2.3 BALANCE AND POSTURAL STABILITY.....	17
2.4 FATIGUE	19
2.4.1 Neuromuscular Fatigue Model.....	19
2.4.2 Energy Supply/Depletion Fatigue Model	23
2.5 DELAYING FATIGUE.....	25
2.6 METHODOLOGICAL CONSIDERATIONS.....	29
2.6.1 Measurement of Lower Extremity Kinematics.....	29

2.6.2	Dynamic Postural Stability Index (DPSI)	30
2.6.3	Electromyography (EMG)	31
2.6.4	Intermittent High-Intensity Exercise Protocol	33
2.6.5	Carbohydrate Feedings.....	34
3.0	METHODOLOGY.....	36
3.1	EXPERIMENTAL DESIGN	36
3.2	SUBJECTS	37
3.2.1	Inclusion Criteria.....	37
3.2.2	Exclusion Criteria.....	38
3.3	SAMPLE SIZE ANALYSIS	38
3.4	INSTRUMENTATION	39
3.4.1	Force Plate.....	39
3.4.2	Motion Analysis System	39
3.4.3	Electromyography (EMG)	40
3.4.4	Heart Rate	41
3.4.5	Anthropometrics	41
3.4.6	Body Composition.....	41
3.4.7	Treadmill	42
3.4.8	Vertec.....	42
3.4.9	Biodex	42
3.4.10	Lactate	42
3.4.11	Rating of Perceived Exertion (RPE)	43
3.4.12	Diet Recall	43

3.5	TESTING PROCEDURES	44
3.5.1	Day 1 Testing	44
3.5.1.1	Anthropometrics and Body Composition	45
3.5.1.2	Peak Vertical Jump (PVJ)	45
3.5.1.3	Peak Treadmill Velocity (PTV)	46
3.5.1.4	IHE Review and Familiarization	46
3.5.2	Day 2 and 3 Testing	46
3.5.2.1	24 Hour Diet Recall	47
3.5.2.2	EMG	47
3.5.2.3	Single-leg Stop-jump and Jump Landing	48
3.5.2.4	Intermittent High-Intensity Exercise (IHE)	50
3.6	DATA REDUCTION	53
3.6.1	Kinematics	53
3.6.2	Dynamic Postural Stability (DPSI)	54
3.6.3	Electromyography (EMG)	55
3.7	STATISTICAL ANALYSIS	55
4.0	RESULTS	57
4.1	SUBJECTS	57
4.1.1	Day 1 Testing	58
4.1.2	Diet Recall	58
4.1.3	Intermittent High-Intensity Exercise Protocol and Treadmill Run to Fatigue	59
4.2	LANDING MECHANICS	63

4.2.1	Knee Flexion at Initial Contact	63
4.2.2	Valgus/Varus at Initial Contact.....	64
4.2.3	Peak Hip Flexion.....	65
4.3	DYNAMIC POSTURAL STABILITY	66
4.3.1	DPSI	67
4.3.2	MLSI.....	68
4.3.3	APSI	69
4.3.4	VSI.....	70
4.4	MUSCLE ACTIVITY	71
4.4.1	EMG Pre-Activity.....	72
4.4.2	EMG Re-Activity	73
5.0	DISCUSSION	75
5.1	DESCRIPTIVE VARIABLES.....	77
5.1.1	Diet Recall	77
5.1.2	Intermittent High-Intensity Exercise Protocol	77
5.2	LANDING MECHANICS.....	80
5.2.1	Knee Flexion at Initial Contact	80
5.2.2	Knee Valgus/Varus at Initial Contact.....	81
5.2.3	Peak Hip Flexion.....	82
5.3	DYNAMIC POSTURAL STABILITY	84
5.4	MUSCLE ACTIVITY	85
5.5	LIMITATIONS AND FUTURE RESEARCH	87
5.6	CONCLUSION	89

APPENDIX A	91
BIBLIOGRAPHY	92

LIST OF TABLES

Table 1. Intermittent High-Intensity Exercise Protocol Timeline	50
Table 2. Subject Demographic Data	57
Table 3. Day 1 Testing Results	58
Table 4. Energy and Macronutrient Comparison between CHO-E and PLA Treatments	59
Table 5. Quarter Completion Time, Heart Rate, and Lactate Descriptive Data and Two-way Repeated Measures Analysis of Variance Results	59
Table 6. Rating of Perceived Exertion (RPE) Descriptive Statistics	61
Table 7. Comparison of Change in Rating of Perceived Exertion (RPE) between Quarters for Treatment Groups	62
Table 8. Kinematic Descriptive Statistics and Two-way Repeated Measures Analysis of Variance Results	63
Table 9. Dynamic Postural Stability Index (DPSI) Descriptive Statistics and Two-way Repeated Measures Analysis of Variance Results	67
Table 10. EMG Descriptive Statistics	72
Table 11. Comparison of Change in EMG Pre-activity Across Time Points Between Treatments	73
Table 12. Comparison of Change in EMG Re-activity Across Time Points Between Treatments	74

LIST OF FIGURES

Figure 1. Mean (± 1 SD) Time to Complete each Quarter	60
Figure 2. Mean (± 1 SD) Heart Rate across Quarters	60
Figure 3. Mean (± 1 SD) Lactate across Quarters	61
Figure 4. Median Rating of Perceived Exertion across Quarters.....	62
Figure 5. Mean (± 1 SD) Knee Flexion at Initial Contact	64
Figure 6. Mean (± 1 SD) Knee Valgus/Varus Angle at Initial Contact.....	65
Figure 7. Mean (± 1 SD) Peak Hip Flexion.....	66
Figure 8. Mean (± 1 SD) Dynamic Postural Stability Index (DPSI) Scores.....	68
Figure 9. Mean (± 1 SD) Medial/Lateral Stability Index (MLSI) Scores.....	69
Figure 10. Mean (± 1 SD) Anterior/Posterior Stability Index (APSI) Scores	70
Figure 11. Mean (± 1 SD) Vertical Stability Index (VSI) Scores.....	71

PREFACE

I have no doubt that I would not be where I am today without the support of my colleagues, friends, and family. I would like to acknowledge some of the many people who have assisted me along the way and have played an instrumental role in the completion of my degree and dissertation research.

First, I would like to thank Dr. Scott Lephart for the opportunity to pursue my doctorate and work at the Neuromuscular Research Laboratory. My time studying and working at the lab under your direction has afforded me many unique experiences that I am very grateful for.

To my committee chair Dr. Timothy Sell and co-chair Dr. Kim Beals, thank you both for your hours of council, advice, and support through this process. I deeply appreciate the time, knowledge, and experience you both devoted to my academic studies and dissertation work. I would like to thank the rest of my committee Dr. John Abt, Dr. Mita Lovalekar, and Dr. Elizabeth Nagle for your willingness to serve on my committee as well as your thoroughness and thoughtful insight on my dissertation project.

I would also like to thank all of my study participants who, quite literally, gave their blood and sweat “for the sake of science”. This research would not have been possible without your time and effort.

A special thanks to all of the NMRL graduate student researchers who I have worked with during my years here at Pitt. Your knowledge and friendship has been invaluable to me and I am thankful for the experiences we have shared. Paul Whitehead and Nicholas Heebner, thank you for your dependable help, time, and expertise during data collection and throughout this whole project.

Finally, to all of my friends and family, thank you for your continued love and support through my life. From the loving prayers of my mother to the laughter and caring from all my family, you have all provided me immense support to help me reach my goals. To my wife, Jillian Darnell, I am so very grateful for you and I am blessed to have you in my life. Your unconditional love and support is a constant source of strength. I love and appreciate you.

1.0 INTRODUCTION

Non-contact, musculoskeletal injuries are among the most common types of injuries experienced in all levels of athletics.⁷⁹ Previous research has identified numerous modifiable risk factors for non-contact musculoskeletal injuries, including deficits in postural stability, strength, proprioception, neuromuscular control, biomechanics, and fatigue.^{76, 96, 152} Fatigue is commonly identified as a contributing factor for musculoskeletal injuries, because it creates a physiological environment in which all of the aforementioned neuromuscular risk factors are negatively altered.⁶⁸ Landing kinematics/kinetics and dynamic postural stability during single-leg tasks are frequently measured to identify risk factors for injury. Fatigue has shown to hinder landing mechanics and stability, increasing an athlete's potential for injury.^{17, 159, 169, 174} Additionally, studies have demonstrated that injuries are more common in the later stages of games, practices, and workouts when individuals are more likely to be fatigued.^{57, 99, 111, 164} Delaying the onset and severity of fatigue has implications for both injury prevention and performance enhancement.

A nutrition fueling strategy, in which individuals consume 3-5ml/kg body weight of a six percent carbohydrate-electrolyte solution every 15-20 minutes, has shown to increase performance and time to fatigue in endurance and intermittent high intensity exercise.^{27, 31, 133, 173, 181} However, the effect of nutrition fueling strategies on preventing or delaying some neuromuscular risk factors affected by fatigue has not been examined. The purpose of this research study is to evaluate the effects of a carbohydrate-electrolyte (CHO-E) feeding compared to a placebo (PLA) on postural

stability, muscle activation patterns, and landing mechanics during and immediately following intermittent high-intensity exercise (IHE) to fatigue.

1.1 FATIGUE AND INJURY

The onset and effects of fatigue are of particular interest in the field of sports medicine for both performance enhancement and injury prevention. Fatigue is commonly associated with an increased risk for injuries during various sporting activities.^{57, 99, 111, 132, 152, 159, 164} Gabbett et al.⁵⁷ found more injuries occur in rugby players during the second half of games, when fatigue may be a contributing factor. In junior and men's hockey significantly more injuries occurred in the third period of matches.^{111, 164} The association between fatigue and injury has also been noted in gymnastics, with a greater number of injuries occurring as the duration of the workout increases.¹³² Liederbach and colleagues⁹⁹ also found an increase in the number of ACL injuries in elite modern and ballet dancers later in the day. A good proportion of the injuries sustained in these studies were non-contact strains and sprains. Non-contact injuries are those that occur without extrinsic contact from another individual, object, or the ground and therefore result primarily from intrinsic factors.^{54, 55, 102, 179} Non-contact injuries are of particular interest for injury prevention research because of the potential for modifying many of the intrinsic risk factors for injury.⁶

The lower extremity is a common site for non-contact injuries, with the majority of injuries being ankle sprains, hamstring strains, and internal knee derangement.⁷⁹ An example of a particularly devastating lower extremity injury would be a complete rupture of the anterior cruciate ligament (ACL). Injuries to the ACL are arguably one of the most prevalent and costly non-contact injuries within athletics.¹¹⁰ It has been reported that over 100,000 athletes sustain non-contact ACL

injury in the United States each year, resulting in estimated healthcare costs of two billion dollars.^{66, 135} Anterior cruciate ligament injuries have a lasting impact on the individual, often causing chronic instability, development of osteoarthritis, and quality of life issues.^{51, 81} Additionally, these injuries cause a significant amount of time loss in sporting activities.¹¹⁰ Given the economic and social impact of ACL injuries, many researchers have examined potential mechanisms for non-contact ACL injuries, and they have attempted to elucidate which of these can be modified to prevent such injuries. Non-contact ACL injuries typically occur at or near full knee extension during acceleration or deceleration motions with reduced hamstring contraction and excessive quadriceps co-contraction.^{68, 69, 75, 160} Additionally, excessive knee valgus load during weight-bearing, decelerating activities also increases the forces at the ACL, increasing the likelihood of injury.^{68, 69, 75, 160}

1.1.1 Functional Joint Stability and the Sensorimotor System

Proper alignment and maintenance of joint stability throughout movement reduces the amount of strain placed on the joints and its surrounding structures. This occurs through a combination of both static and dynamic restraints around the joint contributing to its functional joint stability (FJS), which is the process of a joint remaining in or promptly returning to a proper alignment through equalization of forces and moments.^{140, 141} This occurs through the interaction of both the static and dynamic structures surrounding most joints and is mediated by the sensorimotor system.^{140, 141}

The sensorimotor system is responsible for the central integration and processing of the sensory and motor components involved in maintaining FJS. The process of sensory information arising from peripheral mechanoreceptors and traveling through afferent pathways to the central

nervous system (CNS) is known as proprioception.^{140, 141} This proprioceptive information is then integrated with input from other levels of the nervous system to stimulate efferent motor responses, which is known as neuromuscular control.^{140, 141} This feed-back and feed-forward system is essential for performing coordinated movements and in maintaining joint stability. Impaired proprioceptive feedback to the CNS has been demonstrated to hinder the unconscious activation of dynamic restraints that occur in preparation for and in response to joint movements and loading, thereby altering functional joint stability.¹⁴² Additionally, impairments in the feed-forward mechanisms, occurring in anticipation for joint loading and movement, effect muscle stiffness and activation of the musculotendinous structures that surround the joint, which can also lead to impaired joint stability.¹⁴²

One of the possible physiological mechanisms for fatigue-related injuries involves proprioceptive and neuromuscular control deficits brought on by fatigue, which leads to functional instability, resulting in acute trauma or chronic, repetitive-motion injury.⁹⁷ Proprioceptive and neuromuscular control alterations have been demonstrated through both localized and functional fatigue mechanisms. Localized fatigue protocols focus primarily on fatiguing one muscle or group of muscles through isolated lifting/contracting exercises. Conversely, functional fatigue protocols try to induce fatigue through movements or tasks similar to those seen in athletics or sports. This mechanism has been demonstrated in studies that have shown the effect of fatigue on reaction time, coordination, and motor control.^{95, 108, 184}

The relationship between fatigue and injury is most often drawn from the association that fatigue has on known risk factors for non-contact injuries. Of these risk factors, lower limb muscle activation patterns and biomechanics during jumping and landing tasks, as well as measures of postural stability, have been of particular interest for predicting non-contact ACL injuries.^{69, 96}

Specifically, prospective research has shown certain kinematic variables, such as increased knee valgus and decreased knee flexion angles at initial contact, to increase the risk of ACL injury.^{75, 76} Even more, greater activation of the quadriceps muscles compared to hamstrings prior to and following landing also increases the risk for ACL injury.⁷⁵ Multiple studies have demonstrated that fatigue induces similar changes in kinematics and muscle activation patterns during landing as those shown previously in research to increase the risk for non-contact ACL injuries. These altered biomechanics brought on by fatigue have been demonstrated during landing, running, cutting, and single-leg hop maneuvers.^{5, 24, 45, 126}

1.2 FATIGUE

Fatigue has been widely studied with varying definitions for this phenomenon in different research fields. In the field of sports medicine, fatigue is most commonly described as the inability to maintain a required force production or power output for a given intensity. Typically, this leads to acute decrements in exercise performance, mechanical function, and/or movement patterns. The exact mechanism for the onset of fatigue is complex but can be broadly categorized as either peripheral or central fatigue. Peripheral fatigue results from substrate depletion and/or metabolite accumulation within the muscle leading to impaired translation of motor neural signals to an appropriate force response.^{38, 52, 122} In contrast, central fatigue results from the diminished activation drive from the CNS to the muscle, and may be a function of altered perceptions of effort as well as neurotransmitter and hormonal processes.^{52, 122} During athletics and exercise, a combination of both peripheral and central fatigue is likely to occur synergistically, affecting neuromuscular control and exercise performance.

1.3 NUTRITION AND FATIGUE

The studies on nutrition with regard to fatigue have primarily focused on fueling strategies that increase performance by delaying the onset of fatigue. Carbohydrates (CHO) have been shown to be the most effective macronutrient to increase performance during prolonged and intermittent high intensity exercise.^{28, 35, 90, 173}

The mechanisms in which nutrition, specifically CHO, impacts fatigue are hypothesized to occur in both the peripheral and in the central nervous systems. Depending on exercise intensity and duration, CHO feedings help to maintain blood glucose levels and decrease the reliance on glycogen stores in the muscle and liver. The availability of exogenous carbohydrates (ingested from foods or beverages) is crucial for sparing glycogen stores within the muscle.⁸⁴ Peripherally, depleted muscle glycogen stores lead to fatigue and decreased exercise performance due to the unavailability of immediate energy sources for contracting muscles.⁹⁰ Supplementation with CHO during exercise can help to increase time to fatigue and performance by offsetting the reliance on muscle and liver glycogen stores, preventing hypoglycemia, and allowing for high rates of CHO oxidation needed to sustain exercise intensity.^{34, 35, 84, 90}

The ergogenic effects of CHO during exercise are not limited to preventing substrate depletion. It appears the CHO ingestion, or even the mere presence of CHO in the mouth, also has effects on motor control and exercise performance.^{23, 62, 125, 129} Chambers et al.²³ demonstrated that a CHO mouth rinse during exercise compared to a placebo led to heightened activation of the motor control areas of the brain as well as improved performance during cycling time trials. Further, Gant et al.⁶² were the first to demonstrate the ability of CHO in the mouth to immediately increase the excitability of corticomotor pathway and increase maximum voluntary force production. They suggested that “afference from oral receptors is integrated with descending motor

output,” representing a “novel form of sensorimotor integration”.⁶² This sensorimotor integration from the presence of CHO in the mouth helped to increase corticomotor output to both fatigued and non-fatigued muscles. Despite the possible role that CHO may have on the sensorimotor system, the majority of nutrition research has focused on the ergogenic effects of CHO for improved performance and fatigue resistance.

The role of CHO feedings during exercise and athletic competition may also be associated with injury prevention. A common hypothesis is that if exogenous CHO is available in adequate amounts during exercise, the effects of fatigue would be delayed or lessened and therefore put the athlete at decreased risk for injury.^{90, 155}

1.4 DEFINITION OF THE PROBLEM

More injuries tend to occur in the later stages of games, practices, and during fatigued conditions. Fatigue has also been shown to alter neuromuscular and biomechanical characteristics in a manner that increases an athlete’s risk of non-contact lower extremity injury. Interventions are needed to delay or prevent the effects of fatigue on risk factors for injury. Sports nutrition research has demonstrated the ability to reduce the effects of fatigue and improve athletic performance through CHO feedings. Limited research has examined the capability of nutrition (specifically CHO intake) to attenuate the biomechanical and neuromuscular control alterations that occur as a result of fatiguing exercise.

1.5 PURPOSE

The purpose of this dissertation research is to evaluate the effects of a carbohydrate-electrolyte (CHO-E) feeding compared to a placebo (PLA) on postural stability, muscle activation patterns, and landing mechanics during and immediately following intermittent high-intensity exercise (IHE) to fatigue. Postural stability will be measured using the Dynamic Postural Stability Index (DPSI) score during a single-leg jump landing. Landing mechanics will be measured using a single-leg stop-jump task and muscles activation patterns will be measured during this task using surface electromyography. The CHO feeding will consist of a six percent CHO-E beverage.

1.6 SPECIFIC AIMS AND HYPOTHESES

Specific Aim 1: To determine the effect of consuming a CHO-E beverage during and immediately following IHE on knee flexion at initial contact, varus/valgus angle at initial contact, and peak hip flexion angle during a single-leg stop-jump landing.

Hypothesis 1: CHO-E feedings will maintain knee flexion at initial contact, varus/valgus angle at initial contact, and peak hip flexion angle during a single-leg stop-jump landing closer to baseline as compared to PLA during and immediately following IHE.

Specific Aim 2: To determine the effect of consuming a CHO-E feeding during and immediately following IHE on single-leg jump-landing DPSI scores.

Hypothesis 2: CHO-E feedings will maintain DPSI scores closer to baseline as compared to PLA during and immediately following IHE.

Specific Aim 3: To determine the effect of consuming CHO-E feeding during and immediately following IHE on the pre-activity and re-activity muscle activation patterns of the vastus medialis, vastus lateralis, medial hamstrings, and lateral hamstrings during a single-leg stop-jump landing.

Hypothesis 3: CHO-E feedings will maintain pre-activity and re-activity muscle activation patterns of the vastus medialis, vastus lateralis, medial hamstrings, and lateral hamstrings during a single-leg stop-jump landing closer to baseline as compared to PLA during and immediately following IHE.

1.7 STUDY SIGNIFICANCE

Previous research has shown that CHO feedings can enhance performance and delay time to fatigue during exercise. Research has also demonstrated that exercise fatigue can negatively affect neuromuscular risk factors for non-contact injury. However, the effects of CHO-E feedings on neuromuscular characteristics related to fatigue and risk of non-contact injury have not been studied. The results of this study will help determine if CHO-E feedings during exercise can reduce some of the changes in biomechanics that result from fatigue. This study will be the first to demonstrate a relationship between nutrition fueling and its impact on risk factors of non-contact injury. The results of this study will help to demonstrate the importance of sports nutrition feeding strategies for injury prevention.

2.0 REVIEW OF LITERATURE

2.1 EPIDEMIOLOGY OF LOWER EXTREMITY SPORTS INJURY

Participation in sports, recreational activities, and exercise has long been a part of American culture and is continuing to grow in popularity. Unfortunately, with participation in these types of activities comes an inherent risk of injury. According to a report by the National Institute of Health, approximately three million injuries occur annually in the United States from participation in organized sports, with about 770,000 of these injuries resulting in physician visits and 90,000 requiring hospitalization.¹²⁰ Even more, the Center for Disease Control and Prevention reported that Americans made about 1.5 million emergency department visits in 1999 for injuries sustained while playing basketball, baseball, softball, soccer, or football alone.³⁰ The amount spent for the acute management of these sports injuries has been estimated at 1.3 billion dollars.⁷³

Of all sports injuries the most common sites for injury are the lower extremities.^{29, 65, 88} According to results from US national survey and emergency department data, the lower extremities were the most frequently injured body parts and accounted for 20.2 - 38.9% of injuries.^{29, 65} Junge et al.⁸⁸ tracked injuries during team sports tournaments in the 2004 Olympic Games and reported 50% of all injuries occurring to the lower extremities. Additionally, the authors reported that the ankle (13%) and knee (13%) were tied for the second most frequently injured body part, with sprains, strains and ruptures being one of the most common diagnoses of injury.⁸⁸ Furthermore, in a study examining 16 years of injury surveillance data collected by the National Collegiate Athletic Association for 15 different sports, over 50% of all injuries occurred to the lower extremities.⁷⁹ Of these injuries, the majority were to the knee and ankle.⁷⁹

Given the frequency of lower extremity injury, research has investigated possible risk factors and mechanisms of lower extremity injuries. With clearly established risk factors for injuries known, prevention and intervention strategies can then be implemented to help reduce the incidence of injury. Risk factors for lower extremity injury can broadly be divided into extrinsic (outside the body) and intrinsic (inside the body) risks.¹¹⁵ Extrinsic risks include factors like the environment, weather, playing surface, level of competition, and equipment. Intrinsic factors include previous injury, age, aerobic fitness, musculoskeletal/joint characteristics (flexibility, strength, range of motion, joint laxity), reaction time, and postural stability.¹¹⁵ Lower extremity risk factors can further be classified as either modifiable or non-modifiable risk factors. While non-modifiable risk factors like age, gender, or anatomical alignment may be of interest to some investigators, it is the modifiable risk factors that have the potential for interventions for injury prevention. Fatigue is a condition that negatively alters multiple intrinsic modifiable risk factors for lower extremity injury and has been implicated as a predisposing factor for injury.

2.1.1 Fatigue and Lower Extremity Injury

Epidemiological studies investigating injuries in sports and athletics have identified fatigue during practices and competition as a contributing factor for unintentional musculoskeletal injuries.^{49, 57, 112, 187, 188} Gabbett et al.⁵⁷ studied the incidence of injuries in amateur rugby players over the course of three consecutive seasons. During this time, the authors reported significantly more injuries occurring in the second half of the game compared to the first (70.8% vs. 29.2%). Of these injuries, muscular and joint injuries were the most common injury type accounting for 28.5% and 17.2% of injuries respectively.⁵⁷ The higher incidence of injury in the second half of matches suggests fatigue is a contributing factor for injuries. The findings of Gabbett et al.⁵⁷ are also supported by

additional research indicating increased injury rates in the later stages of competition. In a study examining the incidence of injuries in ice hockey players over a 20 year period, authors discovered the majority of injuries (42%) occurred in the last period of the match.¹¹² Interestingly, when the authors stratified each period into thirds they found the majority (40%) of injuries occurred in the final third of each period.¹¹² The authors hypothesized the increased rate of injuries late in each period and towards the end of matches (when intensity increases) were due to player fatigue.¹¹² Increased incidence of injury in the later stages of games has also been reported during soccer competitions as well.^{49, 138, 187, 188} Woods et al.¹⁸⁷ specifically analyzed the incidence of ankle sprains during soccer matches over the course of two competitive seasons in English professional soccer players. In their study, ankle sprains accounted for 11% of all injuries with over two-thirds of the ankle sprains occurring during competition.¹⁸⁷ The majority of ankle sprains in games occurred during the last 15 minutes of the first and second halves when players are more susceptible to fatigue.¹⁸⁷ The association between fatigue and injury has also been reported during training and practices. In gymnasts, Petrone and colleagues¹³² found a positive relationship between the length of workouts and the total number of injuries. Additionally, in elite ballet and modern dancers, more ACL injuries were experienced later in the day after several hours of activity.⁹⁹

Lower extremity injuries are common during sports competition and training. The majority of injuries often occur later in games and as the duration of training increases, when fatigue is likely a contributing factor.^{99, 111, 112} Injuries to the ankle and knee were the most commonly reported non-contact lower extremity injuries occurring in the late stages of competitions and workouts.^{57, 79, 88, 132, 187} While ankle injuries may be the most prevalent lower extremity injury, knee injuries, like a complete rupture of the ACL are arguably one of the more devastating types

of sports injuries. Anterior cruciate ligament injuries are arguably one of the most prevalent and costly non-contact injury within athletics.¹¹⁰ It has been reported that over 100,000 athletes a year sustain non-contact ACL injury in the United States with an estimated healthcare cost of about two billion dollars.^{66, 135} Anterior cruciate ligament injuries have a lasting impact causing chronic instability, development of osteoarthritis, and quality of life issues.^{51, 81} Additionally, these injuries cause a significant amount of time loss in sporting activities.¹¹⁰ The popularity and negative impact of these injuries have sparked much research to identify possible risk factors. Multiple factors may contribute to the occurrence of these lower extremity injuries, however, for this dissertation, the effect of fatigue on sensorimotor system and its impact on functional joint stability (FJS) of the lower extremity will be examined.

2.2 SENSORIMOTOR SYSTEM AND FUNCTIONAL JOINT STABILITY

In the process of human movement, the sensorimotor system is responsible for the central integration and processing of the sensory and motor components involved in the maintenance of joint stability throughout movement.^{140, 141} Maintenance of FJS throughout movement reduces the amount of strain placed on the joint and surrounding structures.¹⁴⁰⁻¹⁴² This is accomplished through an interaction between the static (ligaments, joint capsule, and bony geometry) and dynamic (surrounding musculature) structures surrounding a joint and is mediated by the sensorimotor system.¹⁴⁰

The sensorimotor system incorporates all of the afferent, efferent, and central integration and processing components involved in maintaining FJS, which encompasses both feed-forward and feedback neuromotor control over the skeletal muscles crossing the joint.^{63, 86, 140} In this

instance, we will be referring to the knee joint. Neuromuscular control over the knee occurs from the unconscious activation of dynamic restraints (skeletal muscle crossing the knee), occurring in preparation for and in response to joint motion and loading for the purpose of maintaining and restoring FJS.^{140, 141} Neuromuscular control occurs from the efferent pathway of the sensorimotor system and is mediated by information transmitted through the afferent pathways. Afferent pathways of the sensorimotor system include visual, vestibular, and somatosensory feedback, which convey input to the three levels of motor control.^{98, 140} The somatosensory system encompasses peripheral mechanoreceptors in cutaneous, articular, and muscle tissues.^{98, 140} The static structures contain mechanoreceptors such as Ruffini receptors, Pacinian corpuscles, Golgi tendon organ like receptors, and free nerve endings that provide afferent information regarding tension and deformation of these structures.^{78, 140, 190} The dynamic structures such as Golgi tendon organs and muscle spindles provide afferent feedback pertaining to muscle tension and change or rate of change in muscle length.^{83, 131, 140} The mechanical stimulus from these peripheral receptors is converted to neural signals and transmitted to the central nervous system to convey information about joint position, kinesthesia, and sense of resistance/force.¹⁴⁰ Collectively, this afferent information from the peripheral receptors is known as proprioception.^{140, 141} This proprioceptive feedback mediates the efferent response and neuromuscular control of the knee. Conversely the feed-forward mechanism describes the anticipatory activation of dynamic restraints and is shaped by previous experiences. The feed-forward mechanism only takes place intermittently until the feedback mechanism is initiated.^{63, 86, 140, 141} This feedback/feed-forward response can be explained using the final common input theory. The information arising from the peripheral receptors in the articular, cutaneous, and muscle tissue converge on the gamma motor neurons, increasing the activation of the gamma motor neuron, which enhances the sensitivity of the muscle spindle.^{85, 140}

The muscle spindle conveys information about the rate and magnitude of length changes within the muscle and plays a significant role in regulating muscle tone or stiffness.^{131, 140, 141} Increased sensitivity of the muscle spindle afferents increases the excitability of the motor-neuron pool, which helps to increase the reflexive neural activation of the muscle.^{131, 140, 141} The reflexive neural activation of the muscle is a component of extrinsic muscle stiffness. Increased muscle stiffness around a joint leads to increased joint stiffness and stability.¹⁴¹ Stiffer muscles are better able to resist joint displacements, transmit loads to the muscle spindles, and enhance the feedback and feed-forward mechanisms for dynamic joint stability.^{70, 100, 106, 141}

Fatigue-associated changes to neuromuscular activity are a major cause of kinematic changes and decreased dynamic joint stability during movement.^{98, 142} Several mechanisms of fatigue have been reported as potential causes for decreases in feed-forward and feedback mechanisms. Decreases in proprioception and neuromuscular control results in altered sensation of joint movement, leading to motor control impairments and functional instability that can result in acute trauma or repetitive motion injury.⁹⁷ Impairments in motor control and functional instability can be measured by muscle activation patterns, postural stability, and kinematic changes that occur during dynamic tasks.¹⁴² Fatigue-induced neuromuscular alterations have been investigated in the lower limb and knee during jumping and landing tasks to determine its effect on known risk factors for lower extremity non-contact injuries. Non-contact knee injuries (specifically ACL injuries) typically occur at or near full knee extension during acceleration or deceleration motions with reduced hamstring contraction and excessive quadriceps co-contraction.^{68, 69, 75, 160} Additionally, excessive knee valgus load during weight-bearing, decelerating activities also increases the forces at the ACL, increasing the likelihood of injury.^{68,}

^{69, 75, 160} Multiple studies have demonstrated that fatigue induces similar alterations in neuromuscular control during jumping and landing tasks.

Santamaria and Webster¹⁵³ conducted a systematic review on the effects of fatigue on single-leg landing biomechanics. Eight studies were included in their analysis, with all of them measuring landing kinematics from a drop or a jumping task before and after a fatiguing exercise. Despite the variations in the fatigue protocols (general vs. local fatigue), the authors concluded that overall, subjects landed with less knee and hip flexion at initial contact post-fatigue than pre-fatigue.¹⁵³ Unfortunately, only three of the eight studies reviewed measured knee kinematics in the frontal plane. Two of the studies reported increased peak knee valgus angles following fatigue, however, due to the limited results no overall effect was shown for fatigue on peak knee valgus angles.¹⁵³ Santamaria and Webster's review demonstrated that fatigue does appear to negatively alter certain biomechanics of the lower limb during single-leg landings that would increase an athlete's risk for injury. The authors suggest that future research should utilize a general fatigue protocol (which better represents the demands of athletic environments) as well as to monitor the progression of fatigue on landing kinematics throughout the fatigue protocol.¹⁵³

Cortes and colleagues³² recently completed a study examining kinematic changes following a functional fatiguing protocol. The researchers measured landing kinematics from a stop-jump and side-cut jump maneuver at three different time points (pre-fatigue, 50% fatigue, and post fatigue).³² The fatiguing protocol incorporated a series of vertical jumps, box step-ups, squats, and a pro agility run. They observed biomechanical changes in both frontal and sagittal kinematics.³² Knee flexion angle at initial contact progressively decreased across all three time points. Additionally, both initial and peak hip flexion decreased from pre-fatigue to post fatigue.

Knee abduction angle at initial contact also decreased from pre-fatigue to post-fatigue, but interestingly, the greatest decrease was seen between pre-fatigue and 50% fatigue.³²

The effects of fatigue on landing can also be seen in the activity patterns of the muscles surrounding the knee joint. James et al.⁸² reported increased EMG activity of the vastus lateralis (VL) and vastus medialis (VM) during the landing phase of a drop jump following fatigue compared to pre-fatigued landings. Additionally, Orishimo and Kremenic¹²⁸ investigated the effects of fatigue on landing biomechanics and EMG during a single-leg hop. The researchers reported significantly increased activation of the VM 100 milliseconds prior to impact in the fatigued state versus non-fatigued.¹²⁸ Both of these researchers identified that as individuals become more fatigued, activation of the quadriceps increased without any significant increase in hamstring activation.^{82, 128} The increase in quadriceps activation without an increase in hamstring activation before landing may explain why, following fatigue, most individuals land with less knee flexion.^{82, 128} These muscle activity patterns seen post fatigue increase an athlete's risk for knee injury.⁷⁵ Excessive quadriceps activation with decreased hamstring co-contraction immediately before or during landing has been identified as a risk factor for ACL injury.⁷⁵

2.3 BALANCE AND POSTURAL STABILITY

Another effect of fatigue on neuromuscular control and functional joint stability has been demonstrated in altered static and dynamic balance during postural maintenance activities. Johnston et al.⁸⁷ reported significantly worse single-leg static balance scores following isokinetic fatigue of the lower extremity. Additionally, static measures of single-leg balance have also shown to increase following repeated anaerobic tests.¹⁹¹ Yaggie et al.^{191, 192} have also discovered

impairments in postural sway following a localized ankle fatigue protocol. Brazen and colleagues¹⁷ measured dynamic balance from a single-leg drop landing following a functional fatigue protocol. The authors reported increased time to stabilization (TTS) in the anterior-posterior and vertical direction following fatiguing exercise.¹⁷ Medial-lateral TTS showed a significant interaction following fatigue however it was not statistically significant.¹⁷ The authors hypothesize that fatigue did not alter TTS in the medial-lateral directions because of the nature of drop landing task most likely was not challenging enough.¹⁷ Wikstrom et al.¹⁷⁴ and Shaw et al.¹⁵⁹ both assessed TTS following a jump landing task before and after fatiguing exercise. Wikstrom reported a significant increase in vertical TTS and Shaw reported no significant difference in anterior-posterior or medial-lateral direction and did not report any values in the vertical direction.^{159, 174} While both of these researchers implemented functional fatigue protocols and a more athletic maneuver to assess stability, Wikstrom hypothesized that TTS measures may not be sensitive enough to detect stability changes caused by fatigue, and has since developed the dynamic postural stability index (DPSI) score as a more sensitive measure of stability.^{174, 177} However, to date limited research has evaluated the effect of fatigue on DPSI scores.

Fatigue is a unique physiological process that impacts many systems of the body. Specifically, fatigue has been demonstrated to affect various aspects of the sensorimotor system and neuromuscular control. These alterations in neuromuscular control cause altered movement patterns, muscle activity, and measures of stability. Alterations in neuromuscular control have been elicited during various fatigue conditions, including both localized and general fatigue states. For a better understanding in the mechanisms involved in fatigue related neuromuscular control changes, a more detailed review of fatigue will be performed.

2.4 FATIGUE

Fatigue is commonly defined as the inability to maintain a required force production or power output for a given intensity.^{1, 16, 21, 38, 107, 121, 122} The onset of fatigue is of particular interest in the field of sports medicine for both performance enhancement and injury prevention. This is because fatigue typically leads to decrements in exercise performance, mechanical functions, and movement patterns. While the definition or consequences of fatigue is commonly agreed upon throughout research, the mechanism or mechanisms in which fatigue occurs has been widely researched. Various models to explain the phenomenon of fatigue have been proposed and include: cardiovascular/anaerobic, energy supply/energy depletion, neuromuscular fatigue, muscle trauma, biomechanical, thermoregulatory, psychological/motivational, central governor, and the complex systems model of fatigue.^{1, 16, 121} While most of these theories describe the process of fatigue in a linear model without much regard to the other systems, the complex systems model of fatigue proposes that the process of fatigue is multifactorial, dependent on multiple systems, and includes both peripheral and central systems.^{1, 121} This dissertation will primarily focus on the neuromuscular and energy supply/energy depletion models of fatigue. The mechanism for these two models of fatigue will be reviewed as well as the interaction that they have on each other.

2.4.1 Neuromuscular Fatigue Model

The ability of the cardiovascular system to supply blood, nutrients, and oxygen to exercising muscles, as well as the muscles' capacity to resynthesize adenosine triphosphate (ATP), are all important mechanisms in exercise performance and fatigue development. However, the excitation, recruitment, and contraction of exercising muscles are also important factors in exercise

performance and fatigue development. The reduction of power production or force development of a muscle or group of muscles, despite an increase in effort, is known as neuromuscular fatigue.⁶⁰ The primary mechanism for neuromuscular fatigue results from a decreased neural drive occurring during exercise. The decrease in neural drive is theorized to occur in two places: in the CNS and in the peripheral nervous system.^{16, 38} The cause of central fatigue refers to the decrease in muscle activation by the CNS. While the exact mechanisms for central fatigue are still inconclusive, decreases in blood glucose, intracortical inhibition due to neurotransmitter imbalances, or afferent feedback are theorized to be major contributors.⁴⁰

Accumulation or depletion of certain neurotransmitters result in a decrease in neural drive and is often linked as a cause of central fatigue.^{16, 107} Serotonin is the most researched neurotransmitter in the area of neuromuscular fatigue. Known for its effect on lethargy and loss of motivation, serotonin can limit central command and motor unit recruitment.^{16, 39, 107} During prolonged exercise, serotonin levels increase in the brain, limiting central neural drive and motor unit recruitment.³⁹ This theory of increased serotonergic activity during prolonged exercise leading to reduced motor unit recruitment was first suggested by Newsholme et al.¹¹⁷. Since then, research has provided fair evidence to support increased serotonin activity during prolonged exercise and its association with fatigue.^{39, 107} Wilson et al.¹⁸⁰ and Davis et al.³⁷ performed similar studies in which subjects consumed medication that increased serotonin activity in the brain and then performed a running or cycling protocol to fatigue. In both studies, participants reported higher ratings of perceived exertion and fatigued sooner on the medication than when taking a placebo.^{37, 180} No significant difference was found in metabolic or cardiovascular function between the two treatments, leading the researchers to conclude the results were primarily due to increased serotonin levels.^{37, 180} While these studies increased serotonin levels through medication, serotonin

levels have been shown to naturally increase during prolonged exercise.^{16, 107} Since serotonin cannot cross the blood-brain barrier, it is synthesized in the brain from its amino acid precursor tryptophan. An increase in the amount of free tryptophan in the brain can increase serotonin synthesis.^{16, 39} Free tryptophan and branch chain amino acids (BCAA) share the same receptor for transport across the blood-brain barrier. Thus, when levels of free tryptophan rise or BCAA levels fall, more tryptophan crosses into the blood-brain barrier to be synthesized into serotonin.^{16, 39} An increase in the free tryptophan to BCAA ratio has been demonstrated during prolonged exercise.³⁹ This is a result of increased BCAA oxidation for energy in contracting muscles, as well as an increase in plasma free fatty acids, which displaces albumin-bound tryptophan, creating more free tryptophan in the plasma.³⁹ Though there is some evidence that central fatigue may occur through altered neurotransmitter synthesis, it is unlikely that this is the sole cause responsible for central fatigue during exercise.

Another mechanism causing a decrease in central neural drive may be from afferent information arising from exercising muscles and the cardiovascular system. Noakes describes this afferent feedback in his review of the central governor theory of fatigue.¹²¹ Decreased neural drive occurs in response to afferent feedback from the periphery as a safety mechanism to prevent over exertion.¹²¹ Group III and IV muscle afferents transmit neural signals to the supraspinal areas about muscle ischemia, hypoxemia, and metabolites which limit the voluntary command of the CNS.^{58, 59, 61} This has been demonstrated in studies using ischemia to prevent recovery from muscle fatigue. The ischemic conditions allow the group III and IV afferents to continue to fire even though nervous command has recovered.⁶¹ Gandevia et al.⁶¹ demonstrated this by using transcranial magnetic stimulation to measure voluntary activation of the CNS. They determined that supraspinal fatigue persisted as long as the muscle stayed ischemic.⁶¹ Furthermore, Bigland-

Ritchie et al.¹¹ have shown that fatigue-induced metabolic changes in the muscle prompt peripheral reflexes (from group III and IV metaboreceptors) that regulate motor-neuron discharge rate. Stimulation of these fatigue-sensitive metaboreceptors has been demonstrated to limit voluntary command by acting upstream of the motor cortex^{134, 167} as well as inhibiting activity of the alpha motor-neurons.^{91, 189} As described previously in the final common input theory, afferent information from the muscle (metaboreceptors and mechanoreceptors) as well as descending supraspinal commands, converge on the gamma motor-neurons, which ultimately influence muscle spindle sensitivity and activation of the alpha motor-neurons, leading to a decrease in muscle force production.

Neuromuscular fatigue also manifests through the peripheral nervous system, where the neural activation of muscles is reduced because of the limited response of the muscle to an electrical stimulus. This is known as the neuromuscular propagation failure theory, and it is thought to occur at the level of the sarcolemma or alpha motor-neuron.^{1, 16} Whereas CNS fatigue is a diminished neural drive from the supraspinal areas, in this theory, muscle force production is reduced through alterations in neuromuscular transmission from a nerve action potential to a muscle action potential.¹⁶ A reduction in the conduction velocity of the action potential from the nerve to the muscle has been attributed to decreases in muscle pH, increases in extracellular potassium (K⁺) and intracellular lactate, and increased intracellular sodium (Na⁺), resulting in reduced EMG activity during prolonged exercise.^{1, 16, 56, 67, 118, 121} During prolonged exercise, muscle membrane excitability may be decreased from a shift of the ionic gradient across the membrane, causing a failure in neuromuscular propagation.^{56, 67, 118}

In summary, the neuromuscular fatigue model proposes that fatigue occurs as a result of diminished neural drive and/or a decreased response of the muscle to an electrical stimulus. This

may happen as a result of neurotransmitter imbalances, afferent feedback from exercising muscles, or ionic/metabolite changes that result in reduced muscle membrane excitability. However, fatigue is also hypothesized to occur within the muscle due to insufficient energy supply or depletion.^{1, 38,}

121

2.4.2 Energy Supply/Depletion Fatigue Model

The energy supply model states that exercise fatigue is a result of insufficient ATP production to working muscles through metabolic pathways like glycolysis, lipolysis, and phosphocreatine.^{40, 67,}

¹²¹ This theory proposes that the ability to generate ATP in the primary metabolic pathway of an activity is a contributing factor for fatigue. Individuals with a greater capacity for ATP production from aerobic lipolysis, for instance, would perform better during a long distance run than those with less of a capacity for aerobic lipolysis. The same would hold true that a sprinter with a greater ability to generate ATP from anaerobic glycolysis and intramuscular phosphorous stores would also be more fatigue resistant. Support for the energy supply model stems from the fact that regular training can up-regulate the enzymes creatine kinase, succinate dehydrogenase, and malate dehydrogenase in energy systems involved in re-phosphorylation.^{18, 72, 183} While intuitively the energy supply hypothesis may make sense, it has not been systematically evaluated and therefore remains uncertain. Enzymatic adaptations to training does not by itself explain changes in performance or fatigue resistance.¹²¹ The main premise of this model is that fatigue occurs as a result of depleted muscle ATP. However, during exercise (even to fatigue), ATP concentrations rarely fall below 40-70% of pre-exercising levels.^{52, 67, 77, 162} Since complete depletion of ATP causes rigor mortis to occur in the muscle, ATP concentrations are likely protected from dropping too low.¹²¹ Therefore, it is doubtful that ATP concentrations play a direct role in exercise fatigue.

It is likely that other factors of exercise fatigue cause a reduction in the utilization of ATP before it has a chance to become depleted.⁵² Specifically, central and peripheral factors of neuromuscular fatigue are thought to occur before complete depletion of ATP. Abnormal levels of ATP may influence afferent feedback to the CNS, decreasing central drive to exercising muscles.^{39, 40} Additionally, buildup of metabolic byproducts from ATP synthesis through anaerobic glycolysis can interfere with cross-bridge cycling and energy production, resulting in peripheral inhibition of muscle contraction.^{52, 121}

A more direct cause of exercise fatigue may be explained through the energy depletion model of fatigue. This model proposes that fatigue during prolonged exercise is a result of depletion of endogenous carbohydrate stores resulting in hypoglycemia.³³ The primary support for this model arises from the association between prolonged fatiguing exercise and muscle and liver glycogen depletion.^{15, 20, 26, 36, 52, 170} Subjects participating in prolonged exercise to fatigue develop very low glycogen stores. However, increased availability of CHO through ingestion during exercise or increased glycogen stores from CHO loading (muscle glycogen super-compensation) allows individuals to exercise longer before fatigue.^{26, 36, 170} The most obvious mechanism for the depletion of CHO and glycogen stores to cause muscle fatigue is that CHO and muscle glycogen are the primary fuel source for sustained high intensity exercise.^{15, 71} This is because CHO oxidation allows for high rates of ATP production.^{44, 163} As CHO fuel sources become depleted, exercise intensity can no longer be maintained due to the lack of CHO substrates available for energy production.^{15, 71} In addition to CHO availability, the rate at which CHO can be oxidized for energy may also be an important factor.^{44, 121}

Another mechanism for muscle fatigue from the energy depletion model is the effect of hypoglycemia on the CNS and neural drive. This was demonstrated by Nybo et al.¹²⁵ when

endurance trained subjects had significantly less force production during a maximum voluntary contraction following prolonged exercise in which their blood glucose levels significantly decreased. Interestingly, force production improved once euglycaemia was achieved through CHO ingestion.¹²⁵ The authors concluded that decreased neural drive from the CNS was responsible for the reduced force development.¹²⁵ The CHO depletion model of fatigue is hypothesized to be responsible for the neurotransmitter imbalances that cause central neuromuscular fatigue. As described earlier in the neuromuscular fatigue model, increased production of serotonin during exercise may decrease neural drive. When CHO stores are depleted during exercise, there is an increased reliance on oxidation of fats and BCAA for energy. This, in turn, increases levels of free-tryptophan in the blood, making free-tryptophan more available for synthesis into serotonin.^{16, 39} Additionally, others have hypothesized that a drop in blood glucose or muscle glycogen levels may provide afferent feedback to the CNS, limiting neural drive.^{16, 39, 121} It appears that the availability of CHO during exercise likely has a role in both the neuromuscular fatigue and energy supply/depletion models of fatigue.

2.5 DELAYING FATIGUE

Studying and understanding the mechanisms of fatigue is beneficial for the development of strategies for delaying fatigue. The ability to prevent or delay the onset of fatigue is of particular interest to coaches, athletes, and researchers. Being more fatigue-resistant could have implications for injury prevention and improved performance. Delaying the effects of neuromuscular fatigue on biomechanics through training programs targeting central control has been suggested by some researchers.^{105, 168} Others look to offset the effects of fatigue through endurance training programs

to make the exercising muscles more fatigue-resistant. Moore and colleagues¹¹³ recently demonstrated the effectiveness of a preseason training program for fatigue resistance of the posterior shoulder muscles in baseball players. While these training programs target more localized systems involved with fatigue, another approach for delaying fatigue has focused on training the cardiovascular system.¹ Improvements in the ability of the cardiovascular system to supply blood, nutrients, and oxygen to exercising muscles, as well as the muscles' capacity to resynthesize ATP, are important factors for exercise performance and fatigue resistance.¹²¹ Interestingly, another method for delaying fatigue is proper nutrition and fueling during exercise. Even well-trained athletes can experience early onset fatigue due to poor hydration or fueling.^{1, 44, 121} This is because adequate nutrition and energy availability has been implicated to affect both central and peripheral factors in fatigue development.^{35, 38, 84, 90}

There is a clear association between muscle metabolism and fatigue. Through manipulation of the process of energy supply or energy depletion, increases in: power output, speed, or time to fatigue can be achieved.^{35, 90} Carbohydrates (CHO) have been shown to be the most effective macronutrient for increased performance during prolonged and intermittent high intensity exercise.^{28, 35, 90, 173}

The mechanisms in which CHO, impact fatigue were outlined in the energy supply/depletion model of fatigue. Depending on exercise intensity and duration, CHO feedings help to maintain blood glucose levels and decrease the reliance on glycogen stores in the muscle and liver. The availability of exogenous carbohydrates is crucial for sparing glycogen stores within the muscle.⁸⁴ In the muscle, depleted glycogen stores lead to fatigue and decreased exercise performance due to the unavailability of immediate energy sources for contracting muscles.⁹⁰ Bergstrom et al.¹⁰ were among some of the first investigators to demonstrate the importance of

muscle glycogen concentrations on exercise performance. After increasing muscle glycogen content through diet, the researchers correlated increased muscle glycogen concentrations with improved endurance capacity.¹⁰ CHO supplementation during exercise can help to increase time to fatigue and performance by offsetting the reliance on muscle and liver glycogen stores, prevent hypoglycemia, and allow for high rates of exogenous CHO oxidation needed to sustain exercise intensity.^{34, 35, 84, 90}

The ergogenic effects of CHO during exercise are not limited to preventing substrate depletion. The ingestion of CHO also appears to have an effect on CNS and neuromuscular control. As described earlier in the substrate depletion model of fatigue, hypoglycemia can lead to increases of free tryptophan and neurotransmitter imbalances leading to CNS fatigue and early exercise termination. Davis and colleagues demonstrated the benefits of CHO ingestion during exercise in decreasing the effects of neurotransmitter imbalances in central fatigue.⁴¹ In their study, participants performed prolonged exercise for 200 minutes, experiencing a drop in blood glucose and a seven-fold increase in plasma free tryptophan.⁴¹ However, when the subjects consumed CHO (at a rate of about 1 g/min), there was no change in blood glucose or plasma free tryptophan, and fatigue was delayed by about 1 hour.⁴¹ Exercise performance and motor control may also be influenced through CHO ingestion or even the presence of CHO in the mouth or intestinal tract.^{23, 62, 125, 129} Chambers et al.²³ demonstrated that a CHO mouth rinse during exercise compared to a placebo led to heightened activation of the motor control areas of the brain as well as improved performance during cycling time trials. Further, Gant et al⁶² was the first to demonstrate the ability of CHO in the mouth to immediately increase the excitability of corticomotor pathway and increase maximum voluntary force production. They suggested that, “afference from oral receptors is integrated with descending motor output,” representing a “novel form of sensorimotor

integration”.⁶² This sensorimotor integration from the presence of CHO in the mouth helped to increase corticomotor output to both fatigued and non-fatigued muscles. These findings help to affirm the role that CHO ingestion has on delaying CNS and neuromuscular fatigue. However, despite the possible role that CHO may have on the sensorimotor system and neuromuscular control, the majority of sports nutrition research has focused on the ergogenic effects of CHO for improving performance through increasing time to fatigue or maintaining power output.

Conversely, the role of CHO ingestion during exercise may also be associated with injury prevention. Schlabach¹⁵⁵ hypothesizes that ingestion of adequate amounts of CHO during exercise would delay fatigue and therefore decrease an athlete’s risk for injury. Given the ergogenic effect that CHO have on central command and muscle metabolism, it is likely that carbohydrates may also play a role in maintaining neuromuscular control and muscle activation patterns crucial for maintaining functional joint stability. Unfortunately, CHO ingestion has only anecdotally been associated with injury prevention. The effects of CHO feedings on neuromuscular characteristics related to fatigue and risk of non-contact injury have not been studied. Therefore, the purpose of this dissertation research is to evaluate the effects of a carbohydrate-electrolyte (CHO-E) feeding compared to a placebo (PLA) on postural stability, muscle activation patterns, and landing mechanics during and immediately following intermittent high-intensity exercise to fatigue. The results of this study will help determine if CHO-E feedings during exercise can reduce some of the changes in lower extremity landing kinematics that result from fatigue. This study will be the first to demonstrate a relationship between nutrition fueling and its impact on risk factors of non-contact injury. The results of this study will help to demonstrate the importance of sports nutrition feeding strategies for injury prevention.

2.6 METHODOLOGICAL CONSIDERATIONS

2.6.1 Measurement of Lower Extremity Kinematics

Three-dimensional (3-D) motion analysis is commonly used in the laboratory setting to capture dynamic movement. Within the laboratory setting marker based systems using retro-reflective markers and infrared cameras are regularly used to calculate hip, knee, and ankle joint angles during movement.^{5, 17, 24, 76} The least invasive marker-based system uses small reflective spheres that are temporarily adhered to the subject's skin over specific anatomical landmarks. More invasive procedures have used markers attached to pins that have been surgically placed in cortical bone.¹³⁹ Using skin based markers may include some error due to skin movement artifact, however, these errors are systematic and repeatable.⁵⁰ The kinematics of skin based marker systems has demonstrated repeatability between various movements within subjects.⁹ Additionally, intra- and inter-day reliability of lower extremity kinematics using skin markers is very high within day, especially for movement in the sagittal plane.⁸⁹ These procedures have been commonly used in research and have demonstrated good to excellent reliability in collecting kinematic data during dynamic movements (within session ICC: 0.933- 0.993; between session ICC: 0.595- 0.922).⁵³

Investigators have employed a variety of dynamic tasks to study lower extremity kinematics and risk factors for non-contact knee injury. Some of these tasks include drop landings, drop jumps, and stop-jumps performed with either one or both legs.^{5, 17, 24, 76, 128, 169} Stop jump movements have been selected because they are more dynamic than drop landings and more closely simulate movements in which non-contact knee injuries commonly occur.⁸ During video analysis of non-contact ACL injuries, it was seen that injuries typically occurred during single-leg landings.¹²⁷ Additionally, these injuries occur during movements that involve a quick deceleration

such as a cutting maneuver or landing from a jump.¹³ Single-leg stop-jumps mimic motions commonly seen in athletics that may highlight an athlete's risk for non-contact injury. Kinematic variables analyzed during the stop-jumps will include knee flexion and varus/valgus angle at initial contact and peak hip flexion angle. These variables were chosen because non-contact ACL injuries typically occur at or near full knee extension with excessive valgus loading during acceleration or deceleration motions.^{68, 69, 75, 160}

2.6.2 Dynamic Postural Stability Index (DPSI)

The ability for an individual to maintain balance when transitioning from a dynamic to a static state is known as dynamic postural stability.⁶⁴ Jump landing tasks and the ability for a subject to stabilize after landing are commonly assessed in research as an aspect of lower extremity motor and postural control.^{148, 149, 151, 159, 174, 177} Decreased postural stability has been identified as a risk factor for lower extremity (ankle and knee) injury in athletic populations.^{2, 104, 150, 161, 172, 178} The DPSI is a measure of how quickly an individual is able to stabilize after a dynamic task. The DPSI is measured using ground reaction force data collected from a force plate and is sensitive to changes of force in the medial-lateral, anterior-posterior, and vertical directions. Additionally, DPSI has been demonstrated to be a precise (SEM = 0.03) measure with excellent test-retest reliability (ICC = 0.96), and is as accurate and precise as time to stabilization measures.¹⁷⁷ Wikstrom and Ross used ground reaction force data from a single-leg jump landing to assess DPSI. The single-leg jump landing protocol for this dissertation is similar to the protocol used by Ross¹⁴⁷⁻¹⁵¹ and Wikstrom.^{175, 176} The protocol will normalize the jump distance according to body height as used by Sell et al.¹⁵⁶ This single-leg jump landing protocol has been used previously in the NMRL and has demonstrated good intersession reliability.¹⁵⁷

2.6.3 Electromyography (EMG)

Measurement of the electrical activity of a muscle can be performed using EMG. During normal movement, in order for muscles to contract, a motor neuron must transmit an impulse (action potential) to the muscle fibers it innervates.^{43,93} This motor neuron and all of the fibers it innervates is known as a motor unit. As the action potential from the motor neuron propagates across the neuromuscular junction to the muscle fibers within the motor unit, electrical activity known as a motor unit action potential (MUAP) is generated.^{43,93} This electrical activity is a result of muscle fiber depolarization and repolarization. The sum of the MUAP of multiple motor units at a specific muscle site is the signal that is usually evaluated for EMG.^{43,93} An increase in the amplitude of EMG signals is seen when the firing rate of an already recruited motor unit is increased or when more motor units are recruited.¹⁸² Bipolar surface electrodes placed on the skin of large superficial muscles are able to detect the MUAP of muscles within a 1-2 cm area around the electrodes.^{43,74,}

93

Possible limitations in recording EMG signals include movement of the electrodes and leads, and crosstalk between muscles which could result in signal noise or contamination.^{74,93} Proper placement and securing of the electrodes on the muscle can help to eliminate causes for signal noise.⁷⁴ Specific and standardized guidelines have been developed by the Surface ElectroMyoGraphy for the Non-Invasive Assessment of Muscles (SENIAM) project for accurately locating and placing electrodes on skeletal muscles for EMG research.⁷⁴

In addition to electrode placement, appropriate data collection and processing techniques are also needed to accurately measure muscle activity using EMG. The Journal of Electromyography and Kinesiology have published guidelines for EMG data collection, filtering, and processing.¹⁰⁹ Filtering applied to EMG signals helps to reduce noise that occurs outside of

the physiologic frequencies of motor activation. A bandpass filter to eliminate frequencies above and below specified ranges (5-10 Hz to 400-500 Hz) is recommended.^{93, 109} Suggested sampling rates of EMG signals should be at least twice the highest frequency cut-off of the bandpass filter.^{93,}

109

Electromyography signals are often processed for interpretation in the time-domain. This is done by first rectifying the data by calculating the absolute value of the EMG signal. The area under the rectified voltage curve can then be calculated across a designated time interval. This process is known as integrated EMG (IEMG) and is measured in volts per second (V/s).¹⁰⁹ In order to standardize IEMG signals, the process of normalization is commonly employed. Normalization is conducted by dividing the amplitude of the signal of interest from one muscle by the amplitude of the signal from a maximal voluntary isometric contraction of the same muscle.¹⁰⁹ Therefore, normalized IEMG is expressed as percent milliseconds (% ms) and represents a percentage of peak activity associated to a designated linear envelope. Normalized IEMG has been used to evaluate the pre-activity and reactivity of muscles during dynamic exercise. Muscular pre-activity is a neuromuscular event that occurs in preparation for an impact, catch, or landing.^{46, 80, 116} It is the continuous build-up of muscle activity that occurs pre-contact to allow the use of muscle-tendon contractile and elastic energy properties to form the initial stiffness of the muscles surrounding a joint.^{46, 80, 116} This initial stiffness contributes to functional joint stability by helping provide adequate joint deceleration to protect the surrounding structures and ligaments from injury during dynamic tasks.^{46, 80, 116} For muscles of the lower extremity, this pre-activity typically occurs 150 milliseconds before contact from a jump or drop landing.^{25, 114} In addition to pre-activity (feed-forward motor control), muscle reactivity has been used as a measure of feedback motor control. Muscle reactivity is regulated by the feedback process of neuromuscular control and occurs after

the onset of joint loading. This dynamic restraint mechanism helps to control joint forces during movement and joint loading that may expose the joint to injury.^{47, 103, 165, 185} The first 150 milliseconds following initial contact from a landing is generally collected to determine the reflexive activity of lower extremity muscles surrounding the knee.^{103, 165} Pre-activity and reactivity of the hamstring and quadriceps muscles are commonly investigated to evaluate the neuromuscular control of the knee joint. Therefore, in order to assess feed-forward and feedback mechanisms of neuromuscular control of the knee, the IEMG activity of the vastus lateralis, vastus medialis, medial hamstring, and lateral hamstring during the 150 millisecond envelope of time directly before and following initial contact of a stop-jump will be collected.

2.6.4 Intermittent High-Intensity Exercise Protocol

Fatigue has shown to hinder landing mechanics and stability, increasing an athlete's potential for injury.^{17, 159, 169, 174} Understanding the onset and severity of fatigue during exercise and activities has implications for both injury prevention and performance enhancement. A variety of different fatiguing exercise protocols have been used in the literature. Wikstrom¹⁷⁴ and Brazen¹⁷ both used a functional fatigue protocol incorporating various jumping, landing, and agility exercises continuously until subjects were fatigued. They then immediately tested subjects on single-leg jumping and balance tasks. While their protocols were effective in eliciting neuromuscular fatigue during functional movements, they did not include any running and therefore do not necessarily simulate athletic training or competition. Others, like Phillips,¹³³ Welsh,¹⁷³ and Winnick¹⁸¹ have used an intermittent high-intensity exercise (IHE) protocol divided into four quarters to better simulate playing conditions of intermittent high intensity sports. Their IHE protocols primarily incorporate shuttle running at various speeds in conjunction with some jumping tasks and an

incremental run to fatigue following the last quarter. The IHE protocol used by Phillips, Welsh, and Winnick did not include many jumping, landing, or agility tasks, and did not include any measures to assess kinematics and neuromuscular fatigue. Therefore, in order to mimic athletic competition and effectively induce neuromuscular fatigue, the IHE protocol used in this study will be a combination of those used by both groups of researchers. The IHE protocol in this dissertation will include treadmill running at various speeds in addition to jumping, landing, and agility exercises. Additionally, the IHE protocol in this dissertation will also incorporate analysis of kinematics, muscle activity, and dynamic postural stability during tasks performed before, during, and after the IHE activity. Pilot research of this protocol in the NMRL has demonstrated the ability for this IHE to induce both fatigue and alterations in landing kinematics. The pilot study demonstrated that the IHE protocol decreased knee flexion angle at initial contact, moved knee angles at initial contact more towards valgus, and decreased peak hip flexion during single-leg stop-jumps.

2.6.5 Carbohydrate Feedings

The carbohydrate feedings for this dissertation will be provided in the form of CHO-E beverages. Carbohydrate feedings were chosen to be given in this form to better simulate game and practice situations in IHE sports where sports drinks are the preferred fueling source.¹⁹ The CHO-E beverages will be a 6% CHO solution with 0.46 g/L of sodium and 0.13 g/L of potassium. The carbohydrate and electrolyte content of the beverages meets the recommendations for sports drinks set by the American College of Sports Medicine, the Academy of Nutrition and Dietetics, and the National Athletic Trainers Association.^{22, 146, 154} These organizations recommend that sports drinks contain between 4-8% CHO and around 0.3-0.7 g/L of sodium and 0.08-0.2 g/L of potassium.^{22,}

^{146, 154} The inclusion of carbohydrates during exercise is recommended for high intensity exercise or activities lasting longer than 60 minutes.^{19, 84} The volume and timing of administration of the CHO-E beverage in this study has been determined in order to optimally provide the right amounts of CHO and volume of fluid for exercise while limiting any gastrointestinal discomfort.^{22, 146, 154} The optimal amount of CHO during exercise is recommended at 30-60g per hour (or about one gram per minute).⁸⁴ Additionally the provision of 15-30g of CHO immediately before high intensity or extended exercise is also recommended.^{19, 84} In order to maintain blood glucose levels during exercise, it is recommended that feedings be spread across the duration of the event in about 15-minute intervals.^{19, 84} The volume of fluid consumed during exercise is mainly dependent on individual sweat rates, however, general guidelines have recommended between 150-350 ml of fluid immediately before and every 10-20 minutes during exercise.^{48, 154}

3.0 METHODOLOGY

3.1 EXPERIMENTAL DESIGN

The study followed a double blind crossover design. Subjects served as their own controls and the sequence in which they received either the treatment (CHO-E beverage) or PLA beverage was counterbalanced.

Independent Variables:

A 6% CHO-E or flavored non-calorie PLA beverage was administered during the exercise sessions. The CHO-E and PLA beverages were identical in volume, taste, appearance, and electrolyte content. Both beverages contained 0.46 g/L of sodium and 0.13 g/L of potassium.

- 6% CHO-E beverage
 - o Total volume: 19 ml/kg body weight spread out across the IHE protocol
- PLA beverage
 - o Total volume: 19 ml/kg body weight spread out across the IHE protocol

Dependent Variables:

- During the stop-jump task
 - o Knee flexion angle at initial contact
 - o Knee valgus/varus angle at initial contact
 - o Peak hip flexion angle
 - o EMG pre-activity of vastus lateralis, vastus medialis, medial hamstring, and lateral hamstring 150 msec. prior to landing

- EMG re-activity of vastus lateralis, vastus medialis, medial hamstring, and lateral hamstring 150 msec. after landing
- During the jump-landing task:
 - Dynamic postural stability index score

3.2 SUBJECTS

Physically active males and females ages 18-35 were recruited for this study. Both males and females were recruited for this study to increase generalizability to athletic populations. Previous research has reported similar effects of fatigue between men and women.^{8, 152} Additionally, research has demonstrated performance benefits in both males and females consuming CHO feedings during exercise.¹⁶⁶ In order to help control for any other underlying differences between sexes an equal number of males and females were recruited for this study. Furthermore subjects were blocked by sex, with an equal number in each block receiving the same treatment sequence. Participants were recruited from the universities, colleges, and communities around the Pittsburgh area. Prior to participating in the study, subjects were provide written informed consent in accordance with the University of Pittsburgh Institutional Review Board. Eligibility was determined by the following inclusion and exclusion criteria:

3.2.1 Inclusion Criteria

- Males and females ages 18-35 years

- Physically active (defined as participating in physical activity a minimum of 4 times per week for at least 45 minutes each session including some high intensity activities)
- Currently participating in competitive organized sports (defined as sports played at moderate to vigorous intensities similar to those seen in hockey, basketball, swimming, soccer, or football and compete against other teams or individuals a minimum of 5 times per year) with a minimum of at least 3 consecutive years of involvement.

3.2.2 Exclusion Criteria

- Any lower extremity musculoskeletal injury within the 6 months prior to day one testing
- Any lower extremity surgery or fracture to dominant limb in the past 4 years or any history of knee surgery
- Head injuries (ex: concussion) within the previous three months
- Any disorder that could affect equilibrium or neuromuscular control
- Any history of pulmonary, vestibular, cardiovascular, or vascular condition contraindicated to vigorous exercise
- Pregnancy (confirmed via history and date of last menses)
- Any allergies to food dyes or adhesive tapes
- Diabetes or any other metabolic disorder

3.3 SAMPLE SIZE ANALYSIS

Sample size was estimated for interaction effects in a two-way repeated measures analysis of variance (crossover design) using pilot data for varus/valgus knee angle and hip flexion kinematic data. The two repeated measures are time and treatment, with time having three levels (baseline, break-3, and break-4) and treatment having two levels (CHO-E and PLA). Based on sample size calculations, 20 physically active adults ages 18-35 were required. To account for subject attrition, an additional four subjects were recruited for this study for a total of 24 subjects.

3.4 INSTRUMENTATION

3.4.1 Force Plate

A force plate (Kistler 9286A, Amherst, NY) was used to collect ground reaction force data and to assess dynamic measures of postural stability. A sampling frequency of 1500 Hz was utilized for all of the dynamic tasks. Force plate data was passed through an amplifier and analog to digital board (Vicon Motion Systems Inc., Centennial, CO) and stored on a personal computer. Vicon Nexus software (Vicon Motion Systems Inc., Centennial, CO) was used to record and synchronize force plate data with motion analysis and EMG data.

3.4.2 Motion Analysis System

A ten camera marker based motion analysis system (Vicon Motion Systems Inc., Centennial, CO) was used to measure lower extremity kinematics. The system uses high-speed cameras with infrared light-emitting-diodes to capture and track light reflected off reflective markers placed on

specific anatomic landmarks of the lower extremity. Eight cameras are mounted on the walls and two are placed on tripods to create the capture volume. Two-dimensional trajectories of the reflective markers are captured by the cameras and combined to create 3D coordinate data using Vicon Nexus software (Vicon Motion Systems Inc., Centennial, CO). The motion analysis system was calibrated according to manufacturer's instructions, using the wand calibration method. Data was collected at a sampling frequency of 250 Hz. Position and angular data collected using this system in our laboratory has been shown to be accurate with a root mean square error of 0.002 m and 0.254° respectively. This system will be used to collect each subject's lower limb kinematics during the stop-jump task.

3.4.3 Electromyography (EMG)

Muscle activation patterns were collected using silver-silver chloride, pre-gelled bipolar surface EMG electrodes (Ambu Inc., Glen Burnie, MD) and an eight channel telemetric system (Noraxon USA Inc, Scottsdale, AZ), using a sampling frequency of 1500 Hz. The recorded EMG signals are relayed from the surface electrodes through leads attached to a small wireless transmitter where the signal is pre-amplified and filtered using a first order high-pass filter set to 10 Hz and a base gain of 400. The pre-amplified signal is then transmitted via radio bands (FCC ID: R8KUGWG4USHN33A) to a wireless transmitter. The wireless transmitter has 11 different Wi-Fi channel frequencies to select from to transmit the signal to a receiver where the digital signal is converted to an analog signal and stored on a personal computer. Signals from the receiver were collected and synchronized with the motion analysis data using Vicon Nexus software.

3.4.4 Heart Rate

Heart rate (beats per minute) data was collected using a Polar heart rate monitor strap and training computer (Polar USA, Lake Success, NY). Heart rate was monitored continuously throughout the entire testing protocol. Heart rate data was collected to ensure subjects are exercising at appropriate intensities.

3.4.5 Anthropometrics

A wall stadiometer and electronic scale (Life Measurement Instruments, Concord, CA) were used to collect subject's height and weight respectively. Leg length was measured with a cloth tape, and knee and ankle width were measured using an anthropometer. Height and weight measurements were collected for demographic information. Lower extremity anthropometric measurements were used for the motion analysis software to build a 3-D model of the subjects in order to calculate joint angles.

3.4.6 Body Composition

Body composition was measured using The BOD POD Body Composition System (Life Measurement Instruments, Concord, CA). The device consists of a single fiberglass structure containing two chambers (reference chamber, test chamber) the test chamber is where the subject sat during testing. Variations in conditions such as chamber size and temperature are accounted for with a two point calibration process that precedes the test and is used for normalization. Body composition was measured for subject demographic data.

3.4.7 Treadmill

A treadmill (Woodway USA Inc., Waukesha, WI) was used to determine peak treadmill velocity and for the running tasks during the IHE protocol.

3.4.8 Vertec

The Vertec Vertical Jump Tester (Sports Imports, Columbus, OH) was used to measure maximal vertical jump height. It consists of a free-standing vertical steel frame with horizontal plastic vanes which are rotated out of the way by the dominant hand to indicate the jump height reached. Vanes are in half-inch increments spanning 24 inches, and the heights of the vanes are adjustable from 6-12 feet.

3.4.9 Biodex

A Biodex System 3 Isokinetic Dynamometer (Biodex Medical Systems, Shirley, NY) was used to isolate and stabilize the quadriceps and hamstring muscles in order to collect 5-second maximum voluntary isometric contractions (MVIC) of the dominant leg.

3.4.10 Lactate

Blood lactate levels were measured using a Lactate Pro 2 (ARKRAY Inc, Japan) test meter and strips. Lactate levels were measured from a small drop of blood obtained from a finger stick. This technology has been proven as a reliable and valid measure of blood lactate levels in exercising

subjects.¹³⁶ Blood lactate levels were measured as a physiological check to monitor that subjects were exercising at appropriate intensities.

3.4.11 Rating of Perceived Exertion (RPE)

The OMNI scale for RPE was used to measure participant's perceived effort during the IHE protocol and PTV tests. The OMNI scale quantifies perceived effort on 0-10 scale using a picture chart with 0 corresponding to "extremely easy" and 10 meaning "extremely hard." The OMNI scale has been demonstrated to be a valid and reliable measure of effort in exercising adults.^{144, 171} The OMNI RPE scale was collected as a way to monitor the subject's perceived effort during exercise.

3.4.12 Diet Recall

The Automated Self-Administered 24-hour recall (ASA24) was used to collect and analyze subject's food consumption. The ASA24 was developed by the National Cancer Institute and utilizes multi-level food probes to accurately assess food types and amounts as well as a triple pass system that cues users to include often forgotten or overlooked items such as beverages and condiments. The program includes pictures of food in multiple portion sizes to assist the user in selecting the appropriate portion size. Diet recalls are analyzed using the USDA's version 4.1 Food and Nutrient Database for Dietary Studies. Data from the ASA24 was used to assure subjects consumed similar diets before each of their test sessions.

3.5 TESTING PROCEDURES

All subjects reported to the Neuromuscular Research Laboratory for three test sessions lasting approximately 120 minutes each. During the first visit to the lab the inclusion and exclusion criteria were reviewed to determine eligibility for the study. All subjects signed an informed consent to participate form, approved by the University of Pittsburgh Internal Review Board prior to participation in this study. Demographic, anthropometric, and baseline measures were then collected. Subjects then perform a familiarization session with the IHE protocol. During this session subjects were also instructed on the procedures to follow prior to their next scheduled test. Prior to testing on visits two and three, subjects were asked to refrain from any exercise 12 hours before their scheduled lab visit. Subjects were also required to fast for 8-hours prior to their scheduled testing time. During their participation in the study, subjects were also be asked to refrain from taking any dietary supplements that may aid in exercise performance.

3.5.1 Day 1 Testing

- Signing informed consent
- Demographics
- Anthropometrics
- Peak vertical jump
- Peak treadmill velocity
- IHE protocol familiarization/review

3.5.1.1 Anthropometrics and Body Composition

A wall mounted stadiometer (Seca, Hanover, MD) and an electronic scale (COSMED USA, Concord, CA) was used to capture standing height and body weight respectively. Body weight was taken with the subject wearing minimum clothing (males = spandex shorts; females = spandex shorts + sports bra OR swimsuit). Body composition was measured using the Bod Pod. Subjects were required to wear a tight fitting bathing suit or spandex outfit with a swim cap covering the hair to reduce air impedance. Subjects entered the Bod Pod and sat within the system for approximately 1-minute. Subjects were asked to breathe regularly and remain motionless during the testing procedure. The specific variables analyzed included percent body fat and fat free mass.

3.5.1.2 Peak Vertical Jump (PVJ)

Peak vertical jump height was measured using the Vertec Vertical Jump Tester (Sports Imports, Hilliard, OH.) and a previously established procedure that demonstrates high reliability (ICC>0.95).¹⁰¹ Subjects stood directly under the Vertec horizontal vanes with both feet flat on the ground, legs and torso straight. To determine their standing vertical reach height, subjects reached up with a straight arm, wrist, and hand to touch the highest vane they could reach. Following this, subjects were instructed to stand on both legs and perform a counter-movement vertical jump as high as they can and touch the highest possible vane. Three sub-maximal warm-up/practice trials were permitted, followed by three maximal-effort measured trials. Peak vertical jump height was calculated as the participant's maximum vertical jump height minus their standing vertical reach height.

3.5.1.3 Peak Treadmill Velocity (PTV)

Peak treadmill velocity (PTV) was measured on a treadmill adapting a previously used protocol.¹²³ Subjects warmed up at a speed comfortable to them for 5 minutes before testing began. The test began at 8 km/hr and a 0% incline. The treadmill speed increased 1.0 km/hr. every minute until volitional fatigue. Peak treadmill velocity was the highest speed that the subjects could maintain for one minute.

3.5.1.4 IHE Review and Familiarization

The order and description of tasks in the IHE protocol was reviewed with each subject. Demonstrations of each task was also provided prior to the subject practicing the movement on their own. Subjects were instructed to go through the IHE protocol at least one time at 50% effort. If subjects felt like they needed more practice at a particular task they were permitted to have additional practice until they felt comfortable with the movements.

3.5.2 Day 2 and 3 Testing

Day two and day three testing were separated by at least one week. Procedures for day two and day three testing were identical, with the exception of the administration of either the CHO-E or PLA beverage. Subjects were required to abstain from any food or drink (with the exception of water) and from any exercise 8 hours prior to day two and day three test sessions. Subjects were also asked to follow the same diet prior to day three testing that they consumed prior to their day two test. Upon arriving at the lab, subjects were asked to change into compression shorts, t-shirt, and athletic shoes.

Day 2/Day 3 Testing:

- 24-hour diet recall
- EMG and marker set up
- Baseline single-leg stop-jump and jump landing
- IHE protocol
- Post single-leg stop-jump and jump landing

3.5.2.1 24 Hour Diet Recall

Subjects completed a 24-hour diet recall using the computer based Automated Self-Administered 24-hour recall (ASA24). Each subject logged in with a unique username and password and recorded everything that they ate or drank in the previous 24 hours. This data was used to ensure the diet before both day two and day three testing was similar.

3.5.2.2 EMG

Muscle activity of the dominant leg (defined as the leg subjects would use to kick a ball as far as possible) was collected from the vastus lateralis, vastus medialis, medial hamstrings, and lateral hamstrings using surface EMG. Electrode placement sites were based on Surface ElectroMyoGraphy for the Non-Invasive Assessment of Muscles (SENIAM) project guidelines.⁷⁴ Electrode placement location was determined by palpating the selected muscle and marking the belly of the muscle with a surgical pen. The vastus medialis electrodes were placed at an oblique angle approximately 80% of the distance between the anterior superior iliac spine (ASIS) and the medial tibiofemoral joint line. The vastus lateralis electrodes were placed about 2/3 the distance between the ASIS and the lateral border of the patella. The electrodes for the medial hamstrings were placed about half the distance between the ischial tuberosity and the tibial medial epicondyle.

Lateral hamstring electrode placement was half of the distance between the ischial tuberosity and the lateral tibial epicondyle. Prior to electrode placement, the subject's skin was shaved, lightly abraded with an emery board, and cleaned with isopropyl alcohol in a small area around the site of electrode placement. Two 22x30 mm silver-silver chloride electrodes (Ambu Inc., Glen Burnie, MD) were placed side-by-side on the belly of the muscle parallel to the muscle fibers. A small wireless transmitter was connected to each pair of electrodes and secured to the skin using double-sided adhesive tape. After manual muscle testing was performed to confirm proper placement of the electrodes, the electrodes and wireless transmitter was secured using surgical tape and pre-wrap. This was done to minimize noise from excessive movement of the transmitter and electrodes. After electrodes and transmitters were secured, a 5-second MVIC test was performed for the quadriceps (vastus medialis and vastus lateralis) and hamstring (medial and lateral) muscle groups. The MVIC test was completed on a Biodex isokinetic dynamometer using the knee flexion and extension attachment with the dynamometer set to isometric mode. The subject was in a seated position with their knee joint center aligned with the axis of rotation of the dynamometer and their leg positioned at 45° of knee flexion. Padded straps were used to secure the subject's torso, waist, and upper thigh (of the tested leg). The subject's lower leg was also secured to the knee flexion/extension attachment using a padded strap. The straps helped to secure the subject and isolate the muscle groups of interest for the MVIC. The MVIC EMG data was used to normalize the respective EMG activity during the dynamic task (single-leg stop-jump).

3.5.2.3 Single-leg Stop-jump and Jump Landing

Prior to testing, anthropometric data including height, weight, leg length, knee width, and ankle width was collected and entered into the motion analysis software. Leg length was measured from the ASIS to the medial malleolus using a cloth tape measure. Knee width was measured with an

anthropometer between the lateral and medial femoral epicondyles. Ankle width was also measured with an anthropometer between the lateral and medial malleoli. Sixteen reflective markers were adhered (using double stick tape) bilaterally to anatomical landmarks of the lower extremities and the pelvis using Vicon Nexus software Plug-in Gait model (Vicon Motion Systems Inc., Centennial, CO). These landmarks included the head of second metatarsal, lateral malleolus, posterior heel, lateral shank, lateral femoral epicondyle, lateral thigh, ASIS, and posterior superior iliac spine.⁸⁹ The lateral thigh markers were placed in line and halfway between the subject's greater trochanter and the lateral femoral epicondyle marker. The lateral shank markers were placed in line and halfway between the lateral femoral epicondyle marker and lateral malleolus markers. Following marker placement, participants stood in anatomical position (with their knees and ankles directly in line with their hip joints) on one force plate for a static capture trial. This static capture served as a baseline reference for joint angle calculations.

Single-leg Stop-jump

The stop-jump task consisted of a single-leg broad jump using the dominant leg, over a distance of 40% of the subject's body height, to a single-leg landing on the same leg, immediately followed by a single-leg vertical jump for maximum height. Data was collected during a one second period following initial contact with the force plate, capturing the initial landing of the task. Each subject was given at least five practice trials but they were able to take as many as needed to become comfortable with the task. Three successful trials of the task was collected and used for data analysis.

Trials were discarded and repeated if subjects failed to land directly on the force plate. Trials were also repeated if the non-weight-bearing leg touched down on the force plate or the ground around the force plate.

Single-leg Jump Landing

Subjects were tested with a single-leg jump landing test that previously has been used in the Neuromuscular Research Laboratory and has demonstrated good intersession reliability.¹⁵⁷ The single-leg jump landing test required subjects to complete a jump in the anterior direction. Subjects were positioned 40% of their body height away from the edge of a force plate. A 30 cm hurdle was placed at the midpoint of this distance. Subjects were instructed to jump in the anterior direction using a two-footed jump take-off over the 30 cm hurdle, land on the force plate with only the dominant limb, stabilize as quickly as possible, place their hands on their hips, and balance for 5 seconds while looking straight ahead. A total of three successful trials were collected and averaged for analyses. Subjects were given three practice trials to familiarize themselves with the task.

Trials were discarded and repeated if subjects failed to jump over or came in contact with the hurdle, hopped on the test leg after landing, the non-weight-bearing leg touched down on the force plate or the ground around the force plate, or if subjects removed their hands from hips for longer than five seconds.

3.5.2.4 Intermittent High-Intensity Exercise (IHE)

Table 1. Intermittent High-Intensity Exercise Protocol Timeline

Baseline	Quarter 1	Break 1	Quarter 2	Break 2 (Half)	Quarter 3	Break 3	Quarter 4	Break 4	Fatigue Run	Post Fatigue
----------	-----------	---------	-----------	----------------	-----------	---------	-----------	---------	-------------	--------------

The IHE procedures were similar to those previously used in the literature.^{17, 133, 173, 174} The IHE protocol was divided into four quarters with a short break between each quarter and 10 minutes

rest at the half. Each quarter was identical, about 10 minutes in length, and included the following activities.

1. 2 minutes treadmill running at 40% PTV
2. Ladder agility drills, 5x each of:
 - a. Sprint through with one foot in each box
 - b. Sprint though with both feet in each box
3. 30 sec. treadmill running at 95% PTV
4. 30 side-to side bounds
 - a. Subjects performed lateral jumps form one marker/cone to another placed 0.9 meters apart.
5. 2 minutes treadmill running at 55% PTV
6. 30 Mini-trampoline jumps
 - a. Subjects performed a series of small consecutive jumps, that incorporated vertical and horizontal movement, on and off a BOSU apparatus (BOSU Fitness, Canton, OH)
7. 30 sec. treadmill running at 95% PTV
8. Mini-hurdle jumps, 5x through
 - a. A series of five, 15 cm hurdles were placed approximately 60 cm apart and subjects jumped over each hurdle with both feet.
9. 1 minute of treadmill running at 75% PTV
10. 5 vertical jumps
 - a. Subjects performed five consecutive countermovement jumps to a height 50% of their PVJ.

The order of testing at baseline, during the breaks, and post fatigue run is as follows:

1. Blood lactate measure
2. Administration of CHO or PLA beverage (Appendix A)
 - a. 5ml/kg body weight 15 minutes prior to start of IHE and at the half
 - b. 3ml/kg body weight between quarters and before incremental run to fatigue
3. Single-leg stop-jump and jump landing
4. 5 second MVIC
 - a. Performed on the Biodex for both leg extension (quadriceps muscles) and flexion (hamstring muscles)
5. Rest
 - a. 10 minute rest at halftime

Following the fourth quarter, subjects also completed an incremental run to fatigue. The incremental run to fatigue followed the same protocol used for the peak treadmill velocity. Subjects consumed 5 ml/kg body weight of CHO-E or PLA 15 minutes before IHE protocol and at the halftime, and 3 ml/kg body weight between quarters and before the incremental run to fatigue. The single-leg stop-jumps and jump landings were performed at 6 time points (before the IHE protocol, between quarters, at the halftime, and before and after the incremental run to fatigue). Blood lactate levels were also collected during these 6 time points. Heart rate was recorded throughout the entire IHE protocol at 15-second intervals.

3.6 DATA REDUCTION

3.6.1 Kinematics

Landing kinematics were calculated using Vicon Nexus software. The sixteen reflective markers placed on specific landmarks of the subject's lower body (using the Plug-in Gait model) were used to create a three-dimensional (3D) lower body model. The Nexus software filtered raw 3D coordinate data using a general cross-validation Woltring filter.¹⁸⁶ The lower body model was constructed in the software program using a static capture of the marker set and the subject's anthropometric measurements of their lower limbs. The lower body model included joint center estimation and a segment local coordinate system definition that was calculated by the software.⁴² The Newington-Gage model calculated hip joint centers from the mean distance between the ASIS markers as well as the distance between each ASIS and the ipsilateral trochanter.⁴² The location of the trochanter was estimated from the subject's leg length measurements. Joint centers for the knee and ankle were calculated using an associated embedded coordinate system and the respective joint widths taken before marker placement. Following calculation of the joint centers, coordinate systems were then calculated and embedded for each joint center. To calculate the segment local coordinate systems the software first used at least three markers to define a segment. Lower body segments were created for the pelvis, thigh, shank, and foot. Each of these segments then had an orthogonal embedded coordinate system calculated for it. Once coordinate systems had been embedded in both the joint centers and the segments, they were aligned using angular offset values obtained during the static capture. The realigned embedded coordinate systems were used to calculate 3D angles of rotation for each segment by defining the orientation of the distal coordinate

system axes relative to the proximal coordinate system axes. Joint angles were then calculated using Euler angles (following a YXZ Euler rotation sequence).^{42, 89}

Further data reduction was completed using a custom Matlab (Mathworks, Natick, MA) script. Knee flexion angle at initial contact, knee valgus/varus angle at initial contact, and peak hip flexion were averaged across all three stop-jumps for each subject. Initial contact was defined as the time point in which the filtered vertical ground reaction force reached a threshold equal to 5% of the subject's bodyweight. This time point was used to identify knee flexion and valgus/varus angles at initial contact. Peak hip flexion angle was identified as the maximum angle of the femur relative to the pelvis during the stance phase of landing. The stance phase of landing was defined as the time from initial contact to the point when peak vertical ground reaction force dropped below 5% of the subject's body weight.

3.6.2 Dynamic Postural Stability (DPSI)

Dynamic postural stability index was calculated using ground reaction force data in the x, y, and z directions collected by the force plates using a custom Matlab program. The DPSI is a composite score of three stability indices: medial lateral stability index (MLSI), anterior-posterior stability index (APSI), and vertical stability index (VSI).¹⁷⁷ The following formula was used to calculate DPSI values:

$$\sqrt{\frac{\sum\left(\frac{0-x}{body\ weight}\right)^2 + \sum\left(\frac{0-y}{body\ weight}\right)^2 + \sum\left(\frac{body\ weight-z}{body\ weight}\right)^2}{number\ of\ data\ points}}$$

The MLSI and APSI were calculated by the mean square deviations of fluctuations around a zero point in the frontal (x) and sagittal (y) axes of the force plate, respectively. The VSI was calculated

by assessing the fluctuations from the subject's bodyweight in the vertical (z) direction of the force plate. All stability indices were calculated using the first three seconds of ground reaction force data following landing on the force plate. The average of three successful trials were used to calculate DPSI scores during each time point (baseline, break 3, and break 4).

3.6.3 Electromyography (EMG)

Raw EMG data from the stop-jump and MVIC tasks were band-pass filtered between 10 and 500 Hz, rectified, and low-pass filtered at 10 Hz using a fourth order, zero-phase shift Butterworth filter.^{93, 109} The enveloped signals from the stop-jump trials were normalized to the mean amplitude of the middle four seconds of the rectified and filtered baseline MVIC data. Pre-activity and re-activity of the vastus medialis and lateralis, and medial and lateral hamstrings were calculated. Pre-activity data was calculated as the normalized integrated EMG (IEMG) signal during the 150 millisecond interval of time prior to initial contact on the force plate during the stop-jump task. Re-activity data was calculated as the normalized IEMG signal during the 150 millisecond interval of time directly following initial contact on the force plate during the stop-jump task. The IEMG pre-activity and re-activity was averaged for each muscle across three stop-jump trials at baseline, break 3 and break 4.

3.7 STATISTICAL ANALYSIS

Descriptive statistics were calculated for all variables. Separate two-way repeated measures (time and treatment as repeated measures) analysis of variance (ANOVA) was performed to study the

effects of time, treatment, and the interaction between time and treatment on the dependent variables. The assumption of normality of the residuals was tested using the Shapiro-Wilk test. If residuals were not normally distributed, appropriate nonparametric tests or data transformations were performed on the dependent variables. If the assumption of sphericity was violated, appropriate corrections were applied. Post hoc analyses was conducted where appropriate. Statistical significance was set at 0.05 (two-sided) a priori. Statistical analysis was conducted using IBM SPSS Statistics 21 (IBM Corporation, Armonk, NY).

4.0 RESULTS

The purpose of this study was to evaluate the effects of a CHO-E feeding compared to a PLA on postural stability, muscle activation patterns, and landing mechanics during and immediately following intermittent high-intensity exercise (IHE) to fatigue.

4.1 SUBJECTS

A total of 24 (12 male, 12 Female) physically active individuals volunteered for this study. Participant's demographic information is displayed in Table 2. Each of the 24 volunteers successfully completed all three days of testing.

Table 2. Subject Demographic Data

Demographic		N	Mean	SD
Age (years)	All	24	23.0	4.0
	Male	12	23.0	4.0
	Female	12	24.0	3.0
Height (centimeters)	All	24	173.3	7.1
	Male	12	177.5	4.5
	Female	12	169.1	6.7
Weight (kilograms)	All	24	72.9	11.5
	Male	12	80.8	10.5
	Female	12	65.1	5.7
Body Fat (percentage)	All	24	18.0	6.4
	Male	12	12.9	4.0
	Female	12	23.1	3.6

N = number of subjects; SD = standard deviation

4.1.1 Day 1 Testing

On the first day of testing subjects performed PTV and PVJ tests to determine appropriate running speeds and jump heights for the IHE protocol performed on Day 2 and 3 of testing. The summary of results from Day 1 testing is presented in Table 3.

Table 3. Day 1 Testing Results

Performance Variable		N	Mean	SD
Peak Treadmill Velocity (kph)	All	24	15.4	1.6
	<i>Male</i>	12	16.4	1.6
	<i>Female</i>	12	14.4	0.9
Peak Treadmill Velocity Time to Fatigue (sec)	All	24	629.3	117.6
	<i>Male</i>	12	697.7	122.9
	<i>Female</i>	12	560.9	60.2
Peak Vertical Jump (cm)	All	24	52.5	12.5
	<i>Male</i>	12	61.5	10.9
	<i>Female</i>	12	43.5	5.5

N = number of subjects; SD = standard deviation; kph = kilometer per hour; sec = seconds; cm = centimeter

4.1.2 Diet Recall

Subjects were asked to complete a 24-hour diet recall after Day 2 and Day 3 test sessions to ensure similar diets were followed before each day of testing. Results from one 24-hour recall from a subject was not available, therefore a total of 23 pairs of recalls were used in the analysis. A paired samples T-test was conducted and revealed no significant ($p > .05$) difference in the diets between the CHO-E and PLA treatment groups in total calorie, carbohydrate, protein, or fat intake. Diet recall results are displayed in Table 4.

Table 4. Energy and Macronutrient Comparison between CHO-E and PLA Treatments

Nutrient	CHO-E Mean (SD)	PLA Mean (SD)	Paired t-test P-Value
Calories	2,473.4 (1,045.0)	2,340.3 (1,105.1)	0.575
Carbohydrate (g)	270.3 (97.2)	256.0 (138.8)	0.629
Protein (g)	109.3 (67.5)	106.9 (59.3)	0.807
Fat (g)	102.6 (59.4)	93.6 (52.8)	0.348

SD = standard deviation; CHO-E = carbohydrate-electrolyte beverage; PLA = placebo beverage

4.1.3 Intermittent High-Intensity Exercise Protocol and Treadmill Run to Fatigue

A two-way repeated measures ANOVA was used to analyze time to complete each quarter, heart rate, and lactate immediately following each quarter during the fatigue protocol. Overall, there was a significant main effect of time on time to complete each quarter ($F(2.3, 52.0) = 18.3, p < 0.001$), heart rate ($F(1.6, 37.4) = 67.3, p < 0.001$), and lactate ($F(3,69) = 3.4, p = 0.022$), but no significant main effect of treatment or time by treatment interaction (Figures 1-3).

Table 5. Quarter Completion Time, Heart Rate, and Lactate Descriptive Data and Two-way Repeated Measures Analysis of Variance Results

Variable	Beverage	Mean (SD)				Interaction	P-Value	
		Quarter 1	Quarter 2	Quarter 3	Quarter 4		Time	Treatment
Time (sec)	CHO-E	559.0 (24.3)	552.6 (22.3)	550.9 (20.4)	551.0 (21.5)	0.554	<0.001	0.632
	PLA	562.4 (24.9)	554.8 (23.8)	552.8 (21.3)	550.7 (22.7)			
Heart Rate (bpm)	CHO-E	170.9 (10.2)	176.9 (9.3)	176.2 (8.8)	176.0 (9.0)	0.325	<0.001	0.730
	PLA	169.5 (11.6)	176.8 (10.1)	176.3 (9.2)	176.3 (9.0)			
Lactate (mmol/L)	CHO-E	7.3 (2.8)	7.6 (3.8)	7.1 (3.9)	6.3 (2.9)	0.086	0.022	0.097
	PLA	6.8 (6.5)	6.5 (3.4)	6.6 (3.3)	6.8 (4.0)			

sec = seconds; bpm = beats per minute; mmol/L = millimoles per liter; CHO-E = carbohydrate-electrolyte beverage; PLA = placebo beverage

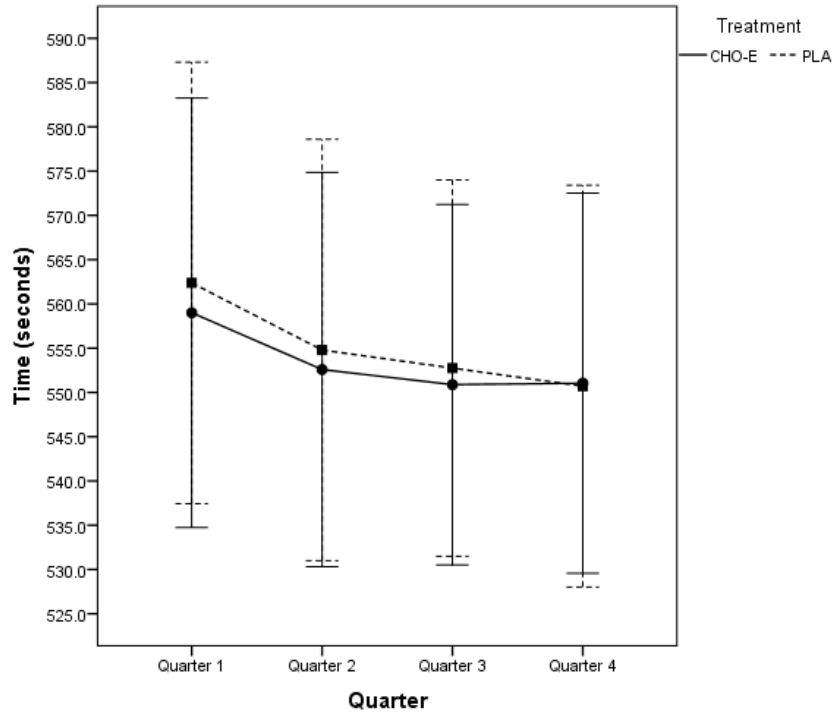


Figure 1. Mean (± 1 SD) Time to Complete each Quarter

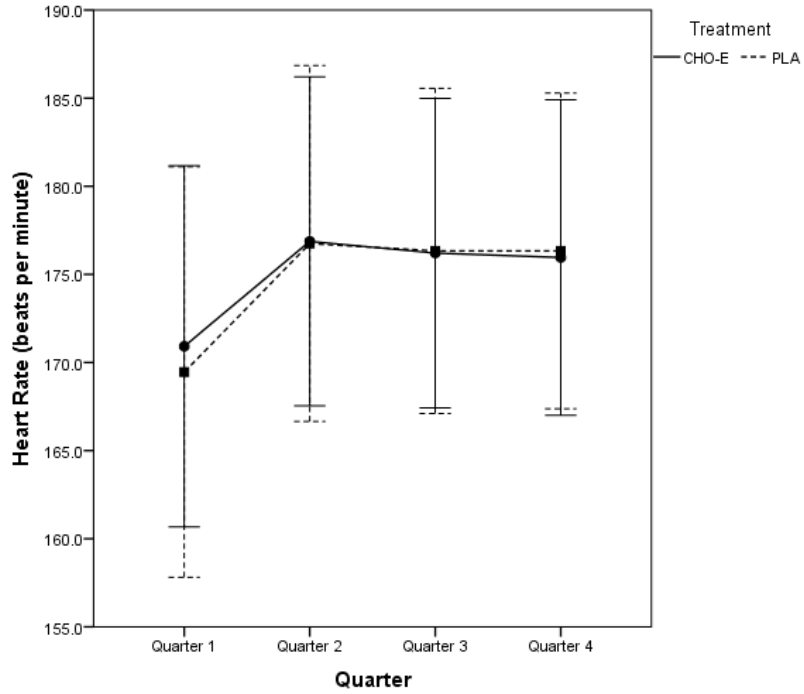


Figure 2. Mean (± 1 SD) Heart Rate across Quarters

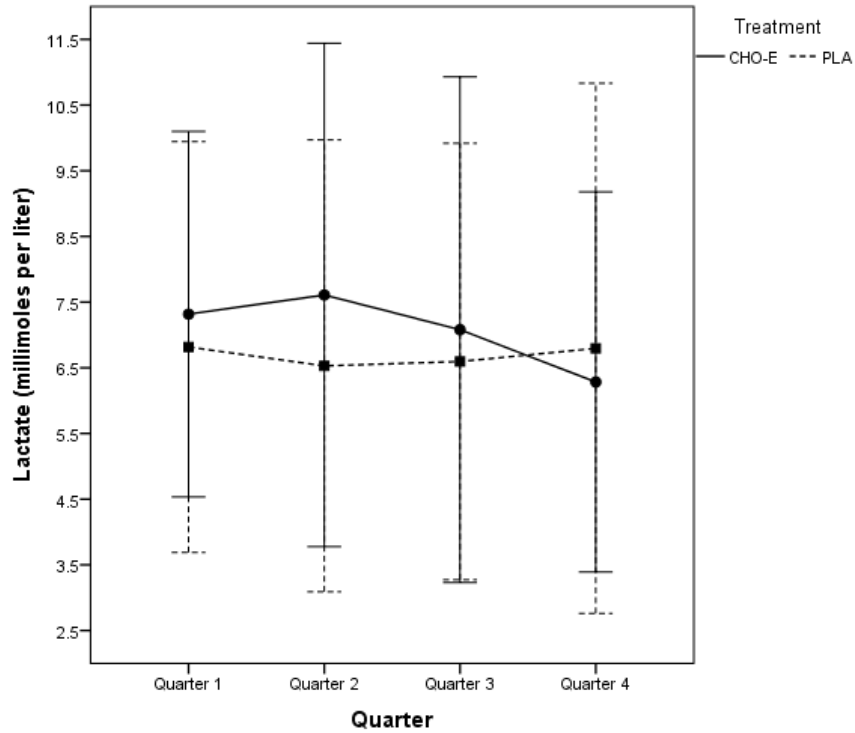


Figure 3. Mean (± 1 SD) Lactate across Quarters

Median RPE was analyzed across all 4 quarters between the CHO-E and PLA groups (Table 6, Figure 4). A Wilcoxon signed ranks test was used to determine if there was any difference in the change in RPE between quarters across treatment groups (Table 7). There was no significant difference in the change of RPE from quarter 1-3 ($p = 0.093$) or from quarter 1-4 ($p = 0.599$) between CHO-E and PLA treatment groups.

Table 6. Rating of Perceived Exertion (RPE) Descriptive Statistics

Beverage	Quarter 1		Quarter 2		Quarter 3		Quarter 4	
	Median	IQR	Median	IQR	Median	IQR	Median	IQR
RPE CHO-E	6	4.0-7.0	7	5.5 - 7.5	7	6.0 - 8.0	8	6.0 - 8.1
PLA	6	4.5 - 7.5	7	6.0 - 8.0	7.5	6.0 - 8.0	8	6.5 - 8.5

CHO-E = carbohydrate-electrolyte beverage; PLA = placebo beverage; IQR = 25th - 75th percentile

Table 7. Comparison of Change in Rating of Perceived Exertion (RPE) between Quarters for

Treatment Groups

Change in RPE	CHO-E			PLA			P-value*
	Mean	SD	Median	Mean	SD	Median	
Quarter 4 - Quarter 3	0.3	0.9	0	0.6	0.8	0.5	0.228
Quarter 3 - Quarter 1	1.5	0.8	1.0	1.1	1.2	1.0	0.093
Quarter 4 - Quarter 1	1.9	1.2	2.0	1.7	1.2	2.0	0.599

*Wilcoxon Signed Ranks Test

SD = standard deviation; CHO-E = carbohydrate-electrolyte beverage; PLA = placebo beverage

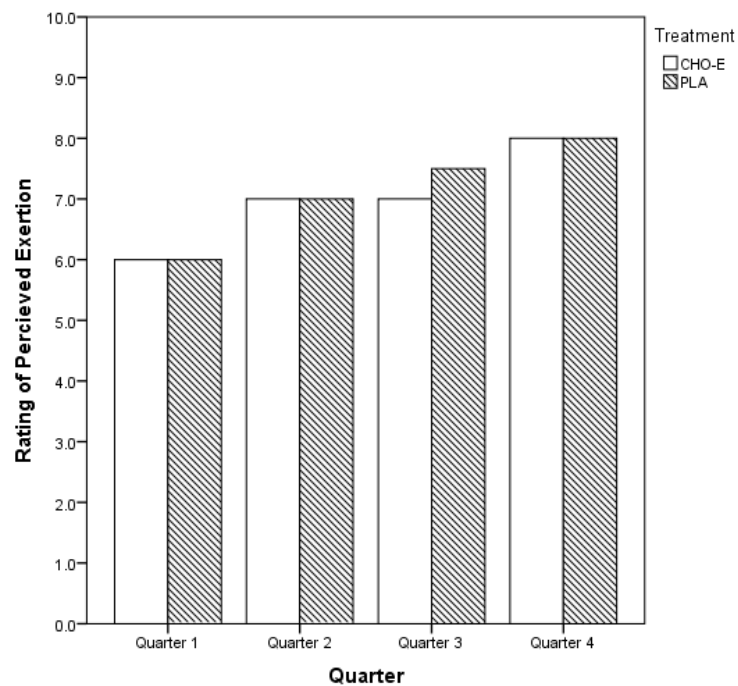


Figure 4. Median Rating of Perceived Exertion across Quarters

Mean time to fatigue during the treadmill run to fatigue was significantly ($p = 0.004$) greater in the CHO-E group (546.75 ± 112.37 sec) compared to the PLA group (520.87 ± 113.78 sec).

4.2 LANDING MECHANICS

Knee flexion angles at initial contact, knee valgus/varus angles at initial contact, and peak hip flexion angles were analyzed during a single-leg stop jump at baseline, break three, and break four of the IHE protocol during the CHO-E and PLA treatment sessions (Table 8).

Table 8. Kinematic Descriptive Statistics and Two-way Repeated Measures Analysis of Variance

Kinematic Variable	Beverage	Mean (SD)			Interaction	P-Value	
		Baseline	Break 3	Break 4		Time	Treatment
Knee Flexion at Initial Contact (degrees)	CHO-E	16.6 (7.9)	13.7 (6.6)	13.8 (4.6)	0.472	0.001	0.441
	PLA	16.5 (5.7)	14.2 (5.5)	15.0 (5.8)			
Knee Valgus/Varus at Initial Contact (degrees)	CHO-E	2.5 (4.1)	2.9 (4.2)	2.1 (3.9)	0.007	0.503	0.872
	PLA	2.9 (3.0)	2.0 (3.3)	2.8 (3.7)			
Peak Hip Flexion (degrees)	CHO-E	42.0 (7.1)	41.3 (7.3)	42.1 (6.3)	0.497	0.497	0.226
	PLA	43.9 (5.5)	42.9 (4.2)	42.6 (5.3)			

SD = standard deviation; CHO-E = carbohydrate-electrolyte beverage; PLA = placebo beverage

4.2.1 Knee Flexion at Initial Contact

There was a significant effect of time on knee flexion at initial contact ($F(2, 46) = 8.179$, $p = 0.001$). There was no effect of treatment ($F(1, 23) = 0.615$, $p = 0.441$) or interaction between treatment and time ($F(2, 46) = 0.763$, $p = 0.472$). Overall as time progressed through the IHE protocol subjects landed with less knee flexion at initial contact, however there was no overall effect of beverage type or beverage type across the breaks on knee flexion angles at initial contact (Figure 5).

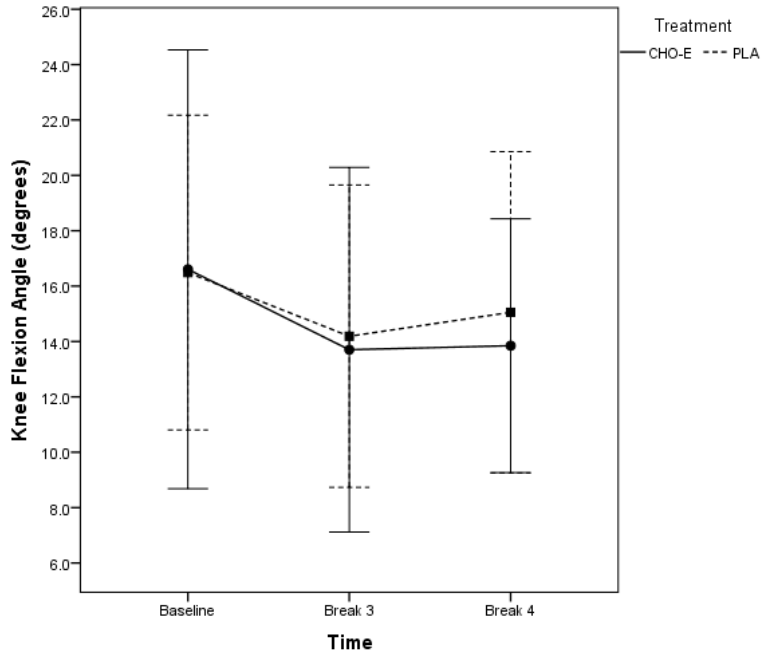


Figure 5. Mean (± 1 SD) Knee Flexion at Initial Contact

4.2.2 Valgus/Varus at Initial Contact

There was no effect of treatment ($F(1, 23) = 0.026, p = 0.872$) or time ($F(1.5, 35.7) = 0.623, p = 0.503$) on valgus/varus angle at initial contact. There was a significant interaction of treatment by time on knee valgus/varus angle at initial contact ($F(2, 46) = 5.474, p = 0.007$) with values for each treatment alternating between time points.

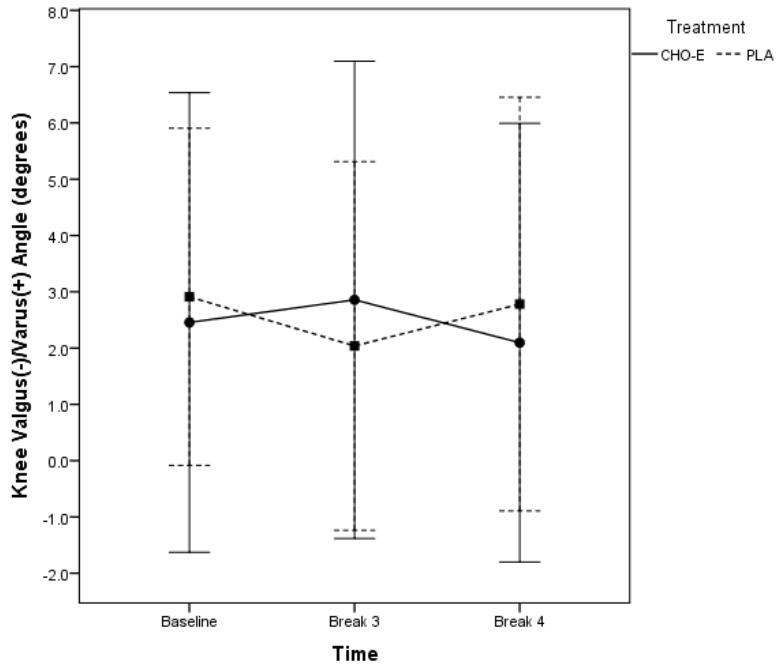


Figure 6. Mean (± 1 SD) Knee Valgus/Varus Angle at Initial Contact

4.2.3 Peak Hip Flexion

There was no effect of treatment ($F(1, 23) = 1.545, p = 0.226$), time ($F(2, 46) = 0.709, p = 0.497$), or interaction of treatment by time ($F(2, 46) = 0.799, p = 0.456$) on peak hip flexion angles.

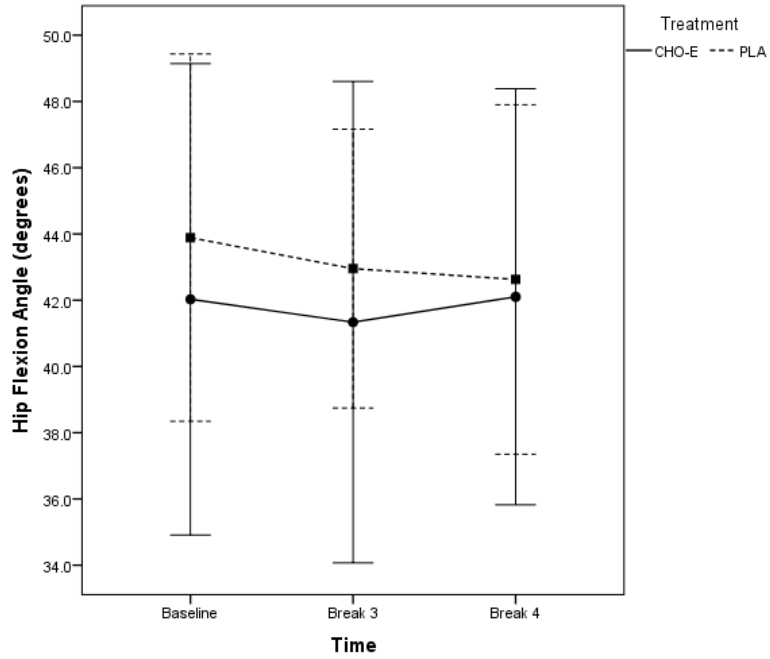


Figure 7. Mean (± 1 SD) Peak Hip Flexion

4.3 DYNAMIC POSTURAL STABILITY

Postural stability during a jump landing task was analyzed using DPSI scores at baseline, break 3, and break 4 of the IHE protocol during both treatment conditions (Table 9).

Table 9. Dynamic Postural Stability Index (DPSI) Descriptive Statistics and Two-way Repeated Measures Analysis of Variance Results

DPSI Score	Beverage	Mean (SD)			Interaction	P-Value	
		Baseline	Break 3	Break 4		Time	Treatment
DPSI	CHO-E	0.3707 (0.0337)	0.3723 (0.0347)	0.3700 (0.0374)	0.023	0.152	0.385
	PLA	0.3752 (0.0376)	0.3661 (0.0327)	0.3641 (0.0329)			
MLSI	CHO-E	0.0325 (0.0062)	0.0312 (0.0079)	0.0292 (0.0056)	0.597	0.005	0.412
	PLA	0.0318 (0.0074)	0.0295 (0.0051)	0.0291 (0.0034)			
APSI	CHO-E	0.1424 (0.0111)	0.1400 (0.0078)	0.1401 (0.0076)	0.982	0.082	0.796
	PLA	0.1420 (0.0092)	0.1398 (0.0084)	0.1399 (0.0076)			
VSI	CHO-E	0.3402 (0.0349)	0.3431 (0.0356)	0.3408 (0.0395)	0.020	0.275	0.414
	PLA	0.3454 (0.0387)	0.3367 (0.0343)	0.3346 (0.0342)			

SD = standard deviation; CHO-E = carbohydrate-electrolyte beverage; PLA = placebo beverage; DPSI = dynamic postural stability index; MLSI = medial/lateral stability index; APSI = anterior/posterior stability index; VSI = vertical stability index

4.3.1 DPSI

Results from the two way repeated measures ANOVA revealed a significant time by treatment interaction between the CHO-E and PLA treatment sessions in overall DPSI scores ($F(2, 46) = 4.091, p = 0.023$). As time progressed DPSI scores from the CHO-E group remained closer to baseline values, while the PLA group's DPSI scores decreased slightly. There was no main effect of treatment ($F(1, 23) = 0.785, p = 0.385$) or time ($F(2, 46) = 1.96, p = 0.152$) on DPSI scores.

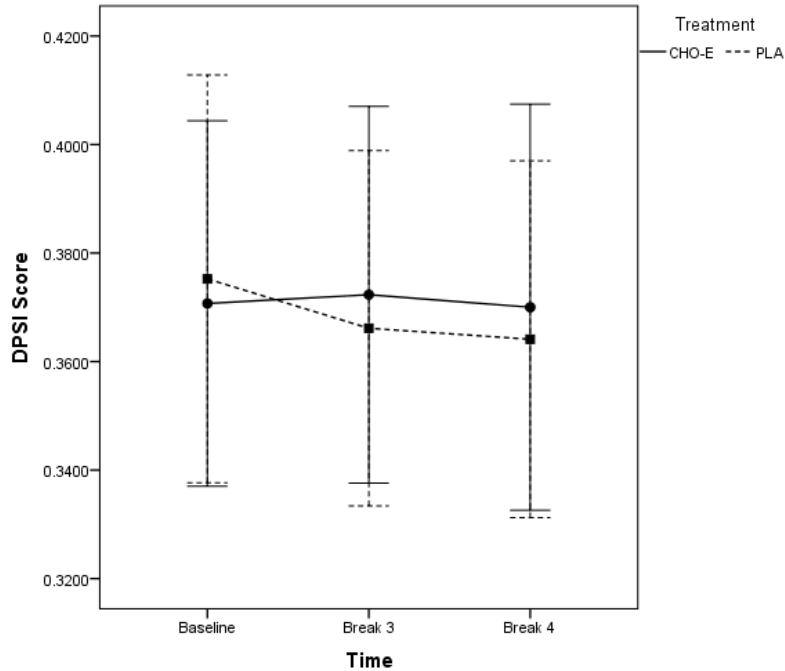


Figure 8. Mean (± 1 SD) Dynamic Postural Stability Index (DPSI) Scores

4.3.2 MLSI

There was a significant effect of time on MLSI scores ($F(2, 46) = 6.038, p = 0.005$). There was no effect of treatment ($F(1, 23) = 0.699, p = 0.412$) or interaction between treatment and time ($F(1.48, 34.03) = 0.425, p = 0.597$). As time progressed through the IHE protocol subject's MLSI scores decreased; however, there was no overall effect of beverage type or beverage type across the breaks on MLSI scores.

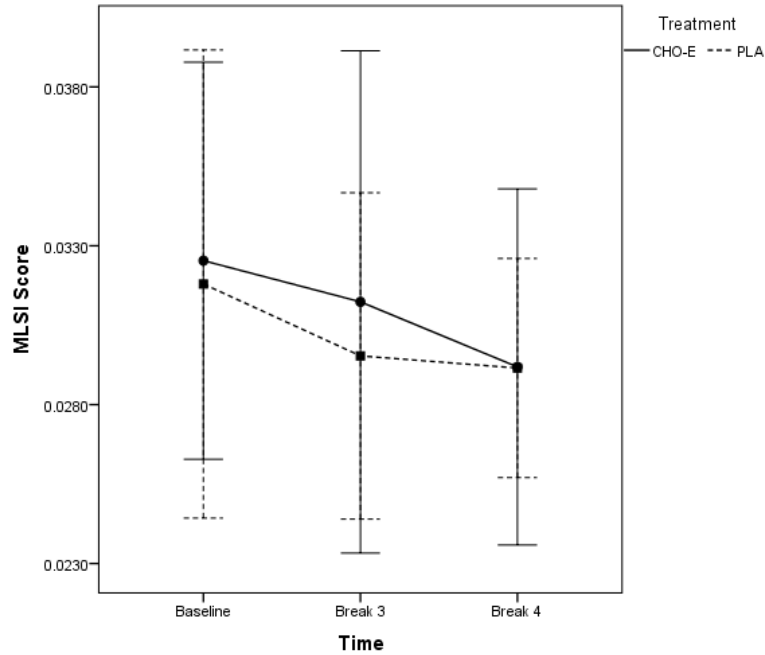


Figure 9. Mean (± 1 SD) Medial/Lateral Stability Index (MLSI) Scores

4.3.3 APSI

There was no effect of treatment ($F(1, 23) = 0.069, p = 0.796$), time ($F(2, 46) = 2.461, p = 0.082$), or interaction of treatment by time ($F(2, 46) = 0.019, p = 0.982$) on APSI scores. From baseline to break four there was no significant change in APSI scores overall or between the treatment groups.

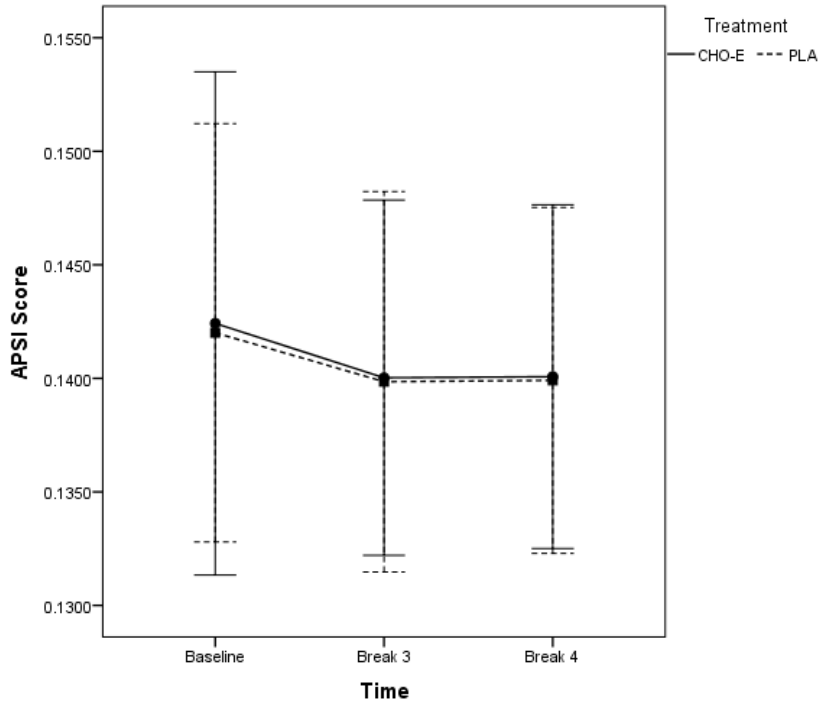


Figure 10. Mean (± 1 SD) Anterior/Posterior Stability Index (APSI) Scores

4.3.4 VSI

A significant time by treatment interaction effect existed between the CHO-E and PLA treatment sessions in VSI scores ($F(2, 46) = 4.250, p = 0.020$). Similar to the DPSI scores the VSI scores of the CHO-E group remained fairly unchanged while the PLA groups scores decreased slightly from baseline to break four. There was no main effect of treatment ($F(1, 23) = 0.691, p = 0.414$) or time ($F(2, 46) = 1.327, p = 0.275$) on DPSI scores.

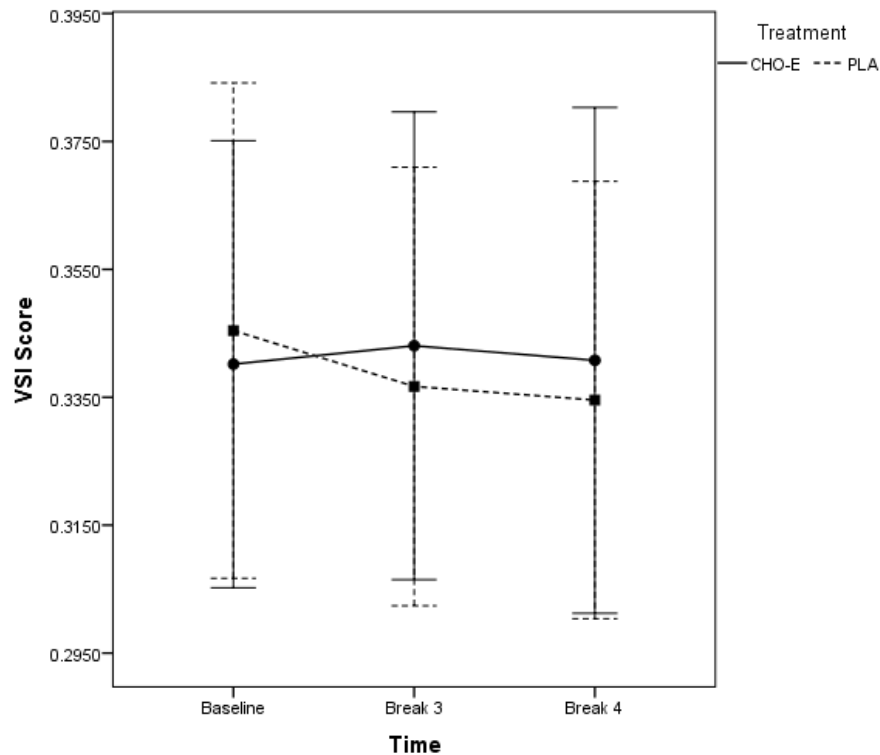


Figure 11. Mean (± 1 SD) Vertical Stability Index (VSI) Scores

4.4 MUSCLE ACTIVITY

Integrated pre-activity and re-activity EMG signals from the of the vastus lateralis (VL), vastus medialis (VM), medial hamstrings (MH), and lateral hamstrings (LH) were collected and analyzed at baseline, break three, and break four between the CHO-E and PLA treatment groups. Assessment of the integrated EMG data using the Shapiro-Wilk test revealed the data was not normally distributed ($p < 0.05$). Data transformations were unsuccessful in normalizing the EMG data. A related samples Wilcoxon Signed Rank test was used to examine median differences between times points between the CHO-E and PLA treatment groups. A summary of the EMG results is presented in Table 10.

Table 10. EMG Descriptive Statistics

Integrated EMG (%MVIC x sec)	Beverage	Baseline		Break 3		Break 4	
		Med	IQR	Med	IQR	Med	IQR
VM	CHO-E	15.053	6.762 - 21.234	9.550	4.99 - 18.382	11.270	5.007 - 17.34
Pre-activity	PLA	13.264	4.756 - 24.695	10.983	7.107 - 18.831	10.771	7.01 - 13.721
VL	CHO-E	10.016	5.155 - 17.827	11.151	5.519 - 15.457	9.382	7.193 - 13.426
Pre-activity	PLA	10.482	3.138 - 22.282	10.667	6.359 - 22.273	10.489	3.897 - 23.677
MH	CHO-E	0.041	0.013 - 0.532	0.026	0.01 - 0.507	0.025	0.009 - 0.448
Pre-activity	PLA	0.036	0.011 - 0.207	0.033	0.016 - 0.16	0.027	0.014 - 0.124
LH	CHO-E	14.875	8.717 - 19.263	10.945	6.317 - 17.991	9.972	4.652 - 17.131
Pre-activity	PLA	10.665	7.147 - 14.716	11.699	8.053 - 14.67	9.618	6.935 - 16.454
VM	CHO-E	9.352	7.53 - 13.024	9.294	6.865 - 14.449	9.351	6.38 - 14.993
Re-activity	PLA	13.880	6.601 - 20.884	8.417	6.46 - 17.286	7.791	5.829 - 14.448
VL	CHO-E	6.010	3.499 - 7.689	4.595	2.658 - 8.081	5.233	2.984 - 7.228
Re-activity	PLA	7.037	3.202 - 10.522	5.158	2.135 - 7.759	5.418	2.658 - 8.64
MH	CHO-E	0.039	0.006 - 0.281	0.054	0.004 - 0.357	0.061	0.005 - 0.31
Re-activity	PLA	0.018	0.007 - 0.105	0.019	0.008 - 0.089	0.020	0.007 - 0.112
LH	CHO-E	6.248	3.607 - 10.994	7.127	2.654 - 10.693	7.405	3.149 - 10.492
Re-activity	PLA	6.373	3.697 - 8.086	6.031	4.869 - 7.681	6.419	4.451 - 9.686

Med = median; IQR = 25th - 75th percentile; %MVIC = percent maximum voluntary isometric contraction; sec = seconds; VM = vastus medialis; VL = vastus lateralis; MH = medial hamstrings; LH = lateral hamstrings; CHO-E = carbohydrate-electrolyte beverage; PLA = placebo beverage

4.4.1 EMG Pre-Activity

There was no significant difference between baseline and break 3, baseline and break 4, or break 3 and break 4 in VM, VL, MH, or LH pre-activity between CHO-E and PLA treatment conditions.

Table 11. Comparison of Change in EMG Pre-activity Across Time Points Between Treatments

Change in Integrated EMG Pre-activity (%MVIC x sec)	Muscle	CHO-E		PLA		P-Value*
		Med	IQR	Med	IQR	
Break 3 - Baseline	VM	-2.636	-6.277 - -1.241	-2.856	-6.016 - 2.001	0.263
	VL	-0.422	-3.706 - 0.905	0.010	-2.743 - 1.286	0.249
	MH	-0.009	-0.027 - 0.001	-0.005	-0.039 - 0	0.858
	LH	-2.472	-4.165 - 1.574	-1.377	-4.995 - 1.987	0.506
Break 4 - Baseline	VM	-2.341	-4.957 - -0.658	-0.502	-7.159 - 2.23	0.168
	VL	-0.421	-3.737 - 2.687	-0.201	-3.451 - 1.474	0.408
	MH	-0.008	-0.083 - -0.001	-0.004	-0.026 - 0.001	0.223
	LH	-2.770	-5.654 - -0.382	-1.964	-7.893 - 0.588	0.570
Break 4 - Break 3	VM	0.327	-2.120 - 1.563	-0.049	-2.024 - 1.503	0.592
	VL	-0.423	-2.459 - 0.981	0.097	-1.097 - 1.944	0.527
	MH	0.000	-0.006 - 0.008	0.000	-0.008 - 0.003	0.935
	LH	-0.835	-3.240 - 0.632	-0.301	-1.451 - 1.234	0.570

*Wilcoxon Signed rank test

Med = median; IQR = 25th - 75th percentile; MVIC = maximum voluntary isometric contraction; sec = seconds; VM = vastus medialis; VL = vastus lateralis; MH = medial hamstrings; LH = lateral hamstrings; CHO-E = carbohydrate-electrolyte beverage; PLA = placebo beverage

4.4.2 EMG Re-Activity

There was no significant difference between baseline and break 3, baseline and break 4, or break 3 and break 4 in VM, VL, MH, or LH re-activity between CHO-E and PLA treatment conditions.

Table 12. Comparison of Change in EMG Re-activity Across Time Points Between Treatments

Change in Integrated EMG Re-activity (%MVIC x sec)	Muscle	CHO-E		PLA		P-Value*
		Med	IQR	Med	IQR	
Break 3 - Baseline	VM	-0.598	-2.735 - 1.355	-1.837	-3.848 - 0.858	0.445
	VL	-0.798	-2.219 - 0.192	-1.556	-2.582 - -0.347	0.961
	MH	0.000	-0.003 - 0.024	0.000	-0.006 - 0.002	0.062
	LH	-0.387	-2.933 - 1.232	0.395	-0.946 - 1.581	0.14
Break 4 - Baseline	VM	-0.022	-1.761 - 1.059	-1.635	-4.887 - 1.124	0.082
	VL	-1.134	-2.225 - 0.963	-1.381	-2.154 - -0.405	0.961
	MH	0.001	-0.001 - 0.027	0.001	0 - 0.013	0.910
	LH	0.171	-1.923 - 1.727	1.602	-0.483 - 2.051	0.306
Break 4 - Break 3	VM	0.712	-1.231 - 1.451	-0.286	-1.307 - 1.997	0.338
	VL	-0.154	-0.658 - 1.247	0.098	-0.705 - 0.881	0.783
	MH	0.001	0.000 - 0.024	0.002	-0.001 - 0.015	0.548
	LH	0.161	-0.483 - 2.211	0.587	-0.745 - 1.694	0.935

*Wilcoxon Signed rank test

Med = median; IQR = 25th - 75th percentile; MVIC = maximum voluntary isometric contraction; sec = seconds; VM = vastus medialis; VL = vastus lateralis; MH = medial hamstrings; LH = lateral hamstrings; CHO-E = carbohydrate-electrolyte beverage; PLA = placebo beverage

5.0 DISCUSSION

This is one of the first studies to examine the capability of nutrition (specifically CHO intake) on attenuating certain biomechanical and neuromuscular control alterations that occur as a result of high intensity exercise. Injury epidemiology has demonstrated that many sports injuries tend to occur in the later stages of games and practices when individuals are more likely to be fatigued.^{49, 57, 112, 187, 188} Fatigue has also been shown to alter neuromuscular and biomechanical characteristics in a manner that increases an athlete's risk of non-contact lower extremity injury.^{76, 96, 152} Previous work in our laboratory has demonstrated the ability of an IHE protocol, mimicking sports competition, to elicit similar changes as fatigue on biomechanical risk factors for non-contact injury. Additionally, sports nutrition research has demonstrated the ability to reduce the effects of fatigue and improve athletic performance through CHO feedings.^{27, 31, 133, 173, 181}

The purpose of this study was to determine whether a CHO-E beverage would have an effect on landing kinematics, balance, and muscle activation during jumping and landing tasks throughout an intermittent high intensity exercise (IHE) protocol. A total of 12 male and 12 female athletes completed the study. All subjects successfully completed all three days of testing. The first day of testing consisted of a familiarization session for the IHE protocol and a treadmill test to determine running speeds used during the IHE protocol. During the second and third days of testing participants performed four quarters of IHE followed by a run to fatigue. The second and third days of testing were separated by at least one week and followed identical protocols with the exception of beverage allocation. The administration of either the CHO-E or PLA beverage was counterbalanced and randomized with an equal number of males and females receiving the same beverage sequence (either CHO-E first or PLA first) across the second and third days of testing.

Landing kinematics, muscle activation, and dynamic postural stability were assessed immediately before the IHE protocol (baseline), after quarter three (break three), and immediately following the fourth quarter (break four) of the IHE protocol. Postural stability was measured using the DPSI score during a single-leg jump landing. Landing kinematics (knee flexion at initial contact, valgus/varus at initial contact, and peak hip flexion) and muscle activity (surface electromyography pre-activity and re-activity of VM, VL, MH, and LH) were measured during a single-leg stop-jump task. Separate two-way repeated measures ANOVA were performed to determine the effects of time, treatment, and the interaction between time and treatment on measures of landing kinematics, postural stability, and muscle activation. We hypothesized that the CHO-E beverage would maintain measures of landing kinematics, postural stability, and muscle activation patterns closer to baseline measures compared to a PLA during intermittent high intensity exercise. Results from our study did not support our hypotheses. Consumption of a CHO-E beverage had no effect on preventing changes in knee flexion at initial contact, peak hip flexion, and muscle activation patterns during landing tasks compared to a PLA throughout four quarters of IHE. A significant interaction effect between beverage and time was found for DPSI scores and knee valgus/varus at initial contact; however, these results may not be clinically significant. Descriptive variables, landing mechanics, dynamic postural stability, muscle activity, and study limitations and future research are discussed with more detail in the subsequent sections.

5.1 DESCRIPTIVE VARIABLES

5.1.1 Diet Recall

A 24-hour diet recall was collected to ensure there were no significant differences in the diet before each day of testing. The analysis of the 24-hour food recall showed no significant difference in total calorie, carbohydrate, protein, or fat intake between the CHO-E and PLA treatment sessions. Additionally, subjects were asked to fast for 10 hours prior to testing. Fasting was verified by asking the subject when they last ate before each testing session. All subjects reported being fasted for 10 hours before starting testing. The results of the 24-hour recall and subjects adhering to the 10 hour fast provide evidence of similar nutritional status prior to each testing session.

5.1.2 Intermittent High-Intensity Exercise Protocol

Time to complete each quarter, heart rate, lactate, and RPE were measured at the end of each quarter to confirm that subjects were exercising at similar intensities between treatment sessions. No significant difference was seen in time to complete each quarter, heart rate, lactate, or RPE between the CHO-E and PLA treatment sessions. Displaying no interaction effect of treatment for time to complete each quarter is consistent with results reported by Welch and Winnick.^{173, 181} Neither researcher reported increased quarter performance between CHO-E and PLA beverages in a similar high intensity exercise protocol.^{173, 181} Average heart rate and lactate values at the end of each quarter were similar between both the CHO-E and PLA testing sessions and are also consistent with values reported by Welch and Winnick,^{173, 181} as well as comparable to those seen in athletes participating in soccer, basketball, and hockey games.^{94, 124, 145} Median ratings of

perceived exertion increased from the first quarter to the fourth quarter. However, there was no significant difference in the change between quarters across the CHO-E or PLA sessions. Overall, the heart rate, RPE, quarter completion time, and lactate data demonstrate that all participants were exercising at high intensities across all four quarters. Additionally, the lack of treatment by time interaction for heart rate, lactate, and quarter completion time indicates that the physiological demands were similar for subjects during each experimental trial.

Following the IHE protocol, during both the CHO-E and PLA testing sessions, subjects completed an incremental treadmill run to fatigue to determine the impact of the beverages on exhaustive exercise. On the days when subjects received the CHO-E beverage they ran significantly longer compared to when they received the PLA beverage. The mean difference between the CHO-E and PLA sessions was 20 seconds. This is similar to the results reported by Winnick and Welsh.^{173, 181} After four quarters of a similar intermittent high intensity exercise protocol, Winnick et al.¹⁸¹ reported subject's time to fatigue with CHO lasting 58 seconds longer than with a placebo beverage. The run to fatigue in Winnick's study consisted of a 20 meter shuttle run alternating between 55% and 120% of maximal oxygen uptake pace until subjects were no longer able to keep pace.¹⁸¹ The fatiguing run in this study consisted of treadmill running with the pace increasing 1 km/h every minute until the subject could no longer continue running. Although the run to fatigue in the current study differs from that used by Winnick and colleagues¹⁸¹, both showed significant improvements in performance to exhaustion with carbohydrate supplementation.

The ability of carbohydrates to delay fatigue during exercise is thought to occur through mechanisms of both the central and peripheral nervous systems. Peripherally, CHO feedings during exercise increases exogenous CHO oxidation, helping to spare muscle glycogen stores and

provide immediate energy sources for contracting muscles in order to sustain exercise intensity.^{34,}
^{35, 84, 90} Additionally, CHO feedings may help delay central fatigue through maintaining blood glucose levels, preventing neurotransmitter imbalances, and increasing corticomotor output.^{39, 62,}
¹²¹ Although the exact mechanisms in which CHO feedings increase time to fatigue during exhaustive exercise is not known, it is likely a factor of both central and peripheral mechanisms. Similar to previous research, the current study was able to demonstrate the ability of CHO-E feedings to increase time to fatigue during exhaustive exercise. During pilot testing in our laboratory the IHE protocol caused a significant decrease in time to fatigue during the incremental treadmill run compared to the baseline incremental treadmill test and also induced changes in neuromuscular characteristics related to fatigue and non-contact lower extremity injury. Following the IHE protocol participants fatigued an average of 65 seconds sooner than when they performed the same treadmill test as part of their baseline measures collected on a previous day. Additionally, during the pilot study, participant's landing kinematics significantly changed from baseline measures to quarter four. The pilot study helped demonstrate the ability of the IHE protocol to induce some level of fatigue and hinder landing kinematics during IHE as well as performance on the treadmill run to exhaustion. During exercise and fatiguing tasks a combination of both central and peripheral mechanism is thought to affect neuromuscular control.^{16, 38} The potential effects that CHO ingestion could have on mitigating neuromuscular changes as a result of fatigue include its ability to peripherally prevent substrate depletion in contracting muscles as well as the potential to maintain or increase activation from the central nervous system to the muscle during exercise.

5.2 LANDING MECHANICS

5.2.1 Knee Flexion at Initial Contact

Decreased knee flexion angle at initial contact has been demonstrated to increase the risk of ACL injury.^{75,76} Additionally, a pilot study in our laboratory has shown decreases in knee flexion angles at initial contact during single-leg stop-jump landings while performing the same IHE protocol used in this study. We hypothesized that compared to a PLA, a CHO-E beverage would maintain knee flexion angles at initial contact closer to baseline values throughout the IHE protocol. Our findings revealed no significant effect of the CHO-E beverage on preventing changes in knee flexion angle at initial contact. Both the CHO-E and PLA groups responded similarly to the IHE protocol, landing with less knee flexion in the later stages of the protocol. Overall knee flexion angles at initial contact significantly decreased from baseline to break three ($p = 0.009$) and from baseline to break four ($p = 0.018$), with no significant difference in knee flexion angles between break three and break four. Mean overall change in knee flexion angles at initial contact from baseline to break three and baseline to break four was 2.6 degrees (95% CI: 0.6 – 4.6) and 2.1 degrees (95% CI: 0.3 – 3.9) respectively. The changes in knee flexion angles at initial contact from baseline measures to late in the IHE protocol are similar to those seen during our pilot investigation as well as results reported by Benjaminse et al.⁸ Benjaminse and colleagues also collected landing kinematics during a single leg stop jump, and reported that participants landed with about two degrees less knee flexion at initial contact following fatigue.⁸ Both the CHO-E and PLA groups experienced a decrease in knee flexion angles at initial contact in the last 2 stages of the IHE protocol. The decrease in knee flexion angle at initial contact is consistent with previous work and other studies investigating the effects of high-intensity exercise and fatigue on knee kinematics.

Neither beverage had a significant effect on preventing changes in knee flexion angles at initial contact as a result of IHE.

5.2.2 Knee Valgus/Varus at Initial Contact

Excessive knee valgus angle during weight-bearing, decelerating activities increases the forces at the knee, increasing the likelihood of injury.^{68, 69, 75, 160} The effect of IHE or fatigue on knee valgus angles at initial contact has demonstrated mixed results, with either an increase or no change in knee valgus at initial contact following fatigue.^{8, 32, 137, 153} During a pilot study of the IHE protocol employed in the current study, subjects displayed knee angles moving towards the valgus direction as time progressed. We hypothesized that compared to a PLA, a CHO-E beverage would maintain knee valgus/varus angles at initial contact closer to baseline values throughout the IHE protocol. In the current study, a statistically significant interaction between treatment and time occurred in knee valgus/varus angle at initial contact, with no significant overall effect of time or treatment. Mean valgus/varus knee angles for our study ranged between 2.0 ± 3.3 and 2.9 ± 4.2 degrees of varus across baseline, break three, and break four in both the CHO-E and PLA groups. Benjaminse and colleagues also experienced similar results with mean initial contact knee angles being in the varus direction in both pre fatigue (1.36 ± 3.0 degrees) and post fatigue (1.0 ± 2.8 degrees) measures.⁸ In both the current study and in Benjaminse's study participants performed a single-leg stop-jump for kinematic analysis. The lack of mean knee angles at initial contact being in valgus alignment in both studies may be due to the inherent nature of the knee to take on more of a varus alignment in single leg stance.³ Although a significant interaction was seen between the CHO-E and PLA groups across the three time points it is difficult to determine if the beverages truly had an effect on knee valgus/varus angles between time points. While the interaction effect

between treatment and time suggests that the beverage effected valgus/varus knee angles between quarters, the mean knee varus angles across all three times points were within one degree of each other, which falls within the error for capturing knee varus/valgus angles.⁵³ Therefore, although the results show statistical significance, it is hard to determine if a clinically significant interaction of treatment by time exists.

5.2.3 Peak Hip Flexion

Previous research has shown that landing in a more upright position (less hip and knee flexion) can increase the strain placed on the ACL, and that fatigue can induce these altered landing mechanics.^{12, 130, 137} Additionally, we have demonstrated in a pilot study that IHE can produce similar decreases in peak hip flexion during landing as fatigue. We hypothesized that compared to a PLA, a CHO-E beverage would maintain peak hip flexion angles closer to baseline values throughout the IHE protocol. In the current study, no overall effect for time, treatment, or interaction of treatment by time was seen for peak hip flexion angles. Our data suggests that there were no significant changes in peak hip flexion angle as a result of beverage consumption or IHE. Mean peak hip flexion angles for both CHO-E and PLA groups ranged between 41.3 ± 7.3 and 43.9 ± 5.5 degrees across all three measurements. These results are similar to the pre-fatigued hip angles reported by Orishimo and Kremenec, who measured landing kinematics during a single leg hop before and after fatiguing exercise.¹²⁸ Although previous research examining kinematics of landing during or following fatigue have shown a decrease in hip flexion angles following fatigue, these studies vary slightly from the present study in either landing tasks and/or exercising mechanisms.^{5, 32, 128, 137} Both Augustsson et al.⁵ and Orishimo and Kremenec¹²⁸ measured hip flexion before and after fatigue during a single-leg hop for distance. While a single-leg hop for

distance is similar to a single leg stop-jump, the landing phase only requires individuals to decelerate from the initial jump, compared to a stop-jump which requires the subject to rapidly decelerate from the first jump and then accelerate into a second vertical jump. Additionally, both teams of researchers used a more localized fatigue protocol consisting of either weighted leg extensions or box step-ups, whereas in the present study a more functional IHE protocol was used.⁵

¹²⁸ On the other hand, Cortes et al.³² and Quammen et al.¹³⁷ both demonstrated greater peak hip flexion angles following more generalized and functional fatigue protocols. However, Quammen et al.¹³⁷ measured landing kinematics during a double-leg stop jump and Cortes et al.³² measured them during a single-leg sidestep-cutting task. Although previous research has demonstrated decreased hip flexion when landing during a variety of tasks, following various fatigue and exercise protocols, in the present study there was no significant effect of time on peak hip flexion. This may be a result of the single-leg stop-jump or of the IHE protocol's ability to alter hip kinematics compared to other fatigue protocols. Even though, there was no overall effect of time on peak hip flexion angle, there was a slight trend in the PLA group with hip flexion angles decreasing from baseline, to break three, to break four. The trend towards decreased peak hip flexion angles in the PLA group resembled results seen during our pilot study of the IHE protocol. However, this trend was not noted in the CHO-E treatment session. Peak hip flexion angles in the CHO-E group decreased slightly from baseline to break three, but returned close to baseline values at break four. Despite these trends, there was not a significant interaction between the treatment and time, suggesting no benefit of the CHO-E beverage on preventing changes in peak hip flexion during IHE.

5.3 DYNAMIC POSTURAL STABILITY

Jump landing tasks and the ability for an individual to stabilize after landing are commonly assessed in research as an aspect of lower extremity motor and postural control.^{148, 149, 151, 159, 174, 177} Decreased postural stability has been identified as a risk factor for lower extremity (ankle and knee) injury in athletic populations.^{2, 104, 150, 161, 172, 178} We hypothesized that compared to a PLA, a CHO-E beverage would maintain DPSI scores closer to baseline values throughout the IHE protocol. Our study revealed no main effect of time or treatment on DPSI scores. We did discover a significant interaction of treatment by time on DPSI scores with the PLA group having lower scores as time progressed through the IHE protocol, while the CHO-E group remained fairly unchanged. These results were unexpected and confirmed through an additional analysis which tested for the presence of a learning effect.

This is one of the first studies to examine the effect of IHE on DPSI scores. Mean DPSI scores across time points and treatments are similar to those reported in the literature in healthy non-exercising males and females.^{156, 158} Additionally, there did not seem to be a significant overall effect of the IHE protocol on DPSI scores and there was a trend for improved scores as time progressed. These results are different than those seen in our pilot study of the IHE protocol, where subjects experienced a trend towards higher DPSI scores later in the exercise protocol. Our hypothesis that CHO-E beverages would maintain DPSI values closer to baseline was partially accepted. The CHO-E group seemingly maintained DPSI scores across all three trials however, the PLA group had improved DPSI scores later in the IHE protocol. It is not clear why the PLA group had better DPSI scores compared to the CHO-E group. Although the PLA group's DPSI scores improved slightly, the change in scores from baseline to break four were small and may not demonstrate a minimal clinically important difference. Additionally, the PLA group may not have

performed the jump landing with as much force as the CHO-E group, and therefore, was able to stabilize quicker. However, it is important to note that when DPSI scores were examined across six time points (baseline, break one, break two, break three, break four, and post fatiguing run) there was no significant interaction effect between treatment and time, but there was a significant overall effect of time. When examined across all six time points the combined DPSI scores of the CHO-E and PLA groups appeared to improve slightly over time. This improvement in overall DPSI scores could again be due to the fact that both groups may have jumped with less force as they progressed through the IHE protocol or they became more familiar with the task.

Very limited research has examined the effect of carbohydrates on measures of balance during exercise or fatigue. One recent study investigated the effect of a 20% maltodextrin solution on the number of balance beam falls in gymnasts following a fatiguing exercise circuit.⁷ The authors reported significantly less balance beam falls after fatiguing exercise in the group consuming the CHO beverage compared to the water only group.⁷ However the authors did not collect any direct measures of balance, strength, or neuromuscular control. Therefore, it is difficult to determine the exact ergogenic effect that CHO ingestion may have had specifically on balance and postural stability to reduce the number of falls.

5.4 MUSCLE ACTIVITY

Feed-forward and feedback activation of the hamstring and quadriceps muscles are important for preparatory and reactive muscle stiffness and contribute to knee functional joint stability.^{80, 116, 165,}

¹⁸⁵ We hypothesized that hamstrings and quadriceps muscle pre-activity and re-activity during a stop-jump would remain closer to baseline values while consuming a CHO-E beverage compared

to a PLA throughout an IHE protocol. Results from this study showed no significant difference between treatments in the IEMG pre-activity or re-activity of the VM, VL, MH, and LH during a single leg stop-jump. Previous studies have reported on the effects of fatigue on feed-forward (pre-activity) and feedback (re-activity) muscle activation during landing tasks.^{82, 128} However, to the author's knowledge, no other published research has examined IEMG pre-activity and re-activity of the VL, VM, MH, and LH during IHE using the same jumping task and timeframes as those employed during this study. Therefore, the following comparisons can only be made to the pattern of EMG activity instead of direct comparison of values. James et al.⁸² measured root mean squared EMG pre-activity (60ms pre-contact) and re-activity (31-60 and 61-90ms post-contact) during a drop landing following two different fatigue protocols. They measured EMG pre-activity and re-activity of the VM, VL, and biceps femoris and found no significant difference between fatigue protocols or between non-fatigued and fatigued conditions.⁸² The authors did find a slight increase in VM and VL re-activity 61-90ms post-contact in fatigued vs non-fatigued conditions, indicating a possible increase in stretch reflex muscle stiffness.⁸² However, due to other changes seen in ground reaction forces and landing mechanics the authors still reported an overall reduction in lower extremity system stiffness following fatigue.⁸² Orishimo and colleagues¹²⁸ investigated the effects of fatigue on EMG activity during a single-leg hop and reported an increase in EMG pre-activity 100ms prior to landing in the VM following fatigue. The authors did not find any significant change in VL or biceps femoris EMG pre-activity following fatigue.¹²⁸ In the present study there was no significant difference in the change in EMG activity from baseline to break three or four between the CHO-E and PLA groups. This indicates that both groups responded similarly to the IHE protocol regardless of beverage type. There appears to be a slight trend towards decreased IEMG pre-activity and re-activity values from baseline to break four in both

groups. In our pilot investigation of the IHE protocol we discovered a similar trend towards decreased IEMG values in the third and fourth quarters. However, in the pilot study IEMG pre-activity of the VM was the only measure that was significantly lower in the later stages of the IHE protocol. Previous research has reported decreases in IEMG due to exercise and fatigue and has suggested it can represent a decrease in motor unit recruitment, muscle force, and/or stiffness.^{4, 92} In the present study we failed to show any difference in IEMG pre or re-activity between the two treatment sessions.

Although, Gant et al.⁶² demonstrated that the ingestion of CHO can immediately increase corticomotor excitability and motor evoked potentials, there was no significant difference in EMG activity between the CHO-E to PLA groups in our study. Gant reported a 30% increase in EMG activity with CHO ingestion compared to a placebo in fatigued forearm muscles during maximum contractions.⁶² The differences between our findings and Gant's may be a result of the submaximal effort of a stop-jump maneuver compared to a maximal voluntary muscle contraction used by Gant and colleagues.⁶² Additionally, they employed a localized fatigue protocol to a small muscle group (forearms), compared to the generalized and systems based IHE protocol used in our study.⁶² It is possible that the effect of IHE on both central and peripheral mechanisms of fatigue might outweigh any potential increase in corticomotor excitability and motor evoked potentials elicited by CHO ingestion.

5.5 LIMITATIONS AND FUTURE RESEARCH

This study has a few potential limitations. Although the IHE protocol caused similar physiological changes in heart rate and lactate as those seen during intermittent high intensity team sports, it did

not completely simulate all movement patterns typically performed in these sports. The IHE protocol involved treadmill running at varying intensities, which is able to produce similar heart rate and lactate responses as those seen in team sports. However, the biomechanics of treadmill running differ slightly than field based running biomechanics.^{119, 143} Additionally, team based intermittent high intensity sports usually contain frequent running and cutting maneuvers. While the IHE protocol in this study had tasks that required cutting or change in direction, there were no running and cutting tasks. The kinematic changes that occurred as a result of this study may only be specific to the laboratory based exercise protocol and may not be representative of kinematic changes seen during a practice or competition. Future research studies should consider a field based IHE protocol in order to increase the generalizability to field based team sports.

Furthermore, the landing kinematics in the present study were analyzed during a single-leg stop-jump. While this type of maneuver closely mimics movements in which non-contact knee injuries typically occur,^{8, 13, 127} the planned nature of the task may not represent unanticipated landing and cutting movements that often occur during intermittent high intensity sports. Previous research has reported increased changes in landing kinematics during unanticipated landings compared to anticipated landings, possibly due to an increased demand for both central and peripheral responses.¹⁴ Future research should investigate a similar study design utilizing unanticipated landing tasks to determine if CHO feedings might have an effect on landing mechanics during a more challenging task requiring decision making.

Another limitation of the present study is that there were no direct measures of endogenous CHO levels before, during, or after the exercise protocol. Therefore, we cannot definitively say if there were any endogenous changes that occurred due to the consumption of the CHO-E versus the PLA beverage. Measurements of blood glucose and/or muscle glycogen levels in future

research might help to highlight any differences that may exist between the CHO-E and PLA group during the exercise protocol.

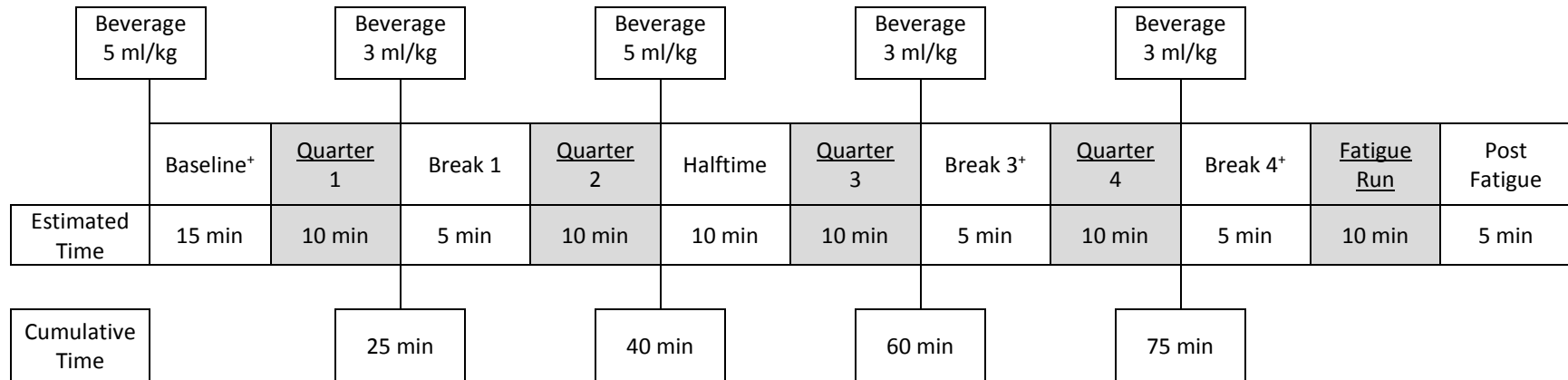
5.6 CONCLUSION

The purpose of this study was to evaluate the effects of a CHO-E beverage compared to a PLA on certain biomechanical and neuromuscular risk factors for injury during jumping and landing tasks throughout an IHE protocol. It was hypothesized that a CHO-E beverage would maintain measures of landing kinematics, postural stability, and muscle activation patterns closer to baseline values compared to a PLA throughout an IHE protocol. The IHE protocol was designed to mimic sports competition and, during pilot testing in our laboratory, it also demonstrated the ability to elicit similar changes as fatigue on biomechanical risk factors for non-contact injury. No significant interaction was found between beverage type (CHO-E or PLA) and time (baseline, break three, and break four) for knee flexion angles at initial contact or peak hip flexion angles during a single-leg stop-jump. Both CHO-E and PLA groups landed with less knee flexion at initial contact as time progressed through the IHE protocol and neither group had a significant change in peak hip flexion. During the stop jump task valgus/varus angles at initial contact demonstrated a statistically significant interaction between beverage type and time; however, the pattern of change between treatment and time was small and did not appear to be clinically meaningful. Additionally, no significant difference was seen between beverage type in EMG pre-activity or re-activity of the quadriceps and hamstring muscles during the single-leg stop-jumps. These results indicate no difference in the ability of CHO-E feedings to effect neuromuscular control mechanisms of muscle activation immediately prior to and following single-leg stop-jump landings during IHE. The

hypothesis that a CHO-E beverage would maintain DPSI values closer to baseline compared to a PLA was partially supported. However, while the DPSI scores at breaks three and four of the CHO-E group remained similar to baseline values, the DPSI scores for the PLA group improved slightly. The slight improvement in DPIS scores in the PLA group was an unexpected finding and may be a result of individuals in the PLA group jumping with less force during the jump landing task. The present study failed to show any significant benefit of consuming a CHO-E beverage in delaying or preventing changes in landing mechanics, muscle activation patterns, or postural stability measures during intermittent high-intensity exercise. Although the CHO-E beverage significantly delayed time to fatigue during an incremental treadmill run, it showed no effect on delaying neuromuscular changes associated with fatigue. It is possible that the mechanisms in which CHO delays fatigue during exhaustive exercise is different than the mechanisms involved in neuromuscular changes related to fatigue. Another explanation may be that the level of fatigue in which CHO consumption is beneficial is different than the threshold of fatigue in which certain neuromuscular changes begin to occur. This is one of the first studies to investigate the effect of nutrition on neuromuscular characteristics and risk factors related to non-contact injuries. While the current study did not demonstrate any benefit of CHO-E feedings on preventing risk factors related to injury, the results from this study should help provide the foundation for future research in nutrition fueling strategies for injury prevention.

APPENDIX A

INTERMITTENT HIGH-INTENSITY EXERCISE FEEDING PROTOCOL



⁺ Time-point for analysis of dependent variables

Time Point	Estimated Duration into IHE protocol	Beverage Volume*	Carbohydrate Amount* (CHO-E beverage)
Baseline	0 min	350 ml (11.8 fl oz)	21 g
Break 1	25 min	210 ml (7.1 fl oz)	12.6 g
Halftime	40 min	350 ml (11.8 fl oz)	21 g
Break 3	60 min	210 ml (7.1 fl oz)	12.6 g
Break 4	75 min	210 ml (7.1 fl oz)	12.6 g
Total	95 min	1,330 ml (44.9 fl oz)	79.8 g

* Assuming 70 kg athlete

BIBLIOGRAPHY

1. Abbiss CR, Laursen PB. Models to explain fatigue during prolonged endurance cycling. *Sports Medicine*. 2005;35(10):865-898.
2. Alonso AC, Greve JMDA, Camanho GL. Evaluating the center of gravity of dislocations in soccer players with and without reconstruction of the anterior cruciate ligament using a balance platform. *Clinics*. 2009;64(3):163-170.
3. Andriacchi TP. Dynamics of knee malalignment. *The Orthopedic clinics of North America*. 1994;25(3):395-403.
4. Arendt-Nielsen L, Mills KR. Muscle fibre conduction velocity, mean power frequency, mean EMG voltage and force during submaximal fatiguing contractions of human quadriceps. *European journal of applied physiology and occupational physiology*. 1988;58(1-2):20-25.
5. Augustsson J, Thomeé R, Linden C, Folkesson M, Tranberg R, Karlsson J. Single leg hop testing following fatiguing exercise: reliability and biomechanical analysis. *Scandinavian Journal of Medicine & Science in Sports*. 2006;16(2):111-120.
6. Bahr R, Holme I. Risk factors for sports injuries — a methodological approach. *British Journal of Sports Medicine*. 2003;37(5):384-392.
7. Batatinha HAP, da Costa CE, de França E, et al. Carbohydrate use and reduction in number of balance beam falls: implications for mental and physical fatigue. *Journal of the International Society of Sports Nutrition*. 2013;10(1):32.

8. Benjaminse A, Habu A, Sell TC, et al. Fatigue alters lower extremity kinematics during a single-leg stop-jump task. *Knee Surgery, Sports Traumatology, Arthroscopy*. 2008;16(4):400-407.
9. Benoit DL, Ramsey DK, Lamontagne M, Xu L, Wretenberg P, Renström P. Effect of skin movement artifact on knee kinematics during gait and cutting motions measured in vivo. *Gait & posture*. 2006;24(2):152-164.
10. Bergström J, Hermansen L, Hultman E, Saltin B. Diet, muscle glycogen and physical performance. *Acta Physiologica Scandinavica*. 1967;71(2-3):140-150.
11. Bigland-Ritchie B, Dawson N, Johansson R, Lippold O. Reflex origin for the slowing of motoneurone firing rates in fatigue of human voluntary contractions. *The Journal of physiology*. 1986;379(1):451-459.
12. Blackburn JT, Padua DA. Sagittal-plane trunk position, landing forces, and quadriceps electromyographic activity. *Journal of Athletic Training*. 2009;44(2):174.
13. Boden BP, Dean GS, Feagin J, Garrett W. Mechanisms of anterior cruciate ligament injury. *Orthopedics*. 2000;23(6):573-578.
14. 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. *Clinical biomechanics*. 2008;23(1):81-92.
15. Bosch AN, Dennis SC, Noakes TD. Influence of carbohydrate loading on fuel substrate turnover and oxidation during prolonged exercise. *Journal of applied physiology*. 1993;74(4):1921-1927.
16. Boyas S, Guével A. Neuromuscular fatigue in healthy muscle: Underlying factors and adaptation mechanisms. *Annals of physical and rehabilitation medicine*. 2011;54(2):88-108.

17. Brazen DM, Todd MK, Ambegaonkar JP, Wunderlich R, Peterson C. The effect of fatigue on landing biomechanics in single-leg drop landings. *Clinical Journal of Sport Medicine*. 2010;20(4):286.
18. Burke ER, Cerny F, Costill D, Fink W. Characteristics of skeletal muscle in competitive cyclists. *Medicine and science in sports*. 1977;9(2):109.
19. Burke LaDV. *Clinical Sports Nutrition*. 4th ed. Australia: Elizabeth Walton; 2010.
20. Burke LM, Kiens B, Ivy JL. Carbohydrates and fat for training and recovery. *Journal of Sports Sciences*. 2004;22(1):15-30.
21. Cairns SP, Knicker AJ, Thompson MW, Sjøgaard G. Evaluation of models used to study neuromuscular fatigue. *Exercise and sport sciences reviews*. 2005;33(1):9-16.
22. Casa DJ, Armstrong LE, Hillman SK, et al. National Athletic Trainers' Association position statement: fluid replacement for athletes. *Journal of Athletic Training*. 2000;35(2):212.
23. Chambers E, Bridge M, Jones D. Carbohydrate sensing in the human mouth: effects on exercise performance and brain activity. *The Journal of physiology*. 2009;587(8):1779-1794.
24. Chappell J, Herman D, Knight B, Kirkendall D, Garrett W, Yu B. Effect of fatigue on knee kinetics and kinematics in stop-jump tasks. *The American journal of sports medicine*. 2005;33(7):1022.
25. Chimera NJ, Swanik KA, Swanik CB, Straub SJ. Effects of plyometric training on muscle-activation strategies and performance in female athletes. *Journal of Athletic Training*. 2004;39(1):24.
26. Coggan AR, Coyle EF. Reversal of fatigue during prolonged exercise by carbohydrate infusion or ingestion. *Journal of applied physiology*. 1987;63(6):2388-2395.
27. Coggan AR, Coyle EF. 1 Carbohydrate Ingestion During Prolonged Exercise: Effects on Metabolism and Performance. *Exercise and sport sciences reviews*. 1991;19(1):1.

28. Conley MS, Stone MH. Carbohydrate ingestion/supplementation for resistance exercise and training. *Sports Medicine*. 1996;21(1):7-17.
29. Conn J, Annest JL, Gilchrist J. Sports and recreation related injury episodes in the US population, 1997–99. *Injury Prevention*. 2003;9(2):117-123.
30. Control NCfIPa. CDC Injury Research Agenda. In: CDC, ed. Atlanta, GA; 2002.
31. Coombes JS, Hamilton KL. The effectiveness of commercially available sports drinks. *Sports Medicine*. 2000;29(3):181-209.
32. Cortes N, Greska E, Kollock R, Ambegaonkar J, Onate JA. Changes in Lower Extremity Biomechanics Due to a Short-Term Fatigue Protocol. *Journal of Athletic Training*. 2013;48(3):306-313.
33. Costill D, Bennett A, Branam G, Eddy D. Glucose ingestion at rest and during prolonged exercise. *Journal of applied physiology*. 1973;34(6):764-769.
34. Costill DL, Bowers R, Branam G, Sparks K. Muscle glycogen utilization during prolonged exercise on successive days. *Journal of applied physiology*. 1971;31(6):834-838.
35. Costill DL, Hargreaves M. Carbohydrate nutrition and fatigue. *Sports Medicine*. 1992;13(2):86-92.
36. Coyle EF, Coggan AR, Hemmert M, Ivy JL. Muscle glycogen utilization during prolonged strenuous exercise when fed carbohydrate. *Journal of applied physiology*. 1986;61(1):165-172.
37. Davis J, Bailey S, Jackson D, Strasner A, Morehouse S. Effects of A Serotonin (5-HT) Agonist During Prolonged Exercise to Fatigue in Humans. *Medicine & Science in Sports & Exercise*. 1993;25(5):S78.
38. Davis JM. Central and peripheral factors in fatigue. *Journal of Sports Sciences*. 1995;13(S1):S49-S53.

39. Davis JM, Alderson NL, Welsh RS. Serotonin and central nervous system fatigue: nutritional considerations. *American Journal of Clinical Nutrition*. 2000;72(2):573S.
40. Davis JM, Bailey SP. Possible mechanisms of central nervous system fatigue during exercise. *Medicine and Science in Sports and Exercise*. 1997;29(1):45-57.
41. Davis JM, Bailey SP, Woods JA, Galiano FJ, Hamilton MT, Bartoli WP. Effects of carbohydrate feedings on plasma free tryptophan and branched-chain amino acids during prolonged cycling. *European journal of applied physiology and occupational physiology*. 1992;65(6):513-519.
42. Davis RB, Ounpuu S, Tyburski D, Gage JR. A gait analysis data collection and reduction technique. *Human Movement Science*. 1991;10(5):575-587.
43. De Luca CJ. The use of surface electromyography in biomechanics. *Journal of applied biomechanics*. 1997;13:135-163.
44. Dennis S, Noakes T, Hawley J. Nutritional strategies to minimize fatigue during prolonged exercise: fluid, electrolyte and energy replacement. *Journal of Sports Sciences*. 1997;15(3):305-313.
45. DERRICK T, DEREU D, MCLEAN S. Impacts and kinematic adjustments during an exhaustive run. *Medicine & Science in Sports & Exercise*. 2002;34(6):998.
46. Dietz V, Noth J, Schmidtbleicher D. Interaction between pre-activity and stretch reflex in human triceps brachii during landing from forward falls. *The Journal of physiology*. 1981;311(1):113-125.
47. Duncan AD, McDonagh MJ. Stretch reflex distinguished from pre-programmed muscle activations following landing impacts in man. *The Journal of physiology*. 2000;526(2):457-468.
48. Dunford M. *Sports nutrition: A practice manual for professionals*: American Dietetic Association; 2006.

49. Ekstrand J, Häggglund M, Waldén M. Injury incidence and injury patterns in professional football: the UEFA injury study. *British Journal of Sports Medicine*. 2011;45(7):553-558.
50. Ferber R, McClay Davis I, Williams D, Laughton C. A comparison of within-and between-day reliability of discrete 3D lower extremity variables in runners. *Journal of Orthopaedic Research*. 2002;20(6):1139-1145.
51. Finsterbush A, Frankl U, Matan Y, Mann G. Secondary damage to the knee after isolated injury of the anterior cruciate ligament. *The American journal of sports medicine*. 1990;18(5):475-479.
52. Fitts R. Cellular mechanisms of muscle fatigue. *Physiological Reviews*. 1994;74(1):49-94.
53. Ford KR, Myer GD, Hewett TE. Reliability of landing 3D motion analysis: implications for longitudinal analyses. *Medicine and Science in Sports and Exercise*. 2007;39(11):2021.
54. Fousekis K, Tsepis E, Poulmedis P, Athanasopoulos S, Vagenas G. Intrinsic risk factors of non-contact quadriceps and hamstring strains in soccer: a prospective study of 100 professional players. *British Journal of Sports Medicine*. 2011;45(9):709-714.
55. Fousekis K, Tsepis E, Vagenas G. Intrinsic Risk Factors of Noncontact Ankle Sprains in Soccer: A Prospective Study on 100 Professional Players. *The American journal of sports medicine*. 2012;40(8):1842-1850.
56. Fowles J, Green H, Tupling R, O'brien S, Roy B. Human neuromuscular fatigue is associated with altered Na⁺-K⁺-ATPase activity following isometric exercise. *Journal of applied physiology*. 2002;92(4):1585-1593.
57. Gabbett TJ. Incidence, site, and nature of injuries in amateur rugby league over three consecutive seasons. *British Journal of Sports Medicine*. 2000;34(2):98-103.
58. Gandevia S. Neural control in human muscle fatigue: changes in muscle afferents, moto neurones and moto cortical drive. *Acta Physiologica Scandinavica*. 1998;162(3):275-283.

59. Gandevia S. Spinal and supraspinal factors in human muscle fatigue. *Physiological Reviews*. 2001;81(4):1725-1789.
60. Gandevia S, Allen G, McKenzie D. Central fatigue. *Fatigue: Neural and Muscular Mechanisms*. 1995;384:281.
61. Gandevia S, Allen GM, Butler JE, Taylor JL. Supraspinal factors in human muscle fatigue: evidence for suboptimal output from the motor cortex. *The Journal of physiology*. 1996;490(Pt 2):529-536.
62. Gant N, Stinear CM, Byblow WD. Carbohydrate in the mouth immediately facilitates motor output. *Brain Research*. 2010;1350:151-158.
63. Ghez C, Hening W, Gordon J. Organization of voluntary movement. *Current opinion in neurobiology*. 1991;1(4):664-671.
64. Goldie P, Bach T, Evans O. Force platform measures for evaluating postural control: reliability and validity. *Archives of physical medicine and rehabilitation*. 1989;70(7):510-517.
65. Gotsch K, Annet J, Holmgren P, Gilchrist J. Nonfatal sports-and recreation-related injuries treated in emergency departments-United States, July 2000-June 2001. *Morbidity and Mortality Weekly Report*. 2002;51(33):736-740.
66. Gottlob CA, Baker Jr CL, Pellissier JM, Colvin L. Cost effectiveness of anterior cruciate ligament reconstruction in young adults. *Clinical orthopaedics and related research*. 1999;367:272-282.
67. Green H. Mechanisms of muscle fatigue in intense exercise. *Journal of Sports Sciences*. 1997;15(3):247-256.
68. Griffin L, Agel J, Albohm M, et al. Noncontact anterior cruciate ligament injuries: risk factors and prevention strategies. *Journal of the American Academy of Orthopaedic Surgeons*. 2000;8(3):141.

69. Griffin L, Albohm M, Arendt E, et al. Understanding and preventing noncontact anterior cruciate ligament injuries. *The American journal of sports medicine*. 2006;34(9):1512.
70. Grillner S. The role of muscle stiffness in meeting the changing postural and locomotor requirements for force development by the ankle extensors. *Acta Physiologica Scandinavica*. 1972;86(1):92-108.
71. Hawley J, Myburgh K, Noakes T, Dennis S. Training techniques to improve fatigue resistance and enhance endurance performance. *Journal of Sports Sciences*. 1997;15(3):325-333.
72. Hellsten Y, Apple FS, Sjödén B. Effect of sprint cycle training on activities of antioxidant enzymes in human skeletal muscle. *Journal of applied physiology*. 1996;81(4):1484-1487.
73. Hergenroeder AC. Prevention of sports injuries. *Pediatrics*. 1998;101(6):1057-1063.
74. Hermens HJ, Freriks B, Merletti R, et al. *European recommendations for surface electromyography*: Roessingh Research and Development The Netherlands; 1999.
75. Hewett TE, Ford KR, Hoogenboom BJ, Myer GD. Understanding and preventing ACL injuries: current biomechanical and epidemiologic considerations-update 2010. *North American journal of sports physical therapy: NAJSPT*. 2010;5(4):234.
76. Hewett TE, Myer GD, Ford KR, et al. Biomechanical measures of neuromuscular control and valgus loading of the knee predict anterior cruciate ligament injury risk in female athletes. *The American journal of sports medicine*. 2005;33(4):492.
77. Hochachka P, McClelland G. Cellular metabolic homeostasis during large-scale change in ATP turnover rates in muscles. *Journal of experimental biology*. 1997;200(2):381-386.
78. Hogervorst T, Brand RA. Current Concepts Review-Mechanoreceptors in Joint Function. *The Journal of Bone & Joint Surgery*. 1998;80(9):1365-1378.

79. Hootman JM, Dick R, Agel J. Epidemiology of collegiate injuries for 15 sports: summary and recommendations for injury prevention initiatives. *Journal of Athletic Training*. 2007;42(2):311.
80. Horita T, Komi P, Nicol C, Kyröläinen H. Interaction between pre-landing activities and stiffness regulation of the knee joint musculoskeletal system in the drop jump: implications to performance. *European Journal of Applied Physiology*. 2002;88(1-2):76-84.
81. Irvine G, Glasgow M. The natural history of the meniscus in anterior cruciate insufficiency. Arthroscopic analysis. *Journal of Bone & Joint Surgery, British Volume*. 1992;74(3):403-405.
82. James CR, Scheuermann BW, Smith MP. Effects of two neuromuscular fatigue protocols on landing performance. *Journal of electromyography and Kinesiology*. 2010;20(4):667-675.
83. Jami L. Golgi tendon organs in mammalian skeletal muscle: functional properties and central actions. *Physiological Reviews*. 1992;72(3):623-666.
84. Jeukendrup AE. Carbohydrate feeding during exercise. *European Journal of Sport Science*. 2008;8(2):77-86.
85. Johansson H, Sjölander P, Sojka P. A sensory role for the cruciate ligaments. *Clinical orthopaedics and related research*. 1991;268:161-178.
86. Johansson R, Magnusson M. Human postural dynamics. *Critical reviews in biomedical engineering*. 1991;18(6):413.
87. Johnston 3rd R, Howard ME, Cawley PW, Losse GM. Effect of lower extremity muscular fatigue on motor control performance. *Medicine and Science in Sports and Exercise*. 1998;30(12):1703-1707.
88. Junge A, Langevoort G, Pipe A, et al. Injuries in team sport tournaments during the 2004 Olympic Games. *The American journal of sports medicine*. 2006;34(4):565-576.

89. Kadaba M, Ramakrishnan H, Wootten M, Gainey J, Gorton G, Cochran G. Repeatability of kinematic, kinetic, and electromyographic data in normal adult gait. *Journal of Orthopaedic Research*. 1989;7(6):849-860.
90. Karelis AD, Smith JEW, Passe DH, Peronnet F. Carbohydrate Administration and Exercise Performance: What Are the Potential Mechanisms Involved? *Sports Medicine*. 2010;40(9):747-763.
91. Kaufman MP, Rybicki KJ, Waldrop TG, Ordway GA. Effect of ischemia on responses of group III and IV afferents to contraction. *Journal of applied physiology*. 1984;57(3):644-650.
92. Komi PV, Tesch P. EMG frequency spectrum, muscle structure, and fatigue during dynamic contractions in man. *European journal of applied physiology and occupational physiology*. 1979;42(1):41-50.
93. Konrad P. The ABC of EMG: A practical introduction to kinesiological electromyography. *Noraxon*. 2005;1.
94. Krstrup P, Mohr M, Steensberg A, Bencke J, Kjær M, Bangsbo J. Muscle and blood metabolites during a soccer game: implications for sprint performance. *Medicine and Science in Sports and Exercise*. 2006;38(6):1165-1174.
95. Lattanzio P, Petrella R, Sproule J, Fowler P. Effects of fatigue on knee proprioception. *Clinical Journal of Sport Medicine*. 1997;7(1):22.
96. Lephart S, Abt J, Ferris C. Neuromuscular contributions to anterior cruciate ligament injuries in females. *Current opinion in rheumatology*. 2002;14(2):168.
97. Lephart S, Henry T. The physiological basis for open and closed kinetic chain rehabilitation for the upper extremity. *Journal of Sport Rehabilitation*. 1996;5:71-87.
98. Lephart SM, Fu FH. *Proprioception and neuromuscular control in joint stability*. Vol 2105: Human Kinetics Champaign, IL; 2000.

99. Liederbach M, Dilgen FE, Rose DJ. Incidence of Anterior Cruciate Ligament Injuries Among Elite Ballet and Modern Dancers A 5-year Prospective Study. *The American journal of sports medicine*. 2008;36(9):1779-1788.
100. Louie JK, Mote Jr C. Contribution of the musculature to rotatory laxity and torsional stiffness at the knee. *Journal of biomechanics*. 1987;20(3):281-300.
101. Manske R, Smith B, Wyatt F. Test-retest reliability of lower extremity functional tests after a closed kinetic chain isokinetic testing bout. *J Sport Rehabil*. 2003;12:119-132.
102. Marshall SW. Recommendations for defining and classifying anterior cruciate ligament injuries in epidemiologic studies. *Journal of Athletic Training*. 2010;45(5):516.
103. McCarthy Persson U, Fleming H, Caulfield B. The effect of a vastus lateralis tape on muscle activity during stair climbing. *Manual Therapy*. 2009;14(3):330-337.
104. McGuine TA, Greene JJ, Best T, Levenson G. Balance as a predictor of ankle injuries in high school basketball players. *Clinical Journal of Sport Medicine*. 2000;10(4):239-244.
105. Mclean S, Samorezov J. Fatigue-induced ACL injury risk stems from a degradation in central control. *Medicine+ Science in Sports+ Exercise*. 2009;41(8):1662.
106. McNair P, Wood G, Marshall R. Stiffness of the hamstring muscles and its relationship to function in anterior cruciate ligament deficient individuals. *Clinical biomechanics*. 1992;7(3):131-137.
107. Meeusen R, Watson P, Hasegawa H, Roelands B, Piacentini MF. Brain neurotransmitters in fatigue and overtraining. *Applied Physiology, Nutrition, and Metabolism*. 2007;32(5):857(858).
108. Melnyk M, Gollhofer A. Submaximal fatigue of the hamstrings impairs specific reflex components and knee stability. *Knee Surgery, Sports Traumatology, Arthroscopy*. 2007;15(5):525-532.

109. Merletti R, Di Torino P. Standards for reporting EMG data. *J Electromyogr Kinesiol.* 1999;9(1):3-4.
110. 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. *The American journal of sports medicine.* 2006;34(6):899-904.
111. Mölsä J, Airaksinen O, Näsman O, Torstila I. Ice hockey injuries in Finland a prospective epidemiologic study. *The American journal of sports medicine.* 1997;25(4):495-499.
112. Mölsä J, Kujala U, Näsman O, Lehtipuu T-P, Airaksinen O. Injury profile in ice hockey from the 1970s through the 1990s in Finland. *The American journal of sports medicine.* 2000;28(3):322-327.
113. Moore SD, Uhl TL, Kibler WB. Improvements in Shoulder Endurance Following a Baseball-Specific Strengthening Program in High School Baseball Players. *Sports Health: A Multidisciplinary Approach.* 2013;5(3):233-238.
114. Mrdakovic V, Ilic DB, Jankovic N, Rajkovic Z, Stefanovic D. Pre-activity modulation of lower extremity muscles within different types and heights of deep jump. *J Sports Sci Med.* 2008;7(2):269-278.
115. Murphy D, Connolly D, Beynon B. Risk factors for lower extremity injury: a review of the literature. *British Journal of Sports Medicine.* 2003;37(1):13-29.
116. Neptune RR, Wright I, Van den Bogert AJ. Muscle coordination and function during cutting movements. *Medicine and Science in Sports and Exercise.* 1999;31:294-302.
117. Newsholme E, Acworth I, Blomstrand E. Amino acids, brain neurotransmitters and a functional link between muscle and brain that is important in sustained exercise. *Advances in myochemistry.* 1987;1987:127-138.
118. Nielsen OB, Clausen T. The Na⁺/K⁺-pump protects muscle excitability and contractility during exercise. *Exercise and sport sciences reviews.* 2000;28(4):159-164.

119. Nigg BM, De Boer RW, Fisher V. A kinematic comparison of overground and treadmill running. *Medicine and Science in Sports and Exercise*. 1995;27(1):98-105.
120. NIH. Conference on Sports Injuries in Youth. Vol NIH publication no. 93-3444. Bethesda, MD: National Institute of Health; 1992.
121. Noakes T. Physiological models to understand exercise fatigue and the adaptations that predict or enhance athletic performance. *Scandinavian Journal of Medicine & Science in Sports*. 2000;10(3):123-145.
122. Noakes T, Gibson ASC, Lambert E. From catastrophe to complexity: a novel model of integrative central neural regulation of effort and fatigue during exercise in humans: summary and conclusions. *British Journal of Sports Medicine*. 2005;39(2):120-124.
123. Noakes T, Myburgh K, Schall R. Peak treadmill running velocity during the VO₂ max test predicts running performance. *Journal of sports sciences*. 1990;8(1):35.
124. Noonan BC. Intragame blood-lactate values during ice hockey and their relationships to commonly used hockey testing protocols. *The Journal of Strength & Conditioning Research*. 2010;24(9):2290-2295.
125. Nybo L. CNS fatigue and prolonged exercise: effect of glucose supplementation. *Medicine and Science in Sports and Exercise*. 2003;35(4):589-594.
126. Nyland J, Caborn D, Shapiro R, Johnson D. Crossover cutting during hamstring fatigue produces transverse plane knee control deficits. *Journal of Athletic Training*. 1999;34(2):137.
127. Olsen O-E, Myklebust G, Engebretsen L, Bahr R. Injury mechanisms for anterior cruciate ligament injuries in team handball a systematic video analysis. *The American journal of sports medicine*. 2004;32(4):1002-1012.
128. Orishimo KF, Kremenec JJ. Effect of fatigue on single-leg hop landing biomechanics. *Journal of applied biomechanics*. 2006;22(4):245.

129. Painelli VS, Nicasastro H, Lancha AH. Carbohydrate mouth rinse: does it improve endurance exercise performance? *Nutrition journal*. 2010;9(1):33.
130. Pandy MG, Shelburne KB. Dependence of cruciate-ligament loading on muscle forces and external load. *Journal of biomechanics*. 1997;30(10):1015-1024.
131. Pearson K, Gordon J. Spinal reflexes. *Principles of Neuroscience*. McGraw-Hill, New York. 2000:713-736.
132. Pettrone FA, Ricciardelli E. Gymnastic injuries: the Virginia experience 1982-1983. *The American journal of sports medicine*. 1987;15(1):59-62.
133. Phillips S, Turner A, Gray S, Sanderson M, Sproule J. Ingesting a 6% carbohydrate-electrolyte solution improves endurance capacity, but not sprint performance, during intermittent, high-intensity shuttle running in adolescent team games players aged 12–14 years. *European Journal of Applied Physiology*. 2010;109(5):811-821.
134. Pitcher JB, Miles TS. Alterations in corticospinal excitability with imposed vs. voluntary fatigue in human hand muscles. *Journal of applied physiology*. 2002;92(5):2131-2138.
135. Prodromos CC, Han Y, Rogowski J, Joyce B, Shi K. A meta-analysis of the incidence of anterior cruciate ligament tears as a function of gender, sport, and a knee injury–reduction regimen. *Arthroscopy: The Journal of Arthroscopic & Related Surgery*. 2007;23(12):1320-1325. e1326.
136. Pyne DB, Boston T, Martin DT, Logan A. Evaluation of the Lactate Pro blood lactate analyser. *European Journal of Applied Physiology*. 2000;82(1-2):112-116.
137. Quammen D, Cortes N, Van Lunen BL, Lucci S, Ringleb SI, Onate J. Two different fatigue protocols and lower extremity motion patterns during a stop-jump task. *Journal of Athletic Training*. 2012;47(1):32.
138. Rahnama N, Reilly T, Lees A. Injury risk associated with playing actions during competitive soccer. *British Journal of Sports Medicine*. 2002;36(5):354-359.

139. Reinschmidt C, Van Den Bogert A, Nigg B, Lundberg A, Murphy N. Effect of skin movement on the analysis of skeletal knee joint motion during running. *Journal of biomechanics*. 1997;30(7):729-732.
140. Riemann BL, Lephart SM. The sensorimotor system, part I: the physiologic basis of functional joint stability. *Journal of Athletic Training*. 2002;37(1):71.
141. Riemann BL, Lephart SM. The sensorimotor system, part II: the role of proprioception in motor control and functional joint stability. *Journal of Athletic Training*. 2002;37(1):80.
142. Riemann BL, Myers JB, Lephart SM. Sensorimotor system measurement techniques. *Journal of Athletic Training*. 2002;37(1):85.
143. Riley PO, Dicharry J, Franz J, Croce UD, Wilder RP, Kerrigan DC. A kinematics and kinetic comparison of overground and treadmill running. *Medicine and Science in Sports and Exercise*. 2008;40(6):1093.
144. Robertson RJ. *Perceived exertion for practitioners: rating effort with the OMNI picture system*: Human Kinetics; 2004.
145. Rodriguez-Alonso M, Fernandez-Garcia B, Perez-Landaluce J, Terrados N. Blood lactate and heart rate during national and international women's basketball. *The Journal of sports medicine and physical fitness*. 2003;43(4):432-436.
146. Rodriguez NR, DiMarco NM, Langley S. Position of the American dietetic association, dietitians of Canada, and the American college of sports medicine: nutrition and athletic performance. *Journal of the American Dietetic Association*. 2009;109(3):509-527.
147. Ross SE, Guskiewicz KM. Time to stabilization: a method for analyzing dynamic postural stability. *Athl Ther Today*. 2003;8(3):37-39.
148. Ross SE, Guskiewicz KM. Examination of static and dynamic postural stability in individuals with functionally stable and unstable ankles. *Clin J Sport Med*. 2004;14(6):332-338.

149. Ross SE, Guskiewicz KM, Gross MT, Yu B. Assessment tools for identifying functional limitations associated with functional ankle instability. *J Athl Train*. 2008;43(1):44-50.
150. Ross SE, Guskiewicz KM, Gross MT, Yu B. Balance measures for discriminating between functionally unstable and stable ankles. *Med Sci Sports Exerc*. 2009;41(2):399-407.
151. Ross SE, Guskiewicz KM, Yu B. Single-leg jump-landing stabilization times in subjects with functionally unstable ankles. *J Athl Train*. 2005;40(4):298-304.
152. Rozzi S, Lephart S, Fu F. Effects of muscular fatigue on knee joint laxity and neuromuscular characteristics of male and female athletes. *Journal of Athletic Training*. 1999;34(2):106.
153. Santamaria LJ, Webster KE. The effect of fatigue on lower-limb biomechanics during single-limb landings: a systematic review. *The Journal of orthopaedic and sports physical therapy*. 2010;40(8):464.
154. Sawka MN, Burke LM, Eichner ER, Maughan RJ, Montain SJ, Stachenfeld NS. American College of Sports Medicine position stand. Exercise and fluid replacement. *Medicine and Science in Sports and Exercise*. 2007;39(2):377-390.
155. Schlabach G. Carbohydrate strategies for injury prevention. *Journal of Athletic Training*. 1994;29(3):244.
156. Sell TC. An examination, correlation, and comparison of static and dynamic measures of postural stability in healthy, physically active adults. *Physical Therapy in Sport*. 2012;13(2):80-86.
157. Sell TC, House AJ, Huang HC, Abt JP, Lephart SM. An Examination, Correlation, and Comparison of Static and Dynamic Measures of Postural Stability in Healthy, Physically Active Adults. *Physical Therapy in Sport (In Press)*. 2011.
158. Sell TC, Pederson JJ, Abt JP, et al. The addition of body armor diminishes dynamic postural stability in military soldiers. *Military Medicine*. 2013;178(1):76-81.

159. Shaw MY, Gribble PA, Frye JL. Ankle bracing, fatigue, and time to stabilization in collegiate volleyball athletes. *Journal of Athletic Training*. 2008;43(2):164.
160. Shimokochi Y, Shultz SJ. Mechanisms of noncontact anterior cruciate ligament injury. *Journal of Athletic Training*. 2008;43(4):396.
161. Söderman K, Alfredson H, Pietilä T, Werner S. Risk factors for leg injuries in female soccer players: a prospective investigation during one out-door season. *Knee Surgery, Sports Traumatology, Arthroscopy*. 2001;9(5):313-321.
162. Spriet LL, Soderlund K, Bergstrom M, Hultman E. Anaerobic energy release in skeletal muscle during electrical stimulation in men. *Journal of applied physiology*. 1987;62(2):611-615.
163. Stepto NK, Martin DT, Fallon KE, Hawley JA. Metabolic demands of intense aerobic interval training in competitive cyclists. *Medicine and Science in Sports and Exercise*. 2001;33(2):303-310.
164. Stuart MJ, Smith A. Injuries in Junior A Ice Hockey A Three-Year Prospective Study. *The American journal of sports medicine*. 1995;23(4):458-461.
165. Swanik CB, Lephart SM, Giraldo JL, DeMont RG, Fu FH. Reactive muscle firing of anterior cruciate ligament-injured females during functional activities. *Journal of Athletic Training*. 1999;34(2):121.
166. Tarnopolsky MA. Females and males: should nutritional recommendations be gender specific? *Schweizerische Zeitschrift für Sportmedizin und Sporttraumatologie*. 2003;51(1):39-46.
167. Taylor JL, Todd G, Gandevia SC. Evidence for a supraspinal contribution to human muscle fatigue. *Clinical and experimental pharmacology and physiology*. 2006;33(4):400-405.
168. Thomas AC, McLean SG, Palmieri-Smith RM. Quadriceps and hamstrings fatigue alters hip and knee mechanics. *Journal of applied biomechanics*. 2010;26(2):159.

169. Tsai LC, Sigward SM, Pollard CD, Fletcher MJ, Powers CM. Effects of Fatigue and Recovery on Knee Mechanics during Side-Step Cutting. *Medicine & Science in Sports & Exercise*. 2009;41(10):1952.
170. Tsintzas O-K, Williams C, Boobis L, Greenhaff P. Carbohydrate ingestion and single muscle fiber glycogen metabolism during prolonged running in men. *Journal of applied physiology*. 1996;81(2):801-809.
171. Utter AC, Robertson RJ, Green JM, Suminski RR, McAnulty SR, Nieman DC. Validation of the Adult OMNI Scale of perceived exertion for walking/running exercise. *Medicine and Science in Sports and Exercise*. 2004;36(10):1776.
172. Wang H-K, Chen C-H, Shiang T-Y, Jan M-H, Lin K-H. Risk-Factor Analysis of High School Basketball-Player Ankle Injuries: A Prospective Controlled Cohort Study Evaluating Postural Sway, Ankle Strength, and Flexibility. *Archives of physical medicine and rehabilitation*. 2006;87(6):821-825.
173. Welsh RS, Mark Davis J, Burke JR, Williams HG. Carbohydrates and physical/mental performance during intermittent exercise to fatigue. *Medicine & Science in Sports & Exercise*. 2002;34(4):723-731.
174. Wikstrom EA, Powers ME, Tillman MD. Dynamic stabilization time after isokinetic and functional fatigue. *Journal of Athletic Training*. 2004;39(3):247.
175. Wikstrom EA, Tillman MD, Chmielewski TL, Cauraugh JH, Naugle KE, Borsa PA. Dynamic postural control but not mechanical stability differs among those with and without chronic ankle instability. *Scand J Med Sci Sports*. 2009;20(1):e137-144.
176. Wikstrom EA, Tillman MD, Smith AN, Borsa PA. A new force-plate technology measure of dynamic postural stability: the dynamic postural stability index. *J Athl Train*. 2005;40(4):305-309.
177. Wikstrom EA, Tillman MD, Smith AN, Borsa PA. A new force-plate technology measure of dynamic postural stability: the dynamic postural stability index. *Journal of Athletic Training*. 2005;40(4):305.

178. Willems TM, Witvrouw E, Delbaere K, Mahieu N, De Bourdeaudhuij I, De Clercq D. Intrinsic Risk Factors for Inversion Ankle Sprains in Male Subjects A Prospective Study. *The American journal of sports medicine*. 2005;33(3):415-423.
179. Williams J. Aetiological classification of injuries in sportsmen. *British Journal of Sports Medicine*. 1971;5(4):228-230.
180. Wilson W, Maughan R. Evidence for a possible role of 5-hydroxytryptamine in the genesis of fatigue in man: administration of paroxetine, a 5-HT re-uptake inhibitor, reduces the capacity to perform prolonged exercise. *Experimental physiology*. 1992;77(6):921-924.
181. Winnick JJ, Davis J, Welsh RS, Carmichael MD, Murphy EA, Blackmon JA. Carbohydrate feedings during team sport exercise preserve physical and CNS function. *Medicine & Science in Sports & Exercise*. 2005;37(2):306.
182. Winter DA. *Biomechanics and motor control of human movement*: Wiley. com; 2009.
183. Wojtaszewski J, Richter EA. Glucose utilization during exercise: influence of endurance training. *Acta Physiologica Scandinavica*. 1998;162(3):351-358.
184. Wojtys E, Wylie B, Huston L. The effects of muscle fatigue on neuromuscular function and anterior tibial translation in healthy knees. *The American journal of sports medicine*. 1996;24(5):615.
185. Wojtys EM, Huston LJ. Neuromuscular performance in normal and anterior cruciate ligament-deficient lower extremities. *The American journal of sports medicine*. 1994;22(1):89-104.
186. Woltring HJ. Smoothing and differentiation techniques applied to 3-D data. *Three-dimensional analysis of human movement*. 1995:79-99.
187. Woods C, Hawkins R, Hulse M, Hodson A. The Football Association Medical Research Programme: an audit of injuries in professional football: an analysis of ankle sprains. *British Journal of Sports Medicine*. 2003;37(3):233-238.

- 188.** Woods C, Hawkins R, Maltby S, Hulse M, Thomas A, Hodson A. The Football Association Medical Research Programme: an audit of injuries in professional football—analysis of hamstring injuries. *British Journal of Sports Medicine*. 2004;38(1):36-41.
- 189.** Woods J, Furbush F, Bigland-Ritchie B. Evidence for a fatigue-induced reflex inhibition of motoneuron firing rates. *Journal of Neurophysiology*. 1987;58(1):125-137.
- 190.** Wyke B. The neurology of joints. *Annals of the Royal College of Surgeons of England*. 1967;41(1):25.
- 191.** Yaggie J, Armstrong WJ. Effects of lower extremity fatigue on indices of balance. *Balance*. 2004;312(322):2004.
- 192.** Yaggie JA, McGregor SJ. Effects of isokinetic ankle fatigue on the maintenance of balance and postural limits. *Archives of physical medicine and rehabilitation*. 2002;83(2):224-228.