Lower Extremity Neuromechanics During a Forward-Side Jump Following Functional Fatigue in Patients with Ankle Instability

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Lower Extremity Neuromechanics During a Forward-Side Jump Following Functional Fatigue in Patients with Ankle Instability

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

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ABSTRACT

Lower Extremity Neuromechanics During a Forward-Side Jump Following Functional Fatigue in Patients with Ankle Instability

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Doctor of Philosophy

Ankle instability (AI) and fatigue impair neuromuscular control as well as dynamic joint stability of the lower extremity. No one has comprehensively examined the effects of AI and fatigue on neuromechanics of the lower extremity during a functional activity. Deficits associated with AI and fatigue could be additive in increasing the risk for injury in patients with AI. PURPOSE: To examine the interaction of AI and fatigue on lower extremity muscle activity, kinematic, and kinetic patterns during a forward-side jump. METHODS: 25 AI (23.3 ± 1.9 yrs, 176.5 ± 10.5 cm, 70.9 ± 11.4 kg), and 25 matched control subjects (23.7 ± 2.5 yrs, 175.0 ± 10.8 cm, 70.3 ± 12.8 kg) were categorized according to the Foot and Ankle Ability Measure (FAAM) (ADL: 84.3 ± 7.6%, Sport: 63.6 ± 8.6%) and the Modified Ankle Instability Instrument (MAII) (3.7 ± 1.2). Fifty-nine reflective markers were placed over anatomical landmarks and eight electromyography (EMG) electrodes were placed on tibialis anterior (TA), peroneus longus (PL), medial gastrocnemius (MG), medial hamstring (MH), vastus lateralis (VL), adductor longus (AL), gluteus medius (GMed), and gluteus maximus (GMax) muscles in the involved leg. Subjects performed five forward-side jumps on a force plate before and after functional fatiguing exercises. To induce fatigue, subjects began 5-min incremental running on a treadmill between 5 and 6 mph. Next, subjects performed 20-second lateral counter movement jumps (CMJ), and 20 vertical CMJs. After each fatigue cycle, subjects performed one max vertical jump. Subjects repeated three exercises until Borg’s rating of perceived exertion (RPE) reached 17 and the vertical jump height fell below 80% of their max jump height. Functional analysis of variance (FANOVA) \((p < 0.05)\) was used to evaluate differences (a group by fatigue interaction) between two conditions (pre- vs post-fatigue) in each group (AI and control) for lower-extremity kinematic, kinetic and neuromuscular patterns. Pairwise comparison functions as well as 95% confidence interval (CI) bands were plotted to determine specific differences. If 95% CI bands did not cross the zero line, we considered the difference significant. RESULTS: Compared to the control group, the AI group demonstrated less range of dorsiflexion, knee and hip flexion motions during early phase of landing after fatigue. For sagittal-plane hip kinetics, subjects with AI decreased the hip extension moment while control subjects increased hip extension moments during landing following functional fatiguing exercise. The AI group showed less reduction of anterior-posterior ground reaction force (AP GRF) during transition phase of a forward-side jump after fatigue compared to control subjects. The AI group decreased EMG amplitude of PL, MH, and GMed while increased VL and GMax during landing after fatigue compared to control subjects. CONCLUSION: AI subjects demonstrated greater impairments in neuromechanical control patterns than a matched control group during a sport movement as fatigue progressed. Compared to AI group, control subjects showed a coordinated joint control strategy after fatigue, increasing joint angles from distal (ankle) to proximal (hip) joints by increasing hip extensor moments during landing from a forward-side jump in an attempt to reduce ground impact force. EMG alterations were consistent with patterns observed in injured patients, which may predispose patients to poor positions associated with lower extremity joint injury. These interactions between neuromuscular fatigue and AI may predispose individuals to lower extremity injuries.

Keywords: ankle instability, fatigue, kinematics, kinetics, electromyography
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Introduction

While studies consistently support the benefits of exercise, the incidence of exercise-related injuries continues to rise. Ankle and knee injuries are of the most common lower extremity injuries during physical activities such as landing, jumping, and cutting. Injury can result in decreased activity in the short term and may have lifelong consequences such as osteoarthritis due to activity restriction. Lateral ankle sprains are one of the most common injuries during sport activities and they can result in ankle instability (AI) characterized by pain, swelling, decreased function, a repetitive “giving away” of the ankle and eventually repeated ankle sprains. Thirty to seventy percent (30%-70%) of patients suffer from AI following an initial lateral ankle sprain. AI is generally attributed to two potential mechanisms: mechanical and functional instabilities. Mechanical ankle instability (MAI) includes either excessive hypermobility or hypomobility of the joint while functional ankle instability (FAI) is more related to sensorimotor deficits such as impaired proprioception, neuromuscular control, impaired postural control, and altered movement patterns. Many studies have reported evidence that subjects with AI display different ankle and knee kinetic, kinematic, and neuromuscular patterns during functional activities compared to normal subjects. For instance, a recent study examining jogging kinematics showed that subjects with AI had greater plantarflexion and inversion during stance phase compared to a control group. In addition, individuals with AI had greater ground reaction force (GRF) and altered patterns of ankle joint moments (inertor moment in a control group and evertor moment in an AI group) during the loading phase of single-leg landing and gait, respectively, compared to the control group. These impairments and alterations may position the ankle with varying loads that could increase susceptibility to recurrent lateral ankle sprain and potential osteoarthritis.
Neuromuscular fatigue has been suggested as a risk factor for lower extremity injury because it can alter lower extremity kinematics, kinetics, and muscle activity during functional activities such as landing, cutting, and jumping. Changes in joint position during landing can affect the magnitudes of the ground reaction force and corresponding joint moments, resulting in more stresses on structures in lower extremity joints. Cortes et al. showed decreases in knee and hip flexion angles at the initial foot contact during both stop-jump and side-cutting tasks after functional fatiguing exercises comprised of jumping and running components. Previous studies have also shown GRF increased during fatigued hopping, landing, and sub-maximum drop jumping. Orishimo et al. reported knee extension moments were significantly decreased while ankle plantarflexion moments increased during single-leg hopping. The lower extremity muscles provide dynamic stability for ankle and knee joints during sports activities. A reduction in the force-generating capacity of muscle may impact the ability of muscles to act as dynamic stabilizers of those joints. Previous studies have shown that most ankle and knee sprains frequently occurred toward the end of the first and second halves of soccer matches, which may suggest that fatigue contributes to altered neuromuscular control of the ankle and knee joints. For instance, neuromuscular fatigue deteriorates motor unit discharge, excitation-contraction coupling, and the sensitivity of muscle receptors, resulting in decreased proprioceptive function. These fatigue-induced impairments in sensorimotor function may be a potential cause for neuromuscular and postural control deficits.

A variety of fatigue protocols has been developed to identify alterations in neuromuscular control of the lower extremity due to fatigue. However, most studies have used isokinetic fatigue protocols (nonweight bearing) to induce muscle fatigue and these protocols involved isolated joint motions and muscle groups. The true nature of muscle function and its effect on
functional joint motions is difficult to assess from isolated forms of isometric, concentric or eccentric contractions since complete volitional exhaustion of a single muscle or muscle group rarely occurs during functional activity. Moreover, previous work has shown that prolonged submaximal fatiguing exercise results in greater central and peripheral muscle fatigue as opposed to short maximal exercise. For these reasons, fatigue protocols that involve total lower extremity actions that incorporate submaximal stretch shortening cycles may better mimic neuromuscular fatigue associated with a competitive athletic environment.

As previously mentioned, contributing factors to AI are multifactorial, such as impaired proprioception and neuromuscular and postural control, and neuromuscular fatigue adversely affects these factors. Neuromuscular fatigue of ankle invertors, impaired eversion force sense, and greater magnitude of force-sense error was shown in subjects with AI. Individuals with AI had greater deficits in both static and dynamic postural control after fatigue-induced running compared to a healthy group. Thus, these results provide evidence that individuals with AI may be more susceptible to the lower extremity injury during sport activity. Deficits in sensorimotor function may gradually change normal movement strategies into definitive patterns (e.g., greater ankle plantarflexion and inversion), inducing altered joint loads. However, previous studies have focused primarily on a component of sensorimotor function rather than outcomes of impaired sensorimotor function such as altered neuromuscular and biomechanical characteristics in the lower extremity during movement. Additionally, most studies have examined these variables (i.e., joint position/force sense, postural control) with static, nonweight-bearing measures rather than dynamic, functional activity including jump landings that involve directional changes. To date, no one has comprehensively examined the interaction of neuromuscular fatigue and AI on lower extremity muscle activity, kinematics, and kinetics
during movements that are representative of those performed in a competitive athletic environment. Deficits associated with AI and fatigue could be additive in increasing the risk for injury in patients with AI. Such a study could also provide insight into mechanisms and rehabilitation programs associated with AI. The purpose of this study was to investigate the interaction of neuromuscular fatigue and AI on lower extremity muscle activity, lower extremity joint mechanics and motion, and ground reaction force during a forward-side jump task.

Methods

Study Design

This study employed a cross-sectional controlled laboratory design in which the AI and control groups reported to the research laboratory for a single data collection session. A pretest, posttest repeated measures statistical design was used to assess differences in the following dependent variables: ankle, knee and hip joint angles, and internal net joint moments, three dimensional ground reaction forces (GRF) and corresponding impulses, and electromyography (EMG) amplitude. These variables were measured during the stance phase of a forward-side jump task before and after fatiguing exercise.

Subjects

Fifty subjects (25 AI and 25 control) participated in this study (Table 1). Subjects were classified into either the AI or control group via the Foot and Ankle Ability Measure (FAAM) and the Modified Ankle Instability Index (MAII) questionnaires. The FAAM consists of the activities of daily living (FAAM-ADL), and sports subscales (FAAM-Sports) (Appendix 1). Inclusion criteria for the AI group consisted of 1) a FAAM-ADL score that was < 75; 2) a FAAM-Sports score that was < 25; 3) at least 2 “yes” answers on questions 4-8 of the MAII; 4) a previous history of at least two acute unilateral ankle sprains, but no significant injury to the
ankle in the previous 3 months; 5) no lower extremity surgery and fracture. The inclusion criteria for the control group consisted of 1) a score of 100% on FAAM-ADL, FAAM-Sports, and MAII; 2) no “yes” answers on questions 4-8 of the modified MAII; 3) no ankle sprain in the most recent 3 years, and; 4) no previous history of surgery in the lower extremity. Control subjects were matched to AI subjects based on age (± 5 years), height (± 5 cm), mass (± 4.5 kg), and gender. Means and standard deviations for self-reported ankle instability questionnaires are reported in Table 2. Control subjects’ involved ankle was determined by matching involved ankle of corresponding AI subjects. Before participation, all subjects reviewed and signed an informed consent form approved by the University’s Institutional Review Board.

Instrumentation

Surface electromyography (sEMG) data were collected at 2500 Hz via a 16-channel Trigno Wireless EMG system (Delsys, Boston, MA). The electrode contacts were made from four 99.9% pure silver bars that are 5mm in length and spaced 10mm apart. The analog EMG data was filtered using standard band-pass real-time processing with cutoffs of 20 and 450 Hz. The common mode rejection ratio was > 80 dB and a gain of 1000 was used for all channels. GRF was recorded (2500 Hz) using an AMTI force platform (OR6-6-1, Advanced Mechanical Technology, Inc., Watertown, MA) embedded in the laboratory floor. Three-dimensional trajectories for reflective markers were determined using ten high-speed video cameras (250 Hz VICON, Oxford Metrics Ltd., Oxford, UK). GRF, kinematic, and sEMG data were synchronized via the VICON hardware. For data collection, subjects dressed in spandex clothing and also wore a standard shoe (Nike T-Lite V RX, Beaverton, OR) during study participation. A Vertec vertical jump tester (Sports Imports, Hilliard, OH) was used to measure the maximum vertical jump height (MVJH).
Procedures

All subjects participated in one testing session. The first part of the session consisted of completing the questionnaires, recording anthropometric data (AI group: 23.3 ± 1.9 yrs, 176.5 ± 10.5 cm, 70.9 ± 11.4 kg; control group: 23.7 ± 2.5 yrs, 175.0 ± 10.8 cm, 70.3 ± 12.8 kg), reviewing data collection procedures with the subjects, and obtaining informed consent. Next, shoes were donned and sEMG electrodes were placed on the involved leg over the following muscles: tibialis anterior (TA), peroneus longus (PL), medial gastrocnemius (MG), medial hamstrings (MH), vastus lateralis (VL), adductor longus (AL), gluteus medius (GMed), and gluteus maximus (GMax). Then the skin was prepped and the electrodes were applied using previously recommended guidelines. The sEMG electrodes were secured with stretch tape. Then the 59 reflective markers were applied to the subject. Single markers were placed on the following landmarks: sternum, acromion processes, C7 spinous process, inferior angles of the scapulae, T7 spinous process, lateral humeral epicondyles, dorsal wrists (mid-styloid processes), posterior-superior iliac spines (PSIS), anterior-superior iliac spines (ASIS), greater trochanters, medial femoral condyles, lateral femoral condyles, medial malleoli, lateral malleoli, posterior heels (three markers), dorsal foot (between base of second and third metatarsals), dorsal 2nd metatarsal heads, 5th metatarsal bases, and insteps (naviculars). Rigid 4-marker clusters were placed on both thighs and shanks, and a 4-marker cluster headband was donned.

After attaching the markers, subjects were instructed to warm-up on a treadmill for 5 minutes prior to data collection. Then two static postures were recorded for 3 seconds. First, an investigator measured subjects’ knee flexion angle with a goniometer and asked them to maintain a position of 45-degree knee flexion for 3 seconds. The sEMG was collected from eight muscles, while the subjects squatted (isometric reference position). The sEMG collected
during this time was used as the reference sEMG amplitude to normalize the subsequently collected sEMG. Second, a static standing posture was recorded; all joint kinematics during this study were reported, relative to this position. Subjects stood with their feet shoulder-width apart and held their arms out to the side parallel with the ground. Next, dynamic trials were collected to estimate functional hip joint center by having subjects perform\textsuperscript{54,55} three forward leg swings (hip flexion and extension) and three lateral swings (hip abduction and adduction) with the knee extended. After all standing trials, ankle and knee joint markers were detached for a forward-side jump task. Then subjects performed three maximum vertical jump trials. The highest vertical jump was recorded as their MVJH.

After establishing MVJH, subjects performed five trials of a forward-side jump on the force plate. Subjects jumped forward 1m to the center of the force plate at a height that was greater than half their shank length, landed on the involved leg, and immediately jumped in the direction of the contralateral side. A rubber band was placed between the subjects’ initial jumping position and the force plate to enforce the jump height in each trial before and after fatiguing protocol. After the prefatigue, forward-side jump trials, each participant underwent a fatigue protocol. They were given verbal encouragement throughout the fatiguing exercises. After the fatigue protocol, subjects immediately performed five trials of the forward-side jump on the force plate.

\textit{Fatigue Protocols}

Neuromuscular fatigue was induced by three fatiguing exercises: 5-minute incremental running, 20 seconds of lateral counter movement jumps (LCMJ), and 20 repetitions of vertical counter movement jumps (VCMJ). During the 5-minute warm-up, the initial treadmill speed was set based on Borg’s RPE scale,\textsuperscript{56} corresponding to subjects’ RPE scale level of 11 (Fairly
light). They began a 5-minute incremental running regimen on the treadmill and the initial speed was increased at a rate of 0.5 mph every minute. After 5 minutes of running, subjects performed 20 seconds of LCMJs with a given cadence of 88 ground contacts per minute, and 20 continuous VCMJs. Ten-second rests were given between fatiguing exercises. After all three fatiguing exercises, subjects immediately performed one trial of maximum vertical jump with a Vertec Vertical Jump device. Fatigue was assessed based on MVJH and Borg’s RPE scale. They completed as many cycles of 5-minute incremental running, 20-second LCMJs, and 20 repetitions of VCMJs until at the end of cycle the subjects’ RPE scale reached 17 (very hard) and the vertical jump height fell below 80% of their MVJH. In this study, fatigue assessment was based on subjective (Borg’s 6 to 20 rate of RPE scale) and objective (reduction in MVJH) measures of neuromuscular fatigue. A previous study showed a good relation between Borg’s RPE level of 17 and a blood lactate concentration (about 7 mmol/L) during exhaustive treadmill running.\textsuperscript{48,57}

\textit{Data Analysis}

In this study, all outcome variables were limited to the stance phase of a forward-side jump task, which was defined as the time on the plate. A 25-N GRF threshold was used to determine initial foot contact and takeoff. “Landing” was defined as the time from initial foot contact to peak knee flexion, while “take-off” was considered the time from peak knee flexion until the foot left the plate.

The raw three-dimensional coordinate data for each reflective marker, for all trials, were digitized using VICON Nexus software (VICON, Oxford Metrics Ltd., Oxford, UK) and then exported to Visual 3D software (C-Motion, Germantown, MD). The coordinate data was then filtered using a 4th order low-pass Butterworth filter with a cut-off frequency of 10 Hz. This cut-
off frequency was determined to be appropriate using a residual analysis technique. After the trajectories were smoothed, a previously described three-dimensional lower-extremity model was created using the static calibration and assigned to all motion trials. This model was used to calculate ankle, knee, and hip joint angles, using a Cardan rotation sequence (X, Y’, Z”). The ankle joint angles were determined as the rotation of Cardan angles of the foot relative to the tibia in an order of (1) plantar-dorsiflexion (x-axis), (2) internal-external (y-axis), and (3) inversion-eversion (z-axis). At the knee, the order of Cardan angles rotation was (1) flexion-extension (x-axis), (2) valgus-varus (y-axis), and (3) internal-external rotation (z-axis). At the hip, the order of Cardan angles rotation was (1) flexion-extension (x-axis), (2) adduction-abduction (y-axis), and (3) internal-external rotation (z-axis).

Synchronized GRF data were also exported to Visual 3D software to facilitate the calculation of joint moments. Three-dimensional net internal joint moments were calculated from the synchronized kinematic, GRF, and anthropometric data using a standard inverse dynamics approach. Three dimensional impulses were calculated during the stance phase of the forward-side jump task by integrating the GRF × time curve, using the trapezoid rule in MatLab (Mathworks, Natick, MA). Synchronized sEMG data were considered from initial foot contact to take-off. The sEMG amplitude was smoothed using the root mean square (RMS) method and a 125-ms window. All sEMG amplitudes were normalized to the aforementioned sEMG reference values. All sEMG data processing was done in MatLab.

Statistical Analysis

A functional data analysis of variance (FANOVA), which was a Bayesian approach similar to principle component analysis, was used. In these approaches, thousands of data points
were compared to a function (a curve of data which represented a normal set of data or “function”), and differences were identified where the curve plus its effect size (in this case 95% confidence intervals) were not equal to zero. These analyses are powerful in their ability to detect whether differences are meaningful and where, in a large data set, differences exist. In this research, functional linear models ($p = 0.05$) were used to evaluate differences between two different conditions (pre- vs postfatigue) for ankle, knee, and hip kinematic, kinetic, EMG, and GRF variables during stance phase of a forward-side jump in subjects with and without AI. In addition, a mixed-design repeated measures FANOVA ($2 \times 2$) was used to test for the main effect and group (AI vs control) by fatigue (pre- vs postfatigue) interactions on the outcome variables of sEMG amplitude, sagittal- and frontal-plane ankle and knee joint angles, moments, and GRFs. An exploratory alpha level of 0.05 was used to determine statistical significance in all comparisons. We used the “fda package” in the statistical program “R” (version 2.15.1) for all functional data analyses. A repeated measures ANOVA for impulses, due to GRF, was conducted in SPSS for Windows, Version 17.0 (SPSS, Inc., Chicago, IL).

Results

Fatigue Protocols

Summary data of fatiguing exercises are presented in Table 3. The average RPE levels in AI (18.2 ± 1.1) and control (17.8 ± 0.7) groups were greater than 17 (very hard) and vertical jump height of all subjects fell below 80% of their maximum vertical jump height immediately after the last cycle of fatigue protocol. There was no statistically significant difference in cycles of fatiguing exercises (AI: 3.8 ± 2.0 vs control: 4.2 ± 2.2) and mean time to fatigue (AI: 27.7 ± 14.5 vs control: 29.6 ± 13.8 min) between AI and control groups.
Joint Angles

For sagittal-plane kinematics at the ankle joint, dorsiflexion angle increased during 85-98% of stance, due to fatigue in the AI group (Figure 1A-B). For the control group, dorsiflexion angle increased during 4-100% of stance phase due to fatigue (Figure 1C-D). We found a group by fatigue interaction ($p < 0.05$) during 11-18% and 66-90% of stance phase during a forward-side jump (Figure 1E). However, no group-by-fatigue interaction was found during stance phase of a forward-side jump in frontal-plane ankle angles (Figure 2E).

For sagittal-plane kinematics at the knee joint in the AI group, fatigue decreased knee flexion angle at initial foot contact (0-7% of stance) and increased between 70-100% of stance (Figure 3A-B). Knee-flexion angle was decreased during 0-8% and increased between 17-100% of stance in the control group following fatigue (Figure 3C-D). A group-by-fatigue interaction ($p < 0.05$) was found during 23-30% of stance (Figure 3E). There was no group-by-fatigue interaction during the stance phase of a forward-side jump in frontal-plane knee angles (Figure 4E).

For sagittal-plane hip joint kinematics, fatigue increased hip flexion angle between 85-100% of stance in the AI group (Figure 5A-B). Hip flexion angle was increased during 13-100% of stance in the control group following fatigue (Figure 5C-D). A significant group-by-fatigue interaction ($p < 0.05$) was found between 21-23% of stance in hip flexion angle (Figure 5E). However, no group-by-fatigue interaction was found during stance phase of a forward-side jump in frontal-plane hip angles (Figure 6E).

Joint Moments

Eversion moment was decreased between 83-100% of stance phase in the AI group following fatigue (Figure 8A-B). Compared to the AI group, a significant decreased eversion
moment was found during 35-50% of stance between pre- and postfatigue in the control group (Figure 8C-D). A group-by-fatigue interaction ($p < 0.05$) was observed for frontal-plane kinetics at the ankle joint during 85-95% of stance phase during a forward-side jump (Figure 8E).

For sagittal-plane kinetics at the hip joint, fatigue decreased the hip extension moment between 25-30% of stance in the AI group (Figure 11A-B). In contrast to the AI group, the hip extension moment was increased during 13-20% of stance in the control group following fatigue (Figure 11C-D). A significant group-by-fatigue interaction ($p < 0.05$) was found between 20-26% of stance in the hip extension moment (Figure 11E). No significant group by fatigue interactions ($p = 0.05$) existed in plantarflexion (Figure 7E), knee extension (Figure 9E), knee valgus/varus (Figure 10E), and hip adduction/abduction (Figure 12E) moments.

**Ground Reaction Forces**

Fatigue decreased anterior-posterior ground reaction force (AP GRF) during 5-90% and 6-83% of stance in AI (Figure 13A-B) and control (Figure 13C-D) groups respectively. A group-by-fatigue interaction ($p < 0.05$) in AP GRF was found between 37-65% of stance phase (Figure 13E). However, there were no group-by-fatigue interactions in medial-lateral (Figure 14E) and vertical (Figure 15E) GRFs.

**Impulses**

No group-by-fatigue interactions were observed in all components of impulses. There were significant fatigue effects in all three impulses ($p < 0.05$). No group main effect was observed (Table 4).

**EMG Amplitude**

For the peroneus longus (PL) muscle, fatigue decreased EMG amplitude of PL between 0-70% of stance in the AI group (Figure 17A-B). PL EMG amplitude was decreased during 17-
50% of stance in the control group following fatigue (Figure 17C-D). A significant group-by-fatigue interaction \((p < 0.05)\) was found between 0-4% of stance in PL muscle (Figure 17E).

VL EMG amplitude was increased during 5-30% of stance phase in the AI group following fatigue (Figure 19A-B) while no significant difference was found in VL EMG amplitude between pre- and postfatigue in the control group (Figure 19C-D). We found a group-by-fatigue interaction \((p < 0.05)\) between 5-25% of stance phase during a forward-side jump (Figure 19E).

For the medial hamstring (MH) muscle, fatigue decreased MH EMG amplitude during 0-14% and 25-73% of stance phase in the AI group (Figure 20A-B). In the control group, MH EMG amplitude was decreased between 23-67% of stance phase following fatiguing exercises (Figure 20C-D). A significant group-by-fatigue interaction \((p < 0.05)\) was observed between 0-9% of stance phase during a forward-side jump (Figure 20E).

In the gluteus medius (GMed) EMG amplitude, the AI group demonstrated decreased GMed EMG amplitude during 0-5%, 40-42% and 90-100% of stance phase (Figure 22A-B). In the control group, EMG amplitude of GMed was decreased between 82-100% of stance phase (Figure 22C-D). We observed a group-by-fatigue interaction \((p < 0.05)\) during 30-35% and 80-85% of stance phase during a forward-side jump (Figure 22E).

For the gluteus maximus (GMax) muscle, in the AI group, fatigue increased EMG amplitude of GMax between 3-24% of stance (Figure 23A-B). EMG amplitude of GMax was decreased during 0-3% and 37-55% of stance in the control group following fatigue (Figure 23C-D). A group-by-fatigue interaction \((p < 0.05)\) was found during 4-11%, 35-53%, and 80-90% of stance (Figure 23E).
We did not observe significant group-by-fatigue interactions \((p = 0.05)\) in EMG amplitude of the tibialis anterior (Figure 16E), medial gastrocnemius (Figure 18E), or adductor longus (Figure 21E) muscles during forward-side jump tasks.

**Discussion**

The major finding of this study is that patients with AI demonstrated different neuromechanical patterns in response to neuromuscular fatigue. Compared to the control group, the AI group exhibited a lower range of dorsiflexion, knee and hip flexion and decreased hip extensor moments during the landing phase of a forward-side jump following fatiguing exercises. Individuals with AI decreased EMG activation of PL, MH, and GMed while increasing activation of VL and GMax during landing after fatigue compared to control subjects. These altered neuromechanical patterns resulted in a greater reduction of AP GRF in the control group than the AI group after functional fatiguing exercises.

**Kinematics**

The control group significantly increased dorsiflexion during the entire stance phase of a forward-side jump following fatiguing exercises (Figure 1C-D). However, no significant change in dorsiflexion due to fatigue was observed during the stance phase of a forward-side jump in the AI group except for the end of the take-off phase (85-98% of stance) (Figure 1A-B). A previous study showed increased dorsiflexion during landing following neuromuscular fatigue. In this study, the ankle was 3.3 degrees more dorsiflexed at the initial foot contact of a single-leg landing during the last cycle of a fatigue protocol. Landing with more dorsiflexion increases ankle stiffness, resulting in greater joint stability. Individuals may use this strategy to maintain stability without having to use as much muscle contraction following fatigue. Increased dorsiflexion is a more stable, closed-packed position of the ankle, providing joint stability by
increasing the articular constraints of the ankle joint.\textsuperscript{64} This may be a protective mechanism; an effort by the individual to compensate for a reduced force of the plantarflexors, preventing excessive supination at foot contact. In addition to the fatigue effect, the AI group did not increase dorsiflexion as much as the control group did after fatigue during landing (Figure 1E). Previously, subjects with AI were shown to exhibit less dorsiflexion during single-leg drop jumps\textsuperscript{65} and treadmill walking and jogging\textsuperscript{21} compared to control subjects. The AI group had about 3 degrees greater plantarflexion from 42 to 51% of the gait cycle (mid to late stance) and less dorsiflexion during the mid-swing phase of jogging compared to the control group.\textsuperscript{21} The lack of dorsiflexion during walking and jogging may be attributed to arthrokinematic restrictions at the talocrural joint.\textsuperscript{21} One possible mechanism of less dorsiflexion in the AI group could be a limited range of motion in posterior talar glide after repetitive inversion ankle sprains.\textsuperscript{65} Therefore, the AI group, with more limited range into the closed-packed position, may be more vulnerable to lateral ankle sprains after neuromuscular fatigue compared to control subjects due to lack of dorsiflexion.

The AI group demonstrated less knee flexion during the landing phase compared to control subjects after functional fatiguing exercises (Figure 3E). A recent study showed that subjects with AI demonstrated less knee flexion during a vertical stop jump task compared to control subjects.\textsuperscript{66} AI is attributed to impaired proprioception and sensorimotor function, leading to deficits in postural and neuromuscular control patterns.\textsuperscript{67,68} Like AI, neuromuscular fatigue may contribute to a reduction in neuromuscular function\textsuperscript{69-71} resulting in altered movement strategies in the lower extremity during landing.\textsuperscript{62,72,73} For these reasons, patients with AI displayed greater fatigue-induced alterations in dynamic postural control compared to normal subjects.\textsuperscript{48,50} Therefore, our finding may indicate that sagittal-plane knee joint kinematics were
more impaired (less displacement into knee flexion) by neuromuscular fatigue in subjects with AI, and they might be more predisposed to lower-extremity injury during activity compared to normal subjects. While no significant group-by-fatigue interaction was found at the initial foot contact of landing, both groups demonstrated a similar landing strategy at the knee joint following fatigue. Both groups decreased knee flexion angle at initial foot contact following functional fatiguing exercises (Figure 3B-D). Similar to the more dorsiflexed ankle, decreased knee flexion may result in increased dependence on skeletal structures to support body mass during a landing task. This may be a compensatory strategy, due to fatigue, resulting from decreased eccentric muscular strength of the knee extensors that are required to control knee flexion during landing. These results are consistent with previous studies examining lower extremity joint angles during initial foot contact following various fatigue protocols. Decreased knee flexion during initial landing could predispose an individual to injury. A previous study suggested that neuromuscular fatigue may result in a more extended knee at the point of peak tibial anterior shear force during a stop-jump task following a series of vertical jumps and 30m sprints. Decreasing knee flexion angle theoretically increases anterior tibial shear force due to the corresponding increase in patella tendon-tibia angle and increased anterior tibial translation due to the quadriceps muscle force; all of which may consequently increase anterior cruciate ligament (ACL) injury risk. In other words, knee flexion angle significantly affects the contribution of anterior shear force to ACL loading, with an increased knee flexion angle resulting in a decrease in ACL loading. In the current study, the knee joint adopted a landing strategy, immediately changing stiff- (extended knee) to soft-landing (flexed knee) during a forward-side jump following neuromuscular fatigue in control group while subjects with AI retained an extended knee posture during landing phase after fatigue. Therefore,
Neuromuscular fatigue is more likely to put AI subjects at knee-joint injury risk than normal subjects.

Neuromuscular fatigue appears to alter the way that control subjects increased hip flexion angle during landing compared to AI subjects (Figure 5E). The present results showed that the control subjects changed from an ankle strategy to a hip strategy, as a result of fatigue; however, the AI subjects did not change as a result of fatigue. Neuromuscular fatigue increased hip flexion angle and changed energy absorption strategy from an ankle to hip joint strategy during single-leg landing tasks. After fatigue, lower extremity joints necessitated kinematic compensatory landing strategies to redistribute postfatigue load by translating from distal to proximal joint motions. With a flexed hip posture, hip extensor muscles (e.g., gluteus maximus) may have optimal length to effectively absorb joint loads during landing to reduce the joint load applied to knee and ankle musculature. The increased range of motion and work at the hip with fatigue indicates a greater reliance on the larger muscles, representing a distal-to proximal redistribution of work. Our ankle and knee kinematic findings show that control subjects demonstrated more close-packed joint positions during the initial phase of landing following neuromuscular fatigue compared to AI subjects. A soft landing strategy, which is characterized by increased knee and hip flexion angles, reduced ground reaction force during mid-phase of landing, resulting in less joint-loading to knee and hip joints. Joint kinematic patterns were changed as fatigue progressed in a way that redistributed energy absorption in the lower extremity from the ankle to the hip. Thus, increased knee and hip flexion, after fatigue, may be an effort to compensate for dorsiflexed ankle position to attenuate shock by dissipating ground reaction force to proximal lower extremity joints during landing. Increased hip flexion suggests that the hip joint plays an important role in decreasing the load of other joints in the lower
extremity following fatigue, perhaps due to the mechanical advantages of its surrounding musculature (greater cross-sectional area, longer muscle fibers, and relatively shorter tendons) compared to that of the knee and ankle joints. Instead of joint configurations, it is knee and hip joint motions that play an important role in reducing the ground reaction forces and knee-joint resultant force during a stop-jump task. Thus, in the present study, the control group who increased range of knee and hip flexion angles after fatigue may reduce the impact forces during mid-phase of landing. Unlike the control group, AI subjects were not able to effectively modify landing strategy from distal (ankle joint) to proximal (hip joint) redistribution of joint angles. Thus a stiffer landing may place greater load on passive structures around lower-extremity joints in the AI group following fatigue compared to the control group.

**Kinetics**

In joint moments, the only significant group-by-fatigue interaction existed in the hip extension moment (Figure 11E). For sagittal-plane hip kinetics, subjects with AI decreased the hip extension moment (Figure 11B) while control subjects increased hip extension moments during the mid-phase of landing following functional fatiguing exercise (Figure 11D). As previously mentioned, the hip joint plays an important role in the dissipation of impact forces. At the hip joint, hip extension moments should be produced by eccentric muscle contractions to control joint motion and to absorb the kinetic energy of the body during landing. A recent study showed that improvement in the strength of the hip extensors changed biomechanical landing strategies, resulting in greater hip flexion angles and increased hip extension moments. Increased hip extension moments allowed for an increased hip flexion angle which may decrease the reliance of the knee extensors to eccentrically control deceleration of landing and cutting. Our findings suggest that exercise-induced decreases in hip extensor moments in the AI group
may not be able to increase hip joint angle, resulting in a stiffer landing pattern compared to the control group. Further, more data are needed to determine if hip extensor training may affect the incidence of reinjury in an AI population.

For AP GRF, the AI group showed less reduction of AP GRF following neuromuscular fatigue compared to control subjects (Figure 13E). As previously mentioned, lower extremity injury frequently occurs during the deceleration phase of landing. This is why we chose a forward-side jump which included sudden deceleration and change in direction. The amount of AP GRF may represent sudden deceleration of the forward jump. Subjects with AI have previously demonstrated greater AP GRF after initial foot contact during a single-leg drop jump compared to control group. AP GRF was the major contributing factor of peak anterior shear force in an AI group during a vertical stop-jump task, landing with less knee flexion. Our data are consistent with these previous findings. Subjects with AI who land with increased AP GRF may be at increased risk for injuries linked to increased anterior shear forces, like ACL injuries. In addition, decreased dorsiflexion, knee and hip flexion may contribute to increased AP GRF due to the position of the center of mass relative to base of support. This finding (less reduction in AP GRF in AI subjects) is consistent with the findings of reduced dorsiflexion, knee and hip range of motion (ROM), and these findings, together, may provide some evidence as to why patients with AI are susceptible to reinjury. Thus, rehabilitation and preventative exercise protocols for subjects with AI should include a landing strategy with increased ROM of dorsiflexion, knee and hip flexion under fatigued conditions. A proper landing strategy after fatigue may reduce AP GRF, resulting in decreased joint loads thereby reducing risk of injury. A previous study that examined effects of fatigue on ground impact forces and joint biomechanics during landing suggested that alterations in landing strategy (increased ROM and a distal-to-
proximal redistribution of extensor moments) may attempt to reduce impact forces (decreased GRFs and impulses).\textsuperscript{83} We think that it is likely that kinetic alteration at the hip joint may be a protective mechanism that can potentially decrease GRF in response to neuromuscular fatigue during landing.

\textit{EMG Amplitude}

At the initial foot contact of stance phase, subjects with AI demonstrated decreased PL EMG amplitude from pre- to postfatigue compared to control subjects (Figure 17E). The peroneal muscles are the primary evertors of the ankle, providing dynamic ankle stability.\textsuperscript{19} Thus, inactivity of these muscles may result in an inability to control sudden and excessive inversion force, resulting in a possible lateral ankle sprain during functional activities.\textsuperscript{84} Other studies have reported that subjects with AI exhibited diminished EMG amplitude of the peroneal muscle during a sudden inversion perturbation during walking,\textsuperscript{85} a lateral shuffle maneuver,\textsuperscript{86} and stop-jump task,\textsuperscript{87} as well as during eccentric contraction of peroneal muscles with an isokinetic dynamometer\textsuperscript{88} compared to control subjects. Neuromuscular fatigue compromises postural control during single-leg standing, indicating sensorimotor deficits.\textsuperscript{43,89,90} Previous research identified a relationship between shank muscle activity patterns and postural sway in a fatigued condition, showing reduced EMG activation of ankle musculature following fatigue.\textsuperscript{91} This suggests that subjects with AI may experience deficits in dynamic stability of the ankle joint during landing compared to control subjects.

Many recent studies have focused on proximal neuromuscular alterations in subjects with AI such as muscle activity of knee and hip musculatures\textsuperscript{92,93} because ankle musculature alone is not enough to provide dynamic ankle joint stability to prevent ankle injury during functional activities.\textsuperscript{94} Subjects with AI exhibited neuromuscular changes of proximal joints.\textsuperscript{66,95,96}
Consistent with the proposed hypotheses, the results of our current study show that subjects with AI demonstrated different EMG activation patterns in proximal musculature during a forward-side jump following functional fatiguing exercises compared to the control group. For knee joint muscles, the AI group demonstrated an increase in VL EMG amplitude (Figure 19E) but a decrease in MH EMG amplitude (Figure 20E) during the initial phase of landing following neuromuscular fatigue compared to the control group. Quadriceps and hamstring muscles provide dynamic stability for the knee joint during physical activity and exercise-induced neuromuscular alterations of these muscles may increase lower-extremity injury risk. Our findings of decreased MH EMG amplitude (Figure 20B and 20D) is consistent with findings of another study, showing that simulated handball match-play reduced EMG amplitude of hamstring muscles during initial landing phase of a side-cutting maneuver. In addition, the current study showed subjects with AI increased VL EMG amplitude (Figure 19B) while decreasing MH EMG amplitude (Figure 20B) following functional fatiguing exercises. This is again consistent with findings of a separate study that reported AI patients demonstrated different arthrogenic muscle responses in the quadriceps and hamstring muscle groups: arthrogenic inhibition of hamstring and facilitation of quadriceps. Therefore, increased VL activation with decreased MH activation patterns may increase quadriceps/hamstring ratios, indicating a quadriceps dominant strategy. This may also lead to increased risk of knee injury, such as ACL sprains, following muscle fatigue.

For the hip joint muscles, the AI group showed greater deficits in GMed EMG amplitude during a forward-side jump following fatigue compared to the control group (Figure 22E). In contrast with GMed activation, the AI group had a greater increase in GMax EMG amplitude compared to the control group following neuromuscular fatigue (Figure 23E). A number of
studies have reported that proximal muscle weakness (i.e., the gluteal muscles) was a result or cause of a lateral ankle sprain. The gluteus medius (primary hip abductor) plays a key role in maintaining balance and postural control and helps ankle muscles to prevent excessive lateral sway during walking, running, and jump landing. For these reasons, decreased GMed activation may cause greater lateral sway, leading to a supinated foot position which is more susceptible to a lateral ankle sprain. Thus, our findings suggest that subjects with AI might experience increased risk of recurrence of lower-extremity injury during sport activities. A previous study showed that subjects with AI demonstrated lower GMax activation during functional activities compared to healthy subjects. This result suggests that patients with AI were not able to use GMax properly to control hip joint motion during functional activities. This alteration in proximal neuromuscular control may be influenced by sensorimotor deficits at the ankle joint. Interestingly, our findings revealed fatigue-induced increases in GMax EMG amplitude in the AI group while no change in the control group was noted following fatigue. Increased recruitment of GMax may be compensation for impairment of hip extensor strength following neuromuscular fatigue. Our fatigue protocol (with running and squatting exercises) demands activation of hip extensors to control lower-extremity joint motions. Our pilot study showed evidence of GMax fatigue (decreased median frequency of GMax) following a fatigue protocol. In addition, single-leg squat and hop exercises required increased activation of GMax to control joint motions. For these reasons, exercise-induced increases in GMax activation in the AI group may be the result of muscle fatigue to compensate for insufficient muscle strength to improve neuromuscular control of landing. Our finding of decreased hip extensor moment is consistent with greater increases in GMax amplitude in the AI group. Our results suggest that subjects with AI showed different neuromuscular control strategies in proximal musculatures
during functional activity following neuromuscular fatigue. Findings of these studies are indicative of central neuromuscular deficits as well as peripheral sensorimotor deficits in subjects with AI.¹⁰¹

In the current study some EMG data did not support kinematic and/or kinetic data. The current study is a comprehensive study of lower-extremity neuromechanics during a functional activity. EMG has been widely used to evaluate muscle activity which might assess the contribution of the muscles at a given phase of joint motions and forces. EMG findings in conjunction with kinematic and kinetic results become more powerful when interpreting movement patterns in research settings as well as clinical settings. Despite the benefits of EMG, there should be discretion with the use of EMG data synchronized with joint torque due particularly to electromechanical delay (EMD), which refers to the phase shift between the EMG onset (electrical activity) and force production (mechanical response).¹⁹,¹⁰⁵ For example, EMD was elongated about 18.7ms after isometric fatiguing exercises of vastus lateralis¹⁰⁶ and EMD increased about 14.2ms after intermittent isometric knee flexor.¹⁰⁷ Furthermore, a study by Hopkins et al. revealed that subjects with AI increased peroneal EMD following inversion perturbation during walking compared to normal subjects.¹⁹ This may be attributed to decreased nerve conduction velocity¹⁰⁸ resulting from traction lesions with initial or repetitive ankle sprains. Future studies might account for EMD to accurately interpret the relationship between muscle activity and biomechanical movement patterns in subjects with AI after neuromuscular fatigue. Further, trunk position may alter lower extremity kinematics and kinetics during a single-leg landing.¹⁰⁹ Identifying trunk motions might explain why a few neuromuscular patterns didn’t fit with lower extremity biomechanical patterns in the current study.
There were several limitations to this study. First, as the current study is a retrospective design, the causality between altered neuromechanical patterns and higher risk of lower-extremity injury is not allowed when interpreting these findings. Consequently, a prospective study should be conducted to identify the cause-effect relationship in AI. Second, our study analyzed sagittal- and frontal-plane biomechanical variables during a forward-side jump which is a three-dimensional motion. Thus, in addition to sagittal and frontal planes, future research should examine landing biomechanics in the transverse plane to better understand neuromechanical alterations in patients with AI. Third, we did not consider upper-body (trunk) motion, which may have affected the results and could account for the disparity between EMG findings and kinematic/kinetic findings. Lastly, while our functional fatigue protocol tried to mimic sport activities including running and jump-landing with stretch-shortening cycles, it still does not capture all aspects of actual sport events.

Conclusions

Subjects with AI demonstrated different neuromechanics than control subjects during a forward-side jump following neuromuscular fatigue. Compared to the control group, the AI group did not show a coordinated joint control strategy during a forward-side jump; i.e., increasing joint angles from distal (ankle) to proximal (hip) joints to compensate for muscle fatigue in an attempt to reduce ground impact force. It appears that subjects with AI showed greater fatigue-induced neuromechanical alterations of proximal joints. In addition, EMG alterations were consistent with patterns observed in injured patients, which may predispose patients to poor positions associated with lower extremity joint injury. These results suggest that subjects with AI have greater impairments in neuromechanical control patterns than a matched
control group during a sport movement as fatigue progresses. These interactions between neuromuscular fatigue and AI may predispose individuals to lower extremity injuries.
References


Table 1. Subject demographic data

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<th>Group</th>
<th>Age (Years)</th>
<th>Height (cm)</th>
<th>Mass (kg)</th>
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<tr>
<td></td>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
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<td>AI</td>
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<td>176.5 (10.5)</td>
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<td>Control</td>
<td>23.7 (2.5)</td>
<td>175 (10.8)</td>
<td>70.3 (12.8)</td>
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Table 2. Ankle instability questionnaire scores in AI and control groups

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<tr>
<th>Group</th>
<th>FAAM-ADL (%)</th>
<th>FAAM-Sport (%)</th>
<th>MAII</th>
<th># of Ankle Sprain</th>
<th>Duration (Month)</th>
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<tr>
<td></td>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
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<td>AI</td>
<td>84.3 (7.6)</td>
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<td>3.7 (1.2)</td>
<td>4.8 (3.0)</td>
<td>20.1 (18.3)</td>
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<td>100 (0.0)</td>
<td>100 (0.0)</td>
<td>0.0 (0.0)</td>
<td>0.4 (1.0)</td>
<td>80 (58.1)</td>
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Table 3. Summary of fatigue protocol

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<th>Group</th>
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<th>Time to fatigue (min) Mean (SD)</th>
<th>RPE (level) Mean (SD)</th>
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<td>17.8 (0.7)</td>
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Table 4. Means and standard deviations for three-dimensional impulses based on group (control/AI) by fatigue (pre-/postfatigue) ANOVAs

<table>
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<th>Impulse (N·s)</th>
<th>Prefatigue</th>
<th>Postfatigue</th>
<th>Group Effect (p)</th>
<th>Fatigue Effect (p)</th>
<th>Interaction (p)</th>
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<td>Control</td>
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<td>M-Impulse</td>
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<td>697 (126)</td>
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<td>V-Impulse</td>
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<td>1927 (472)</td>
<td>1754 (531)</td>
<td>1825 (433)</td>
<td>0.455</td>
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</table>

*Indicates statistically significant fatigue main effect (p < .05)
Figure 1. Plantar- and dorsiflexion (PF/DF) angle during stance phase of a forward-side jump before and after functional fatiguing exercises in AI (A) and control (C) groups. Mean difference in PF/DF angle in AI (B), and control (D) groups between pre- and postfatigue during stance phase of a forward-side jump. (E) represents group by fatigue interaction. Mean differences (bold solid curve) and corresponding 95% CI (shaded area) are plotted as a function of time. When the shaded area does not overlap with the zero line (bold horizontal dotted line), a significant difference (p < 0.05) is indicated between pre- and postfatigue. Vertical dotted line indicates peak knee flexion angle.
Figure 2. Inversion (IV) angle during stance phase of a forward-side jump before and after functional fatiguing exercises in Al (A) and control (C) groups. Mean difference in IV angle in Al (B), and control (D) groups between pre- and postfatigue during stance phase of a forward-side jump. (E) represents group by fatigue interaction. Mean differences (bold solid curve) and corresponding 95% CI (shaded area) are plotted as a function of time. When the shaded area does not overlap with the zero line (bold horizontal dotted line), a significant difference (p < 0.05) is indicated between pre- and postfatigue. Vertical dotted line indicates peak knee flexion angle.
Figure 3. Knee flexion (KF) angle during stance phase of a forward-side jump before and after functional fatiguing exercises in AI (A) and control (C) groups. Mean difference in KF angle in AI (B), and control (D) groups between pre- and postfatigue during stance phase of a forward-side jump. (E) represents group by fatigue interaction. Mean differences (bold solid curve) and corresponding 95% CI (shaded area) are plotted as a function of time. When the shaded area does not overlap with the zero line (bold horizontal dotted line), a significant difference (p < 0.05) is indicated between pre- and postfatigue. Vertical dotted line indicates peak knee flexion angle.
Figure 4. Knee valgus/varus (VL/VR) angle during stance phase of a forward-side jump before and after functional fatiguing exercises in Al (A) and control (C) groups. Mean difference in VL/VR angle in Al (B), and control (D) groups between pre- and postfatigue during stance phase of a forward-side jump. (E) represents group by fatigue interaction. Mean differences (bold solid curve) and corresponding 95% CI (shaded area) are plotted as a function of time. When the shaded area does not overlap with the zero line (bold horizontal dotted line), a significant difference ($p < 0.05$) is indicated between pre- and postfatigue. Vertical dotted line indicates peak knee flexion angle.
Figure 5. Hip flexion (HF) angle during stance phase of a forward-side jump before and after functional fatiguing exercises in Al (A) and control (C) groups. Mean difference in HF angle in Al (B), and control (D) groups between pre- and postfatigue during stance phase of a forward-side jump. (E) represents group by fatigue interaction. Mean differences (bold solid curve) and corresponding 95% CI (shaded area) are plotted as a function of time. When the shaded area does not overlap with the zero line (bold horizontal dotted line), a significant difference (p < 0.05) is indicated between pre- and postfatigue. Vertical dotted line indicates peak knee flexion angle.
Figure 6. Hip abduction (ABD) angle during stance phase of a forward-side jump before and after functional fatiguing exercises in Al (A) and control (C) groups. Mean difference in ABD angle in Al (B), and control (D) groups between pre-and postfatigue during stance phase of a forward-side jump. (E) represents group by fatigue interaction. Mean differences (bold solid curve) and corresponding 95% CI (shaded area) are plotted as a function of time. When the shaded area does not overlap with the zero line (bold horizontal dotted line), a significant difference (p < 0.05) is indicated between pre- and postfatigue. Vertical dotted line indicates peak knee flexion angle.
Figure 7. Plantarflexion (PF) moment during stance phase of a forward-side jump before and after functional fatiguing exercises in AI (A) and control (C) groups. Mean difference in PF moment in AI (B), and control (D) groups between pre- and postfatigue during stance phase of a forward-side jump. (E) represents group by fatigue interaction. Mean differences (bold solid curve) and corresponding 95% CI (shaded area) are plotted as a function of time. When the shaded area does not overlap with the zero line (bold horizontal dotted line), a significant difference (p < 0.05) is indicated between pre- and postfatigue. Vertical dotted line indicates peak knee flexion angle.
Figure 8. Eversion (EV) moment during stance phase of a forward-side jump before and after functional fatiguing exercises in AI (A) and control (C) groups. Mean difference in EV moment in AI (B), and control (D) groups between pre- and postfatigue during stance phase of a forward-side jump. (E) represents group by fatigue interaction. Mean differences (bold solid curve) and corresponding 95% CI (shaded area) are plotted as a function of time. When the shaded area does not overlap with the zero line (bold horizontal dotted line), a significant difference ($p < 0.05$) is indicated between pre- and postfatigue. Vertical dotted line indicates peak knee flexion angle.
Figure 9. Knee extension (KE) moment during stance phase of a forward-side jump before and after functional fatiguing exercises in AI (A) and control (C) groups. Mean difference in KE moment in AI (B), and control (D) groups between pre- and postfatigue during stance phase of a forward-side jump. (E) represents group by fatigue interaction. Mean differences (bold solid curve) and corresponding 95% CI (shaded area) are plotted as a function of time. When the shaded area does not overlap with the zero line (bold horizontal dotted line), a significant difference ($p < 0.05$) is indicated between pre- and postfatigue. Vertical dotted line indicates peak knee flexion angle.
Figure 10. Knee valgus/varus (VL/VR) moment during stance phase of a forward-side jump before and after functional fatiguing exercises in AI (A) and control (C) groups. Mean difference in VL/VR moment in AI (B), and control (D) groups between pre- and postfatigue during stance phase of a forward-side jump. (E) represents group by fatigue interaction. Mean differences (bold solid curve) and corresponding 95% CI (shaded area) are plotted as a function of time. When the shaded area does not overlap with the zero line (bold horizontal dotted line), a significant difference (p < 0.05) is indicated between pre- and postfatigue. Vertical dotted line indicates peak knee flexion angle.
Figure 11. Hip extension (HE) moment during stance phase of a forward-side jump before and after functional fatiguing exercises in AI (A) and control (C) groups. Mean difference in HE moment in AI (B), and control (D) groups between pre- and postfatigue during stance phase of a forward-side jump. (E) represents group by fatigue interaction. Mean differences (bold solid curve) and corresponding 95% CI (shaded area) are plotted as a function of time. When the shaded area does not overlap with the zero line (bold horizontal dotted line), a significant difference (p < 0.05) is indicated between pre- and postfatigue. Vertical dotted line indicates peak knee flexion angle.
Figure 12. Hip adduction/abduction (ADD/ABD) moment during stance phase of a forward-side jump before and after functional fatiguing exercises in AI (A) and control (C) groups. Mean difference in ADD/ABD moment in AI (B), and control (D) groups between pre- and postfatigue during stance phase of a forward-side jump. (E) represents group by fatigue interaction. Mean differences (bold solid curve) and corresponding 95% CI (shaded area) are plotted as a function of time. When the shaded area does not overlap with the zero line (bold horizontal dotted line), a significant difference (p < 0.05) is indicated between pre- and postfatigue. Vertical dotted line indicates peak knee flexion angle.
Figure 13. Anterior-posterior ground reaction force (AP GRF) during stance phase of a forward-side jump before and after functional fatiguing exercises in AI (A) and control (C) groups. Mean difference in AP GRF in AI (B), and control (D) groups between pre- and postfatigue during stance phase of a forward-side jump. (E) represents group by fatigue interaction. Mean differences (bold solid curve) and corresponding 95% CI (shaded area) are plotted as a function of time. When the shaded area does not overlap with the zero line (bold horizontal dotted line), a significant difference (p < 0.05) is indicated between pre- and postfatigue. Vertical dotted line indicates peak knee flexion angle.
Figure 14. Medial-lateral ground reaction force (ML GRF) during stance phase of a forward-side jump before and after functional fatiguing exercises in AI (A) and control (C) groups. Mean difference in ML GRF in AI (B), and control (D) groups between pre- and post-fatigue during stance phase of a forward-side jump. (E) represents group by fatigue interaction. Mean differences (bold solid curve) and corresponding 95% CI (shaded area) are plotted as a function of time. When the shaded area does not overlap with the zero line (bold horizontal dotted line), a significant difference \((p < 0.05)\) is indicated between pre- and postfatigue. Vertical dotted line indicates peak knee flexion angle.
Figure 15. Vertical ground reaction force (V GRF) during stance phase of a forward-side jump before and after functional fatiguing exercises in AI (A) and control (C) groups. Mean difference in V GRF in AI (B), and control (D) groups between pre- and postfatigue during stance phase of a forward-side jump. (E) represents group by fatigue interaction. Mean differences (bold solid curve) and corresponding 95% CI (shaded area) are plotted as a function of time. When the shaded area does not overlap with the zero line (bold horizontal dotted line), a significant difference (p < 0.05) is indicated between pre- and postfatigue. Vertical dotted line indicates peak knee flexion angle.
Figure 16. EMG amplitude for tibialis anterior (TA) during stance phase of a forward-side jump before and after functional fatiguing exercises in AI (A) and control (C) groups. Mean difference in EMG amplitude for TA in AI (B), and control (D) groups between pre- and post-fatigue during stance phase of a forward-side jump. (E) represents group by fatigue interaction. Mean differences (bold solid curve) and corresponding 95% CI (shaded area) are plotted as a function of time. When the shaded area does not overlap with the zero line (bold horizontal dotted line), a significant difference (p < 0.05) is indicated between pre- and post-fatigue. Vertical dotted line indicates peak knee flexion angle.
Figure 17. EMG amplitude for peroneus longus (PL) during stance phase of a forward-side jump before and after factional fatiguing exercises in Al (A) and control (C) groups. Mean difference in EMG amplitude for PL in Al (B), and control (D) groups between pre- and postfatigue during stance phase of a forward-side jump. (E) represents group by fatigue interaction. Mean differences (bold solid curve) and corresponding 95% CI (shaded area) are plotted as a function of time. When the shaded area does not overlap with the zero line (bold horizontal dotted line), a significant difference ($p < 0.05$) is indicated between pre- and postfatigue. Vertical dotted line indicates peak knee flexion angle.
Figure 18. EMG amplitude for medial gastrocnemius (MG) during stance phase of a forward-side jump before and after functional fatiguing exercises in AI (A) and control (C) groups. Mean difference in EMG amplitude for MG in AI (B), and control (D) groups between pre- and postfatigue during stance phase of a forward-side jump. (E) represents group by fatigue interaction. Mean differences (bold solid curve) and corresponding 95% CI (shaded area) are plotted as a function of time. When the shaded area does not overlap with the zero line (bold horizontal dotted line), a significant difference (p < 0.05) is indicated between pre- and postfatigue. Vertical dotted line indicates peak knee flexion angle.
Figure 19. EMG amplitude for vastus lateralis (VL) during stance phase of a forward-side jump before and after functional fatiguing exercises in AI (A) and control (C) groups. Mean difference in EMG amplitude for VL in AI (B), and control (D) groups between pre- and postfatigue during stance phase of a forward-side jump. (E) represents group by fatigue interaction. Mean differences (bold solid curve) and corresponding 95% CI (shaded area) are plotted as a function of time. When the shaded area does not overlap with the zero line (bold horizontal dotted line), a significant difference (p < 0.05) is indicated between pre- and postfatigue. Vertical dotted line indicates peak knee flexion angle.
Figure 20. EMG amplitude for medial hamstring (MH) during stance phase of a forward-side jump before and after functional fatiguing exercises in AI (A) and control (C) groups. Mean difference in EMG amplitude for MH in AI (B), and control (D) groups between pre- and postfatigue during stance phase of a forward-side jump. (E) represents group by fatigue interaction. Mean differences (bold solid curve) and corresponding 95% CI (shaded area) are plotted as a function of time. When the shaded area does not overlap with the zero line (bold horizontal dotted line), a significant difference (p < 0.05) is indicated between pre- and postfatigue. Vertical dotted line indicates peak knee flexion angle.
Figure 21. EMG amplitude for adductor longus (AL) during stance phase of a forward-side jump before and after functional fatiguing exercises in AI (A) and control (C) groups. Mean difference in EMG amplitude for AL in AI (B), and control (D) groups between pre- and postfatigue during stance phase of a forward-side jump. (E) represents a group by fatigue interaction. Mean differences (bold solid curve) and corresponding 95% CI (shaded area) are plotted as a function of time. When the shaded area does not overlap with the zero line (bold horizontal dotted line), a significant difference (p < 0.05) is indicated between pre- and postfatigue. Vertical dotted line indicates peak knee flexion angle.
Figure 22. EMG amplitude for gluteus medius (GMed) during stance phase of a forward-side jump before and after fictitional fatiguing exercises in Al (A) and control (C) groups. Mean difference in EMG amplitude for GMed in Al (B), and control (D) groups between pre- and postfatigue during stance phase of a forward-side jump. (E) represents group by fatigue interaction. Mean differences (bold solid curve) and corresponding 95% CI (shaded area) are plotted as a function of time. When the shaded area does not overlap with the zero line (bold horizontal dotted line), a significant difference (p < 0.05) is indicated between pre- and postfatigue. Vertical dotted line indicates peak knee flexion angle.
Figure 23. EMG amplitude for gluteus maximus (GMax) during stance phase of a forward-side jump before and after functional fatiguing exercises in AI (A) and control (C) groups. Mean difference in EMG amplitude for GMax in AI (B), and control (D) groups between pre- and postfatigue during stance phase of a forward-side jump. (E) represents group by fatigue interaction. Mean differences (bold solid curve) and corresponding 95% CI (shaded area) are plotted as a function of time. When the shaded area does not overlap with the zero line (bold horizontal dotted line), a significant difference (p < 0.05) is indicated between pre- and postfatigue. Vertical dotted line indicates peak knee flexion angle.