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The Effects of Abdominal Training on Postural Control, Lower Extremity Kinematics, Kinetics, and Muscle Activation

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THE EFFECTS OF ABDOMINAL TRAINING ON POSTURAL CONTROL, LOWER EXTREMITY KINEMATICS, KINETICS, AND MUSCLE ACTIVATION

by

Matthew J. Gage

A dissertation submitted to the faculty of
Brigham Young University
in partial fulfillment of the requirements for the degree of

Doctor of Philosophy

Department of Exercise Sciences
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BRIGHAM YOUNG UNIVERSITY

GRADUATE COMMITTEE APPROVAL

of a dissertation submitted by

Matthew J. Gage

This dissertation has been read by each member of the following graduate committee and by majority vote has been found to be satisfactory.

Date	J. Ty Hopkins, Chair
Date	Iain Hunter
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Date	David D. Draper
Date	William Myrer
Date	Richard Sudweeks

BRIGHAM YOUNG UNIVERSITY

As chair of the candidate's graduate committee, I have read the dissertation of Matthew J. Gage in its final form and have found that (1) its format, citations, and bibliographical style are consistent and acceptable and fulfill university and department style requirements; (2) its illustrative materials including figures, tables, and charts are in place; and (3) the final manuscript is satisfactory to the graduate committee and is ready for submission to the university library.

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ABSTRACT

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Matthew J. Gage

Department of Exercise Sciences

Doctor of Philosophy

Context: Abdominal training may decrease the risk of lower extremity injuries through improved balance and postural control. Objective: To determine the effect of an eight-week abdominal-training program on center of pressure, lower extremity joint angles, and abdominal muscle activation during a single-leg drop landing. The effects of abdominal training on abdominal muscle thickness was assessed. Design: A cohort research design. Setting: Research laboratory. Other Participants: Sixty healthy physically active college-aged students participated. They were divided into three groups: Control, Chronic ankle instability (CAI), and Healthy. Nineteen Control (age = 22.0 ± 2.72 yrs, mass = 74.1 ± 13.8 kg, height = 172.6 ± 11.3 cm, BMI = 24.8 ± 3.1 %), 21 CAI (age = 22.1 ± 2.3 yrs, mass = 77.6 ± 14.0 kg, height = 175.4 ± 12.3 cm, BMI = 25.1 ± 2.6 %), and 20 healthy (age = 22.9 ± 3.4 yrs, mass = 70.9 ± 15.6 kg, height = 172.2 ± 8.9 cm, BMI = 23.7 ± 3.3 %). Subjects in the CAI group had a history of CAI and functional ankle instability (FAI). The Ankle Instability Index and the Functional Ankle Ability Measure were used to self-report CAI and FAI respectively. Interventions: The CAI and Healthy groups participated in an eight-week abdominal-training program while the Control group maintained their normal activities of daily living and level of physical activity. Main Outcome Measures: Abdominal muscle thickness was measured biweekly throughout the study. Center of pressure excursion, muscle activation, vertical ground reaction force, and lower extremity joint angles were measured during a single-leg drop landing, pre- and postabdominal training. Results: Muscle thickness at rest increased in the rectus abdominis and external oblique muscles follow training. Eight weeks of abdominal training decreased vertical ground reaction forces and muscle activation down the lower kinetic chain. Center of pressure excursion and velocity were increased following training. Conclusions: Eight-weeks of abdominal training increased abdominal muscle thickness. Training improved neuromuscular efficiency throughout the kinetic chain and may have improved dynamic postural control. Our data also suggest CAI subjects may utilize both feedforward and feedback mechanisms to maintain postural control. Key Words: ankle instability, abdominal training, balance, functional ankle instability, and vertical ground reaction force

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Table of Contents

List of Tables

Tables

List of Figures

Figures

THE EFFECTS OF ABDOMINAL TRAINING ON POSTURAL CONTROL, LOWER EXTREMITY KINEMATICS, AND MUSCLE ACTIVATION

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ABSTRACT

Context: Abdominal training may decrease the risk of lower extremity injuries through improved balance and postural control. Objective: To determine the effect of an eight-week abdominal-training program on center of pressure, lower extremity joint angles, and abdominal muscle activation during a single-leg drop landing. The effects of abdominal training on abdominal muscle thickness was assessed. Design: A cohort research design. Setting: Research laboratory. Other Participants: Sixty healthy physically active college-aged students participated. They were divided into three groups: Control, Chronic ankle instability (CAI), and Healthy. Nineteen Control (age = 22.0 ± 2.72 yrs, mass = 74.1 ± 13.8 kg, height = 172.6 ± 11.3 cm, BMI = 24.8 ± 3.1 %), 21 CAI (age = 22.1 ± 2.3 yrs, mass = 77.6 ± 14.0 kg, height = 175.4 ± 12.3 cm, BMI = 25.1 ± 2.6 %), and 20 healthy (age = 22.9 ± 3.4 yrs, mass = 70.9 ± 15.6 kg, height = 172.2 ± 8.9 cm, BMI = 23.7 ± 3.3 %). Subjects in the CAI group had a history of CAI and functional ankle instability (FAI). The Ankle Instability Index and the Functional Ankle Ability Measure were used to self-report CAI and FAI respectively. Interventions: The CAI and Healthy groups participated in an eight-week abdominal-training program while the Control group maintained their normal activities of daily living and level of physical activity. Main Outcome Measures: Abdominal muscle thickness was measured biweekly throughout the study. Center of pressure excursion, muscle activation, vertical ground reaction force, and lower extremity joint angles were measured during a single-leg drop landing, pre- and postabdominal training. Results: Muscle thickness at rest increased in the rectus abdominis and external oblique muscles follow training. Eight weeks of abdominal training decreased vertical ground reaction forces and muscle activation down the lower kinetic chain. Center of pressure excursion and velocity were increased following training. Conclusions: Eight-weeks of abdominal training increased abdominal muscle thickness. Training improved neuromuscular efficiency throughout the kinetic chain and may have improved dynamic postural control. Our data also suggest CAI subjects may utilize both feedforward and feedback mechanisms to maintain postural control. Key Words: ankle instability, abdominal training, balance, functional ankle instability, and vertical ground reaction force

INTRODUCTION

In the 1960s, Freeman et al[1-3] coined the term “functional instability” to describe the neuromuscular deficits they observed at the ankle and foot. These deficits were believed to be responsible for repeated “giving way” episodes. Functional ankle instability is thought to be one of the components of chronic ankle instability (CAI).[4] Freeman[2] hypothesized that when an injury to the ankle occurs, deafferentiation of the afferent nerves may result which could contribute to CAI. Other researchers have observed decreased proprioception,[2, 5, 6] joint position sense,[6-9] strength,[9-14] coordination,[2, 8] balance,[15, 16] postural control[17-25] and increased peroneal muscle latency[26, 27] in CAI subjects. The cause of CAI remains unclear despite the extensive research in this area.

Due to the complexity of these findings, researchers have begun to study the relationship between proximal joints/muscles and CAI. Numerous studies support the theory that CAI subjects use proximal muscles to compensate for distal neuromuscular deficits.[28-33] Activation of the gluteus medius (GMed),[28] gluteus maximus,[29, 31] and biceps femoris[31] have been reported to be altered in CAI subjects compared to healthy subjects. The GMed activated earlier in CAI than healthy subjects following a perturbation,[28] and subjects with a history of a severe ankle sprain demonstrated delayed gluteus maximus and earlier biceps femoris activation.[29, 31] In addition to altered proximal muscle activation, arthrogenic muscle inhibition of the hamstrings and facilitation of the quadriceps were observed in CAI subjects, demonstrating altered motorneuron pool excitability of proximal muscles.[34] Gribble et al[33] reported a disruption in sagittal postural control during a dynamic task in CAI subjects, which was most notable in joints proximal to the ankle. Smaller reach distances and knee-flexion angles were also observed in CAI subjects.[32, 33] While these studies support the idea that proximal muscle contraction patterns are altered in CAI, none of these studies assessed subjects during dynamic functional movement.

Since different tasks have been used to assess the relationship between CAI and proximal joints/muscles it is difficult to make comparisons. Some of the tasks used were a prone leg extension, single-leg perturbation, maximal voluntary contraction, and Star Excursion Balance Test.[28, 29, 32-34] These tasks are not dynamic functional tasks that would be observed during an athletic event. Therefore, we chose to use a single-leg drop landing for our study. A single-leg drop landing provides a controlled representation of landings that occur during athletic events. It has been reported that abdominal muscle activation and center of pressure excursion (COPd) increased as the level of difficulty increased during a single-leg drop landing in healthy subjects.[35] A single-leg drop landing places greater demands on the kinetic chain in comparison to previously used tasks.

It is unknown if the kinetic chain responds neuromuscularly proximal to distal or distal to proximal during dynamic movement. A distal to proximal response would support what Freeman hypothesized (feedback mechanism). When the ankle moves, the somatosensory system informs the central nervous system of the movement, then a signal is sent to the muscles around the ankle to respond to its movement. However, a proximal to distal response (feedforward mechanism) would suggest that training the proximal muscles may assist in the prevention and/or treatment of CAI. Based on these aforementioned studies, it appears that CAI subjects do not use a feedback-only mechanism to maintain postural control.[36] A more comprehensive theoretic model that includes both feedback and feedforward mechanisms may be more appropriate. Understanding how the feedback and feedforward mechanisms interrelate may aid in preventing CAI. If feedforward mechanisms are involved in helping CAI subjects maintain dynamic postural control, it would be logical to think that training the muscles proximal to the ankle may improve postural control.

Abdominal or "core" training is thought to improve balance, postural control, and reduce the risk of lower extremity injuries.[37-40] This theory is supported by the fact that subjects with a history of lower extremity injuries required greater trunk muscle recruitment to stabilize the body during dynamic tasks compared to healthy subjects.[41] Recently, researchers observed improved postural control in healthy subjects following a six week core training program.[39] Following training those subjects demonstrated greater reach distance, and peak excursion during a Star Excursion Balance Test.[39] These studies are consistent with the feedforward ideas suggested earlier. However, further research is required to comprehensively assess how abdominal training affects postural control and the risk of lower extremity injuries.

The purpose(s) of our study was to determine if muscle activation, center of pressure (COP), and kinematics differed between groups during a single-leg drop landing pre- and postabdominal training. Another purpose of this study was to determine if morphological changes occur to abdominal muscle thickness at rest and during abdominal hollowing (AH) between & within groups.

METHODS

Design

Two different cohort designs were utilized to analyze changes in the dependent variables (abdominal muscle thickness, muscle activation, COP, sagittal lower extremity joint angles, and vertical ground reaction force). Separate 3 x 5 designs (group x time) were used to analyze abdominal muscle thickness at rest and during AH. A 3 x 2 design (group x time) was used to analyze COP excursion, lower extremity joint angles, muscle activation, and vertical ground reaction force (VGRF) during single-leg drop landings. These measurements were taken pre- and postabdominal training.

Participants

Seventy-five physically active subjects of both genders were recruited to participate in our study. They were divided equally into three groups (Control, Healthy & CAI). The Healthy and CAI groups participated in an eight-week abdominal-training program while the Control subjects were asked to maintain their activities of daily living (ADL) without increasing their current level of physical activity.

Inclusion criteria for the CAI group were a history of 1) at least one substantial ankle sprain with the initial sprain occurring more than 12 months ago, 2) the ankle "giving way" during functional activities (CAI), and 3) functional ankle instability. Three questionnaires were used to determine if subjects fit the inclusion criterion set for the CAI group. The Ankle Instability Index determined if subjects had CAI. Subjects had to answer “yes” to at least two of questions four through eight on the Ankle Instability Index to be classified as having CAI. The Functional Ankle Ability Measure

(FAAM) ADL and FAAM Sport questionnaires allowed subjects to self-report functional ankle instability. Functional ankle instability was self-reported if subjects scored greater than or equal to 90% on the FAAM ADL scale and 80% on the FAAM Sport Scale. Subjects in the Control and Healthy groups were randomly assigned and matched by gender and leg dominance with a CAI subject.

Exclusion criteria were a history of cardiovascular or neurological disorder, mechanical ankle instability, childbirth or pregnancy within the past two years; abdomen, low back, or lower extremity injury/pain within the past year that restricted the subject's ability to be physically active; abdominal, low back, or lower extremity surgery within the past two years; or regular participation in an abdominal-training program. Regular participation was defined as performing abdominal training exercises three or more times a week. Subjects were excluded during the study if they sustained an abdomen, low back, or lower extremity injury that restricted their ADL or if they missed two abdominal thickness measurement or training sessions (supervised or unsupervised). Failure to return the weekly exercise log at the supervised training session was also grounds for exclusion from this study.

Fifteen of the 75 subjects were unable to complete the eight-week study for a variety of reasons. Six Control group subjects did not complete the study due to time commitment (1), illness (1), and instrument malfunction (4). Four CAI subjects failed to complete the study because of time commitment (3) and illness (1). Five Healthy group subjects were unable to complete the study because of time commitment (3) and failure to complete abdominal workouts (2). Thus 60 subjects completed the study, 19 Control (age = 22.0 ± 2.72 yrs, mass = 74.1 ± 13.8 kg, height = 172.6 ± 11.3 cm, BMI = 24.8 ± 3.1 %), 21 CAI (age = 22.1 ± 2.3 yrs, mass = 77.6 ± 14.0 kg, height = 175.4 ± 12.3 cm, BMI = 25.1 ± 2.6 %), and 20 Healthy (age = 22.9 ± 3.4 yrs, mass = 71.0 ± 15.6 kg, height = 172.2 ± 8.9 cm, BMI = 23.7 ± 3.3 %). All of the subjects read and signed the approved informed consent form prior to data collection.

Instrumentation

Ultrasound Imaging

We used the LOGIQ P5 Laser Doppler Ultrasound (General Electric, Piscataway, NJ, USA) with a linear phased array probe (45 x 10 mm footprint; 7 to 12 MHz frequencies) to measure abdominal muscle thickness at rest and during abdominal hollowing (AH). Probe frequency was set at 10 MHz with a gain of 70 for all measurements.[42]

Previous ultrasound imaging research focused on establishing reliability,[43-49] validity,[50] and a correlation between muscle activation and abdominal muscle thickness changes. Ultrasound imaging provides a noninvasive instrument to measure abdominal thickness at rest and during AH.[51-53] It is also a reliable[43-49] and valid[50] instrument to measure changes in abdominal thickness. Lateral abdominal muscle thickness was measured previously by the primary investigator over four weeks with good to excellent intrarater (ICC= 0.89-0.96) and intersession reliability (ICC= 0.90-0.94).[46] A correlation between changes in abdominal muscle thickness and muscle activation exist during sub-maximal activities.

Electromyography

The Delsys Myomonitor IV System (Delsys Inc., Boston, MA) was used to measure muscle activation of the transverse abdominis (TrA) /internal oblique (IO), external oblique (EO), GMed, vastus medialis (VM), and peroneus longus (PL) muscles. These measurements were collected using Delsys surface electromyography (EMG) sensors (DE-2.1, Delsys Inc., Boston, MA). The Myomonitor IV System is a wireless unit. Therefore we had to account for a delay in signal transmission because all wireless systems have a delay. Delsys estimated the delay to be approximately 60 ms. We accounted for the delay 60 ms delay while we processed the muscle activation data. Electromyography data were collected at 1250 Hz. The input impedance of the amplifier was $>10^{15}$ megohm//0.2 pF, with a common mode rejection ratio of 90 dB, high and low pass filters of 20 and 450Hz, a signal to noise ratio of -92 dB, and a gain of 1000.

Five “good” single-leg drop landing trials were used to determine mean and peak normalized muscle activation values. Matlab software (R2008b, The Mathworks, Inc, Natick, MA) processed the raw muscle activation data postcollection. All muscle activation data were integrated and smoothed using a root mean square (RMS) algorithm with a 50 ms moving window. Mean and peak muscle activation data were normalized to reference values.

Force Plate

An AMTI OR6-5 force plate (Newton, MA) was used to measure ground reaction force and COP during single-leg drop landings. The sampling rate for ground reaction force and COP data was set at 1250 Hz. Vertical ground reaction force identified the time window. The time window ran from initial contact until the VGRF reached the subject's mass a second time following initial contact. During this time window of the single-leg drop landings; kinematic, COP, and muscle activation data were analyzed. Center of pressure was calculated three different ways: total excursion length (COPd), mean and peak center of pressure velocity (COPv).

Kinematics

We used the Vicon motion analysis system and the plug-in gait (Vicon, Centennial, CO) model to measure joint angles of the ankle, knee, and hip in the sagittal plane during single-leg drop landings. Total joint excursion and peak flexion of the ankle, knee, and hip were measured during the time window previously discussed in this section under "Force Plate." The analog output features of the Vicon Nexus system synchronized COP, muscle activation, and kinematic data. Kinematic data were collected at 250Hz using six Vicon MX13+ cameras running on Nexus 1.3 software (Vicon, Centennial, CO).[54] Two different camera set-ups were utilized during data collection; set-up was determined by leg dominance. Figures 1 & 2 illustrate the camera set-ups used for left and right leg dominant subjects.

Procedures

Orientation Session

Subjects completed the required paperwork (consent form, Ankle Instability Index, FAAM ADL & Sport) and were familiarized with how to perform AH and a single-leg drop landing. A physical exam of the ankle was completed by an experienced certified athletic trainer (12 years) to assess for mechanical ankle instability.

Subjects were taught and practiced how to correctly perform AH in the supine hook-lying position (Figure 3). The following standardized instructions were given to every subject prior to performing AH, "gently pull your umbilicus towards the table without moving your spine and maintain normal breathing."[55] Ultrasound imaging confirmed the correct performance of AH. Visual biofeedback via ultrasound imaging was provided to some subjects (~ 5) if they were unable to correctly perform AH. Subjects had to correctly perform three consecutive AH maneuvers without visual feedback, prior to the end of the orientation session.[45] Feedback was not provided during data collection.

Subjects then learned and practiced how to perform a single-leg drop landing from a 35 cm platform (Figure 4) onto their dominant leg. Leg dominance was defined as the leg the subject planted to kick a ball. All subjects were given standardized single-leg drop landing instructions. The standardized instructions are in Table 1. Single-leg drop landings were repeatedly practiced and an investigator visually determined if the drop landing was correctly performed. Subjects were required to correctly perform three consecutive single-leg drop landings prior to the completion of the session.

Pre- and Posttraining Data Collection Sessions

The pretraining data collection session occurred approximately three weeks after the orientation session. Abdominal muscle thickness was measured at rest and during AH. The remaining four dependent variables (COP, kinematics, muscle activation, and VGRF) were measured during five good single-leg drop landings. A good trial was defined as the subject landing on their dominant leg while maintaining balance for approximately three seconds without losing his/her balance. Failed landings were not included; prior to the study the maximum number of failed drop landings allowed was set at ten. All of the subjects were able to perform five "good" landings within ten trials. Approximately two minutes elapsed between trials, during this time the investigators reviewed the kinematic data.

Postural control was assessed using COPd and COPv. Kinematics measured total excursion of the ankle, knee, and hip joints along with mean and peak joint flexion angles. Muscle activation of the TrA/IO, EO, GMed, VM, and PL muscles were measured to assess muscle activation. The sequences of the pre- and posttraining data collection sessions are in Tables 2 and 3, respectively.

Abdominal thickness measurements. Thickness of the rectus abdominis (RA), EO, IO, and TrA muscles were measured using ultrasound imaging. The RA was only measured at rest while the EO, IO, and TrA were measured at rest and during AH. Rectus abdominis thickness was only measured at rest because it was not possible to simultaneously measure the RA and lateral abdominal muscles (EO, IO, & TrA) during AH with one ultrasound probe. Subjects refrained from eating or exercising for a minimum of one hour prior to all abdominal muscle thickness measurements.

Abdominal muscle thickness (RA, EO, IO, TrA) measurements were taken with subjects in a supine hook-lying position on a plinth.[43] Their hips and knees were flexed to approximately 45° and 90°, respectively.[44] The RA measurement site was lateral to the linea alba at the thickest point of the muscle and level with the umbilicus.[49] The thickest point was visibly identified by the primary investigator. Lateral abdominal muscle thickness measurements were taken level with the umbilicus and medial to the mid-axillary line on the subject's dominant side.[56] This site provided the clearest ultrasound image of the EO, IO, and TrA (Figure 5). Immediately after each measurement site was identified, a line was placed on the subject's skin to identify the measurement sites of RA and the lateral abdominal muscles. A Sharpie® marker was used to place these lines at the lateral edge of the probe (Figure 6). The lateral edge of the probe was aligned with these lines to standardize ultrasound head placement for future measurements. Each subject was provided with a Sharpie® marker to re-mark the measurement lines throughout the eight-week study. Abdominal muscle thickness was measured biweekly throughout the eight-week training program at weeks 0 (pretraining), 2, 4, 6, and 8 (posttraining). These measurements were taken on the same day and at the same time throughout the eight-week study. All muscle thickness measurements were taken by the primary investigator.

Five separate images of the RA and lateral abdominal muscles (EO, IO, and TrA) were obtained at rest, followed by five measurements of the lateral abdominal muscles during AH. Subjects held the AH maneuver for approximately six seconds to provide the primary investigator time to capture an image. Abdominal hollowing images were obtained at peak TrA thickness, which was visibly determined by the primary investigator. Approximately 30 seconds elapsed between image captures. The ultrasound imaging software's internal calipers were used to quantify muscle thickness.

To standardize abdominal thickness measurements a 25 x 18 cm transparency with a vertical center line was placed over the computer screen to identify the middle of the frozen images (Figure 7). Thickness measurements started where the superficial fascial layer and center line intersected (Figure 7).[49, 55, 57] The perpendicular distance between the superficial and deep fascial layers represented the muscle's thickness. Each image was analyzed separately. These thickness values were averaged for statistical analysis.

Electromyography, lower extremity joint angles, and center of pressure. Center of pressure, electromyography, and lower extremity joint angles during five good single-leg drop landings were measured pre- and postabdominal-training program. These variables were measured during the time window discussed previously in the methods section under Force Plate.

Surface EMG sensors were placed over the TrA/IO, EO, GMed, VM, and PL after the skin was prepped. Over the electrode site, the skin was abraded with fine sandpaper and cleansed with an alcohol wipe prior to electrode placement; correct placement was confirmed through manual muscle testing. All electrodes were aligned parallel with the orientation of muscle fibers, and placed approximately midway between the innervation zone and the insertion of the distal tendon.[58] Table 4 describes the placement and direction of these electrodes. Electromyography measured muscle activation during single-leg drop landings and while reference values were obtained. Reference values were used to normalize drop landing muscle activation pre- and postabdominal training.

Reference values were then obtained for each of these muscles using manual muscle testing. Two, five-second practice trials were given to each subject to familiarize them with the reference value position and contraction. Thereafter, muscle activation of the three, five-second trials were averaged to calculate the reference values for each muscle. The mean reference value was used to normalize the pre- and posttraining drop landing muscle activation data. Reference values for the TrA/IO were collected by having subjects perform the AH maneuver the same way they did when muscle thickness measurements were taken. Reference values of the EO, GMed, VM, and PL were obtained during maximal contractions. Figures 8 – 12 demonstrate how subjects were positioned to obtain each muscle's reference value.

Reflective markers were placed over lower extremity anatomical landmarks to measure kinematic data of the ankle, knee, and hip during the single-leg drop landings. Twenty single reflective markers were placed on every subject. The single markers were placed bilaterally over the 5th metatarsal styloid process, on the dorsum between the 2nd and 3rd phalanges, lateral malleoli, calcaneus (posterior middle), knee joint line (lateral), greater trochanter, anterior superior iliac spine, and posterior superior iliac spine. The markers over the 5th styloid processes and greater trochanters were reference markers that were used to assist in filling gaps post data collection. Figure 13 illustrates reflective marker placement for anterior, lateral, and posterior views. Anthropometric measurements were then taken and entered into Vicon Nexus. Subjects wore spandex clothing (shirt and shorts) and a standardized pair of Nike T-Lite V shoes (Nike Inc., Beaverton, OR) that we provided during data collection. Spandex clothing allowed reflective markers to be placed more accurately over anatomical landmarks and reduced the chance of loose clothing covering up markers during data collection.

Total joint excursion and peak joint angles were measured pre- and posttraining while subjects completed five good single-leg drop landings. The joint angles of interest were ankle dorsiflexion, knee and hip flexion.

Abdominal Training

The Healthy and CAI groups were taught the eight-week abdominal-training program when pretraining data collection was completed. Subjects performed the training program three days a week with one day of rest between workouts. One workout each week was completed under the direct supervision of the investigators, while the remaining two workouts were done on their own. These subjects were required to complete a weekly abdominal training exercise log; this log was returned to the investigators every week at the weekly training session.

The exercises focused on training the EO, IO, TrA, and RA muscles. Table 5 provides a summary of the abdominal-training program by weeks. The exercises chosen were based upon previously reported muscle activation of the abdominal musculature (RA, IO/TrA, and EO) during rehabilitative exercises.[59] Abdominal hollowing was performed during all of the exercises in an attempt to preferentially activate the TrA. The abdominal-training program included five different exercises: curl-up, side-bridge, sit-up with rotation, lower abdominal series (LAS), and prone-bridge.

Curl-up. Subjects laid on the floor/table in the supine hook-lying position with arms resting at their side (Figure 14). Subjects were instructed to: “1) perform AH, 2) bring chin to chest, 3) lift and slide arms forward, and 4) curl the trunk until the inferior angles of the scapula were off the floor/table” [60] (Figure 15).

Side-bridge. Instructions given to subjects for the side-bridge were, "1) assume a side lying position on one side, 2) place the elbow closest to the floor/table at a 90° angle underneath the shoulder with the forearm flat on the floor/table, 3) place the opposite arm along the upper side of the body, 4) perform AH, and 5) lift the pelvis towards the ceiling and 6) return to the side lying position" (Figures 16 and 17). This exercise was performed bilaterally.

Sit-up with rotation. Subjects started in the supine hook-lying position with their arms crossed against their chest. The instructions were to "1) lift the trunk off the floor/table, 2) rotate as the trunk was flexed until the left elbow touched the right knee or the right elbow touched the left knee, 3) return to the starting position, and 4) repeated steps 1 and 2 to the opposite side" (Figures 18-21).

Lower abdominal series (LAS). This series consists of five different levels of exercises that progress in difficulty. Four of those levels were included in this eight-week program. Level four of the LAS was skipped because previously individuals stated that level 3 was harder than level four. Therefore, we chose to eliminate level four. Level one was considered the easiest and level five the most difficult. This exercise focused on training the TrA. Abdominal hollowing was performed throughout these exercises. Common mistakes individuals made throughout these exercises included holding one's breath, contracting the gluteal and hamstring muscles, lifting the head, and abdominal pouching. Abdominal pouching is the visible contraction of the RA instead of hollowing the abdominal cavity.[61] The starting position for all of the LAS exercises was the supine hook-lying position. The levels are illustrated in Figures 22-25. The instructions provided to the subjects for each level are described in Table 6.

Prone-bridge. The prone-bridge exercise was added to the training program during the fourth week. It provided some variation to the program and increased the demands placed on the abdominal muscles. Subjects started prone with their elbows under their shoulders (Figure 26). They lifted their pelvis until they reached the peak position, which was when the shoulders, pelvis, and ankles were in a straight line (Figure 27). The peak position was held for the assigned time.

STATISTICAL ANALYSIS

SPSS 16.0 (SPSS Inc., Chicago, IL, USA) was used to manage and analyze the data. The dependent variables were muscle thickness, COP, muscle activation, kinematics, and VGRF, while the independent variables were group and time. The means of five trials were averaged and used for statistical analysis of the dependent variables. Differences between groups prior to the abdominal-training program were assessed using a one-way analysis of variance (ANOVA). A General Linear Model repeated measures ANOVA was used to analyze abdominal muscle thickness, COP excursion, muscle activation, and kinematic differences between groups following the eight-week abdominal-training program. Tukey-Kramer post-hoc multiple comparison tests were performed to make pairwise contrasts between groups. A simple *t-test* with a Bonferonni adjustment determined if a difference existed within groups, pre- and postabdominal training. The partial eta^2 statistic was used to report effect size.

RESULTS

The results were separated into the “pretraining data” and “posttraining data” sections. Data for the five dependent variables were reported under each of those sections. The data reported in the pretraining data section represent the differences observed between groups prior to training. Results under the posttraining data section are the differences observed between and within each group over time (eight weeks).

Pretraining Data

Abdominal Muscle Thickness

Mean muscle thickness values at rest and during AH are in Tables 7 & 8. Muscle thickness of the RA, EO, IO, and TrA at rest and during AH were not different between groups at rest (RA: $F_{(57, 2)} = 1.17, p = 0.318$; EO: $F_{(57, 2)} = 0.77, p = 0.468$; IO: $F_{(57, 2)} = 0.55, p = 0.582$; TrA: $F_{(57, 2)} = 1.16, p = 0.319$) or during AH (EO: $F_{(57, 2)} = 1.04, p = 0.360$; IO: $F_{(57, 2)} = 0.33, p = 0.722$; TrA: $F_{(57, 2)} = 0.18, p = 0.834$).

Kinematics

Joint excursion and peak flexion angle means along with effect size for the ankle (dorsiflexion), knee, and hip joint are in Table 9. Mean joint excursion (Ankle: $F_{(57, 2)} = 1.82, p = 0.172$; Knee: $F_{(57, 2)} = 0.30, p = 0.742$; Hip: $F_{(57, 2)} = 0.49, p = 0.613$) and peak joint angles (Ankle: $F_{(57, 2)} = 0.60, p = 0.550$; Knee: $F_{(57, 2)} = 0.15, p = 0.864$; Hip: $F_{(57, 2)} = 0.13, p = 0.878$) were not different between groups.

Electromyography

Mean and peak muscle activation values for the TrA/IO, EO, GMed, VM, and PL muscles are in Tables 10 and 11. A difference between groups was observed in peak and mean TrA/IO (peak: $F_{(57, 2)}$= 3.28, p = 0.045; mean: $F_{(57, 2)}$= 3.29, p = 0.044), EO (peak: $F_{(57, 2)}$=: $F_{(57, 2)}$= 7.25, p = 0.002; mean: $F_{(57, 2)}$= 13.06, p < 0.001), and GMed (peak: $F_{(57, 2)}$= 3.20, p = 0.048; mean: $F_{(57, 2)}$= 3.14, p = 0.051). There was no difference between groups for peak and mean VM (peak: $F_{(57, 2)}$ = 0.42, p = 0.657; mean: $F_{(57, 2)}$ = 0.25, p = 0.780) and PL (peak: $F_{(57, 2)}$ = 0.36, p = 0.698; mean: $F_{(57, 2)}$ = 0.26, p = 0.772) muscle activation values.

Pair-wise post-hoc comparisons revealed the CAI group had greater mean and peak TrA/IO (peak: p = 0.041; mean: p = 0.039) and EO (peak: p = 0.022; mean: p = 0.002) muscle activation than the Control group. The CAI group had greater mean and peak EO (peak: p = 0.002; mean: p < 0.001), and GMed (peak: p = 0.037; mean: p = 0.040) muscle activation than the Healthy group. No differences were observed between groups for mean and peak VM and PL muscle activation.

Center of Pressure

Tables 12 and 13 contain the means and effect size for the COP variables (COPd, peak and mean COPv, and time window pre training). Peak and mean COPv (peak: $F_{(57, 2)}$ = 9.16, p < 0.001; mean: $F_{(57, 2)}$ = 8.31, p = 0.001), were different between groups. No difference in COPd *(*$F_{(57, 2)}$ = 1.26, p = 0.291) or time window *(*$F_{(57, 2)}$ = 0.88, p = 0.422) were observed between groups.

Pair-wise comparisons showed the Control group had greater COPv (peak p < 0.001; mean: p = 0.001) than the Healthy group. Control subjects also had greater peak COPv (p < 0.001) than CAI subjects. Chronic ankle instability subjects had greater mean COPv than Healthy subjects (p = 0.021).

Vertical Ground Reaction Force

Peak and mean VGRF were reported in Table 14. A difference between groups was observed for peak VGRF $(F_{(57,2)} = 3.43, p = 0.039)$ but not mean VGRF $(F_{(57,2)} = 1.68, p = 0.195)$. The CAI group demonstrated greater peak VGRF than the Healthy group ($p = 0.033$).

Posttraining Data

Abdominal Muscle Thickness

Posttraining mean muscle thickness values at rest and during AH along with effect size are in Tables 7 and 8. An interaction between time and group existed for RA thickness at rest ($F_{(8,4)} = 4.07, p < 0.001$). The CAI ($p < 0.001$) and Healthy ($p < 0.001$) group's thickness increased while the Control group was unchanged. Post-hoc comparisons revealed no difference in RA thickness between the combined mean thicknesses of the three groups before and after training. Mean EO thickness at rest increased in the CAI ($p < 0.001$) and Healthy ($p = 0.002$) groups. External oblique thickness was greater in the CAI group than the Control group ($p = 0.013$). No changes were observed in IO and TrA muscle thickness at rest (IO: $F_{(8,4)} = 0.33, p = 0.857$; TrA: $F_{(8,4)} = 1.33, p = 0.261$).

During AH, thickness changes were observed in the EO, IO, and TrA muscles. An interaction was ($F_{(8,4)} = 3.84, p < 0.001$) present between EO thickness and group. Post-hoc tests revealed that EO thickness increased in both the CAI ($p < 0.001$) and Healthy ($p < 0.001$) groups following the eight-week abdominal-training program. The CAI ($p = 0.003$) and Healthy ($p = 0.050$) groups had thicker EO muscles during AH than the Control group. Mean IO and TrA thickness during AH did not change between (IO: $F_{(2, 1)} = 0.26, p = 0.774$; TrA: $F_{(2, 1)} = 0.073, p = 0.930$) or within (IO: $F_{(8, 4)} = 1.21, p = 0.306$; TrA: $F_{(8, 4)} = 1.47, p = 0.214$) groups. Despite the lack of within group significance, the Healthy group's IO ($p = 0.004$) and TrA ($p = 0.033$) thickness during AH increased.

Kinematics

Joint excursion and peak flexion angle means along with effect size following training are in Table 9 for the ankle (dorsiflexion), knee, and hip joint. An interaction existed between group and time for ankle excursion. No differences existed between or within the groups for ankle excursion (within: $F_{(2, 1)} = 0.63, p = 0.430$; between: $F_{(2, 1)} = 1.27, p = 0.289$) and peak ankle angle (within: $F_{(2, 1)} = 3.78, p = 0.057$; between: $F_{(2, 1)} = 0.020, p = 0.980$). The Control group's peak ankle angle differed between pre- and postmeasurements ($p = 0.005$). Knee excursion differed within groups ($F_{(2, 1)} = 5.24, p = 0.026$). No differences existed between or within groups for peak knee angle (within: $F_{(2, 1)} = 0.24, p = 0.627$; between: $F_{(2, 1)} = 0.30, p = 0.741$), hip excursion (within: $F_{(2, 1)} = 1.05, p = 0.311$; between: $F_{(2, 1)} = 0.64, p = 0.534$): and peak hip angle (within: $F_{(2, 1)} = 0.34, p = 0.564$: between: $F_{(2, 1)} = 0.045, p = 0.956$).

Electromyography

Tables 10 and 11 contain posttraining muscle activation values (peak and mean) and effect size for all muscles. A difference was present within groups for peak ($F_{(2, 1)} = 5.40, p = 0.024$) and mean ($F_{(2, 1)} = 4.36, p = 0.041$) TrA activation. The CAI group's peak (p = 0.002) and mean (p = 0.003) TrA/IO muscle activation decreased significantly following the training program.

Peak and mean EO muscle activation differed between groups (peak: $F_{(2, 1)}$ = 3.50, p = 0.037; mean: $F_{(2, 1)}$ = 4.28, p = 0.018). The CAI group had greater peak and mean EO muscle activation than the Control (peak: p = 0.084; mean: p = 0.072) and Healthy (peak: p = 0.054; mean: p = 0.023) groups. Peak and mean EO muscle activation data decreased following abdominal training in both the CAI and Healthy groups, however, it was not significant.

Peak and mean GMed decreased in all groups and group differences were observed (peak: $F_{(2, 1)}$ = 5.14, p = 0.009; mean: $F_{(2, 1)}$ = 6.43, p = 0.003). The CAI group had greater peak and mean GMed (peak: p = 0.007; mean: p = 0.002) muscle activation than the Healthy group.

Peak ($F_{(2, 1)}$ = 18.20, p < 0.001) and mean ($F_{(2, 1)}$ = 23.11, p < 0.001) VM activation decreased within the CAI and Healthy groups. A decrease in peak (CAI: p = 0.003; Healthy: p = 0.009) and mean VM muscle activation (CAI: p =0.008; Healthy: p =0.001) were observed in the CAI and Healthy groups.

A difference within groups for peak ($F_{(2, 1)}$ = 4.65, p = 0.035) and mean ($F_{(2, 1)}$ = 4.21, p = 0.045) PL activation existed, however, post-hoc testing showed no statistical difference.

Center of Pressure

Posttraining means and effect size for COPd and COPv are displayed in Table 12. Differences within groups existed for COPd ($F_{(2, 1)} = 12.97, p = 0.001$). Center of pressure distance increased in the CAI (p =0.053) and Healthy groups (p =0.009).

An interaction was present between group and time on the mean COPv variable ($F_{(2, 1)} = 18.72, p < 0.001$). Mean COPv demonstrated within group differences ($F_{(2, 1)} = 50.98, p < 0.001$). The CAI ($p < 0.001$) and Healthy ($p < 0.001$) group's mean COPv increased after the training program. Peak COPv differences existed within ($F_{(2, 1)} = 35.32, p < 0.001$) and between ($F_{(2, 1)} = 18.98, p < 0.001$) groups. The Control group had greater peak COPv than the CAI ($p = 0.032$) and Healthy ($p< 0.001$) groups, while the CAI group had greater mean COPv than the Healthy group ($p = 0.002$). Peak COPv increased in the CAI and Healthy groups (CAI: $p < 0.001$; Healthy: $p = 0.004$).

Posttraining means for the time window are in Table 13. An interaction between time window and group was present when pre- and posttraining measurements were compared ($F_{(2, 1)} = 3.41, p = 0.040$). The Control group's time window increased ($p = 0.015$).

Vertical Ground Reaction Force

The mean and peak VGRF means and effect size following training are in Table 14. Peak and mean VGRF varied within groups (peak: $F_{(2, 1)} = 5.45, p = 0.023$; mean: $F_{(2, 1)} = 4.14, p = 0.047$). The CAI group's VGRF decreased following abdominal training (peak:, $p = 0.002$; mean: $p = 0.006$).

DISCUSSION

Abdominal Thickness

Mean thickness values observed prior to training were similar to those previously reported in healthy subjects.[49, 62] Abdominal muscle thickness was not different between groups prior to abdominal-training. Following the eight-week abdominal-training program, the CAI and Healthy groups demonstrated increased RA and EO muscle thickness at rest and during AH. Thickness changes were not observed in the TrA or IO muscles despite the focus placed on performing AH in an attempt to activate the TrA during all training exercises.

Increased thickness or morphological changes are a sign of increased strength.[63] Although IO and TrA thickness at rest and during AH was unchanged, it does not mean strengthening did not occur. Strength increases can occur without morphological changes.[63-65] Strength increases observed during the first four to six weeks of any training program are largely due to neurological adaptations.[63] Changes in RA and EO thickness may indicate that the eight-week training program strengthened those muscles in the CAI and Healthy groups. The TrA and IO muscles may have become stronger without morphological changes. It may take longer than eight weeks of training to observe thickness changes in the TrA and IO muscles. Another explanation for no TrA and IO thickness changes was that subjects relied more on their global (RA and EO) than local abdominal muscles (IO and TrA) during the training exercises.

Our data demonstrate that eight weeks of training using the training program discussed in this study is enough time to see morphological changes in the RA and EO.

The IO and TrA muscles may have been strengthened even though there were no changes in thickness. This could be evident by the reduction in TrA/IO muscle activation following training.[66] A reduction in muscle activation is a sign of increased strength.[66] Therefore, even though TrA/IO thickness did not increase at rest we still believe the TrA and IO were strengthened.

Kinematics

The Control group differed between pre- and postmeasurements of peak knee flexion. Knee excursion differed within groups although post-hoc tests revealed no difference. Kinematic differences were previously observed in the sagittal plane between CAI and Healthy subjects.[67, 68] Limited dorsiflexion was previously reported during landing as a potential cause of CAI.[68] Our CAI subjects did not demonstrate deficits in mean or peak dorsiflexion. Therefore our data are inconsistent with prior research that stated CAI subjects may not dorsiflex their ankle as much as healthy subjects. However, we defined a shorter time window to analyze kinematics than previously used; this may explain why no differences were observed between groups in this study.

People use an ankle, hip, or combination (hip and ankle) strategy during landing to maintain postural control.[69] An ankle strategy is identified by less joint excursion or a stiff landing while a hip strategy demonstrates greater lower extremity joint excursion.[69] The ankle landing strategy places greater demands on the ankle and lower leg musculature while the hip and combination strategies transfer more energy up the kinetic chain.[69]

It has been reported that CAI and healthy subjects use different landing strategies.[18, 70, 71] Chronic ankle instability subjects use an ankle strategy while healthy subjects utilize a hip or combination strategy during single-leg landings to maintain postural control.[67, 69, 72] A soft landing or hip strategy is identifiable by greater knee flexion.[69] Prior to abdominal training, it does not appear that our CAI group used only an ankle strategy. This is inconsistent with previously published research.[67, 69, 72]

Although not significant, changes in knee joint kinematics occurred following abdominal-training. The CAI and Healthy groups increased peak knee flexion angles following training. Despite the lack of significance greater peak knee flexion is consistent with the idea that training may allow individuals to transition from an ankle strategy to a combination strategy.

Electromyography

Muscle activation varied between groups pre- and postabdominal training. The CAI group had greater peak and mean proximal muscle activation than the Control (TrA/IO and EO) and Healthy (EO and GMed) groups prior to training. Chronic ankle instability subjects may have relied more on their proximal muscles to maintain postural control than the Control and Healthy groups. This could be due to learned compensatory strategies to account for neuromuscular deficits and/or it may suggest the use of a feedforward mechanism. Vastus medialis and PL activation were not different between groups before training.

Decreased muscle activation was observed in the CAI and Healthy groups following training. Muscle activation decreased in all of the muscles, however only the

TrA/IO, VM, and PL were significantly reduced. The CAI group had decreased TrA/IO, VM, and PL activation while Healthy subjects had decreased VM activity. Decreased muscle activation following training indicates improved neuromuscular efficiency.[66] The CAI and Healthy groups were able to perform the same task (single-leg drop landing) with the recruitment of fewer motor units/muscle fibers following abdominal-training. This supports our theory that the TrA and IO were strengthened despite no change in their thickness at rest. Therefore neuromuscular efficiency was improved by abdominal-training. External oblique and GMed muscle activation remained greater in the CAI than Healthy group.

Prior research observed differences in proximal muscle activation between healthy subjects and those with a history of ankle sprains.[28, 29, 31] A delay in gluteus maximus activation was observed by Bullock-Saxton et al[29] in previously injured subjects. Beckman and Buchanan[28] observed that subjects with a history of ankle sprains activated their GMed earlier in response to a perturbation than healthy controls. Both of those studies assessed the onset of muscle activation and did not report amplitudes. We did not assess the onset of muscle activation. However, we did observe greater proximal muscle activation in CAI subjects than Control (pretraining: TrA/IO and EO) and Healthy (pretraining: TrA/IO and EO; posttraining: EO and GMed) subjects.

This may suggest that CAI subjects use a combination of an ankle and hip landing strategy. This is consistent with recently published research that stated CAI subjects may not use an ankle strategy only to maintain postural control.[24, 67] If CAI subjects rely on more than an ankle strategy, this would suggest that they may use a feedback and feedforward mechanism. If CAI subjects use both feedback and feedforward mechanisms then neuromuscular changes should be observed down the lower kinetic chain during a task. We observed a decrease in EMG amplitude following training, this suggests improved neuromuscular efficiency.[66, 73] Improved neuromuscular efficiency is represented physiologically by a decrease in the number of motor units required to perform a task.[66] The CAI subjects in our study demonstrated less motor unit recruitment while performing a single-leg drop landing following training than prior to training. This suggests a change in the central nervous system motor strategies. A change in the central nervous system indicates CAI subjects may use a feedforward mechanism to maintain postural control in addition to a feedback mechanism. Therefore our data are consistent with the theory that CAI subjects may use both feedback and feedforward mechanisms to maintain postural control. If this is the case, clinicians may want to focus rehabilitation efforts on the entire kinetic chain following an ankle injury.

We observed no differences in peak or mean PL and VM activation prior to training. One potential explanation for no differences in muscle activation may be due to the preactivation (feedforward mechanism) of muscles prior to initial contact. Therefore a feedforward mechanism may compensate for lower extremity deficits. Prior research observed quadriceps facilitation and inhibition of the hamstrings during maximal voluntary contractions in CAI subjects.[34] Facilitation of a muscle increases muscle activation above its normal amplitude. Quadriceps facilitation may explain why we observed no difference in VM muscle activation. Although a similar landing study reported decreased PL activation prior to initial contact but no differences post-initial contact.[68] We did not assess muscle activation prior to initial contact; however, our data are consistent with no differences in PL activation post-initial contact.[68] If PL deficits exist prior to initial contact and do not following contact this may suggest the combination of feedforward and feedback mechanisms actually assist in maintaining dynamic postural control during a single-leg drop landing. Although deficits in PL activation prior to initial contact may also suggest the PL is not activating properly prior to landing. This suggests the feedback and feedforward mechanisms used to maintain postural control may be affected by CAI, therefore increasing the risk of recurrent ankle sprains. Further research is needed to determine the relationship between feedforward and feedback mechanisms in CAI subjects.

Muscle activation changed following the eight-week abdominal-training program. The CAI and Healthy subjects demonstrated decreased muscle activation. Decreased muscle activation during a single-leg drop landing indicates the lower extremity neuromuscular system became more efficient after abdominal training in performing the same task. They did not have to recruit as many muscles fibers to maintain postural control posttraining. Decreased muscle activation in CAI (VM and PL) and Healthy (VM) subjects suggest that abdominal training does influence muscle activation down the lower kinetic chain. Increased gluteus maximus and medial hamstring muscle activation were previously observed when subjects performed AH during prone hip extension.[74] A more efficient neuromuscular system (greater endurance) may decrease the risk of lower extremity injuries during landing by providing a more stable base over time.

Vertical Ground Reaction Force

We observed greater peak and mean VGRF in the CAI group than the Healthy group prior to abdominal-training. The CAI and Healthy groups' peak and mean VGRF were decreased following training.

Previous researchers reported differences in ground reaction forces between CAI and healthy subjects.[72] Researchers previously observed that CAI subjects had greater peak VGRF and reached those peaks sooner than healthy subjects.[72] We did not assess the timing of peak force. Our data before training were consistent with previous research that demonstrated that CAI subjects generate greater VGRF than Healthy subjects.

The greater VGRF observed in CAI subjects may help explain why they experience recurrent ankle sprains.[72] Following abdominal-training, the CAI group's mean and peak VGRF decreased. This demonstrates that eight weeks of abdominal training can decrease the forces that act on the foot and ankle to cause injury. If VGRF can be reduced in CAI subjects through abdominal-training, the likelihood of recurrent ankle sprains may also be prevented or reduced. Therefore, clinicians may want to include abdominal training as part of their patient's ankle rehabilitation programs.

Center of Pressure

The Control and CAI groups had greater mean COPv than the Healthy group prior to training. Peak COPv was also greater in the Control group than the CAI and Healthy groups during pretraining data collection. There was no difference between groups for COPd before training. After training, the Control group had greater peak COPv than both the CAI and Healthy groups. Center of pressure excursion and COPv (peak and mean) was increased in the CAI and Healthy groups following abdominal-training. We speculate abdominal training made the spine more stable by decreasing center of gravity variation. This allowed subjects to deal with greater speeds during landing (increased COPv) and potentially more movement in the lower kinetic chain (increased COPd), which would be characteristic of a softer landing (decreased VGRF).

At first glance it would appear that COPd and COPv (peak and mean) became worse following training. Prior research concluded that increased COPd and COPv represent a decline in postural control.[75] However, when you consider COPd and COPv (peak and mean) increased while VGRF decreased following abdominal training, an increase in COP may be a positive outcome. We theorize increased COPd and COPv in conjunction with decreased VGRF during a functional dynamic task may represent improved dynamic postural control. Chronic ankle instability subjects have demonstrated greater VGRF postinitial contact compared to healthy controls.[68] Greater VGRF is thought to contribute to CAI.[68] Therefore if abdominal training decreased VGRF and increased COPd and COPv, we theorize dynamic postural control may actually be improved due to the relationship between VGRF and COP. Increased COPd and COPv suggest the CAI and Healthy groups were able to travel faster (COPv) with greater COPd following training. This may represent improved dynamic postural stability during a dynamic functional task even though it is contrary to previous COP data. It is important to recognize that most COP research has been completed during a static task, while we used a dynamic task.

Our data contradict previous research that states differences in force plate measures exist between Healthy and CAI subjects prior to training. Although there seems to be some confusion with regard to which COP measurement is the most accurate at detecting postural control changes. Previously it was reported that COPd may be a more sensitive measure of postural control than other measurement methods.[22] This may be limited to a static stance. However, a recent study set out to determine the most accurate force-plate measure to distinguish between CAI and Healthy subjects. Ross et al[76] concluded that medial/lateral ground reaction force standard deviation and anterior/posterior time to stabilization were the most accurate at discriminating between CAI and Healthy subjects. That may be why our CAI group's COPd, and COPv were not different than the Control and Healthy groups prior to training.

Limitations

Like all research studies ours had limitations. Center of pressure was the only measure of postural control used. It would have been beneficial to have another measure of postural control to compare with our COP data. The use of surface electrodes to measure muscle activation is another limitation. Surface electrodes provide an estimation of the muscle activation of muscle fibers only underneath the electrode.[73] Due to the depth and location of the TrA and IO, muscle activation for those muscles can not be reported separately without the use of fine-wire electrodes. Another limitation is the abdominal training may have also trained other muscles (eg. erector spinae, multifidi, hip flexors, gluteal, quadriceps, hamstrings muscles).

Future Research

Future research needs to focus on understanding the role abdominal muscles have during landing in healthy and injured subjects. It would be beneficial to determine the time required to achieve hypertrophy of the TrA and IO at rest. This would assist clinicians in determining the length of their patients' training programs. Further studies should determine what effect training CAI subjects how to land has on lower extremity muscle activation and postural control. Our study measured kinematic changes only in the sagittal plane; future research should assess frontal plane changes following training. Further research needs to determine if decreasing VGRF results a decreased risk of lower extremity injuries.

CONCLUSIONS

Eight weeks of abdominal training resulted in changes in RA and EO muscle thickness, muscle activation (TrA/IO, VM, and PL), and postural control for the CAI and Healthy groups. No significant kinematic differences were observed. Our training program caused thickness changes to occur in the RA and EO muscles at rest and during AH but not the IO and TrA. Training decreased muscle activation of the proximal and distal muscles. This suggests abdominal training may improve neuromuscular function down the lower kinetic chain by potentially enhancing the capabilities of feedforward mechanisms. Our data are consistent with the hypothesis that CAI subjects use feedforward and feedback mechanisms to maintain postural control.

This study demonstrated the importance of training muscles proximal to the ankle in an attempt to prevent and reduce CAI. Clinicians must not train only the foot and lower leg musculature following an ankle sprain but they should consider training the entire kinetic chain.

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Table 1. Single-Leg Drop Landing Instructions

Steps	Instructions
1	Step up onto the 35 cm platform
2	Place your hands approximately 6” (15.2 cm) above your hips to avoid covering up the reflective markers
3	Move the dominant limb in front of the platform
4	Lean forward and drop off the platform; guide the dominant foot towards the center of the force plate as you drop; prior to landing pull the nondominant foot away from the platform
5	Land as you normally would
6	Upon landing stand erect on your dominant limb; locate the camera directly in front of you with your eyes; maintain your balance for approximately five seconds

Table 2. Sequence of the Pretraining Data Collection Session

Data Collection Timeline
1. Demographic data was collected
2. Subjects were assigned to a group
3. Subjects practiced correctly performing AH
4. Abdominal muscle thickness measurement sites were identified & marked with a permanent marker
5. Subjects correctly performed three consecutive AH, confirmed via ultrasound imaging
6. Took five RA muscle thickness measurements at rest
7. Took five lateral abdominal muscle thickness measurements at rest
8. Took five lateral abdominal muscle thickness measurements during AH
9. Placed surface EMG electrodes & reflective markers on the subject
10. Anthropometric measurements were taken
11. Subjects practiced performing a single-leg drop landing
12. Subjects correctly performed three consecutive single-leg drop landings
13. Five single-leg drop landing trials were performed for data collection (COP, EMG, & kinematics)
14. The CAI and Healthy groups were instructed on how to perform the abdominal strengthening program

Abbreviation: AH, abdominal hollowing; COP, center of pressure; EMG, electromyography; USI, ultrasound imaging

Table 3. Sequence of the Posttraining Data Collection Session

Data Collection Timeline
1. Took five RA muscle thickness measurements at rest
2. Took five lateral abdominal muscle thickness measurements at rest
3. Took five lateral abdominal muscle thickness measurements during AH
3. Placed surface EMG electrodes & reflective markers on the subject
4. Anthropometric measurements were taken
5. Subjects practiced performing a single-leg drop landing
6. Subjects correctly performed three consecutive single-leg drop landings
7. Five single-leg drop landing trials were performed for data collection (COP, EMG, & kinematics)

Abbreviation: AH, abdominal hollowing; COP, center of pressure; EMG, electromyography

Table 4. Surface Electrode Placement

Muscle	Electrode Direction	Electrode Placement
External Oblique	Oblique	Approximately 12-15 cm lateral to the umbilicus[77]
Internal Oblique / Transverse Abdominis	Transverse	2cm medial and inferior to the anterior superior iliac spine[77]
Gluteus Medius	Longitudinal	Halfway between the greater trochanter and lateral most aspect of the iliac crest[59, 78]

Table 5. Eight-Week Abdominal-Training Program

Week One	Repetitions	Week Five	Repetitions
Level 1 LAS	2 sets of 10	Level 3 LAS	2 sets of 10
Curl-up	2 sets of 10	Curl-up	2 sets of 20
Side-bridge	2 sets of 10	Side-bridge	2 sets of 20
Sit-up with rotation	2 sets of 10	Sit-up with rotation	2 sets of 20
		Prone-bridge	3 sets of 10s
Week Two	**Repetitions**	**Week Six**	**Repetitions**
Level 1 LAS	3 sets of 10	Level 3 LAS	3 sets of 10
Curl-up	3 sets of 10	Curl-up	3 sets of 20
Side-bridge	3 sets of 10	Side-bridge	3 sets of 20
Sit-up with rotation	3 sets of 10	Sit-up with rotation	3 sets of 20
		Prone-bridge	2 sets of 15s
Week Three	**Repetitions**	**Week Seven**	**Repetitions**
Level 2 LAS	2 sets of 10	Level 5 LAS	2 sets of 10
Curl-up	2 sets of 15	Curl-up	2 sets of 25
Side-bridge	2 sets of 15	Side-bridge	2 sets of 25
Sit-up with rotation	2 sets of 15	Sit-up with rotation	2 sets of 25
		Prone-bridge	3 sets of 15s
Week Four	**Repetitions**	**Week Eight**	**Repetitions**
Level 2 LAS	3 sets of 10	Level 5 LAS	3 sets of 10
Curl-up	3 sets of 15	Curl-up	3 sets of 25
Side-bridge	3 sets of 15	Side-bridge	3 sets of 25
Sit-up with rotation	3 sets of 15	Sit-up with rotation	3 sets of 25
Prone-bridge	2 sets of 10s	Prone-bridge	4 sets of 15s

Abbreviation: LAS, Sahrmann lower abdominal series; s, seconds

Table 6. Description of Lower Abdominal Series

LAS Level	Description
1	Subjects lifted the first leg to 90° of hip flexion followed by the 2nd leg, the knees flexed as the hips flexed; the first leg was then lowered to the starting position, followed by the second leg
2	Subjects lifted the first leg to 90° of hip flexion followed by the 2nd leg, the knees flexed as the hips flexed; the heel of the first leg was then lowered and slid across the floor/table until it became straight; the first leg returned to 90° of hip flexion by sliding the heel across the table; this was repeated by the second leg
3	Subjects lifted the first leg to 90° of hip flexion followed by the 2nd leg, the knees flexed as the hips flexed; the first leg was then straightened without the heel touching the floor/table until it became straight; the first leg returned to 90° of hip flexion; this was repeated by the second leg
4	Subjects started with both legs straight; both legs were then flexed to 90° of hip flexion, the knees flexed as the hips flexed; the legs were not allowed to touch the floor/table; both legs were straightened without touching the floor/table

Abbreviation: LAS, Sahrmann lower abdominal series

Table 7. Abdominal Muscle Thickness at Rest[a] (mm)

	RA @ rest		EO @ rest		IO @ rest		TrA @ rest	
	Pre	Post[*]	Pre	Post	Pre	Post	Pre	Post
Control	10.7 ± 1.9	10.7 ± 2.0	6.1 ± 1.2	6.2 ± 1.3[ǀ]	9.4 ± 2.2	9.2 ± 2.2	3.8 ± 0.9	3.7 ± 0.9
CAI	11.9 ± 2.9[‡]	12.8 ± 2.9[‡]	6.7 ± 1.6[§]	7.9 ± 2.1[§ǀ]	10.0 ± 3.3	9.7 ± 2.1	4.0 ± 1.3	3.8 ± 0.9
Healthy	11.3 ± 2.5[†]	12.9 ± 2.8[†]	6.3 ± 1.4[‖]	7.3 ± 1.9[‖]	9.2 ± 2.5	9.6 ± 2.8	3.6 ± 0.7	3.7 ± 0.8
Effect size[b]	0.076		0.018		0.009		0.016	

Abbreviations: mm, millimeter(s); Post, posttraining data collection; Pre, pretraining data collection

[a] Values are expressed as mean ± standard deviation

[b] Partial eta^2 statistic was used to report effect size

[*] Interaction existed between rectus abdominis thickness and group, $p < 0.001$

[†] Rectus abdominis thickness increased within groups between the pre- and posttraining data collection sessions; $p < 0.002$

[‡] Rectus abdominis thickness increased within groups between the pre- and posttraining data collection sessions; $p < 0.001$

[§] External oblique thickness increased within groups between the pre- and posttraining data collection sessions; $p < 0.001$

[‖] External oblique thickness increased within groups between the pre- and posttraining data collection sessions; $p = 0.002$

[ǀ] CAI ≥ Control; $p = 0.013$

Table 8. Abdominal Muscle Thickness during Abdominal Hollowing[a] (mm)

	Pre	Post	Pre	Post	Pre	Post
Control	6.2 ± 1.4	6.3 ± 1.4[§‖]	10.6 ± 2.7	11.0 ± 3.3	5.8 ± 1.0	6.0 ± 1.3
CAI	6.9 ± 1.5[†]	8.3 ± 2.4[†§]	11.4 ± 3.9	11.2 ± 2.5	5.9 ± 1.8	5.9 ± 1.4
Healthy	6.5 ± 1.6[‡]	7.8 ± 1.7[‡‖]	11.0 ± 3.4[|]	12.3 ± 4.3[|]	5.6 ± 1.2	6.3 ± 1.6
Effect size[b]	0.177		0.009		0.003	

Abbreviations: mm, millimeter(s); Post, posttraining data collection; Pre, pretraining data collection

[a] Values are expressed as mean ± standard deviation

[b] Partial eta^2 statistic was used to report effect size

[*] An interaction existed between group and time for external oblique thickness; $p < 0.001$

[†] External oblique thickness increased within the CAI group between the pre- and posttraining data collection sessions; $p < 0.001$

[‡] External oblique thickness increased within the Healthy group between the pre- and posttraining data collection sessions; $p < 0.001$

[§] CAI ≥ Control; $p = 0.003$

[‖] Healthy ≥ Control; $p = 0.050$

[|] Internal oblique thickness increased within the Healthy group between the pre- and posttraining data collection sessions; $p = 0.004$

Table 9. Lower Extremity Joint Excursion and Peak Angles[a] (°)

	Ankle Excursion		Ankle Peak	
	Pre	Post*	Pre	Post
Control	42.6 ± 15.9	45.9 ± 12.6	18.7 ± 7.1†	24.0 ± 9.9†
CAI	43.3 ± 13.1	41.0 ± 14.9	20.9 ± 7.1	21.4 ± 6.9
Healthy	50.4 ± 14.1	46.6 ± 11.7	21.5 ± 10.3	21.7 ± 8.6
Effect size[b]	0.043		0.001	

	Knee Excursion		Knee Peak	
	Pre	Post	Pre	Post
Control	29.0 ± 7.1	25.8 ± 6.7	33.3 ± 7.2	30.9 ± 9.6
CAI	27.8 ± 9.7	25.4 ± 9.8	33.3 ± 11.1	35.9 ± 20.2
Healthy	26.7 ± 9.7	25.6 ± 10.5	31.8 ± 10.8	34.4 ± 12.8
Effect size[b]	0.004		0.010	

	Hip Excursion		Hip Peak	
	Pre	Post	Pre	Post
Control	16.9 ± 15.5	13.7± 8.3	34.8 ± 16.6	30.0 ± 17.0
CAI	16.0 ± 10.1	16.5 ±11.0	33.7 ± 15.6	33.2 ± 15.4
Healthy	13.5 ± 6.6	12.7 ± 7.7	32.4 ± 11.8	34.5 ± 12.3
Effect size[b]	0.022		0.002	

Abbreviations: °, degree(s); Post, posttraining data collection; Pre, pretraining data collection

[a] Values are expressed as mean ± standard deviation

[b] Partial eta^2 statistic was used to report effect size

* An interaction existed between group and time for peak ankle excursion; *p* = 0.052

† Peak ankle angle increased within the Control group between the pre-and posttraining data collection sessions; *p* = 0.005

Table 10. Mean Normalized EMG[a] (%)

	Transverse Abdominis/Internal Oblique		External Oblique	
	Pre	Post	Pre	Post
Control	311.7 ± 176.2[*]	378.7 ± 510.3	194.5 ± 101.8[†]	138.3 ± 97.1
CAI	855.0 ± 1038.2[*,‖]	365.0 ± 292.3[‖]	328.4 ± 163.6[†,‡]	287.9 ± 602.5[ǀ]
Healthy	503.6 ± 481.5	332.8 ± 572.3	148.7 ± 56.3[‡]	127.7 ± 187.0[ǀ]
Effect size[b]	0.060		0.131	

	Gluteus Medius		Vastus Medialis	
	Pre	Post	Pre	Post
Control	91.2 ± 69.0	70.5 ± 20.0	520.5 ± 719.5	202.6 ± 82.7
CAI	109.4 ± 62.2[§]	91.8 ± 43.6[¶]	611.3 ± 478.4[∞]	219.7 ± 145.8[∞]
Healthy	65.6 ± 29.9[§]	50.2 ± 23.4[¶]	665.8 ± 726.5[‼]	157.4 ± 66.7[‼]
Effect size[b]	0.184		0.006	

	Peroneus Longus	
	Pre	Post
Control	323.7 ± 739.0	126.8 ± 110.4
CAI	356.1 ± 836.0	126.0 ± 81.2
Healthy	212.8 ± 257.5	107.8 ± 54.1
Effect size[b]	0.012	

Abbreviations: %, percent; Post, posttraining data collection; Pre, pretraining data collection

[a] Values are expressed as mean ± standard deviation

[b] Partial eta^2 statistic was used to report effect size

[*] CAI ≥ Control; $p \leq 0.07$

[†] CAI ≥ Control; $p \leq 0.00$

[‡] CAI ≥ Healthy; $p \leq 0.02$

[§] CAI ≥ Healthy; $p \leq 0.04$

[‖] TrA/IO activation decreased between the pre- and posttraining data collection sessions; $p = 0.003$

[ǀ] CAI ≥ Healthy; $p = 0.02$

[¶] CAI ≥ Healthy; $p \leq 0.00$

[∞] VM activation decreased between the pre- and posttraining data collection sessions; $p = 0.02$

[‼] VM activation decreased between the pre- and posttraining data collection sessions; $p = 0.02$

Table 11. Peak Normalized Muscle Activation[a] (%)

	Transverse Abdominis/Internal Oblique		External Oblique	
	Pre	Post	Pre	Post
Control	675.8 ± 345.3[*]	786.9 ± 1084.4	434.3 ± 277.8[†]	298.8 ± 201.5
CAI	1876.1 ± 2354.4[*‖]	771.6 ± 546.7[‖]	824.3 ± 692.6[†‡]	568.5 ± 1146.0[ǀ]
Healthy	1062.3 ± 993.8	622.6 ± 874.9	312.2 ± 174.4[‡]	368.1 ± 812.5[ǀ]
Effect size[b]	0.074		0.109	

	Gluteus Medius		Vastus Medialis	
	Pre	Post	Pre	Post
Control	215.8 ± 191.4	195.9 ± 164.1	980.9 ± 1315.7	457.9 ± 200.1
CAI	288.6 ± 265.1[§]	217.9 ± 146.3[¶]	1371.7 ± 1510.5[∞]	458.9 ± 254.0[∞]
Healthy	134.9 ± 68.5[§]	106.2 ± 47.1[¶]	1195.9 ± 1162.9[‼]	370.5 ± 300.1[‼]
Effect size[b]	0.153		0.015	

	Peroneus Longus	
	Pre	Post
Control	676.4 ± 1549.9	269.7 ± 239.8
CAI	795.1 ± 1756.1	254.1 ± 142.5
Healthy	432.1 ± 508.1	217.6 ± 123.8
Effect size[b]	0.015	

Abbreviations: %, percent; Post, posttraining data collection; Pre, pretraining data collection

[a] Values are expressed as mean ± standard deviation

[b] Partial eta^2 statistic was used to report effect size

[*] CAI ≥ Control; $p = 0.08$

[†] CAI ≥ Control; $p = 0.02$

[‡] CAI ≥ Healthy; $p \leq 0.00$

[§] CAI ≥ Healthy; $p = 0.04$

[‖] TrA/IO activation decreased between the pre- and posttraining data collection sessions; $p = 0.002$

[ǀ] CAI ≥ Healthy; $p = 0.05$

[¶] CAI ≥ Healthy; $p = 0.01$

[∞] VM activation decreased between the pre- and posttraining data collection sessions; $p = 0.02$

[‼] VM activation decreased between the pre- and posttraining data collection sessions; $p = 0.03$

Table 12. Center of Pressure Excursion[a] (mm) and Velocity[a] (mm/s)

	COPd		COPv Mean		COPv Peak	
	Pre	Post	Pre	Post[¶]	Pre	Post
Control	32.6 ± 20.5	36.9 ± 9.8	70.0 ± 14.7[*]	68.5 ± 13.2	932.4 ± 448.7[‡,§]	1110.3 ± 190.3[∫,⁄]
CAI	26.5 ± 4.6[‖]	31.7 ± 6.8[‖]	65.8 ± 15.3[†,∞]	82.0 ± 11.0[∞]	698.1 ± 120.5[‡,¦]	1059.5 ± 273.9[¦,∫,‾]
Healthy	30.3 ± 6.1[|]	37.6 ± 9.2[|]	50.8 ± 16.6[*,†,‼]	81.3 ± 14.2[‼]	567.9 ± 104.4[§,∩]	794.6 ± 126.2[∩,⁄,‾]
Effect size[b]	0.080		0.076		0.040	

Abbreviations: mm, millimeters; mm/s; millimeters per second; Post, posttraining data collection; Pre, pretraining data collection

[a] Values are expressed as mean ± standard deviation

[b] Partial eta^2 statistic was used to report effect size

[*] Control ≥ Healthy; $p \leq 0.00$

[†] CAI ≥ Healthy; $p = 0.01$

[‡] Control ≥ CAI; $p = 0.02$

[§] Control ≥ Healthy; $p \leq 0.00$

[‖] A trend towards increased COPd existed between the pre- and posttraining data collection sessions; $p = 0.053$

[|] COPd increased between the pre- and posttraining data collection sessions; $p = 0.009$

[¶] An interaction existed between group and time for the mean COPv; $p \leq 0.001$

[∞,‼] Mean COPv increased between the pre- and posttraining data collection sessions; $p \leq 0.001$

[¦] Peak COPv increased between the pre- and posttraining data collection sessions; $p \leq 0.001$

[∩] Peak COPv increased between the pre- and posttraining data collection sessions; $p = 0.004$

[∫] Control ≥ CAI; $p = 0.03$

[⁄] Control ≥ Healthy; $p \leq 0.001$

[‾] CAI ≥ Healthy; $p = 0.002$

Table 13. Data Collection Time Window[a] (ms)

	Time Window	
	Pre	Post[*]
Control	0.39 ± 0.1[†]	0.44 ± 0.2[†]
CAI	0.34 ± 0.1	0.35 ± 0.1
Healthy	0.38 ± 0.1	0.36 ± 0.1
Effect size[b]	0.061	

Abbreviations: ms, milliseconds; Post, posttraining data collection; Pre, pretraining data collection

[a] Values are expressed as mean ± standard deviation

[b] Partial eta^2 statistic was used to report effect size

[*] An interaction between time and group existed on time window; $p = 0.04$

[†] Time window increased between the pre- and posttraining data collection sessions; $p = 0.015$

Table 14. Mean and Peak Vertical Ground Reaction Force Data[a, b] (Newtons)

	Peak VGRF		Mean VGRF	
	Pre	Post	Pre	Post
Control	2216 ± 418.6	2217.5 ± 436.0	1171.8 ± 252.6	1149.6 ± 265.6
CAI	2459.6 ± 430.8[*†]	2286.1 ± 583.3[†]	1275.9 ± 257.7[‡]	1197.8 ± 321.5[‡]
Healthy	2085.9 ± 534.1[*]	2039.7 ± 473.5	1122.0 ± 309.3	1124.2 ± 294.1
Effect size[b]	0.073		0.030	

Abbreviations: Post, posttraining data collection; Pre, pretraining data collection; VGRF, vertical ground reaction force

[a] Values are expressed as mean ± standard deviation

[b] Partial eta^2 statistic was used to report effect size

[*] CAI ≥ Healthy; $p \leq 0.03$

[†] Peak VGRF decreased between the pre- and posttraining data collection sessions; $p = 0.002$

[‡] Mean VGRF decreased between the pre- and posttraining data collection sessions; $p = 0.006$

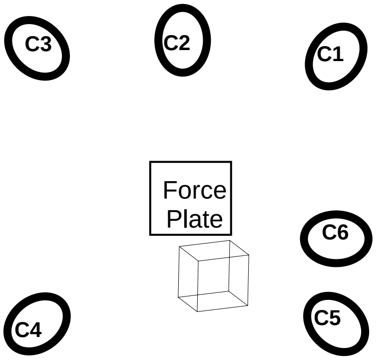

Figure 1. Camera Set-up for Left Leg Dominance
Abbreviations: C1-6, cameras 1-6

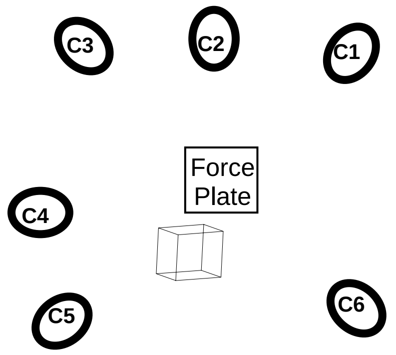

Figure 2. Camera Set-up for Right Leg Dominance
Abbreviations: C1-6, cameras 1-6

Figure 3. Supine Hook-lying Position

Figure 4. Single-leg Drop Landing

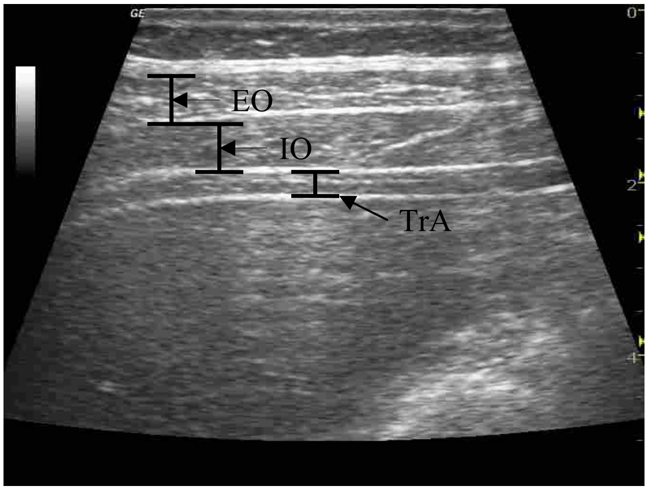

Figure 5. Image of Lateral Abdominal Muscles

Figure 6. Standardized Streak Used Ultrasound Transducer Head Placement

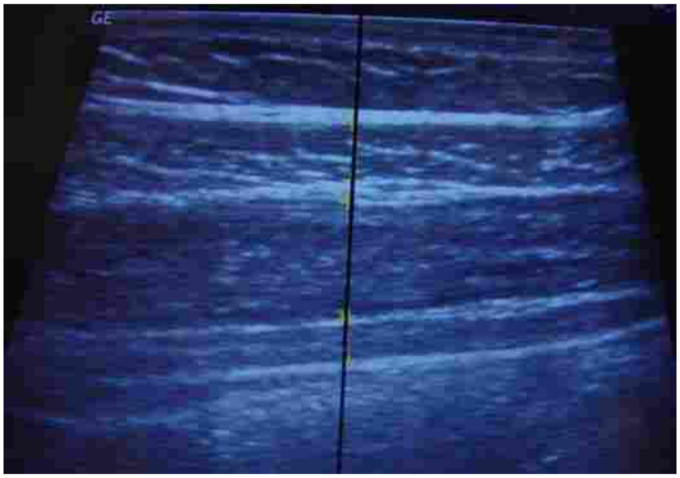

Figure 7. Grid Overlay and Measurement Procedure

Figure 8. Transverse Abdominis / Internal Oblique Reference Position

Figure 9. External Oblique Reference Position

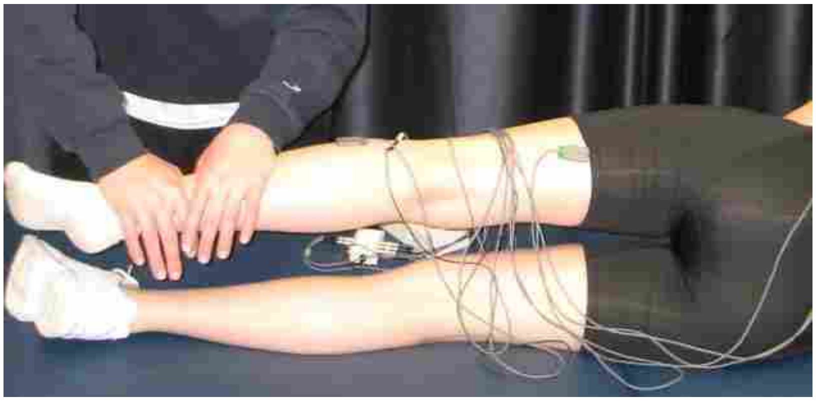

Figure 10. Gluteus Medius Reference Position

Figure 11. Vastus Medialis Reference Position

Figure 12. Peroneus Longus Reference Position

Figure 13. Anterior, Lateral, and Posterior Views of Plug-in Gait Marker Placement

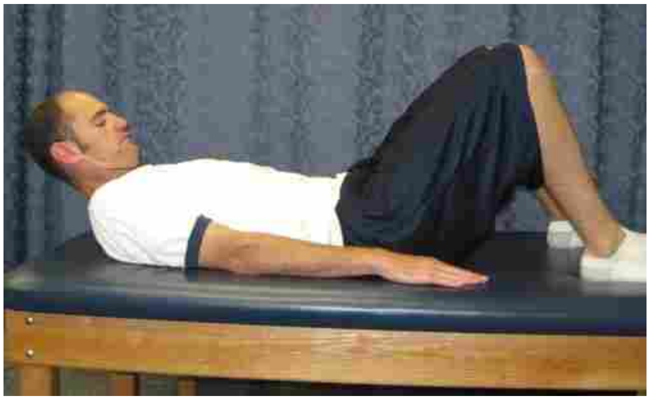

Figure 14. Curl-up Starting Position

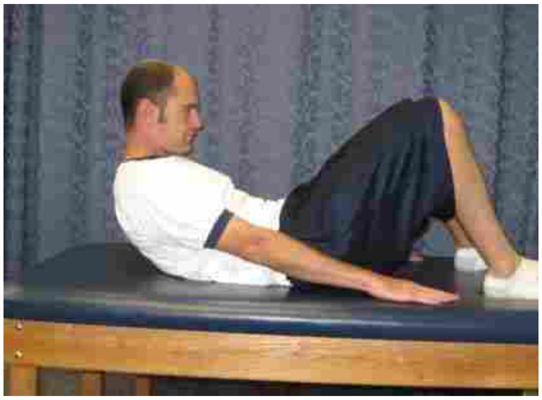

Figure 15. Curl-up Peak Position

Figure 16. Side-Bridge Starting Position

Figure 17. Neutral Spine Position


Figure 18. Sit-up with Rotation Starting Position

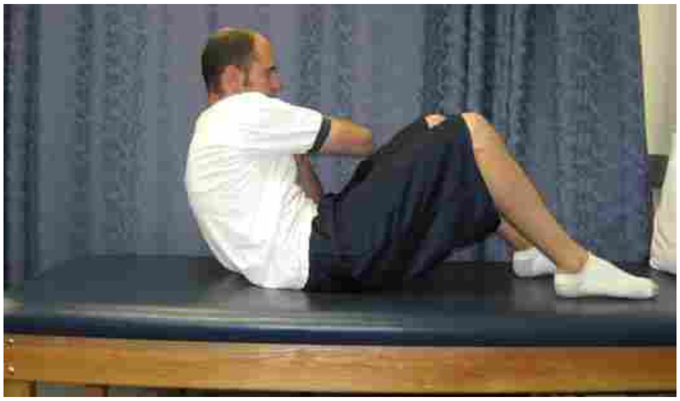

Figure 19. Right Elbow to Left Knee

Figure 20. Return to Starting Position

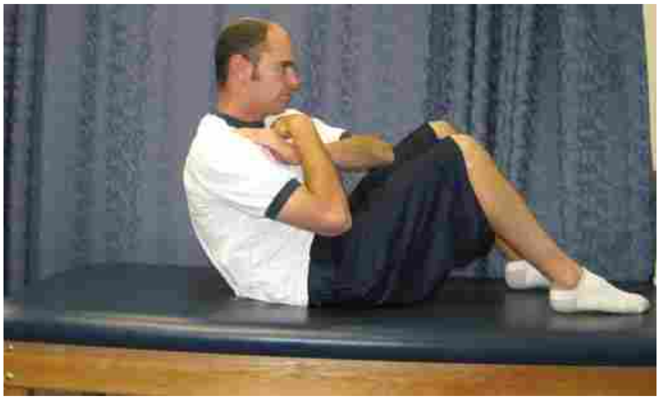

Figure 21. Left Elbow to Right Knee

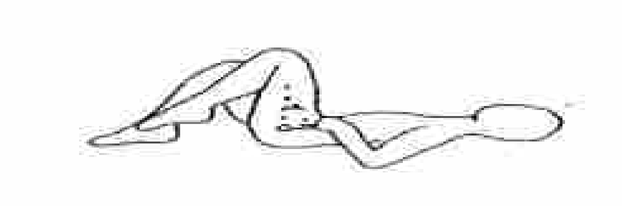

Figure 22. Level 1 Lower Abdominal Series

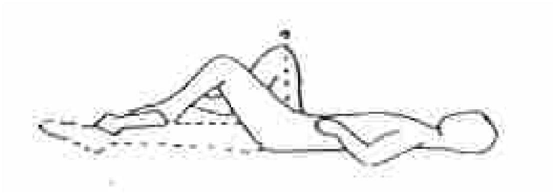

Figure 23. Level 2 Lower Abdominal Series

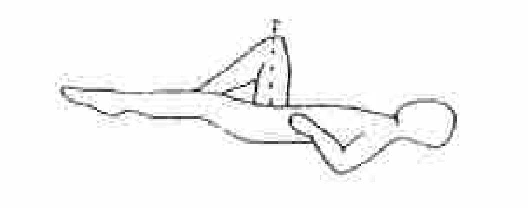

Figure 24. Level 3 Lower Abdominal Series

Figure 25. Level 5 Lower Abdominal Series

Figure 26. Prone-Bridge Starting Position

Figure 27. Prone-Bridge Peak Position

Appendix A

Prospectus

Chapter 1

Introduction

The ankle is the most frequently injured lower extremity joint in the human body.[79, 80] Approximately 85% of all ankle sprains occur to the lateral ligaments.[81] Ankle sprains frequently occur during both activities of daily living and athletic events. Athletes miss more games and practices from an ankle injury than any other injury.[80] It was reported that approximately 30 to 40% of the individuals who sprain their ankle will develop chronic ankle instability.[1, 2]

Three terms commonly used to classify ankle instability are chronic, functional, and mechanical. Chronic ankle instability (CAI) is where individuals suffer from recurrent bouts of lateral ankle sprains.[82] Researchers believe CAI is due to two types of instability dysfunctions.[83, 84] These dysfunctions are known as functional and mechanical ankle instability. Functional ankle instability (FAI) refers to recurrent ankle joint laxity due to neuromuscular deficits.[82] This ankle instability is self-reported through various questionnaires. Functional ankle instability subjects report at least one substantial ankle sprain that occurred greater than one year ago and have “giving way” episodes.[85] Mechanical ankle instability (MAI) is the result of deformities to bony or ligamentous structures of the ankle and/or foot caused by the initial or recurrent injury(s).[82, 86] Some patients with ankle instability have signs and/or symptoms of both FAI and MAI.[84, 87] The majority of ankle instability research has centered on the implications of FAI because without surgical intervention it is believed little can be done to change MAI.[86] Although FAI has been studied for years, its causes and effects remain unclear.

Many factors are believed to contribute to FAI. Some of the most common are decreased proprioception,[2, 5, 6] joint position sense,[6-9] strength,[9-14] and increased peroneal muscle latency.[26, 27] Individuals with FAI have also demonstrated decreased coordination,[2, 8] balance,[15, 16] and postural control.[17-25] Although these risk factors have been demonstrated by FAI subjects, researchers have also observed no deficits.[5, 9-12, 88-97] All of these variables are thought to make FAI patients prone to CAI. However, a consensus regarding the importance of these risk factors or how they may interact with each other has not been reached. Due to the complexity of these risk factors, researchers have begun to study the interrelationship between proximal joints and muscles and FAI. Both how FAI might effect the proximal joints and muscles and how FAI subjects may use these muscles to compensate for ankle instability during dynamic movements.[28-33]

Functional ankle instability subjects have demonstrated compensatory landing strategies compared to healthy control subjects.[18, 70, 71] They land in a more erect or stiff-legged position than healthy subjects.[70] This is referred to as an ankle-landing strategy because it requires the ankle joint and surrounding muscles to absorb greater energy than the knee and hip. Therefore, FAI subjects may place greater demands on the injured ankle joint and surrounding muscles in an attempt to maintain balance or postural control. Healthy subjects use a combination of landing strategies (ankle, hip, and/or combined),[69] which allows energy to be transferred up the kinetic chain for energy absorption.[69, 98] As energy is transferred up the kinetic chain, the knee and hip joints along with their surrounding muscles reduce the amount of energy absorbed by the ankle. This serves to take some of the stress off the ankle and surrounding muscles.[69, 99-102] How the compensatory landing strategy of FAI subjects affects their proximal muscle activation is unclear.

Altered proximal muscle activation and strength deficits were observed in subjects with CAI. Subjects with FAI have demonstrated altered gluteus medius (GMed),[28] gluteus maximus (GMax),[29, 31] and biceps femoris (BF)[31] muscle activation. Arthrogenic muscle inhibition of the quadriceps and hamstrings was also observed in CAI subjects. This may explain why proximal strength deficits exist in CAI subjects.[34] A correlation between ankle/foot injuries and ipsilateral hip abductor and adductor muscle weakness exists.[103] Subjects with a history of a lower extremity injury or low back pain have exhibited decreased hip extensor strength.[104] Gribble et al[33] observed CAI and fatigue may disrupt postural control during a dynamic task, this was most notable in joints proximal to the ankle.[33] Hip abductor and extensor weakness in subjects with a lower extremity injury required greater recruitment of trunk stabilizer muscles during dynamic tasks.[41] The involvement of proximal joints and muscles superior to the pelvis is unknown, thus further research is required.

It was theorized that abdominal muscle strength may play a role in reducing the risk of lower extremity injuries.[105] Few research studies however, have assessed the relationship between core muscles and lower extremity injury. Trunk muscle involvement increased with the presence of GMed weakness.[41] Healthy subjects increased abdominal muscle activation as the difficulty of static and dynamic tasks increased.[35] Hodges and Richardson[106, 107] observed that the transversus abdominis (TrA) muscle activated prior to any lower or upper extremity limb movement. These studies suggest a feedforward mechanism may be utilized to activate the TrA prior to movement. Gluteus maximus muscle activation increased when abdominal hollowing (AH) was performed during a prone straight leg raise; however, this study did not assess GMed muscle activation.[74] Abdominal hollowing is a strengthening exercise that isolates the contraction of the TrA muscle.[55] Currently however, it is unknown how abdominal muscle activation is affected by a lower extremity injury such as ankle instability.

The purpose(s) of our study are to first determine if muscle activation, center of pressure, and kinematics differ between FAI and healthy subjects during a single-leg drop landing. These variables will then be measured following an eight-week abdominal strengthening program undertaken by healthy and FAI subjects to determine what effect abdominal strengthening has on muscle activation, center of pressure (COP) excursion, and kinematics. A better understanding of how abdominal and lower extremity muscles interact during single-leg drop landing may aid in finding a possible contributor to ankle instability and could change the way clinicians rehabilitate FAI patients.

RESEARCH QUESTIONS

1. Does COP excursion, muscle activation, muscle thickness, and kinematics differ between healthy and FAI subjects during a single-leg drop landing?
2. Does an eight week abdominal strengthening program improve postural control in healthy and FAI subjects?

3. Does abdominal strengthening increase peak and mean electromyography (EMG) amplitude of lateral abdominal (internal oblique /TrA, external oblique) and lower extremity muscles (GMed and BF) during a single-leg drop landing?
4. Do morphological changes individually occur to the external oblique (EO), internal oblique (IO), and TrA during an eight week abdominal strengthening program?
5. Does abdominal strengthening change ankle, knee, or hip joint angles (frontal and sagittal planes) during a single leg drop landing?

RESEARCH HYPOTHESES

1. Functional ankle instability subjects will demonstrate deficits in COP excursion, muscle activation, and kinematics compared to healthy controls preabdominal strengthening.
2. An eight week abdominal strengthening program will decrease COP excursion, increase muscle activation and kinematics in healthy and functional ankle instability subjects.
3. Abdominal muscle strengthening will increase mean and peak EMG amplitude of the IO/TrA and EO muscles, GMed, and BF muscles during a single-leg drop landing
4. The thickness of the EO, IO, and TrA at rest and during AH will increase during an eight-week strengthening program.

5. Abdominal strengthening will decrease hip adduction, and increase knee flexion and dorsiflexion in FAI subjects during a single-leg drop landing.
6. A positive correlation will exist between abdominal muscle thickness during AH and muscle activation during a single-leg drop landing.

OPERATIONAL DEFINITIONS

Abdominal hollowing – subjects gently pull their umbilicus towards the plinth or floor without moving their spine and maintaining normal breathing while lying supine.[55]

Arthrogenic muscle inhibition – the presynaptic ongoing reflex inhibition of musculature surrounding a joint following distention or damage to structures of that joint.[108]

Balance – ability to maintain equilibrium by controlling the center of gravity over its base of support.[109]

Center of pressure – assesses body equilibrium and postural control.

Chronic ankle instability – condition where individuals suffer from recurrent bouts of lateral ankle sprains.[36]

Coordination – ability of muscles and muscle groups to perform complicated movements.[109]

Core – includes the hip, pelvis, and lumbar spine along with any muscles or soft tissue that inserts or originates on these bony structures.

Correct abdominal hollowing – a significant increase in TrA thickness, with minimal or no increase in EO and IO thicknesses.

Dominant limb – the limb an individual would plant with while kicking a ball.[58]

Electromyography – an instrument used to assess the electrical activity of muscle tissue.[110]

Energy absorption – amount of work absorbed by the joints and muscles.

Force sense – ability to reproduce or detect a given amount of force.[89, 111]

Functional ankle instability – condition referring to recurrent ankle joint instability due to neuromuscular deficits.

Ground reaction force – the sum of all forces applied to a surface (ie., foot).[110]

Healthy – No abdominal, low back, or lower extremity injury in the past year or surgery in the past two years.

Initial contact – the first contact between the foot and ground or force plate.

Joint position sense – posture of a segment and joint in space.[112]

Kinematics – the study of motion without regards to what caused the motion.[110]

Kinesthesia – ability to detect movement.[89]

Kinetics – study of forces such as work, energy, impulse, momentum, and power acting on human body.[110]

Lateral abdominal muscles – the EO, IO, and TrA, commonly referred to as the lateral abdominal wall muscles.

Mechanical ankle instability – condition caused by deformities to bony or ligamentous structures of the ankle and/or foot.[82, 86]

Muscle thickness – the perpendicular distance between the superficial and deep fascial lines of the muscles; thickness measurements will not include the fascial boundary.

Postural control – the ability to control the position of the body in space for the dual purposes of stability and orientation.[75]

Proprioception – the ability to provide feedback to the nervous system through joint position sense, kinesthesia, sense of resistance (force).[112]

Physically active – participate in 30 minutes of aerobic activity a minimum of 3 times per week.

Regular participation – performing abdominal strengthening exercises three times per week.

Single-leg drop landing – landing on the dominant leg from a 35 cm platform onto the force plate while the nondominant limb is nonweight bearing.

X-axis – measures forces in the medial-lateral direction.[110]

Y-axis – measures forces in the anterior-posterior direction.[110]

Z-axis- measures forces in the vertical direction.[110]

ASSUMPTIONS

This study is based on the following assumptions:

1. Center of pressure, muscle activation, and kinematics will be different between FAI and healthy subjects.

2. Subjects will honestly answer the foot and ankle ability measure (FAAM) questionnaire.
3. Subjects will complete all of the abdominal strengthening exercise protocol three times a week and will provide honest responses on the weekly exercise log.
4. Surface EMG electrodes placed over the IO/TrA represent muscle activation of both the IO and TrA.
5. Center of pressure is an accurate indirect measure of balance and postural control.
6. Reflective markers will be accurately placed over anatomical landmarks.
7. Eight weeks of abdominal strengthening is enough time to observe change in abdominal muscle thickness at rest and during AH.

DELIMITATIONS

1. All subjects will be physically active.
2. All subjects will be free from any neurological or cardiovascular disorders.
3. No history of pregnancy or childbirth in the past two years.
4. Subjects will self-report CAI using the AII questionnaire.
5. Subjects will self-report FAI using the FAAM questionnaire.
6. Subjects other than the FAI group will have no history of an abdomen, low back, or lower extremity injury in the past year.
7. Subjects will have no history of a surgery to the abdomen, low back, or lower extremity in the past two years.

LIMITATIONS

1. Center of pressure excursion is the only measure we are using to represent balance and postural control.
2. Surface EMG is an estimation of the muscle activation of muscle fibers only underneath the electrode.
3. Individual muscle activation of IO and TrA can not be determined.
4. The abdominal strengthening exercises may strengthen more than just the abdominal muscles (eg. erector spinae, multifidi, hip flexors, gluteal, quadriceps, hamstrings muscles) which may contribute to the findings.

Chapter 2

Review of Literature

Ankle instability and sprains continue to frequently occur despite the knowledge gained from extensive research. Numerous contributing factors have been associated with chronic ankle instability (CAI). The majority of functional ankle instability (FAI) studies have only assessed the involvement of the lower leg musculature. Researchers recently began to assess the involvement of proximal joints and musculature in FAI subjects. This research indicates that proximal musculature may have an important role in both preventing and rehabilitating FAI.

The relationship between hip musculature and ankle instability has been attributed to somateosensory deficits and/or spinal, and supraspinal input.[36] It remains unclear how a distal injury may affect the feedback and feedforward mechanisms. Assuming the feedforward mechanism is the primary connection between proximal musculature and distal injuries, there is a need for further research to assess the role of other proximal "core" muscles.

The relationship between ankle instability and lateral abdominal muscles has not been assessed. In athletic events the upper body is required to move in multiple directions and planes. To maintain postural control during dynamic movements the spine must be stabilized, otherwise postural control may be diminished; therefore, increasing the risk of injury. Since the lateral abdominal muscles assist in providing spinal stabilization during dynamic movements it is logical to assess the relationship between the lateral abdominal muscles and ankle instability. This study will assess the connection between this muscle group and FAI.

The outline below identifies the topics contained in this literature review.

DATABASES AND KEYWORDS SEARCHED
ANKLE INJURY STATISTICS
ANKLE INSTABILITY
Mechanical Ankle Instability
Functional Ankle Instability
CONTRIBUTORS OF FUNCTIONAL ANKLE INSTABILITY
Proprioception
    Kinesthesia.
    Joint Position Sense.
    Sense of Resistance.
Motorneuron Pool Excitability
    Arthrogenic Muscle Inhibition.
Lower Extremity Muscle Activation
    Peroneal Muscle Group Latency.
    Altered Electromyography.
Lower Extremity Strength Deficits
    Eversion Strength.
    Inversion Strength.
    Plantar Flexion Strength.
Postural Control
    Sway.
    Center Of Pressure.
    Time to Boundary.
    Time to Stabilization.
    Dynamic Postural Stability Index.
    Equitest.
    Balance Error Scoring System.
    Star Excursion Balance Test.
    Single-Leg Balance Test.
Biomechanics
    Landing Types.
    Landing Strategies.
    Energy Absorption During Landing.
    Perturbations.
    Muscle Activation.
    Ankle Instability and Landing.
    Gender Differences.

PROXIMAL ANATOMICAL STRUCTURES AND LOWER EXTREMITY INJURY
Hip Musculature
  Ankle Instability.
  Abdominal Musculature.
CENTRAL NERVOUS SYSTEM
Brain
Spinal Cord
Feedback
Feedforward
CONTEMPORARY THEORY: ANKLE INSTABILITY & SOMATOSENSORY DEFICITS
CORE STABILITY
Musculature
REHABILITATION PROGRAMS
Ankle Rehabilitation
Abdominal Strengthening Exercises
INSTRUMENTATION
Electromyography
Force Plate
Kinematics
Ultrasound Imaging

DATABASES AND KEYWORDS SEARCHED

CINAHL, MEDLINE, Pre-CINAHL, and SPORTDiscus databases were searched from 1965 - present. Additional literature was gathered from citations in articles, textbooks, and symposiums. Keywords searched include the following terms:

Abdominal muscles
Abdominal strengthening
Ankle
Ankle instability
Arthrogenic muscle inhibition
Balance
Center of pressure
Chronic ankle instability
Coordination
Core stability
Core strengthening
Electromyography (EMG)
Energy absorption
External oblique
Feedback
Feedforward
Force sense
Functional ankle instability
Gluteus maximus
Gluteus medius
Hip musculature
Internal oblique
Joint position sense
Landing strategies
Mechanical ankle instability
Muscle activation
Peroneal
Postural control
Strength
Training
Transverse abdominis
Ultrasound imaging

ANKLE INJURY STATISTICS

Approximately 50% of all sports related injuries occur to the lower extremity.[113] The ankle is the most commonly injured joint in the lower extremity[114] and human body.[81, 113-115] Epidemiological research assessed the frequency of ankle injuries in both collegiate[113] and high school athletes,[79, 80, 114] along with the general public.[115, 116] The rate of ankle injuries vary depending on gender, type of participation (game or practice), and sport. Ankle injuries are most often associated with sports requiring a combination of jumping and change of direction during running.[80] Males injured their ankles more in football[113, 114] and basketball[80, 113, 115] than any other sports. It was reported that 45% of ankle injuries in basketball occur during landing.[116] Soccer and volleyball were the sports that reported the greatest incident of ankle injuries for females.[114, 115] Typically more ankle injuries occur during competition than practice.[80] Over 50% of those who sprain their ankle do not seek professional treatment.[116]

Epidemiological studies have identified some other contributing factors associated with ankle injuries. These factors include history of injury,[116] being overweight,[117] wearing air celled shoes,[116] not stretching prior to participation,[116] FAI,[1, 2] and mechanical ankle instability (MAI).[86] A history of an ankle sprain was the strongest predictor of an ankle injury, individuals with a previous ankle injury were five times more likely to reinjure their ankle.[116] An ankle injury was 4.3 times greater when air celled shoes were worn.[116] Failing to stretch prior to exercise made individuals 2.6 times more likely to suffer an ankle injury.[116] Although some contributing factors have been identified, controversy remains regarding the importance of these factors.[118] Table 1 summarizes the epidemiological studies discussed in this section.

Table 1. Summary of Epidemiological Studies

Authors/Year	Instrument(s)	Results	Conclusion
Fernandez et al, 2007[114]	1-year HS Epidemiology	52.8% of injuries were to the LE F(x) most common in ankle (41.8%) Ankle (40%) was the most common LE joint injured	LE injuries commonly occur in HS athletes Gender & team-specific patterns exist FB had the highest rate of LE injury among boys Soccer highest rate for girls
Freeman, 1965[1]	1-year Epidemiology	AP instability & adhesion formation are not associated with FAI No subtalar motion, calf weakness, or tib-fib injury ↑ observed	Subjects demonstrated MAI FAI is not initiated by MAI or adhesion formation Cause of FAI unknown
Freeman et al, 1965[2]	Epidemiology	Proprioceptive deficits in 25% of patients Coordination exercises ↓ risk of FAI initially & post Coordination group had lower incidence of FAI & proprioception deficit	Proprioceptive deficits demonstrated immediately after injury Deficits should persist in FAI patients FAI & deficits may decrease w/ motor coordination rehab Coordination rehab virtually eliminates FAI symptoms & deficits Deficits are associated with FAI
Garrick, 1977[81]	Epidemiology		85% of all sprains are lateral Lateral ligaments are most commonly injured body part
Garrick & Requa, 1988[115]	Epidemiology	Ankle & foot injuries comprise 25.2% of injuries Basketball had highest incident of ankle injuries	Foot/ankle are the most commonly injured body parts VB had the greatest # of ankle sprains
Hootman et al, 2007[113]	Epidemiology	More than 50% of all injuries were to the LE in games & practices Spring FB & MBB had the highest rates of ankle sprains	More attention should be given to injury prevention research that is applicable to all types of LE injuries Ankle sprains were the most common injury (15%)
McHugh et al, 2006[117]	1-leg stance on tilt board Handheld dynamometer	Females had better balance Females sprain their ankles more No balance, hip abductor, adductor, or flexion strength differences between FAI & control subjects	Balance and hip strength were not significant indicators for noncontact ankle sprains Previous injury & overweight ↑ risk Hip weakness in FAI subjects is likely a consequence of injury & not causative factor
McKay et al, 2000[116]	Questionnaire Recreational basketball players	45% of ankle injuries occurred during landing 56.8% did not seek professional treatment 73% reported previous ankle injury	History of ankle injuries was the strongest predictor CAI subjects are 5x likely to reinjure Air cell shoes are 4.3x more likely to injure their ankle Not stretching made players 2.6x likely to become injured

Nelson et al, 2007[80]	1-year Epidemiology	Ankle injuries cause athletes miss ≤ 7 days (51.7%), 7 to 21 days (33.9%), ≥ 22 days (10.5%)	Ankle injuries occur more in competition than practice Boys' basketball had the highest rate of ankle injury Sports that combine jumping & swift Δof direction while running were most often associated with ankle injuries

ANKLE INSTABILITY

Mechanical Ankle instability

Little research has been done to assess how mechanical deficits following an ankle injury contribute to MAI. Research has identified some of the factors that may contribute to MAI.[86] Pathological laxity, arthrokinematic restrictions, degenerative changes, and synovial changes are some of the contributing factors that may cause MAI.[82, 86] Hypermobility and hypomobility are the result of these mechanical changes following injury.[86] The talocrural, inferior tibiofibular, and subtalar joints are common sites for these mechanical changes.[82, 86] Additional research discussed the relationship between FAI and MAI.[82, 84, 119] Some CAI subjects have demonstrated symptoms of both FAI and MAI, this showed that FAI and MAI are not exclusive.[84, 119] This makes it difficult to identify the type of instability affecting a patient. Typically MAI is identified through clinical exams and other diagnostic tests (ie., stress radiography, magnetic resonance imaging).[86] Mechanical ankle instability is initially treated with conservative rehabilitation.[86] Rehabilitation includes providing external support[120] (ie., taping or bracing) to the ankle joint and functional rehabilitation.[86] However, if symptoms persist in MAI patients, a surgical intervention may need to be done to repair the mechanical deformities[86]; although, researchers observed that MAI patients have less laxity than FAI patients.[83] Therefore, the relationship between FAI and MAI remains unclear and further research is needed to determine what role MAI has in FAI patients.[83] Since both mechanical and functional deficits contribute to CAI,[119] further research also needs to determine what role FAI has in MAI patients.

Functional ankle instability

Functional ankle instability has been described as sensorimotor or neuromuscular deficits that coincide with recurrent ankle sprains.[36, 82] Freeman and colleagues[1-3] noticed ligamentous injuries to the ankle and foot often led to proprioceptive deficits. Joint instability was believed to be the result of proprioceptive deficits. Since the 1960s, extensive research has been completed on FAI. The majority of the research has assessed possible contributing factors associated with FAI. There are a number of factors associated with FAI which adds to the complexity of this multifaceted dysfunction.

CONTRIBUTORS OF FUNCTIONAL ANKLE INSTABILITY

Some of the factors associated with FAI include but are not limited to deficits in proprioception (force sense,[89, 111, 121] joint position sense,[6, 9, 122-127] kinesthesia,[125, 128, 129]), motorneuron pool excitability,[34, 130] sensory changes,[30] muscle activation,[26, 27, 29, 31, 131-133] strength,[13, 14, 124, 134, 135] and postural control.[6, 15, 17, 19, 21-25, 33, 128, 136, 137] Functional ankle instability subjects have demonstrated the use of a compensatory landing strategy. This includes greater hip adduction than healthy control and the use of primarily an ankle landing strategy. The compensatory strategies are thought to be an attempt to maintain postural control. Some believe FAI subjects may use the proximal muscles and joints to reduce the stress placed on the ankle and risk of CAI.[67, 68, 72, 138, 139] All of the contributors of FAI mentioned above will be discussed in greater detail below.

Proprioception

Proprioception is frequently used incorrectly to refer to kinesthesia, joint position sense, balance, and other sensorimotor system terms.[112] Riemann & Lephart,[112] stated there are three aspects to conscious proprioceptive senses: kinesthesia, joint position sense (JPS), and sense of resistance or force. In an attempt to eliminate confusion regarding proprioception, these three areas will be discussed separately.

Kinesthesia. Diminished kinesthesia was observed in subjects with an acute ankle sprain[90] and CAI.[9, 128, 129, 140, 141] Subjects rated their perceived kinesthesia deficits, while blinded observers also rated their deficits.[128, 129] Forkin et al[128] and Garn and Newton[129] used a kinesthesiometer to assess kinesthesia and observed that the uninjured limb detected passive plantar-flexion better than the injured limb. No difference was observed when a motorized kinesthesiometer was used to measure perceived passive plantar-flexion and dorsiflexion between CAI and healthy subjects.[88] As velocity increased, perceived displacements became smaller.[88] No difference was observed in detection of passive plantar-flexion and dorsiflexion at different velocities when the ankle was taped, therefore, ankle bracing and taping do not enhance kinesthesia.[88, 141] The ability to detect passive inversion and eversion motion was less following an acute ankle sprain and in CAI subjects when compared to healthy controls.[9, 90, 140] Detection errors measured one week post-acute ankle sprain were greater than errors at 6 and 12 weeks post-injury.[90] This demonstrated that detection errors decreased over time, although the injured limb made more errors at 12 weeks post-acute ankle sprain than the control group.[90] It can be concluded that kinesthesia deficits may contribute to FAI. Table 2 summarizes the kinesthesia studies discussed in this section.

Table 2. Summary of Kinesthesia Research Studies

Author, Year	Instrument(s)	Results	Conclusion
Forkin et al, 1996[128]	Kinesthesiometer	Subjects detected passive plantar-flexion uninjured (158/165) ankle movement > than injured (143/165)	Balance & kinesthetic deficits are common in gymnasts with MAS
Garn & Newton, 1988[129]	Kinesthesiometer	The injured limb had ↓ passive plantar-flexion kinesthesia	Kinesthetic deficits exist in the injured ankle joints of athletes with a history of MAS
Jerosch et al, 1995[141]	1-leg jumping course 1-leg stance test Angle reproduction	Difference between injured & healthy ankle joints 1-jumping errors Difference between injured & healthy ankle joints 1-leg stance Reproduction errors > in injured ankle	Ankle braces reduced 1-leg jumping errors Taping makes no difference Difference between injured & uninjured ankle joints for all 3 tests
Konradsen et al, 1998[90]	Inversion angle position	Injured limb > angle error than uninjured Injured angle errors > @ 1wk than @ 6 & 12 wks	Acute ankle injuries result in Δ of inversion ankle position Injured limb has > error @ 12 wks compared to healthy limb
Lentell et al, 1995[9]	Passive movement sense	> passive inversion movement sense in involved ankles ($p \leq .05$)	> passive inversion movement sense & talar tilt present in the involved limb
Refshauge et al, 2003[140]	Kinesthesiometer with a motor	70% passive inversion & eversion detection ↑ with velocity CAI group had worse detection than controls	Ability to detect inversion & eversion is impaired in CAI subjects
Refshauge et al, 2000[88]	Kinesthesiometer with a motor	No difference in kinesthesia between (healthy & CAI) groups & velocity No difference in kinesthesia between taped & untapped groups & velocity	Smaller displacements perceived as ↑ in velocity Ability to detect passive plantar-flexion & dorsiflexion is not impaired in CAI subjects Ankle taping did not enhance kinesthesia

Joint position sense. Many researchers have assessed JPS in CAI subjects. Deficits in JPS was determined to be a possible predictor of ankle joint injuries.[142] Subjects with CAI demonstrated an increase in ankle repositioning errors for inversion[7, 122, 123, 125, 126, 141] and plantar flexion.[6] The greatest repositioning errors occurred near the beginning and end of inversion range of motion.[122, 123, 127] A positive relationship` existed between severity of injury and JPS deficits.[127] Both active and passive JPS deficits exist in CAI subjects.[122]

A positive relationship existed between plantar-flexion repositioning errors and postural sway in subjects with multiple ankle sprains, both plantar-flexion repositioning errors increased as postural sway increased.[6] Subjects with CAI demonstrated an increase in knee joint angle repositioning errors when compared to a healthy control group.[123] This suggested a distal injury such as CAI may alter proximal joint(s) and muscle function.

Others applied interventions to CAI subjects to determine if JPS errors could be decreased. Konradsen and Magnusson[125] observed that a ten-minute warm-up run on a treadmill reduced ankle replication errors. Ankle taping and a variety of ankle braces were applied to subjects to determine if an external support device could improve JPS during ankle angle reproduction, single leg stance test, and single leg stance position test. Ankle taping did not improve JPS; however, ankle braces may improve JPS.[141] A ten-week elastic-tubing strengthening program increased strength and decreased inversion, dorsiflexion, and plantar-flexion JPS.[124]

A study by Docherty et al[89] contradicts the relationship between ankle instability and JPS. Docherty et al,[89] reported no relationship between FAI and both active inversion and eversion JPS. However, this study did demonstrate a positive correlation between FAI and force sense.[89] Table 3 summarizes the joint position sense studies discussed in this section.

Table 3. Summary of Joint Position Sense Research Studies

Author, Year	Instrument(s)	Results	Conclusion
Boyle & Negus, 1998[122]	Active & passive JPS (pedal goniometer)	No difference for uninjured group (passive) 30% and 90% positions (active) were different > error @ 30% active JPS Difference between groups' passively in all positions Difference between groups' active JPS @ 30%	Healthy subjects no difference in active or passive JPS Active & passive JPS deficits exist in CAI subjects
Docherty et al, 2006[89]	Force Sense Active JPS	No relationship between inversion or eversion active JPS & FAI Force sense positively correlated with FAI	Low load force sense deficits are present in FAI subjects
Docherty et al, 1998[124]	Electric goniometer Handheld dynamometer	Training group ↑ strength & JPS No eversion JPS effect but there was a dorsiflexion JPS effect	Ankle strengthening improves inversion & plantar flexion JPS FAI subjects had ↑ strength, inversion, dorsiflexion, and plantar flexion JPS
Fu & Hui-Chan, 2005[6]	Passive ankle repositioning	↑ in ankle repositioning errors in subjects with bilateral MAS	Ankle repositioning & postural sway in stance ↑ with MAS + relationship between repositioning & postural sway
Glencross & Thorton, 1981[127]	Goniometer	Difference in MES between injured vs. healthy limbs ($p<.01$) Difference in MES between joint angles	> error @ largest angles of movement Error > for most severely injured group
Jerosch et al, 1995[141]	1-leg jump course 1-leg stance position test Angle reproduction	Ankle braces ↓ 1-leg jumping & stance errors Difference between injured & healthy ankle joints During 1-leg jumping & stance errors Reproduction errors > in injured ankle	Difference between injured & uninjured ankle joints for all 3 tests Ankle taping makes no difference Ankle braces may improve JPS
Konradsen & Magnusson, 2000[125]	Electronic torsion Goniometer	Absolute inversion error > on affected side compared to uninvolved limb & healthy group Difference in absolute error before & after warm-up	No difference between sides Injured limb had > inversion replication errors Warming up ↓ error by 38%
Konradsen, 2002[8]	Literature review	Δ in JPS & kinesthesia found in CAI may ↑ injury risk	Balance & coordination training can restore the ↑ uncertainty of joint positioning to normal levels
Nakasa et al, 2008[126]	Goniometer footplate	Difference in inversion angle replication error between ankles of the unstable group & healthy group	FAI had JPS deficits compared to healthy volunteers
Payne et al, 1997[142]	ROM JPS-Biodex Strength-Biodex	8 out of 42 basketball players sprained an ankle during a season Proprioception was a predictor of ankle injury Strength & ROM failed as injury predictors	Ankle joint proprioceptive deficits can be used to predict ankle injuries in females Male & female basketball players have similar injury rates

Tsiganos et al, 2008[123]	JPS (isokinetics)	CAI subjects have > error than healthy control group (30° $p< .001$, 45° $p< .023$, & 70° $p <.05$) CAI dominant limb > knee angle error than control dominant CAI dominant & non-dominant ↓ error from 30° to 45° to 70° knee angle	Integrating proximal joint assessment to a distal injury may improve rehabilitation of CAI
Willems et al, 2002[7]	Active & passive JPS (Biodex)	Instability group was less accurate during active position sense near maximal inversion	CAI group underestimated the reference angle CAI may have inappropriate foot positioning CAI due to diminished JPS

Sense of resistance. Sense of resistance or force sense is reportedly one of the earliest methods used to assess proprioception.[89, 111] It is comprised of two different senses: effort and tension.[111] The effort sense is primarily a central mechanism with the influence of the peripheral system.[143] Tension placed on the muscle is physiologically monitored by the peripheral system especially the Golgi tendon organs.[143]

Few research studies have monitored force sense in an injured population. Functional ankle instability subjects have demonstrated force sense deficits.[89, 111, 121] A relationship between force sense and muscle stiffness was observed, especially between the involved limb stiffness and contralateral reproduction.[144] The relationship between low-load eversion force sense errors and both the number of "giving way" episodes and ankle instability index was assessed, a positive correlation was observed between FAI and force sense errors.[111] Greater eversion force sense errors were observed in FAI subjects.[89, 121] These studies demonstrated that FAI subjects had difficulty replicating eversion forces. Currently there is a clear consensus that FAI subjects have difficulty detecting force sense. Therefore, force sense deficits are thought to impair joint stability, although further research needs to be done to confirm this theory.[121] Table 4 summarizes the sense of resistance studies discussed in this section.

Table 4. Summary of Sense of Resistance Research Studies

Author, Year	Instrument(s)	Results	Conclusion
Arnold & Docherty, 2006[111]	Force Sense AII	Eversion force sense errors were + correlated with # of giving way episodes & AII (ipsilateral)	FAI subjects struggled to replicate eversion forces Larger errors were related to giving way & perceived ankle instability
Docherty et al, 2006[89]	Force Sense Active JPS	No relationship between inversion or eversion active JPS & FAI Force sense positively correlated with FAI	Low load force sense deficits are present in FAI subjects
Docherty & Arnold, 2006[121]	Force Sense	Absolute & variable errors different between groups FAI subjects had > absolute & variable force sense errors	FAI is associated with deficits to reproduce a given force This deficit may impair an individual's ability to provide joint stability
Docherty et al, 2004[144]	Force Sense JPS Stiffness	No correlation between force sense & JPS or JPS & stiffness Contralateral reproduction correlated w/ involved limb stiffness	Force sense is correlated with involved limb stiffness
McCloskey et al, 1974[143]	Force Sense	Difficult for subjects to match weights, ↑ with movement Difficult for subjects to match tension	Sense of effort exists & is separate from peripheral sense of tension It is unknown what peripheral receptors are responsible for conscious appreciation of tension

Motorneuron Pool Excitability and Sensory Change

Arthrogenic Muscle Inhibition. Arthrogenic muscle inhibition (AMI) is a limiting factor in joint injury rehabilitation.[108] Joint injury, immobilization, pain, muscle atrophy and weakness are some of the factors which influence or are effected by AMI.[108] Muscle inhibition decreases the number of motor units available to perform a muscle contraction, which increases the risk of CAI and a prolonged rehabilitation.[108]

The Hoffmann reflex (H-reflex), H_{max}:M_{max} ratio, and central activation ratio (CAR) are common techniques used to measure motorneuron (MN) availability. The H-reflex measures α-MN recruitment following an electrical stimulation of a peripheral nerve.[145] The H:M_{max} ratio normalizes the maximal H-reflex to maximum M-response. This ratio indicates the number of MNs capable of being activated.[130] Central activation ratio is the ratio between a maximal voluntary contraction (MVC) and a MVC with an external electrical superimposed burst applied.[34] A small CAR value indicates greater AMI.

Research studies have assessed the relationship between AMI and FAI. McVey et al[130] measured the H-reflex and M-response of the anterior tibialis, peroneals, and soleus bilaterally in FAI and healthy subjects. Functional ankle instability subjects demonstrated smaller peroneal and soleus H:M_{max} ratios on the injured side compared to the healthy side.[130] No side-to-side differences were observed in the healthy subjects H:M_{max} ratios.[130] Sedory et al[34] assessed the arthrogenic muscle response of the hamstrings and quadriceps in healthy and CAI subjects using CAR. Chronic ankle instability subjects exhibited quadriceps facilitation in the injured limb compared to the uninjured limb and healthy group.[34] However, the hamstrings CAR value was smaller for the CAI group compared to the control group.[34] These studies provide data to support the theory that AMI is present in FAI subjects.[34, 130] A distal joint injury, such as FAI, may alter the response of proximal muscles during movement. Arthrogenic muscle inhibition of the lower leg and thigh musculature may be due to FAI. The role proximal muscles have in FAI subjects needs to be researched further.

Functional ankle instability subjects also demonstrated difficulty in detecting sensory changes. Bullock-Saxton[30] used two-point discrimination and vibration to assess sensory changes in FAI subjects compared to a healthy control group. Two-point discrimination and vibration threshold perception were not identified as well by FAI subjects.[30] Functional ankle instability subjects took longer to identify two-point discrimination and vibration had to be increased to reach vibration perception threshold.[30]

Arthrogenic muscle inhibition was present in the lower leg and further up the kinetic chain in FAI subjects. The combination of AMI and the difficulty to identify sensory changes suggest that the central and peripheral nervous systems may be affected by FAI. Table 5 summarizes the motorneuron pool excitability and sensory change studies discussed in this section.

Table 5. Motorneuron Pool Excitability and Sensory Change

Author, Year	Instrument(s)	Results	Conclusion
Bullock-Saxton 1995[30]	Vibration 2- point discrimination	Higher the vibration frequency, the lower the perception level ↑ vibration needed to reach threshold of perception (FAI) Injured group did not identify 2-point discrimination as well as uninjured	Side to side differences present in vibration, 2-point discrimination, & balance Sensory changes are consequential on ankle injury Motor control is likely to be affected by ankle sprains
Hopkins & Palmieri, 2004[146]	Kinematic Kinetic EMG	PL RMS amplitude decreased following effusion & 30-min post effusion No changes in TA & soleus	↓ joint torque after ankle joint effusion ↓ muscle function accompanies ankle joint effusion in the form of ↓ plantar-flexion torque & PL activation
McVey et al, 2005[130]	H:M ratio	Soleus & peroneal H:M ratio smaller in injured limb of FAI subjects No difference in TA of FAI group Control group had no differences	Depressed H:M ratio suggest AMI in the injured limb musculature
Palmieri et al, 2004[147]	H-reflex M-response H:M ratio	Soleus H-reflex & H:M ratios lower before injection M-wave ↑ immediately after injection PL H-reflex & M-wave were lower before injection No difference in PL & TA H:M-ratios TA H-reflex & M-wave ↑ after injection	MN excitability is facilitated in the PL, soleus, & TA after joint effusion Facilitation is a reaction that helps to maintain postural control & locomotion
Palmieri et al, 2003[148]	COP distance MPF	COP path ↓ after effusion	Postural control improved following joint effusion Additional somatosensory feedback, neural drive or ↑ capsule tension are possible explanations
Sedory et al, 2007[34]	CAR EMG	Quadriceps CAR greater in involved limbs vs. uninvolved; both hamstrings CAR lower in CAI group	Demonstrates change in proximal and neurological related unilateral CAI

Lower Extremity Muscle Activation

Peroneal Muscle Group Latency. The peroneal muscles have been the target of extensive CAI research. Contraction of the peroneus brevis and longus muscles are responsible for ankle eversion. They are thought to be the primary muscles capable of preventing inversion ankle sprains. Functional ankle instability subjects however, have demonstrated increased peroneal muscle latency.[26, 131, 133, 149, 150] Increased peroneal muscle latency is believed to make FAI subjects prone to CAI.[26, 133]

Peroneal muscle latency was measured primarily by using a trap-door mechanism during static stance. This mechanism was an attempt to simulate the mechanism of injury for an inversion ankle sprain. Along with prolonged peroneal muscle latency, the unstable ankle accelerated into inversion faster than the stable ankle.[26] To compensate for the prolonged peroneal latency, FAI subjects demonstrated ankle dorsiflexion, knee flexion, hip adduction, and flexion.[131] Lofvenberg et al[133] assessed the reaction times of the peroneus longus (PL) and tibialis anterior (TA). Prolonged peroneal reaction time was observed in FAI subjects compared to healthy controls, but not when the injured limb was compared to the uninjured limb.[133] This indicated FAI may affect both limbs and it may be better to compare FAI subjects to a healthy control group instead of their uninjured limb. [133] The TA muscle did not demonstrate any latency changes.[133] Ankle taping[150] and injecting local anesthetic into the sinus tarsi[149] decreased the reaction time of the peroneal muscles. However, facilitation of the soleus, PL, and TA was observed following an injection of a saline solution into the ankle capsule. [147] Therefore, it was suggested the MN-pool excitability may increase following an injection of saline solution into the ankle joint capsule.[147]

Subjects have also demonstrated no increase in peroneal muscle latency.[90-95] These studies used a similar trap-door methodology to measure latency. No difference in peroneal latency was observed between limbs[90, 91, 93] or groups.[93-95] Peroneal reaction time was not affected three weeks postacute ankle sprain.[90] A flaw of this study was that reaction time was not measured until three weeks post-injury, so it is unknown if a difference in reaction time existed immediately following an acute sprain.

Leg dominance, inversion angle, and rehabilitation influence peroneal muscle latency.[91, 92, 94] Peroneal latency increased as the trap door angle increased.[92] This study also theorized that leg dominance may influence peroneal latency more than an injury. [92] All the subjects were right dominant and the right peroneal latency was greater than the left side.[92] Rehabilitation programs may allow peroneal muscle function to return to normal.[91, 94]

A consensus has not been reached regarding peroneal latency in FAI subjects. One reason there may not be a consensus is that the static trap-door mechanism may not simulate what occurs during dynamic movement (ie., jogging, running, landing).[151]

Konradsen et al[132] used electromechanical delay (EMD) to assess the role of the peroneal, hamstrings, and quadriceps muscles as a defense mechanism to prevent inversion ankle sprains during static stance and walking in healthy subjects. A trap-door mechanism was used in this study to assess EMD at three different starting positions: 10° inversion, neutral, and 10° eversion. The 10° inversion position produced the fastest muscle activation out of the three starting positions. Subjects in this study demonstrated a median of 20° ankle dorsiflexion, 30° knee flexion, and 25° of hip flexion to correct for the dropping of the trap-door.[132] This study observed peroneal muscle activity prior to the hamstrings and quadriceps muscle activation.[132]

The authors concluded that the reflex reaction to sudden inversion was initiated at the peripheral level followed by spinal or supraspinal centers.[132] Both the peripheral and supraspinal responses were thought to be too slow to prevent an inversion ankle sprain at heel strike.[132] Table 6 summarizes the peroneal muscle latency studies discussed in this section.

Table 6. Summary of Peroneal Muscle Latency

Author, Year	Instrument(s)	Results	Conclusion
No Prolonged Latency			
Ebig et al, 1997[91]	SEMG	No difference in PL & TA mean firing rates between stable & unstable ankles No difference between TA & PL No difference between stable & unstable for all motions	FAI may not result in a diminished PL or TA response Subjects participated in a rehab program
Fernandes et al, 2000[92]	SEMG	↑ latency as tilt angle increased Latency differed by sides, right < left injured and healthy groups had no difference in PL latency	Ankle instability does not influence PL latency Side dominance may influence latency
Isakov et al, 1986[93]	SEMG Latency	Latency times did not differ by leg or group	Stretch inversion motion has no role in protecting the ankle
Johnson & Johnson, 1993[94]	Inversion platform SEMG	No difference between 3 groups or between affected & unaffected ankle	Rehabilitation appears to enable normal peroneal function in both surgical & nonsurgical lateral ankle sprains
Konradsen et al, 1998[90]	SEMG	PRT did not differ between limbs @ 3wks post-acute sprain	Peroneal reaction time to sudden inversion was not affected 3wks post injury
Vaes et al, 2002[95]	SEMG	1st deceleration time was shorter in unstable ankle Latency, total inversion time, 2nd deceleration time, & EMD were not different	Unstable ankles have less control of inversion speed Peroneals do not have longer latency
Prolonged Latency			
Khin Myo, et al, 1999[149]	BAPS board SEMG	Sense of instability was gone postinjection for all subjects PRT was 82.0 ms & 69.3 ms pre- & postinjection	Difference existed between groups before injection & within the FAI group before & after injection Inflammation may suppress peroneal gamma-MN activity & cause FAI & shorten PRT
Karlsson & Andeasson, 1992[150]	SEMG	Difference in MAI between stable & unstable ankle joints Unstable ankles had > PRT than stable ankle Taping ↓ PRT in the unstable ankle	Tape has an effect on proprioceptive ankle function & may improve FAI
Konradsen & Ravn, 1990[131]	SEMG Electric goniometer	All subjects reacted with ankle dorsiflexion, knee flexion, hip adduction & flexion	Prolonged reaction time in unstable ankle

Konradsen et al, 1997[132]	SEMG 2-D kinematics	Peroneal reflex latency median was 48ms Knee flexion reached median of 30° Median hip flexion was 25°	Dynamic response to sudden inversion involves both peripheral reaction & a central mediated strategy A uniform reaction pattern exists for both unilateral & contralateral limbs Only anticipated muscle activity, static stabilizer strength, or external support can prevent an injury
Lofvenberg et al, 1995[133]	SEMG	No difference in median PRT between CAI group contralateral and ipsilateral limbs Median ipsilateral PRT was longer in CAI group when compared to healthy group	Delayed proprioceptive response to sudden angular displacement of the ankle can be 1 of the causes of CAI
Vaes et al, 2001[26]	Accelerometer SEMG	Unstable ankles ↓total supination time ↑ PL latency in unstable ankle (P=0.017)	Unstable ankle has shorter total supination time, less efficient deceleration Peroneals protect the ankle less

EMD- electromechanical delay; FAI- functional ankle instability; IEMG- integrated electromyography; MN- motor neuron; ms- milliseconds; Nm- Newton meters; PL-peroneus longus; PRT- peroneal reaction time; SEMG- surface electromyography; TA- tibialis anterior; wk- week

Altered Electromyography. Subjects with FAI have demonstrated altered peroneal muscle activation during walking compared to healthy subjects.[27, 138] Delahunt et al[138] analyzed electromyography (EMG) of the PL, rectus femoris, TA, and soleus. The PL was the only muscle to demonstrate altered muscle activation in FAI subjects. Muscle activation was analyzed at three different times: preheel strike, at heel strike, and postheel strike.[138] The FAI subjects had greater rectus femoris activation during preheel strike and increased PL EMG postheel strike.[138] Functional ankle instability subjects had greater inversion throughout all three time periods with a decrease in foot to ground clearance.[138] The combination of altered PL activation and biomechanical factors make FAI subjects prone to CAI.[138]

Side-to-side comparisons of unilateral FAI subjects demonstrated that the PL muscle was activated for a shorter time on the injured side.[27] The timing of the PL is altered following an injury, makes FAI subjects prone to injury.[27] Table 7 summarizes the altered lower extremity muscle activation studies discussed in this section.

Table 7. Altered Lower Extremity Muscle Activation

Peroneals			
Author, Year	Instrument(s)	Results	Conclusion
Santilli et al, 2005[27]	Kinematics SEMG	No correlation between sides & PL activity Injured side ↓ PL activation (22.8%) time during stance phase vs. uninjured (37.6%)	Timing of the PL Δ after injury ↓ PL activity may reduce protection of lateral ankle sprains
Delahunt et al 2006[138]	SEMG Kinematic	FAI ↑ inversion @ pre-IC, IC, & post-IC FAI ↓ foot-floor clearance during terminal swing FAI ↑PL EMG post-HS FAI ↑RF EMG pre-HS	FAI subjects demonstrate altered ankle joint neuromuscular control & kinematics ↑ PL activity may result from Δ in preprogrammed feedforward motor control

Lower Extremity Strength Deficits

Eversion Strength. The strength of the evertors has been the primary focus of researchers studying strength deficits in FAI subjects.[152]Isometric eccentric eversion strength deficits existed following an acute inversion ankle sprain.[90] Eversion strength deficits remained at three weeks postacute sprain when compared to the uninjured limb. However, at the 12-week follow-up, evertor strength in the injured limb was 96% as strong as the uninjured limb.[90] It was not documented if the subjects participated in a rehabilitation program or not. Therefore, it is an assumption that evertor muscle strength will be close to normal after 12 weeks postacute sprain. This study used the uninjured limb of FAI subjects as the control.

Bush[134] observed a surprising isometric eversion strength difference between sides. The eversion strength of the injured limb was 8% greater than the healthy limb. This is surprising because the strength deficit was actually on the healthy limb. This may have occurred because the subjects in this study were collegiate athletes who had returned to full activity; therefore, they probably participated in a rehabilitation program. If rehabilitation was only performed to the injured limb, this may have contributed to the injured limb being stronger.

Strength of the lower leg muscles were measured using an isokinetic device, decreased eversion strength was observed.[7, 13, 153] A relationship existed between strength ratio (eccentric evertor/concentric inversion peak torque) deficits and CAI. Chronic ankle instability subjects demonstrated muscle weakness compared to a healthy control group strength.[13, 153] The peak torque ratio increased as velocity increased except at 180°/s & 240°/s in both groups.[13] Eccentric evertor peak torque and strength ratios were different between the CAI and healthy groups near the ends of range of motion.[153] The CAI group had lower peak evertor strength throughout the range of motion.[153] Strength deficits were also observed in a CAI group when eversion peak torque values were normalized to body weight.[7] Willems et al[7] observed that CAI subjects had evertor muscle weakness and decreased JPS. Porter et al[154] used strength ratios (peak torque/body weight) and time to peak torque to assess dorsiflexor and evertor strength in FAI subjects. No strength or time to peak torque differences were observed between FAI and healthy subjects.[154]

Although the previous studies observed eversion strength differences, other studies have observed no differences in eversion strength.[5, 9-12, 96, 97] Inversion and eversion mean peak torque values were measured isokinetically and isometrically, no difference was observed between the limbs of CAI subjects.[97] The authors concluded that muscle weakness was not a major contributing factor to CAI.[97] Bernier et al [96] assessed eccentric inversion and eversion strength and postural sway. No difference was observed in postural sway or strength between the FAI and healthy groups.[96] However, inversion peak torque was different between the dominant and nondominant limbs in the healthy control group, suggesting that limb dominance may be an indicator of strength.[96]

No difference in concentric and eccentric peak torques,[10, 12, 155] eversion or inversion strength[5, 155] were observed in FAI subjects. These studies concluded that FAI do not appear to have eversion[5, 9, 10, 12, 155] or inversion[5, 10, 96, 97] strength deficits. Ryan[5] observed no difference in eversion strength, however, observed that FAI subjects had a decrease in balance. McKnight and Armstrong[10] observed no strength difference during all ankle motions.[10]

Inversion Strength. Invertor strength was observed to be less than evertor strength.[12, 14] Subjects with an acute ankle sprain demonstrated greater invertor weakness than those with CAI.[14] invertor muscle weakness may be a contributing factor to acute ankle sprains and CAI. This is an area that needs further research, but at this time the majority of researchers have observed inversion strength deficits.

Plantar-flexion Strength. Plantar-flexion strength deficits have been observed.[135, 156] Peak torque differences were observed between limbs and groups.[135] The injured side of FAI subjects demonstrated decreased plantar-flexion range of motion and hip abductor strength, a correlation existed between hip abductor and extensor strength.[156] One study observed no difference in plantar-flexion strength.[10] Plantar-flexor weakness may influence the risk of an ankle injury. Table 8 summarizes the lower extremity strength studies discussed in this section.

Table 8. Summary of Lower Extremity Strength Studies

Author, Year	Instrument(s)	Results	Conclusion
Kaminski & Hartsell, 2002[152]	Literature review		Ankle strength deficits are not highly correlated with CAI
McKnight & Armstrong, 1997[10]	Isokinetics Goniometer	No difference between groups for any ROM No differences in strength or work measurements between groups	No differences in AROM, strength, or work measurements between groups
Evertors			
Bernier et al, 1997[96]	Balance system Isokinetic	No difference in 1-leg postural sway or eversion strength between limbs Difference between dominant & nondominant limbs in healthy	Postural sway & PT are not affected by FAI
Bush, 1996[134]	Strain gauge Inclinometer Force plate Platforms	No difference between injured & healthy side in proprioception, ROM, & sway Evertor strength on the injured side was 8% ≥ healthy side Both ankles of the injured group are weaker than the control group	Evertor strength was the only significant predictive factor Evertor weakness is a strong predictor of an ankle sprain
Kaminski et al, 1999[155]	Isokinetic dynamometer	Concentric PT ↓ as velocity ↑ No difference in concentric, eccentric, or isometric eversion strength between groups	Unless an obvious weakness to evertors exist, strengthening may be a waste of time and energy Eversion muscle strength deficits were not found
Konradsen et al, 1998[90]	SEMG	PRT did not differ between FAI & healthy groups Injured ankle eversion strength @ 3 wks was ↓ than healthy	Acute inversion injuries result in marked Δ to assess inversion position Peroneal reaction time to sudden inversion was not affected 3 wks post injury
Lentell et al, 1995[9]	Passive JPS Isokinetics (PT)	> passive motion in involved ankles No difference in PT between ankles	No evertor weakness present > passive JPS & talar tilt present in the involved limb
Lentell et al, 1990[97]	Isokinetics	No PT difference between involved & uninvolved ankles Isometric & isokinetic PT was symmetrical across population ↓ balance on injured limb	Evertor or invertor weakness are not associated with CAI Balance deficits associated with FAI limb
Ryan, 1994[5]	Isokinetics UBE	Evertor mean strength was not different between stable (19.2 Nm) & unstable ankles (18.8 Nm) Proprioceptive differences between affected (4.0 s) & unaffected ankles (1.8 s)	MAI is frequently absent from FAI subjects Evertor weakness was not present Impaired proprioception contributes to FAI
Munn et al, 2003[12]	Isokinetic	No eccentric or concentric evertor strength deficit was found in the injured limb Max eccentric inversion strength was ≤ eccentric eversion Eccentric inversion torques for injured limb were 12% >	FAI is not associated with evertor strength deficits Invertor strength deficits were found Weak invertors may contribute to FAI

Ryan, 1994[5]	Isokinetics UBE	Evertor mean strength was not different between stable (19.2 Nm)& unstable ankles (18.8 Nm) Balance differences existed between affected (4.0 s) & unaffected ankles (1.8 s)	MAI is frequently absent from FAI subjects Evertor weakness was not present Impaired proprioception contributes to FAI
Wilkerson et al, 1997[14]	Isokinetic	> invertor than evertor deficits for PT & average power Acute group had > deficits than chronic group	Lateral ankle ligament injury may be associated with invertor deficits Rehab may restore evertor/invertor strength relationship
Strength ratios			
Hartsell & Spaulding, 1999[13]	Isokinetic dynamometer	Unstable ankle was weaker eccentrically & concentrically for inversion & eversion	CAI & muscle weakness co-exist
Porter et al, 2002[154]	Isokinetic PT TPT	No difference in concentric dorsiflexion PT/BW ratio & TPT between FAI limbs & group No difference in concentric eversion PT/BW ratio & TPT between FAI limbs & group TPT values were @ 240° s were slower that 120° s	No concentric strength or TPT differences in CAI subjects
Willems et al, 2002[7]	Active & passive JPS (Biodex) Peak torque	No difference for active or passive JPS Instability group had lower eversion strength values than control & other 3 groups	CAI group underestimated the reference angle CAI may have inappropriate foot positioning No relationship between invertor strength & sprains Evertor strength differences observed Diminished proprioception & evertor weakness
Yildiz et al, 2003[153]	Isokinetics	Eccentric evertor/concentric invertor strength ratios were lower in CAI group @ 15° & 20° Eccentric evertor PT near end range were ↓ in CAI Both CAI & healthy groups had ↓ concentric invertor PT values Strength ratios ↑ @ end range	CAI group has differences in strength (E_{ecc}/I_{con} & eccentric evertor)@ end ROM End ROM strength values are most valuable Evertor strength weakness may predispose recurrent ankle injuries
Hip			
Friel et al, 2006[156]	Goniometer Handheld dynamometer	Hip abductors weaker on involved side Plantar-flexion ROM < on injured side Hip abductor & extensors correlated	CAI subjects have weaker hip abductors
Plantar-Flexors			
Fox et al, 2008[135]	Isokinetics	Difference between FAI limb & matched control PT Difference between sides of control group PT	Deficit in plantar flexion PT in FAI subjects

Postural Control

Functional ankle instability is believed to foster poor postural control. Poor postural control is the inability to maintain stability during dynamic or static movement.[157] Many different clinical and research techniques are used to assess postural control. The force plate is the most common instrument used to assess postural control in research. Measurements that use a force plate include sway, center of pressure (COP) excursion, Time to Boundary (TTB), Time to Stabilization (TTS), and the Dynamic Postural Stability Index (DPSI). Other measurement methods used clinically and in research include the Biodex Balance System, Balance Error Scoring System (BESS), Single-Leg Stance Test, and Star Excursion Balance Test (SEBT). Due to the wide variety of measurement techniques used to assess postural control, this section will be organized by measurement technique in an attempt to compare study results.

Sway. Assessing sway in the anterior-posterior and medial-lateral directions provides an objective measure to assess postural control. The theory that FAI subjects have poor postural control compared to healthy controls was not confirmed using sway.[158-160] However, subjects with higher sway values were at a greater risk of an ankle injury during the following competitive season.[160] It was concluded that FAI subjects may have a built in compensatory mechanism to cope with poor balance.[160] Ankle taping did not increase stability or decrease sway.[159] Another study measured single-leg stance sway and TTS during a single-leg jump.[158] Sway values did not differ between groups, but it took FAI subjects longer to stabilize.[158] Postural control deficits may not be present during a static task like the single-leg stance. Therefore, a more dynamic task such as the single-leg jump may need to be used to assess postural control changes. It can be concluded that sway measurements may not truly represent changes in postural control for FAI subjects.

Center of Pressure. Center of pressure is widely used to assess postural control. Postural control deficits were observed during a single-leg stance in FAI[22, 161] and acute ankle sprain subjects.[23, 162] Subjects demonstrated an increased COP excursion velocity (COP_V) bilaterally following an acute sprain during a single leg stance.[23] This indicated that an acute sprain may result in a centrally mediated mechanism which contribute to bilateral postural control deficits.[23] These deficits were observed at the following time periods: 1-day, 7-days & 21-days postinjury with the injured limb having a greater COP_V.[23] Center of pressure distance may be more sensitive than other measurement methods at measuring postural control changes.[22] Decreased stability was observed two years following an initial ankle injury.[22] Peroneal latency and ankle position are highly correlated with COP.[161, 162] Mitchell et al[162] observed the unstable ankle in subjects with an acute sprain had greater COP excursion when vision was eliminated. Functional ankle instability subjects used different strategies to maintain postural control compared to a healthy control group.[161] When the ankle could no longer maintain postural control, FAI subjects used their hip to make corrections.[161] The hip correction strategy was used when larger corrections were needed to maintain stability.[161] There seems to be some confusion regarding which COP method is most accurate at detecting postural control changes. Despite this confusion postural control deficits were observed in postacute ankle sprain and in FAI subjects during a single-leg stance. As the difficulty of a task increased, it appears that the proximal muscles play a greater role in maintaining postural control.[35]

Time to Boundary. Chronic ankle instability subjects demonstrated postural control deficits during the TTB measurement.[21, 136] Both genders of CAI subjects have demonstrated postural control deficits using the TTB test.[21, 136] Researchers assessed postural control in healthy and CAI female subjects using both COP and TTB during single-leg stance.[21] Chronic ankle instability subjects demonstrated bilateral postural control deficits using the TTB measurement.[21] The CAI subjects had lower TTB scores and increased COP_V excursion compared to the healthy control.[21] It was concluded the TTB test detects postural control deficits better than COP excursion.[21] Gender differences were not seen during a single-leg stance with eyes open or closed using TTB.[136] The authors theorized that CAI may place constraints on the sensorimotor system during prolonged single leg stance.[136] Therefore, CAI subjects may have to alter their postural control strategies.[136]

Time to Stabilization. The TTS measurement during a jump landing was different between healthy and FAI individuals.[163, 164] Functional ankle instability subjects took longer to stabilize following a jump landing than healthy subjects.[18, 137, 158, 164] Researchers observed greater TTS scores during a jump protocol compared to a step protocol.[163] Therefore, a jump protocol will detect dynamic stability deficits better than a step protocol.[163] The ability to control movement decreased during a jump protocol, therefore, a step protocol may allow the subject to accurately repeat the motion. It was suggested that TTS should be used to detect dynamic postural control changes.[158, 164] Time to Stabilization can detect poor postural control in FAI subjects during a single-leg jump landing.

Dynamic Postural Stability Index. The DPSI is a relatively new measurement technique used to assess postural control deficits. It is a reliable and practical measurement of dynamic postural control that provides a comprehensive measurement of dynamic stability in three directions.[165] Wikstrom et al[24] used DPSI to assess postural control between FAI and healthy subjects. Functional ankle instability subjects demonstrated differences in the anterior-posterior, vertical, and DPSI indexes compared to a healthy group. The authors concluded the DPSI is a reliable measurement to assess postural control and suggested FAI subjects use a nonankle strategy to maintain stability.[24] The altered strategy may predispose FAI subjects to CAI.[24]

Equitest. The Equitest was used to assess the subject's response to a standardized perturbation.[6, 20] Subjects with bilateral ankle sprains demonstrated an increase in postural sway following a perturbation.[6] A positive relationship was also seen between ankle repositioning and postural sway in collegiate basketball players during single leg stance.[6] Pintsaar et al[20] monitored postural corrections during three different perturbations (small, medium, and large) between three groups (healthy, FAI, and MAI). No difference in reaction time was observed between groups, however, the reaction time to a medium perturbation was less than a small perturbation.[20] The primary finding of this study was ankle function is related to coordination.[20] The ankle was responsible for making small postural control corrections while the hip corrected larger postural adjustments.[20] This study supported the theory that impaired neuromuscular function is responsible for altered postural control strategies.[20] McGuine et al[15] used the NeuroCom System to assess the balance of high school basketball players prior to their season.[15] Subjects with higher preseason postural sway scores were seven times more likely to sprain their ankle.[15] This suggested if postural control can be improved, the risk of a lower extremity injury may decrease.

Balance Error Scoring System. The BESS is a clinical test used to assess an individual's static balance during three stance positions (double-leg, single-leg, and tandem). It has been used to measure balance differences in healthy[166] and FAI suubjects.[17] Functional ankle instability subjects made more balance errors than the healthy subjects during three balance tasks (single-leg on floor, tandem stance on foam, and single-leg stance on foam).[17] The BESS is a reliable method to measure postural control changes in FAI subjects.[17]

Star Excursion Balance Test. This test is becoming a widely used method to assess dynamic balance clinically and in research. The SEBT measures reach distance in eight directions (anterolateral, anterior, anteromedial, medial, posteromedial, posterior, posterolateral, and lateral) during single-leg stance. High reliability was obtained for the SEBT when three reach directions (anterior, posteromedial, and posterolateral) were used.[167] The SEBT may be used as a preparticipation test to predict lower extremity injuries in high school basketball players.[167] A decrease in the functional reach test, SEBT, and Biodex balance index was observed following an acute ankle sprain.[168] These three balance test scores were highly correlated.[168] It was concluded that an acute ankle sprain affected unconscious proprioception more than conscious proprioception.[168] This emphasizes the importance of the feedforward mechanism. Researchers have also used the SEBT to assess the effect of fatigue on reach distance in CAI subjects.[32, 33, 169] Unilateral CAI subjects had a decreased reach distance when standing on the injured limb compared to a matched control group.[169] Following a fatiguing protocol CAI subjects displayed a decrease in SEBT reach distances.[32, 33] Knee flexion angles were also less after fatiguing; this indicated the neuromuscular deficits associated with CAI affected the proximal joints.[33] It is believed that the neuromuscular deficits observed at the ankle in CAI subjects also occur to the proximal joints.[32, 33] The SEBT is used by clinicians and researchers to measure dynamic postural control deficits in CAI subjects.

Single-Leg Balance Test. The single-leg balance test has been assessed in different ways. Sometimes it is quantified by the length of time an individual can maintain balance on one limb or it may be judged by the individual or an observer. Subjects with FAI or a history of a severe unilateral ankle sprain demonstrated poor balance when the injured limb was compared to the healthy control during a Single-Leg Stance Test.[5, 30] Functional ankle instability subjects spent 2.2 seconds longer out of balance on the injured limb.[5] Severe ankle sprain subjects had a 5.7 second difference between injured and uninjured sides. Motor function was impaired by both FAI and a severe ankle sprain.[5, 30]

A decrease in single-leg balance was perceived by subjects with a history of ankle sprains and observed by judges.[128, 129] The majority of subjects with a history of multiple ankle sprains perceived better balance on the uninjured limb. [128, 129] Judges were blinded and observed decreased balance on the injured limb.[128, 129] Table 9 summarizes the postural control studies discussed in this section.

Table 9. Summary of Postural Control Studies

Author, Year	Instrument(s)	Results	Conclusion
Sway			
Tropp et al, 1984[159]	Sway	No stability difference b/w FAI & healthy groups 6 wks ankle disk training ↑ stability Ankle taping did not improve stability	Ankle injury along does not produce FAI Taping does not effect stability 6 wks ankle disk training ↑ stability & ↓ "giving way" feeling
Tropp et al, 1984[160]	Sway	Ankle injury history did not affect sway ↑ sway=↑ injury risk	Injured group not at higher risk of instability than uninjured FAI @ no > risk of ankle injury FAI may compensate for disturbanced of balance
Ross & Guskiewicz, 2004[158]	Sway TTS	Mean A/P & M/L sway during 1- leg stance does not differ between groups (FAI & healthy) A/P & M/L TTS differed	FAI took longer to stabilize Suggest using TTS to assess FAI individuals
Time to Stabilization			
Brown et al, 2004[18]	Biodex, Vertec, Force plate, EMG	No difference in JPS error scores in 4 directions TTS = ↑ in FAI subjects No EMG difference 200 ms before landing Soleus activated differently between groups in 1000 ms after landing	No difference in replicating joint angles Difference in landing patterns between FAI & healthy FAI subjects land dorsiflexed
Ross & Guskiewicz, 2003[164]	TTS	TTS with a jump landing can differentiate between FAI & healthy FAI limbs take longer to stabilize	TTS with a jump landing provides a way to identify dynamic postural control deficits
Ross & Guskiewicz, 2004[158]	Sway TTS	Mean A/P & M/L sway during 1-leg stance does not differ between groups (FAI & healthy) A/P & M/L TTS differed	FAI took longer to stabilize Suggest using TTS to assess FAI individuals
Ross et al, 2005[137]	TTS	TTS longer for the FAI group TTS reliability is moderate to poor in A/P & M/L directions	TTS is longer in FAI group Ankle instability may impair the subjects' ability to stabilize after a 1-leg landing
Wikstrom et al, 2005[163]	Force plate	Jump protocol produced > TTS scores in the vertical direction than the step protocol	Jump protocol will be more successful detecting differences in dynamic stability than a step down
Center of Pressure			
Cornwall & Murrell, 1991[22]	COP distance	Postural sway greater in FAI No difference between AP & ML directions	FAI patients are less stable during a single-leg stance COP distance may be more sensitive to changes versus other studies Instability evident 2 yrs following injury

Evans et al 2004[23]	COP_V Acute sprains	Postural control deficits in both the injured & healthy ankle after 1 day Deficit also noted on day 7 & 21 in both ankles	Bilateral impairments in postural control during 1-leg stance after ankle injury Bilateral deficits indicate centrally mediated mechanism with acute joint injury > postural control deficits in injured limb
Mitchell et al, 2008[162]	COP	UA > lateral & medial sway without vision than SA than the DA & NDA Correlation between PL & PB reaction times & lateral, medial, & anterior sway	FAI subjects have sway deficits Relationship between PL & PB reaction times & sway in UA
Tropp & Odenrick, 1988[161]	COP SEMG	COP is highly correlated to position of ankle & peroneal activity Corrections made @ hip	Different strategies exist for maintaining equilibrium in 1-leg stance Hip is used to correct disequilibrium Hip strategy makes larger corrections possible When ankle can no longer maintain postural control – hip strategy is used
DPSI			
Wikstrom et al, 2005[163]	DPSI	DPSI was highly reliable & precise between sessions Mean 10s> 5s>3s interval trials	DPSI is a reliable & practical measure of postural control 3 s interval is best to study athletic performance
Wikstrom et al 2007[24]	DPSI 1-leg hop test	Difference in A/P, vertical, & DPSI stability indexes FAI were deficient in these indexes	Ankle instability causes motor control changes, forcing FAI to use a nonankle strategy, & predispose them to injury DPSI is sensitive enough to measure dynamic postural stability in FAI & healthy
TTB			
Hertel & Olmstead-Kramer, 2007[21]	COPV, TTB	CAI group had ↓TTB and ↑COPV	Postural control deficits were noted bilaterally using TTB Suggests centrally mediated postural control in CAI
McKeon & Hertel, 2008[136]	TTB	No group by gender interaction or gender main effects CAI group observe deficits in 4 of 6 measures with EC	CAI may place > constraints on the sensorimotor system during 1-leg stance May indicate diminished ability to respond effectively to postural control demands in CAI
Equitest			
Fu & Hui-Chan, 2005[6]	SOT	↑ postural sway (SOT) in injured subjects	Basketball players with MAS have ↑ ankle repositioning & postural sway during stance A positive relationship was found between repositioning & postural sway

Fu & Hui-Chan, 2007[71]	SEMG of TFL, TA, & PER	TFL activates later than TA during landing in subjects with BMAS	BMAS use different landing strategies Change in prelanding EMG noted @ ankle and hip
Pintsaar et al, 1996[20]	Equitest	No latencies differences among 3 groups Latency was shorter for medium than for small translations	Ankle corrects small perturbations Hip corrects larger perturbations Impaired function is related to change in strategy
Ryan, 1994[5]	Isokinetics UBE	Evertor mean strength was not different between stable (19.2 Nm) & unstable ankles (18.8 Nm) Proprioceptive differences between affected (4.0 s) & unaffected ankles (1.8 s)	MAI is frequently absent from FAI subjects Evertor weakness was not present Impaired proprioception contributes to FAI
Van Deun et. al., 2007[70]	EMG Force plate	Later ankle, hip, & hamstring onset during 2-leg to 1-leg stance CAI subjects used similar muscle activation patterns Controls adjusted their activation patterns to the condition Muscle activation seemed proximal to distal	Lower extremity activation patterns vary between healthy & CAI CAI use 1-landing strategy CAI activate ankle, knee, & hip later than control
Balance			
BESS			
Bressel et al, 2007[166]	BESS SEBT	Gymnasts 55% less errors than basketball group BESS Soccer group 7% further reach than basketball group SEBT	Basketball group less static balance than gymnastic group Basketball group inferior dynamic balance than soccer group
Docherty et al, 2006[17]	BESS	FAI scored higher errors 1-leg on floor, tandem stance on foam, & 1-leg on foam	Postural control deficits were in FAI using the BESS BESS is reliable at measuring postural control changes in FAI patients
SEBT			
Akbari et al, 2006[168]	SEBT FRT Biodex balance system	Injured limb had a ↓ FRT, SEBT, & balance index during unilateral stance on injured limb Strong relationship existed between all balance tests	The unconscious part of proprioception is more severely affected than the conscious part
Gribble et al, 2004[33]	Isokinetics Kinematics SEBT	CAI had smaller reach distance & knee-flexion angles Fatigue amplified this trend	CAI & fatigue disrupted dynamic postural control, most notably joints proximal to the ankle Neuromuscular deficits associated with CAI result in similar changes to proximal neuromuscular control
Gribble et al, 2007[32]	SEBT Kinematics Ankle & lunge fatigue	Lunge fatigue, CAI, & Δ in knee & hip flexion predicted ~49% of % MAXD CAI predicted 20% of medial % MAXD CAI predicted 18% of anterior % MAXD	Isolated ankle fatigue did not cause different responses between groups Functional fatigue protocols may expose deficits in dynamic postural control caused by neuromuscular control alterations in proximal joints present in CAI subjects

Olmstead et al, 2002[169]	SEBT	CAI had ↓ reach distance compared to matched control group & uninjured side CAI reached less standing on the injured side	SEBT may be an effective means to determine reach deficits between & within unilateral CAI subjects
Pliskey et al, 2006[167]	SEBT	SEBT is reliable (ICC 0.82 to 0.87) Anterior right/left reach distance > 4cm = 2.5x likely to sustain a lower extremity injury Girls with a composite reach ≤ 94% 6.5x likely to have a lower extremity injury	The SEBT is a reliable measure to assess balance & may predict possible injury.
Robinson & Gribble, 2008[170]	Normalized SEBT	Increased excursion distance (7), hip flexion (4), & knee flexion (5) occurred out of the 8 reach directions	Maximum reach distance & 1-leg angular displacement achieved stability within 4 practice trials Recommend reducing the # of practice trials from 6 to 4
1- leg stance			
Bullock-Saxton 1995[30]	1-leg stance	5.7s difference between injured group's injured side vs. uninjured side	Side to side differences present in vibration, 2-point discrimination, & balance Sensory changes are consequential on ankle injury Motor control is likely to be affected by ankle sprains
Forkin et al, 1996[128]	1-leg balance (EO & EC)	9 of 11 subjects perceived balance better on uninjured d Observers judged EO balance better in 4 & 5 subjects Observers judged EC balance better in 7 of 11 subjects	Balance & kinesthetic deficits are common in gymnasts with MAS
Garn & Newton, 1988[129]	1-leg balance test	13 subjects perceived ↓ balance on injured vs. healthy side Observer 16 subjects had ↓ balance injured vs. healthy side	Kinesthetic deficits exist in the injured ankle joints of athletes with a history of MAS
McGuine et al, 2000[15]	NeuroCom (postural sway)	Higher preseason postural sway scores corresponded with increased ankle sprains; those with poor balance were 7x likely to sprain their ankle	Preseason postural sway can be used to predict ankle injuries in high school basketball players

Biomechanics

Landing Types. Three types of landings have been discussed in the literature. A stiff landing occurs when the peak knee flexion angle is less than 90°, therefore, a stiff landing places individual's in a more erect posture.[69, 98] An erect posture places additional stress on the ankle joint compared to a soft landing. Greater hip and knee flexion occur during a soft landing, knee flexion greater than 90° is representative of a soft landing.[98] Greater demands are placed on the proximal joints and muscles during a soft landing compared to a stiff landing.[69, 98] It is believed that individuals choose between a stiff or soft landing, however, individuals have used a combination of these landing types.[171] The combination of landing strategies is referred to as the preferred or normal landing type of landing.[172]

Landing Strategies. Individuals use different landing strategies to maintain postural control following an unexpected perturbation. The ankle and hip strategies are discussed in the literature.[173] An ankle strategy is primarily used to make corrections to small perturbations or during static movements such as a single-leg stance. The hip strategy is used to correct for larger perturbations or during dynamic tasks where the center of mass is displaced outside of the body's base of support. A combination of these strategies are used to maintain balance as velocity increases following a backward translation.[171] Healthy subjects use a hip or preferred landing strategy during dynamic tasks which allow them to perform a soft landing while FAI subjects use the ankle strategy which produces a stiff landing.

Energy Absorption During Landing. Energy absorption varied depending on impact velocity and type of landing performed.[69, 101] As landing height and demands placed on the lower extremity were increased, subjects altered their landing strategy.[98, 101] Athletes may not use their full energy absorption capabilities during every task.[171] The ankle absorb the majority of the energy dispersed throughout the kinetic chain during a stiff landing.[69, 98, 171] A soft landing allowed the proximal joints and surrounding muscles to absorb more energy, therefore, taking some stress off the ankle.[69, 98, 171] Energy absorption differences between FAI and healthy controls have not been reported. Anterior cruciate ligament reconstruction (ACLr) subjects demonstrated a stiff landing compared to healthy subjects. This required ACLr subjects to use an ankle landing strategy.[174] Knee energy absorption is a predictor of leg impedance in female athletes.[172]

Energy absorption can be altered through training and changing abdominal postures.[101, 175] McNitt-Gray[101] compared energy absorption between elite male gymnasts and recreational athletes following a drop landing. Gymnasts used larger ankle and hip extensor moments to absorb energy. Kulas et al[175] concluded abdominal postures (AH and pelvic tilt) may influence lower extremity energy absorption.[175] Training individuals to maintain an abdominal posture may allow them to increase energy absorption up the kinetic chain and improve postural control. The relationship between abdominal muscles and lower extremity injury needs to be researched further.

Perturbations. The kinematics of the ankle, knee, and hip were measured under varying conditions. Riemann et al[176] assessed the corrective actions of the ankle, knee, hip, and trunk in healthy subjects during a single-leg stance on different surfaces (firm, foam, and multiaxial) with eyes open and closed. Closed eyes make it harder to maintain balance.[176] Proximal joint involvement increased, as the difficulty of the task increased.[176] This study did not assess corrective actions on the foam and multiaxial surfaces with the eyes closed.

Following a medial-lateral perturbation to the pelvis or shoulder the center of mass was displaced in the direction of the perturbation.[177] Displacement was corrected by hip joint movement followed by the ankle joint.[177] The contralateral hip reacted first to a shoulder perturbation while the ipsilateral hip reacted first to a pelvis perturbation.[177] Many subjects overshot the correction of the perturbations prior to returning to the starting position.[177] This suggested the central nervous system (CNS) initiated the corrective action to a perturbation prior to the completion of the perturbation, indicating a feedforward mechanism.[177] The direction and location of the perturbation dictated the corrective response not the magnitude.[177, 178]

Henry et al[178] assessed the corrective response of healthy subjects to lateral and anterior/posterior perturbations. An anterior perturbation was a two stage correction (ankle & hip displaced, then return to neutral) while posterior and lateral perturbations had a three stage correction (hip & ankle displaced, hip angle returns, ankle & hip return to initial position).[178] Controlling the center of mass required both the ankle and hip.[178] The tensor fascia latae contracted first during lateral perturbations while distal muscles activated first with anterior and posterior perturbations.[178]

Location of the trunk during a forward lunge dictated the involvement of the ankle, knee, and hip.[179] Joint angles and impulses were assessed during a forward lunge with the trunk in three different positions (flexed, normal, and extended).[179] Greater peak dorsiflexion, knee flexion and less hip flexion occur when the trunk is extended.[179] An increase in GMax and BF muscle activation was observed with a flexed trunk.[179]

Muscle Activation. Neuromuscular function was compared between elite triple jumpers and recreational active subjects during two-drop jump heights (40 cm & 80 cm).[180] The triple jumpers jumped higher at both heights than the control group, this demonstrated triple jumpers had more efficient neuromuscular control.[180] Jumpers activated their vastus lateralis and gastrocnemius muscles earlier than the control group.[180] Greater knee flexion was observed in the triple jumpers than the controls.[180] This may indicate trained athletes use a hip landing strategy while recreationally active subjects use an ankle strategy to land from a drop jump.[180]

Wikstrom et al[181] assessed neuromuscular control differences between successful and failed jump landings. The muscles (vastus medialis, semimembranosis, lateral gastrocnemius, and TA) were activated earlier with greater amplitude pre- and postlanding during successful landings.[181] Activation patterns were proximal to distal with the vastus medialis activating first and the TA last.[181] Therefore, successful and failed landings use feedforward and feedback mechanisms, respectively. The authors suggested the failure of hip musculature may be the cause of a failed jump landing.[181]

Ankle Instability and Landing. Chronic ankle instability subjects have demonstrated altered biomechanics during landing and walking compared to healthy control subjects. Ankle dorsiflexion, inversion, knee flexion, and hip external rotation were altered in FAI subjects.[67, 68] Functional ankle instability subjects had greater dorsiflexion and knee flexion from 20 milliseconds (ms) prelanding to 60 ms postlanding during single-leg jumps. [67] These results indicate the differences between groups are not due to reflexive changes.[67] The authors suggested FAI and healthy subjects use different feedforward mechanisms during landing.[67] Less dorsiflexion was observed in FAI subjects postinitial contact (90 ms-200 ms).[68] Prior to initial contact, healthy subjects had greater hip external rotation compared to FAI subjects.[68] Functional ankle instability subjects are not as efficient at controlling ankle motion compared to healthy subjects.[68] They theorized neuromuscular impairments of FAI are not limited to the ankle, but will transmit up the kinetic chain to proximal joints and muscles.[68] Proximal joints and muscles may have an important role in maintaining postural control in FAI subjects, however, further research is needed.

Chronic ankle instability subjects demonstrated increased inversion during walking and a more lateral COP trajectory while running.[139, 182] Greater inversion was demonstrated by CAI subjects from 100 ms preheel strike to 200 ms postheel strike.[139] Chronic ankle instability subjects performed ankle inversion from 5 ms pre- and postheel strike while healthy subjects performed eversion.[139] Throughout the 200 ms postheel strike, CAI subjects exhibited an evertor moment while the control subjects demonstrated an invertor moment.[139] Chronic ankle instability subjects also had higher inversion angular velocity than controls at heel-strike.[139] Subjects with CAI demonstrated a more lateral COP trajectory than subjects with a history of one lateral ankle sprain.[182] Authors agree altered biomechanics place the ankle/foot in a position that will increase the likelihood of a CAI.

Differences in ground reaction forces (GRF) were observed between FAI subjects and healthy subjects. Peak anterior and lateral GRF occurred 10 to 13 ms earlier FAI than control subjects during a jump landing.[72] Time-averaged GRF was different between groups following initial contact.[72] Delahunt et al[68] observed increased vertical, medial, and posterior GRF postinitial contact. Functional ankle instability reached peak posterior GRF sooner than healthy subjects.[68] This suggests the entire kinetic chain may be affected by FAI.[68] Altered GRF is contributed to deficits in feedforward motor control.[72] These data suggested FAI subjects may have an altered feedforward mechanism.

Gender Differences. Biomechanical differences were observed between genders during dynamic tasks (walking, running, landing, and a single-leg squat). Females demonstrated an increased walking cadence and decreased stride length compared to males.[183, 184] Women displayed increased hip flexion and decreased knee extension during walking.[184] Their knees absorbed more energy while walking than males.[184] Greater oblique pelvic movement and less normalized vertical center of mass (COM) displacement was exhibited by women during walking.[183] Aging intensified oblique pelvic movement and COM displacement differences between genders.[183]

Female runners demonstrated increased peak hip adduction, internal rotation, and knee abduction angles.[185] Females absorbed more energy in the frontal and transverse planes than males.[185] Males and females use different lower extremity mechanics during running.[185]

Healthy men and women demonstrated different biomechanics during a single-leg squat.[186] Women demonstrated greater rectus femoris muscle activation and increased ankle dorsiflexion, pronation, hip adduction, flexion, and external rotation compared to males.[186] Greater knee valgus at the beginning and end of a single-leg squat were observed in females.[186] Gender differences were observed between both one and two-leg landings.[54, 99, 102, 187, 188] At initial contact during a two-leg landing, females had greater knee extension and plantar flexion angles than their male counterparts.[99] Females use their ankles and knees to absorb energy more than males and this could be why females are prone to injuries.[99, 186]

Females demonstrated less hip and knee flexion during a single-leg jump landing and they reached peak hip and knee flexion angles sooner than males.[102] The results of this study also demonstrated females use their ankles to absorb the majority of energy during a single-leg jump landing.[102] Decreased hip and knee flexion along with shorter time to peak angles may make females more prone to lower extremity injuries.

Russell et al[188]assessed knee angle and GMed activation differences between genders in healthy subjects during a single-leg drop landing. Females landed in knee valgus while males landed in varus at initial contact.[188] Males demonstrated greater knee varus than females at maximal knee flexion.[188] The authors concluded women had greater valgus stress placed on their knees than men and GMed activation was not different between genders.[188]

Jacobs et al[187] assessed peak torque, endurance capacity, and peak joint displacement of the hip and knee during a single-leg landing in healthy adults. Women demonstrated decreased peak torques and increased valgus knee peak joint displacement compared to their male counterparts.[187] The peak torque of women was correlated with hip flexion, adduction, and knee valgus displacement.[187] Altered biomechanics during single-leg landings may increase a women's risk of knee injury.[187] Hip abductor strength is thought to have an important role in neuromuscular control.[187]

Hart el al.[189] assessed lower extremity muscle activation in healthy subjects during a single-leg jump and landing. This study demonstrated that GMed muscle activation was greater in male division I collegiate soccer players than females.[189] No difference was observed in other lower extremity muscles (BF, vastus lateralis, and medial gastrocnemius).[189] Males use their hip muscles more to absorb energy during a single-leg landing than females.[189] This may make females more susceptible to lower extremity injuries than males. Table 10 summarizes the biomechanics studies discussed in this section.

Table 10. Summary of Biomechanics Research Studies

Authors/ Year	**Instrument(s)**	**Results**	**Conclusion**
Ankle Instability			
Caulfield & Garrett, 2004[72]	Jump landing GRF	Timing of GRF peaks varied between groups Peak lateral & anterior GRF occurred 10 to 13 ms earlier in FAI Time-averaged GRF differed between groups post-IC	Disordered force patterns in FAI subjects likely result in repeated injury due to additional stress on the ankle Disordered patterns most likely due to deficits in feedforward motor control
Caulfield & Garrett, 2001[67]	1-leg jumps Kinematics	FAI subjects had > dorsiflexion & exhibited > knee flexion than controls during 20 ms prior to 60 ms post landing	These timing differences indicate the results are not reflexive Feedforward programs are different between FAI & control subjects
Delahunt et al 2006[68]	SEMG (RF, PL, TA, SO) Kinematic Strain gauge	FAI ↓ PL activity pre-IC FAI ↑inversion pre-IC FAI ↓ dorsiflexion post-IC FAI ↓ angular velocity post-IC FAI ↓ hip external rotation pre-IC FAI ↑ vertical GRF (35-60 ms) medial GRF (85-105 ms), posterior GRF (75-90 ms) post-IC	FAI reached peak posterior GRF sooner than control FAI ↓ dorsiflexion FAI not as efficient as control @ reaching dorsiflexion Neuromuscular impairment are not confined to the ankle, but transmit up the kinetic chain
Monaghan et al, 2006[139]	Kinematic Kinetics	CAI subjects were more inverted from 100 ms pre-HS to 200 ms post-HS CAI invert during 5 ms pre-& post-HS, while healthy evert CAI exhibited an evertor moment while control invert during 200 ms post-HS CAI have higher angular velocity than controls @ HS	Δ in kinematics & kinetics are likely to result in ↑ stress being applied to ankle joint structures during HS & loading response phases of gait
Energy Absorption			
Decker et al, 2002[174]	Force plate Kinematics	ACLr subjects land with hip & knee more extended & ankle plantar flexed ACLr had > energy absorption from the knee & ankle than hip	Landing strategies are preselected & can be designed to mediate stresses to a specific joint
Devita & Skelly, 1992[69]	Kinetics Energetics	> joint flexion in preparation for soft landing (hip & knee 9° more, plantar flexed 5° less) Soft landings require > work (hip-54% & knee-46%) Muscles absorb more during a soft landing Ankle absorbs 14% more in stiff landings (plantar flexors absorb more energy)	Muscular system absorbed more energy during soft landings than stiff Ankle absorbs the most followed by knee then hip

Kulas et al, 2005[175]	Kinematics	Abdominal postures can be reliably performed during a 1-leg landing	Abdominal postures may have an influence on lower extremity energy absorption
Kulas et al, 2006[172]	Kinematics	Knee energy absorption during stabilization accounted for 55.1% Hip absorption @ stabilization was 8.3% Ankle absorption @ impact was 7.1%	Leg impedance ↑ from soft to preferred to stiff landing Leg impedance was primarily explained by knee energy absorption.
McNitt-Gray, 1993[101]	Kinematic Kinetics	Extensor moment ↑ with impact velocity ↑ Energy absorption ↑ with impact velocity ↑	Balance control is associated with ankle or hip adjustments Drop landing kinetic Δwith ↑ in impact velocity Gymnasts & recreational athletes dissipate energy differently
Self & Paine, 2001[190]	Kinematics	Stiff landings had the highest peak vertical forces & accelerations Achilles tendon peak force highest @ stiff plantar flexed landings	Athletes may not use full energy absorbing potential during sporting events
Zhang et al, 1998[98]	Kinematics Kinetics	GRF ↑as landing height ↑ Peak hip moment & power were later than the ankle & knee Peak hip moment & power were > than ankle & knee Ankle muscles ↑ work with ↑ heights ↑ landing stiffness = ↑ ankle muscle contribution & ↓ hip contribution Hip & knee ROM ↓ with stiff landings	↑ height, ↑ biomechanical responses Ankle is less capable of energy absorption than hip & knee Knee & hip involvement Δwith landing strategy Hip & knee energy absorption ↑ as mechanical demands ↑ Shift from ankle to hip strategy as landing height ↑
Perturbation			
Farrokhi et al, 2008[179]	Kinematics Kinetics	LTE dorsiflexion ≥ NL & LTF LTE knee extensor impulse ≥ LTF LTE plantar-flexor impulse > during LTF Peak LTE knee flexion angle ≥ LTF Peak LTE hip flexion angle ≤ NL LTF hip flexion angle ≥ NL & LTE LTF hip extensor impulse ≥ NL & LTE	The location of the trunk during a forward lunge dictates muscle involvement LTF ↑ hip extensor involvement > GMax & biceps femoris activation for LTF
Henry et al, 1998[191]	SEMG Force plate Motion analysis	Anterior translation has a 2-stage pattern (ankle & hip displaced, then return to neutral) Lateral & posterior translation 3-stage pattern (hip & ankle displaced, hip angle returns, & ankle & hip return back to initial position	Control of COM requires ankles & hips TFL was 1st activated w/ lateral translation Distal muscles recruited 1st w/ A/P translations

Riemann et al, 2003[176]	Kinematic	Difference between joints for FIEC (ankle>hip>knee>trunk), FOEO (ankle>knee & hip>trunk), MAEO (ankle>knee) Within joint differences: ankle(FIEC>FOEO>MAEO>FIEO), knee(FIEC>FOEO> FIEO & MAEO), hip(FIEC>FOEO> FIEO & MAEO), & trunk(FIEC> FOEO & MAEO>FIEO)	FIEC required>correction FIEO required<correction As task became more challenging ↑ reliance on proximal joints Task rank FIEO<MAEO<FOEO<FIEC
Rietdyk et al, 1999[177]	Kinematics COP	Hip joint movement & moment occurred 1st followed by ankle Contralateral hip 1st active with shoulder perturbation Ipsilateral hip 1st moment & angle to become active for pelvis perturbation	Trunk movement was dependent upon perturbation location Many subjects overshoot in the opposite direction before returning to the stationary position CNS initiates response before perturbation is fully developed Perturbation location dictates response not magnitude
Muscle Activation			
Farrokhi et al, 2008[179]	SEMG	LTF ↑ GMax & biceps femoris EMG LTE ↑ dorsiflexion angle & ↓ peak hip flexion angle	LTF ↑ hip extensor impulse & recruitment LTE did not alter LE joint impulse or activation
Konradsen et al, 1997[132]	SEMG 2-D kinematics	Peroneal reflex latency median was 48 ms Knee flexion reached median of 30° Median hip flexion was 25° A uniform reaction pattern exists for both unilateral & contralateral limbs	Dynamic response to sudden inversion involves both peripheral reaction & a central mediated strategy Only anticipated muscle activity, static stabilizer strength, or external support can prevent an injury
Runge et al, 1999[171]	SEMG kinematics	Activation ↑ as velocity ↑ Anterior muscle activation ↑ (RA, STER, RF) as backward translation is reached Knee flexion & peak hip flexion ↑ as velocity ↑ Velocity threshold of hip torques emerged the same time as RA EMG	Ankle & Hip strategies are mixed as velocity increases EMG patterns during fast translations are indicative of combined ankle & hip action
Viitasalo et al, 1998[180]	SEMG Goniometer	Triple jumpers 32% higher @ .40 m & 34% @ .80 m VL & gastrocnemius had earlier preactivity than controls EMG did not differ b/w drop heights DJ80 had > angles than DJ40	Jumpers have more efficient neuromuscular system than controls Jumpers better able to resist > speeds & GRF
Wikstrom et al, 2008[181]	SEMG 1-leg jump	Greater activation times, preparatory & reactive EMG Successful landings muscle activation times (VM, SM, LG, TA), preparatory EMG (VM, SM, LG), reactive EMG (VM, SM, LG, TA)	Successful jump landing trials had earlier activation & reactive EMG Activation patterns were proximal to distal Preparatory activation plays a > role Failed trials could be caused by hip musculature failure

Gender			
Decker et al, 2003[99]	Kinematics Kinetics	Females had > knee extension & plantar-flexion angles @ IC Females have > peak angular velocities Females used their ankle & knee to absorb energy more than males	Knee is primary energy absorbing joint Females absorb more energy with their ankle & knee Supports why females are more prone to ACL injuries Males reached peak knee extensor moment sooner than females Males had > hip power than females
Ferber et al, 2003[185]	Kinematics	Female runners > peak hip adduction, internal rotation, & knee abduction > hip frontal & transverse plane negative work > hip frontal plane (-) work, & peak hip adduction velocity Females > knee abduction angle	Females exhibit different lower extremity mechanics in the frontal & transverse planes @ the hip & knee compared to men
Hart et al, 2007[189]	SEMG Kinematics	GMed activation < in Div. I soccer athletes (2.62 to 7.17) Males > GMed activation than females	Gender specific force absorbing strategies while landing arise from hip muscles Neuromuscular strategies to attenuate the forces of 1-leg landings may involve more hip activity in males than females
Jacobs et al, 2007[187]	PT %E PJD	Women lower PT than males Women PT correlated with hip flexion, adduction, & knee valgus PJD during landing	No gender difference in %E Hip abductor strength may play an important role in neuromuscular control of the women's knee
Kerrigan et al, 1998[184, 188]	Kinematics Kinetics	Female cadence is ≥ males Stride length is < in females > peak knee absorption in females	Females had > hip flexion & less knee extension before initial contact
Russell et al, 2006[155]	Force plate SEMG Kinematics	Women had > valgus @ IC & remained in more valgus than males during 1-leg drop landing At MKF, men (15.26°) had > varus (3.13°) than females GMed activation > at MKF than IC	Women land in greater knee valgus than men, GMed does not differ between sexes in healthy subjects Timing of GM activation is of > importance than level of activation GMed activation did not differ between sexes
Schmitz et al, 2007[102]	Kinematics Kinetics	Females less hip (60%) & knee (36%) ROM than males during landing Females shorter time to peak hip (52%) & knee (22%) flexion Females 9% > peak normalized vertical GRF	Females absorb > energy @ the ankle than males Females use less total hip & knee flexion & have shorter peak flexion values during 1-leg drop landing
Smith et al, 2002[183]	COM Pelvic obliquity, rotation, tilt	Women had > cadences, and shorter stride length Woman had > pelvic obliquity & lower normalized vertical COM	No gender differences in walking velocity Aging intensifies gender differences

Zeller et al, 2003[186]	SEMG Kinematics 1-leg squat	Women ↑ankle dorsiflexion, pronation, hip adduction, flexion, external rotation, & ↓ trunk lateral flexion Women start & end in more valgus Women activated their rectus femoris more than males	Women place their lower extremity & activate their muscles in a way that may ↑ the risk of an ACL injury

%E- endurance capacity; ACLr – anterior cruciate ligament reconstruction; COM-center of mass; FIEC – firm surface, eyes closed; FIEO – firm surface, eyes open; FOEO – foam surface, eyes open; GMed- gluteus medius; GRF- ground reaction force; HS- heel strike; IC – initial contact; MAEO – multiaxial, eyes open; MKF- maximal knee flexion; ms – milliseconds; PJD- peak joint displacement; PT- peak torque; ROM- range of motion; SEMG- surface electromyography

PROXIMAL ANATOMICAL STRUCTURES AND LOWER EXTREMITY INJURY

Hip Musculature

The relationship between proximal anatomical structures and lower extremity injury is not fully understood.[192] Nicholas et al[103] assessed the strength of five lower extremity muscle groups (quadriceps, hamstrings, hip abductors, adductors, and hip flexors) in subjects with different lower extremity pathology (ankle and foot problems, ligamentous instability, patellar lesions, intrarticular defects, arthritis, and back). Different lower extremity pathology was associated with specific muscle weakness.[103] Subjects with ankle and foot problems demonstrated a strong correlation between ipsilateral hip abductor and adductor weakness.[103] Ipsilateral quadriceps weakness was related to knee ligament laxity.[103] Patellar lesion subjects displayed the greatest overall ipsilateral weakness; the quadriceps, hamstrings, and hip flexors all had weakness.[103] The subjects with intraarticular defects and back groups demonstrated ipsilateral quadriceps weakness.[103]

The relationship between hip strength and patellofemoral pain syndrome was assessed, subjects demonstrated decreased hip abductor and external rotator strength compared to a healthy control group.[192] Although strength differences were observed further research is required to understand the relationship between hip weakness and patellofemoral pain syndrome.[192]

Researchers observed hip strength differences in subjects with a lower extremity or low back injury.[41, 104, 193] Females demonstrated side-to-side hip muscle weakness in subjects with lower extremity injuries.[104, 193] Right hip abductor and left hip extensor muscles were stronger than the opposing limb.[104] The muscles of intercollegiate athletes with a history of a lower extremity injury displayed decreased strength on the left compared to the right and hip extensor muscles were weaker than the abductors.[193] A distal injury may affect neuromuscular control and foster compensatory strategies.[193]

Nadler et al[194] assessed functional performance of subjects with a history of lower extremity injury using the 20-meter shuttle run during preparticipation physicals. Freshman with a history of lower extremity injury were slower than those without a history of injury.[194] Female subjects were slower on average and the nonfreshman athletes were faster than the freshman, but no difference was observed between nonfreshman regardless of injury history.[194] The authors concluded kinetic chain deficits may last long after recovery and hip muscles are important at transferring forces up the kinetic chain.[194] They also theorized core strengthening may improve shuttle run times.[194]

Ankle Instability. Individuals with CAI demonstrated altered hip muscle activation. The gluteal muscles were the primary hip muscles previously assessed in CAI subjects. Subjects with CAI activated their GMed muscle later than healthy control subjects during right or left ankle perturbations.[28] The contralateral GMed muscle was activated prior to the ipsilateral GMed in both groups (ankle instability & healthy).[28] Greater pelvic displacement was measured in one subject on the perturbation side, therefore, a greater stretch was placed on the ipsilateral side compared to the contralateral side.[28] Chronic ankle instability subjects altered their GMed muscle activation as a compensatory strategy in an attempt to maintain postural control. This response was probably due to a polysynaptic reflex instead of a supraspinal signal.[28]

Chronic ankle instability subjects demonstrated hip abductor weakness on the injured side. A correlation was observed between hip abductor and extensor strength.[156] This study suggested that both the GMed and GMax may be affected by CAI.[156] The authors suggested that comprehensive ankle rehabilitation programs should include hip strengthening.[156]

Subjects that suffered an ankle sprain demonstrated altered GMax muscle activation. The GMax activated later than healthy subjects during a prone-leg extension.[29, 31] A correlation between muscle function and sensory changes was observed in subjects with a history of unilateral ankle sprains.[31] It was concluded that changes in local sensation and proximal muscle function are associated with ankle sprains;[31] therefore, the authors suggest that ankle rehabilitation protocols should be holistic in nature instead of focusing on a specific location such as the lower leg.[31] Altered muscle (contra-lateral & ipsilateral erector spinae, GMax, and biceps femoris) activation patterns have been observed between healthy subjects and those with a history of ankle sprains.[29] The GMed muscle activated later and for a shorter amount of time in subjects that previously sprained their ankle compared to the healthy control group.[29]

Abdominal Musculature. It has been theorized that the core musculature may have an important role in the prevention of lower extremity injuries.[37, 38, 105, 195] As previously discussed, recent research has focused on the relationship between lower extremity injuries and the hip musculature. However, research has not directly assessed the relationship between the remaining core muscles and lower extremity injuries. Therefore, further research is required to understand the behavior of the remaining core musculature.[37, 38, 105, 195] The abdominal muscles are of particular interest because these muscles stabilize the spine during activity along with creating trunk motion.

A study assessed the differences in core stability between genders and history of injury. Core stability was measured in 140 collegiate basketball players (80 females and 60 males) over a two-year period.[38] The researchers used a handheld dynamometer to assess isometric hip abduction and external rotation strength.[38] They also measured core stability using the lumbar extensor endurance, side bridge, and straight-leg-lowering test.[38] Although these are commonly used measures to assess core stability all but the straight-leg-lowering test involve an isometric contraction. Therefore, these may not be the best measures to assess core stability. A gender difference was observed; males demonstrated greater core stability than females.[38] Those subjects who suffered a lower extremity injury also demonstrated less core stability compared to the healthy subjects.[38] The authors concluded core stability is important to prevent lower extremity injury, especially in females.[38] Table 11 summarizes the proximal anatomical structures and ankle instability studies discussed in this section.

Table 11. Summary of Proximal Anatomical Structures and Ankle Instability

Authors, Year	Instrument(s)	Results	Conclusion
Ortiz, et al, 2006[105]	Literature Review	Defines the core Provides training ideas	Core stabilization & strengthening programs are thought to promote ↑ lumbo-pelvic-hip stability & ↑ neuromuscular recruitment This is thought to ↓low back & lower extremity injuries
Willson, et al, 2005[37]	Literature Review	Core stability maintains low back health & prevents knee injuries Defines core stability	Lower extremity injuries may diminish core stability
Beckman & Buchanan, 1995[28]	EMG latency	GMed activates sooner in FAI than healthy during same side perturbation Contralateral GMed activates before ipsilateral	FAI GMed activates prior to healthy Contralateral GMed activates first followed by ipsilateral
Bolga, et al, 2008[192]	Strength Kinematics	PFPS subjects have ↓ strength (hip external rotation & abductor torque) No difference in hip and knee angles	Additional research is required to understand the relationship between hip weakness & PFPS
Bullock-Saxton, 1994[31]	SEMG	Difference of vibration @ 3 frequencies in injured ankle > vibration was required by the ankle injury group GMax onset later in ankle injury group Earlier onset of hams & GMax in healthy subjects	Local sensory and proximal muscle function Δ associated ankle sprains Correlation between sensory and muscle function Holistic approach is recommended
Bullock-Saxton, 1995[30]	SEMG	Injured group activation pattern different than healthy GMax activation was delayed Activation time was < for injured than healthy	Altered afferent input from ankle injury may influence CNS motor plan
Friel, et al, 2006[156]	Goniometer Handheld dynamometer	Hip abductors weaker on involved side Plantar-flexion ROM < on injured side Hip abductor & extensors correlated	CAI subjects have weaker hip abductors
Leetun, et al, 2004[38]	Isometric hip abduction, external rotation LET Side bridge test SLLT	Males demonstrated > core stability than females Athletes who suffered injuries generally demonstrated lower core stability	Females displayed decreased hip external rotation & side bridge compared to males Highlights the importance of proximal stabilization for lower extremity injury prevention
Nadler, et al, 2000[104]	Dynamometer	L extensor group stronger than R (females w/o injury were 10.9% stronger) R abductors were stronger than L	Females demonstrated side-to-side hip strength differences Athletes with previous LE injury or LBP were found to have differences in hip strength as compared with athletes w/o

		Males had no side-to-side differences	injury
Nadler, et al, 2002[193]	Dynamometer	Difference in ratio of max LA/LE in athletes with LE injury No difference b/w max RA/RE	More strength dysfunction on L as compared to R hip in athletes with LE injury Greater torque on L Hip extensors appeared weaker Reflects distal injury may affect muscle weakness, firing patterns, central inhibition, & compensatory strategy
Nadler, et al 2002[194]	20 m shuttle run	Freshman w/history of LE injury had slower shuttle runs No difference in nonfreshman regardless of injury	Kinetic chain deficits may last long after symptomatic recovery Hip musculature plays a role in transferring forces from the LE up towards the spine Core strength may improve shuttle run times
Nadler, et al, 2002[41]	Dynamometer	No Δ in LBP occurrence R Δ hip extensor stronger than L on average (*P* =.0001) Females w/weaker L hip abductors had > chance of LBP	Program Δ hip extensor strength Need exists for gender specific programs Weak hip abductors cause increased trunk involvement Hip abductors help maintain stability in midstance
Nicholas, et al, 1976[103]	Manual muscle tests Cybex II	Strong correlation between ankle & foot problems & ipsilateral hip abductors & adductors	Specific weaknesses found with certain conditions Injured leg weaker than control LE injuries may affect remote
Zazulak, et al, 2007[196]	APR & PPR	3 year prospective study Interaction between gender & knee injuries APR deficits observed in female subjects compare to control No difference in PPR 2.9-fold ↑ in knee injury (*P* = .005), 3.3 ↑ in ligament/ meniscus injury (*P* = .007)	Lends credence to association between ↓ neuromuscular control of body's core & ↑ knee injury risk Healthy females had better APR than males ↓ active core proprioception predicted knee injury risk in females

GMed – gluteus medius; L – left, , LE- lower extremity, LBP – low back pain, w/o – without, LET - Lumbar Endurance test, PFPS- patellofemoral pain syndrome, R – right, SLLT - Straight leg lowering test

CENTRAL NERVOUS SYSTEM

Brain

The brain is composed of six major divisions. These divisions are the cerebrum, diencephalon, midbrain, cerebellum, pons, and medulla oblongata.[66] The cerebrum is divided into two cerebral hemispheres which contain three sections (cerebral cortex, white matter, and basal ganglia).[197] Layers of neuron cell bodies make up the cerebral cortex. White matter is composed of myelinated axons that serve as the pathway by which the cerebral cortex communicates with the rest of the central nervous system.[197] The diencephalon is composed of the thalamus and hypothalamus.[66] The thalamus serves as the relay station for sensory information provided to the cerebral cortex.[66, 197] Hypothalamus is the homeostasis center and is controlled primarily by the autonomic nervous system.[66] The midbrain is part of the brainstem and controls eye movements, and relays auditory and visual reflexes.[197] Ascending and descending pathways cross through the midbrain to and from the forebrain.[197] The cerebellum is posterior to the brainstem and coordinates movement and balance.[197] Another part of the brainstem is the Pons; it is the transfer station between the cerebellum and cerebrum.[197] The final part of the brain stem is the medulla oblongata. It transfers information from the spinal cord to the rest of the brain. Descending fibers of the medulla oblongata are associated with motor function while ascending fibers are sensory in nature.[197]

Spinal cord

The spinal cord is the link between the peripheral nervous system and the brain. It contains sensory and motor neurons involved with reflexes, ascending, and descending pathways.[197] Each vertebra contains gray matter, unmyelinated cell bodies, with a dorsal and ventral nerve root.[66] Signals from skeletal muscles to the spine enter through the vertebra's dorsal nerve root while the ventral root sends information from the central nervous system (CNS) to muscles.[66] Information coming from the muscles or joints is referred to as feedback (afferent) and travel to the spine via sensory fibers.[66, 197]

Feedback

Within the joints and muscles of the body there are many different receptors that supply feedback to the nervous system. Muscle spindles signal change in muscle length and rate of change.[197] Spindles are long encapsulated structures that contain intrafusal muscle fibers.[66, 197] There are two types of intrafusal fibers: nuclear bag and nuclear chain. Nuclear bag fibers are further divided into dynamic and static bag fibers.[197] The nuclear bag fibers are swollen and clustered centrally.[197] Dynamic bag fibers are sensitive to the rate of change in muscle length while static bag fibers are sensitive only to change in muscle length. Nuclear chain fibers are not swollen and form a line/row, they are sensitive to changes in muscle length.[197] Two types of sensory fibers are associated with intrafusal fibers: Type Ia and II.[197] Type Ia fibers are associated with nuclear bag fibers while type II fibers are associated with nuclear chain fibers.[197] Type II fibers react to the muscle spindle being stretched but do not respond to the rate of being stretched.[197] Gamma MNs (γ-MN) signal the peripheral nervous system when the central region of nuclear bag and chain fibers are stretched.[197] Dynamic and static γ-MN maintain spindle sensitivity and length, respectively.[197] The frequency of action potentials sent along the type Ia fibers increase as intrafusal fibers are stretched.[197]

Golgi tendon organs (GTOs) are another sensory receptor located in muscle fibers. They are located in the musculotendinous junction and are composed of free nerve endings.[197] Type Ib sensory fibers relay information from the GTOs to the peripheral nervous system. Goli tendon organs are slow to fire and accommodate and regulate muscle tension. A signal sent from the GTOs to the spine will inhibit alpha MNs (α-MN).[197]

Other sensory receptors sometimes referred to as joint receptors are the ruffini corpuscles, pacinian corpuscles, and nociceptors. Ruffini corpuscles relay sensory information regarding joint position and displacement, angular velocity, and intra-articular pressure.[73] Pacinian corpuscles monitor mechanical stress and detect joint acceleration.[73] Nociceptors also known as free-nerve endings inform the nervous system when a joint is placed under abnormal stress or there is pain.[73]

Feedforward

Feedforward (efferent) refers to the signals that originate in the CNS and transcend to extremity musculature. Efferent signals communicate with the motor system through spinal tracts. The medial spinal tracts are responsible for transferring information regarding postural and gross motor movement.[197]

There are five medial spinal tracts: medial corticospinal, tectospinal, medial reticulospinal, medial vestibulospinal, and lateral vestibulospinal. The medial corticospinal tract originates in the cortex and descends bilaterally through the thoracic vertebrae to affect the shoulder, neck, and trunk muscles.[197] The tectospinal pathway begins in the brain stem (messencephalon) and controls eye and head movement. The medial reticulospinal tract controls postural and limb extensor muscles and begins in the Pons. The medial vestibulospinal tract originates at the medulla oblongata and affects upper back and neck muscles. The lateral vestibulospinal tracts descend ipsilaterally the full length of the spine from the brainstem to facilitate extensor muscles while inhibiting flexors.[197] This tract's function is to control balance and posture.[197]

Lateral spinal tracts assist with motor function by affecting distal limb movements. The lateral corticospinal tract is the primary motor control pathway. It begins in the cortex. The rubrospinal tract begins in the midbrain and controls arm, but not leg motions. The lateral reticulospinal is another tract that originates in the medulla oblongata. It connects directly to γ-MN and affects posture.

CONTEMPORARY THEORY: ANKLE INSTABILITY AND SOMATEOSENSORY DEFICITS

For years researchers have theorized ankle instability was due to joint deafferentation which solely affected the feedback system. Freeman[1-3] proposed this theory in the 1960s. Although many still believe in Freeman's theory a more recent theory has been suggested. The contemporary theory is a more comprehensive theory that includes both feedforward and feedback mechanisms.[36] Since recent research has demonstrated ankle instability, subjects have altered sensorimotor control or function in proximal musculature, the contemporary theory may be more accurate. Further research is required to support this theory.

CORE STABILITY

Core stability has become a frequently used term by allied health care professionals, strength and conditioning coaches, and those in fitness professions. Two problems exist: 1) the core has not been clearly defined, and 2) professionals have used terms interchangeably (core stability, core strength, trunk stabilization, lumbar stabilization) without clear definitions. King[198] defined the "core" as a cylinder that extends inferiorly from the superior rib cage to the inferior aspect of the pelvis. Others included the spine, pelvis, proximal lower extremity, and abdominal structures as part of the core.[195] Akuthota and Nadler[199] defined the superior portion of the core as the diaphragm, pelvic girdle inferiorly, the abdominal muscles anteriorly, and the paraspinal and gluteal muscles posteriorly.

Core stability is the ability to control the position and motion of the trunk over the pelvis during physical activity.[195] Willson et al[37] defined core stability as, "the ability of the lumbopelvic-hip complex to prevent buckling of the vertebral column and return it to equilibrium following a perturbation." It was suggested the variations in core and core stability definitions are due to the complexity of this region.[200] The muscles of the core are thought to work in conjunction with each other to provide stability to the spine, trunk, and the extremities during dynamic and static movements. It remains unclear how all of these muscles work together to achieve stability.

Musculature

Over 45 different muscles are included in our definition of the core; they function as stabilizers of the spine and pelvis or assist with the movement of the thigh, trunk, or upper extremity. These muscles include the latissimus dorsi, hamstrings, quadriceps, hip abductors, hip flexors, hip external rotators, gluteal, paraspinal, and abdominal muscles or groups. TABLES 12-14 indicate each muscle's origin, insertion, innervation, and function.

Every researcher included the abdominal muscles as part of the core. Bergmark[201] separated abdominal and back muscles into two groups, called local and global systems. The function of muscles in the local system is to stabilize the spine during movement.[201] Local system muscles (multifidi, interspinal, intertransversii, medial quadratus lumborum, and TrA) originate or insert onto a vertebrae according to Bergmark.[201] The psoas is the primary hip flexor during non-weight bearing and stabilizes the spine during weight bearing activities.[201]

The global system muscles (erector spinae, EO and IO, RA, quadratus lumborum, psoas, latissimus dorsi) reduce the force transferred to the lumbar spine and local system.[201] These muscles also serve as primary movers to change pelvis, trunk, and limb position during movement.[201]

Table 12. Thigh Musculature

Hamstrings				
Muscles	**Origin**	**Insertion**	**Innervation**	**Function**
Semitendinosus	ischial tuberosity	medial flare of tibia	Sciatic (tibial), L4-S2	knee flexion, extends & medial rotation hip
Semimembranosus	ischial tuberosity	medial flare of tibia	Sciatic (tibial), L4-S3	knee flexion, extends & medial rotation hip
Biceps femoris	*Long head*:sacrotuberous ligament, ischial tuberosity *Short head*: linea aspera, proximal 2/3 supracondylar line	fibular head, lateral tibia condyle	*Long:* Sciatic (tibial), L5-S3, *Short:* Sciatic (peroneal), L5-S2	knee flexion, lateral rotation, long assists w/ hip lateral rotation
Quadriceps				
Vastus lateralis	intertrochanteric line, greater trochanter	proximal border of patella, tibial tuberosity	Femoral, L2-4	knee extension
Vastus intermedius	proximal 2/3 of femur, distal linea aspera	proximal border of patella, tibial tuberosity	Femoral, L2-5	knee extension
Vastus medialis	distal 1/2 of intertrochanteric line	proximal border of patella, tibial tuberosity	Femoral, L2-6	knee extension
Hip Flexors				
Rectus femoris	ASIS, above acetabulum	proximal border of patella, tibial tuberosity	Femoral, L2-7	extend knee, flexes hip
Psoas major	Ventral T12-L5 transverse processes	lesser trochanter	Lumbar plexus, L1-4	hip flexion
Psoas minor	T12-L1 vertebrae	iliopectineal eminence, arcuate line	Lumbar plexus, L1-2	hip flexion
Iliacus	iliac fossa, iliac crest, sacroiliac ligaments, sacrum	lesser trochanter	Femoral, L1-4	hip flexion
Sartorius	ASIS	medial flare of tibia	Femoral, L2-4	flex, lateral rotate, & abduct hip, flex & medial rotate knee
Tensor fascia latae	iliac crest, ASIS	IT band	Superior gluteal, L4-S1	flex, medial rotate, & abduct hip, knee extension
Hip Adductors				
Pectineus	pubic tubercle	pectineal line of femur	Femoral & Obturator, L2-4	hip adduction
Adductor magnus	pubic ramus, ischial ramus, & ischial tuberosity	medial gluteal tuberosity, adductor tubercle	Obturator, L2-4 & sciatic L4-S1	hip adduction

Gracilis	symphasis pubis, pubic bone	medial flare of tibia,	Obturator, L2-4	hip adduction
Adductor brevis	inferior pubic ramus	pectineal line & linea aspera of femur	Obturator, L2-4	hip adduction
Adductor longus	pubic crest/symphasis	linea aspera	Obturator, L2-4	hip adduction
Hip Lateral Rotators				
Piriformis	S1-S4, sacrotuberous ligament	greater trochanter	Sacral plexus, L5-S2	lateral rotation
Quadratus femoris	ischial tuberosity	quadrate line, intertrochanteric crest	Sacral plexus, L4-S2	lateral rotation
Obturator internus	posterior pelvis (obturator foramen)	greater trochanter	Sacral plexus, L5-S2	lateral rotation
Obturator externus	pubis & ischium	trochanteric fossa	Obturator, L3-4	lateral rotation
Gemellus superior	ischial spine	obturator internus tendon, greater trochanter	Sacral plexus, L5-S2	lateral rotation
Gemellus inferior	ischial tuberosity	obturator internus tendon, greater trochanter	Sacral plexus, L4-S2	lateral rotation
Gluteals				
Gluteus minimus	ilium b/w gluteal lines	greater trochanter	Superior gluteal, L4-S1	hip abduction, medial rotation, flexion
Gluteus medius	ilium b/w gluteal lines	greater trochanter	Superior gluteal, L4-S1	hip abduction, medial rotation, flexion
Gluteus maximus	posterior gluteal line, sacrum, coccyx, sacrotuberous ligament	IT band, gluteal tuberosity	Inferior gluteal, L5-S2	hip extension, lateral rotation, adduction/abduction

Table 13. Back and Shoulder Musculature

Back Muscles				
Muscles	**Origin**	**Insertion**	**Innervation**	**Function**
Iliocostalis lumborum	sacral medial crest, T11-L5 spinous process, iliac crest, supraspinous lig.,sacral lateral crest	inferior angle of lower 6 or 7 ribs	Spinal	Extension, draws ribs down
Longissimus thoracis	lumbar transverse process, anterior thoracolumbar fascia	all thoracic transverse processes, lower 9-10 ribs	Spinal	Extension, lateral flexion, ribs downward
Spinalis thoracis	T11-12, L1-2 spinous processes	T1-8 spinous processes	Spinal	Extension
Multifidi	*Sacral region*: posterior sacrum, posterior iliac spine, posterior-sacroiliac ligaments	spans 2-4 vertebrae above last into spinous processes	Spinal	Extension, Rotation
Rotares	vertebrae transverse processes	spinous process of above vertebrae	Spinal	Extension, Rotation
Interspinales	pairs between spinous process		Spinal	Extension
Intertransversarii ant. & post.	between transverse processes		Spinal	Lateral flexion
Quadratus lumborum	Iliac crest, iliolumbar ligament	Last rib, lumbar transverse processes	Spinal	Alone, lateral flexion of vertebral column; Together, depression of thoracic rib cage
Latissimus dorsi	T6-12 spinous process, ribs 8-12, thoracolumbar fascia	intertubercle groove	thoracodorsal, C6-8	medial rotation, adduction, extension, assists w/ anterior/lateral pelvis tilt

Table 14. Abdominal Musculature

Muscles	**Origin**	**Insertion**	**Fiber Direction**	**Innervation**	**Function**
Rectus abdominis	Pubic crest and symphasis	costal cartilages of the fifth -7th rib and xiphoid process	vertical	T5-T12, ventral rami	trunk flexion
External oblique					
Anterior fibers	5-8 ribs, serratus anterior	linea alba	oblique downward and medially	T5-T13	*Bilateral*:flexion, compression *Unilateral*:rotation
Lateral fibers	9-12 ribs	mesh w/ serratus anterior & latissimus dorsi	oblique downward and medially:downward anteriorly	T5-T12	*Bilateral*:flexion *Unilateral*:rotation
Internal oblique					
Lower anterior	lateral 2/3 of inguinal ligament, iliac crest	pubic crest, pectineal line, linea alba	transversely across lower abs	T7-L1, iliohypogastric, ilioinguinal, ventral rami	compress & support lower abdominal viscera w/ TrA
Upper anterior	anterior 1/3 of iliac crest	linea alba	obliquely medially and upward	T7-L1, iliohypogastric, ilioinguinal, ventral rami	*Bilateral*:flexion & compress vicera *Unilateral*:rotate vertebrae
Lateral	middle 1/3 of iliac crest & thoracolumbar fascia	10-12 ribs, linea alba	obliquely upward and medially but more upward than anterior fibers	T7-L1, iliohypogastric, ilioinguinal, ventral rami	*Bilateral*:flexion *Unilateral*:rotation
Transverse abdominis	ribs 6-12;thoracolumbar fascia; iliac crest; lateral 1/3 inguinal ligament	linea alba, pubic crest, pecten pubis	transverse	T7-L1, iliohypogastric, ilioinguinal, ventral rami	flattens ab wall & compress viscera, upper assists w/ breathing(expiration)
Diaphragm					
Sternal part	xiphoid process, costal part: costal cartilages ribs 6-12, TrA,	central tendon		phrenic, C3-5	separates thoracic and abdominal cavities, primary respiration muscle (inspiration -contract, exhalation- relax)
Lumbar part	lumbar vertebrae, lateral arcuate ligament from vertebrae to transverse processes , and 12th rib	central tendon		phrenic, C3-6	separates thoracic and abdominal cavitities, primary respiration muscle (inspiration -contract, exhalation- relax)

REHABILITATION PROGRAMS

Ankle Rehabilitation

The focus of ankle rehabilitation programs have been to improve strength and balance following an ankle sprain. Functional ankle instability subjects demonstrated improvements in joint position sense, peak torque, Single-Leg Stance Test, and other functional measures following strength training programs.[124, 202] Subjects performed strengthening exercises 3 times a week for 6 weeks. [124, 202] Docherty et al[124] used rubber tubing exercises while Sekir et al[202] used an isokinetic training device.

Other six-week ankle-strength training performed on FAI subjects did not prove to be beneficial.[203, 204] Kaminski et al[204] assigned FAI subjects to four training groups (strength, proprioception, strength and proprioception, and control) and assessed strength using peak torque and peak-torque ratios after the training program. Isokinetic strength, muscle fatigue, and single-leg balance were not effected by the training program.[204]

Subjects with acute ankle sprains and CAI demonstrated improvements in balance, coordination, and postural sway following a balance training program.[16, 19, 205-211] Many different types of balance training programs were used in research. Balance training using an ankle disk was assessed over 6 and 10 week periods.[159, 205, 206] Ankle disc training decreased postural sway and FAI, therefore, reducing the chance of CAI.[159, 206] Displacement of the hip was decreased following six weeks of ankle disc training.[205] The postural improvements observed after ankle disc training, stress the importance of central programming.[205]

Balance board training programs were effective at reducing ankle sprains and FAI.[209, 210] Although the training program reduced the risk of CAI, it did, however, increase the risk of a knee injury in subjects with a history of an overuse knee injury.[209] Residual effects (painless walking, running, and edema) of an acute ankle sprain were not affected by the training program.[210] Other balance training programs used the Biodex Balance System (Biodex Inc., Shirley, NY) for training and assessment.[207, 208] A four-week balance-training program was an effective way to reduce sway and improve balance index scores.[207, 208]

Subjects that participate in balance training are less likely to injure their ankle.[16] The risk of an ankle sprain in basketball and soccer players decreased following a training program which included closed chain exercises.[16] Center of pressure excursion was decreased and reach distances of CAI subjects were increased following balance training.[19] Holme et al[211] observed JPS, postural sway, and strength deficits six weeks postacute ankle sprain, no differences were observed four months post-injury.[211] It was concluded that supervised rehabilitation may decrease CAI.[211] Rasool and George[212] observed that SEBT reach distance increased after two and four weeks of a single-leg dynamic balance exercise program in healthy subjects, the researchers suggested that improved postural control may be due to central processing.[212]

Abdominal Strengthening Exercises

Abdominal muscles are thought to play an important role in stabilizing the spine and pelvis.[213] Abdominal hollowing, curl-up, pelvic-tilt, and sit-up are some of the exercises that are commonly used to strengthen the abdominal muscles. Additional equipment such as Swiss balls and foam rollers are frequently used to provide variation within a program and increase the level of difficulty.

There is no one strengthening exercise that will strengthen all of the abdominal and core muscles.[214] Therefore, it is important for clinicians and researchers to use multiple exercises with their patients or subjects to improve stability, balance, proprioception, and motor control.[214] To do this clinicians and researchers need to know what muscles are contracted during specific strengthening exercises.

Abdominal hollowing exercises have demonstrated preferential activation of the TrA muscle.[55, 215] The TrA/IO was voluntarily recruited prior to other abdominal muscles during AH or bracing exercises.[215] Internal oblique muscle activation was increased during AH while EO and RA muscle activation remained unchanged or decreased.[216, 217] These studies did not assess TrA muscle activation.

Researchers assessed the role of AH during landing and its effect on lower extremity muscle activation.[74, 172, 175] Abdominal hollowing decreased anterior tilt of the pelvis and increased GMax and medial hamstring muscle activation during prone hip extension.[74] Contraction of the TrA may increase muscle activation in muscles distal to the trunk, however, erector spinae muscle activation decreased during AH.[74] Further research is needed to assess how a voluntary contraction the TrA effects lower extremity muscle activation.

Kulas et al[175] assessed leg spring stiffness and relative energy absorption during three different abdominal postures (control, AH, pelvic-tilt). Subjects were able to reliably maintain these postures during a single-leg landing.[175] Males activated their TrA/IO before the RA and IO and produced greater TrA/IO muscle activation than females while landing on both feet.[218] The TrA/IO was activated more in males than females, prior to landing, however, females demonstrated greater TrA/IO activation following landing than males.[218] These studies suggest abdominal posture can be maintained during dynamic tasks, however, males and females activate their TrA/IO at different times during landing.[175, 218]

Clinicians and researchers used the curl-up exercise to assess abdominal muscle strength. The RA is activated more during a curl-up exercise than any other abdominal muscle.[60, 215, 217, 219] Wohlfahrt et al[220] assessed abdominal muscle strength dynamically with the maximum number of curl-ups and isometrically using the Sahrmann lower abdominal strengthening program. The number of curl-ups a subject could perform was associated with their ability to maintain an isometric static contraction.[220] greater stability was achieved when strengthening exercises were performed at a slower pace, therefore, the speed at which exercises are performed may effect abdominal muscle strength.[220] The RA and EO muscles were activated during lateral flexion, curl-up, and sit-up exercises.[221] Variation in muscle activation was observed between subjects during these exercises.[221]

Researchers have assessed core muscle activation during various other core strengthening exercises. Richardson et al[60] observed pelvic tilt exercises with one and two leg lowering initiated RA and IO muscle activation. They concluded trunk flexion exercises are performed by the RA with assistance from the IO, while there was little erector spinae (ES) activation.[60] The EO and ES muscle activation increased with trunk rotation. [60]

Ekstrom et al[59] assessed EMG of core, trunk, hip, and thigh muscles (RA, EO, ES, multifidi, GMax, GMed, vastus medialis obliquus, and hamstring) during nine rehabilitation exercises (hip abduction, bridge, bridge with knee extended, side bridge, prone-bridge, superman, lateral step, lunge, and dynamic edge). The greatest GMed muscle activation was demonstrated during the side bridge exercise, while the GMax muscle activation was greatest during the superman exercise (quadruped arm/lower extremity lift).[59] Lateral step-up and lunge exercises recruited the vastus medialis oblique more than any other muscles.[59] Hamstrings demonstrated the greatest muscle activation during the unilateral bridge and superman exercises.[59] Four exercises demonstrated greater muscle activation in the erector spinae (longissimus thoracis) and multifidi than any other exercises (bridge with knee extended, side-bridge, bridge, and the superman exercise).[59] The abdominal muscles (RA and EO) were recruited more during the prone-bridge and side-bridge exercises.[59] The authors suggested these exercises could be used in a core strengthening program depending upon the needs of the patient.[59]

Muscle activation of the RA, EO, ES, and multifidi were measured during three exercises (pelvic-tilt, AH, and level one of the Sahrmann series).[222] The EO demonstrated greater amplitude for all exercises while the RA did not differ between exercises.[222] Erector spinae muscle activation was greater than the multifidi during these three exercises.[222] The pelvic tilt exercise had greater EO muscle activation than AH and Sahrmann series, however, the Sahrmann series recruited the EO more than AH.[222] Rectus abdominus muscle activation was lower during AH than the other exercises.[222]

Core stabilization is essential because abdominal muscle activity is synchronized with lower extremity movement during dynamic tasks.[223] A comprehensive core strengthening program was suggested to enhance lumbo-pelvic stability and postural control.[219, 224] Based on the EMG data discussed above abdominal strengthening exercises should be chosen dependent upon the imbalances/weaknesses observed in the patient or the musculature researchers want to study. Further research is required to understand how abdominal muscles interact with lower extremity joints and muscles. Table 15 summarizes the rehabilitation studies discussed in this section.

Table 15. Summary of Rehabilitation Research

Author, Year	**Instrument(s)**	**Results**	**Conclusion**
Balance Programs			
Bernier & Perrin, 1998[207]	Balance system JPS	JPS ↑ with training Passive JPS ↑ than active max inversion No difference in sway index b/w groups	Balance can be improved in FAI with 6 wks training Unclear if JPS can be improved
Gauffin et al, 1988[205]	Sway	Sway ↓ after disk training ↓ in hip angle after training	Central motor programs are important
Hale et al, 2007[19]	COP_V SEBT	CAI had > COPV between injured & healthy limbs CAI subjects had ↓ lateral, posterior-medial, & posterior-lateral reach in SEBT Rehab improved SEBT reach	SEBT is reliable & able to detect limitations between sides SEBT is sensitive enough to monitor change from rehab
Hoffman & Payne, 1995[206]	BAPS training Force plate	Sway improved from ankle disk training for X & Y parameter Difference between the sway of experimental & control groups	Ankle disk training ↓ FAI & reinjury 10wks disk training ↓ healthy subjects sway
Holme et al, 1999[211]	Position sense Isometric strength Postural sway	Training group had side-to-side strength (plantar flexion, eversion, inversion) & postural sway differences @ 6 wks post injury Injured control group had side to side strength (plantar flexion, eversion, inversion, & inversion) & postural sway differences @ 6 wks No side to side differences @ 4 months Control group 29% reinjury & training group 7% reinjury	Side to side strength & postural sway deficits exist @ 6wks Differences normalize by 4 months Supervised PT may result in a ↓ ankle sprain reinjury
McGuine & Keene, 2006[16]	Epidemiological study	Ankle sprains lower for intervention group; athletes with prior ankle sprains 2x as likely to resprain their ankle	Balance training reduces the risk of basketball and soccer players spraining an ankle; balance training included functional closed chain exercises
Rasool & George, 2007[212]	SEBT	Reach distance increased in the trained leg @ 2 wks & greater @ 4 wks	possible improved control of COP, automatic postural response patterns, attention (central processing); balance training improves reach in all directions; suggest cross-over training effect
Rozzi et al, 1999[208]	Biodex Balance System	Posttraining scores were better than pretraining scores for both subjects with unstable & stable ankles @ high & low resistance	Balance training is an effective method to improve joint proprioception & single-leg balance in subjects with stable & unstable ankles
Tropp et al, 1984[160]	Sway	No stability difference b/w FAI & healthy groups 6 wks ankle disk training ↑ stability Ankle taping did not improve stability	Ankle injury alone does not produce FAI Taping does not effect stability 6 wks ankle disk training ↑ stability & ↓ "giving way"

			feeling
Verhagen et al, 2004[209]	Balance board program	Fewer ankle sprains in the intervention group > reduction of ankle sprain risk in CAI Balance board training ↑ risk of knee injury in players with a history of overuse knee injury	Balance board training is effective for prevention of ankle sprains
Wester et al, 1996[210]	Wobble board training Volumetric measurement	Acute ankle sprain edema ↓@ the same rate for the training & non-training group No training group had > recurrent sprains	Wobble board training ↓ recurrent sprains & preventing FAI
Strength Programs			
Docherty et al, 1998[124]	Electric goniometer Handheld dynamometer	Training group ↑ strength & JPS No eversion JPS effect but there was a dorsiflexion JPS effect	Ankle strengthening improves inversion & plantar flexion JPS FAI subjects had ↑ strength, inversion, dorsiflexion, and plantar flexion JPS
Kaminski et al, 2003[204]	4 training groups	No difference in PT & PT ratios pre & posttraining Training groups did not effect strength	6 wks of strength & proprioception training had no effect on isokinetic strength measures Further research is needed
Powers et al, 2004[203]	f_{med} COP	Strength & proprioceptive training had no effect on fatigue or static balance during single leg task	Poor training program Strength, proprioceptive and a combination of the 2 training programs did not ↑ postural control in FAI 6 wk training may not be long enough
Sekir et al, 2007[202]	Strength JPS Functional tests 6wk training	Invertor PT lower in injured ankle compared to healthy JPS error higher in injured ankle Injured 1-leg stance test ↓ time Functional performance tests longer on injured Isokinetic training 3 days a week improved all variables	Concentric & eccentric isokinetic training improved these parameters Only concentric invertor strength deficits present in FAI subjects

Wk(s)- week(s);

INSTRUMENTATION

Electromyography

The number of action potentials sent along the sacrolemma to the neuromuscular junction are measured using electromyography (EMG).[73] Neural changes (increased motor unit recruitment) due to strengthening programs are also measured using EMG, increased EMG activity suggested greater motor-unit recruitment and firing rates.[66]

Surface and in-dwelling or fine-wire electrodes are two types of electrodes used to measure muscle activation.[66, 73] There are benefits and drawbacks to using each of these types of electrodes. Surface electrodes are convenient to use, require a noninvasive procedure, are cost effective, and measures a larger portion of the muscle electrical activity. The drawback to surface electrodes is cross-talk; you can not differentiate between the muscles that produced the electrical activity during an activity.[73] The benefit of indwelling or fine-wire electrodes is the elimination of cross-talk. Indwelling electrodes sample a smaller number of motor units compared to surface electrodes, this is viewed as either a benefit or drawback depending upon the purpose of the research study. A major drawback to fine-wire EMG use is an invasive procedure is required. Depending on the anatomical structures being studied this methodology could prove to be difficult for the researcher and possibly painful for the subject.

Force Plate

The force plate is an instrument used in biomechanics research. It measures the forces applied to it and ground reaction forces.[110] Most force plates are three dimensional and measure forces on three different axes (x, y, and z). The x-axis measures forces in the medial-lateral direction, while the y-axis assesses forces in the anterior-posterior directions, and the z-axis measures forces in the vertical or superior direction.

Center of pressure is an indirect measurement of balance and postural sway.[75] It is the accumulation of forces that are applied to a certain location of the body, typically the foot, during activity. There are many different COP techniques used to assess balance, these include mean sway amplitude, maximum sway amplitude, minimum sway amplitude, peak-to-peak amplitude, sway path, sway velocity root mean square (RMS) amplitude, and RMS velocity.[75] Center of pressure excursion refers to the total distance traveled during a set amount of time.[75] An increase in COP excursion is thought to indicate postural control deficits.[211] It was concluded the length of the COP path does not provide useful information for clinicians and researchers.[75] Center of pressure velocity (COP_v) was reliable between sessions during double-leg stance. An increase in COP_v is believed to represent decreased postural control.[75] The research remains unclear what COP excursion and velocity actually measure and its accuracy.[75] It is theorized that COP_v is a measure of central postural control, which may indicate the response to maintaining postural control.

Kinematics

Range of motion, displacement, and power can be calculated using 3-dimensional kinematics.[110] Prior to data collection a stationary wand with reflective markers are waved and placed where the data collection will be performed. This calibrates the equipment and defines the global coordinate system. The reflective markers placed on anatomical landmarks of the subject are then viewed by cameras to monitor motion. Computer software is used in conjunction with measurements between markers and of the subject to calculate body segments.

Ultrasound Imaging

Ultrasound imaging (USI) is a diagnostic tool researchers began to use to assess lateral abdominal muscle (EO, IO, and TrA) behavior.[51, 57, 225-230] It is also been referred to as rehabilitative or real-time ultrasound imaging in the literature.[57, 226, 231] Prior to USI, EMG was used to quantify abdominal muscle behavior through muscle activation.[51, 229] Ultrasound imaging provides a non-invasive method to quantify abdominal muscle behavior (muscle thickness) without the limitations of EMG.

Researchers have compared muscle thickness values obtained using ultrasound imaging with magnetic resonance imaging and EMG.[50, 51, 53] The validity of using ultrasound imaging to measure lateral abdominal muscle thickness was assessed by comparing lateral abdominal muscle thickness values obtained with ultrasound imaging to those of MRI, a correlation (ICC=0.78-0.95) existed between the two instruments.[50] Researchers also correlated (R^2=0.87) changes in abdominal muscle activation measured by EMG with changes in TrA muscle thickness measured by ultrasound imaging.[51, 53] These three studies have provided a degree of validity for USI.

The reliability of ultrasound imaging was reported for with-in and between raters and sessions.[43, 44, 48, 49] Intraclass correlation coefficient (ICC) ranged between 0.66-0.99 for intrarater intrasession reliability.[43, 44] Reported ICC values for intrarater intersession reliability ranged from 0.80-0.99.[48, 49] Interrater reliability was assessed and excellent ICC values were obtained while measuring TrA thickness at rest and during contraction.[225]

Chapter 3

Methods

STUDY DESIGN

A 3 x 2 factorial design will be used to assess COP excursion (distance & velocity), muscle activation (IO/TrA, EO, GMed, & BF), and kinematics (ankle, knee, & hip joint angles) before and after an eight week abdominal strengthening. Figure 1 diagrams the 3 x 2 research design. The independent variables are group (control, healthy, and FAI) x time (pre- & posttraining). A 3 x 2 x 6 factorial design will be used to assess lateral abdominal muscle thickness (EO, IO, and TrA) biweekly throughout an eight-week abdominal-strengthening. The independent variables are group (control, healthy, and FAI) x type of contraction (relaxed and during AH) x time (0, 2, 4, 6, 8, and 9 weeks). Figure 2 diagrams the 3 x 2 x 6 research design. Dependent variables include abdominal muscle thickness (EO, IO, RA, and TrA), COP excursion (distance and velocity), muscle activation (peak and mean amplitudes), and kinematics (ankle, hip, & knee joint angles) in the frontal and sagittal planes. The dependent variables are repeated measures. Abdominal muscle thickness will be measured every two weeks throughout the eight week abdominal strengthening program. Center of pressure excursion, muscle activation, and kinematic data will be assessed pre- and posttraining.

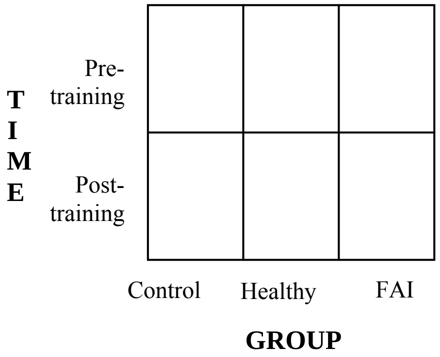

Figure 1. 3x 2 Study Design

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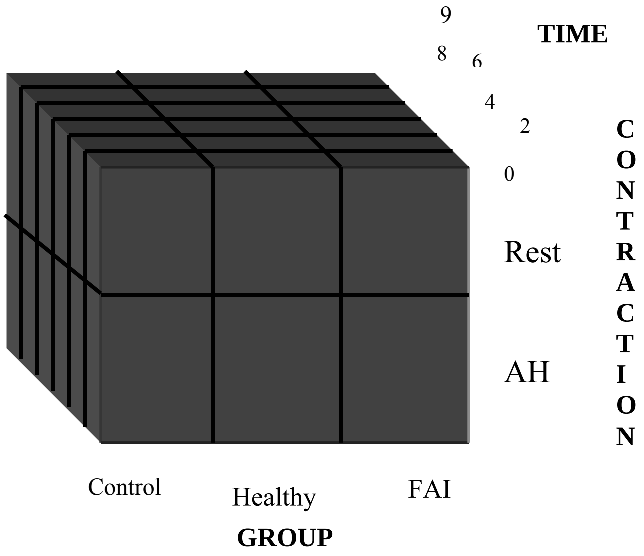


Figure 2. 3 x 2 x 6 Study Design

SUBJECTS

Seventy-five physically active female and male college-age subjects will be divided into three groups (control (C), healthy with abdominal strengthening (Healthy), and FAI with abdominal strengthening (FAI)). Twenty-five subjects with a history of FAI will be assigned to the FAI group. The FAI subjects will self-report CAI using the Ankle Instability Index (AII) and FAI with the Foot and Ankle Ability Measure (FAAM) questionnaires, respectively. Subjects in the control and healthy groups will be matched by gender and leg dominance with a subject in the FAI group. If an FAI subject's injured limb is their dominant limb, they will be matched with a subject in the control and healthy group whose dominant limb is the same as the FAI subjects. Fifty subjects will be randomly assigned to the control or healthy groups after they are matched. Exclusion criterion will be a history of cardiovascular or neurological disorder, childbirth or pregnancy within the past two years; abdomen, low back, or lower extremity injury or pain within the past year that restricted the subject's ability to be physically active; abdomen, low back, or lower extremity surgery within the past two years; or regularly participation in an abdominal strengthening program. Regular participation is defined as performing abdominal strengthening exercises three times per week or more. All subjects that qualify to participate in this study will read and sign a university approved informed consent form prior to data collection.

Subjects will also be excluded from the study if they sustain an abdomen, low back, or lower extremity injury during the study that restricts their activities of daily living or miss two abdominal thickness measurement data collection sessions, two strength training sessions, or fail to return a weekly exercise log.

INSTRUMENTS

Ultrasound Imaging

Lateral abdominal muscle thickness will be measured using the LOGIQ P5 Laser Doppler Ultrasound (General Electric, Piscataway, NJ, USA) with a linear phased array probe (45 x 10 mm footprint; 7 to 12 MHz frequencies) at rest and during AH. Probe frequency will be set at 10 MHz with a gain of 70 for all measurements.[42] Rectus abdominis (RA) muscle thickness will be measured only at rest because it is impossible to measure EO, IO, TrA and RA thickness simultaneously during AH with one probe. The external oblique, IO, and TrA measurement site is midway between the mid-axillary line and level with the umbilicus. The RA measurement site is lateral to the linea alba, thus RA thickness during AH will not be measured. Subjects will be asked to refrain from eating or exercising for a minimum of one hour prior to abdominal thickness measurements.

Electromyography

The BIOPAC MP150 System (BIOPAC Inc., Goleta, CA) will be used to measure muscle activation. These measurements will be collected using surface electrodes with an inter-electrode distance set at approximately 2 cm.[58] The electrodes will be aligned parallel with the muscle fibers, and placed approximately midway between the innervation zone and the insertion of the distal tendon.[58] Signals will be amplified (DA100B, BIOPAC Inc., Goleta, CA) from disposable, pregelled Ag-AgCl electrodes (EL-503, BIOPAC Inc., Goleta, CA).[58] Electromyography data will be collected at 1250 Hz. The input impedance of the amplifier will be 1.0 megohm, with a common mode rejection ratio of 90 dB, high and low pass filters of 10 and 500Hz, a signal to noise ratio of 70 dB, and a gain of 2000.[58]

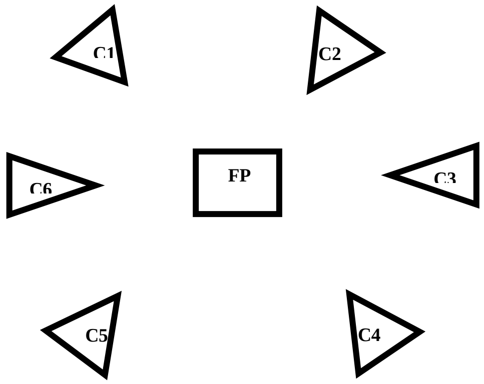
Due to the close proximity of IO and TrA muscles, it is impossible to determine which muscle is activated when surface electrodes are used. Therefore, the muscle activation of the IO and TrA will be reported together instead of individually. The use of surface electrodes to measure muscle activation for the IO and TrA has demonstrated good to excellent reliability and validity.[77]

Force Plate

A force plate (AMTI OR6-5, Newton, MA) will be used to measure COP excursion during a single-leg drop landing. The sampling rate will be set at 1250 Hz. Center of pressure excursion velocity and total excursion length will both be reported because it is unclear which one is a better measure of COP excursion.[22, 23]

Kinematics

Kinematic data will be collected at 250Hz using six Vicon MX13+ cameras running on Nexus 1.3 software (Vicon, Centennial, CO).[54] Kinematic, COP excursion, and EMG data will be synchronized using triggering devices. The proposed camera set-up is diagramed in Figure 3 with the force plate located in the center. Subjects will wear spandex clothing (shirt and shorts) during data collection. This will allow reflective markers to be placed more accurately over anatomical landmarks reducing the chance of loose clothing covering up markers during data collection.



Abbreviations: C1-6 – Camera Number; FP- Force plate

Figure 3. Kinematic Camera Placement

Reflective markers will be placed on each subject to measure joint angles of the ankle, knee, and hip in both the frontal and sagittal planes. The marker set will be a modification of a previously used marker set used to measure kinematic data during landing.[54] Good reliability was reported for measuring kinematic data during a drop vertical jump landing with this marker set.[232] A modification of this marker set will be used to improve accuracy of the thigh and lower leg position measurements. This includes using a marker cluster to replace the single markers on the thigh and lower leg.

The custom marker set-up will include single and cluster reflective markers placed over the lower extremity. Twenty-two single reflective markers and four cluster markers that contain four markers will be placed on each subject. The single markers will be placed over the following anatomical landmarks: 5th metatarsal styloid process, between the 2nd and 3rd phalanges, talus (anterior middle), medial and lateral malleoli, calcaneus (posterior middle), knee joint line (medial & lateral), greater trochanter, anterior superior iliac spine (ASIS), and posterior superior iliac spine (PSIS). A 4-marker reflective cluster will be placed over the right and left medial flare of the tibia and anterior aspect of both thighs. Figure 4 illustrates reflective marker placement for anterior, lateral, and posterior views.

Posterior View

Anterior View

Lateral View

Figure 4. Kinematic Marker Placement

PROCEDURES

Figure 5 provides the timeline for the data collection sessions with the time between sessions. The first session will be an orientation session that will familiarize the subjects with the study's procedures prior to pre-training data collection. Thirty-five days after the orientation session subjects will report for the pre-training data collection session. Two weeks after pre-training data collection, abdominal muscle thickness measurements will continue. Day 2 through day 6 of data collection sessions will consists of abdominal muscle thickness measurements. These sessions will be 14 days apart at the same time of day. The seventh day of data collection will be the post-training data collection session. This session will occur within 1 week after the completion of the 8th week of the abdominal strengthening program.

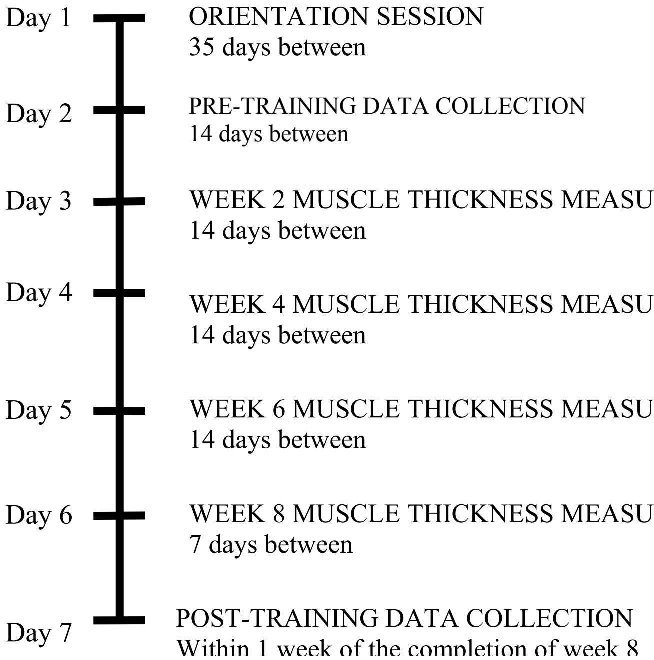


Figure 5. Procedural Timeline

Orientation Session

Subjects will participate in two orientation sessions. The first session will occur three weeks prior to the data collection session. It will involve completing paperwork, teaching subjects skills (AH and drop landing), and providing subjects time to practice the skills. The study will be explained to the subjects, and then they will read the approved informed consent form. Upon completion any remaining questions will be answered before subjects sign the consent form. Subjects will complete the Modified Ankle Instability Index (AII), Foot and Ankle Ability Measure (FAAM) and FAAM sport questionnaires followed by a physical exam of the ankle joint to assess for mechanical ankle instability. The AII and FAAM questionnaires will determine if subjects qualify for the FAI group. To qualify for the FAI group subjects must: 1) have a history of at least one substantial ankle sprain with the initial ankle sprain occurring greater than 12 months ago, 2) complain of the ankle "giving way" during functional activities, this is assessed with questions 4 through 8 on the AII 3) answer yes to at least 2 of those questions. Subjects will self-report FAI by scoring less than or equal to 90% on the FAAM ADL scale and 80% on the FAAM Sport scale. The AII, FAAM, and directions for scoring the FAAM are included in Appendix A1.

Subjects will be taught how to perform AH in the supine hook-lying position (Figure 6) and a single-leg drop landing (Figure 7). The following standardized instructions will be given to every subject prior to performing AH, “gently pull the umbilicus towards the plinth or floor without moving your spine while maintaining normal breathing”.[55] When the rater and subject believe AH is being performed correctly, it will be confirmed using ultrasound imaging. If a subject is unable to correctly perform AH, ultrasound imaging will be used to provide visual biofeedback to assist subjects until they learn how to correctly perform AH. Visual feedback will not be provided during data collection. Subjects have to correctly perform three consecutive AH maneuvers, prior to the end of the orientation session.[45]


[redacted]

Figure 6. Supine Hook-lying Position

Figure 7. Single-leg Drop Landing

Subjects will then be taught how to perform a 35 cm single-leg drop landing. The instructions given to the subjects for a drop landing will be to 1) place the dominant limb in front of the platform, 2) lean forward placing the dominant foot over the center of the force plate and descend while pulling the nondominant foot away from the platform prior to landing, and 3) upon landing locate the black “X” on the wall with your eyes and stand erect on the dominant limb for approximately five seconds with your hands on your hips. The “X” on the wall will be at eye level and approximately 15 feet directly in front of the subject. Subjects will practice this skill until he/she can correctly perform it. An investigator will visually determine if the drop landing is performed correctly. Subjects are required to correctly perform three consecutive single-leg drop landings prior to the completion of the first orientation session. Table 15 summarizes the sequence of the first orientation session.

Table 15. Steps of Orientation Sessions

First Orientation Session
1. Explain the study to the subject
2. Subjects read through the IRB informed consent form and sign it when their questions are answered
3. Subject completes the AII and FAAM questionnaires
4. Primary investigator performs physical exam of the ankle to rule out MAI
5. Subjects will learn and practice how to correctly perform AH
6. Subjects must perform 3 consecutive correctly performed AH, confirmed via USI
7. Subjects will learn and practice how to correctly perform a single-leg drop landing
8. Subjects must perform 3 consecutive correctly performed single-leg drop landings

The second orientation session will occur in conjunction with the first day of data collection. Subjects will review the skills taught and learned at the first orientation session. They will correctly perform three consecutive AH and drop landings prior to data collection. The healthy and FAI groups will be instructed on the abdominal strengthening program when baseline data collection is completed. Subjects are expected to complete a weekly abdominal strengthening exercise log; this log will be returned to the investigators each week at the weekly training session.

Data Collection

Table 16 outlines the sequence of the predata collection session. Muscle thickness measurements will be measured at rest and during AH with subjects in a supine hook- lying position on a plinth.[43] Their hips and knees will be flexed to approximately 45° and 90°, respectively.[44] All measurements will be taken level with the umbilicus and medial to the mid-axillary line on the subject's dominant side.[56]

Table 16. Sequence of Data Collection Session

Data Collection Timeline
1. Collect demographic data
2. Subject is assigned to a group
3. Subjects will practice correctly performing AH
4. Identify & mark the USI site with a marker
5. Subjects correctly perform three consecutive AH, confirmed via ultrasound imaging
6. Take five muscle thickness measurements at rest
7. Take five muscle thickness measurements during AH
8. Place surface electrodes & reflective markers on the subject
9. Subject will practice performing a single-leg drop landing
10. Subjects will perform three consecutive single-leg drop landings
11. Five single-leg drop landing trials performed for data collection (COP, EMG, & kinematics)
12. Introduce and instruct subjects on the abdominal strengthening program

The measurement site is the location that provides the clearest ultrasound image of the EO, IO, and TrA (Figure 8). Immediately after identifying the measurement site, a line will be

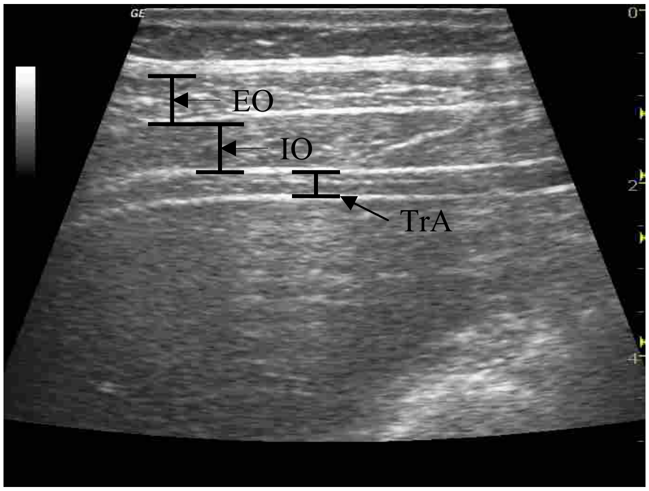


Figure 8. Clearest Image of Lateral Abdominal Musculature.

placed on the subject's skin at the lateral edge of the probe with a permanent marker when the site is determined (Figure 9). The lateral edge of the probe will be aligned with this line to standardize ultrasound head placement for following measurements (Figure 10). Each subject will be provided with a Sharpie® marker to remark the measurement line throughout the eight week study. Abdominal muscle thickness will be assessed biweekly on the same day and at the same time throughout the eight-week study.

Figure 9. Mark to Standardize Ultrasound Transducer Head Placement.

Figure 10. Probe Placement for Thickness Measurements

Three images of the EO, IO, RA, and TrA muscles will be obtained at rest, followed by three measurements of the lateral abdominal muscles during AH. Each image will be analyzed separately. Subjects will hold the AH maneuver for approximately six seconds; this provides the rater time to capture an image. Abdominal hollowing images will be obtained during peak TrA thickness, which the rater will visibly determine. Approximately 30 seconds will elapse between image captures. The ultrasound imaging software's internal calipers will be used to measure muscle thickness.

A 25.2 x 18 cm transparent grid will be positioned over the computer screen to identify the middle of the frozen images (middle line of grid) (Figure 11). The perpendicular line will start where the fascial layers and middle line of grid intersected (Figure 11).[49, 55, 57] This location on the image is referred to as the intersection point. A research assistant will record the thickness values and erase them after every image to blind the rater. The three thickness values at rest and during AH will be averaged for statistical analysis.

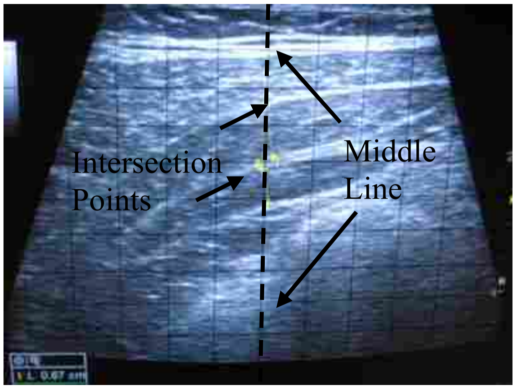


Figure 11. Grid Overlay and Measurement Procedure

Electromyography, COP excursion, and kinematic data will be collected while subjects perform five single-leg drop landing. The mean and peaks of five good single-leg drop landing trials will be used for data analysis. A good trial consists of the subject being able to stand on their single leg for three seconds following landing without losing his/her balance. Failed landings will not be included; the maximum number of drop landings allowed will be ten. If five good trials can not be performed within ten trials the subjects will be excluded from the study.

These measurements will be taken during the first week and within one week after the completion of the abdominal strengthening program. Muscle activation of the lateral abdominal (EO, IO/TrA) and lower extremity muscles (GMed and BF) will be assessed from 500 ms pre-drop landing until 1 second post-drop landing. Table 17 describes the direction and placement of the surface EMG electrodes. The skin will be abraded with a fine sandpaper block and cleansed with an alcohol wipe prior to placing the electrodes; correct placement will be confirmed using manual muscle tests.

Table 17. Surface Electrode Placement

Muscle	Electrode Direction	Electrode Placement
External Oblique	Oblique	Approximately 12-15 cm lateral to the umbilicus[77]
Internal Oblique / Transverse Abdominis	Transverse	2cm medial and inferior to the anterior superior iliac spine[77]
Gluteus Medius	Longitudinal	Halfway between the greater trochanter and lateral most aspect of the iliac crest[59, 78]
Biceps Femoris	Longitudinal	Approximately 50% of the distance between the ischial tuberosity to the head of the fibula[233]

Joint angles of the ankle, knee, and hip will be measured using kinematics. Table 18 provides a list of the joints, measurements, and the time measurements will be taken during kinematic data collection. The mean degrees of ankle, hip, and knee range of motion (ROM) will be measured at initial contact and peak ROM will be measured when it is reached during the single-leg drop landing. Two minutes rest will be given to the subject between trials. Center of pressure excursion distance and velocity will be assessed for one second post-drop landing.

Table 18. Kinematic Measurements

Joint	Measurement	Time
Ankle		
	Dorsiflexion	Initial contact & Peak
	Plantar-flexion	Initial contact
Knee		
	Flexion	Initial contact & Peak
	Extension	Initial contact
Hip		
	Flexion	Initial contact & Peak
	Extension	Initial contact
	Abduction	Initial contact & Peak
	Adduction	Initial contact & Peak

Abdominal Strengthening Program

The abdominal-strengthening program will be eight weeks long. Both the healthy and FAI groups will complete this strengthening program. Subjects will perform the abdominal strengthening program three days a week with one day of rest between strengthening sessions. One of the three strengthening sessions each week will be under the direct supervision of the investigators in the modalities lab, while the remaining two days will be done on their own. An exercise log will be maintained by the subjects and returned to the investigators at the weekly supervised strengthening session. If a subject misses two strengthening sessions or fail to turn in the weekly exercise log he/she will be excluded from the study.

The exercises are focused on strengthening the lateral abdominal muscles and RA. Table 19 provides a summary of the strengthening exercises and repetitions included in this study by weeks. The exercises included in this program were based upon previously reported muscle activation of the abdominal musculature (RA, IO/TrA, and EO) during rehabilitative exercises.[59] Subjects will perform AH during all of the exercises in an attempt to preferentially activate the IO and TrA.

Table 19. Eight Week Abdominal Strengthening Program

Week One	**Repetitions**
Level 1 LAS	2 sets of 10
Curl-up	2 sets of 10
Side bridge	2 sets of 10
Sit-up with rotation	2 sets of 10
Week Two	
Level 1 LAS	3 sets of 10
Curl-up	3 sets of 10
Side bridge	3 sets of 10
Sit-up with rotation	3 sets of 10
Week Three	
Level 2 LAS	2 sets of 10
Curl-up	2 sets of 15
Side bridge	2 sets of 15
Sit-up with rotation	2 sets of 15

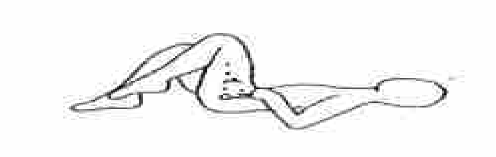
Week Five	**Repetitions**
Level 3 LAS	2 sets of 10
Curl-up	2 sets of 20
Side bridge	2 sets of 20
Sit-up with rotation	2 sets of 20
Prone bridge	3 sets of 10s
Week Six	
Level 3 LAS	3 sets of 10
Curl-up	3 sets of 20
Side bridge	3 sets of 20
Sit-up with rotation	3 sets of 20
Prone bridge	2 sets of 15s
Week Seven	
Level 5 LAS	2 sets of 10
Curl-up	2 sets of 25
Side bridge	2 sets of 25
Sit-up with rotation	2 sets of 25
Prone bridge	3 sets of 15s

Week Four	
Level 2 LAS	3 sets of 10
Curl-up	3 sets of 15
Side bridge	3 sets of 15
Sit-up with rotation	3 sets of 15
Prone bridge	2 sets of 10s

Week Eight	
Level 5 LAS	3 sets of 10
Curl-up	3 sets of 25
Side bridge	3 sets of 25
Sit-up with rotation	3 sets of 25
Prone bridge	4 sets of 15s

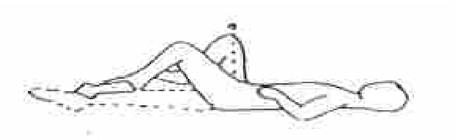
Lower abdominal series (LAS). This series consists of five different levels of exercises that progress in difficulty. Four of these levels will be part of this eight-week program. Level one is considered the easiest and level five is the most difficult. Abdominal hollowing will be performed throughout these exercises. Common mistakes individuals make throughout these exercises include holding one's breath, contracting the gluteal and hamstring muscles, lifting the head, and abdominal pouching. Abdominal pouching is the visible contraction of the RA instead of hollowing the abdominal cavity.[61] The hook-lying position is the starting position for all of the lower abdominal series (LAS) exercises. The levels are illustrated and described below.

<u>Level 1</u>



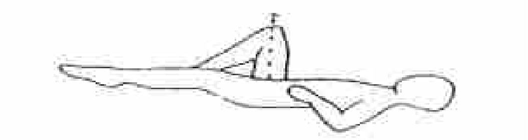
Lift 1 leg to 90° of hip flexion; lift the 2nd leg to 90° of hip flexion, lower leg 1 followed by leg 2 to the starting position

<u>Level 2</u>



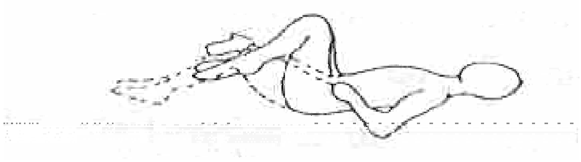
Lift 1 leg, then the 2nd leg to 90° hip flexion; touch heel 1 to table, slide it along the floor/table until it is straight; return leg 1 to 90° hip flexion; repeat with second leg; lower leg 1 to starting position; lower leg 2 to the starting position

<u>Level 3</u>



Lift both legs to 90° hip flexion; keep leg 1 at 90° of hip flexion; lower leg 2 just above the floor/table without touching; extend the leg out above floor/table; return leg 1 to 90° of hip flexion; repeat this with second leg; lower both legs together to the starting position

<u>Level 5</u>



Start with both legs straight; flex hips until 90° of hip flexion is achieved; reverse the process while keeping the heels above the table; lower the heels once the legs are extended

Curl-Up. Subjects will lie on the floor/table in the supine hook-lying position with arms resting at their side (Figure 12). Subjects will, "1) perform AH, 2) lift and slide arms forward, 3) bring chin to chest, and 4) curl the trunk until the inferior angles of the scapula are off the floor/table"[60] (Figure 13).

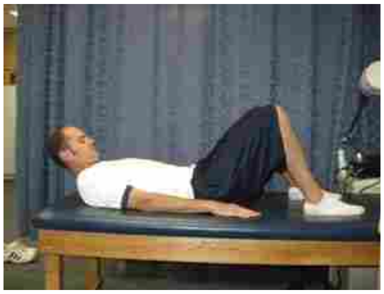


Figure 12. Curl-up Starting Position

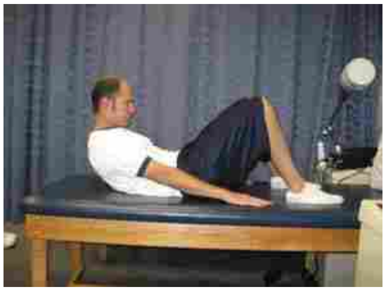


Figure 13. Curl-up Peak Position

Side-Bridge. Instructions given to the subjects for side-bridge are, "1) assume a side lying position on one side, 2) place the elbow closest to the floor at a 90° angle underneath the shoulder with the forearm flat on the floor/table, 3) place the opposite arm along the upper side of the body, 4) perform AH, and 5) lift the pelvis towards the ceiling until a neutral spine is achieved" (Figures 14 & 15). This exercise is performed bilaterally.

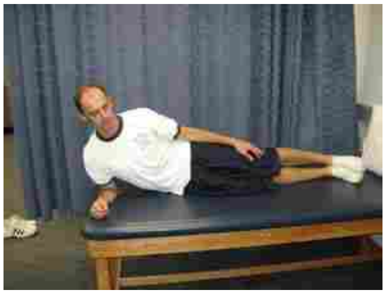


Figure 14. Side-Bridge Starting Position

Figure 15. Neutral Spine Position

Sit-Up with Rotation. Subjects start in the hook-lying position with their arms crossed against their chest. The instructions are to "1) lift the trunk off the floor/table, 2) rotate until the left elbow touches the right knee or the right elbow touches the left knee, 3) return to the starting position, and 4) repeated steps 1 and 2 to the opposite side" (Figures 16-19). The direction of rotation will alter every other sit-up and set.

Figure 16. Sit-up Starting Position

Figure 17. Right Elbow to Left Knee

Figure 18. Return to Starting Position

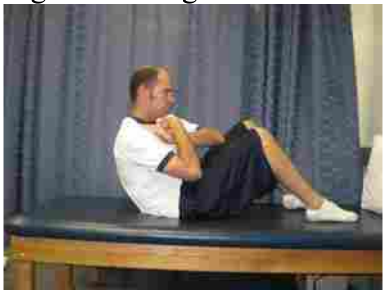


Figure 19. Left elbow to Right Knee

Prone-Bridge. The prone-bridge exercise is added to the strengthening program during the fourth week. It is addition will provide some variation to the program and increase the demands placed on the abdominal muscles. Subjects will begin in the starting position (Figure 20), prone with their elbows under their shoulders. They will lift their pelvis until they reach the peak position, which is when the shoulders, pelvis, and ankles are in a straight line (Figure 21). The peak position will be held for approximately 3 seconds.

Figure 20. Prone-Bridge Starting Position

Figure 21. Prone-Bridge Peak Position

Data Analysis

SPSS 16.0 (SPSS Inc., Chicago, IL, USA) will be used to analyze and manage the data. Repeated measures ANOVA will be used to analyze abdominal muscle thickness, COP excursion, EMG, and kinematic data. A Tukey-Kramer post-hoc multiple comparison test will be performed to determine pair wise contrasts.

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Appendix A1

Ankle Instability Questionnaires

Modified Ankle Instability Instrument

1. Have you ever sprained an ankle? ☐Yes ☐No
    a. Have you sprained your right ankle? _________
    b. Have you sprained your left ankle? __________

2. Have you ever seen a doctor for an ankle sprain? ☐Yes ☐No

3. Did you ever use a device (such as crutches) because you could not bear weight due to an ankle sprain? ☐Yes
    ☐No

4. Does your ankle ever feel unstable while walking on a flat surface? ☐Yes ☐No

5. Does your ankle ever feel unstable while walking on uneven ground? ☐Yes ☐No

6. Does your ankle ever feel unstable during recreational or sport activity?
    ☐Yes ☐No ☐N/A

7. Does your ankle ever feel unstable while going *up* stairs? ☐Yes ☐No

8. Does your ankle ever feel unstable while going *down* stairs? ☐Yes ☐No

9. Have you ever had rehabilitation on your ankle due to a sprain? ☐Yes ☐No

10. Have you ever had an injury to your knee? ☐Yes ☐No
    If yes, please explain

Side (Right or Left)	Injury	Date
_________________	________________________________	_________
_________________	________________________________	_________

11. Have you ever had an injury to your leg below the knee? ☐Yes ☐No
    If yes, please explain

Side (Right or Left)	Injury	Date
_________________	________________________________	_________
_________________	________________________________	_________

Number of previous ankle sprains:
LEFT: __________ RIGHT: __________

How long since your last ankle sprain?
LEFT: __________ RIGHT: __________

Foot and Ankle Ability Measure (FAAM)

Please answer **every question** with one response that most closely describes to your condition within the past week. If the activity in question is limited by something other than your foot or ankle mark not applicable (N/A).

	No difficulty at all	Slight difficulty	Moderate difficulty	Extreme difficulty	Unable to do	N/A
Standing	☐	☐	☐	☐	☐	☐
Walking on even ground	☐	☐	☐	☐	☐	☐
Walking on even ground without shoes	☐	☐	☐	☐	☐	☐
Walking up hills	☐	☐	☐	☐	☐	☐
Walking down hills	☐	☐	☐	☐	☐	☐
Going up stairs	☐	☐	☐	☐	☐	☐
Going down stairs	☐	☐	☐	☐	☐	☐
Walking on uneven ground	☐	☐	☐	☐	☐	☐
Stepping up and down curbs	☐	☐	☐	☐	☐	☐
Squatting	☐	☐	☐	☐	☐	☐
Coming up on your toes	☐	☐	☐	☐	☐	☐
Walking initially	☐	☐	☐	☐	☐	☐
Walking 5 minutes or less	☐	☐	☐	☐	☐	☐
Walking approximately 10 mins	☐	☐	☐	☐	☐	☐
Walking 15 minutes or greater	☐	☐	☐	☐	☐	☐

Because of your **foot and ankle** how much difficulty do you have with:

	No difficulty at all	Slight difficulty	Moderate difficulty	Extreme difficulty	Unable to do	N/A
Home Responsibilities	☐	☐	☐	☐	☐	☐
Activities of daily living	☐	☐	☐	☐	☐	☐
Personal care	☐	☐	☐	☐	☐	☐
Light to moderate work (standing, walking)	☐	☐	☐	☐	☐	☐
Heavy work (push/pulling, climbing, carrying)	☐	☐	☐	☐	☐	☐
Recreational activities	☐	☐	☐	☐	☐	☐

How would you rate your current level of function during your usual activities of daily living from 0 to 100 with 100 being your level of function prior to your foot or ankle problem and 0 being the inability to perform any of your usual daily activities?

.0%

FAAM Sports Scale

Because of your **foot and ankle** how much difficulty do you have with:

	No difficulty at all	Slight difficulty	Moderate difficulty	Extreme difficulty	Unable to do	N/A
Running	☐	☐	☐	☐	☐	☐
Jumping	☐	☐	☐	☐	☐	☐
Landing	☐	☐	☐	☐	☐	☐
Starting and stopping quickly	☐	☐	☐	☐	☐	☐
Cutting/lateral movements	☐	☐	☐	☐	☐	☐
Low impact activities	☐	☐	☐	☐	☐	☐
Ability to perform activity with your normal technique	☐	☐	☐	☐	☐	☐
Ability to participate in your desired sport as long as you would like	☐	☐	☐	☐	☐	☐

How would you rate your current level of function during your sports related activities from 0 to 100 with 100 being your level of function prior to your foot or ankle problem and 0 being the inability to perform any of your usual daily activities?

.0 %

Overall, how would you rate your current level of function?

☐ Normal ☐ Nearly normal ☐ Abnormal ☐ Severely abnormal

Scoring Instructions for the FAAM

The ADL and Sports subscales are scored separately.
The response to each item on the ADL subscale is scored from 4 to 0, with 4 being “no difficulty” and 0 being “unable to do”. N/A responses are not counted. The score on each of the items are added together to get the item score total. The total number of items with a response is multiplied by 4 to get the highest potential score. If the subject answers all21 items, the highest potential score is 84. If one item is not answered the highest score is80, if two are not answered the total highest score is 76, etc. The item score total is divided by the highest potential score. This value is then multiplied by 100 to get a percentage. A higher score represents a higher level of physical function.

The Sports subscale is scored the same as above, 4 being “no difficulty at all” to 0 being “unable to do”. The score on each item are added together to get the item score total. The number of items with a response is multiplied by 4 to get the highest potential score. If the subject answers all 8 items the highest potential score is 32. If one item is no answered the highest potential score is 28, if two are not answered the highest potential score is 24, etc. The item score total is divided by the highest potential score. This value is multiplied by 100 to get a percentage. A higher score represents a higher level of physical function.

Psychometric Information

Relates to scores out of 100 percentage points

	ADL subscale	**Sports subscale**
Error associated with a one time measurement95% confidence	**7** points	**10** points
Minimal detectable difference over a four week period 95% confidence	**6** points	**12** points
*Minimal Clinically Important Difference	**8** points	**9** points

* The Minimal Clinically Important Difference is the score distinguished patients who felt they improved with physical therapy from those who felt they did not improve over a four week period.