2017

Prediction of Acute and Recurrent Ankle Sprains in Athletes

Ryan S. McCann
University of Kentucky, mccannatc59@yahoo.com
Digital Object Identifier: https://doi.org/10.13023/ETD.2017.056

STUDENT AGREEMENT:

I represent that my thesis or dissertation and abstract are my original work. Proper attribution has been given to all outside sources. I understand that I am solely responsible for obtaining any needed copyright permissions. I have obtained needed written permission statement(s) from the owner(s) of each third-party copyrighted matter to be included in my work, allowing electronic distribution (if such use is not permitted by the fair use doctrine) which will be submitted to UKnowledge as Additional File.

I hereby grant to The University of Kentucky and its agents the irrevocable, non-exclusive, and royalty-free license to archive and make accessible my work in whole or in part in all forms of media, now or hereafter known. I agree that the document mentioned above may be made available immediately for worldwide access unless an embargo applies.

I retain all other ownership rights to the copyright of my work. I also retain the right to use in future works (such as articles or books) all or part of my work. I understand that I am free to register the copyright to my work.

REVIEW, APPROVAL AND ACCEPTANCE

The document mentioned above has been reviewed and accepted by the student's advisor, on behalf of the advisory committee, and by the Director of Graduate Studies (DGS), on behalf of the program; we verify that this is the final, approved version of the student's thesis including all changes required by the advisory committee. The undersigned agree to abide by the statements above.

Ryan S. McCann, Student

Dr. Phillip A. Gribble, Major Professor

Dr. Esther Dupont-Vertsteegden, Director of Graduate Studies

PREDICTION OF ACUTE AND RECURRENT ANKLE SPRAINS IN ATHLETES

DISSERTATION

A dissertation submitted in partial fulfillment of the requirements for the degree of Doctor of Philosophy in Rehabilitation Sciences in the College of Health Sciences at the University of Kentucky

By

Ryan Sean McCann

Lexington, Kentucky

Co-Directors: Dr. Phillip A. Gribble, Associate Professor of Athletic Training
and Dr. Brian W. Noehren, Associate Professor of Physical Therapy

Lexington, Kentucky

ABSTRACT OF DISSERTATION

PREDICTION OF ACUTE AND RECURRENT ANKLE SPRAINS IN ATHLETES

Ankle sprains are not only among the most common sport-related injuries, but also associated with a high rate of recurrence. While prevention is a favorable approach to reducing the incidence of index and recurrent ankle sprains, identifying individuals at greater risk may improve allocation of preventative resources. This dissertation aimed to accomplish the following through three aims: 1) determine the ability of baseline clinical tests to predict acute lateral ankle sprain (LAS) in an understudied athletic population, 2) describe the degree of residual impairments and activity limitations in athletes returning to play from a LAS, and 3) determine the ability of patient- and disease-oriented outcomes to predict recurrent ankle sprains in athletes returning to play in the same competitive season.

In the first aim, baseline anterior star excursion balance test scores (SEBT-ANT) and isometric hip extension strength (HEXT) were not useful predictors of LAS in collegiate women's soccer players. Participant height produced a prediction model for LAS with excellent sensitivity (0.88) and moderate specificity (0.51). The diagnostic odds ratio (DOR=7.50) and area under the receiver operating characteristic curve (AUROC=0.73) further established the predictive utility of height for injury. Taller collegiate women's soccer players may be less able to resist external moments exerted on the body, potentially increasing LAS risk.

For the second aim, athletes returning to play from a LAS reported low self-reported function based on scores from the Foot and Ankle Ability Measure activity of daily living (FAAM-ADL) and sport (FAAM-S) subscales. Additionally, participants demonstrated significantly lower ankle dorsiflexion range of motion and SEBT-ANT scores, and significantly greater ankle joint swelling and ligamentous laxity of the involved limb compared to the uninvolved limb. The residual impairments and activity limitations exhibited by athletes returning to play may offer a means of identifying individuals at increased risk for recurrent injury and chronic ankle instability.

In the third aim, athletes that sustained a recurrent ankle sprain in the same competitive sport season exhibited greater height, mass, and body mass index (BMI) compared to those that did not sustain a recurrent injury. ROC curve analyses and DORs further validated the predictive utility of height (AUROC=0.71, DOR=4.93), mass

(AUROC=0.75, DOR=12.21) and BMI (AUROC=0.71, DOR=9.48). A clinical evaluation of pain, ankle joint swelling, ligamentous laxity, ankle dorsiflexion range of motion, SEBT-ANT scores, FAAM-ADL scores, and FAAM-S scores at return to play (RTP) failed to predict recurrent injury status. Similar to the first study, athletes with larger stature may have decreased ability to reverse momentum in the presence of injurious forces. Athletic trainers can use information from this dissertation to determine which athletes are at elevated risk for an acute and recurrent ankle sprain, and ultimately facilitate improved allocation of resources for injury prevention.

KEYWORDS: ankle sprain, injury prediction, clinical evaluation, disease-oriented outcomes, patient-oriented outcomes, return to play

Ryan S. McCann
Student's Signature

April 3, 2017
Date

PREDICTION OF ACUTE AND RECURRENT ANKLE SPRAINS IN ATHLETES

By

Ryan Sean McCann

Phillip A. Gribble, PhD
Co-Director of Dissertation

Brian W. Noehren, PhD
Co-Director of Dissertation

Esther Dupont-Vertsteegden, PhD
Director of Graduate Studies

April 3, 2017
Date

DEDICATION

This work is dedicated to my parents, Sean and Marjorie McCann, and brother, Colin McCann, who have shown endless support for my education and professional goals.

ACKNOWLEDGMENTS

I owe tremendous thanks to many individuals for their guidance and support as I completed this dissertation. First, I must thank my mentor, Dr. Phillip Gribble, for being the epitome of what I consider a doctoral advisor to be. Dr. Gribble's knowledge, experience, ambitiousness, and commitment to his students have made for an excellent educational experience. I also owe many thanks to our other Wildcat Ankle Researchers, Dr. Masafumi Terada and Kyle Kosik, who have been heavily involved in and valuable to my work. I also must thank my three other doctoral committee members for their dedication to this dissertation; Drs. Brian Noehren, Carl Mattacola, and Philip Westgate have all contributed highly valuable input to these projects.

I would also like to thank a several other faculty members who have added significantly to my doctoral education, including Drs. Tim Butterfield, Jennifer Howard, Abbey Thomas, and Tim Uhl. I have also interacted with many peers during my doctoral studies, and I am thankful that many have added to my education and become my friends. I also want to thank a number of athletic trainers who have facilitated this work in their clinical settings: Laurie Blunk, Gretchen Buskirk, Andrew Carlson, Caroline Crowley, Rachel Evans, Emma Gay, Jamey Gay, Lydia Heebner, Allison Isham, Dylan Koesling, Randy McGuire, Margaret Pelton, Taylor Spyker, Danielle Torp, Dan Volpe, and Jenni Williams. Without their help, this type of valuable clinical research could not exist.

Lastly, I want to thank all of my friends and colleagues who were not directly involved in this work, but who provided continual support over the years. They led me to believe I was capable of accomplishing this goal, and helped me stay in high spirits while working towards it.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

Chapter 1: Introduction

Background

Each year, approximately 600,000 to 1 million United States emergency department visits occur due to ankle sprains.[1,2] In half of all cases, physical activity is the source of traumatic injury.[2] With over 8 million combined student-athletes participating annually, the National Collegiate Athletic Association (NCAA) along with high school athletics contribute a significant proportion of the total record of ankle sprains,.[3,4] High school student-athletes sustain over 326,000 ankle injuries in the US annually,[5] over 80% of which are ankle sprains.[6] Among a selection of 15 NCAA sports, approximately 11,000 ankle ligament injuries occur annually, representing up to 15% of all injuries.[7]

As many as 96% of ankle sprains consist of a lateral ankle sprain (LAS), marked by damage to the lateral ankle ligaments.[1,8,9] Aside from high rates of index injuries, widespread recurrence elevates concern for LASs. Konradsen et al.[10] reported that within seven years of a LAS, 19% of patients report recurrent injuries or complain of susceptibility to recurrent injuries. Braun[11] reported that approximately 19% of patients with an ankle sprain sustain a recurrent injury between 6 and 18 months later. Recurrent LAS combined with episodes of ankle "giving way" and feelings of instability comprise the condition known as chronic ankle instability (CAI).[12-14] Among those with a history of LAS, 32-74% report having one or more characteristics consistent with CAI.[10,11,15] Furthermore, approximately 31 and 19% of high school and collegiate athletes, respectively, are estimated to have CAI.[16] Other long-term consequences of LAS include decreased physical activity,[17,18] decreased health-related quality of life,[17] and post-traumatic osteoarthritis.[19]

Prominent injury rates and subsequent long-term consequences have inspired widespread initiatives to prevent LASs. Investigators have previously identified potentially effective prevention strategies for LAS,[20,21] but their implementation commonly suffers from limited time and resources.[22] A number of investigators have attempted to identify risk factors that predict individuals predisposed to LAS in order to direct preventative resources to those most in need. Previous LAS appears to be the most consistently identified risk factor for LAS,[23-31] but its lack of modifiability has created a need to identify other outcomes with strong predictive value. Many outcomes, including ankle range of motion,[24,27,32-39] ankle ligamentous laxity,[23,26,27,32,33,40] ankle muscular strength,[32,33,35,37-40] and body mass index,[24,26,31,34,38-43] have been widely studied, but exhibit inconsistent predictive utility for LASs.

Among clinical assessments, reduced postural control performance has perhaps displayed the greatest consistency as an effective predictor of LAS.[44] Investigators have utilized a variety of clinical and laboratory measures of static and dynamic postural control to confirm the predictive value of postural control performance, but they have often done so with specific athletic populations. Different athletic populations are likely to differ in postural control performance,[45-47] and thus, future studies may need to establish test scores that identify high and low injury risk for understudied athletic populations. Furthermore, other outcomes, such as hip muscular strength[29,48] and ankle-specific patient reported outcomes[24,26,27] have been studied sparsely as predictors of LAS, also with mixed results. Their inclusion in prospective investigations of previously unstudied populations will provide clearer evidence for their ability, or lack thereof, to predict LASs.

Although effective LAS prediction is achievable, the models are unlikely to be perfect. Even the strongest clinical tests will occasionally misclassify athletes as low-risk, potentially leading clinicians to withhold valuable preventative care from those individuals. While clinicians may be unable to predict every index LAS with baseline assessments of neuromusculoskeletal deficiencies, acute injuries are associated with an assortment of structural and functional impairments and activity limitations that may predispose a patient to recurrent LAS. Moreover, many associated impairments and activity limitations remain unresolved by the time patients with a LAS resume pre-injury activities. Two reports found that over 70% of patients with a history of LAS experience at least one residual sequela six months to four years after injury.[11,15] Specifically, patients complained of pain, swelling, weakness, perceived instability, reduced physical function, and recurrent injury at long-term follow-ups. Meanwhile, Medina McKeon et al.[49] reported the median time for return-to-play (RTP) was three days for a first-time LAS and one day for a recurrent LAS. While the collective findings of these studies[11,15,49] suggest that residual impairments and activity limitations are present after RTP, that conclusion is limited in that none of the investigations actually conducted clinical evaluations relative to their patients' time of RTP. As RTP represents a critical time in which injured athletes resume high-risk activity, identifying which impairments and activity limitations consistently present beyond RTP may offer information regarding potential factors that cause some patients to sustain recurrent injuries.

Prediction and prevention of recurrent injury may act as an additional safeguard from long-term consequences of LAS. A number of investigators have attempted to predict recurrent LAS through assessment of various outcomes after an acute LAS and at

long-term follow-ups. In a systematic review of 4 studies,[24,27,50,51] Pourkazemi et al.[52] reported that patients with a grade 2 ankle sprain had approximately 2.6 times greater odds of sustaining a recurrent sprain than patients with a grade 1 or 3 injury. However, the authors cautioned the interpretation of that finding, as the grading system varied between studies, and concerns arose regarding validity of the grading systems. Pooled data from two studies using balance and perceived instability as predictor variables could not identify a significant prediction model for recurrent ankle sprain.[52]

In contrast, Doherty et al.[53] found an effective prediction model for CAI development 1-year post-injury in which predictors consisted of the inability to perform jumping and landing tasks 2-weeks post-injury, and lower self-reported function and dynamic postural control 6-months post-injury. However, others have found limited predictive utility with post-injury assessments of injury grade,[52] previous injury history,[24,27] age,[54] weight-bearing status,[54] mechanism of injury,[54] pain,[54] and presence of syndesmosis involvement.[55] Also within those studies, assessments of BMI,[54] previous injury history,[54,55] injury grade,[54,55] self-reported function and instability,[52,54] ligamentous laxity,[55] dorsiflexion range of motion,[24,54] static postural control,[52,53] dynamic postural control,[53] functional performance,[54] and functional movement kinematics[53] failed to exhibit predictive utility for recurrent LAS or CAI. While these investigations have reported valuable findings regarding prediction of recurrent LAS, the limited collection of studies inhibits widespread clinical applicability.

Perhaps most notably, no study has attempted to predict recurrent LAS in high school and collegiate athletes, despite large contributions to the total volume of LAS incidents from those populations. Additionally, the current body of work has not

considered the predictive value of residual sequelae relative to the re-initiation of sporting activity. While immediate post-injury sequelae may be pertinent to the risk of recurrent LAS, impairments and activity limitations that remain when the patient has returned to high-risk physical activity may be more relevant. Thus, investigators may achieve prediction of recurrent LAS more effectively by evaluating the presence of impairments and activity limitations as the patient resumes pre-injury physical activity levels.

The Problem

A number of LAS prediction models have effectively identified athletes at elevated risk for injury with baseline clinical tests. Since clinical tests may vary among different athletic populations,[45,46] researchers must continue to establish LAS prediction models for athletes of different sports and levels of competition. While prediction models offer a strategy for efficient allocation of injury prevention resources, no model is perfect, and clinicians will occasionally misidentify and subsequently deny preventative care to high-risk athletes unintentionally. Therefore, some athletes will continue to sustain ankle sprains, likely leading to various structural and functional impairments and activity limitations. Which specific impairments and activity limitation consistently last beyond patients' RTP is currently unknown, but they may provide clues as to why certain patients sustain recurrent injury after resuming high-risk physical activity. Previous studies have attempted to produce prediction models for recurrent LAS utilizing post-injury assessments of impairments and activity limitations. However, no study has done so in athletes returning to sport. The ability of clinicians to identify athletic patients at elevated risk for recurrent LAS will be valuable for RTP decision-making. Clinicians can

extend efforts to target residual impairments and activity limitations relevant to recurrent LAS before granting RTP clearance.

Purposes

This dissertation includes three purposes all related to using clinical outcomes to understand the risk of sustaining acute acute LAS. The first purpose was to develop a prediction model for acute LAS injuries in a previously unstudied population (collegiate women's soccer players), utilizing primary outcomes of dynamic postural control and isometric hip strength as well as secondary demographic outcomes as potential predictors. The second purpose was to describe the presence of residual structural and functional impairments and activity limitations in athletes with an acute LAS following clearance for RTP. The third purpose was to develop a prediction model for recurrent ankle sprains in athletes, utilizing assessments of structural and functional impairments and activity limitations at RTP as potential predictors for a repetitive acute LAS injury during a competition season.

Experimental Aims and Hypotheses

Specific Aim 1: To examine the influence of baseline clinical outcome measures (dynamic postural control performance, isometric hip strength, and participant demographics) on the estimated odds of sustaining a LAS in collegiate women's soccer players during the subsequent competitive sport season.

Hypothesis 1: Collegiate women's soccer players with lower baseline dynamic postural control performance and isometric hip strength as well as increased height, body mass, and body mass index (BMI) will have greater estimated odds of sustaining a LAS during the subsequent competitive sport season.

Specific Aim 2.1: To quantify potential deficiencies in clinical outcomes (ankle joint pain, ankle swelling, ankle ligamentous laxity, dorsiflexion range of motion, dynamic postural control, and self-reported function) at RTP in athletic patients with an acute LAS.

Hypothesis 2.1: Patients will exhibit greater ankle swelling and ankle ligamentous laxity, and lower dorsiflexion range of motion and dynamic postural control performance in the involved limb compared to the uninvolved limb at RTP. Additionally, patients will self-report meaningful degrees of pain and activity limitations in the involved limb at RTP.

Specific Aim 2.2: To compare clinical outcomes (ankle joint pain, ankle swelling, ankle ligamentous laxity, dorsiflexion range of motion, dynamic postural control, and self-reported function) between patients with higher and lower injury severity and analyze associations between the number of days of immobilization and rehabilitation following the acute LAS and the degree of impairment and activity limitation.

Hypothesis 2.2: Patients with lower injury severity and more days of immobilization and supervised therapeutic exercise sessions will demonstrate lower pain, ankle swelling and ankle ligamentous laxity, and greater dorsiflexion range of motion, dynamic postural control performance, and self-reported function at RTP.

Specific Aim 3.1: To examine the influence of clinical outcomes (ankle joint pain, ankle swelling, ankle ligamentous laxity, dorsiflexion range of motion, dynamic postural control, and self-reported function) at RTP on the estimated odds of sustaining a recurrent ankle sprain in athletes during the same competitive sport season.

Hypothesis 3.1: Patients with greater ankle joint pain, ankle swelling, and ankle ligamentous laxity and lower dorsiflexion range of motion, dynamic postural control, and self-reported function and instability at RTP will have greater estimated odds of sustaining a recurrent ankle sprain during the same competitive sport season.

Specific Aim 3.2: To examine the influence of demographics (age, height, mass, BMI) and clinical case outcomes (injury grade, percentage of season remaining, previous injury history, days to return to play [DRTP], immobilization, rehabilitation, and usage of prophylactic ankle supports for RTP) on the estimated odds of sustaining a recurrent ankle sprain in athletes during the same competitive sport season.

Hypothesis 3.2: Patients with greater age, height, mass, BMI, injury grade, percentage of season remaining, previous injury history, and DRTP and lower days of immobilization, therapeutic exercise sessions, and usage of prophylactic ankle supports for RTP will have greater estimated odds of sustaining a recurrent ankle sprain during the same competitive sport season.

Operational Definitions

Activity Limitation: Reduced ability to engage in specific activities.

Ankle Sprain: Traumatic injury resulting in mechanical strain of ligaments of the ankle joint. The lateral ankle ligaments, medial ankle ligaments, inferior tibiofibular ligaments, syndesmosis, and/or subtalar ligaments are subject to damage.

Dynamic Postural Control: An individual's ability to maintain their center of mass over a stable base of support while simultaneously executing a functional task.

Functional Impairment: Disability of physiological capacity of body systems.

Lateral Ankle Sprain: Traumatic inversion injury resulting in damage to the lateral ankle ligaments (anterior talofibular, calcaneofibular, and/or posterior talofibular).

Return to Play: Resumption of unrestricted sporting activity following injury.

Self-Reported Function: Patient's perceived capacity to execute activities; assessed with the Foot and Ankle Ability Measure activity of daily living and sport subscales.

Structural Impairment: Disability of specific anatomical parts, such as limbs and joints.

Delimitations

1. Participants were high school and collegiate athletes over 13 years of age.
2. Participants were cleared for full sport participation prior to undergoing testing.
3. Participants in the second and third studies did not have a fracture, other lower extremity injuries, or surgical treatment in addition to the ankle sprain.
4. All ankle sprains resulted in a minimum of one day of activity time-loss.
5. A certified athletic trainer evaluated and treated all injured participants.
6. One certified athletic trainer with over seven years of professional experience conducted independent evaluations of patients with an acute ankle sprain.
7. Independent evaluations of patients with an acute ankle sprain occured in the athletic training facility of each patient's school.

Limitations

1. The small sample sizes of athletes in each study may not represent the overall population.
2. The RTP criteria varied among treating ATs.
3. RTP evaluations occurred in a window 48 hours before and after the actual RTP date.

4. The follow-up period for recurrent ankle sprains was the remainder of the competitive sport season, which compared to other studies, was relatively short; it also caused variability in the follow-up times between patients.

Chapter 2: Review of the Literature

Introduction

The purpose of this literature review is to 1) describe the pathology known as lateral ankle sprain (LAS), 2) discuss current evidence regarding functional impairments and activity limitations associated with LAS, and 3) discuss research regarding the predictive utility of disease- and patient-oriented outcomes for recurrent LAS and chronic ankle instability (CAI).

Lateral Ankle Sprain

Epidemiology

Ankle sprains are an extremely common musculoskeletal pathology, accounting for an estimated 600,000 to 1 million emergency department visits in the United States annually.[1,2] Physical activity is the most common source of ankle sprains, accounting for approximately half of such injuries.[2] The actual incidence of ankle sprains among physically active individuals may be severely underestimated, as McKay et al.[30] reported that over half of high school basketball players with an ankle injury do not seek care from a medical professional, and thus, go undocumented. In the US, nearly 500,000 student-athletes participate in National Collegiate Athletic Association (NCAA) athletics annually,[4] and approximately 7.8 million student-athletes participate in high school athletics annually,[3] resulting in a significant contribution to the incidence of ankle sprains. A previous study of 15 NCAA-sponsored sports reported ankle sprains represented an estimated 15% of all injuries, equating to approximately 11,000 ankle sprains per year.[7] A more recent epidemiological study of 25 NCAA-sponsored sports determined LAS was the most common injury, accounting for approximately 7% of all

injuries.[56] In that report, the authors estimated that approximately 16,000 LASs occur annually, equating to a rate of nearly 5 per 10,000 athlete-exposures.[56] While lower than the previously reported 83 ankle sprains per 10,000 athlete exposures,[7] the more recent study included only LASs and also included additional sports with very low LAS rates.[56] Among high school athletics, up to 22% of all injuries involve the ankle joint.[5,57] Approximately 87% of those injuries are diagnosed as a ligament sprain, indicating that ankle sprains account for up to 19% of all injuries among high school athletes.[5] In an investigation of ankle ligament injuries in nine US high school sports across six years, Swenson et al.[58] estimated that nearly 17% of all high school sport injuries were ankle sprains. That estimate equated to approximately 228,000 ankle sprains per year and an injury rate over 3 per 10,000 athlete-exposures. Of the ankle sprains in high school sports, 85% involved the anterior talofibular ligament (ATFL), supporting previous evidence that the vast majority of ankle sprains are classified as a LAS.[8] Others[1,9] have estimated that as many as 91-96% of all ankle sprains involve the lateral ankle ligaments.

Concern for the high rate of LAS in high school and collegiate athletes is elevated by high rates of recurrent injury. Approximately 10% of injuries among high school athletes are recurrent in nature, and approximately 25% of recurrent injuries consist of ankle ligament sprains, representing the most common recurrent injury.[59] Konradsen et al. [10] reported that within seven years of a LAS, 19% of patients report the recurrence of injuries or complain of susceptibility to recurrent injuries. Braun[11] reported that approximately 19% of patients with an ankle sprain sustain a recurrent injury between 6 and 18 months later. Recurrent LAS combined with episodes of ankle "giving way" and feelings of instability comprise the condition known as chronic ankle instability (CAI).[12-

[14] Among those with a history of LAS, 32-74% report having one or more characteristics consistent with CAI.[10,11,15] Furthermore, approximately 31 and 19% of high school and collegiate athletes, respectively, are estimated to have CAI.[16]

Mechanism of Injury

Lateral ankle sprains typically occur due to excessive rearfoot supination combined with external rotation of the proximal segments during a weight-bearing task.[60] Rearfoot supination is a multiplanar motion composed of ankle plantarflexion, subtalar inversion, and internal rotation of the foot.[61] These combined movement patterns result in the center of pressure (COP) moving laterally on the plantar aspect of the foot, as well as medially relative to the ankle joint axis of rotation.[60] In this position, a ground reaction force creates an external supination moment at the ankle.[60] Pronation moments can be elicited both externally, as with prophylactic ankle supports, and internally, as with the peroneal muscles and lateral ankle ligaments, in order to counteract the external supination moment. A net supination moment of sufficient magnitude will exert stress on the lateral ankle ligaments, potentially causing strain or deformation. During an acute LAS, maximum ankle inversion may be reached as quickly as 40 ms after initial ground contact.[62] However, a reactive internal eversion moment generated by the peroneal muscles is estimated to take 126 ms following detection of a potentially injurious perturbation.[63] Since the sensorimotor system may be unable to react quickly enough to protect against injury, a LAS may be partially attributable to poor preparatory motor planning. However, responsiveness to a perturbation or sudden external inversion moment may still play an important role in protection against LAS. Contrary to previous findings,[62,63] Vaes et al.[64] estimated that total inversion time in participants subjected to a

sudden 50° inversion was 105-110 ms. Furthermore, they estimated the motor response to occur in approximately 80 ms, indicating responsive motor control may potentially limit the degree of damage, depending on the loading rate.

Several investigators have conducted motion analysis of live accidental LAS incidents using 3D motion capture equipment and of filmed LAS incidents using model-based image matching, both of which have provided valuable information regarding mechanisms by which a LAS may occur. The studies commonly noted exaggerated ankle inversion and internal rotation motion during the injury incident.[65-70] Some found increased plantarflexion motion as well,[67,68,70] which supports commonly held perceptions of LAS injury mechanisms. However, others did not observe increased plantarflexion position, indicating it is not a necessary component of the injury mechanism and may be dependent on the task.[65,66,69] A landing task may be more conducive to plantarflexion motion during a toe-to-heel landing, whereas running and cutting tasks involve less vertical motion and more medial-lateral motion.[65] Fong et al.[66] collected plantar pressure data during a cutting task and described center of pressure (COP) shifts toward the forefoot and lateral aspect of the foot, creating a traumatic inversion torque. Kristianslund et al.[68] also reported a more laterally positioned COP relative to the plantar aspect of the foot during the injury trial. In a similar task, Gehring et al.[67] described suppressed activation of the tibialis anterior and peroneus longus muscles 40 and 44 ms following initial ground contact, respectively, followed by bursts that exceeded the activation of the non-injury trials. They concluded that such altered muscle activation patterns might contribute to injurious joint positioning. Proximal to the ankle joint, Gehring et al.[67] noted that the participant exhibited greater hip flexion and

less knee flexion than the non-injury trials. Similarly, Terada and Gribble[70] identified reduced peak knee and hip flexion angles, reduced sagittal plane knee energy dissipation, and a higher, laterally shifted center of mass (COM). Thus, the occurrence of an acute LAS may be partially attributable to a more erect lower extremity landing position, which may limit the ability of the body to attenuate external moments after ground contact. Additionally, it appears as though positioning of the ankle as well as more proximal joints plays a role in the occurrence of a LAS.

Clinical Presentation

A LAS is most commonly recognized by damage to the lateral ankle ligaments. The injury may also involve a number of soft tissue structures including the ankle joint capsule, ankle muscles and tendons, syndesmosis, nerves, and other foot and ankle ligaments.[71] As with other acute musculoskeletal injuries, trauma associated with a LAS can initiate an inflammatory response,[72] and thus, cardinal signs of inflammation, including pain and swelling are commonly present. In addition, mechanical and functional deficiencies commonly arise as the result of structural changes and sensorimotor impairments. Many structural alterations, functional impairments, and activity limitations can be assessed objectively, while subjective assessments provide additional information.

Pain

Following acute trauma, the local release of inflammatory mediators stimulates free nerve endings in soft tissue structures.[73] The presence of inflammatory mediators leads to sensitization, or a reduction in the threshold for nociceptor activation.[74] As a result, higher rates of nociceptive transmissions are sent through afferent Aδ and C nerve

fibers.[73,74] Pain signals terminate in a number of brain centers, each of which correspond to specific sensations or motor responses associated with pain.[75]

Nilsson[76] found that 100% of patients with a LAS reported pain for at least 24 hours after the acute injury. Pain is likely to persist through the acute healing phase and into the subacute stage, with 75-93% of patients complaining of spontaneous pain for at least 2 weeks after a LAS.[77] Pain levels vary following a LAS, and may be related to the severity of the injury. Zammit and Herrington[78] reported that patients with a mild or moderate LAS had mean pain levels of 4.9 on a 10 cm visual analogue scale (VAS) within 24 hours of visiting the emergency department. Boyce et al.[79] reported mean pain levels of 5.3-6.2 on a 10 cm VAS in patients within 24 hours of a moderate or severe LAS. Similarly, patients presenting to general practitioners with a LAS complained of mean pain levels at rest of 3.5-4.5 (50-64%) and mean pain levels during activity over 5 (71%) on a 7 cm VAS.[80] Conversely, Eisenhart et al.[81] found mean pain levels reaching 6.5-7.3 on a VAS in patients within 24 hours of sustaining a mild or moderate ankle sprain. However, they did not differentiate between lateral, medial, or syndesmotic sprains, which may have accounted for more severe pain presentations in their participants.[81] Aside from ankle sprain type or severity, pain may vary based on the conditions under which it is measured. Bleakley et al.[82] found that patients visiting the emergency department with a mild or moderate acute ankle sprain reported mean pain levels of 23-26 (out of 100) at rest and 54-58 during activity. Similarly, van Rijn et al.[83] noted that patients' pain ranged from 2-8 on a 10 cm VAS depending the activity, which ranged from rest to running on a rough surface.

Long-term pain is commonly present in patients with a LAS. Braun[11] examined over 400 patients with a LAS between 6 and 18 months after the acute injury and found that over 50% complained of residual ankle pain. Approximately 23% of patients reported moderate to severe ankle pain in that time.[11] In a similar study, Anandacoomarasamy and Barnsley[15] evaluated 19 patients with a LAS 1-4 years post-injury, and found that 47% complained of residual pain. Verhagen et al.[84] reported that 27-35% of LAS patients complained of ankle pain 9 months following the acute injury, and 17-22% reported residual ankle pain 6.5 years post-injury. Van Rijn et al.[85] conducted a systematic review of studies examining the clinical course of a LAS and concluded that 5-33% of patients experience pain for at least 1 year and 5-25% of patients experience pain for at least 3 years. Although pain is not a requisite for the classification of CAI,[12-14] Wright et al.[86] reported that individuals with CAI more commonly experience pain at end-range ankle inversion. Therefore, persistent pain following a LAS may have implications for prolonged dysfunction and perceived instability.

Swelling

Joint swelling is another common marker of acute inflammation following a traumatic musculoskeletal injury. Tissue damage causes a release of inflammatory mediators that stimulate vasodilation and vascular permeability, which respectively elevate local blood flow and promote migration of inflammatory cells to the injury site.[72] This collection of substances, referred to as exudate, moves to the injury site in a fluid form, causing local swelling or edema.[61] During the initial inflammatory response, neutrophils and macrophages are largely present and active in phagocytosis, or the

consumption of damaged cells.[87] Neutrophil counts peak within 48 hours and may remain present for 1-2 weeks following injury.[87] Macrophages peak 5-7 days after injury and remain present more than 2 weeks after injury.[87] The proliferation and maturation phases of healing coincide with a reduction (but not necessarily complete absence) of acute inflammation.[87]

Ankle joint swelling is present in 75-100% of patients for up to 2 weeks following a LAS.[77] Using a bimalleolar girth measurement, Boyce et al.[79] reported limb-to-limb swelling differences of approximately 1.4 cm within 24 hours of a LAS. Pugia et al.[88] employed a figure-of-eight girth measurement to assess swelling, and found that within 10 days of sustaining a LAS, patients demonstrated increased girth of the involved ankle by nearly 1.8 cm. In patients with a LAS, swelling commonly persists and may also contribute to prolonged dysfunction. Braun[11] noted that approximately 36% of patients exhibited swelling 6-18 months following injury. Anandacoomarasamy and Barnsely[15] had very similar results, with 37% of patients reporting swelling 1-4 years post-injury. Verhagen et al.[84] reported that 26-31% of LAS patients complained of ankle swelling 9 months following the acute injury, and 21-33% reported residual ankle swelling 6.5 years post-injury.

Ankle Ligamentous Laxity

The anterior talofibular ligament (ATFL) is the most commonly damaged ankle ligament during a LAS, with the calcaneofibular ligament (CFL) and posterior talofibular ligament (PTFL) being sites of secondary and tertiary damage, respectively.[71,89] Operative findings of 27 patients with an acute LAS revealed 100% had a complete ATFL rupture, 17 (63%) had a complete CFL rupture, 7 (26%) had a partial CFL

rupture.[89] Fallat et al.[71] reported that among 547 patients with a LAS, 453 cases (83%) involved the ATFL, 366 cases (67%) involved the CFL, and 187 cases (34%) involved the PTFL. The anterior drawer test is a common clinical test of ligamentous laxity, and a greater degree of translation is generally considered a sign of damage to the ATFL.[90] Greater laxity on the inversion talar tilt test is considered a sign of damage of the CFL.[90] Ankles with a history of LAS commonly exhibit greater degrees of joint laxity on anterior drawer and inversion talar tilt tests than ankles with no history of LAS.[91] Others have reported joint laxity in the anterior drawer test only,[92,93] which may be attributed to the increased likelihood of ATFL damage compared to the CFL.

A review of studies tracking ligamentous laxity changes longitudinally in patients with a LAS determined that remodeling and recovery of mechanical stability are expected to take a minimum of 6 weeks to 3 months.[90] Full recovery of mechanical stability commonly takes up to 1 year.[90] Among 242 patients with an acute LAS, Broström[94] reported that 28-31% actually exhibited ligamentous laxity on the anterior drawer test for greater than 1 year. Additionally, several investigators have noted ligamentous laxity in individuals with CAI or characteristics resembling CAI.[92,95-97] However, residual ligamentous laxity is not a requisite for CAI classification,[12-14] and its contribution to CAI development is questionable. Hubbard et al.[95] noted that individuals with CAI displayed greater anterior drawer and inversion talar tilt laxity compared to controls, and 31% of group membership variance was explained by ligamentous laxity. Conversely, Wikstrom et al.[98] determined that ankle joint stiffness, which is partially influenced by ligamentous laxity, did not differ between those with CAI and LAS copers. They postulated that if

ligamentous laxity is not a predisposing factor to recurrent LAS, it may just be a common, inconsequential sequela observed following acute inversion trauma.[98]

Dorsiflexion Range of Motion Deficits

Restricted dorsiflexion range of motion (ROM) is another prominent impairment following an acute LAS.[99] Limited dorsiflexion ROM is likely attributable to one or more structural alterations, such as plantarflexor tightness, immobility of the posterior talocrural joint capsule, or positional faults of the talus or fibula. Posterior talocrural capsular immobility may be recognized by the inability to translate the talus posteriorly relative to the tibia.[91,100] During normal sagittal plane talocrural motion, the talus translates anteriorly with plantarflexion and posteriorly with dorsiflexion. Thus, a posterior capsular restriction may restrict posterior talar translation, subsequently limiting dorsiflexion ROM. Similarly, after a LAS, damage to the ATFL may cause the talus to subluxate anteriorly, creating an anterior positional fault.[91] As the anterior end of the wedge-shaped talus is wider than the posterior end,[61] the anteriorly positioned talus may resist returning to its normal position. The anterior talar fault results in an anteriorly positioned talocrural axis of rotation, limiting the ability of the talus to glide posteriorly, mechanically blocking dorsiflexion ROM.[91] This structural alteration has not previously been measured in acutely sprained ankles, but the presence of an anterior talar fault has been reported in individuals with chronic ankle instability (CAI).[101] Wikstrom and Hubbard[101] noted that the injured ankles of individuals with CAI had a more anteriorly positioned talus compared to their uninjured contralateral limbs and those of healthy controls.

Dorsiflexion ROM may also be limited by arthrokinematic restrictions between the distal fibula and tibia. Normally, the distal fibula translates posteriorly on the tibia during ankle dorsiflexion.[102] Tension on the ATFL during a LAS is thought to pull the distal fibula anteriorly, creating a positional fault, altered arthrokinematics, and ROM deficits.[91,103] Anterior faults of the distal fibula have been reported in over 80% of patients with a subacute LAS.[104,105] In addition, Hubbard et al.[106] identified more anteriorly positioned distal fibulas in individuals with CAI compared to their uninvolved limbs and healthy controls. The fibular positional fault is directly related to the degree of ankle joint swelling,[104,105] and thus, may be partially corrected with swelling reduction.[104] However, this association may also be related to the injury severity, in which a more severe LAS may be inclined to demonstrate increases in both swelling and positional faults.

Mechanical restrictions arising from a LAS may have other effects on dorsiflexion ROM. Plantarflexed ankle positions caused by positional faults have been postulated to promote adaptive shortening of the triceps surae and Achilles tendon when engaging in functional movements.[99] Tightness of the gastrocnemius-soleus complex may also be partially attributed to immobilization in the acute healing stages.[107] Terada et al.[107] conducted a systematic review to determine the most effective methods for correcting dorsiflexion ROM following an ankle sprain, and concluded static stretching resulted in the greatest improvements. The noted benefits of static stretching on dorsiflexion ROM lend support to the existence of plantarflexor tightness in ankle-injured populations.

Postural Control Deficits

Patients with an acute LAS commonly exhibit postural control deficits in static and dynamic conditions.[108-114] Static postural control tests measure the ability of an individual to maintain his or her center of gravity over the base of support with as little movement as possible, whereas dynamic postural control tests measure the ability of an individual to maintain his or her center of gravity over the base of support while performing a functional movement. Although postural control deficits are common in the acute stages of recovery, reports have varied in regards to how long this impairment may persist. At 1-day and 2-weeks post-injury, Hertel et al.[113] reported that individuals with an acute LAS had increased center of pressure excursion (COP) velocity and length, as well as an increased range of COP excursion in the involved limb compared to the uninvolved limb during a static single-leg stance. No limb-to-limb differences were noted at a 4-week follow-up.[113] Similarly, Evans et al.[112] found reduced static postural control performance in the acute stages of recovery and up 3 weeks following a LAS. However, no deficits were found at a 4-week follow-up.[112] Additionally, Holme et al.[114] reported that deficits in static postural control were resolved within 4 months in both patients who did and did not engage in supervised rehabilitation. Doherty et al.[109-111] identified reduced static and dynamic postural control performance in individuals within 2 weeks of sustaining a LAS compared to healthy controls. Unlike previous authors, they found that postural control deficits persisted for up to 6 months post-injury.[115,116] At a 1-year follow-up, those who developed CAI retained postural control deficits, while those classified as LAS copers did not.[117,118] Thus, the aforementioned discrepancies in postural control resolution may be explained by the study participants' tendencies to develop or avoid CAI. Postural control deficits are among the most commonly reported

functional deficits in individuals with CAI,[98,117-126] and the current definition of CAI requires a minimum of 12 months since the initial LAS and a minimum of 3 months since the most recent LAS.[12-14] Therefore, postural control impairments may persist in many individuals for months or years after an acute LAS.

The first reports of postural control deficiencies in an ankle-injured population were made by Freeman et al.[127] They proposed that during a LAS, mechanoreceptors within the lateral ankle ligaments incur damage, resulting in deafferentation. Type II and III mechanoreceptors, responsible for sensing initial joint motion and end-range motion, respectively, are abundant in the lateral ankle ligaments.[128] Several studies have investigated measures of postural control in participants following anesthesia injections in the ATFL and lateral ankle joint capsule. Hertel et al.[129] reported participants exhibited greater medial-lateral center of pressure (COP), but no changes in postural sway distance or joint position sense. In a similar study, Konradsen et al.[130] also identified no changes in postural sway, but did find reduced passive joint position sense. De Carlo and Talbot[131] actually noted an increase in average time in balance in individuals receiving an anesthesia injection. This unexpected finding may have occurred due to a learning effect in the postural control task or limited demand on the Type III mechanoreceptors during the task. Collectively, these findings suggest that ligamentous deafferentation does not entirely explain postural control losses in ankle-injured populations.[132]

Impaired postural control may also be affected by arthrogenic muscle responses throughout the lower extremity as a means of promoting disuse and protecting the injured limb. Perceived pain may cause supraspinal centers of motor control to activate

inhibitory signaling known as pre-synaptic inhibition. In this case, corticospinal tract axons create synapses with Ia afferent axons.[133] These axoaxonic synapses are GABAergic, in which the inhibitory neurotransmitter gamma-Aminobutyric acid (GABA) is released from the descending corticospinal axon to the Ia afferent, ultimately decreasing the excitability of the post-synaptic spinal interneuron or alpha motoneuron (αMN).[133] Hass et al.[120] demonstrated that individuals with a history of CAI had impaired control of plantar center of pressure (COP) during a gait initiation task, which is controlled by the motor cortex. Thus, supraspinal influences of neural inhibition are likely present in ankle-injured populations. However, due to the study's retrospective design, the authors could not confirm whether motor control impairments were a result or cause of prior ankle injuries.

Recurrent inhibition has been described as a "gain regulator" that tempers motor responses to excessive sensory input.[134] The primary regulators of recurrent inhibition are Renshaw cells, which synapse with motor axon collaterals branching from the αMN. Renshaw cells subsequently synapse with and inhibit αMNs, gamma motoneurons (γMNs), and spinal interneurons. While over-active recurrent inhibition has not been documented in patients with an acute LAS, Sefton et al.[135] utilized a conditioned Hoffman-reflex (H-reflex) stimulus followed by a second H-reflex stimulus to determine the degree of recurrent inhibition in the soleus muscle of patients with CAI. Bussel and Pierrot-Deseilligny[136] explained that a conditioned H-reflex stimulus activates the recurrent inhibition pathway in the αMNs of interest. The second H-reflex stimulus 10 ms later is then prone to the residual post-synaptic inhibition, and results in depressed muscular activation.[136] Sefton et al.[135] found that recurrent inhibition of the soleus was

present in single- and double-leg stance conditions in patients with CAI compared to healthy, matched controls. Along with other measures of postural control and neural excitability, the degree of recurrent inhibition was utilized in a discriminant functional analysis and contributed significantly to the ability to differentiate between those with and without CAI.[135]

Gamma loop dysfunction is yet another possible pathway of neural inhibition contributing to sensorimotor deficits following a LAS. Although it has not been directly investigated in ankle-injured individuals, there is evidence to suggest gamma loop dysfunction contributes to persistent functional impairments in this population. Within skeletal muscle, length and rate of length change in extrafusal fibers are detected by muscle spindle fibers.[132] Contractile units within muscle spindles regulate their sensitivity to sensory stimuli,[126,132] adjusting their feedback to the central nervous system, which is necessary for the generation of appropriate motor responses. The γMN innervates muscle spindle fibers, but likely receives supraspinal influences,[137] which may be a source of inhibition following a musculoskeletal pathology.[120,133] Renshaw cells are another direct influence of the γMN,[134] potentially introducing recurrent inhibition within the gamma loop. Additionally, damage to joint mechanoreceptors likely affect gamma loop dysfunction. Konishi et al.[138] noted abnormal muscular strength and activation responses in anterior cruciate ligament-injured and knee anesthetized participants compared to controls following prolonged knee vibration. Neurotransmitter depletion or an elevated Ia fiber threshold leading to reduced muscular strength and activation is normally expected to occur after a prolonged vibration, but the experimental groups demonstrated the opposite effect. The authors postulated that the influence of the γMN

on Ia afferents may result in abnormal motor responses in the presence of gamma loop dysfunction. As this was observed in participants with injured or anesthetized knee ligaments, the contribution of joint mechanoreceptor impairment to gamma loop dysfunction was supported.

While the magnitude of each inhibitory pathway's contribution to neuromuscular alterations is unknown, studies of muscular activation and strength related to postural control in ankle-injured participants support the presence of inhibitory influences. Feger et al.[139] reported decreased activation of the tibialis anterior muscle during a dynamic postural control task in participants with CAI. When transitioning from double- to single-leg stance, van Duen et al.[140] noted that individuals with CAI displayed increased onset latency of ankle, knee, and hip musculature. In patients with acute ankle sprains, Doherty et al. identified a greater hip-dominant strategy of postural control that persisted in those who developed CAI 1 year post-injury.[118,141] Similarly, Rios et al.[124] noted reduced ankle muscular activation and increased hip and spine muscular activation during a dynamic postural control task in individuals with CAI. The authors postulated that residual ankle muscular dysfunction may have resulted in increased reliance in alternative strategies to maintain balance.[118,124,141] McCann et al.[122] identified decreased dynamic postural control and isometric hip strength in individuals with CAI compared to LAS copers and controls. Additionally, the CAI group's postural control performance was directly related to isometric hip strength, whereas the other groups' was not.[122] Therefore, a shift to a more proximal postural control strategy may exist in ankle-injured individuals, but the proximal musculature may also suffer from inhibition, further limiting motor control. Along with effects on proximal musculature, inhibitory pathways

may also effect function on the uninjured contralateral limb. Evans et al.[112] prospectively measured static postural control bilaterally in collegiate athletes. Those suffering a subsequent LAS exhibited postural control deficits in the injured and uninjured ankle 1 day post-injury, suggesting neuromuscular limitations were mediated by the central nervous system.[112] While these studies are evident of muscular inhibition in those with a history of LAS, many were conducted in participants with CAI as opposed to an acute injury. Further inquiry is required to fully understand the impact of inhibition on postural control in acutely injured individuals.

Reduced Self-Reported Function & Stability

While patients with a LAS commonly exhibit objective structural and functional impairments, subjective measures of function and health-related quality of life (HRQOL) are considered an important component of injury evaluation, and often reveal additional impairments and limitations.[142] Examples of such validated ankle-specific patient-reported outcomes (PROs) include the Foot and Ankle Ability Measure (FAAM),[143,144] Foot and Ankle Disability Index (FADI),[145,146] Ankle Joint Functional Assessment Tool (AJFAT),[147,148] and Functional Ankle Outcome Score (FAOS).[149] The FAAM is perhaps the most commonly used instrument for evaluating functional limitations in patients with an acute LAS. The FAAM consists of two subscales emphasizing limitations with activities of daily living (FAAM-ADL) and sports (FAAM-S). Each FAAM subscale is scored on a 100-point scale, with a score of 100 equating to no functional limitations. Klykken et al.[150] reported that 10 patients with an acute LAS in the past 24-72 hours had mean scores of 63% on the FAAM-ADL and 35% on the FAAM-S. Croy et al.[151] found that within 2 weeks of sustaining a LAS, patients reported scores of 65-70% on the

FAAM-ADL and 35-40% on the FAAM-S. Significant improvements were noted at 3-week (FAAM-ADL = 85-90%; FAAM-S = 60-65%) and 6-week (FAAM-ADL = 90-95%; FAAM-S = 70-75%) follow-ups.[151] Similar to the previous study,[151] Doherty et al.[109,111,152,153] noted that patients reported scores of 57-70% on the FAAM-ADL and 32-40% on the FAAM-S within 2 weeks of sustaining an acute LAS. At 6-month follow-ups, Doherty et al.[115,116,154,155] noted improvements (FAAM-ADL = 96%; FAAM-S = 83-87%), but their levels of self-reported function were still significantly less than those of uninjured individuals.

The FADI is a similar instrument to the FAAM with nearly identical questions and a division into two subscales (FADI-ADL and FADI-S), both of which are scored on a 100-point scale. The primary difference is that the FADI-ADL has additional items regarding pain that are not included on the FAAM-ADL. Cosby et al.[156] reported FADI-ADL scores of 73% and FADI-S scores of 82% in patients with an acute LAS. However, they did not specify the amount of time between the injury episode and collection of outcome measures.[156] Hubbard and Cordova[157] found that patients reported mean FADI-ADL scores of 68% and FADI-S scores of 46% within 3 days of sustaining a mild or moderate LAS. Both scores were significantly lower than those of the uninjured contralateral limb and matched limb of a healthy control group. At an 8-week follow-up, patients reported mean FADI-ADL scores of 88% and FADI-S scores of 72%, both of which were significantly lower than the uninjured contralateral limb and matched limb of a healthy control group.[157]

The FAOS assesses a patient's symptoms and functional limitations in the previous week.[149] It consists of 42 items separated into five domains: symptoms, pain,

function with ADLs, function with sport and recreation, and quality of life. Each domain is evaluated on a 100-point scale with higher scores representing favorable outcomes. Aiken et al.[158] evaluated 50 patients with an acute ankle sprain and noted deficient scores related to symptoms (58%), pain (59%), function with ADLs (63%), function with sport (33%), and quality of life (39%) 4 days following an initial emergency department visit. Although improved, deficient scores related to symptoms (67%), pain (78%), function with ADLs (89%), function with sport (67%), and quality of life (63%) were still present 30 days following the emergency department visit.[158]

De Bie[77] produced an ankle-specific measurement of self-reported function, commonly referred to as the Ankle Function Score (AFS).[159] The instrument includes metrics of pain, instability, weight-bearing status, swelling, and gait patterns that are combined to produce a single score out of 100.[77] While selected arbitrarily and not yet validated, patients with scores over 75% are considered healed.[77] Van Middlekoop et al. reported that patients with an acute LAS had an average AFS score of 42%.[159] Similarly, van Rijn et al.[83] reported mean AFS scores of 39% in patients with an acute LAS. In addition to evaluating functional limitations of acutely injured individuals, these ankle-specific PROs have also been used to describe the degree of perceived function in patients that have and have not developed CAI following a LAS.[160]

Identification of CAI is commonly accomplished with various instruments, including the Cumberland Ankle Instability Tool (CAIT),[161,162] Ankle Instability Instrument (AII),[163] and the Identification of Functional Ankle Instability (IdFAI) questionnaire.[164,165] The CAIT mostly addresses issues related to perceived instability. The AII does as well, but with increased emphasis on previous injuries and their

management. The IdFAI contains components of the CAIT and AII. However, the CAIT, AII, and IdFAI cannot detect functional impairments, so PROs such as the FAAM and FADI are commonly used in conjunction with those intended to designate injury status.

According to the International Ankle Consortium (IAC), reporting scores less than 75% in three or more domains of the FAOS is representative of CAI.[12-14] Additionally, scores less than 90% and 80% on the FAAM-ADL and FAAM-S, respectively, are standard levels of self-reported dysfunction used to describe individuals with CAI.[12-14,143] However, these measures are not considered an absolute necessity for CAI classification.[12-14] Doherty et al.[117,118,166,167] reported scores of approximately 96% on the FAAM-ADL and 86% on the FAAM-S in individuals with CAI. Others[168,169] have reported scores of 89-94% on the FAAM-ADL and 76-94% on the FAAM-S in individuals with CAI. Terada et al.[170] found that individuals with CAI reported FAAM-ADL and FAAM-S scores ranging from 90-97% and 79-94%, respectively, depending on whether they experienced recurrent injuries, perceived instability, or both. Similarly, when the FADI was used to describe the level of perceived function and not to classify injury status, individuals with CAI reported scores of 89-93% on the FADI-ADL and 75-84% on the FADI-S.[95,171]

Return to Play

The time of return to play (RTP) is a critical instance in treatment of athletes with a recent injury. At RTP, a previously injured body part resumes unrestricted exposure to activities that increase risk of recurrent injury. Thus, when making RTP decisions, treating clinicians must carefully consider attributes of the patient's recovery that may further contribute to elevated risk for recurrent injury. Unfortunately, treatment

recommendations for a LAS have previously been overly simplistic, consisting of protection, rest, ice, compression, elevation (PRICE), and basic guidelines for weight bearing and ROM.[158,172] These components of LAS care are valuable, but may not address all functional neuromuscular impairments associated with the injury. Punt et al.[18] reported that using basic at-home rehabilitation instructions insufficiently corrected ankle ROM and strength deficits and gait alterations in patients 4 weeks post-LAS. Additionally, regularly providing these rudimentary treatment recommendations may contribute to perceptions of a LAS as a benign injury, potentially reducing patient adherence to rehabilitation and limiting the thoroughness of care.[173] Therefore, the presence of residual structural and functional impairments and activity limitations discussed in the previous section may be partially due to insufficient care in the acute stages of recovery. Contemporary recommendations for LAS care are more comprehensive, incorporating manual therapy, functional rehabilitation targeting neuromuscular control, follow-up management, and RTP considerations.[174,175] Athletes are recommended to refrain from RTP until self-reported function and functional performance measures have returned to normal. Additionally, the use of prophylactic ankle supports following RTP is recommended to mechanically stabilize the joint.

Athletic trainers (ATs) commonly introduce therapeutic interventions as the standard of care for an acute LAS, yet many investigations continue to report that symptoms often persist in patients for months or even years after injury.[11,15] Often, these residual complaints vastly exceed typical timeframes for RTP. Nelson et al.[5] conducted an epidemiological study of over 900 ankle injuries in high school athletes that had access to a staff AT. Although the investigation included other conditions than LAS,

83% of reported ankle injuries consisted of LAS. They estimated that over 50% of high school athletes with an ankle injury reach RTP within 7 days, and 85% reach RTP within 21 days. Medina McKeon et al.[49] conducted a time-to-event analysis aiming to gauge the association between injury history and RTP following a LAS. In over 200 high school athletes, the median time for RTP was 3 days for an index LAS and 1 day for a recurrent LAS. Furthermore, probabilities for RTP within 7 days of injury were 86% for an index LAS and 94% for a recurrent LAS. The large potential overlap between residual impairments and RTP has caused concern that insufficient care for a LAS may contribute to recurrent injury or CAI after the athlete has reached RTP. Ardern et al.[176] argued that waiting for all residual impairments to subside before permitting RTP may result in favorable injury-specific outcomes, but may not be in the best interest of the patient. Additionally, the influence of each impairment on the recurrence of LAS or development remains unknown. Thus, the optimal degree of impairment resolution required to prevent recurrent LAS and CAI also remains unknown.

Prospective Injury Prediction

Vast rates of musculoskeletal injury, particularly LAS, and their long-term consequences have led to widespread initiatives to prevent index and recurrent injuries. Effective prevention strategies for index and recurrent LAS have been identified previously,[20,21] as have interventions designed to eliminate characteristics of CAI,[177] but their implementation may often suffer from the same factors that limit care of acutely injured athletes: limited time and resources.[22] As a potential means for improving efficient allocation of preventative resources, a number of investigators have attempted to identify risk factors that predict individuals predisposed to index and recurrent LAS.

Establishing effective prediction models can allow intervention resources to be allocated to patients with the greatest risk for injury. Additionally, prediction models can lead to modified interventions that target impairments most relevant to LAS occurrence.

Lateral Ankle Sprain Prediction

Efforts to predict an index LAS have relied heavily on prospective study designs, in which outcomes are assessed at baseline, and then injuries are tracked for a specified follow-up period. Often, intrinsic participant characteristics that are routinely evaluated following an acute ankle sprain make up the primary outcomes assessed at baseline. A variety of statistical models are suitable for determining how or if variations in a population's outcomes can influence subsequent injury occurrences.

Previous Injury History

Perhaps the LAS risk factor most commonly reported in prospective studies is a previous history of LAS. Ekstrand and Gillquist[25] found significantly higher rates of previous LAS in adult soccer players that sustained a LAS during 1 year of injury surveillance (47%) compared to those that did not sustain a LAS in the same time (25%). Kofotolis et al.[28] also prospectively examined a large cohort of amateur soccer players and determined that those with a previous LAS had nearly 2 times greater odds of sustaining a LAS during 2 years of subsequent observation. In another study of amateur male soccer players,[26] previous history of LAS was again the strongest predictor of LAS, increasing the odds of injury approximately 23%. McKay et al.[30] prospectively studied a sample of over 10,000 high school basketball players and found that athletes with a previous LAS had nearly 5 times greater odds of sustaining a LAS. Arnason et al.[23] found a similar increase in the odds (~5x) of sustaining an ankle sprain in adult male

soccer players with a previous ankle sprain. McHugh et al.[29] found that the rate of grade 2 and 3 LASs was over 4 times greater in high school athletes with a previous history of LAS. In high school football players, Tyler et al.[31] noted a significantly greater ankle sprain incidence rate in participants with a previous history of ankle sprain. Among physically active college students, de Noronha et al.[24] reported that a history of previous ankle sprain increased the likelihood of injury throughout a 1-year follow-up period. Collectively, these studies suggest that a previous musculoskeletal injury may affect the long-term mechanical integrity and sensorimotor control surrounding the joint, potentially increasing the risk for recurrent injury. Hiller et al.[27] studied the predictive utility of a previous LAS on the contralateral limb. They utilized a survival analysis and actually determined that a history of LAS on one limb was associated with nearly 4 times greater odds of sustaining a contralateral LAS within a 13-month follow-up period. While previous LAS has mostly been confirmed as a risk factor for subsequent LAS, a few other studies have failed to identify predictive utility of injury history in athletic populations.[34,178,179]

Postural Control

Other researchers have studied more modifiable outcomes as potential risk factors for LAS. Early work by Freeman et al.[127] initially uncovered a link between CAI and postural control deficits. As a result, a number of investigators have studied reduced postural control as a predictor of LAS. Trojian and McKeag[179] utilized a single-leg, eyes closed static balance test as a baseline assessment in high school in collegiate athletes. The test was measured as a dichotomous (pass/fail) outcome, in which an inability to maintain balance or feelings of instability were criteria for failure. They found that a

failed test was associated with 2.5 times greater odds of sustaining an ankle sprain compared to those with a passed test. Hrysomallis et al.[180] also utilized a single-leg stance task to test elite Australian football players, but on an unstable surface. Above average medial-lateral COP excursions were also associated with approximately 2.5 times greater odds of sustaining an ankle injury. Tropp et al.[181] reported that physically active males with lower static postural control performance measured with stabilometric recordings had significantly greater risk of sustaining a LAS. McGuine et al.[42] noted that high school basketball players had approximately 7 times as many LASs when found to have poor single-leg postural sway scores. Using a similar postural control assessment, Wang et al.[37] reported that every 1 mm increase in postural sway variation in high school basketball players was associated with a 22% increase in odds of sustaining an ankle injury. Henry et al.[34] found that soccer players with longer double-leg static stability times on a wobble board had significantly lower odds of sustaining an ankle injury; odds of injury reduced 57% for every 1-second increase in stabilization time.

Reduced dynamic postural control has also demonstrated predictive utility for LAS. In a cohort of over 600 high school and collegiate football players, Gribble et al.[41] identified predictive utility in the star excursion balance test (SEBT). Specifically, those athletes with anterior reach scores below 67% had nearly 3 times greater odds of sustaining a LAS during the subsequent season. Similarly, Plisky et al.[182] conducted baseline screening with the SEBT in over 200 high school basketball players and found that athletes' odds of lower extremity injury grew more than 2 times with limb-to-limb differences over 4 cm on the anterior reach. Additionally, the odds of lower extremity injury were over 6 times greater in athletes with scores under 94% on the composite

SEBT. Another previous study[24] demonstrated that physically active college students with lower posterolateral SEBT scores were more likely to sustain a LAS within 1 year of baseline testing. Hiller et al.[27] reported the inability to balance on demipointe for 5 seconds was predictive of ankle injuries in dance and ballet students, but its strength as a predictor was less than that of previous injury history. Willems et al.[38,39] conducted a series of static and dynamic postural control tests in college students and found poor performance in some measures of dynamic postural control were associated with a greater likelihood of sustaining a LAS. However, many other measures of postural control were not influential of injury status.[38,39] Although others have failed to find a significant relationship between poor postural control and LAS,[26,29,33,40] Witchalls et al.[44] conducted a meta-analysis and found that athletes who sustained a subsequent LAS consistently had worse postural control performance.

Dorsiflexion Range of Motion

Dorsiflexion ROM is necessary for establishing a close-packed position and attenuation of external forces during deceleration, and thus, has been an outcome of interest when attempting to predict a LAS. Passive dorsiflexion ROM can be assessed in weight-bearing and non-weight-bearing conditions, both of which are uniquely important to functional movement. A number of studies[32,33,37-39] assessed passive non-weight-bearing dorsiflexion ROM in a prone position with the knee in full extension and flexed to 90° in various physically active populations. Only one study[38] found that dorsiflexion ROM in the knee-extended condition was associated with risk of LAS. Payne et al.[35] assessed active non-weight-bearing dorsiflexion ROM in collegiate basketball players, but found it was not associated with subsequent ankle sprains.

Passive weight-bearing dorsiflexion ROM is commonly assessed with the weight-bearing lunge test (WBLT). Pope et al.[36] utilized the WBLT in a baseline screening of military recruits, and obtained the degree of dorsiflexion ROM through trigonometric calculations. They reported that recruits with scores at lower end of the range (~34°) had 5-times greater risk of sustaining an ankle sprain than those with average dorsiflexion ROM. Several other authors have reported that WBLT scores were not predictive of ankle injuries in physically active populations.[24,27,34] Others have also reported a lack of injury prediction utility with unspecified methods of dorsiflexion ROM assessment.[26,40]

Ankle Ligamentous Laxity

Ankle ligamentous laxity has been measured in several prospective studies. Beynnon et al.[33] examined the anterior drawer and talar tilt tests on NCAA athletes using a 3-point scale and a dichotomous (positive/negative) grading, respectively. According to Cox regression analyses, neither test was associated with rates of ankle injuries. Using similar methods, Baumhauer et al.[32] reported alike findings. Arnason et al.[23] also found no predictive utility for ankle injuries with the anterior drawer and talar tilt tests, but it was unclear how the tests were scored. Other researchers[27,40] have assessed the anterior drawer on a multi-point grading scale in physically active populations, and have not found predictive utility for ankle injuries. Engebresten et al.[26] also assessed the anterior drawer as a dichotomous outcome in male soccer players and found it did not impact the odds of sustaining an ankle injury.

Ankle Muscular Strength

A number of studies have investigated the value of ankle muscular strength as a predictor of ankle sprains, and mixed results have been reported. Fousekis et al.[40]

assessed soccer players' concentric and eccentric isokinetic strength of the dorsiflexors and plantarflexors at 60°/s. They found that those with limb-to-limb isokinetic dorsiflexion and plantarflexion strength asymmetries over 15% had nearly 9 times greater odds of sustaining an ankle sprain. Willems et al.[38,39] assessed concentric and eccentric isokinetic strength of the dorsiflexors, plantarflexors, invertors, and evertors at 30 and 120°/s. Male college students with lower concentric dorsiflexion strength at 30°/s were at greater risk for sustaining a LAS.[38] This finding suggested that impaired strength may limit the patient's ability to establish a close-packed position. However, females with greater concentric dorsiflexion strength at 120°/s were actually found to be at greater risk for sustaining a LAS.[39] These contradictory results suggested that ankle dorsiflexion strength might be of little importance to ankle sprain risk. Baumhauer et al.[32] also measured concentric isokinetic 4-direction ankle strength in collegiate athletes. Participants that sustained a LAS had a higher eversion-to-inversion strength ratio compared to those that remained uninjured. Additionally, within the injured group, the injured limb displayed greater plantarflexion strength, a lower dorsiflexion-to-plantarflexion strength ratio, and a lower eversion-to-inversion strength ratio. Like the findings of Willems et al.,[38,39] this study's results are contradictory, as the influence of eversion-to-inversion strength on injury status differs from between- to within-group comparisons. Other studies[33,35,37] examined isokinetic 4-direction ankle strength, but were unable to find an association with subsequent injury status. Collectively, these results suggest that ankle muscular strength tests may be an inappropriate predictors of ankle sprains.

Hip Muscular Strength

As several studies have noted reduced hip muscular strength in individuals with a history of LAS,[122,183,184] some investigators have attempted to determine the predictive utility of hip muscular strength. McHugh et al.[29] initially conducted baseline assessments of isometric hip flexion, abduction, and adduction strength in high school athletes. Hip muscular strength did not differ between those with and without a subsequent LAS, and variations in hip muscular strength did not affect the odds of sustaining a LAS. More recently, de Ridder et al.[48] prospectively assessed isometric hip strength in youth male soccer players. Using a principal-component Cox regression analysis, they found a significant hazard ratio, indicating that increased hip extension strength was associated with reduced rates of LAS over 3 consecutive competitive seasons. They postulated that reduced hip extensor function might impair an individual's ability to attenuate external forces during deceleration, potentially increasing loads on static structures, such as ligaments. More work is needed to establish the utility of neuromuscular impairments in joints proximal to the ankle as predictors of LAS. As these studies included participants with previous injuries, further prospective inquiry will confirm whether widespread neuromuscular impairments simply predispose individuals to index injuries or arise as result of peripheral musculoskeletal injury.

Patient-Reported Outcomes

Subjective measures of ankle function, stability, and pain are considered valuable components of an ankle sprain evaluation. However, they have been studied seldom as predictors of subsequent ankle sprains. Two prospective studies[24,27] included the CAIT as a measure of perceived ankle stability. Both reported that CAIT scores did not possess any predictive utility for future ankle sprains in physically active populations.

Engebresten et al.[26] incorporated the FAOS as a part of baseline screening of amateur male soccer players, but also found no predictive utility for ankle sprains. Although these three studies had similar findings, a multitude of ankle-specific PROs are commonly used in clinical practice and research, each potentially containing a unique level of predictive value. Thus, the predictive strength of many PROs (FAAM, FADI, IdFAI, AII, AJFAT) remains unknown.

Body Mass Index

Aside from ankle-specific outcomes, researchers have searched for other intrinsic risk factors that may be modifiable through targeted interventions. Body mass index (BMI) is one such outcome, commonly investigated due to its contribution to larger moments of inertia in the lower extremity.[43] Essentially, greater body mass and longer trunk and extremity segments may reduce an individual's ability to resist external moments exerted on the body, potentially increasing injury risk.[43] However, the literature regarding BMI's ability to predict ankle sprains has been inconsistent. Fousekis et al.[40] noted that soccer players with a BMI over 23.1 kg/m^2 had 8-times greater odds of sustaining an ankle sprain. Gribble et al.[41] reported that high school and collegiate football players with a BMI over 26.7 kg/m^2 had 2-times greater odds of sustaining a LAS. Tyler et al.[31] found that ankle sprain incidences in high school football players increased as BMI increased from classifications of normal, risk for overweight, and overweight, defined by normative data. When considered with injury history, they found that the combination of overweight classification and a previous ankle sprain increased the risk of ankle sprain 19 times compared to those with a normal weight classification and no previous ankle sprain. Henry et al.[34] determined that soccer players in the top

tertile of BMI scores had greater risk of ankle injuries than those in the middle tertile. No participants in the lowest tertile sustained an ankle injury. Despite a significant univariate logistic regression model, BMI did not contribute to a multivariate regression model, and thus, the investigators considered it a less valuable risk factor. Others have failed to find predictive utility for ankle injuries using BMI altogether.[24,26,38,39,42]

Recurrent Lateral Ankle Sprain Prediction

Although valuable prediction models for ankle sprains have been produced, discovery of a perfect model is likely unrealistic. As a result, a number of injuries will continue to occur, some with long-term consequences, including recurrent injury and chronic instability. Therefore, prediction and prevention of recurrent LAS and CAI may be equally as important to long-term musculoskeletal health. Only Malliaropoulos et al.[51] has attempted to predict recurrent injury in athletes following an acute LAS. A novel 4-grade severity scale was utilized as the primary predictor of recurrent LAS in a cohort of over 200 elite track and field athletes. Within 2 years of the acute LAS, patients with a grade II injury (29%) sustained the highest rates of recurrent injury. Patients with grade I (14%) and grade IIIA (5.6%) had significantly lower rates of recurrent injury. Higher recurrent injury rates in patients with a grade II LAS compared to those with a grade I LAS were attributed to more severe trauma, likely increasing vulnerability to further trauma. The reason for lower recurrent injury rates in patients with a grade III LAS was unclear, but the authors postulated that patients with grade III injuries might receive more comprehensive care due to the extensive damage incurred. Additionally, their recovery required more activity time-loss, and thus, may have reduced the likelihood of recurrent injury within 2 years.

Chronic Ankle Instability Prediction

Similar to the aims of Malliaropoulos et al.,[51] several investigators have utilized clinical outcomes to predict the development of CAI or CAI-like characteristics in individuals following an acute LAS. Doherty et al.[53] conducted the only prospective study designed to predict those that subsequently develop CAI under the contemporary definition described by the International Ankle Consortium.[12-14] Eight-two patients with a first-time LAS underwent evaluation with a battery of self-reported ankle function and stability questionnaires, laboratory-based biomechanical tests, and clinically-applicable functional performance tests 2 weeks, 6 months, and 12 months post-injury. Logistic regression models were utilized to determine the ability of the various outcomes to predict classifications of CAI or LAS coper at 12 months. At 2-weeks, the inability to perform jumping and landing tasks was associated with greater odds of developing CAI. At 6-months, lower scores on the FAAM-ADL and SEBT posterolateral reach were associated with greater odds of developing CAI.

Gerber et al.[55] conducted evaluations of 96 military cadets following an ankle sprain. They were unable to predict CAI under its current definition, as they conducted the study before the development of the contemporary CAI definition. However, the investigators attempted to determine associations between clinical outcomes immediately following injury and chronic dysfunction 6 months post-injury. Within 24 hours of injury, each cadet was evaluated by the mechanism of injury, ankle injury history, pain (VAS), physical function (VAS), joint stability (anterior drawer, talar tilt, squeeze, and external rotation tests), ROM, muscular strength, swelling, and palpation. Favorable outcomes at 6 months coincided with an absence of pain, complete return of self-reported

function, and a functional hop test score within 20% of the contralateral limb's score. They found the factor most closely associated with chronic dysfunction was involvement of the syndesmosis.

As done in the previous study,[55] O'Connor et al.[54] aimed to predict recovery (as opposed to operationally defined CAI) at 4 weeks and 4 months in 85 patients with an acute ankle sprain. Potential predictor variables consisted of age, BMI, mechanism of injury, ankle injury history, weight-bearing status, medial joint-line pain, pain on the WBLT and lateral hop test. The primary outcome was the score on the Karlsson, a survey instrument designed to assess perceived ankle function. They found that 34% of ankle function at 4 weeks was explained by higher age, injury grade, and weight-bearing status at baseline. Additionally, 20% of ankle function at 4 months was explained by higher age, weight-bearing status, and mechanism of injury at baseline. Lastly, 49% of ankle function at 4 months was explained by pain on the WBLT and medial joint-line pain at 4 weeks.

Another study[50] examined differences in pain, mobility, and instability of 15 children with varying grades of acute ankle sprains, determined by magnetic resonance imaging. Final evaluations were conducted 8 months following the acute injury. As a secondary analysis, the authors found no differences in the clinical outcomes existed between patients with grade 2 and grade 3 ankle sprains at the final follow-up.

Pourkazemi et al.[52] examined several aforementioned studies[24,27,50,51] in a systematic review aiming to identify predictors of CAI following an initial acute ankle sprain. Pooled data from two studies using balance and perceived instability as predictor variables could not identify a significant prediction model for recurrent ankle sprain.

Data from the other two studies suggested that injury severity explained 7-11% of recurrent injury status. Patients with a grade 2 sprain had approximately 2.6 times greater odds of sustaining a recurrent sprain than patients with a grade 1 or 3 injury. However, the authors cautioned the interpretation of that finding, as the grading system varied between studies, and concerns regarding validity were present. This systematic review, like other aforementioned studies, attempted to predict those who will develop CAI after an acute ankle sprain, but the primary outcome variable was recurrent injury. Although recurrent injury is included in the CAI definition, it does not encapsulate the entire classification, and thus, may further limit the utility of the prediction models.

Chapter 3: Acute Lateral Ankle Sprain Prediction in Collegiate Women's Soccer Players

INTRODUCTION

Over 488,000 student-athletes participated in National Collegiate Athletic Association (NCAA) sponsored sports during the 2014-2015 academic year, approximately 43% of which were females.[4] While the NCAA's participation rate has risen annually, sport-related injury rates have remained steady,[185] likely leading to a greater total number of injuries. Across all collegiate women's sports, soccer has the highest injury rate during competition,[7] with the lower extremity accounting for approximately 70% of the total injuries.[185] Lateral ankle ligament sprains (LASs) are the most commonly reported injuries, resulting in 10 or more days of activity loss in collegiate women's soccer.[185] Along with time loss, there is added concern for recurrent injury,[186] decreased neuromuscular control,[18,115,116] decreased physical activity,[17,18] decreased health-related quality of life,[17] and post-traumatic osteoarthritis[19] in individuals with a history of LAS.

Prevention of LAS and subsequent long-term consequences may be accomplished through training programs designed to enhance neuromuscular control. Specifically in athletic populations, the use of neuromuscular training protocols has previously demonstrated effectiveness for preventing LASs.[21] However, a numbers needed to treat analysis performed by McKeon and Hertel[187] found that up to 44 athletes were required to undergo training in order to prevent one LAS. While successful injury prevention is likely achievable, prospective determination of which participants are at greater risk for an acute lower extremity injury likely enhances the efficiency of neuromuscular training protocols, as those at greater risk may have a greater degree of responsiveness.[188]

Furthermore, risk assessment will perhaps identify individuals' specific impairments, which clinicians can target through neuromuscular training interventions.

Previously, investigators have produced prediction models for LAS risk in athletic populations with the use of the Star Excursion Balance Test (SEBT).[41,182] The SEBT is a multi-directional lower extremity reaching task, typically used to test dynamic postural control.[119] Previous findings suggest the SEBT may be an effective predictor of injuries when simplified to just the anterior reach (SEBT-ANT).[41,182] Plisky et al.[182] found value in the SEBT composite score as well as the SEBT-ANT reach as individual predictors of general lower extremity injuries in high school basketball players. Gribble et al.[41] reported that SEBT-ANT performance was the most useful SEBT component for the prediction of LASs in high school and collegiate football players. Furthermore, Thorpe and Ebersole[189] suggested that SEBT-ANT performance is a useful tool for assessing the effectiveness of a prevention program and tracking improvement of dynamic postural control in collegiate women's soccer players. However, to our knowledge there is limited evidence reporting LAS prediction capability of SEBT-ANT performance in collegiate women's soccer players.

Measures of isometric hip strength have also demonstrated utility for lower extremity injury prediction in collegiate athletes.[190,191] Specifically, investigators have identified isometric hip abduction and external rotation as valuable injury predictors,[190,191] as they are likely influential to neuromuscular control in the frontal and transverse planes, respectively. Reduced isometric hip extension strength (HEXT) has recently been identified as a predictor of LAS in youth soccer players.[48] As the gluteus maximus functions in multi-planar lower extremity neuromuscular control,[192-195] HEXT

may efficiently and broadly represent posterolateral hip muscular strength and the ability to position the entire lower extremity to avoid injury.

In addition to isolated and functional performance tests, simple measures of height and mass have demonstrated usefulness in LAS prediction models.[2,31,34,40,41,43] Greater body mass index (BMI), calculated from height and mass, likely increases the body's moments of inertia and reduces an individual's ability to resist external forces.[43] Due to the simplicity of their measurement, demographics are viable co-variates for any injury prediction analysis. No previous investigators have developed a model of LAS risk for collegiate women's soccer players, but the SEBT-ANT, HEXT, and demographics may possess potential injury prediction value for that population. Therefore, the purpose of this study was to develop a prediction model for acute LAS injuries in collegiate women's soccer players, utilizing primary outcomes of SEBT-ANT and HEXT as well as secondary demographic outcomes as potential predictors.

METHODS

Participants

A convenience sample of 26 NCAA Division I women's soccer players from a single university volunteered for participation in this prospective cohort study. We conducted the study over two consecutive fall collegiate soccer seasons. Fourteen of the participants were on the rosters both seasons, each accounting for two separate entries (28 total) in the full sample. Fifteen participants each accounted for a single entry, bringing the total sample of cases examined during the two years to 43 (19.7 ± 1.1 years, 166.8 ± 3.7 cm, 60.8 ± 4.4 kg). Inclusion criteria consisted of full medical clearance for participation in sporting activities. Within one week prior to the beginning of pre-season

practices, each participant reported for testing in the university athletic training facility. Each participant reviewed and signed an informed consent document approved by the university institutional review board.

Procedures

Following study enrollment, each participant underwent bilateral testing of the SEBT-ANT and HEXT. Participants accounting for two entries repeated testing at the beginning of the second year. Height (cm) and mass (kg) were measured using a standard physician beam scale (Detecto 339 Eye Level Physician Scale; Detecto Scale Company; Webb City, MO). We calculated body mass index (BMI) from the participant height and mass (kg/m^2). The order of testing limbs and task performance was randomized.

Star Excursion Balance Test Anterior Reach Assessment

First, participants' leg length (cm) were measured from the anterior superior iliac spine to the distal end of the lateral malleolus for each limb. The examiner then instructed each participant how to perform the SEBT-ANT (Figure 3.1). Participants were required to maintain a single-leg stance, with the distal end of the second toe placed at the 0 mark of a metric tape measure adhered to the floor. While maintaining the stance heel flat against the floor and hands on hips, participants reached for maximum distance with the non-stance limb in the anterior direction. Participants were allotted four practice trials,[196] followed by three test trials. After a 1-minute rest interval, the second limb underwent testing in the same manner. The average of three trials for each limb was normalized as a percentage of stance leg length (%LL) and utilized for statistical analysis.

Previous studies[119,197,198] reported excellent intrarater reliability (ICC = 0.89-0.95) and good to excellent interrater reliability (ICC = 0.76-0.89) for the SEBT-ANT.

Isometric Hip Extension Strength Assessment

Participants lay prone on a treatment table with hips in a neutral position and the knee of the test limb flexed to 90°. The examiner placed a hand-held dynamometer (MicroFET 2, Hoggan Health Industries, Inc, West Jordan, UT) over the posterior thigh, 5.08 cm proximal to the lateral knee joint line (Figure 3.2).[199] Peak HEXT assessment occurred as participants extended their hip, gradually ramping up intensity for the first three seconds, then giving maximum effort for the fourth and fifth seconds. The examiner maintained the dynamometer position manually. A single practice trial preceded three test trials, with 30-second rest intervals between trials. After a 1-minute rest interval, the second limb underwent assessment in the same manner. We averaged peak torque across three test trials (kg) for each limb and normalized it as a percentage of body mass (%BM). Excellent test-retest reliability (ICC = 0.99) has been previously demonstrated for HEXT averaged across 3 trials.[200] A similar variation of isometric hip extension strength assessment demonstrated good to excellent intrarater reliability (ICC = 0.77-0.93) and good interrater reliability (ICC = 0.65).[201]

Throughout the course of the subsequent soccer season, the certified and licensed athletic trainer (AT) responsible for providing care to the team recorded LAS injuries sustained by the participants. A LAS must have 1) occurred during a team practice or competition, 2) required care by medical personnel, and 3) resulted in at least one day of missed soccer activity. The team AT facilitated baseline data collection, but was blinded to baseline performances of each participant.

Statistical Analysis

The involved limb of participants who sustained a LAS injury during the course of the season was included in the statistical analysis. A randomly selected limb of each uninjured participant underwent statistical analysis. Independent t-tests and Cohen's *d* effect sizes with 95% confidence intervals (CIs) compared the physical function tests (SEBT-ANT and HEXT) and demographics (age, height, mass, and BMI) between injured and uninjured participants. Effect sizes were interpreted as small: $d = 0.20 – 0.49$, moderate: $d = 0.50 – 0.79$, and large: $d \geq 0.80$.[202]

Separate forward binary logistic regression analyses assessed the influence of SEBT-ANT, HEXT, along with any significantly different demographics on the estimated odds of sustaining a LAS. We employed a Receiver Operating Characteristic (ROC) curve to plot the predictive utility (sensitivity vs. 1-specificity) of each value observed for each outcome. From the ROC curve, we obtained the area under the ROC curve (AUROC), a singular quantitative representation of the overall predictive value of each variable, with 95% confidence intervals. The AUROC can range from 0 to 1, with 0.5 representing an absence of predictive power, and 1 representing perfect predictive power.[203] From ROC curves demonstrating predictive utility, we identified cutoff scores that maximized sensitivity and specificity for the predictor variable. We utilized Fisher's exact test to determine the strength of association between the predicted group classification (based on the cutoff score) and the observed injury status. We calculated sensitivity, specificity, positive and negative likelihood ratios (+LR, -LR), and the diagnostic odds ratio (DOR) for cutoff scores. Statistical significance was set *a priori* at

P<0.05. All statistical analyses were conducted using IBM SPSS version 22 (IBM Corporation, Armonk, NY).

RESULTS

Our participants participated in 18 games and 54 practices in the first injury-tracking season, and 21 games and 51 practices in the second injury-tracking season. In that time, 8 participants sustained a LAS. Independent t-tests revealed no statistically significant group differences for age, mass, BMI, SEBT-ANT, or HEXT (Table 1). However, a significant t-test and large effect size indicated the injured group was taller than the uninjured group and was subsequently added to the predictive model with our primary clinical outcome measures (SEBT-ANT and HEXT) in separate forward binary logistic regression analyses (Table 2). Height was the only significant predictor of injury status. The odds ratio suggested that an increase in 1 cm of height was associated with a 30% increase in the odds of sustaining a LAS. The ROC curve analyses further demonstrated moderate predictive utility of height (AUROC = 0.73 [0.58, 0.89]; P = 0.04) (Figure 3.3) and poor predictive utility of SEBT-ANT (AUROC = 0.51 [0.27, 0.75]; P = 0.93) (Figure 3.4) and HEXT (AUROC = 0.62 [0.42, 0.83]; P = 0.29) (Figure 3.5). A cutoff score for height that maximized sensitivity and specificity (167.6 cm) within the ROC curve produced a significant Fisher's exact test (P = 0.05) (Table 3.3). Predicted and actual injury status based on the height cutoff score are in Table 3.4. Associated sensitivity, specificity, +LR, -LR, and DOR calculated from the 2-by-2 contingency table are in Table 3.5. We identified excellent sensitivity (0.88) and low to moderate specificity (0.51) within the model. A favorable DOR (7.5) indicated that

participants with height equal to or greater than 167.6 cm had 7.5 times greater odds of sustaining a LAS than participants less than 167.6 cm in height.

DISCUSSION

The primary finding of this study is that participant height was an effective predictor of LAS among collegiate women's soccer players. Specifically, those athletes equal to or taller than 167.6 cm in height had 7.5 times greater odds of sustaining a LAS than those below 167.6 cm in height. Its predictive value supports previous studies reporting participant height as an effective predictor of ankle injuries. Waterman et al.[2] reported taller military academy cadets were at greater risk of sustaining an ankle sprain. Similarly, Milgrom et al.[43] found that taller infantry recruits were more prone to LASs. They postulated that taller stature may contribute to larger moments of inertia in the lower extremity.[43] Essentially, longer trunk and extremity segments may reduce the ability of an individual to resist external moments exerted on the body, potentially increasing injury risk.[43] The aforementioned studies[2,43] only found associations between height and injury in male participants, but our findings suggest height may also be pertinent to LAS risk in females. While greater height may be relevant to LAS risk, its lack of modifiability limits applicability to injury prevention strategies. Elevated body mass can also increase moments of inertia, but the lack of differences between injured and uninjured participants suggests body mass had little influence over injury risk in this population. Furthermore, the lack of body mass differences likely limited the ability of BMI to differentiate those that did and did not sustain a LAS.

Surprisingly, SEBT-ANT performance failed to demonstrate predictive utility for LAS in this sample. Plisky et al.[182] examined a cohort of over 200 high school basketball

players and reported that participants with side-to-side SEBT-ANT differences over 4 cm had more than two times greater estimated odds of sustaining a lower extremity injury. Gribble et al.[41] conducted a study of over 600 football players and identified significant differences and a moderate effect size (d = 0.55) for SEBT-ANT between those who did and did not subsequently sustain a LAS. Additionally, the previous study found that athletes with an SEBT-ANT score below 67.2% had nearly three times greater estimated odds of sustaining a LAS compared to those with scores greater than or equal to 67.2%.[41] In the current study, we found that the average SEBT-ANT scores for our injured and uninjured groups were both greater than the cutoff score previously suggested by Gribble et al.[41] Athletes of varying sports and levels of competition have previously demonstrated differing SEBT-ANT performance,[45-47] suggesting the SEBT-ANT may have varying predictive value for LASs among different athletic populations (i.e. male football players vs. female soccer players). Thus, investigators may need to establish predictive value of the SEBT-ANT for specific populations in the future. Another possible explanation for our inability to produce a robust LAS prediction model with the SEBT-ANT was the small sample (n = 43) compared to those of previous studies.[41,182]

Like the SEBT-ANT, HEXT also exhibited poor predictive utility for LAS in collegiate women's soccer players. Hip extensor strength is influential to multi-planar hip alignment,[194] which may subsequently affect multi-planar position and injury risk of more distal joints.[190,191] Thus, we expected athletes with lower HEXT to be predisposed to LASs. Recently, de Ridder et al.[48] conducted a prospective assessment of isometric hip strength in 133 male youth soccer players. They reported that athletes with HEXT less than the sample average sustained LASs 10% earlier than those with HEXT greater

than the sample average. They postulated that athletes with reduced HEXT may be less able to dissipate impact forces during functional tasks, potentially directing that force to non-contractile structures, such as the ankle ligaments. Conversely, McHugh et al.[29] conducted baseline isometric hip strength assessments in 169 high school athletes and found that no measure possessed predictive value for ankle sprains. While their results support our findings, they utilized only measures of hip flexion, abduction, and adduction strength in their study, and thus, they could not confirm nor deny the predictive value of HEXT. Similar to our findings for SEBT-ANT, we must consider the potential effect of differing population characteristics and sample sizes on the predictive utility of HEXT. Future studies should continue to explore the effectiveness of HEXT and SEBT-ANT for predicting LASs in various athletic populations with greater sample sizes.

Clinical Implications

Among collegiate women's soccer players, the SEBT-ANT and HEXT may lack the ability to predict those who will sustain a LAS. However, a simple measure of participant height may effectively predict injury status. Clinically, the strength of height as a LAS predictor is its ease of assessment, but it is clearly limited by its lack of modifiability. Although height itself is not malleable, this simple demographic characteristic may be an important catalyst for targeted intervention. For example, preventative measures such as prophylactic ankle supports and postural control training are viable options for LAS prevention,[204,205] and perhaps may be particularly valuable for taller athletes. While prophylactic ankle supports and postural control training are associated with significant cost and time demands, respectively, identification of a strong

risk factor (i.e. height) will allow clinicians to allocate preventative resources to those with the greatest predisposition to LAS.

Limitations

Certain notable limitations are present within this study. First, our study was specific to NCAA Division I women's soccer players and may not be applicable to those participating in other sports and levels of competition. Furthermore, the sample of convenience population was potentially small (observed power for comparisons of group means < 0.20 for SEBT-ANT and HEXT), raising the possibility of type II error. Lastly, we focused on a limited collection of potential predictor variables (SEBT-ANT, HEXT, and demographics). Examining additional SEBT reach directions (i.e., posteromedial and posterolateral), multiple measures of hip strength (i.e., flexion, abduction, external rotation), and other performance measures (i.e., flexibility) may provide more insight into the evolution of these prediction models.

CONCLUSIONS

Participant height demonstrated predictive value for LAS among collegiate women's soccer players, whereas SEBT-ANT and HEXT did not. Longer trunk and segment lengths may impair an athlete's ability to resist external forces, potentially increasing the likelihood of sustaining a LAS. Clinicians should consider collegiate women's soccer players for interventions designed to prevent LAS.

Table 3.1. Comparisons of Demographics, SEBT-ANT, and HEXT between Injured and Uninjured Participants

	Injured (n=8)	**Uninjured (n=35)**	**Independent T-Test**	**Cohen's *d* (95%CI)**
Age (years)	19.8 ± 1.2	19.6 ± 1.1	t_{41} = -0.27, P = 0.79	0.18 (-0.59, 0.94)
Height (cm)	169.2 ± 2.3	166.3 ± 3.7	t_{41} = -2.87, **P = 0.01**	**0.83 (0.03, 1.60)**
Mass (kg)	60.7 ± 6.1	60.6 ± 4.1	t_{41} = 0.05, P = 0.96	0.02 (-0.75, 0.79)
BMI (kg/m^2)	21.2 ± 2.2	22.0 ± 1.5	t_{41} = 1.23, P = 0.22	-0.49 (-1.25, 0.30)
SEBT-ANT (%LL)	68.5 ± 6.3	69.0 ± 6.3	t_{41} = -0.20, P = 0.84	-0.08 (-0.85, 0.69)
HEXT (%BM)	42.3 ± 6.3	44.5 ± 7.8	t_{41} = 0.76, P = 0.45	-0.29 (-1.06, 0.48)

Abbreviations: BMI = body mass index; SEBT-ANT = star excursion balance test anterior reach (normalized to a percentage of stance leg length [%LL]); HEXT = isometric hip extension strength (normalized to a percentage of body mass [%BM])

Table 3.2. Separate Binary Logistic Regression Analyses

Variables	Odds Ratio (95%CI)	P-Value
Height	**1.30 (1.00, 1.70)**	**0.05**
SEBT-ANT	1.01 (0.89, 1.15)	0.84
HEXT	0.96 (0.87, 1.06)	0.44

Table 3.3. Fisher's Exact Test for Height

Height (cm)	LAS	No LAS
≥ 167.6	7	17
< 167.6	1	18

Table 3.4. Diagnostic Statistics of Height Cutoff Score (167.6 cm)

Quantities	Formula		Results
Sensitivity	true positive/(true positive + false negative)	7/8	0.88
Specificity	true negative/(true negative + false positive)	17/35	0.51
+LR	sensitivity/(1-specificity)	0.88/0.49	1.80
-LR	(1-sensitivity)/specificity	0.12/0.51	0.24
DOR	+LR/-LR	1.89/0.21	7.50

Abbreviations: +LR = positive likelihood ratio; -LR = negative likelihood ratio; DOR = diagnostic odds ratio

Figure 3.1. Star Excursion Balance Test Anterior Reach (SEBT-ANT)

Figure 3.2. Isometric Hip Extension Strength (HEXT)

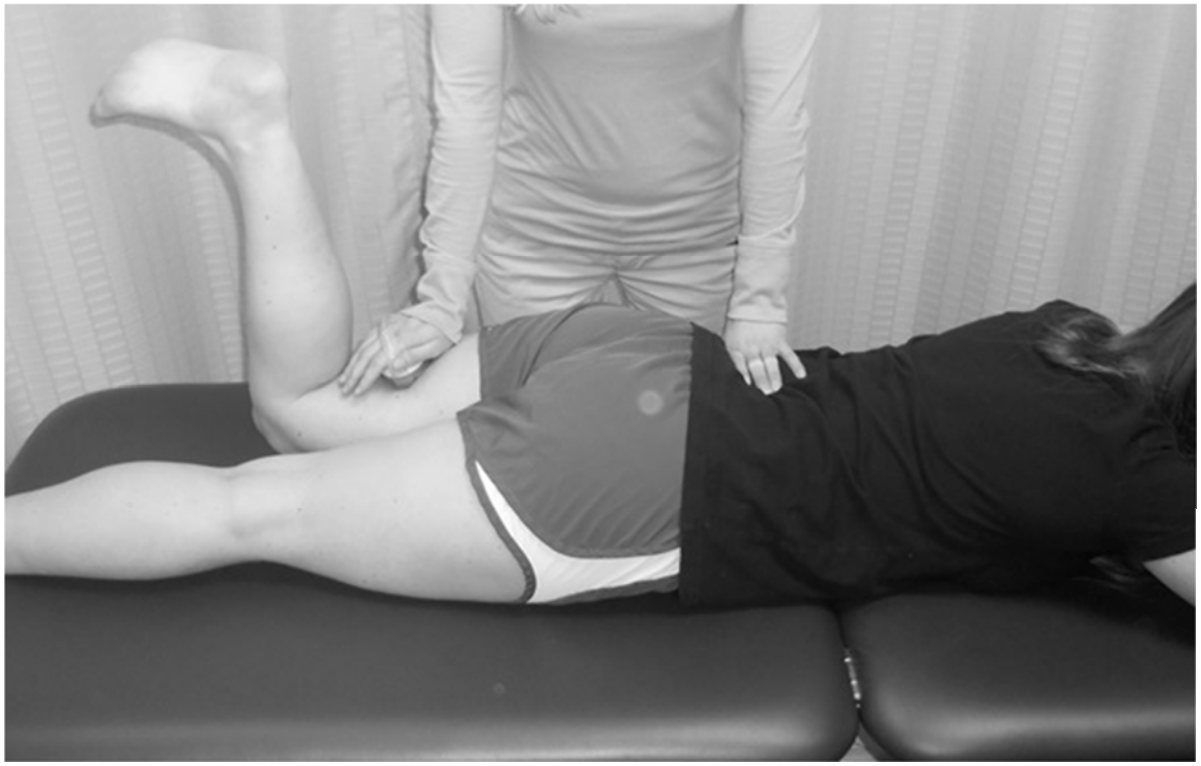


Figure 3.3. Height ROC Curve

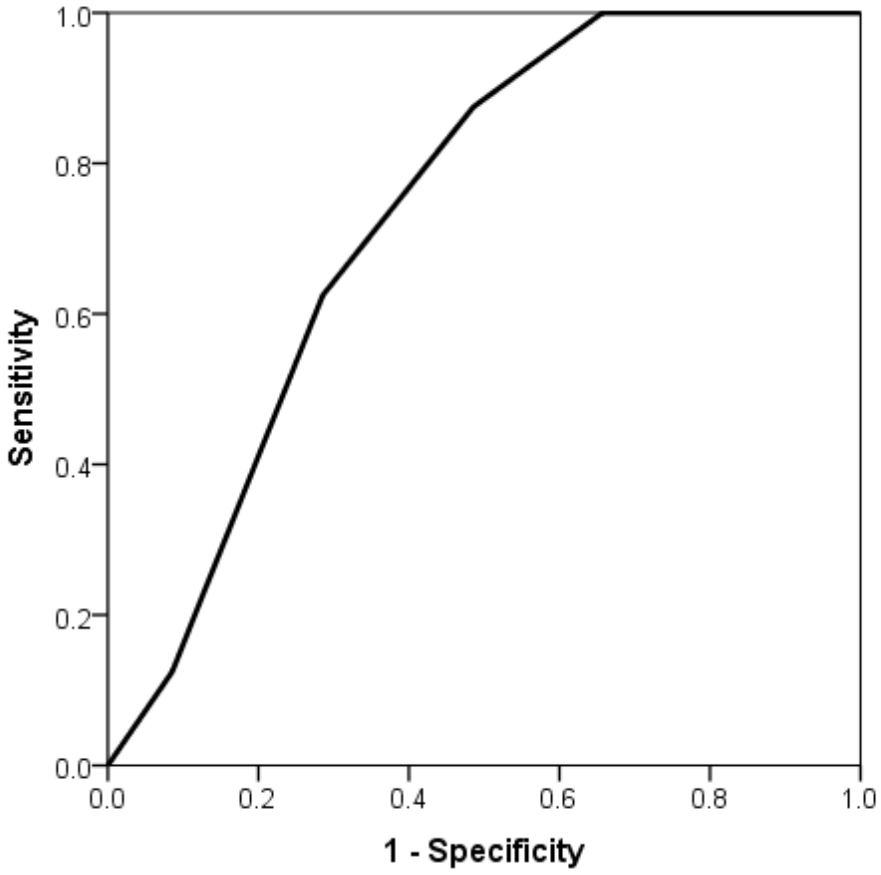


Figure 3.4. SEBT-ANT ROC Curve

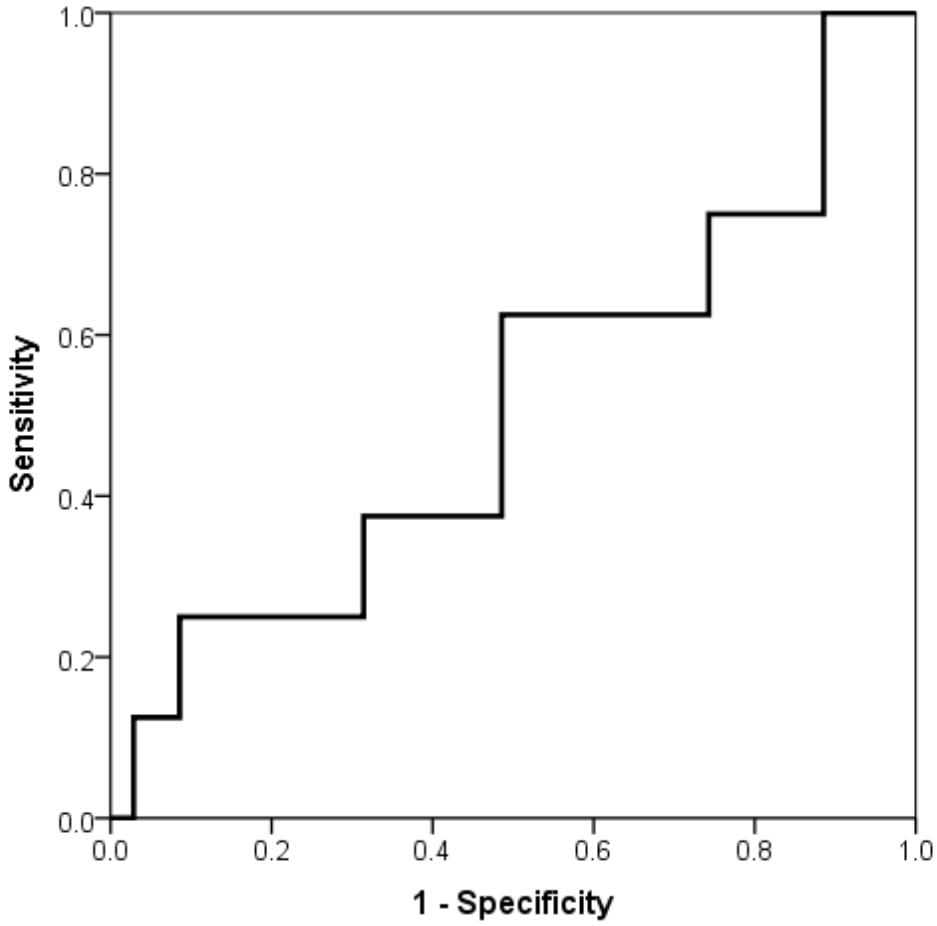
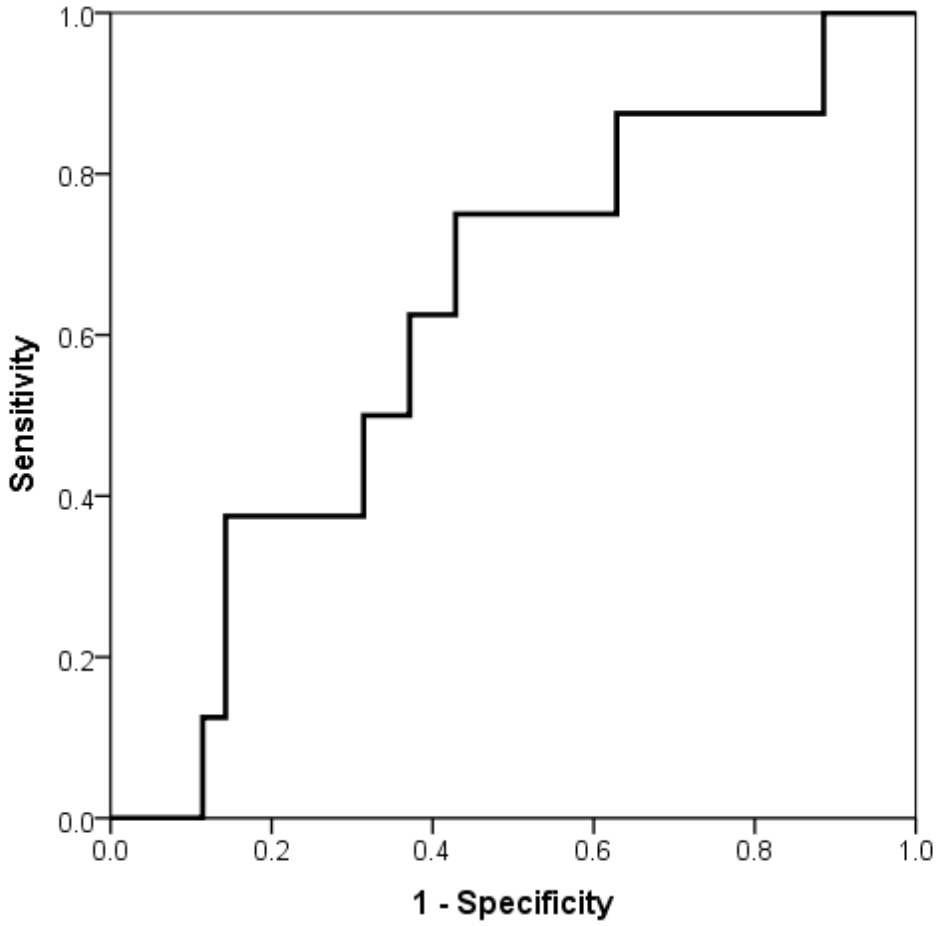


Figure 3.5. HEXT ROC Curve



Chapter 4: Residual Impairments and Activity Limitations at Return to Play from a Lateral Ankle Sprain

INTRODUCTION

Athletic activity is the most common source of ankle sprains, accounting for nearly 50% of these injuries.[2] Over 326,000 ankle injuries occur annually among US high school student-athletes,[5] with ankle sprains accounting for over 80% of all ankle injuries.[6] In collegiate athletics, ankle ligament injuries account for up to 15% of all injuries.[7] Among all ankle ligament sprains, as many as 96% will be classified as a lateral ankle sprain (LAS).[9]

The sequelae of a LAS usually consist of structural and functional impairments as well as activity limitations. Varying combinations of perceived instability, pain, edema, decreased ankle range of motion (ROM), ankle ligamentous laxity, and dynamic postural control deficits are commonly present in the acute stages of LAS recovery. Over 70% of patients with a history of LAS experience at least one residual symptom six months to four years after injury.[11,15] The significant concern regarding these residual sequela is intensified by widespread development of chronic ankle instability (CAI), marked by recurrent LAS, perceived instability, and "giving way" episodes for at least six months following an index LAS.[12-14] Medina McKeon et al.[49] reported the median time for RTP was three days for a first-time LAS and one day for a recurrent LAS. Subsequently, it is likely that a large proportion of patients with a LAS resume pre-injury activities before associated impairments are expected to be resolved.

The long-term consequences following a LAS are perhaps more common than many clinicians realize, and it is possible that RTP often occurs before all impairments have fully resolved. However, it remains unclear which outcomes are consistently

deficient following clearance for RTP. As RTP represents a critical time in which injured athletes resume high-risk activity, identifying which impairments and activity limitations most consistently last beyond RTP may offer information regarding potential factors that cause some patients to sustain recurrent injuries and develop CAI. Therefore, the purpose of this study was to describe the presence of residual structural and functional impairments, as well as activity limitations, in athletes with an acute LAS following clearance for RTP. We hypothesized that athletes with an acute LAS would exhibit significant impairments in the involved ankle compared to the uninvolved ankle at RTP. Additionally, we expected patients to self-report meaningful degrees of activity limitations relative to accepted norms at RTP. To account for a potential influence from previous LAS history in the uninvolved limb on limb-to-limb comparisons, we aimed to assess differences in outcomes in the uninvolved limbs between participants with and without a previous LAS.

As the degree of patients' residual sequelae may be dependent on injury severity and care provided by clinicians, we included secondary purposes, comparing outcomes between patients with higher and lower injury severity and exploring associations between the number of days of immobilization and rehabilitation following the acute LAS and the degree of impairment and activity limitation. For this secondary aim, we hypothesized that patients with lower injury severity and more days of immobilization and supervised therapeutic exercise sessions would demonstrate lower structural and functional impairments and activity limitations at RTP.

METHODS

Participants

As a sample of convenience, we recruited 50 patients (F=15, M=35; 17.6±3.6yrs; 178.3±11.5cm; 85.3±27.0kg) with an acute LAS from athletic training facilities of local high schools and colleges. Inclusionary criteria consisted of the presence of an acute LAS that occurred during organized sporting activity, was evaluated by an AT, and resulted in at least one day of activity time-loss. Exclusionary criteria consisted of the presence of fracture, the presence of additional lower extremity injuries, or surgical treatment for the injury. Participants over the age of 18 read and signed an informed consent document approved by the university institutional review board. Participants under 18 provided assent, while a parent or legal guardian signed the informed consent document.

Procedures

We utilized a case series study design to assess structural and functional impairments and activity limitations of student-athletes at RTP following a LAS. After an athlete sustained a LAS, the treating AT dictated decisions for care at RTP of each patient. While the treating ATs reported some heterogeneous RTP criteria across clinical sites, "mild to no pain during running" was a consistent minimal standard utilized for RTP by all of the treating clinicians. The treating AT contacted the primary investigator to conduct an independent clinical evaluation of the following primary outcome measures within 48 hours of RTP: self-reported physical function, ankle joint pain, edema, ankle dorsiflexion ROM, ankle ligamentous laxity, and dynamic postural control. The independent evaluations took place in the athletic training facility of each patient's school. The treating AT provided documentation of secondary outcome measures of

injury grade, previous injury history, days to return-to-play (DRTP), days of immobilization, and sessions of therapeutic exercise.

Injury Grade

We provided the treating ATs with a grading scale to utilize during the initial injury evaluation.[206,207] A LAS presenting with little to no pain and swelling, and minimal loss of weight bearing ability and mechanical stability received a severity grade of "1". A LAS presenting with moderate pain and swelling, and moderate loss of weight bearing ability and mechanical stability received a severity grade of "2". A LAS presenting with severe pain and swelling, and severe loss of weight bearing ability and mechanical stability receive a severity grade of "3".

Previous Injury History

We recorded the quantity and date of previous LASs for each participant. If medical documentation was unavailable for review, we asked patients to self-report previous injuries.

Self-Reported Physical Function Assessment

At RTP, we assessed self-reported physical function of the involved limb using the Foot and Ankle Ability Measure activity of daily living (FAAM) and sport (FAAM-S) subscales, each reported as a percentage.[144]

Ankle Joint Pain Assessment

Each participant reported pain in the ankle region with a 100 mm visual analogue scale (VAS). We used three conditions for pain assessment: non-weight-bearing, single-leg stance, and four walking steps. A previous study of post-operative patients reported

that pain measured with a VAS could be interpreted as none (< 5/100), mild (5-44/100), moderate (45-74/100), and severe (> 74/100).[208]

Edema Assessment

We conducted bilateral figure-of-eight girth measurements to assess edema accumulation (Figure 4.1).[209] The examiner placed the patient's ankle in neutral dorsiflexion and instructed the participant to maintain that position. The examiner then wrapped a tape measure around the ankle, beginning midway between the tibialis anterior tendon and the lateral malleolus. The tape measure tracked across the anterior ankle and passed just distally to the navicular tuberosity. After the tape measure passed under the plantar aspect of the foot, it passed just proximally to the base of the fifth metatarsal. The tape measure continued across the anterior aspect of the ankle, and wrapped around the shank just distally to the medial and lateral malleoli. After passing the lateral malleolus, the figure-of-eight concluded at its starting point. The total distance in centimeters represented the girth of the measured ankle.

Ankle Dorsiflexion ROM Assessment

We conducted bilateral weight-bearing lunge tests (WBLT) to assess ankle dorsiflexion ROM (Figure 4.2).[210] The patient faced the wall with the hallux and heel of the test limb in-line on top of a tape measure fixed perpendicular to the wall. The patient lunged forward, keeping the foot flat against the floor, and touched the anterior aspect of the knee to the wall. We allowed the patient to place their hands on the wall and non-test limb on the floor for support. If the patient successfully touched their knee to the wall, we incrementally moved the foot away from the wall, and repeated the test. The goal of the test was to contact the anterior knee to the wall with the hallux at the furthest distance

possible, indicating a greater degree of dorsiflexion ROM. We permitted up to five trials to determine maximum performance.

Ligamentous Laxity Assessment

We utilized anterior drawer and talar tilt tests to assess lateral ankle ligament laxity.[32] We conducted the anterior drawer test with the ankle in approximately 10° of plantar flexion. The examiner grasped the distal lower leg with one hand and the posterior calcaneus with the other. While stabilizing the lower leg, the examiner exerted an anteriorly directed force on the heel and assessed the degree of laxity during anterior talar translation.[32,92] We conducted the talar tilt with the ankle in a neutral sagittal plane position. The examiner grasped the distal lower leg with one hand and the inferior calcaneus with the other. While stabilizing the lower leg, the examiner inverted the ankle and assessed the degree of lateral joint laxity. We graded laxity in both tests on a four-point scale: 0=no laxity, 1=mild laxity, 2=moderate laxity, and 3=severe laxity.[92]

Dynamic Postural Control Assessment

We utilized the anterior reach of the star excursion balance test (SEBT-ANT) to assess dynamic postural control (Figure 4.3).[119] We selected the SEBT-ANT due to its superior efficacy for LAS prediction compared to the other reach directions.[41] The patient maintained a single-leg stance on the test limb while reaching for maximum distance in the anterior direction with the non-test limb. The examiner instructed the patient to gently touch the tape measure with the most distal aspect of the reaching limb while maintaining a single-leg base of support on the test limb, and then return to double-leg stance. The patient's hands were required to remain on their hips and the stance heel was required to remain in contact with the floor. Four practice trials were performed,[196]

followed by three test trials. We normalized the average SEBT-ANT score for each limb as a percentage of stance leg length, measured from the ASIS to the distal end of the medial malleolus.[119]

Days to Return-to-Play

The total number of days from the onset of injury until the participant returned to unrestricted sporting activity as determined by the treating AT represented DRTP. We did not provide guidelines for RTP decisions to the treating ATs.

Immobilization

We recorded the utilization of devices such as crutches, walking boots, and splints that limited use of the injured ankle as the total number of days in which at least one device was in use.

Rehabilitation

We recorded the total number of therapeutic exercise sessions conducted under direct supervision of a health care professional.

Statistical Analysis

We compared figure-of-eight girth, WBLT, and SEBT-ANT scores between limbs using paired t-tests. Cohen's *d* effect sizes (weak [$0.2 \leq d < 0.5$], moderate [$0.5 \leq d < 0.8$], strong [$d > 0.8$]) and 95% confidence intervals assessed the magnitude of differences.[202] We compared ligamentous laxity between limbs using a non-parametric Wilcoxon Signed Rank test. As a consideration for a potential influence of a history of previous LAS in the uninvolved limb on between-limb comparisons, we assessed differences figure-of-eight girth, WBLT, and SEBT-ANT in the uninvolved limbs between participants with and without a previous LAS with separate independent t-tests.

To explore the potential impact of varying injury severity on the primary outcomes, we conducted independent t-tests of continuous primary outcomes between patients with a low (grade 1) and high (grade 2-3) LAS severity. Mann-Whitney U tests compared ligamentous laxity between patients with low and high grade injuries. To explore the potential impact of varying treatment strategies on the primary outcomes, we assessed the associations of days of immobilization and the number of rehabilitation session with pain, FAAM scores, FAAM-S scores, and limb-to-limb differences of the figure-of-eight girth measurement, WBLT, and SEBT-ANT with Pearson product moment correlations and associated coefficients of determination. Larger limb-to-limb differences equated to greater degrees of swelling and worse WBLT and SEBT-ANT scores in the involved limb. We compared days of immobilization and the number of clinician-supervised rehabilitation session between those with varying injury grades and ligamentous laxity using separate non-parametric Kruskal-Wallis tests. In the event of a significant Kruskal-Wallis test, we utilized Mann-Whitney U tests for pairwise comparisons. Significance was set *a priori* at $P<0.05$. All statistical analyses were conducted with IBM SPSS Statistics, version 22 (IBM Corporation, Armonk, NY).

RESULTS

We evaluated 50 patients with a LAS from 10 different clinical sites. Among 50 patients, 29 competed in high school athletics and 21 competed in collegiate athletics. Frequencies of LAS by sport are reported in Table 4.1. Eighteen patients (36%) had at least one previous LAS on the involved limb, the most recent of which occurred on average 21.8±26.0 months previously. Seven patients (14%) had at least one previous LAS on the uninvolved limb, the most recent of which occurred on average 18.4±11.4

months previously. All patients reported mild to no pain during running as a common minimal standard for RTP.

The average DRTP for the entire cohort was 12.7±10.0 days (Table 4.2). Descriptive statistics of each primary and secondary outcome variable are presented in Table 4.3. At the time of RTP, twenty-nine participants (58%) had FAAM scores below 90%, and 36 (72%) had FAAM-S scores below 80%, thresholds of which are consistent with self-reported function of individuals with CAI.[12-14] Twenty-six patients (52%) reported both a FAAM score below 90% and a FAAM-S score below 80%. Thirty-five patients (70%) reported the presence of pain in at least one of the tested conditions. Sixteen patients (32%) reported pain during the non-weight-bearing condition, 34 (68%) reported pain during the single-leg stance condition, and 30 (60%) reported pain during the walking condition. The vast majority of patients (96%) reported none to mild pain during the three conditions (range=0-41/100). Only two patients reported moderate pain; one during the single-leg stance condition (49/100), and the other during the walking condition (52/100).

Patients had significantly greater figure-of-eight girth measurements (t_{49}=5.51, P<0.01, d=0.16 [-0.23, 0.55]), and significantly lower WBLT (t_{49}=-7.14, P<0.01, d=-0.61 [-1.01, -0.21]) and SEBT-ANT scores (t_{49}=-4137, P<0.01, d=-0.46 [-0.86, -0.06]) on the involved limb compared to the uninvolved limb. Patients had significantly greater ligamentous laxity with the anterior drawer test on the involved limb compared to the uninvolved limb (Z=-3.36, P<0.01), but did not demonstrate limb-to-limb differences with the talar tilt test (Z=-1.67, P=0.10).

No significant differences in figure-of-eight girth (t_{48}=0.96, *P*=0.34, *d*=0.39 [-0.42, 1.19]), WBLT (t_{48}=-0.08, *P*=0.94, *d*=-0.05 [-0.85, 0.75]), and SEBT-ANT (t_{48}=-0.13, *P*=0.89, *d*=-0.05 [-0.85, 0.75]) existed between patients with and without a previous history of LAS on the uninvolved limb.

Twenty-five injuries (50%) received a severity grade of "1", 24 (48%) received a grade of "2", and one (2%) received a grade of "3". Comparisons of low and high LAS grades demonstrated that patients with a grade 1 LAS had lower figure-of-eight girth asymmetries and WBLT asymmetries compared to those with a grade 2 or 3 LAS (Table 4.4). Mann-Whitney U tests indicated that ligamentous laxity did not differ between patients with higher and lower LAS severity on anterior drawer (Grade 1: 1.0±0.5, Grade 2: 1.3±0.6, Z = -1.14, P = 0.26) or talar tilt tests (Grade 1: 1.1±0.4, Grade 2: 1.3±0.5, Z = -1.15, P = 0.25). No other primary outcomes differed among patients with low and high injury grades.

Thirty-eight patients (76%) utilized at least one immobilization device, and 15 participants (30%) utilized multiple devices. Nineteen patients (38%) utilized crutches, 21 (42%) utilized a walking boot, 13 (26%) utilized a semi-rigid brace. Correlation analyses indicated that increased days of immobilization was associated with increased ankle joint swelling and SEBT-ANT asymmetries, and decreased WBLT asymmetry at RTP (Table 4.5). Days of immobilization did not differ among those with varying joint laxity on the anterior drawer ($\chi^2_{(3)}$=1.13, *P*=0.77) or talar tilt test ($\chi^2_{(2)}$=0.50, *P*=0.78), but days of immobilization was different among those with differing injury grades ($\chi^2_{(2)}$=19.06, *P*<0.01). Patients with a grade 1 LAS had significantly fewer days of immobilization compared to those with a grade 2 LAS (Z=-4.13, *P*<0.01).

Forty-six patients (92%) underwent at least one clinician-supervised therapeutic exercise session. An increased number of supervised rehabilitation sessions was associated with higher FAAM scores and increased SEBT-ANT aysmmetry at RTP (Table 4.6). Rehabilitation sessions did not differ among those with varying joint laxity on the anterior drawer ($\chi^2_{(2)}$=3.27, *P*=0.20) or talar tilt test ($\chi^2_{(2)}$=1.34, *P*=0.51). Rehabilitation sessions were different among those with differing injury grades ($\chi^2_{(2)}$=15.71, *P*<0.01). Patients with a grade 1 LAS had significantly fewer rehabilitation sessions compared to those with a grade 2 LAS (Z=-3.74, *P*<0.01).

DISCUSSION

The primary finding of this study was that athletic patients returning to sporting activity following an acute LAS presented with residual disease- and patient-oriented dysfunction. The average DRTP was over 12 days, but that timeframe did not coincide with the resolution of all structural and functional impairments and activity limitations. Early RTP is common among athletes, with reports that 94% high school athletes with have RTP within 10 days post-LAS; but on average LAS patients experience RTP within approximately 3 days.[49] Furthermore, Nelson et al.[5] estimated that over 50% of high school athletes with a LAS reach RTP within 7 days, and 85% reach RTP within 21 days.

Among the clinical sites we drew patients from in the current study, the only criteria for RTP unanimously identified by the treatment clinicians was "mild to no pain during running." Various other criteria related to specific impairments and functional performance measures received inconsistent consideration. Not surprisingly, we identified differences in days of immobilization and the number of supervised rehabilitation sessions between those with varying injury grades. Thus, clinicians are

likely inclined to treat a more severe LAS with greater volumes of protection and therapeutic exercise. However, despite potential consideration for injury severity, a number of our participants' impairments and activity limitations persisted at the time that RTP was designated.

Participants reported marked deficits in their perceived ability to complete activities of daily living and sport-specific tasks, as measured with the FAAM and FAAM-S, respectively. The FAAM and FAAM-S are valid indicators of physical function in those with leg, foot, and ankle injuries.[144] Thus, the deficiencies noted by our participants are attributable to lower extremity musculoskeletal pathology, such as the recent LAS. Furthermore, scores below 90% on the FAAM and 80% on the FAAM-S, both of which were demonstrated by the majority of our participants, are suggested to distinguish those with CAI.[12-14] If left unresolved, this degree of perceived limitations may coincide with the onset of CAI, particularly in individuals resuming high-risk sporting activities.

Also of note, participants' involved limbs had significant deficits in weight-bearing ankle dorsiflexion ROM compared to the uninvolved leg, which was further substantiated by a moderate effect size and confidence intervals that did not cross zero. Although seven participants had a history of LAS on the uninvolved limb, they did not demonstrate statistically different WBLT scores on the uninvolved limb, and thus this likely did not influence limb-to-limb comparisons. Patients with an acute LAS may experience persistent dorsiflexion ROM restrictions for up to four weeks after the initial injury.[99] Aiken et al.[158] tested active dorsiflexion ROM of patients with acute ankle sprains, and discovered restrictions persisted at least four days following emergency

department discharge, but resolved by 30 days following discharge. As our patients' average RTP occurred approximately 12 days post-injury, the residual dorsiflexion ROM restrictions we found agree with previous literature.[99,158] Furthermore, the dorsiflexion ROM displayed in our participants' involved limbs remained less than that previously reported in individuals with CAI.[123] Meanwhile, patients' uninvolved limb dorsiflexion ROM resembled that of healthy controls and LAS copers from that previous study.[123] Persistent dorsiflexion ROM restrictions in those with CAI can negatively impact functional knee and hip mobility,[211] attenuation of ground reaction forces during landing,[211] and dynamic postural control.[212] Sufficient dorsiflexion ROM is also necessary for establishment of a closed-packed position of the ankle and protection of the lateral ankle ligaments during functional tasks. Therefore, continued dorsiflexion ROM restrictions at RTP may warrant concern for recurrent injury and CAI development.

Patients exhibited residual dynamic postural control deficits, indicated by significant limb-to-limb differences in SEBT-ANT scores and a moderate effect size with confidence intervals that did not cross zero. Like the WBLT, uninvolved limb SEBT-ANT performance was not influenced by previous LAS history, and thus, limb-to-limb comparisons we observed are not confounded by the injury history. Postural control may continue to improve for up to four weeks after an acute LAS,[113] supporting our finding that postural control deficits are often unresolved at RTP. Reduced dynamic postural control is a verified risk factor for LAS in high school and collegiate athletes,[41] and has also been demonstrated in individuals with CAI.[119,123] Our participant's involved and uninvolved SEBT-ANT scores both resembled that of individuals with CAI,[117] which may have partially masked the magnitude of dynamic postural control deficits that

actually existed in our patients. Consequently, the balance impairments observed in patients at RTP are reason for further alarm, as they may precede recurrent injury as well as persistent deficits in health-related quality of life.

One of two ligamentous laxity tests (anterior drawer) exposed decreased mechanical stability in the involved limb compared to the uninvolved limb at RTP. Mechanical joint stability may require six to 12 weeks to recover following a LAS,[90] substantiating our finding of increased ligamentous laxity after a 12-day average time-loss. Increased laxity observed during the anterior drawer test is generally considered a sign of damage to the anterior talofibular ligament (ATFL), which is the primarily damaged ankle ligament during a LAS. A positive talar tilt test is considered a sign of damage of the calcaneofibular ligament (CFL), which is damaged secondarily to the ATFL. Consequently, our participants' CFLs may have incurred less damage, limiting the amount of mechanical instability we observed with the talar tilt test. Although we observed an increase in ligamentous laxity in this cohort, it may ultimately have little contribution to long-term consequences, as the International Ankle Consortium has not emphasized it as an essential component of CAI.[12-14]

Prominent degrees of swelling and pain were not present in the patients at RTP. Although we identified a statistically significant difference in ankle joint edema between limbs at RTP, it was associated with a negligible effect size, indicating the difference likely has little clinical meaningfulness. Our examination of potential confounding factors suggests the small limb-to-limb difference likely was not due to previous LAS in the uninvolved limbs. Previously, ankle joint swelling has raised concern, partially due to a potential association with decreased ankle ROM. As a secondary analysis, we

explored the relationship between the percent change in ankle girth (relative to the uninvolved limb) and the percent change in WBLT scores (relative to the uninvolved limb), and found that they were not related (r=-0.08, *P*=0.59). Ankle joint swelling raised further concern due to its potential influence on arthrogenic muscle responses in lower leg musculature, but recent evidence indicates ankle joint swelling following an acute LAS likely has little contribution to neural excitability of lower leg musculature.[150] Although the degree of pain experienced by our participants at RTP may not immediately impact sensorimotor function, pain may persist for months or even years after injury.[15] It is unclear how long-lasting pain may influence functional movement patterns and physical activity levels.

To analyze the influence of injury severity on clinical presentation at RTP, patients with grades 2 and 3 were combined due to the occurrence of only one grade 3 LAS in our cohort. Our participants exhibited an equal number of low (grade 1) and high (grade 2-3) severity LASs. As expected, severity appeared to be a strong factor behind the degrees of swelling and WBLT deficits remaining at RTP. The presence of swelling was an explicit component of our grading criteria, and our findings suggest the presence of residual swelling will likely be greater in patients with a more severe ankle sprain. Although dorsiflexion ROM was not an explicit criterion for grading, WBLT deficits at RTP also appear to be greater in patients with a more severe injury. Surprisingly, pain, ligamentous laxity, postural control, and self-reported function at RTP did not differ among injury grades. While our criteria for grading specifically included pain and ligamentous laxity, their presence after the initial injury may not be indicative of pain and laxity that present at RTP. Classification of dynamic postural control and self-reported

function were not clearly included in the grading scale, and appear to be deficient at RTP, regardless of injury severity. Clinicians should consider that an overall injury grade might be an insufficient determinant of all impairments and activity limitations likely to exist at RTP.

The majority of participants utilized at least one form of immobilization and underwent at least one clinician-supervised therapeutic exercise session. Some authors recommend short-term use of immobilization devices following a LAS,[213] whereas other treatment guidelines heavily favor early functional rehabilitation over immobilization.[214] One proposed benefit of early, aggressive therapy is that it allows for earlier RTP.[214] Our data indicate that longer durations of immobilization were associated with smaller deficits in dorsiflexion ROM, but also increased ankle joint swelling and SEBT-ANT deficits at RTP. However, we cannot conclude that longer durations of immobilization had a causative effect on these impairments at RTP, as we have no documentation of the outcomes immediately following the acute injury. Our data do show that swelling and postural control deficits commonly persist following the use of an immobilization device and that continued rehabilitation is necessary between removal of the immobilization device and RTP. The number of supervised rehabilitation sessions was directly related to FAAM scores and the magnitude of SEBT-ANT deficits, indicating that those undergoing more rehabilitation exhibited greater self-reported function related to activities of daily living, but also more pronounced SEBT-ANT deficits at RTP. Similar to our results regarding immobilization, we cannot conclude causation without documentation of deficits immediately post-injury. However, increased involvement in rehabilitation likely benefited participants' self-reported function directly, as it is unlikely

that patients with fewer activity limitations engaged in more rehabilitation. Conversely, participants with greater SEBT-ANT deficits immediately post-injury most likely underwent more rehabilitation sessions, but not enough to resolve their more severe impairments. As the number of rehabilitation sessions was not related to any other functional impairment or the FAAM-S, this particular metric of rehabilitation may not be suitable for predicting outcomes. Instead, the type, frequency, and duration of rehabilitation may be more appropriate for developing models for identifying the outcomes of this patient cohort.

Clinical Implications

Clinicians should be aware that athletes' structural and functional impairments and activity limitations following a LAS often persist at RTP. Additionally, the presence of each impairment and activity limitation at RTP is not necessarily related to greater injury severity, days of immobilization, or number of therapeutic exercise sessions. In order to optimize patient care, clinicians likely need to develop LAS treatment plans based on regular assessments of impairments and limitations specific to each patient, rather than designing general treatment protocols based on commonly observed sequelae.[215] However, this strategy might not be widely exercised by clinical ATs, as we observed an oversimplified common standard for RTP along with residual impairments and activity limitations. As it remains unclear which outcomes contribute most to recurrent LAS and CAI, clinicians should aim to resolve all residual structural and functional impairments and activity limitations. Although patients likely benefit from more extensive care, the treatment provided by ATs may still be more effective than common, rudimentary treatment guidelines. The patients in our study had far higher rates

of immobilization use (88% vs. 28%) and therapeutic exercise (92% vs. 6%) compared to 700,000 patients seeking professional care for a LAS as reported in the literature.[9] At 12 days post-injury, our participants displayed self-reported functional deficits similar to those reported on the Foot and Ankle Outcome Score survey by ankle sprain patients 30 days after emergency department discharge.[158] Additionally, those engaging in early clinician-supervised rehabilitation have previously demonstrated favorable ankle muscular strength and postural control six weeks post-injury and a lower recurrent injury rate 12 months post-injury, compared to patients following standard emergency department guidelines.[114] Thus, while underlying impairments and activity limitations may commonly persist following RTP, access to AT-supervised care is likely advantageous to patients with a LAS.

Limitations

We must acknowledge several limitations within this study. First, the size of the cohort may not accurately depict the total population of student-athletes with a LAS, and future studies could benefit from an expanded sample size. Although we identified a minimal standard for RTP utilized by the treating ATs, other components of the ATs' RTP criteria varied, which may have influenced our findings. While we aimed to illustrate patients' impairments and limitations at the time of RTP, due to logistic concerns, the actual evaluation occurred up to 48 hours before or after the actual RTP date. We did not assess the VAS, FAAM, and FAAM-S on the uninvolved limbs, and thus, we assumed the degree of pain and self-reported function on the involved limbs was comparable to a healthy, uninvolved ankle. While we attempted to document the volume of rehabilitation completed by the cohort, we did not include unsupervised rehabilitation

sessions. Finally, we did not collect information regarding the type, duration, and intensity of the cohort's therapeutic exercises, which may have partially explained varying outcomes in our sample.

CONCLUSIONS

In conclusion, athletic patients with an acute LAS presented with residual impairments and activity limitations related to self-reported function, dorsiflexion ROM, ankle joint laxity, and dynamic postural control at the time of RTP. As resumption of sporting activities did not coincide with complete resolution of structural and functional impairments and activity limitations, clinicians may need to consider if expanded care is necessary before returning patients with a LAS to high-risk activity. The impact of these impairments and activity limitations on long-term consequences remains unknown, and follow-up studies should investigate these common clinical tests as prospective predictors of recurrent injury and CAI.

Table 4.1. Ankle Sprain Frequency by Sport

Sport	Number of Ankle Sprains
Football	24
Basketball	10
Soccer	9
Baseball	2
Volleyball	2
Lacrosse	1
Dance	1
Riflery	1

Table 4.2. Days to Return-to-Play by LAS Grade

LAS Grade	DRTP (Mean ± SD)
1 (n=25)	6.6 ± 6.5
2 (n=24)	17.9 ± 8.9
3 (n=1)	36

Table 4.3. Descriptive Statistics

Outcome Measure	Mean ± SD
FAAM (%)	85.3 ± 11.2
FAAM-S (%)	67.5 ± 18.3
Pain, non-weight-bearing (#/100)	4.4 ± 8.5
Pain, single-leg stance (#/100)	13.0 ± 12.7
Pain, walking (#/100)	10.8 ± 13.9
Figure-of-8, involved (cm)	*54.7 ± 5.2
Figure-of-8, uninvolved (cm)	53.8 ± 5.0
Figure-of-8, asymmetry (cm)	0.9 ± 1.0
WBLT, uninvolved (cm)	*6.8 ± 3.5
WBLT, involved (cm)	9.1 ± 4.0
WBLT, asymmetry (cm)	2.3 ± 2.4
Anterior drawer, involved (grade 0-3)	*1.1 ± 0.5
Anterior drawer, uninvolved (grade 0-3)	0.9 ± 0.5
Talar tilt, involved (grade 0-3)	1.2 ± 0.4
Talar tilt, uninvolved (grade 0-3)	1.1 ± 0.4
SEBT-ANT, involved (% leg length)	*57.9 ± 5.9
SEBT-ANT, uninvolved (% leg length)	60.9 ± 6.0
SEBT-ANT, asymmetry (% leg length)	2.8 ± 4.3
Immobilization (days)	5.7 ± 6.3
Supervised rehabilitation (sessions)	7.6 ± 6.7

*significantly different from uninvolved limb; FAAM = Foot and Ankle Ability Measure, activity of daily living subscale; FAAM-S = Foot and Ankle Ability Measure, sport subscale; WBLT = weight bearing lunge test; SEBT-ANT = anterior reach of the star excursion balance test; RTP = return to play

Table 4.4. Comparisons between High and Low Injury Severity

	Grade 1 (n=25)	**Grade 2-3 (n=25)**	**Independent T-Test**	**Cohen's d Effect Size**
FAAM (%)	83.9 ± 12.5	86.9 ± 9.4	t_{48} = -0.95, P = 0.35	-0.27 (-0.82, 0.29)
FAAM-S (%)	68.9 ± 17.3	65.0 ± 18.8	t_{48} = 0.75, P = 0.46	0.22 (-0.34, 0.77)
Pain, non-weight-bearing (#/100)	4.5 ± 9.4	3.4 ± 6.8	t_{48} = 0.49, P = 0.63	0.13 (-0.42, 0.69)
Pain, single-leg stance (#/100)	12.0 ± 12.1	12.1 ± 13.3	t_{48} = 0.04, P = 0.97	-0.01 (-0.56, 0.55)
Pain, walking (#/100)	12.3 ± 15.3	8.2 ± 12.1	t_{48} = 1.05, P = 0.30	0.30 (-0.26, 0.85)
Figure-of-8, involved (cm)	54.6 ± 5.4	54.9 ± 4.5	t_{48} = -0.23, P = 0.82	-0.06 (-0.61, 0.50)
Figure-of-8, uninvolved (cm)	54.1 ± 5.2	53.8 ± 4.4	t_{48} = 0.19, P = 0.85	0.06 (-0.49, 0.62)
Figure-of-8, asymmetry (cm)	*0.5 ± 0.9	1.1 ± 1.0	t_{48} = -2.11, **P = 0.04**	**-0.63 (-1.19, -0.05)**
WBLT, uninvolved (cm)	7.2 ± 3.2	6.5 ± 3.8	t_{48} = 0.67, P = 0.51	0.20 (-0.36, 0.75)
WBLT, involved (cm)	8.8 ± 3.1	9.5 ± 4.7	t_{48} = -0.69, P = 0.49	-0.18 (-0.73, 0.38)
WBLT, asymmetry (cm)	*1.6 ± 1.8	3.0 ± 2.5	t_{48} = 2.33, **P = 0.02**	**-0.64 (-1.20, -0.06)**
SEBT-ANT, involved (% leg length)	58.8 ± 5.7	56.8 ± 6.3	t_{48} = 1.16, P = 0.25	0.33 (-0.23, 0.89)
SEBT-ANT, uninvolved (% leg length)	60.2 ± 4.7	60.9 ± 7.4	t_{48} = -0.37, P = 0.71	-0.11 (-0.67, 0.44)
SEBT-ANT, asymmetry (cm)	1.3 ± 3.8	3.6 ± 4.5	t_{48} = -1.96, P = 0.06	-0.55 (-1.11, 0.02)

Table 4.5. Pearson Product Moment Correlations and Coefficients of Determination of Days of Immobilization

Outcome	r	R^2	Significance
FAAM	0.05	0.00	0.76
FAAM-S	-0.15	0.02	0.31
Pain NWB	-0.06	0.00	0.69
Pain SLS	-0.07	0.00	0.63
Pain walking	-0.11	0.01	0.45
Figure-of-8, asymmetry (cm)	0.31	0.10	0.03[a]
WBLT, asymmetry (cm)	-0.44	0.19	<0.01[a]
SEBT-ANT, asymmetry (% leg length)	0.35	0.12	0.01[a]

[a] Statistically significant correlation ($P<0.05$)

Table 4.6. Pearson Product Moment Correlations and Coefficients of Determination of Clinician-Supervised Rehabilitation Sessions

Outcome	r	R^2	Significance
FAAM	0.29	0.09	0.04[a]
FAAM-S	-0.07	0.01	0.65
Pain NWB	-0.07	0.01	0.64
Pain SLS	-0.08	0.01	0.59
Pain walking	-0.10	0.01	0.51
Figure-of-8, asymmetry (cm)	0.16	0.03	0.28
WBLT, asymmetry (cm)	-0.08	0.01	0.58
SEBT-ANT, asymmetry (% leg length)	0.34	0.11	0.02[a]

[a] Statistically significant correlation ($P<0.05$)

Figure 4.1. Figure-of-Eight Girth Measurement

Figure 4.2. Weight-Bearing Lunge Test (WBLT)

Figure 4.3. Star Excursion Balance Test Anterior Reach (SEBT-ANT)

Chapter 5: Clinical Determinants of Recurrent Ankle Sprain following Return to Play

INTRODUCTION

High school student-athletes in the United States sustain over 326,000 ankle injuries annually,[5] 80% of which are ankle sprains.[6] Athletes competing in National Collegiate Athletic Association (NCAA) sports account for 11,000 to 16,000 ankle sprains annually,[7,56] which represents 15% of all injuries in that population.[7] In addition to the high incidence of acute ankle sprains, recurrent injuries are also common, making up approximately 16% of all ankle sprains.[58] Konradsen et al. [10] reported that within seven years of a LAS, 19% of patients report the recurrence of injuries or complain of susceptibility to recurrent injuries. Braun[11] reported that approximately 19% of patients with an ankle sprain sustain a recurrent injury between 6 and 18 months later. Recurrent injuries, along with episodes of "giving way" and feelings of instability, contribute to a common condition known as chronic ankle instability (CAI).[12-14] The repetitive nature of ankle sprains has contributed to a prominent financial burden and a negative impact on neuromuscular control, physical activity levels, health-related quality of life, and joint health.[216]

Vast rates of acute and recurrent ankle sprains have led to widespread initiatives for injury prevention. Effective prevention strategies for index and recurrent ankle sprains have been identified previously,[20,21] as have interventions designed to eliminate characteristics of CAI,[177] but their implementation often suffers from limited time and resources.[22] As a potential means for improving efficient allocation of preventative resources, a number of investigators have attempted to identify risk factors that predict individuals predisposed to ankle sprains. Despite the development of numerous effective

prediction models for acute ankle sprains, as yet, there is no perfect prediction model. This equates to many clinical tests possessing susceptibility to misclassify patients as low-risk, potentially leading to absence of preventative care.

Prediction and prevention of recurrent ankle sprains may act as an additional safeguard from long-term consequences of the initial injury. Ankle sprains are associated with a number of impairments and activity limitations, such as pain, swelling, ligamentous laxity, reduced range of motion (ROM), reduced postural control, and perceived dysfunction and instability. When left unresolved, the influence of these outcomes on recurrent injuries is unknown. Few investigators have attempted to identify risk factors for recurrent ankle sprains using clinical outcomes, and thus far, injury severity has been the only one to demonstrate usefulness.[52] Doherty et al.[53] aimed to predict CAI development 1-year post-injury, and found that the inability to perform jumping and landing tasks 2-weeks post-injury, and lower self-reported function and dynamic postural control 6-months post-injury were the strongest risk factors.

While these investigations have reported valuable findings regarding prediction of recurrent ankle sprains and CAI, the limited collection of studies inhibits widespread clinical applicability. Perhaps most notably, no study has attempted to predict recurrent ankle sprains in high school and collegiate athletes, despite large contributions to the total volume of ankle sprain incidents from those populations. Additionally, the current body of work has not considered the predictive value of residual sequelae relative to the re-initiation of sporting activity. While immediate post-injury sequelae may be pertinent to the risk of recurrent ankle sprains, clinicians commonly introduce therapeutic interventions to correct impairments and activity limitations in the sub-acute stages

before return to play (RTP) is considered. Consequently, investigators may achieve prediction of recurrent ankle sprains more effectively by evaluating the presence of impairments and activity limitations as the patient is granted RTP status and resumes high-risk physical activity.

Therefore, the purpose of this study was to determine the ability of clinical measures of pain, swelling, ligamentous laxity, dorsiflexion ROM, dynamic postural control, and self-reported function and instability to predict recurrent ankle sprains in athletes during the same competitive season after RTP from an acute ankle sprain. We hypothesized that patients with greater ankle joint pain, ankle swelling, and ankle ligamentous laxity and lower dorsiflexion range of motion, dynamic postural control, and self-reported function and stability at RTP would have greater estimated odds of sustaining a recurrent ankle sprain during the same competitive sport season. Additionally, we aimed to examine the predictive value secondary outcomes (age, height, mass, body mass index [BMI], injury grade, percentage of season remaining, previous injury history, days to return to play [DRTP], immobilization, rehabilitation, and use of prophylactic ankle supports for RTP) that may also influence recovery from an ankle and susceptibility to recurrent injury. We hypothesized that patients with greater age, height, mass, BMI, injury grade, percentage of season remaining, previous injury history, and DRTP and lower days of immobilization, therapeutic exercise sessions, and usage of prophylactic ankle supports for RTP would have greater estimated odds of sustaining a recurrent ankle sprain during the same competitive sport season.

METHODS

Participants

In this prospective cohort study, we recruited 64 patients (F: 11, M: 49; 17.9 ± 3.3yrs; 178.8 ± 10.7cm; 85.3 ± 24.3kg) with an acute ankle sprain from athletic training facilities of high schools and colleges in central Kentucky. Inclusionary criteria consisted of the presence of an acute ankle sprain (lateral, medial, or syndesmotic) that occurred during organized sporting activity, evaluation by an athletic trainer (AT), and at least one day of activity time-loss. Exclusionary criteria consisted of the presence of fracture, the presence of additional lower extremity injuries, or surgical treatment for the injury. The treating AT notified participants and parents, when applicable, of their study eligibility following injury diagnosis. Participants over the age of 18 read and signed an informed consent document approved by the University of Kentucky's institutional review board. Participants under 18 provided assent, while a parent or legal guardian signed the informed consent document.

Procedures

Following the diagnosis of an ankle sprain, the treating AT and/or physician determined each participant's care and RTP decisions. We defined RTP as a resumption of unrestricted sporting activity. As the participant neared RTP, the treating AT contacted the primary investigator (PI) to schedule an independent clinical evaluation in the athletic training facility of each participant's school. The PI conducted evaluations no more than 48 hours before or after the actual RTP date. Primary outcomes assessed during the evaluation consisted of ankle joint pain, ankle edema, ankle dorsiflexion ROM, ankle ligamentous laxity, dynamic postural control, and self-reported physical function and stability. We also documented secondary outcome measures, including age, height, mass, BMI, injury grade, percentage of season remaining, previous injury history,

days to return to play (DRTP), immobilization, rehabilitation, and use of prophylactic ankle supports for RTP.

Pain Assessment

We measured pain with a 100 millimeter visual analogue scale (VAS), with opposite ends labeled "no pain" and "worst pain ever."[217] Participants made a mark on the location of the line that best represented the immediate intensity of pain. We quantified pain by the distance in millimeters from the participant's mark to the lowest end of the VAS. Participants reported pain in a non-weight-bearing position (NWB), single-leg stance (SLS), and after walking four steps. Pain was interpreted as none (< 5/100), mild (5-44/100), moderate (45-74/100), and severe (>74/100), based on a previous study of pain in post-operative patients.[208] Bijur et al.[218] reported excellent reliability (ICC = 0.95-0.98) of the VAS in patients with acute pain.

Edema Assessment

We assessed edema accumulation in the injured ankle with a figure-of-eight girth measurement (Figure 4.1).[219] The PI placed the participant's ankle in neutral dorsiflexion and instructed the participant to maintain that position. The PI then wrapped a tape measure around the ankle, beginning midway between the tibialis anterior tendon and the lateral malleolus. The tape measure tracked across the anterior ankle and passed just distally to the navicular tuberosity. After the tape measure passed under the plantar aspect of the foot, it passed just proximally to the base of the fifth metatarsal. The tape measure continued across the anterior aspect of the ankle, and wrapped around the shank just distally to the medial and lateral malleoli. After passing the lateral malleolus, the figure-of-eight concluded at its starting point. The total distance in centimeters

represented the girth of the measured ankle. We utilized two variables to represent edema formation: 1) the raw measurement (cm) of the involved limb, and 2) asymmetry, calculated from the difference (cm) between the involved and uninvolved limb. A previous study reported excellent intra-rater (ICC = 0.99) and inter-rater reliability (ICC = 0.99) for the figure-of-eight girth measurement.[209]

Ankle Dorsiflexion ROM Assessment

We examined ankle dorsiflexion ROM bilaterally with the weight-bearing lunge test (WBLT) (Figure 4.2).[210,220] To prepare the WBLT, we fixed a tape measure to the floor, perpendicular to a wall. The participant faced the wall with the hallux and heel of test limb in-line on top of the tape measure. The participant lunged forward, keeping the foot flat against the floor, and touched the anterior aspect of the knee to the wall. We allowed the participant to place their hands on the wall and non-test limb on the floor for support. If the participant successfully touched their knee to the wall, we incrementally moved the foot away from the wall, and repeated the test. The goal of the test was to contact the anterior knee to the wall with the hallux at the furthest distance possible, indicating a greater degree of dorsiflexion ROM. We permitted up to five trials to determine maximum performance. Similar to the edema assessment, we utilized two variables to represent dorsiflexion ROM: 1) the raw WBLT scores (cm) of the involved limb, and 2) asymmetry, calculated from the WBLT difference (cm) between the involved and uninvolved limb. In a systematic review of WBLT reliability studies, Powden et al.[221] reported good to excellent intra-rater (ICC = 0.65-0.99) and inter-rater reliability (ICC = 0.80-0.99).

Ligamentous Laxity Assessment

We employed the anterior drawer and talar tilt tests to evaluated ankle ligamentous laxity. We conducted the anterior drawer test with the ankle in approximately 10° of plantar flexion.[32] The PI grasped the distal lower leg with one hand and the posterior calcaneus with the other. While stabilizing the lower leg, the examiner exerted an anteriorly directed force on the heel and assessed the degree of laxity during anterior talar translation.[32,92] We conducted the talar tilt with the ankle in a neutral sagittal plane position. The examiner grasped the distal lower leg with one hand and the inferior calcaneus with the other. While stabilizing the lower leg, the examiner inverted the ankle and assessed the degree of lateral joint laxity.[92] The PI utilized an eversion talar tilt test if the participant had sustained a medial ankle sprain (MAS). We graded laxity in both tests on a four-point scale: 0 = no laxity, 1 = mild laxity, 2 = moderate laxity, and 3 = severe laxity.[92] In addition to the overall degree of laxity, we assessed each test as a dichotomous (+/-) variable, in which a greater degree of laxity on the involved limb compared to the uninvolved limb represented a positive test.

To our knowledge, no previous study has reported reliability of the anterior drawer and talar tilt tests. Thus, prior to the study's onset, the PI examined 10 healthy volunteers on two separate occasions, two weeks apart. We calculated weighted Kappa coefficients to determine intra-rater reliability of the PI.[222] Nearly perfect intra-rater agreement was demonstrated on the talar tilt test ($\kappa_w = 0.89$, $P < 0.01$), and fair intra-rater agreement was demonstrated on the anterior drawer test ($\kappa_w = 0.40$, $P = 0.04$).

Dynamic Postural Control Assessment

We assessed dynamic postural control bilaterally using the anterior reach of the star excursion balance test (SEBT-ANT) (Figure 4.3). We selected SEBT-ANT due to its

superior efficacy for ankle sprain prediction compared to the other reach directions.[41] We utilized previously reported criteria for SEBT-ANT administration.[119,223,224] To prepare the SEBT-ANT, we fixed a tape measure to the floor directly anterior to the participant, and positioned the participant's second toe at the zero mark. The participant maintained a single-leg stance on the test limb while reaching for maximum distance in the anterior direction with the non-test limb. The PI instructed the participant to gently touch the tape measure with the most distal aspect of the reaching limb while maintaining a single-leg base of support on the test limb, and then return to double-leg stance. The participant's hands were required to remain on their hips and the stance heel was required to remain in contact with the floor. Four practice trials were performed,[196] followed by three test trials. The average SEBT-ANT score for each limb was normalized as a percentage of stance leg length (%LL).[119] Again, we utilized two variables to represent dynamic postural control: 1) the normalized SEBT-ANT scores (%LL) of the involved limb, and 2) asymmetry, calculated from the SEBT-ANT difference (%LL) between the involved and uninvolved limb. Hertel et al.[198] reported excellent intra-rater reliability (ICC = 0.88-0.95) of the SEBT-ANT. Gribble et al.[197] reported excellent inter-rater reliability (ICC = 0.88) of normalized SEBT-ANT scores.

Self-Reported Physical Function and Stability Assessments

We assessed each participant's self-reported physical function relative to the involved limb using the Foot and Ankle Ability Measure activity of daily living (FAAM-ADL) and sport (FAAM-S) subscales. Both subscales are scored as a percentage, with a score of 100 associated with no loss of self-reported physical function due to the involved ankle, whereas a score of 0 associated with a complete loss of self-reported physical

function due to the involved ankle. Martin et al.[144] reported excellent test-retest reliability on the FAAM-ADL (ICC = 0.89) and FAAM-S subscales (ICC = 0.87).

We assessed self-reported ankle stability using the Identification of Functional Ankle Instability (IdFAI) questionnaire. Higher scores are associated with lower levels of self-reported ankle stability. Guarev et al.[164] reported that individuals between ages 20-30 had excellent test-retest reliability (ICC = 0.98) on the IdFAI.

Injury Grade

Each ankle sprain received an overall severity grade by the treating AT during the initial injury evaluation. A severity grade of 1 was marked by little to no pain and swelling, and minimal loss of weight bearing ability and mechanical stability. A severity grade of 2 was marked by moderate pain and swelling, and moderate loss of weight bearing ability and mechanical stability. A severity grade of 3 was marked by severe pain and swelling, and severe loss of weight bearing ability and mechanical stability.

Previous Injury History

The treating AT provided documentation of the number of previous ankle sprains sustained on each of the participant's limbs. If medical documentation was unavailable for review, we asked participants to recall previous injuries.

Percentage of Season Remaining

We included the percentage of season remaining as an exploratory variable; to our knowledge, no other author has previously investigated its predictive utility for injury. The potential influence on the conservativeness of the treatment plan led to its inclusion. We calculated percentage of season remaining from the following formula:

$$\frac{\text{\# days from injury onset until the last regular season competition}}{\text{\# of days from the 1st pre-season practice to the last regular season competition}} \times 100$$

Immobilization

We documented the total number of days each participant utilized at least one immobilization device (crutches, walking boot, splint, and compression wrap) that limited use of the injured ankle.

Rehabilitation

We recorded the total number of therapeutic exercise sessions conducted under direct supervision of a health care professional (i.e. AT, physical therapist).

Days to Return to Play

We defined days to RTP (DRTP) as the total number of days from the onset of injury until the participant returned to unrestricted sporting activity as determined by the treating AT or physician. We provided no guidelines for RTP decision-making to the treating ATs.

Use of Prophylactic Ankle Supports

Following the conclusion of the participants' competitive seasons, we categorized participants as those who did and did not intend to use prophylactic ankle supports (i.e. braces, tape) following RTP.

Recurrent Injury Tracking

Following RTP, the treating AT tracked recurrent ankle sprains on the involved limb for the remainder of the competitive season. At the conclusion of the participants' competitive seasons, we assigned participants to either a Recurrent Injury (RI) or No

Recurrent Injury (NRI) group. Those in the RI group sustained at least one additional ankle sprain of any type on the involved limb before the conclusion of the competitive season. Like the initial injury, recurrent ankle sprains must have occurred during organized sporting activity, undergone evaluation by an AT, and resulted in at least one day of activity time-loss. Members of the NRI group sustained no recurrent ankle sprains on the involved limb for the remainder of the competitive season.

Statistical Analysis

We compared continuous primary and secondary outcomes (pain, ankle joint swelling, ankle dorsiflexion ROM, dynamic postural control, patient-reported outcomes, age, height, mass, BMI, percentage of season remaining, DRTP, days of immobilization, and number of rehabilitation sessions) between groups with separate independent t-tests. Additionally, we utilized Cohen's *d* effect sizes to gauge the magnitude of group differences for continuous variable. Effect sizes were interpreted as small (d = 0.20-0.49), moderate (d = 0.50-0.79), and large (d > 0.80).[202] We conducted separate Fisher's exact tests to determine the strength of association between the recurrent injury status and dichotomous categorical variables (ligamentous laxity, previous ankle sprain history, and use of prophylactic ankle supports). Separate non-parametric Mann-Whitney U tests examined differences in ligamentous laxity and injury grade between groups.

Separate forward binary logistic regression analyses assessed the influence of each significantly different outcome on the estimated odds of sustaining a recurrent ankle sprain in the same competitive season. We also conducted logistic regression analyses of any non-significant primary outcomes. We employed a Receiver Operating Characteristic (ROC) curve to plot the predictive utility (sensitivity vs. 1-specificity) of

each value observed for the continuous primary and secondary outcomes. For outcomes that differed between groups, we obtained the area under the ROC curve (AUROC), a singular quantitative representation of the overall predictive value of each variable, with 95% confidence intervals. The AUROC can range from 0 to 1, with 0.5 representing an absence of predictive power, and 1 representing perfect predictive power.[203] Also from the ROC curves, we identified cutoff scores that maximized sensitivity and specificity for each predictor variable. We conducted Fisher's exact tests to determine the strength of association between the predicted group classification based on the cutoff score and observed injury status. We calculated sensitivity, specificity, positive and negative likelihood ratios (+LR, -LR), and the diagnostic odds ratio (DOR) for cutoff scores. Statistical significance was set *a priori* at $P<0.05$. All statistical analyses were conducted using IBM SPSS version 23 (IBM Corporation, Armonk, NY).

RESULTS

We evaluated 64 patients with an ankle sprain from 12 different clinical sites. We excluded four patients from statistical analyses after they transferred or discontinued participation in athletics before the end of the competitive season. Therefore, the final analyses included 60 patients with an ankle sprain. Thirty-seven (62%) competed in high school athletics and 27 (38%) competed in collegiate athletics. Frequencies of ankle sprains by sport are reported in Table 5.1. Fifty-four participants (90%) sustained a lateral ankle sprain (LAS), four (7%) sustained a medial ankle sprain (MAS), and two (3%) sustained a syndesmotic ankle sprain (SAS). Thirty injuries (50%) received a severity grade of "1," 28 (47%) received a grade of "2," and two (3%) received a grade of

"3." Following RTP, six patients (10%) sustained a recurrent ankle sprain before the conclusion of the competitive season (Table 5.2).

Patients in the RI groups demonstrated significantly greater height and mass compared to the NRI group (Table 5.3). Large effect sizes further substantiated the group differences in height and mass. No other continuous outcomes differed between groups. Group membership had no significant associations with dichotomous categorical outcomes (Table 5.4). Mann-Whitney U tests found greater ligamentous laxity on the talar tilt in the NRI group compared to the RI group, but no differences in anterior drawer laxity or injury grade (Table 5.5).

Logistic regression analyses indicated that greater height and mass were significantly associated with greater odds of being in the RI group (Table 5.6). Odds ratios indicated that every increase in height by 1 cm was associated with a 13% increase in the estimated odds of sustaining a recurrent ankle sprain; every increase in mass by 1 kg was associated with a 4% increase in recurrent injury odds. No other primary outcome produced a significant logistic regression model.

ROC curve analyses further demonstrated moderate predictive values for height and mass, although mass did not reach a degree of statistical significance (Table 5.7). The AUROCs for height and mass are depicted in Figures 1 and 2. The AUROCs for primary outcomes that did not differ between groups are depicted in Appendix A. From the ROC curves, we obtained cutoff scores for height (191.0 cm) and mass (100.0 kg) that maximized sensitivity and specificity of each test. Fisher's exact tests revealed significant associations between recurrent injury status and cutoff scores for height and

mass (Table 5.8). Cutoff scores for height and mass demonstrated moderate to moderate to excellent sensitivity and specificity and favorable diagnostic odds ratios (Table 5.9).

DISCUSSION

The primary finding of this study was that height and mass were effective predictors of recurrent ankle sprain in athletes returning to sporting activity in the same competitive season in which a previous ankle sprain occurred. Height demonstrated the best predictive value, as patients taller than 191.0 cm had over 16 times greater odds of sustaining a recurrent sprain than those shorter than 191.0 cm. Additionally, patients weighing at least 100.0 kg had over eight times greater odds of sustaining a recurrent sprain than those below 100.0 kg. These findings are likely attributable to larger mass and length of the lower extremity segments that increase inertial resistance of the ankle joint and reduce the ability of the individual to reverse momentum in the presence of an external inversion or eversion moment.[31,43] The use of self-reported height and mass is a potential limitation of our study, but others have discovered that self-reported estimates are highly correlated with instrumented measures of height and mass.[225,226] The simplicity of these measures is an obvious advantage to implementation of our findings into clinical practice. Athletic trainers in any setting can collect height and mass data and confidently determine which patients are in need of added preventative care during the remainder of the season.

No previous studies have utilized participant height or mass as predictors of recurrent ankle sprain in high school and collegiate athletes, but others have utilized such measures as potential predictors of acute ankle sprains. Waterman et al.[2] reported that male military cadets that sustained an ankle sprain during various organized physical

activities had greater height, mass, and BMI compared to those that did not sustain an ankle sprain. Similarly, Milgrom et al[43] found that male military recruits that sustained a LAS were taller and heavier than those that did not sustain a LAS. Two additional studies[30,33] explored the value of height and mass as separate predictors of ankle sprains in athletes, but neither variable was effective. Investigators have more frequently studied BMI as an injury predictor, with several authors reporting that elevated BMI significantly increased risk for ankle sprains in athletes.[31,34,40,41] Although, BMI did not demonstrate predictive value for recurrent ankle sprains in our study, statistical trends and a moderate effect size suggest BMI may have exhibited greater predictive utility in a larger sample. Tyler et al.[31] found that ankle sprain incidences increased in high school football players as BMI increased from normal, risk for overweight, and overweight classifications. When considered with injury history, they found that the combination of an overweight classification and a previous ankle sprain increased the risk of recurrent ankle sprain 19 times compared to those with a normal weight classification and no previous ankle sprain. The findings of Tyler et al.[31] may be pertinent to our study, as each member of our RI group sustained a recent ankle sprain, which may have compounded negative effects of potentially elevated BMI. Despite these previous findings, the consensus regarding the predictive value of BMI for ankle sprains has remained inconclusive, as others have failed to produce an effective prediction model.[24,26,38,39,42]

We selected the primary outcomes due to their common deficiencies following an acute ankle sprain, ease of implementation in a multitude of clinical settings, and potential modifiability. However, the outcomes surprisingly exhibited little to no predictive value for recurrent ankle sprains in our study. Talar tilt laxity (0-3) on the

involved limb demonstrated potential predictive value in group comparisons. However, the logistic regression analyses failed to produce a useful prediction model. Additionally, the observed difference opposed our expectation, as members of the RI group appeared to have lower joint laxity. This may indicate that lower talar tilt laxity provides a false sense of recovery in patients with an ankle sprain and that its absence is not an appropriate determinant of preparedness for RTP. Contrary to this idea, Gerber et al.[55] noted that ankle ligamentous laxity possessed no predictive utility for CAI development.[55] Others[23,32,33] also reported that the talar tilt test was not an effective predictor of acute ankle sprains. Since the current study produced a futile odds ratio with an infinite confidence interval, our sample was likely too small to produce a robust prediction model with talar tilt laxity. Thus, our findings regarding this outcome should be interpreted cautiously.

The three pain assessments, figure-of-eight, WBLT, SEBT-ANT, FAAM, and IdFAI demonstrated no predictive utility for recurrent ankle sprain in athletes. Conversely, Gerber et al.[55] reported that military cadets that avoided CAI after an ankle sprain were pain-free. Additionally, O'Connor et al.[54] found that 49% of ankle function, measured with the Karlsson questionnaire, at 4 months was explained by pain on the WBLT and medial joint-line pain at 4 weeks. While these studies support pain as a useful indicator of future outcomes, our study differed in the timing and method of pain assessment, as well as the length of follow-up. Thus, the predictive value of pain for long-term consequences of ankle sprains should continue to be examined.

This study is the first to examine ankle joint swelling as a potential risk factor for acute or recurrent ankle sprains. While swelling is a common sign of trauma to ankle

ligaments, our findings agree with others suggesting it has little contribution to impaired function,[88] and thus, should receive limited consideration for RTP decisions. Previous studies have reported mixed results regarding our other primary outcomes as risk factors for acute and recurrent ankle sprains. Several studies[24,27,34,36] were unable to predict acute ankle sprains utilizing the WBLT. Additionally, Plante and Wikstrom[123] found that WBLT scores did not differ between individuals with CAI and LAS copers, and thus, may be unable to predict recurrent injury in those with a previous ankle sprain. Our findings may support a lack of predictive value for the WBLT. However, the previous study also found that those with CAI had impaired WBLT scores compared to healthy controls,[123] and our RI and NRI groups' involved limb WBLT scores both resembled those of the CAI cohort (7.8±4.3). Since we tracked our patients with impaired dorsiflexion ROM for a relatively short follow-up period, the patients may not have been exposed to enough risk to realize the true predictive value of the WBLT for recurrent injury.

Two previous studies[41,224] reported that decreased SEBT-ANT scores led to increased risk of acute ankle sprains. In our study, the SEBT-ANT scores did not predict recurrent injury, but our RI and NRI groups' SEBT-ANT scores both resembled those of individuals with CAI.[117,122] As a lower SEBT-ANT score is a potential risk factor for ankle sprains, the poor performance of both groups may indicate that we need a longer follow-up period to determine when the SEBT-ANT can demonstrate predictive value. Conversely, another study found that lower SEBT-ANT scores simply did not increase acute ankle sprain risk, but lower SEBT posterolateral (SEBT-PL) scores did.[24] Similarly, Doherty et al.[53] reported that a lower SEBT-PL score was a significant risk

factor for CAI, while a lower SEBT-ANT score was not. Plante and Wikstrom[123] found that the posteromedial (SEBT-PM) score was the only direction capable of distinguishing between those with CAI and LAS copers. Future studies should examine the predictive value of the three-direction SEBT over an extended follow-up period after RTP.

One previous study[53] investigated the FAAM as a predictor of CAI after an acute ankle sprain, with only the FAAM-ADL subscale demonstrating usefulness. The FAAM and IdFAI are both capable of distinguishing individuals with and without CAI,[143,165] but our results indicate that their evaluation at RTP cannot detect who will sustain a recurrent ankle sprains in a short follow-up period. Similar to the WBLT and SEBT-ANT, FAAM and IdFAI scores for the RI and NRI groups both resembled those of individuals with CAI,[143,165] potentially indicating that a longer follow-up period is needed to realize the predictive value of the FAAM and IdFAI. Future studies should also examine the ability of patient-reported outcomes to predict recurrent ankle sprains in athletes over longer follow-up periods following RTP.

Clinical Implications

Clinicians should be aware that athletes with larger stature are at greater risk of sustaining a recurrent ankle sprain following RTP from a previous ankle sprain in the same competitive season. Patient height and mass are useful metrics of this characteristic, but height is apparently the strongest. Patients exhibiting height and mass over the corresponding cutoff score should be considered candidates for additional preventative care. Weight can be safely modified in athletes,[227] but reductions may cause sport performance to suffer in certain athletes (i.e. football linemen). In such cases, clinicians may utilize established alternative means of preventing ankle sprains and

correcting CAI, such as prophylactic ankle supports[20] and postural control training.[177,205,228] Although the majority of our injured participants engaged in therapeutic exercise and expressed an intent to wear prophylactic ankle supports, we did not document the volume of postural control training performed or the actual usage of prophylactic ankle supports. Therefore, we cannot make any definitive conclusions about the effects of these rehabilitative and preventative strategies on injury recurrence in our sample.

Limitations

Several notable limitations are present in this study. First, the small sample size, particularly among the RI group may not be representative of all high school and collegiate athletes that sustain a recurrent ankle sprain. Additionally, the small sample limited our ability to analyze the predictive utility of two primary outcomes (pain-NWM and talar tilt laxity) completely. We intended to evaluate patients' residual impairments and activity limitations at the time of RTP, but scheduling conflicts caused the actual evaluations to occur up to 48 hours before or after the actual RTP date. The follow-up period (end of the competitive season) was relatively short compared to other studies aiming to predict recurrent ankle sprains or CAI, and it varied across the sample, which may have prevented some patients with residual impairments from sustaining a recurrent injury.

CONCLUSIONS

High school and collegiate athletes with greater height and mass had greater odds of sustaining recurrent ankle sprain during the same season. Taller and heavier patients will likely benefit from established interventions designed to prevent ankle sprains, such

as postural control training and prophylactic ankle supports. While our findings suggest that weight-loss therapy may also reduce the odds of sustaining a recurrent ankle sprain, further inquiry is required to confirm this effect. Ankle joint pain, swelling, ligamentous laxity, dorsiflexion range of motion, dynamic postural control, and self-reported function and instability were not effective predictors of recurrent ankle sprain during the same competitive season after RTP from a previous ankle sprain. As several of our patients' outcomes (dorsiflexion range of motion, dynamic postural control, and self-reported function and instability) resembled those of individuals with CAI, their predictive value may be realized with longer follow-up periods.

Table 5.1. Ankle Sprain Frequency by Sport

Sport	Number of Ankle Sprains
Football	25
Basketball	15
Soccer	9
Baseball	3
Lacrosse	3
Volleyball	2
Dance	1
Riflery	1
Wrestling	1
Total	60

Table 5.2. Recurrent Ankle Sprain Characteristics

Case	Sex	Sport	Initial Injury	Recurrent Injury	Weeks After RTP
1	M	Collegiate Football	Right LAS	Right LAS	2
2	M	Collegiate Football	Left LAS	Left LAS	2
3	M	High School Basketball	Left LAS	Left LAS	10
4	F	High School Soccer	Right LAS	Right LAS	21
5	M	High School Football	Right LAS	Right LAS	4
6	M	Collegiate Football	Left MAS	Left LAS	3

Table 5.3. Comparison of Continuous Outcomes between Recurrent Injury (RI) and No Recurrent Injury (NRI) Groups

Predictor Variable	RI (n=6)	NRI (n=54)	Independent T-Test	Cohen's *d* (95%CI)
Pain, NWB (#/100)	0.0 ± 0.0	4.2 ± 9.0	t_{58} = 1.47, P = 0.26	-0.49 (-1.33, 0.37)
Pain, SLS (#/100)	13.5 ± 12.0	9.7 ± 11.7	t_{58} = -0.76, P = 0.45	0.32 (-0.53, 1.17)
Pain, 4 steps (#/100)	14.8 ± 18.5	9.7 ± 13.5	t_{58} = -0.86, P = 0.40	0.36 (-0.46, 1.21)
Figure-of-8, involved (cm)	57.5 ± 4.5	54.4 ± 5.0	t_{58} = -1.46, P = 0.15	0.62 (-0.23, 1.47)
Figure-of-8 asymmetry (cm)	0.7 ± 1.2	1.0 ± 0.9	t_{58} = 0.55, P = 0.59	-0.32 (-1.16, 0.53)
WBLT, involved (cm)	7.9 ± 4.5	6.9 ± 3.5	t_{58} = -0.63, P = 0.53	0.28 (-0.57, 1.12)
WBLT asymmetry (cm)	2.5 ± 2.1	2.4 ± 2.3	t_{58} = 0.11, P = 0.91	0.04 (-0.80, 0.89)
SEBT-ANT, involved (%LL)	58.5 ± 2.2	58.0 ± 6.8	t_{58} = -0.17, P = 0.87	-0.13 (-0.97, 0.72)
SEBT-ANT asymmetry (%LL)	2.1 ± 3.4	2.6 ± 4.0	t_{58} = 0.32, P = 0.75	-0.12 (-0.97, 0.72)
FAAM-ADL (%)	81.6 ± 7.4	86.8 ± 10.7	t_{58} = 1.15, P = 0.25	-0.50 (-1.34, 0.36)
FAAM-S (%)	67.7 ± 10.2	65.2 ± 19.6	t_{58} = -0.30, P = 0.76	0.13 (-0.71, 0.97)
IdFAI	19.2 ± 10.1	18.8 ± 7.7	t_{58} = -0.10, P = 0.92	0.05 (-0.79, 0.89)
Age (years)	18.3 ± 3.7	17.9 ± 3.3	t_{58} = -0.33, P = 0.74	0.12 (-0.73, 0.96)
Height (cm)	188.2 ± 10.0	177.5 ± 10.4	t_{58} = -2.39, **P = 0.02**	**1.03 (0.16, 1.88)**
Mass (kg)	107.0 ± 34.7	82.5 ± 22.5	t_{58} = -2.39, **P = 0.02**	**1.03 (0.15, 1.88)**
BMI (kg/m^2)	29.8 ± 7.9	25.9 ± 5.5	t_{58} = -1.56, P = 0.12	0.68 (-0.18, 1.52)

Predictor Variable	RI (n=6)	NRI (n=54)	Independent T-Test	Cohen's *d* (95%CI)
% Season Remaining	65.7 ± 28.7	62.0 ± 32.0	t_{58} = -0.27, P = 0.79	0.12 (-0.73, 0.96)
DRTP	11.5 ± 12.6	13.9 ± 11.9	t_{58} = 0.47, P = 0.64	-0.20 (-1.04, 0.65)
Immobilization (days)	5.7 ± 5.1	6.1 ± 6.3	t_{58} = 0.15, P = 0.88	-0.06 (-0.91, 0.78)
Rehabilitation (sessions)	5.5 ± 4.7	8.7 ± 8.3	t_{58} = 0.92, P = 0.36	-0.40 (-1.24, 0.45)

Abbreviations: BMI = body mass index; SEBT-ANT = star excursion balance test anterior reach (normalized to a percentage of stance leg length [%LL]); HEXT = isometric hip extension strength (normalized to a percentage of body mass [%BM])

Table 5.4. Association between Recurrent Injury Status and Dichotomous Categorical Variables.

Predictor Variable	Outcomes	RI (n=8)	NRI (n=52)	Fisher's Exact Test
Anterior Drawer Laxity	+	2	14	P = 0.65
	-	4	40	
Talar Tilt Laxity	+	0	9	P = 0.58
	-	6	45	
Sex	Female	1	16	P = 0.67
	Male	5	38	
Level of Competition	High School	3	30	P = 1.00
	Collegiate	3	24	
Previous Ankle Sprain (involved limb)	Yes	2	19	P = 1.00
	No	4	35	
Previous Ankle Sprain (uninvolved limb)	Yes	3	9	P = 1.00
	No	3	45	
Prophylactic Ankle Support for RTP	Yes	6	47	P = 1.00
	No	0	7	

Table 5.5. Comparison of Polytomous Categorical Variables between Recurrent Injury (RI) and No Recurrent Injury (NRI) Groups

Predictor Variable	RI (n=6)	NRI (n=54)	Mann-Whitney U Test
Anterior Drawer (0-3)	1.0, 0.0	1.0, 0.0	Z = -0.65, P = 0.51
Talar Tilt (0-3)	1.0, 0.3	1.0, 1.0	Z = -2.03, **P = 0.04**
Injury Grade (1-3)	1.0, 1.0	2.0, 1.0	Z = -0.98, P = 0.33

Descriptive statistics presented as median, interquartile range.

Table 5.6. Separate Binary Logistic Regression Analyses

Predictor Variable	Odds Ratio (95%CI)	Significance
Height	**1.13 (1.01, 1.25)**	**0.03**
Mass	**1.04 (1.00, 1.07)**	**0.03**
Pain, NWB	0.00 (0.00, ∞)	0.99
Pain, SLS	1.29 (0.67, 2.51)	0.45
Pain, 4 steps	1.26 (0.74, 2.16)	0.39
Figure-of-8, involved	1.13 (0.96, 1.34)	0.16
Figure-of-8 asymmetry	0.77 (0.31, 1.92)	0.87
WBLT, involved	1.08 (0.85, 1.38)	0.53
WBLT asymmetry	0.98 (0.67, 1.43)	0.91
Anterior Drawer (0-3)	0.61 (0.12, 3.01)	0.54
Anterior Drawer (+/-)	1.43 (0.24, 8.67)	0.70
Talar Tilt (0-3)	0.00 (0.00, ∞)	1.00
Talar Tilt (+/-)	0.00 (0.00, ∞)	1.00
SEBT-ANT, involved	3.18 (0.00, ∞)	0.87
SEBT-ANT asymmetry	0.96 (0.77, 1.21)	0.75
FAAM-ADL	0.96 (0.89, 1.03)	0.26
FAAM-S	1.01 (0.96, 1.06)	0.76
IdFAI	1.01 (0.90, 1.12)	0.92

Table 5.7. AUROC Analyses

Predictor Variable	AUROC	Significance
Height	0.78 (0.57, 0.99)	**P = 0.03**
Mass	0.73 (0.50, 0.97)	P = 0.06
Pain, NWB	0.67 (0.49, 0.85)	P = 0.18
Pain, SLS	0.62 (0.41, 0.83)	P = 0.32
Pain, 4 steps	0.58 (0.32, 0.83)	P = 0.55
Figure-of-8, involved	0.69 (0.45, 0.92)	P = 0.14
Figure-of-8 asymmetry	0.57 (0.30, 0.83)	P = 0.59
WBLT, involved	0.58 (0.33, 0.83)	P = 0.53
WBLT asymmetry	0.52 (0.27, 0.78)	P = 0.86
SEBT-ANT, involved	0.53 (0.38, 0.67)	P = 0.84
SEBT-ANT asymmetry	0.51 (0.26, 0.76)	P = 0.94
FAAM-ADL	0..71 (0.56, 0.86)	P = 0.10
FAAM-S	0.51 (0.33, 0.69)	P = 0.93
IdFAI	0.52 (0.27, 0.77)	P = 0.88

Table 5.8. Fisher's Exact Test for Height, Mass, and BMI Cutoff Scores

Predictor Variable	Outcomes	RI (n=6)	NRI (n=54)	Fisher's Exact Test
Height (cm)	≥ 191.0	4	6	**P = 0.01**
	< 191.0	2	48	
Mass (kg)	≥ 100.0	4	11	**P = 0.01**
	< 100.0	2	43	

Table 5.9. Diagnostic Statistics of Cutoff Scores

Quantity	Formula	Height (191.0 cm)	Mass (100.0 kg)
Sensitivity	true positive/(true positive + false negative)	4/6 = 0.67	4/6 = 0.67
Specificity	true negative/(true negative + false positive)	48/54 = 0.89	43/54 = 0.80
+LR	sensitivity/(1-specificity)	0.67/0.11 = 6.01	0.67/0.20 = 3.35
-LR	(1-sensitivity)/specificity	0.33/0.89 = 0.37	0.33/0.80 = 0.41
DOR	+LR/-LR	6.01/0.37 = 16.24	2.32/0.19 = 8.17

Abbreviations: +LR = positive likelihood ratio; -LR = negative likelihood ratio; DOR = diagnostic odds ratio

Figure 5.1. Height ROC Curve

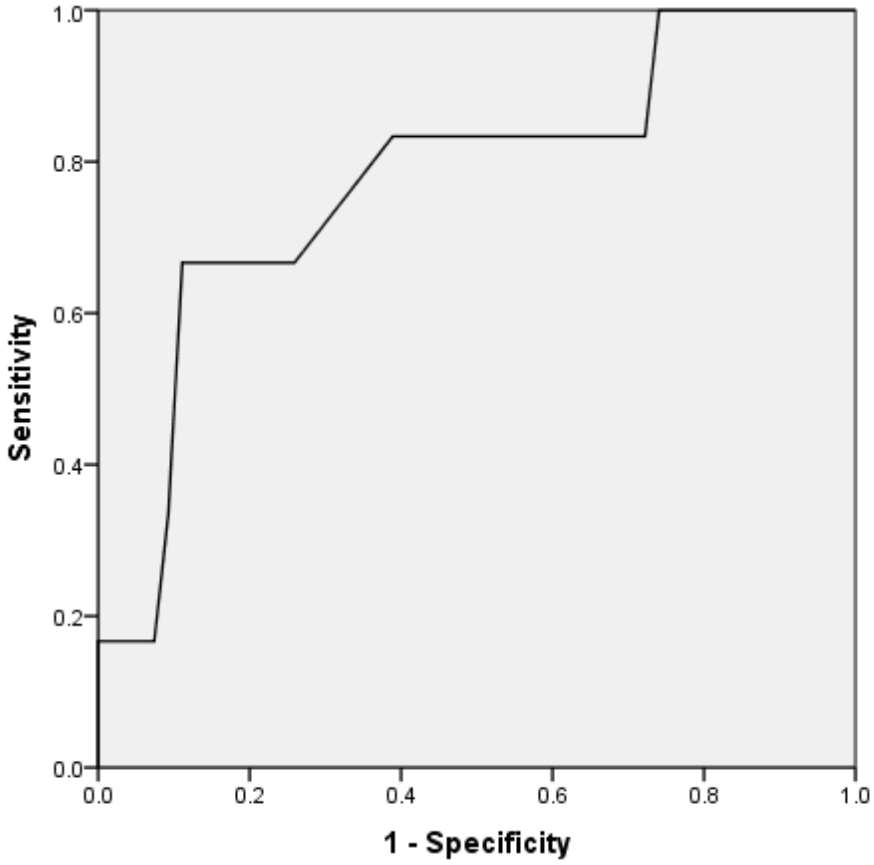
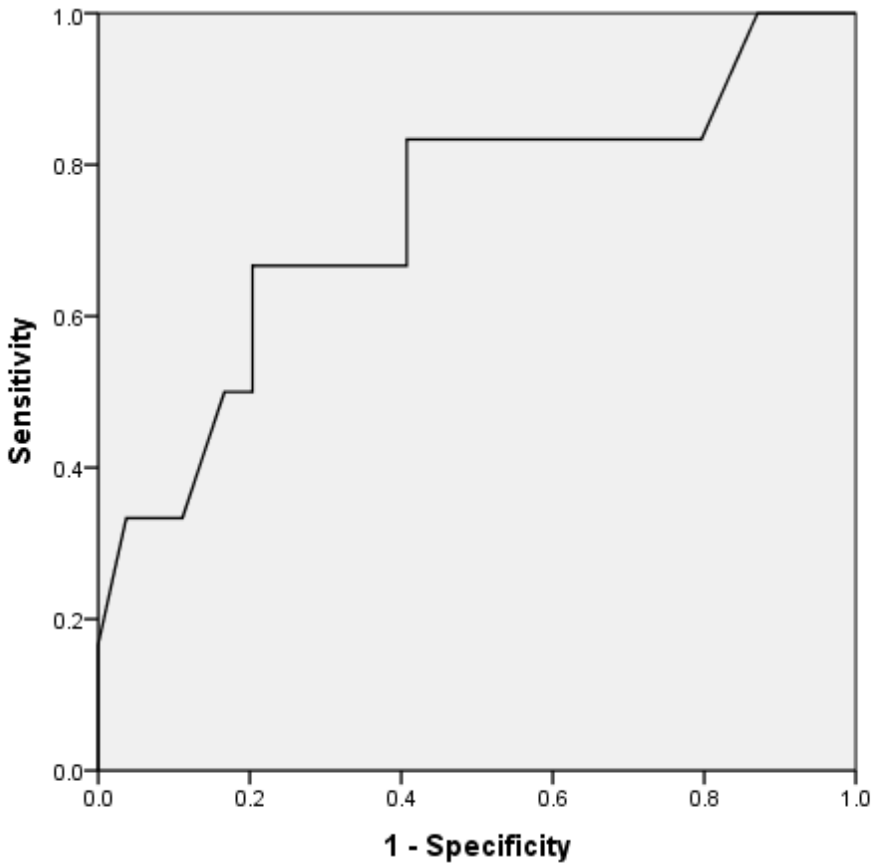


Figure 5.2. Mass ROC Curve



Chapter 6: Summary

The purposes of this dissertation were 1) to develop a prediction model for acute LAS injuries in a previously unstudied population (collegiate women's soccer players) utilizing primary outcomes of dynamic postural control and isometric hip strength as potential predictors, 2) describe the presence of residual impairments and activity limitations in athletes with an acute LAS following clearance for RTP, and 3) develop a prediction model for recurrent ankle sprains in athletes, utilizing assessments impairments and activity limitations at RTP as potential predictors. Here, we summarize our findings pertaining to the hypotheses outlined in Chapter 1:

Purpose 1: To develop a prediction model for acute LAS injuries in a previously unstudied population (collegiate women's soccer players), utilizing primary outcomes of dynamic postural control and isometric hip strength as well as secondary demographic outcomes as potential predictors.

Hypothesis 1: Collegiate women's soccer players with lower baseline dynamic postural control performance and isometric hip strength as well as increased height, body mass, and body mass index (BMI) will have greater estimated odds of sustaining a LAS during the subsequent competitive sport season

> Finding: Greater height was a significant risk factor for LAS in collegiate women's soccer players. Dynamic postural control measured with the SEBT-ANT, isometric hip extension strength, age, body mass, and BMI provided no predictive value for LASs in that population.

Purpose 2: To describe the presence of residual structural and functional impairments and activity limitations in athletes with an acute LAS following clearance for RTP.

Hypothesis 2.1: Patients will exhibit greater ankle swelling and ankle ligamentous laxity and lower dorsiflexion range of motion and dynamic postural control performance in the involved limb compared to the uninvolved limb at RTP. Additionally, patients will self-report meaningful degrees of pain and activity limitations in the involved limb at RTP.

Finding: Patients with an acute LAS presented with residual impairments and activity limitations related to self-reported function, dorsiflexion ROM, ankle joint laxity, and dynamic postural control at the time of RTP. Pain and ankle joint swelling were also commonly present, but not to a clinically meaningful degree.

Hypothesis 2.2: Patients with lower injury severity and more days of immobilization and supervised therapeutic exercise sessions will demonstrate lower pain, ankle swelling and ankle ligamentous laxity, and greater dorsiflexion range of motion, dynamic postural control performance, and self-reported function at RTP.

Finding: Patients with higher injury severity presented with greater swelling and dorsiflexion ROM asymmetries at RTP than those with lower injury severity. Greater days of immobilization was associated greater swelling and dynamic balance asymmetries, but lower dorsiflexion ROM asymmetries at RTP. A greater number of therapeutic exercise sessions was associated with greater self-reported function, but greater dynamic balance asymmetries at RTP.

Purpose 3: To develop a prediction model for recurrent ankle sprains in athletes, utilizing assessments of structural and functional impairments and activity limitations at RTP as potential predictors.

Hypothesis 3.1: Patients with greater ankle joint pain, ankle swelling, and ankle ligamentous laxity and lower dorsiflexion range of motion, dynamic postural control, and

self-reported function and instability at RTP will have greater estimated odds of sustaining a recurrent ankle sprain during the same competitive sport season.

Finding: Clinical measures of ankle joint pain, swelling, ligamentous laxity, dorsiflexion range of motion, dynamic postural control, and self-reported function and instability at RTP provided no predictive value for recurrent ankle sprains in athletes at RTP following a previous ankle sprain in the same competitive sport season.

Hypothesis 3.2: Patients with greater age, height, mass, BMI, injury grade, percentage of season remaining, previous injury history, and DRTP and lower days of immobilization, therapeutic exercise sessions, and use of prophylactic ankle supports for RTP will have greater estimated odds of sustaining a recurrent ankle sprain during the same competitive sport season.

Finding: Greater height and mass were strong predictors of recurrent ankle sprains in athletes during the same competitive season. Age, BMI, injury grade, percentage of season remaining, previous injury history, DRTP, days of immobilization, therapeutic exercise sessions, and use of prophylactic ankle supports were not significant predictors of recurrent ankle sprain during the same competitive sport season.

Synthesis and Application of Results

The first study builds upon a large body of work that has aimed to predict acute LASs in athletes. Our study is the first to produce a LAS prediction model in collegiate women's soccer players, a population that has among the highest risk for LASs.[7,185] We found that those participants with height over 167.6 cm were at greater risk of sustaining

a LAS during the course of the season. The benefit of this finding is that clinical settings with limited preventative resources will be able to target this subset of the population that is at higher risk for LAS. While height itself is not modifiable, alternative interventions such as prophylactic ankle supports[20] and postural control training[205,228] are viable options for LAS prevention in taller athletes. Dynamic postural control was apparently not deficient in our cohort, as the SEBT-ANT may have not been the best measure of dynamic postural control in collegiate women's soccer players. However, evidence exists that postural control training is an effective prevention and intervention strategy for LAS.[21,114,229] Perhaps other SEBT reach directions may be better identifiers of LAS risk in certain populations.[24] Similarly, we found HEXT to have no predictive value for LAS in our sample, but other measures of hip strength (abduction, external rotation) may have been more relevant to LAS risk.[122,183] Future research should examine the predictive utility of various measures of postural control and muscular strength, as well as other modifiable clinical outcomes in collegiate women's soccer players.

Many useful LAS prediction models such as ours for collegiate women's soccer players exist, but none can perfectly identify risk from baseline clinical impairments. However, LASs are commonly repetitive,[216] so prediction of recurrent injuries may protect against the long-term consequences of LASs. An acute LAS can produce a number of impairments and activity limitations that may persist beyond RTP, potentially increasing risk of LAS. Thus, in the second study, we aimed to identify which structural and functional impairments and activity limitations most consistently last in athletes past RTP. We found that high school and collegiate athletes commonly present with residual impairments and activity limitations related to self-reported function, dorsiflexion ROM,

ankle joint laxity, and dynamic postural control at the time of RTP. However, the presence of residual sequelae at RTP was not influenced by greater injury severity, days of immobilization, or number of therapeutic exercise sessions in all cases. These findings suggest there is a pattern of ATs addressing numerous clinical outcomes insufficiently in athletes before RTP. However, we could not determine the impact of these deficiencies on long-term consequences from these findings, so we recommend that clinicians aim to resolve all residual structural and functional impairments and activity limitations in athletes prior to RTP. Until the relevance of all sequelae are established, care for a LAS may be optimized by assessing each impairment and limitation of the patient, then designing an individualized treatment protocol based on the evaluation.[215]

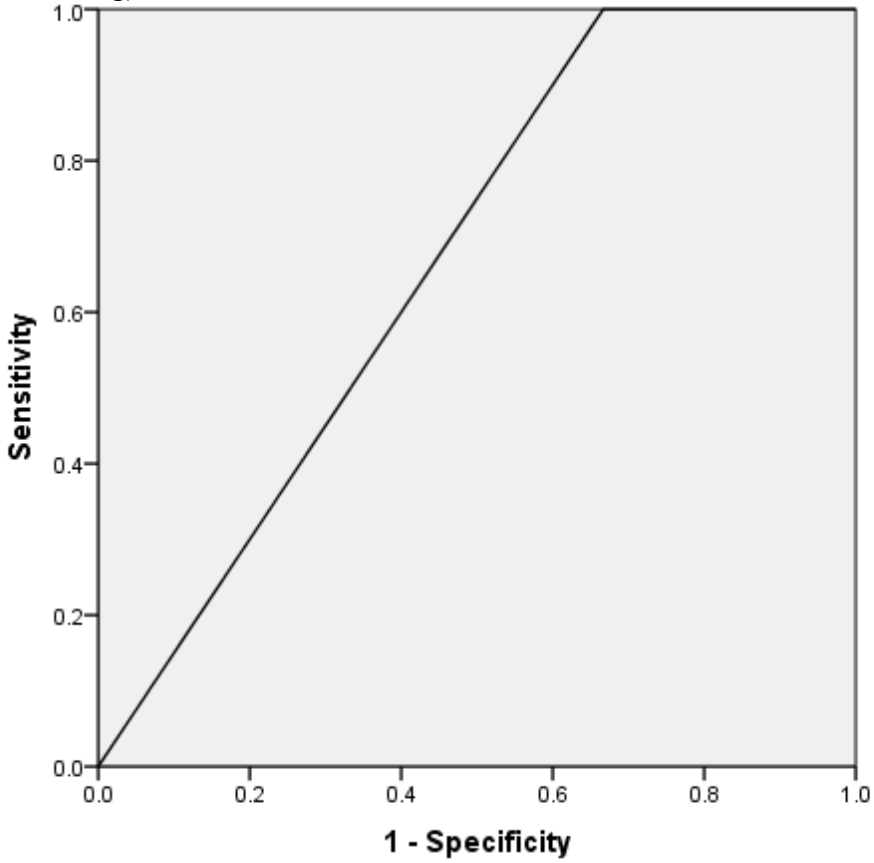
After confirming that athletes commonly RTP without a complete resolution of impairments and limitations, the third study aimed to determine the ability of those outcomes to predict a recurrent ankle sprain in the same competitive season. Previous investigators have identified injury severity, dynamic postural control, and self-reported function as potential predictors of recurrent ankle sprains or CAI,[52,53] but none have done so in high school and collegiate athletes. In our sample, we found that clinical measures of pain, swelling, ligamentous laxity, dorsiflexion ROM, dynamic postural control, and self-reported function and instability at RTP did not predict recurrent ankle sprains during the same competitive sport season in athletes. However, our patients in RI and NRI groups both had dorsiflexion ROM, dynamic postural control, and self-reported function and instability resembling that of patients with CAI, potentially indicating that longer follow-up periods are needed to expose patients to risk and realize the full predictive value of these outcomes. Despite finding no predictive value in the primary outcomes,

increased height and mass were strong risk factors for recurrent ankle sprains. Others have reported similar findings, suggesting that increased physical stature leads to increased inertial resistance of the ankle joint and reduced ability to reverse momentum in the presence of an external inversion or eversion moment.[31,43] We recommend that larger athletes that have sustained an ankle sprain undergo additional care before RTP in order to preventative a recurrent injury. Weight and BMI can be safely modified in athletes,[227] but in those athletes that may be negatively affected (i.e. football linemen), clinicians should utilize alternative means of preventing ankle sprains and correcting CAI, such as prophylactic ankle supports[20] and postural control training.[177,205,228]

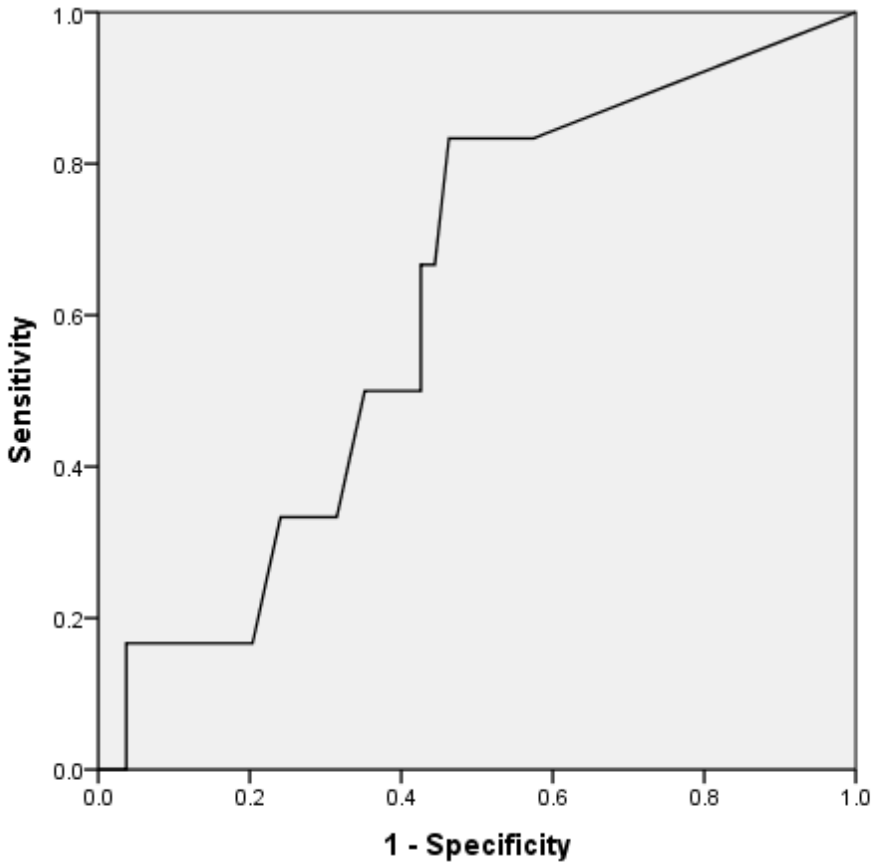
In conclusion, we found that increased height was a risk factor for acute ankle sprains and increased height and mass were risk factors for recurrent ankle sprains in athletes. Those athletes exhibiting such characteristics should undergo additional care to prevent long-term consequences of ankle sprains. While ankle sprains are associated with a number of other impairments and activity limitations at RTP, they are not predictive of recurrent ankle sprains in the same competitive sport season. However, the impact of those outcomes on recurrent ankle sprains over a longer period remains unknown. Future investigations are needed to investigate expanded timelines and understand the sequela of chronicity development in athletes that sustain an ankle sprain.

Appendix A – ROC Curves for Primary Outcomes in Chapter 5

Pain (non-weight-bearing) ROC Curve



Pain (single-leg stance) ROC Curve



Pain (4 steps) ROC Curve

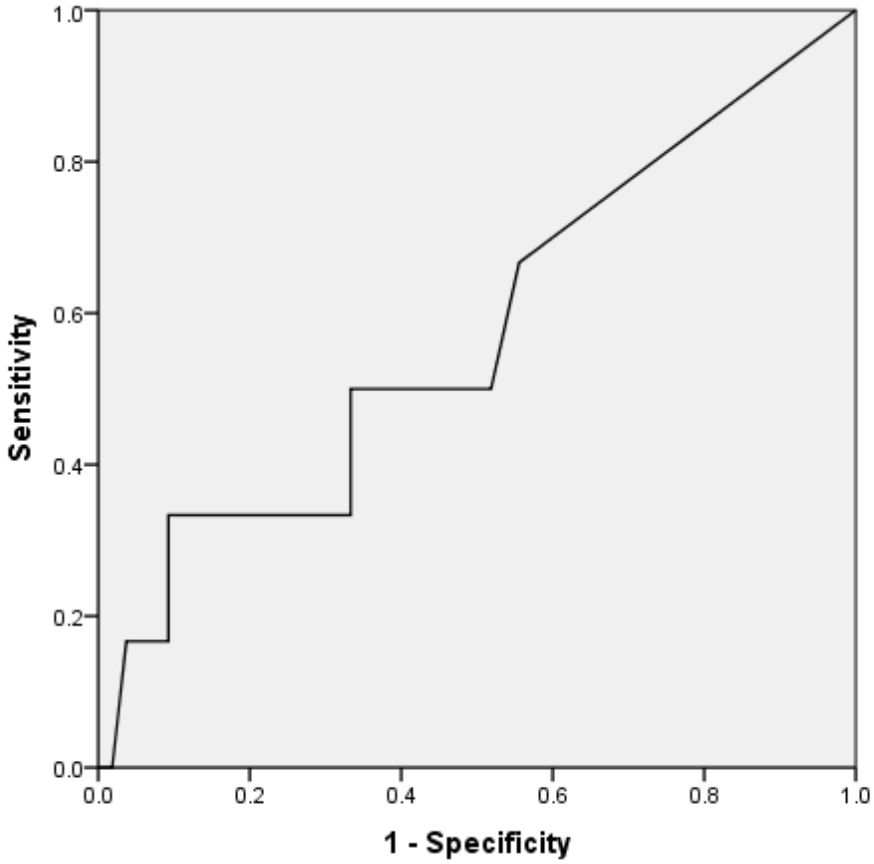


Figure-of-8 (involved) ROC Curve

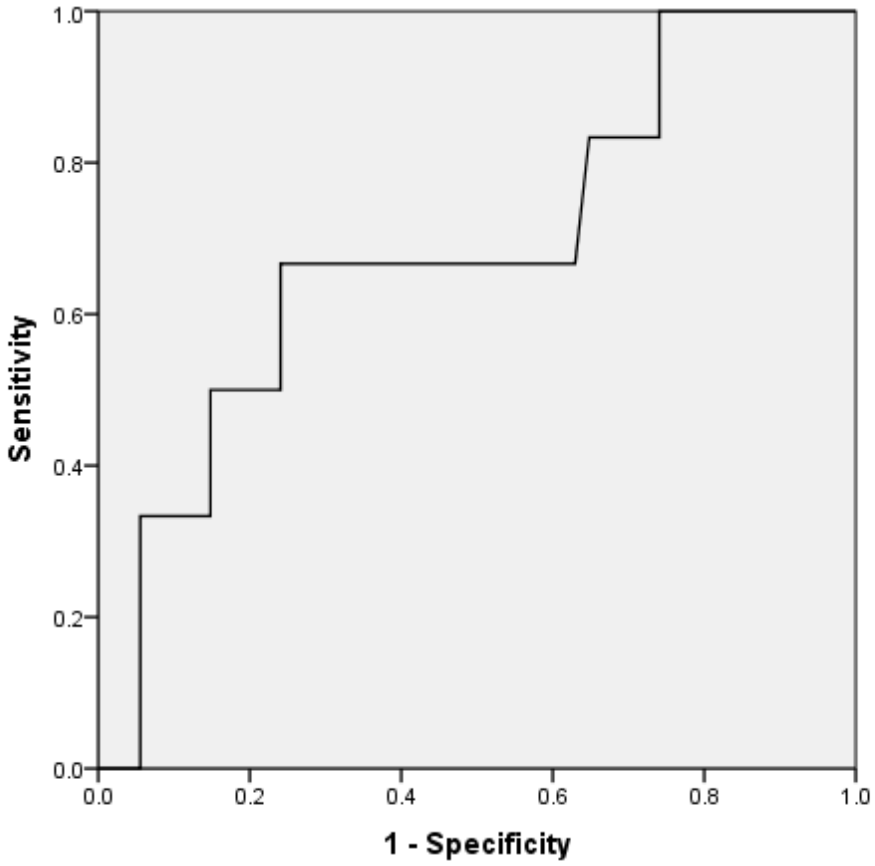
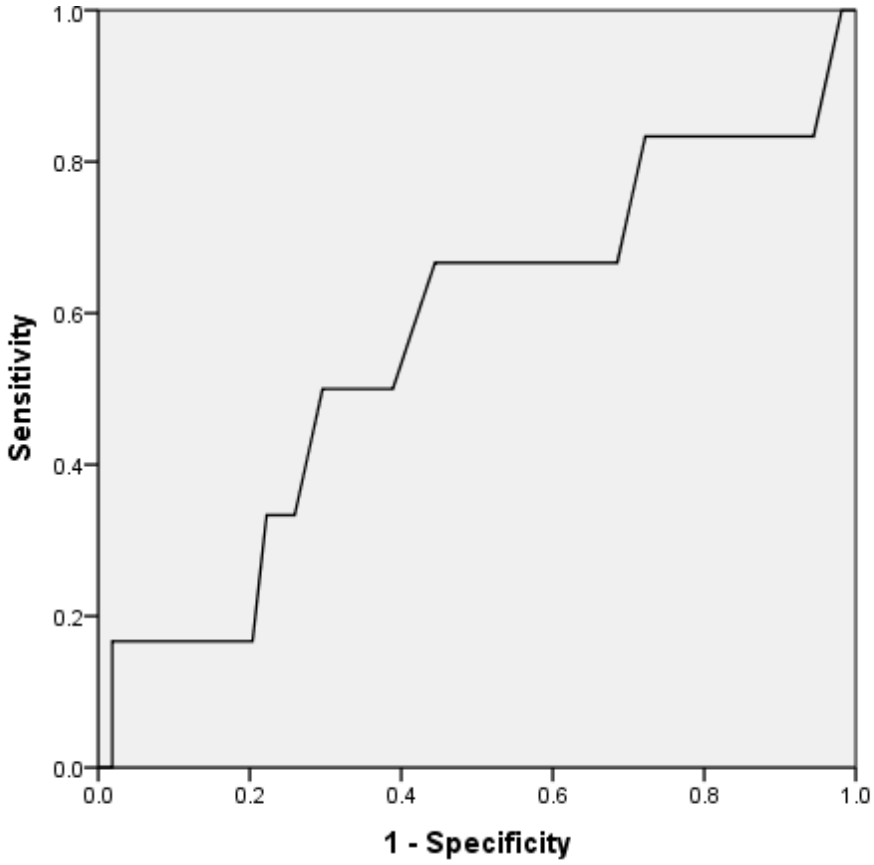
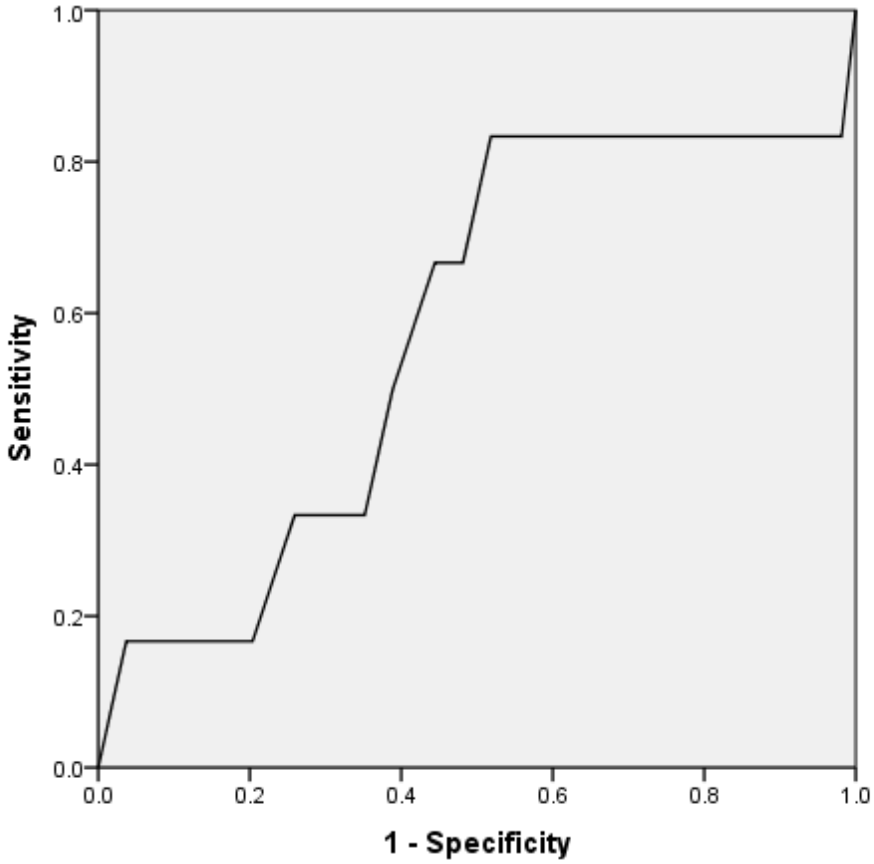


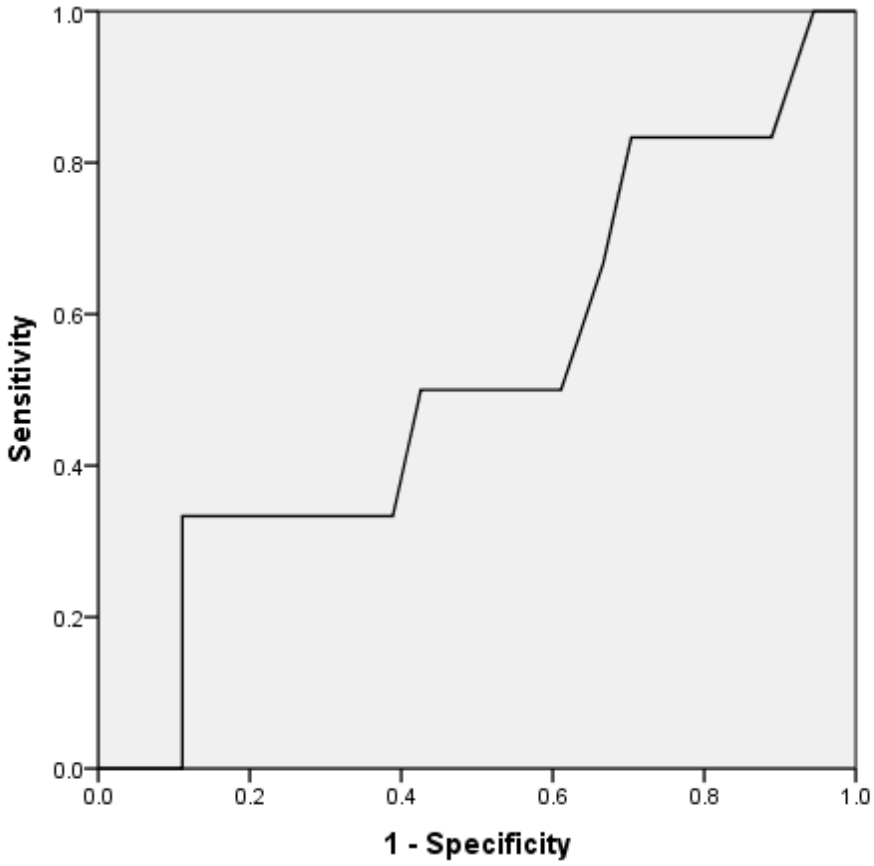
Figure-of-8 Asymmetry ROC Curve



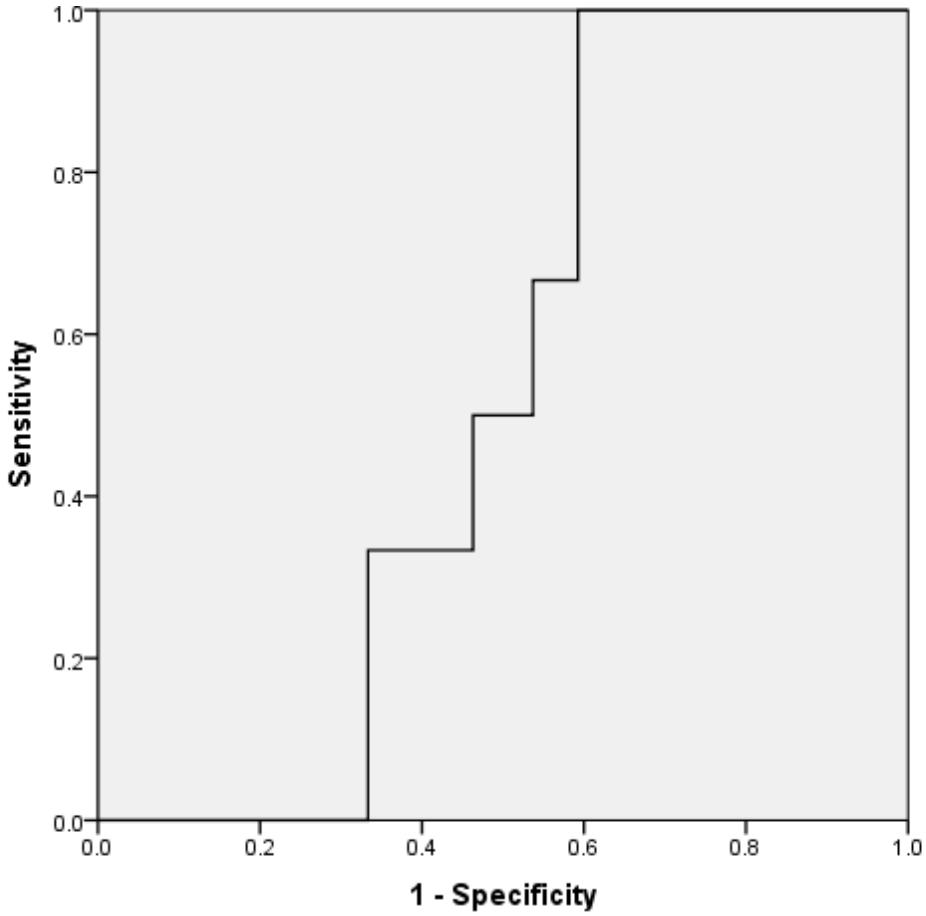
WBLT (involved) ROC Curve



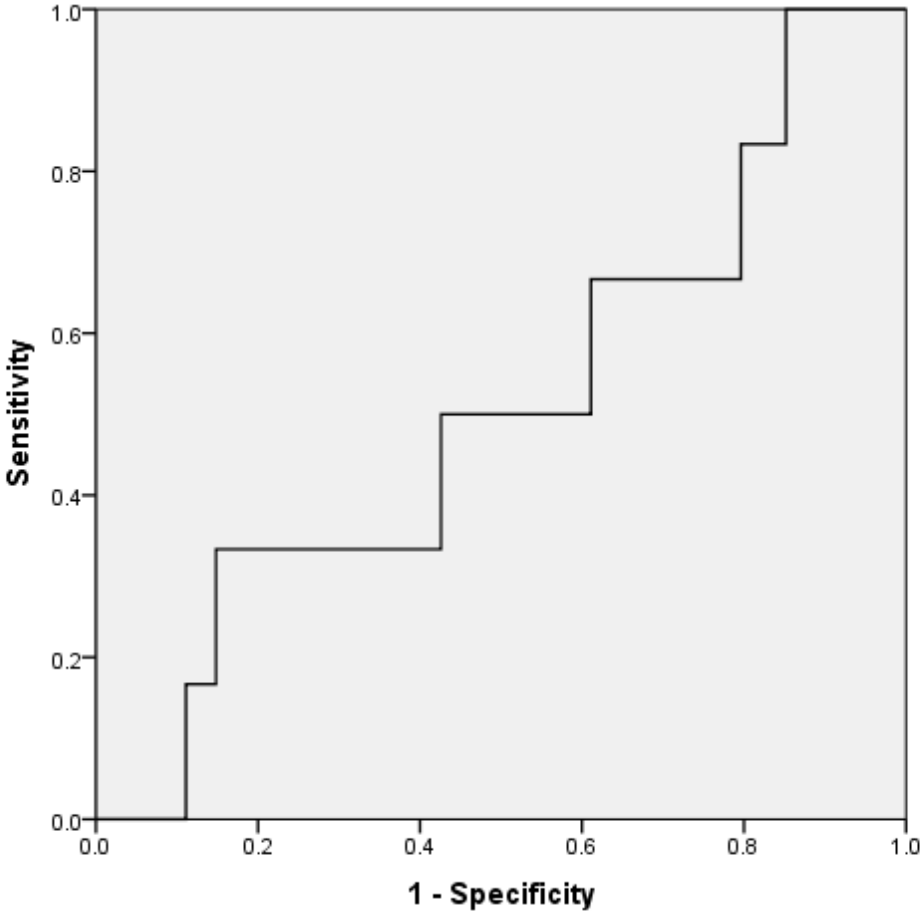
WBLT Asymmetry ROC Curve



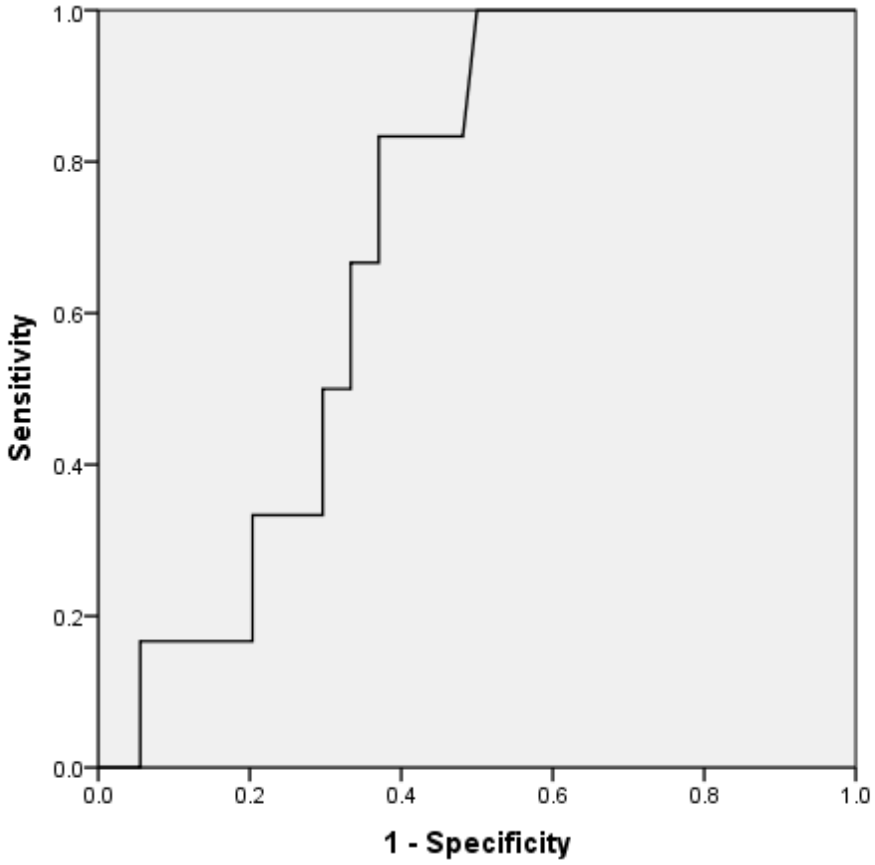
SEBT-ANT (involved) ROC Curve



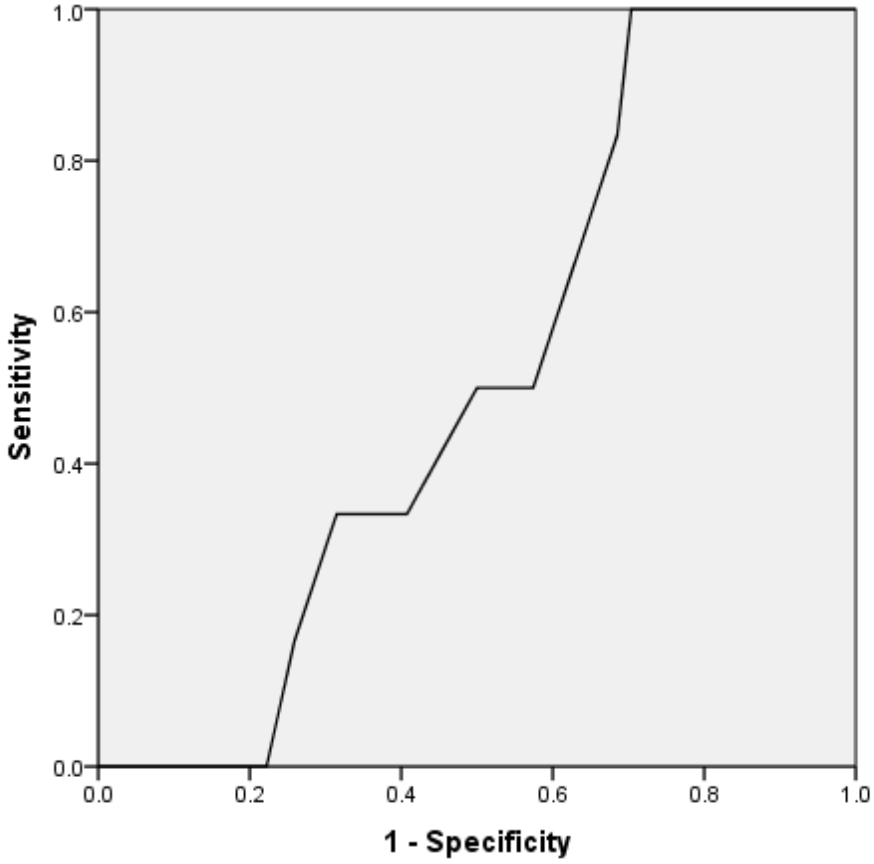
SEBT-ANT Asymmetry ROC Curve



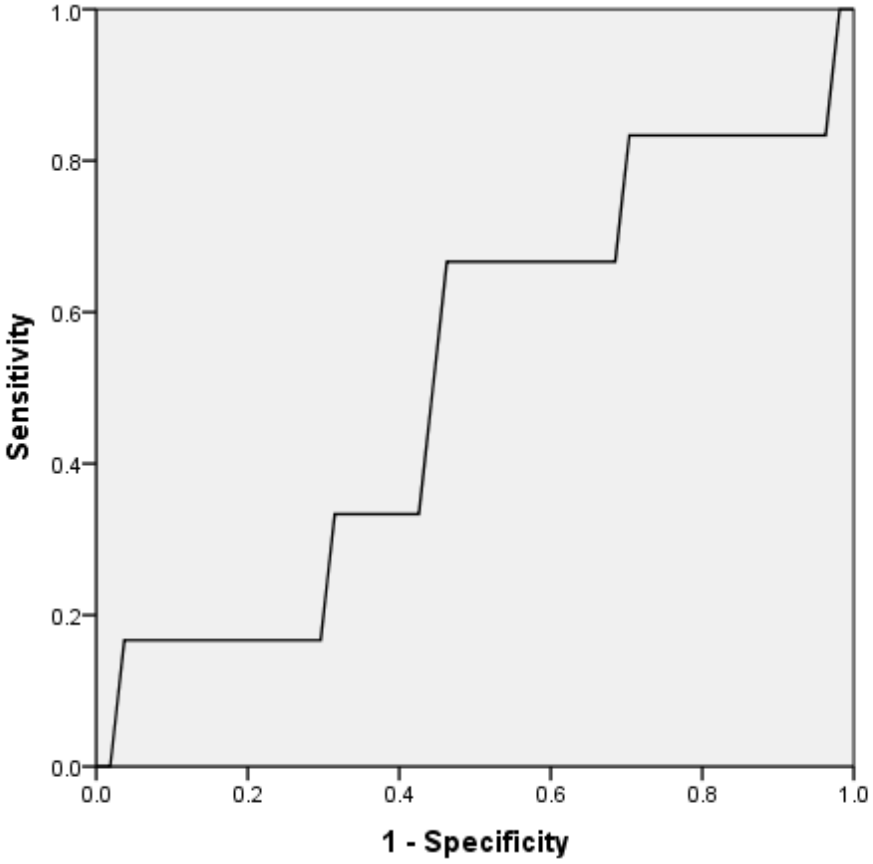
FAAM-ADL ROC Curve



FAAM-S ROC Curve



IdFAI ROC Curve



References

1. Shah S, Thomas AC, Noone JM, Blanchette CM, Wikstrom EW. Incidence and Cost of Ankle Sprains in United States Emergency Departments. *Sport Health.* 2016;8(6):547-552.
2. Waterman BR, Owens BD, Davey S, Zacchilli MA, Belmont PJ, Jr. The epidemiology of ankle sprains in the United States. *J Bone Joint Surg Am.* 2010;92(13):2279-2284.
3. *NFHS Handbook 2015-16.* Indianapolis, IN: National Federation of State High School Associations; 2015.
4. *1981-82–2014-15 NCAA Sports Sponsorship and Participation Rates Report.* Indianapolis, IN: National Collegiate Athletic Association;2015.
5. Nelson AJ, Collins CL, Yard EE, Fields SK, Comstock RD. Ankle injuries among United States high school sports athletes, 2005-2006. *J Athl Train.* 2007;42(3):381-387.
6. Fong DT, Hong Y, Chan LK, Yung PS, Chan KM. A systematic review on ankle injury and ankle sprain in sports. *Sports Med.* 2007;37(1):73-94.
7. Hootman JM, Dick R, Agel J. Epidemiology of collegiate injuries for 15 sports: summary and recommendations for injury prevention initiatives. *J Athl Train.* 2007;42(2):311-319.
8. Doherty C, Delahunt E, Caulfield B, Hertel J, Ryan J, Bleakley C. The Incidence and Prevalence of Ankle Sprain Injury: A Systematic Review and Meta-Analysis of Prospective Epidemiological Studies. *Sports Med.* 2013;44(1):123-140.
9. Feger MA, Glaviano NR, Donovan L, et al. Current Trends in the Management of Lateral Ankle Sprain in the United States. *Clin J Sport Med.* In Press.
10. Konradsen L, Bech L, Ehrenbjerg M, Nickelsen T. Seven years follow-up after ankle inversion trauma. *Scand J Med Sci Sports.* 2002;12(3):129-135.
11. Braun BL. Effects of ankle sprain in a general clinic population 6 to 18 months after medical evaluation. *Arch Fam Med.* 1999;8(2):143-148.
12. Gribble PA, Delahunt E, Bleakley C, et al. Selection criteria for patients with chronic ankle instability in controlled research: a position statement of the International Ankle Consortium. *Br J Sports Med.* 2014;48(13):1014-1018.
13. Gribble PA, Delahunt E, Bleakley C, et al. Selection criteria for patients with chronic ankle instability in controlled research: a position statement of the International Ankle Consortium. *J Orthop Sports Phys Ther.* 2013;43(8):585-591.
14. Gribble PA, Delahunt E, Bleakley CM, et al. Selection criteria for patients with chronic ankle instability in controlled research: a position statement of the International Ankle Consortium. *J Athl Train.* 2014;49(1):121-127.
15. Anandacoomarasamy A, Barnsley L. Long term outcomes of inversion ankle injuries. *Br J Sports Med.* 2005;39(3):e14.
16. Tanen L, Docherty CL, Van Der Pol B, Simon J, Schrader J. Prevalence of chronic ankle instability in high school and division I athletes. *Foot Ankle Spec.* 2014;7(1):37-44.
17. Houston MN, Van Lunen BL, Hoch MC. Health-related quality of life in individuals with chronic ankle instability. *J Athl Train.* 2014;49(6):758-763.

18. Punt IM, Ziltener JL, Laidet M, Armand S, Allet L. Gait and physical impairments in patients with acute ankle sprains who did not receive physical therapy. *PM R.* 2014;7(1):34-41.
19. Valderrabano V, Hintermann B, Horisberger M, Fung TS. Ligamentous posttraumatic ankle osteoarthritis. *Am J Sports Med.* 2006;34(4):612-620.
20. Dizon JM, Reyes JJ. A systematic review on the effectiveness of external ankle supports in the prevention of inversion ankle sprains among elite and recreational players. *J Sci Med Sport.* 2010;13(3):309-317.
21. Schiftan GS, Ross LA, Hahne AJ. The effectiveness of proprioceptive training in preventing ankle sprains in sporting populations: a systematic review and meta-analysis. *J Sci Med Sport.* 2015;18(3):238-244.
22. McGuine TA, Hetzel S, Pennuto A, Brooks A. Basketball coaches' utilization of ankle injury prevention strategies. *Sport Health.* 2013;5(5):410-416.
23. Arnason A, Sigurdsson SB, Gudmundsson A, Holme I, Engebretsen L, Bahr R. Risk factors for injuries in football. *Am J Sports Med.* 2004;32(1 Suppl):5S-16S.
24. de Noronha M, Franca LC, Haupenthal A, Nunes GS. Intrinsic predictive factors for ankle sprain in active university students: a prospective study. *Scand J Med Sci Sports.* 2013;23(5):541-547.
25. Ekstrand J, Gillquist J. Soccer injuries and their mechanisms: a prospective study. *Med Sci Sports Exerc.* 1983;15(3):267-270.
26. Engebretsen AH, Myklebust G, Holme I, Engebretsen L, Bahr R. Intrinsic risk factors for acute ankle injuries among male soccer players: a prospective cohort study. *Scand J Med Sci Sports.* 2010;20(3):403-410.
27. Hiller CE, Refshauge KM, Herbert RD, Kilbreath SL. Intrinsic predictors of lateral ankle sprain in adolescent dancers: a prospective cohort study. *Clin J Sport Med.* 2008;18(1):44-48.
28. Kofotolis ND, Kellis E, Vlachopoulos SP. Ankle sprain injuries and risk factors in amateur soccer players during a 2-year period. *Am J Sports Med.* 2007;35(3):458-466.
29. McHugh MP, Tyler TF, Tetro DT, Mullaney MJ, Nicholas SJ. Risk factors for noncontact ankle sprains in high school athletes: the role of hip strength and balance ability. *Am J Sports Med.* 2006;34(3):464-470.
30. McKay GD, Goldie PA, Payne WR, Oakes BW. Ankle injuries in basketball: injury rate and risk factors. *Br J Sports Med.* 2001;35(2):103-108.
31. Tyler TF, McHugh MP, Mirabella MR, Mullaney MJ, Nicholas SJ. Risk factors for noncontact ankle sprains in high school football players: the role of previous ankle sprains and body mass index. *Am J Sports Med.* 2006;34(3):471-475.
32. Baumhauer JF, Alosa DM, Renstrom AF, Trevino S, Beynnon B. A prospective study of ankle injury risk factors. *Am J Sports Med.* 1995;23(5):564-570.
33. Beynnon BD, Renstrom PA, Alosa DM, Baumhauer JF, Vacek PM. Ankle ligament injury risk factors: a prospective study of college athletes. *J Orthop Res.* 2001;19(2):213-220.
34. Henry T, Evans K, Snodgrass SJ, Miller A, Callister R. Risk Factors for Noncontact Ankle Injuries in Amateur Male Soccer Players: A Prospective Cohort Study. *Clin J Sport Med.* 2016;26(3):251-258.

35. Payne KA, Berg K, Latin RW. Ankle injuries and ankle strength, flexibility, and proprioception in college basketball players. *J Athl Train.* 1997;32(3):221-225.
36. Pope R, Herbert R, Kirwan J. Effects of ankle dorsiflexion range and pre-exercise calf muscle stretching on injury risk in Army recruits. *Aust J Physiother.* 1998;44(3):165-172.
37. Wang HK, Chen CH, Shiang TY, Jan MH, Lin KH. Risk-factor analysis of high school basketball-player ankle injuries: a prospective controlled cohort study evaluating postural sway, ankle strength, and flexibility. *Arch Phys Med Rehabil.* 2006;87(6):821-825.
38. 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. *Am J Sports Med.* 2005;33(3):415-423.
39. Willems TM, Witvrouw E, Delbaere K, Philippaerts R, De Bourdeaudhuij I, De Clercq D. Intrinsic risk factors for inversion ankle sprains in females--a prospective study. *Scand J Med Sci Sports.* 2005;15(5):336-345.
40. Fousekis K, Tsepis E, Vagenas G. Intrinsic risk factors of noncontact ankle sprains in soccer: a prospective study on 100 professional players. *Am J Sports Med.* 2012;40(8):1842-1850.
41. Gribble PA, Terada M, Beard MQ, et al. Prediction of lateral ankle sprain risk in football players using clinical modifiable factors. *Am J Sports Med.* 2016;44(2):460-467.
42. McGuine TA, Greene JJ, Best T, Leverson G. Balance as a predictor of ankle injuries in high school basketball players. *Clin J Sport Med.* 2000;10(4):239-244.
43. Milgrom C, Shlamkovitch N, Finestone A, et al. Risk factors for lateral ankle sprain: a prospective study among military recruits. *Foot Ankle.* 1991;12(1):26-30.
44. Witchalls J, Blanch P, Waddington G, Adams R. Intrinsic functional deficits associated with increased risk of ankle injuries: a systematic review with meta-analysis. *Br J Sports Med.* 2012;46(7):515-523.
45. Butler RJ, Southers C, Gorman PP, Kiesel KB, Plisky PJ. Differences in soccer players' dynamic balance across levels of competition. *J Athl Train.* 2012;47(6):616-620.
46. McCann RS, Kosik KB, Beard MQ, Terada M, Pietrosimone BG, Gribble PA. Variations in Star Excursion Balance Test Performance Between High School and Collegiate Football Players. *J Strength Cond Res.* 2015;29(10):2765-2770.
47. Stiffler MR, Sanfilippo JL, Brooks MA, Heiderscheit BC. Star Excursion Balance Test Performance Varies by Sport in Healthy Division I Collegiate Athletes. *J Orthop Sports Phys Ther.* 2015;45(10):772-780.
48. de Ridder R, Witvrouw E, Dolphens M, Roosen P, Van Ginckel A. Hip Strength as an Intrinsic Risk Factor for Lateral Ankle Sprains in Youth Soccer Players: A 3-Season Prospective Study. *Am J Sports Med.* 2016: In Press.
49. Medina McKeon JM, Bush HM, Reed A, Whittington A, Uhl TL, McKeon PO. Return-to-play probabilities following new versus recurrent ankle sprains in high school athletes. *J Sci Med Sport.* 2014;17(1):23-28.
50. Endele D, Jung C, Bauer G, Mauch F. Value of MRI in diagnosing injuries after ankle sprains in children. *Foot Ankle Int.* 2012;33(12):1063-1068.

51. Malliaropoulos N, Ntessalen M, Papacostas E, Longo UG, Maffulli N. Reinjury after acute lateral ankle sprains in elite track and field athletes. *Am J Sports Med.* 2009;37(9):1755-1761.
52. Pourkazemi F, Hiller CE, Raymond J, Nightingale EJ, Refshauge KM. Predictors of chronic ankle instability after an index lateral ankle sprain: a systematic review. *J Sci Med Sport.* 2014;17(6):568-573.
53. Doherty C, Bleakley C, Hertel J, Caulfield B, Ryan J, Delahunt E. Recovery From a First-Time Lateral Ankle Sprain and the Predictors of Chronic Ankle Instability: A Prospective Cohort Analysis. *Am J Sports Med.* 2016;44(4):995-1003.
54. O'Connor SR, Bleakley CM, Tully MA, McDonough SM. Predicting functional recovery after acute ankle sprain. *PloS one.* 2013;8(8):e72124.
55. Gerber JP, Williams GN, Scoville CR, Arciero RA, Taylor DC. Persistent disability associated with ankle sprains: a prospective examination of an athletic population. *Foot Ankle Int.* 1998;19(10):653-660.
56. Roos KG, Kerr ZY, Mauntel TC, Djoko A, Dompier TP, Wikstrom EA. The Epidemiology of Lateral Ligament Complex Ankle Sprains in National Collegiate Athletic Association Sports. *Am J Sports Med.* In Press.
57. Shankar PR, Fields SK, Collins CL, Dick RW, Comstock RD. Epidemiology of high school and collegiate football injuries in the United States, 2005-2006. *Am J Sports Med.* 2007;35(8):1295-1303.
58. Swenson DM, Collins CL, Fields SK, Comstock RD. Epidemiology of U.S. high school sports-related ligamentous ankle injuries, 2005/06-2010/11. *Clin J Sport Med.* 2013;23(3):190-196.
59. Swenson DM, Yard EE, Fields SK, Comstock RD. Patterns of recurrent injuries among US high school athletes, 2005-2008. *Am J Sports Med.* 2009;37(8):1586-1593.
60. Fuller EA. Center of pressure and its theoretical relationship to foot pathology. *J Am Podiatr Med Assoc.* 1999;89(6):278-291.
61. Whiting WC, Zernicke RF. *Biomechanics of Musculoskeltal Injury.* 2nd ed. Champaign, IL: Human Kinetics; 2008.
62. Ashton-Miller JA, Ottaviani RA, Hutchinson C, Wojtys EM. What best protects the inverted weightbearing ankle against further inversion? Evertor muscle strength compares favorably with shoe height, athletic tape, and three orthoses. *Am J Sports Med.* 1996;24(6):800-809.
63. Konradsen L, Voigt M, Hojsgaard C. Ankle inversion injuries. The role of the dynamic defense mechanism. *Am J Sports Med.* 1997;25(1):54-58.
64. Vaes P, Duquet W, Van Gheluwe B. Peroneal Reaction Times and Eversion Motor Response in Healthy and Unstable Ankles. *J Athl Train.* 2002;37(4):475-480.
65. Fong DT, Ha SC, Mok KM, Chan CW, Chan KM. Kinematics analysis of ankle inversion ligamentous sprain injuries in sports: five cases from televised tennis competitions. *Am J Sports Med.* 2012;40(11):2627-2632.
66. Fong DT, Hong Y, Shima Y, Krosshaug T, Yung PS, Chan KM. Biomechanics of supination ankle sprain: a case report of an accidental injury event in the laboratory. *Am J Sports Med.* 2009;37(4):822-827.

67. Gehring D, Wissler S, Mornieux G, Gollhofer A. How to sprain your ankle - a biomechanical case report of an inversion trauma. *J Biomech.* 2013;46(1):175-178.
68. Kristianslund E, Bahr R, Krosshaug T. Kinematics and kinetics of an accidental lateral ankle sprain. *J Biomech.* 2011;44(14):2576-2578.
69. Mok KM, Fong DT, Krosshaug T, et al. Kinematics analysis of ankle inversion ligamentous sprain injuries in sports: 2 cases during the 2008 Beijing Olympics. *Am J Sports Med.* 2011;39(7):1548-1552.
70. Terada M, Gribble PA. Jump Landing Biomechanics During a Laboratory Recorded Recurrent Ankle Sprain. *Foot Ankle Int.* 2015;36(7):842-848.
71. Fallat L, Grimm DJ, Saracco JA. Sprained ankle syndrome: prevalence and analysis of 639 acute injuries. *J Foot Ankle Surg.* 1998;37(4):280-285.
72. Scott DT, Lam FY, Ferrell WR. Acute joint inflammation--mechanisms and mediators. *Gen Pharmacol.* 1994;25(7):1285-1296.
73. Besson JM. The neurobiology of pain. *Lancet.* 1999;353(9164):1610-1615.
74. Carr DB, Goudas LC. Acute pain. *Lancet.* 1999;353(9169):2051-2058.
75. Derbyshire SW, Jones AK, Gyulai F, Clark S, Townsend D, Firestone LL. Pain processing during three levels of noxious stimulation produces differential patterns of central activity. *Pain.* 1997;73(3):431-445.
76. Nilsson S. Sprains of the lateral ankle ligaments. *J Oslo City Hosp.* 1983;33(2-3):13-36.
77. de Bie RA, de Vet HC, van den Wildenberg FA, Lenssen T, Knipschild PG. The prognosis of ankle sprains. *Int J Sports Med.* 1997;18(4):285-289.
78. Zammit E, Herrington L. Ultrasound therapy in the management of acute lateral ligament sprains of the ankle joint. *Phys Ther Sport.* 2005;6:116-121.
79. Boyce SH, Quigley MA, Campbell S. Management of ankle sprains: a randomised controlled trial of the treatment of inversion injuries using an elastic support bandage or an Aircast ankle brace. *Br J Sports Med.* 2005;39(2):91-96.
80. O'Hara J, Valle-Jones JC, Walsh H, et al. Controlled trial of an ankle support (Malleotrain) in acute ankle injuries. *Br J Sports Med.* 1992;26(3):139-142.
81. Eisenhart AW, Gaeta TJ, Yens DP. Osteopathic manipulative treatment in the emergency department for patients with acute ankle injuries. *J Am Osteopath Assoc.* 2003;103(9):417-421.
82. Bleakley CM, O'Connor SR, Tully MA, et al. Effect of accelerated rehabilitation on function after ankle sprain: randomised controlled trial. *BMJ.* 2010;340:c1964.
83. van Rijn RM, Willemsen SP, Verhagen AP, Koes BW, Bierma-Zeinstra SM. Explanatory variables for adult patients' self-reported recovery after acute lateral ankle sprain. *Phys Ther.* 2011;91(1):77-84.
84. Verhagen RA, de Keizer G, van Dijk CN. Long-term follow-up of inversion trauma of the ankle. *Arch Orthop Trauma Surg.* 1995;114(2):92-96.
85. van Rijn RM, van Os AG, Bernsen RM, Luijsterburg PA, Koes BW, Bierma-Zeinstra SM. What is the clinical course of acute ankle sprains? A systematic literature review. *Am J Med.* 2008;121(4):324-331 e326.
86. Wright CJ, Arnold BL, Ross SE, Ketchum J, Ericksen J, Pidcoe P. Clinical examination results in individuals with functional ankle instability and ankle-sprain copers. *J Athl Train.* 2013;48(5):581-589.

87. Smith C, Kruger MJ, Smith RM, Myburgh KH. The Inflammatory Response to Skeletal Muscle Injury: Illuminating Complexities. *Sports Med.* 2008;38(11):947-969.
88. Pugia ML, Middel CJ, Seward SW, et al. Comparison of acute swelling and function in subjects with lateral ankle injury. *J Orthop Sports Phys Ther.* 2001;31(7):384-388.
89. Staples OS. Ruptures of the fibular collateral ligaments of the ankle. Result study of immediate surgical treatment. *J Bone Joint Surg Am.* 1975;57(1):101-107.
90. Hubbard TJ, Hicks-Little CA. Ankle ligament healing after an acute ankle sprain: an evidence-based approach. *J Athl Train.* 2008;43(5):523-529.
91. Denegar CR, Hertel J, Fonseca J. The effect of lateral ankle sprain on dorsiflexion range of motion, posterior talar glide, and joint laxity. *J Orthop Sports Phys Ther.* 2002;32(4):166-173.
92. Hertel J, Denegar CR, Monroe MM, Stokes WL. Talocrural and subtalar joint instability after lateral ankle sprain. *Med Sci Sports Exerc.* 1999;31(11):1501-1508.
93. McCann RS, Kosik KB, Terada M, Gribble PA. Residual Impairments and Activity Limitations at Return to Play from a Lateral Ankle Sprain. *Sport Health.* Submitted February 2017: In Review.
94. Broström L. Sprained ankles. V. Treatment and prognosis in recent ligament ruptures. *Acta Chir Scand.* 1966;132(5):537-550.
95. Hubbard TJ, Kramer LC, Denegar CR, Hertel J. Contributing factors to chronic ankle instability. *Foot Ankle Int.* 2007;28(3):343-354.
96. Lentell G, Baas B, Lopez D, McGuire L, Sarrels M, Snyder P. The contributions of proprioceptive deficits, muscle function, and anatomic laxity to functional instability of the ankle. *J Orthop Sports Phys Ther.* 1995;21(4):206-215.
97. Louwerens JW, Ginai AZ, van Linge B, Snijders CJ. Stress radiography of the talocrural and subtalar joints. *Foot Ankle Int.* 1995;16(3):148-155.
98. 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.* 2010;20(1):e137-144.
99. Youdas JW, McLean TJ, Krause DA, Hollman JH. Changes in active ankle dorsiflexion range of motion after acute inversion ankle sprain. *J Sport Rehabil.* 2009;18(3):358-374.
100. Vicenzino B, Branjerdporn M, Teys P, Jordan K. Initial changes in posterior talar glide and dorsiflexion of the ankle after mobilization with movement in individuals with recurrent ankle sprain. *J Orthop Sports Phys Ther.* 2006;36(7):464-471.
101. Wikstrom EA, Hubbard TJ. Talar positional fault in persons with chronic ankle instability. *Arch Phys Med Rehabil.* 2010;91(8):1267-1271.
102. Grindstaff TL, Beazell JR, Sauer LD, Magrum EM, Ingersoll CD, Hertel J. Immediate effects of a tibiofibular joint manipulation on lower extremity H-reflex measurements in individuals with chronic ankle instability. *J Electromyogr Kinesiol.* 2011;21(4):652-658.

103. Kavanagh J. Is there a positional fault at the inferior tibiofibular joint in patients with acute or chronic ankle sprains compared to normals? *Man Ther.* 1999;4(1):19-24.
104. Fukuhara T, Sakamoto M, Nakazawa R, Kato K. Anterior Positional Fault of the Fibula after Sub-Acute Anterior Talofibular Ligament Injury. *J Phys Ther Sci.* 2012;24:115-117.
105. Hubbard TJ, Hertel J. Anterior positional fault of the fibula after sub-acute lateral ankle sprains. *Man Ther.* 2008;13(1):63-67.
106. Hubbard TJ, Hertel J, Sherbondy P. Fibular position in individuals with self-reported chronic ankle instability. *J Orthop Sports Phys Ther.* 2006;36(1):3-9.
107. Terada M, Pietrosimone BG, Gribble PA. Therapeutic interventions for increasing ankle dorsiflexion after ankle sprain: a systematic review. *J Athl Train.* 2013;48(5):696-709.
108. Akbari M, Karimi H, Farahini H, Faghihzadeh S. Balance problems after unilateral lateral ankle sprains. *J Rehabil Res Dev.* 2006;43(7):819-824.
109. Doherty C, Bleakley C, Hertel J, Caulfield B, Ryan J, Delahunt E. Postural control strategies during single limb stance following acute lateral ankle sprain. *Clin Biomech (Bristol, Avon).* 2014;29(6):643-649.
110. Doherty C, Bleakley C, Hertel J, Caulfield B, Ryan J, Delahunt E. Balance failure in single limb stance due to ankle sprain injury: an analysis of center of pressure using the fractal dimension method. *Gait Posture.* 2014;40(1):172-176.
111. Doherty C, Bleakley CM, Hertel J, Caulfield B, Ryan J, Delahunt E. Laboratory Measures of Postural Control During the Star Excursion Balance Test After Acute First-Time Lateral Ankle Sprain. *J Athl Train.* 2015;50(6):651-664.
112. Evans T, Hertel J, Sebastianelli W. Bilateral deficits in postural control following lateral ankle sprain. *Foot Ankle Int.* 2004;25(11):833-839.
113. Hertel J, Buckley WE, Denegar CR. Serial Testing of Postural Control After Acute Lateral Ankle Sprain. *J Athl Train.* 2001;36(4):363-368.
114. Holme E, Magnusson SP, Becher K, Bieler T, Aagaard P, Kjaer M. The effect of supervised rehabilitation on strength, postural sway, position sense and re-injury risk after acute ankle ligament sprain. *Scand J Med Sci Sports.* 1999;9(2):104-109.
115. Doherty C, Bleakley C, Hertel J, Caulfield B, Ryan J, Delahunt E. Dynamic Balance Deficits 6 Months Following First-Time Acute Lateral Ankle Sprain: A Laboratory Analysis. *J Orthop Sports Phys Ther.* 2015;45(8):626-633.
116. Doherty C, Bleakley C, Hertel J, et al. Inter-joint coordination strategies during unilateral stance 6-months following first-time lateral ankle sprain. *Clin Biomech (Bristol, Avon).* 2015;30(2):129-135.
117. Doherty C, Bleakley C, Hertel J, Caulfield B, Ryan J, Delahunt E. Dynamic balance deficits in individuals with chronic ankle instability compared to ankle sprain copers 1 year after a first-time lateral ankle sprain injury. *Knee Surg Sports Traumatol Arthrosc.* 2016;24(4):1086-1095.
118. Doherty C, Bleakley C, Hertel J, et al. Lower Limb Interjoint Postural Coordination One Year after First-Time Lateral Ankle Sprain. *Med Sci Sports Exerc.* 2015;47(11):2398-2405.

119. Gribble PA, Hertel J, Plisky P. Using the Star Excursion Balance Test to assess dynamic postural-control deficits and outcomes in lower extremity injury: a literature and systematic review. *J Athl Train.* 2012;47(3):339-357.
120. Hass CJ, Bishop MD, Doidge D, Wikstrom EA. Chronic ankle instability alters central organization of movement. *Am J Sports Med.* 2010;38(4):829-834.
121. Linens SW, Ross SE, Arnold BL, Gayle R, Pidcoe P. Postural-stability tests that identify individuals with chronic ankle instability. *J Athl Train.* 2014;49(1):15-23.
122. McCann RS, Crossett ID, Terada M, Kosik KB, Bolding BA, Gribble PA. Hip strength and star excursion balance test deficits of patients with chronic ankle instability. *J Sci Med Sport.* Submitted July 2016: In Review.
123. Plante JE, Wikstrom EA. Differences in clinician-oriented outcomes among controls, copers, and chronic ankle instability groups. *Phys Ther Sport.* 2013;14(4):221-226.
124. Rios JL, Gorges AL, dos Santos MJ. Individuals with chronic ankle instability compensate for their ankle deficits using proximal musculature to maintain reduced postural sway while kicking a ball. *Hum Mov Sci.* 2015;43:33-44.
125. Wikstrom EA, Fournier KA, McKeon PO. Postural control differs between those with and without chronic ankle instability. *Gait Posture.* 2010;32(1):82-86.
126. Hertel J, Olmsted-Kramer LC. Deficits in time-to-boundary measures of postural control with chronic ankle instability. *Gait Posture.* 2007;25(1):33-39.
127. Freeman MA, Dean MR, Hanham IW. The etiology and prevention of functional instability of the foot. *J Bone Joint Surg Br.* 1965;47(4):678-685.
128. Michelson JD, Hutchins C. Mechanoreceptors in human ankle ligaments. *J Bone Joint Surg Br.* 1995;77(2):219-224.
129. Hertel JN, Guskiewicz KM, Kahler DM, Perrin DH. Effect of Lateral Ankle Joint Anesthesia on Center of Balance, Postural Sway, and Joint Position Sense. *J Sport Rehabil.* 1996;2:111-119.
130. Konradsen L, Ravn JB, Sorensen AI. Proprioception at the ankle: the effect of anaesthetic blockade of ligament receptors. *J Bone Joint Surg Br.* 1993;75(3):433-436.
131. de Carlo MS, Talbot RW. Evaluation of ankle joint proprioception following injection of the anterior talofibular ligament. *J Orthop Sports Phys Ther.* 1986;8(2):70-76.
132. Riemann BL. Is There a Link Between Chronic Ankle Instability and Postural Instability? *J Athl Train.* 2002;37(4):386-393.
133. Goulding M, Bourane S, Garcia-Campmany L, Dalet A, Koch S. Inhibition downunder: an update from the spinal cord. *Curr Opin Neurobiol.* 2014;26:161-166.
134. Hultborn H, Lindstrom S, Wigstrom H. On the function of recurrent inhibition in the spinal cord. *Exp Brain Res.* 1979;37(2):399-403.
135. Sefton JM, Hicks-Little CA, Hubbard TJ, et al. Sensorimotor function as a predictor of chronic ankle instability. *Clin Biomech (Bristol, Avon).* 2009;24(5):451-458.
136. Bussel B, Pierrot-Deseilligny E. Inhibition of human motoneurons, probably of Renshaw origin, elicited by an orthodromic motor discharge. *J Physiol.* 1977;269(2):319-339.

137. Johansson H, Sjolander P, Sojka P. Receptors in the knee joint ligaments and their role in the biomechanics of the joint. *Crit Rev Biomed Eng.* 1991;18(5):341-368.
138. Konishi Y, Fukubayashi T, Takeshita D. Possible mechanism of quadriceps femoris weakness in patients with ruptured anterior cruciate ligament. *Med Sci Sports Exerc.* 2002;34(9):1414-1418.
139. Feger MA, Donovan L, Hart JM, Hertel J. Lower extremity muscle activation during functional exercises in patients with and without chronic ankle instability. *PM R.* 2014;6(7):602-611; quiz 611.
140. van Deun S, Staes FF, Stappaerts KH, Janssens L, Levin O, Peers KK. Relationship of chronic ankle instability to muscle activation patterns during the transition from double-leg to single-leg stance. *Am J Sports Med.* 2007;35(2):274-281.
141. Doherty C, Bleakley C, Hertel J, et al. Inter-joint coordination strategies during unilateral stance following first-time, acute lateral ankle sprain: A brief report. *Clin Biomech (Bristol, Avon).* 2015;30(6):636-639.
142. Kirshner B, Guyatt G. A methodological framework for assessing health indices. *J Chronic Dis.* 1985;38(1):27-36.
143. Carcia CR, Martin RL, Drouin JM. Validity of the Foot and Ankle Ability Measure in athletes with chronic ankle instability. *J Athl Train.* 2008;43(2):179-183.
144. Martin RL, Irrgang JJ, Burdett RG, Conti SF, Van Swearingen JM. Evidence of validity for the Foot and Ankle Ability Measure (FAAM). *Foot Ankle Int.* 2005;26(11):968-983.
145. Hale SA, Hertel J. Reliability and Sensitivity of the Foot and Ankle Disability Index in Subjects With Chronic Ankle Instability. *J Athl Train.* 2005;40(1):35-40.
146. Martin RL, Burdett RG, Irrgang JJ. Development of the Foot and Ankle Disability Index (FADI) [abstract]. *J Orthop Sports Phys Ther.* 1999;29:A32-A33.
147. Ferretti A, Papandrea P, Poggini L, Falez F. Third-degree lesions of the external compartment of the ankle: results of conservative treatment. *Ital J Orthop Traumatol.* 1991;17(1):41-53.
148. Haywood KL, Hargreaves J, Lamb SE. Multi-item outcome measures for lateral ligament injury of the ankle: a structured review. *J Eval Clin Pract.* 2004;10(2):339-352.
149. Negahban H, Mazaheri M, Salavati M, et al. Reliability and validity of the foot and ankle outcome score: a validation study from Iran. *Clin Rheumatol.* 2010;29(5):479-486.
150. Klykken LW, Pietrosimone BG, Kim KM, Ingersoll CD, Hertel J. Motor-neuron pool excitability of the lower leg muscles after acute lateral ankle sprain. *J Athl Train.* 2011;46(3):263-269.
151. Croy T, Saliba S, Saliba E, Anderson MW, Hertel J. Talofibular interval changes after acute ankle sprain: a stress ultrasonography study of ankle laxity. *J Sport Rehabil.* 2013;22(4):257-263.
152. Doherty C, Bleakley C, Hertel J, Caulfield B, Ryan J, Delahunt E. Lower extremity function during gait in participants with first time acute lateral ankle sprain compared to controls. *J Electromyogr Kinesiol.* 2015;25(1):182-192.

153. Doherty C, Bleakley C, Hertel J, Caulfield B, Ryan J, Delahunt E. Single-leg drop landing motor control strategies following acute ankle sprain injury. *Scand J Med Sci Sports.* 2015;25(4):525-533.
154. Doherty C, Bleakley C, Hertel J, Caulfield B, Ryan J, Delahunt E. Single-leg drop landing movement strategies 6 months following first-time acute lateral ankle sprain injury. *Scand J Med Sci Sports.* 2015;25(6):806-817.
155. Doherty C, Bleakley C, Hertel J, et al. Coordination and symmetry patterns during the drop vertical jump, 6-months after first-time lateral ankle sprain. *J Orthop Res.* 2015;33(10):1537-1544.
156. Cosby NL, Koroch M, Grindstaff TL, Parente W, Hertel J. Immediate effects of anterior to posterior talocrural joint mobilizations following acute lateral ankle sprain. *J Man Manip Ther.* 2011;19(2):76-83.
157. Hubbard TJ, Cordova M. Mechanical instability after an acute lateral ankle sprain. *Arch Phys Med Rehabil.* 2009;90(7):1142-1146.
158. Aiken AB, Pelland L, Brison R, Pickett W, Brouwer B. Short-term natural recovery of ankle sprains following discharge from emergency departments. *J Orthop Sports Phys Ther.* 2008;38(9):566-571.
159. van Middelkoop M, van Rijn RM, Verhaar JA, Koes BW, Bierma-Zeinstra SM. Re-sprains during the first 3 months after initial ankle sprain are related to incomplete recovery: an observational study. *J Physiother.* 2012;58(3):181-188.
160. Houston MN, Hoch JM, Hoch MC. Patient-Reported Outcome Measures in Individuals With Chronic Ankle Instability: A Systematic Review. *J Athl Train.* 2015;50(10):1019-1033.
161. Hiller CE, Refshauge KM, Bundy AC, Herbert RD, Kilbreath SL. The Cumberland ankle instability tool: a report of validity and reliability testing. *Arch Phys Med Rehabil.* 2006;87(9):1235-1241.
162. Wright CJ, Arnold BL, Ross SE, Linens SW. Recalibration and validation of the Cumberland Ankle Instability Tool cutoff score for individuals with chronic ankle instability. *Arch Phys Med Rehabil.* 2014;95(10):1853-1859.
163. Docherty CL, Gansneder BM, Arnold BL, Hurwitz SR. Development and reliability of the ankle instability instrument. *J Athl Train.* 2006;41(2):154-158.
164. Gurav RS, Ganu SS, Panhale VP. Reliability of the Identification of Functional Ankle Instability (IdFAI) Scale Across Different Age Groups in Adults. *N Am J Med Sci.* 2014;6(10):516-518.
165. Simon J, Donahue M, Docherty C. Development of the Identification of Functional Ankle Instability (IdFAI). *Foot Ankle Int.* 2012;33(9):755-763.
166. Doherty C, Bleakley C, Hertel J, Caulfield B, Ryan J, Delahunt E. Locomotive biomechanics in persons with chronic ankle instability and lateral ankle sprain copers. *J Sci Med Sport.* 2015.
167. Doherty C, Bleakley C, Hertel J, Caulfield B, Ryan J, Delahunt E. Single-leg drop landing movement strategies in participants with chronic ankle instability compared with lateral ankle sprain 'copers'. *Knee Surg Sports Traumatol Arthrosc.* 2016;24(4):1049-1059.
168. Arnold BL, Wright CJ, Ross SE. Functional ankle instability and health-related quality of life. *J Athl Train.* 2011;46(6):634-641.

169. Hubbard-Turner T, Turner MJ. Physical Activity Levels in College Students With Chronic Ankle Instability. *J Athl Train.* 2015;50(7):742-747.
170. Terada M, Bowker S, Hiller CE, Thomas AC, Pietrosimone B, Gribble PA. Quantifying levels of function between different subgroups of chronic ankle instability. *Scand J Med Sci Sports.* In Press.
171. Marshall PW, McKee AD, Murphy BA. Impaired trunk and ankle stability in subjects with functional ankle instability. *Med Sci Sports Exerc.* 2009;41(8):1549-1557.
172. Cooke MW, Lamb SE, Marsh J, Dale J. A survey of current consultant practice of treatment of severe ankle sprains in emergency departments in the United Kingdom. *Emerg Med J.* 2003;20(6):505-507.
173. McCann RS, Gribble PA. Resilience and Self-Efficacy: A Theory-Based Model of Chronic Ankle Instability. *Int J Athl Ther Train.* 2016;21:32-37.
174. Kaminski TW, Hertel J, Amendola N, et al. National Athletic Trainers' Association position statement: conservative management and prevention of ankle sprains in athletes. *J Athl Train.* 2013;48(4):528-545.
175. Martin RL, Davenport TE, Paulseth S, Wukich DK, Godges JJ. Ankle Stability and Movement Coordination Impairments: Ankle Ligament Sprains. *J Orthop Sports Phys Ther.* 2013;43(9):A1-40.
176. Ardern CL, Bizzini M, Bahr R. It is time for consensus on return to play after injury: five key questions. *Br J Sports Med.* 2016;50(9):506-508.
177. Kosik KB, McCann RS, Terada M, Gribble PA. Therapeutic interventions for improving self-reported function in patients with chronic ankle instability: a systematic review. *Br J Sports Med.* 2017;51:105-112.
178. Hagglund M, Walden M, Ekstrand J. Previous injury as a risk factor for injury in elite football: a prospective study over two consecutive seasons. *Br J Sports Med.* 2006;40(9):767-772.
179. Trojian TH, McKeag DB. Single leg balance test to identify risk of ankle sprains. *Br J Sports Med.* 2006;40(7):610-613; discussion 613.
180. Hrysomallis C, McLaughlin P, Goodman C. Balance and injury in elite Australian footballers. *Int J Sports Med.* 2007;28(10):844-847.
181. Tropp H, Ekstrand J, Gillquist J. Stabilometry in functional instability of the ankle and its value in predicting injury. *Med Sci Sports Exerc.* 1984;16(1):64-66.
182. Plisky PJ, Rauh MJ, Kaminski TW, Underwood FB. Star Excursion Balance Test as a predictor of lower extremity injury in high school basketball players. *J Orthop Sports Phys Ther.* 2006;36(12):911-919.
183. Friel K, McLean N, Myers C, Caceres M. Ipsilateral hip abductor weakness after inversion ankle sprain. *J Athl Train.* 2006;41(1):74-78.
184. Nicholas JA, Strizak AM, Veras G. A study of thigh muscle weakness in different pathological states of the lower extremity. *Am J Sports Med.* 1976;4(6):241-248.
185. Dick R, Putukian M, Agel J, Evans TA, Marshall SW. Descriptive epidemiology of collegiate women's soccer injuries: National Collegiate Athletic Association Injury Surveillance System, 1988-1989 through 2002-2003. *J Athl Train.* 2007;42(2):278-285.
186. Beynnon BD, Murphy DF, Alosa DM. Predictive Factors for Lateral Ankle Sprains: A Literature Review. *J Athl Train.* 2002;37(4):376-380.

187. Sugimoto D, Myer GD, McKeon JM, Hewett TE. Evaluation of the effectiveness of neuromuscular training to reduce anterior cruciate ligament injury in female athletes: a critical review of relative risk reduction and numbers-needed-to-treat analyses. *Br J Sports Med.* 2012;46(14):979-988.
188. Myer GD, Ford KR, Brent JL, Hewett TE. Differential neuromuscular training effects on ACL injury risk factors in"high-risk" versus "low-risk" athletes. *BMC Musculoskelet Disord.* 2007;8:39.
189. Thorpe JL, Ebersole KT. Unilateral balance performance in female collegiate soccer athletes. *J Strength Cond Res.* 2008;22(5):1429-1433.
190. Khayambashi K, Ghoddosi N, Straub RK, Powers CM. Hip Muscle Strength Predicts Noncontact Anterior Cruciate Ligament Injury in Male and Female Athletes: A Prospective Study. *Am J Sports Med.* 2016;44(2):355-361.
191. Leetun DT, Ireland ML, Willson JD, Ballantyne BT, Davis IM. Core stability measures as risk factors for lower extremity injury in athletes. *Med Sci Sports Exerc.* 2004;36(6):926-934.
192. Distefano LJ, Blackburn JT, Marshall SW, Padua DA. Gluteal muscle activation during common therapeutic exercises. *J Orthop Sports Phys Ther.* 2009;39(7):532-540.
193. Hollman JH, Galardi CM, Lin IH, Voth BC, Whitmarsh CL. Frontal and transverse plane hip kinematics and gluteus maximus recruitment correlate with frontal plane knee kinematics during single-leg squat tests in women. *Clin Biomech (Bristol, Avon).* 2014;29(4):468-474.
194. Hollman JH, Hohl JM, Kraft JL, Strauss JD, Traver KJ. Modulation of frontal-plane knee kinematics by hip-extensor strength and gluteus maximus recruitment during a jump-landing task in healthy women. *J Sport Rehabil.* 2013;22(3):184-190.
195. Preece SJ, Graham-Smith P, Nester CJ, et al. The influence of gluteus maximus on transverse plane tibial rotation. *Gait Posture.* 2008;27(4):616-621.
196. Robinson RH, Gribble PA. Support for a reduction in the number of trials needed for the star excursion balance test. *Arch Phys Med Rehabil.* 2008;89(2):364-370.
197. Gribble PA, Kelly SE, Refshauge KM, Hiller CE. Interrater reliability of the star excursion balance test. *J Athl Train.* 2013;48(5):621-626.
198. Hertel J, Miller SJ, Denegar CR. Intratester and Intertester Reliability During the Star Excursion Balance Test. *J Sport Rehabil.* 2000;9:104-116.
199. Ireland ML, Willson JD, Ballantyne BT, Davis IM. Hip strength in females with and without patellofemoral pain. *J Orthop Sports Phys Ther.* 2003;33(11):671-676.
200. Thorborg K, Petersen J, Magnusson SP, Holmich P. Clinical assessment of hip strength using a hand-held dynamometer is reliable. *Scand J Med Sci Sports.* 2010;20(3):493-501.
201. Kelln BM, McKeon PO, Gontkof LM, Hertel J. Hand-held dynamometry: reliability of lower extremity muscle testing in healthy, physically active,young adults. *J Sport Rehabil.* 2008;17(2):160-170.
202. Cohen J. Statistical power analysis for the behavioral sciences. 2nd ed. Hillsdale, N.J.: L. Erlbaum Associates; 1988:77-83.

203. Swets JA. Measuring the accuracy of diagnostic systems. *Science.* 1988;240(4857):1285-1293.
204. McGuine TA, Hetzel S, Wilson J, Brooks A. The effect of lace-up ankle braces on injury rates in high school football players. *Am J Sports Med.* 2012;40(1):49-57.
205. McGuine TA, Keene JS. The effect of a balance training program on the risk of ankle sprains in high school athletes. *Am J Sports Med.* 2006;34(7):1103-1111.
206. Chinn L, Hertel J. Rehabilitation of ankle and foot injuries in athletes. *Clin Sports Med.* 2010;29(1):157-167.
207. Lynch SA. Assessment of the Injured Ankle in the Athlete. *J Athl Train.* 2002;37(4):406-412.
208. Jensen MP, Chen C, Brugger AM. Interpretation of visual analog scale ratings and change scores: a reanalysis of two clinical trials of postoperative pain. *J Pain.* 2003;4(7):407-414.
209. Tatro-Adams D, McGann SF, Carbone W. Reliability of the figure-of-eight method of ankle measurement. *J Orthop Sports Phys Ther.* 1995;22(4):161-163.
210. Chisholm MD, Birmingham TB, Brown J, Macdermid J, Chesworth BM. Reliability and validity of a weight-bearing measure of ankle dorsiflexion range of motion. *Physiother Can.* 2012;64(4):347-355.
211. Hoch MC, Farwell KE, Gaven SL, Weinhandl JT. Weight-Bearing Dorsiflexion Range of Motion and Landing Biomechanics in Individuals With Chronic Ankle Instability. *J Athl Train.* 2015;50(8):833-839.
212. Terada M, Harkey MS, Wells AM, Pietrosimone BG, Gribble PA. The influence of ankle dorsiflexion and self-reported patient outcomes on dynamic postural control in participants with chronic ankle instability. *Gait Posture.* 2014;40(1):193-197.
213. van den Bekerom MP, Kerkhoffs GM, McCollum GA, Calder JD, van Dijk CN. Management of acute lateral ankle ligament injury in the athlete. *Knee Surg Sports Traumatol Arthrosc.* 2013;21(6):1390-1395.
214. Kerkhoffs GM, Rowe BH, Assendelft WJ, Kelly KD, Struijs PA, van Dijk CN. Immobilisation for acute ankle sprain. A systematic review. *Arch Orthop Trauma Surg.* 2001;121(8):462-471.
215. Donovan L, Hertel J. A new paradigm for rehabilitation of patients with chronic ankle instability. *Phys Sportsmed.* 2012;40(4):41-51.
216. Gribble PA, Bleakley CM, Caulfield BM, et al. Evidence review for the 2016 International Ankle Consortium consensus statement on the prevalence, impact and long-term consequences of lateral ankle sprains. *Br J Sports Med.* 2016;50(24):1496-1505.
217. Katz J, Melzack R. Measurement of pain. *Surg Clin North Am.* 1999;79(2):231-252.
218. Bijur PE, Silver W, Gallagher EJ. Reliability of the visual analog scale for measurement of acute pain. *Acad Emerg Med.* 2001;8(12):1153-1157.
219. Mawdsley RH, Hoy DK, Erwin PM. Criterion-related validity of the figure-of-eight method of measuring ankle edema. *J Orthop Sports Phys Ther.* 2000;30(3):149-153.

220. Bennell KL, Talbot RC, Wajswelner H, Techovanich W, Kelly DH, Hall AJ. Intra-rater and inter-rater reliability of a weight-bearing lunge measure of ankle dorsiflexion. *Aust J Physiother.* 1998;44(3):175-180.
221. Powden CJ, Hoch JM, Hoch MC. Reliability and minimal detectable change of the weight-bearing lunge test: A systematic review. *Man Ther.* 2015;20(4):524-532.
222. Viera AJ, Garrett JM. Understanding interobserver agreement: the kappa statistic. *Fam Med.* 2005;37(5):360-363.
223. Hertel J, Braham RA, Hale SA, Olmsted-Kramer LC. Simplifying the star excursion balance test: analyses of subjects with and without chronic ankle instability. *J Orthop Sports Phys Ther.* 2006;36(3):131-137.
224. Plisky PJ, Gorman PP, Butler RJ, Kiesel KB, Underwood FB, Elkins B. The reliability of an instrumented device for measuring components of the star excursion balance test. *N Am J Sports Phys Ther.* 2009;4(2):92-99.
225. Kreatsoulas C, Hassan A, Subramanian SV, Fleegler EW. Accuracy of Self-Reported Height and Weight to Determine Body Mass Index Among Youth. *J Child Adolesc Behav.* 2014;2(1):1-3.
226. Nieto-Garcia FJ, Bush TL, Keyl PM. Body mass definitions of obesity: sensitivity and specificity using self-reported weight and height. *Epidemiology.* 1990;1(2):146-152.
227. Turocy PS, DePalma BF, Horswill CA, et al. National Athletic Trainers' Association position statement: safe weight loss and maintenance practices in sport and exercise. *J Athl Train.* 2011;46(3):322-336.
228. McHugh MP, Tyler TF, Mirabella MR, Mullaney MJ, Nicholas SJ. The effectiveness of a balance training intervention in reducing the incidence of noncontact ankle sprains in high school football players. *Am J Sports Med.* 2007;35(8):1289-1294.
229. Wester JU, Jespersen SM, Nielsen KD, Neumann L. Wobble board training after partial sprains of the lateral ligaments of the ankle: a prospective randomized study. *J Orthop Sports Phys Ther.* 1996;23(5):332-336.

Vita

Ryan Sean McCann, MSEd, ATC, CSCS

Place of Birth: Cincinnati, OH, January 6, 1986

Certificate or Specialty Board Licensure:
National Athletic Trainers' Association Board of Certification #070802037 (since 2008)

I. Education

2014-present University of Kentucky, Lexington, KY
Doctor of Philosophy, Rehabilitation Sciences
Expected Completion: May 2017
Dissertation: Prediction of Acute and Recurrent Ankle Sprains in Athletes
Advisor: Phillip A. Gribble, FNATA, PhD, ATC

2008-2010 Old Dominion University, Norfolk, VA
Master of Science in Education, Athletic Training
Thesis: The Effects of an Anterior Cruciate Ligament Prevention Program and Retention Period on Lower Extremity Biomechanics
Advisor: James A. Onate, FNATA, PhD, ATC

2004-2008 Northern Kentucky University, Highland Heights, KY
Bachelor of Science in Athletic Training

II. Professional Experience

2016-present Outreach Athletic Trainer, Nicholas County High School, Carlisle, KY
University of Kentucky Orthopaedics & Sports Medicine, Lexington, KY

2015-present PRN Athletic Trainer
Bluegrass Orthopaedics, Lexington, KY

2013-2014 Part-time Athletic Trainer
Premier Volleyball Academy, Maumee, OH

2010-2013 Outreach Athletic Trainer, Grassfield High School, Chesapeake, VA
The Children's Hospital of the King's Daughters, Norfolk, VA

2008-2010 Graduate Assistant Athletic Trainer
Old Dominion University, Norfolk, VA

III. Scholastic and Professional Honors

2015 National Athletic Trainers' Association Research & Education Foundation, Doctoral Grant
Title: Clinical Determinants of Recurrent Injury following Index Ankle Sprain

2010	National Athletic Trainers' Association Research & Education Foundation, Master's Poster Award Winner *Title*: A strength and agility training program reduces knee valgus angle at initial contact
2008	Outstanding Athletic Training Student of the Year Award Northern Kentucky University
2007	Athletic Training Achievement Scholarship Northern Kentucky University

IV. Professional Publications

McCann RS, Kosik K, Terada M, Beard MQ, Buskirk GE, Gribble PA. Associations between Functional and Isolated Performance Measures in Collegiate Women's Soccer Players. *Journal of Sport Rehabilitation*. In Press.

Kosik K, Terada M, **McCann R**, Boland S, Gribble P. Comparison of Two Rehabilitation Protocols on Patient and Disease Oriented Outcomes in Chronic Ankle Instability Individuals. *International Journal of Athletic Therapy & Training*. Submitted May 2016: In Press.

Kosik KB, **McCann RS**, Terada M, Gribble PA. Therapeutic Interventions for Improving Self-Reported Function in Patients with Chronic Ankle Instability: A Systematic Review. *British Journal of Sports Medicine*. 2017, 51:105-112.

Kosik KB, Terada M, Drinkard CP, **McCann RS**, Gribble PA. Potential Corticomotor Plasticity in those with and without Chronic Ankle Instability. *Medicine & Science in Sport & Exercise*. 2017, 49(1):141-149.

Terada M, Kosik KB, **McCann RS**, Gribble PA. Diaphragm Structural Integrity and Contractility in Individuals with and without Chronic Ankle Instability. *Medicine & Science in Sport & Exercise*. 2016, 48(10):2040-2045.

McCann RS, Gribble PA. Resilience and Self-Efficacy: A Theory-Based Model of Chronic Ankle Instability. *International Journal of Athletic Therapy & Training*. 2016, 21(3):32-37.

Gribble PA, Terada M, Beard MQ, Kosik K, Lepley A, **McCann RS**, Pietrosimone BG, Thomas A. Prediction of lateral ankle sprain risk in football players using clinical modifiable factors. *American Journal of Sports Medicine.* 2016, 42(2):460-467.

McCann RS, Kosik K, Beard MQ, Terada M, Pietrosimone BG, Gribble PA. Variations in Star Excursion Balance Test Performance between High School and Collegiate Football Players. *Journal of Strength and Conditioning Research*. 2015, 29(10):2765-2770.

McCann R, Cortes N, Van Lunen BL, Greska E, Ringleb S, Onate JA. Neuromuscular changes following and injury prevention program for ACL injuries. *International Journal of Athletic Therapy & Training*. 2011, 16(4):16-20.