Quadriiceps and Hamstrings Fatigue Alters Hip and Knee Mechanics

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Neuromuscular fatigue exacerbates abnormal landing strategies, which may increase noncontact anterior cruciate ligament (ACL) injury risk. The synergistic actions of quadriceps and hamstrings (QH) muscles are central to an upright landing posture, though the precise effect of simultaneous fatigue of these muscles on landing and ACL injury risk is unclear. Elucidating neuromechanical responses to QH fatigue thus appears important in developing more targeted fatigue-resistance intervention strategies. The current study thus aimed to examine the effects of QH fatigue on lower extremity neuromechanics during dynamic activity. Twenty-five healthy male and female volunteers performed three single-leg forward hops onto a force platform before and after QH fatigue. Fatigue was induced through sets of alternating QH concentric contractions, on an isokinetic dynamometer, until the first five repetitions of a set were performed at least 50% below QH peak torque. Three-dimensional hip and knee kinematics and normalized (body mass $\times$ height) kinetic variables were quantified for pre- and postfatigue landings and subsequently analyzed by way of repeated-measures mixed-model ANOVAs. QH fatigue produced significant increases in initial contact (IC) hip internal rotation and knee extension and external rotation angles ($p < .05$), with the increases in knee extension and external rotation being maintained at the time of peak vertical ground reaction force (vGRF) ($p < .05$). Larger knee extension and smaller knee flexion and external rotation moments were also evident at peak vGRF following fatigue ($p < .05$). Females landed with greater hip flexion and less abduction than males at both IC and peak vGRF as well as greater knee flexion at peak vGRF ($p < .05$). The peak vGRF was larger for females than males ($p < .05$). No sex $\times$ fatigue effects were found ($p > .05$). Fatigue of the QH muscles altered hip and knee neuromechanics, which may increase the risk of ACL injury. Prevention programs should incorporate methods aimed at countering QH fatigue.

**Keywords:** kinetics, kinematics, muscle

Approximately 200,000 anterior cruciate ligament (ACL) injuries occur in the United States each year, with females being 2–8 times more susceptible to a rupture than their male counterparts (Griffin et al., 2006). Exposure to this injury typically precipitates surgical reconstruction and a difficult and lengthy rehabilitation program, with athletes rarely returning to their preinjury competitive levels (Myklebust et al., 2003). The ACL injured patient is also predisposed to significant long-term risk through the related progression of osteoarthritis (Kannus & Jarvinen, 1989; McDaniel & Dameron, 1983). Therefore, elucidating the underlying mechanisms of noncontact ACL injury is crucial so that targeted prevention modalities may be developed and implemented.

An underlying factor potentially linked to the risk of noncontact ACL injury that has recently gained substantial research attention is that of neuromuscular fatigue (Chappell et al., 2005; McLean et al., 2007; Orishimo & Kremenic, 2006). Neuromuscular fatigue manifests through combined central and peripheral processes and is largely inevitable in sports, where high intensity efforts are often required for extended periods. Such fatigue-related manifestations typically present as altered proprioception (Miura et al., 2004), delayed muscle responses (Wojtys et al., 1996), and altered biomechanics, offering the potential for injurious changes within the resultant neuromechanical profile. Epidemiological data also suggest that neuromuscular fatigue may contribute directly to ACL injury risk, with more injuries occurring at the beginning or end of the season or toward the end of the half or match when athletes (e.g., football players, soccer players) may succumb to the physiological demands of intense training and/or competition (Bradley et al., 2002; Hawkins & Fuller, 1999).

Considering stability of the knee during dynamic landings is governed largely by the combined actions of the quadriceps and hamstrings (QH) muscles, understanding the effect of fatigue on this activation strategy,
therefore, appears vital to a deeper understanding of injury risk (Besier et al., 2003; Lloyd & Buchanan, 1996; Schippelein & Andriacchi, 1991). The QH antagonistic response is believed to reduce the risk of ACL injury by limiting anterior tibial translation. Quadriceps and hamstrings co-contraction has also been shown to provide dynamic stability to the frontal plane at the knee (Lloyd & Buchanan, 2001), potentially protecting against excessive knee abduction or adduction loading, which have been shown to increase ACL injury risk (Hewett et al., 2005). In addition, the QH, in their roles as knee and hip extensors, respectively, absorb energy upon impact, creating a muscle dominant landing strategy and protecting the ACL. The QH are also important for successful hip control strategies, in particular, those governing sagittal and transverse plane hip motions (Delp et al., 1994, 2001). The fatigue levels achieved during moderate-to-intense physical activity within these muscle groups, therefore, may compromise landing strategy, increasing ACL injury risk. Nyland et al. (1997), for example, found that knee extension angles and moments increased substantially on ground contact of a crossover cut task following both isolated quadriceps and isolated hamstrings fatigue. Further, Augustsson et al. (2006) found similar quadriceps fatigue-induced increases in hip extension angles and moments at peak vertical ground reaction force (vGRF). Fatigue-induced modifications in hip and knee joint control strategies associated with sports landing postures also appear more pronounced in females than in males (McLean et al., 2007), thus suggesting that neuromuscular fatigue may be implicated directly in the sex-based disparity in ACL injury rates.

Current programs targeted at preventing ACL injuries focus primarily on modifying the biomechanical causes of injury. While such steps are warranted, that ACL injury rates and the associated sex-disparity have not diminished in spite of these programs suggests that solely targeting the biomechanical causes of ACL injury is not sufficient (Agel et al., 2005). Further, as fatigue may exacerbate hazardous biomechanics, ACL injury prevention methods may ultimately benefit from countering fatigue-related modifications in the underlying muscle activation strategy. Before such steps can be taken, however, a precise understanding of how fatigue affects the resultant landing strategy is needed. Previous neuromuscular fatigue studies have provided important insight here; however, these studies have not enhanced the understanding of the role that specific muscles and muscle groups have in generating these postfatigue neuromechanical adaptations. Given this, and considering the previously mentioned role of the QH muscle group on dynamic stabilization of the lower extremity (Besier et al., 2003; Lloyd & Buchanan, 1996; Schippelein & Andriacchi, 1991), the purpose of the current study was to examine the effects of combined QH muscle fatigue on neuromechanical adaptations of the lower extremity during a specific dynamic landing posture. In addition, we set out to determine whether neuromechanical adaptations, which result from fatigue, are more pronounced in females when compared with males. We hypothesized that QH fatigue would lead to a stiff landing strategy, whereby subjects would land with greater hip and knee extension and with larger sagittal plane loads. Further, we theorized that subjects would land in a position of dynamic valgus, with increased hip adduction and internal rotation as well as knee abduction and external rotation, with a corresponding increase in joint loading. We also hypothesized that these neuromechanical adaptations resulting from fatigue would be more pronounced in females than in males.

**Methods**

**Subjects**

Forty-two healthy male and female recreational volunteers, aged 18–22 were recruited to participate in this study, with data from 13 males (age = 20.31 ± 0.85 years; height = 1.8 ± 0.1 m; mass = 76.0 ± 8.9 kg) and 12 females (age = 20.33 ± 1.33 years; height = 1.7 ± 0.1 m; mass = 58.3 ± 7.7 kg) being analyzed. An a priori power analysis revealed that 11 subjects of each sex would be required to achieve 80% statistical power with an alpha level of 0.05. Subject exclusion criteria included (1) any previous history of knee injury or surgery; (2) current knee pain; (3) current hip or ankle injury; (4) and/or history of lower extremity injury in the past six months. The medical school institutional review board at the University of Michigan approved the study and informed consent was obtained from all subjects before participation.

**Instrumentation**

The movements of the lower extremity segments were tracked with a three-dimensional motion capture system (Vicon MX, Oxford Metrics Ltd., Oxford, UK). Eight cameras captured lower extremity motion at a frequency of 200 Hz. Subjects landed on a force platform (OR 6–7; Advanced Medical Technology, Inc, Watertown, MA), which was located in the middle of the capture volume for the cameras and used to collect ground reaction force data. Ground reaction force data were sampled at 1200 Hz and were synchronized with the Vicon system for simultaneous collection.

An isokinetic dynamometer (Cybex 340, Cybex International, Inc., Medway, MA, USA) sampling at 50 Hz was used to monitor peak torque and induce muscle fatigue. All contractions were performed on the dynamometer at a speed of 180°/s through a knee range of motion of 0–100° (Bellew & Fenter, 2006).

**Testing Procedures**

A pre/posttest design was used in this investigation. For each subject, three-dimensional hip and knee kinetic and kinematic data were recorded during three successfully executed forward hops, both before and after exposure to a QH fatigue protocol. Upon reporting for testing, all subjects were outfitted with 17 precisely attached
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retro-reflective markers (Figure 1) and a stationary video recording was obtained (McLean et al. 2004) before the initiation of the prefatigue forward hops. Five retro-reflective markers (bilateral posterior superior iliac spines as well as dominant limb gluteus maximus, distal shank, and heel markers) were removed during the fatiguing exercise and replaced before the execution of the postfatigue forward hops. To account for this, a second stationary video recording was captured following the completion of the postfatigue forward hops.

**Forward Hopping Procedures.** Each hop consisted of a single-leg takeoff and landing (Lephart et al., 2002) and was used to simulate rapid deceleration, a common component of the ACL injury mechanism, that would occur during sport (Boden et al., 2000). A successful hopping trial required subjects to jump forward off and land on their dominant leg (the leg with which they would kick a ball) on the force platform. The distance hopped was determined by each individual subject’s leg length, defined as the tip of the greater trochanter to the tip of the lateral malleolus (Webster et al., 2004). Hop distance was normalized to $1 \times$ leg length in an attempt to equalize task difficulty across subjects. Each subject was given three practice hops at the beginning of the testing session for familiarization with the task.

**Fatigue Procedures.** Before initiating the fatigue protocol, knee flexion/extension peak torque of each subject was recorded so that it could later be used to quantify percentage strength degradation and, ultimately, denote fatigue. Specifically, subjects performed a series of five alternating QH maximum voluntary concentric contractions (MVCC) (Bellew & Fenter, 2006). The highest peak torque value for both flexion and extension was recorded (baseline peak torque). Subjects were then provided with 2 min of rest before the fatiguing exercises were initiated (Bellew & Fenter, 2006). To fatigue the QH muscle groups, subjects performed alternating QH MVCC until the torque measured in both muscle groups dropped below 50% of the subject’s baseline peak torque value for three consecutive repetitions (Ochsendorf et al., 2000). Once this point was achieved, subjects were given 20 s of rest and then asked to resume the alternating QH MVCC until the torque again dropped below 50% of the subject’s baseline peak torque value. This process of MVCC and rest was repeated until fatigue was achieved, corresponding to when the first five repetitions of a QH MVCC set were performed below 50% of the subject’s baseline peak torque in both muscle groups. There was no limit on the number of MVCC a subject could perform. Following the fatigue protocol, subjects immediately completed three additional forward hops, adopting exactly the same

![Figure 1](image.png) — Marker locations used for the kinematic model.
procedure as that used for the prefatigue trials. Immediately following the final postfatigue hopping trial, subjects had knee flexion/extension peak torque reassessed in the same manner as before fatigue.

**Kinematic and Kinetic Data Collection and Analysis.**

Joint rotations were quantified during each trial based on the 3-D coordinates of the retro-reflective markers. Lower limb segment joint centers were subsequently defined similar to our previous work (McLean et al., 2007). The 3-D marker trajectories recorded during each dynamic landing trial were processed by Visual 3-D software (Version 3.9, C-motion, Inc., Rockville, MD, USA) to solve for the generalized coordinates of each frame. Joint rotations were calculated using a Cardan rotation sequence (Grood & Suntay, 1983) and were expressed relative to each subject’s neutral position (McLean et al., 2007). Synchronous 3-D ground reaction force data and the above kinematic data were filtered with a zero-lag Butterworth filter at a 12-Hz cutoff frequency (McLean et al., 2007) and submitted to a standard inverse dynamics analysis (Bresler & Frankel, 1950). Segment inertial properties were defined in accordance with the work of Dempster (1955). Intersegmental moments at the hip and knee were expressed as flexion-extension, adduction-abduction and internal-external rotation moments with respect to the cardanic axes of their respective joint coordinate systems (Grood and Suntay, 1983; Wu et al., 2002). Kinetic outputs were normalized to body mass and height (Willson & Davis, 2008) and subsequently converted to represent external moments acting on or about the joint. That is, an external knee abduction moment acts to produce a knee abduction rotation.

Joint and hip 3-D joint rotations were recorded at IC, being defined as the first instance vGRF data exceeded 10 N (McLean et al., 2007), and at peak vGRF. Joint moment data were recorded at peak vGRF only. Peak vGRF was chosen as a temporal marker as this represents a time during landing when injury may occur (Ireland, 2002). All angles and moments were averaged for the three pre- and postfatigue hopping trials obtained for each subject and subsequently submitted to statistical analyses.

**Statistical Analysis**

Sagittal, frontal, and transverse plane hip and knee kinematics and kinetics recorded at IC and peak vGRF were analyzed by way of 2 × 2 ANOVAs. Each model had one between-subjects factor, sex (female and male), and one within-subjects factor, fatigue state (prefatigue and postfatigue). Peak vGRF, time to peak vGRF, quadriceps torque, and hamstring torque were similarly subjected to 2 × 2 ANOVAs. Bonferroni multiple comparison procedures were used to make all post hoc comparisons. Effect sizes were calculated using Cohen’s d. The experimentwise type I error rate for the hypotheses associated with each response variable was ≤ .05. Statistical analyses were performed using the Statistical Package for the Social Sciences (SPSS) 10.1 software (SPSS, Inc., Chicago, IL, USA). Effect sizes were calculated in Microsoft Excel 2007 (Microsoft Corporation, Redmond, WA, USA).

**Results**

**Joint Angles at Initial Contact**

There was no interaction between subject sex and fatigue state for any joint angle at IC (p > .05). QH fatigue altered hip transverse plane joint angles at IC (p = .02) but did not affect hip angles in the sagittal (p = .49, d = 0.14) or frontal planes (p = .20, d = 0.28) (Table 1). When fatigued, subjects displayed more internal rotation at ground contact (Figure 2). Subject sex influenced hip joint angle in the sagittal (p = .03) and frontal planes (p = .006), but not in the transverse plane (p = .38), with females landing in a position of greater hip flexion and males in a position of greater hip abduction at IC (Figure 3). Quadriceps and hamstrings fatigue also altered knee transverse (p = .05) and sagittal (p < .001) plane angles at IC but did not affect knee angles in the frontal plane (p = .91, d = 0.02). Subjects landed in a position of greater knee extension and external rotation at ground contact following fatigue (Figure 4). Subject sex did not influence

<table>
<thead>
<tr>
<th>Joint Angle (°)</th>
<th>Prefatigue</th>
<th>Postfatigue</th>
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<tr>
<td></td>
<td>Female</td>
<td>Male</td>
</tr>
<tr>
<td>IC Hip Flex/Ext†</td>
<td>31.5 (8.3)</td>
<td>26.1 (4.0)</td>
</tr>
<tr>
<td>IC Hip Add/Abd†</td>
<td>−3.8 (4.1)</td>
<td>−8.5 (3.9)</td>
</tr>
<tr>
<td>IC Hip Int/Ext rotation*</td>
<td>−1.5 (7.6)</td>
<td>1.9 (7.1)</td>
</tr>
<tr>
<td>IC Knee Ext/Flex*</td>
<td>−14.9 (6.1)</td>
<td>−11.9 (4.4)</td>
</tr>
<tr>
<td>IC Knee Add/Abd</td>
<td>−2.0 (2.6)</td>
<td>−1.2 (1.7)</td>
</tr>
<tr>
<td>IC Knee Int/Ext rotation*</td>
<td>1.2 (3.4)</td>
<td>−0.5 (2.2)</td>
</tr>
</tbody>
</table>

*Indicates statistically significant difference between fatigue conditions (p < .05).
†Indicates statistically significant difference between the sexes (p < .05).

**Table 1 Hip and knee angles at initial contact for pre- and postfatigue states**

Note: All values are presented as mean (standard deviation). For all joint angles, the first angle listed is positive (e.g., at IC the hip is in a flexed position as indicated by the positive values).
Figure 2 — Effect of fatigue state on mean (± SD) transverse plane hip joint angle during the one-legged hop. Subjects displayed greater hip internal rotation when fatigued at initial ground contact (Time = 0).

Figure 3 — Effect of sex on mean (± SD) sagittal and frontal plane hip joint angles during the one-legged hop. Females landed with more hip flexion and males with greater hip abduction at IC and peak vGRF. The vertical line indicates time of peak vGRF for both males and females.

Figure 4 — Effect of fatigue state on mean (± SD) sagittal and transverse plane knee joint angles during the one-legged hop. Prefatigue subjects landed with greater knee flexion and internal rotation at IC and peak vGRF. The dashed vertical line represents the time of peak vGRF prefatigue, while the solid vertical line indicates time of peak vGRF postfatigue.

Joint Angles at Peak Vertical Ground Reaction Force

There was no interaction between subject sex and fatigue state for any hip or knee joint angle at peak vGRF (p > .05). Fatigue state did not affect hip joint angle in any plane at peak vGRF (sagittal p = .33, d = 0.20; frontal p = .14, d = 0.31; transverse p = .17, d = 0.26; Table 2). At the knee, subjects demonstrated greater extension (p = .002) and external rotation angles (p = .02) when fatigued (Figure 4). There was no effect of fatigue on knee adduction/abduction angle (p = .75, d = 0.05). Subject sex influenced hip sagittal (p = .04) and frontal (p = .006) plane angles, with females demonstrating greater hip flexion, while males demonstrated greater hip abduction at peak vGRF (Figure 3). Further, subject sex influenced knee sagittal plane angle (p = .04), with females demonstrating a greater knee flexion angle than males at peak vGRF (Figure 5). There was no sex difference in hip joint angle in the transverse plane (p = .41, d = 0.24) or in knee joint angle in the frontal (p = .47, d = 0.72) or transverse planes (p = .45, d = 0.22).
Table 2  Hip and knee angles at peak vertical ground reaction force for pre- and postfatigue states

<table>
<thead>
<tr>
<th>Joint Angle (°)</th>
<th>Prefatigue</th>
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<th>Postfatigue</th>
<th></th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Female</td>
<td>Male</td>
<td>Female</td>
<td>Male</td>
</tr>
<tr>
<td>Peak vGRF Hip Flex/Ext†</td>
<td>33.4 (9.8)</td>
<td>27.6 (4.3)</td>
<td>33.0 (10.7)</td>
<td>25.0 (8.3)</td>
</tr>
<tr>
<td>Peak vGRF Hip Add/Abd†</td>
<td>–1.1 (5.3)</td>
<td>–6.4 (4.2)</td>
<td>–3.3 (2.4)</td>
<td>–7.1 (5.4)</td>
</tr>
<tr>
<td>Peak vGRF Hip Int/Ext rotation</td>
<td>0.6 (8.7)</td>
<td>3.4 (6.6)</td>
<td>2.9 (7.4)</td>
<td>4.4 (8.6)</td>
</tr>
<tr>
<td>Peak vGRF Knee Ext/Flex*†</td>
<td>–28.1 (11.1)</td>
<td>–22.9 (5.3)</td>
<td>–23.0 (10.3)</td>
<td>–15.6 (5.5)</td>
</tr>
<tr>
<td>Peak vGRF Knee Add/Abd</td>
<td>–3.1 (6.0)</td>
<td>–1.2 (2.6)</td>
<td>–2.1 (4.9)</td>
<td>–1.7 (3.7)</td>
</tr>
<tr>
<td>Peak vGRF Knee Int/Ext rotation*</td>
<td>1.0 (4.2)</td>
<td>–0.4 (2.3)</td>
<td>–1.8 (3.7)</td>
<td>–2.8 (7.1)</td>
</tr>
</tbody>
</table>

Note: All values are presented as mean (standard deviation). For all joint angles, the first angle listed is positive (e.g., at peak vGRF the hip is in a flexed position as indicated by the positive values).

*Indicates statistically significant difference between fatigue conditions (p < .05).
†Indicates statistically significant difference between the sexes (p < .05).

Discussion

In this study, we set out to examine the effects of QH muscle fatigue on kinetics and kinematics of the hip and knee during the performance of a single-leg forward hop. Previous research (McLean et al., 2007; Padua et al., 2006) has shown that neuromuscular fatigue of the lower extremity may increase the risk of ACL injury by perpetuating abnormal, and potentially hazardous,
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Table 3  Hip and knee moments at peak vertical ground reaction force for pre- and postfatigue states

<table>
<thead>
<tr>
<th>Joint Moment (N m/kg m)</th>
<th>Prefatigue</th>
<th></th>
<th>Postfatigue</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Female</td>
<td>Male</td>
<td>Female</td>
<td>Male</td>
</tr>
<tr>
<td>Peak vGRF Hip Flex/Ext</td>
<td>0.74 (0.22)</td>
<td>0.75 (0.79)</td>
<td>0.33 (0.74)</td>
<td>0.77 (1.15)</td>
</tr>
<tr>
<td>Peak vGRF Hip Add/Abd</td>
<td>0.91 (0.25)</td>
<td>0.63 (0.32)</td>
<td>0.75 (0.27)</td>
<td>0.65 (0.84)</td>
</tr>
<tr>
<td>Peak vGRF Hip Int/Ext rotation</td>
<td>0.32 (0.15)</td>
<td>0.28 (0.19)</td>
<td>0.34 (0.17)</td>
<td>0.30 (0.35)</td>
</tr>
<tr>
<td>Peak vGRF Knee Ext/Flex*</td>
<td>–0.74 (0.68)</td>
<td>–0.86 (0.60)</td>
<td>–0.43 (0.46)</td>
<td>–0.16 (0.24)</td>
</tr>
<tr>
<td>Peak vGRF Knee Add/Abd</td>
<td>0.09 (0.31)</td>
<td>0.22 (0.20)</td>
<td>0.14 (0.19)</td>
<td>0.25 (0.41)</td>
</tr>
<tr>
<td>Peak vGRF Knee Int/Ext rotation*</td>
<td>–0.16 (0.14)</td>
<td>–0.11 (0.09)</td>
<td>–0.11 (0.12)</td>
<td>–0.04 (0.09)</td>
</tr>
</tbody>
</table>

*Indicates statistically significant difference between fatigue conditions (p < .05).

Note: All values are presented as mean (standard deviation). For all joint moments the first moment listed is positive (e.g., there is a flexion moment at the hip at peak vGRF as indicated by the positive values).

Figure 6 — Effect of fatigue state on mean (± SD) normalized sagittal and transverse plane knee joint moments during the one-legged hop. Prefatigue subjects landed in with a smaller knee flexion and external rotation moment. The dashed vertical line represents the time of peak vGRF prefatigue, while the solid vertical line indicates time of peak vGRF postfatigue.

The fatigue model we employed used simultaneous fatigue of the QH muscles in isolation from the rest of the lower extremity. Further, fatigue was defined as a percent decrease in the subject’s maximum strength (Ochsendorf et al., 2000), which allowed all subjects to be fatigued to the same level. Though previous researchers have chosen to fatigue each of the QH muscle groups separately (Nyland et al., 1997), we felt that simultaneous landing strategies. The combined QH muscle activation strategy is important for lower extremity control during dynamic activities. Hence, elucidation of the neuromechanical adaptations that ensue following QH fatigue may afford targeted prevention strategies that can more effectively counter the neuromuscular demands associated with postures elicited within a realistic sports environment.
QH fatigue was more realistic to athletic activity. We acknowledge that general lower extremity fatigue models used previously (Augustsson et al., 2006; Borotikar et al., 2008; Chappell et al., 2005; Madigan & Pidcoe, 2003; McLean et al., 2007) may be more realistic to sport than our fatigue model, as they may provide an understanding of how the muscles of the lower extremity work together to stabilize the limb. However, fatiguing muscle groups in isolation may afford the contributions of specific muscle groups to dynamic limb stability to be delineated. Determining which muscle groups are most responsible for imposing injurious postures and loads on the lower extremity in a fatigued state may help reduce the incidence of fatigue-related injury. Specifically, this knowledge can lend to the development of injury prevention programs focused on reducing the fatigability of those muscle groups, thereby limiting the deleterious effects of fatigue.

Quadriceps and hamstrings muscle fatigue had minimal influence on hip control, with subjects only increasing IC hip internal rotation angle when fatigued. This finding is in partial agreement with our hypothesis that subjects would increase hip internal rotation angle and consistent with that of Borotikar et al. (2008) who similarly noted an increase in hip internal rotation following repetitive, fatigue-inducing squatting exercises. While QH musculature was not fatigued exclusively in their study, it seems intuitive that their chosen squatting tasks would have still induced substantial QH fatigue, a point alluded to by the authors (Borotikar et al., 2008). The QH muscle group is considered to have, at most, a very small moment arm at the hip in the transverse plane (Pressel & Lengsfeld, 1998). We thus suspect that the increase in hip internal rotation following QH fatigue may represent a compensatory neuromuscular strategy, whereby the muscles acting directly at the hip became more active to account for the decreased capacity of the QH to decelerate the body’s center of mass. We did not gather muscle activation data and cannot quantify the specific hip muscle activation changes that may have occurred; however, we would speculate that increased activity of the tensor fascia latae, iliopsoas, and/or gluteals may have been involved. In our future work, we intend to incorporate an analysis of the associated muscle activation patterns to test these hypotheses explicitly.

Limited modifications were identified in sagittal plane hip motions postfatigue directly contradicting our original premise. In accordance with our hypotheses, however, subjects contacted the ground following fatigue with a more extended knee joint and remained in more extension with a smaller knee external flexion moment at peak vGRF. Previous studies (Augustsson et al., 2006; McLean et al., 2007) have demonstrated a fatigue-induced “stiffening” of the stance limb upon landing, whereby subjects displayed more extended hip and knee postures. This increased stiffness has been proposed to prevent lower limb collapse and potentially aids in preventing joint injury (McNitt-Gray et al., 2001). Similar to our current findings, however, Orishimo and Kremenic (2006) failed to observe fatigue-induced alterations in the sagittal plane hip strategy during performance of a single-leg forward hop. As the rectus femoris and the hamstrings musculature have sagittal plane moment arms acting at the hip, we theorized that fatigue of these muscles may alter hip flexion/extension mechanics. The lack of change in hip mechanics may stem from the ability of the nonfatigued hip flexors (iliacus, psoas major, and tensor fascia latae) and extensors (gluteals) to maintain prefatigue postures and loads. Future studies examining muscle activation strategies pre- and postfatigue would provide further insight.

While increased knee joint stiffness may serve as a protective mechanism against collapse of the knee, increased knee joint loading, combined with an extended knee posture, has been touted extensively to cause injury to the ACL (Boden et al., 2000; DeMorat et al., 2004). Landing in an upright posture in the presence of QH fatigue may require increased input from the passive joint structures (e.g., the ACL) in preserving knee stability (Besier et al., 2003). Maintaining a flexed hip posture on landing may place the monoarticular hip extensors at a more optimal length to become the primary absorber during the landing phase, hence minimizing the need for QH activity. The flexed hip posture demonstrated by our subjects, therefore, may have been a compensatory mechanism to decrease the load that would otherwise be placed on the ACL and protect it from injury. It is possible that the postfatigue load redistribution evident at the hip may have been aided further by a similar redistribution of load at the ankle joint. Considering, however, that we did not currently examine ankle kinetics, this contention remains speculative.

The fact that knee external rotation angle at peak vGRF increased following fatigue despite a concomitantly smaller external rotation moment at the same time point appears somewhat counterintuitive. Given that the hamstrings control tibial rotation (Nyland et al., 1999), fatigue-induced increases in external rotation angle may have resulted directly from hamstrings fatigue. The increased external rotation angle may also be connected to the increase in hip IR angle, wherein subjects generated greater knee external rotation to maintain a more neutral lower limb alignment during landing. Worth noting, however, is that knee joint laxity is also increased in the presence of reasonable fatigue (Wojtys et al., 1996), which in turn may compromise spinal reflex mechanisms, and hence muscle contributions to joint stability (Lattanzio & Petrella, 1998). If the latter is indeed true, then reduced external rotation moments may have represented an adaptive strategy to offset further increases in external rotation angles in an increasingly loose knee joint. Given that axial tibial rotation angles and loads are known to induce substantial ACL loading (Kanamori et al., 2000; Markolf et al., 1995), and have been implicated directly within the injury mechanism (Griffin et al., 2006; Ireland, 1999), the relationship between knee joint rotational laxity and associated muscle activation strategies in the presence of fatigue warrants further study.
The reduced external rotation moment at the knee also could have resulted from a decrease in the knee flexion angle following fatigue. A smaller knee flexion angle may have altered the moment arm of the ground reaction force about the knee, yielding a smaller net moment and, therefore, reducing the external rotation moment. Further, altered foot position at IC may have contributed to the reduced external rotation moment at the knee, since we know that ankle pronation/supination angle influences tibial rotation moment (Bellchamber & van den Bogert, 2000). Again, incorporating ankle data in future studies would provide further insight here.

The lack of change in hip joint abduction angles at IC and peak vGRF and loads peak vGRF following fatigue directly contradicts our initial hypotheses, and further, the majority of previous research examining this relationship (Chappell et al., 2005; Kernozek et al., 2008; McLean et al., 2007). Worth noting, however, is that unlike these previous investigations, we did not fatigue hip abductor or adductor musculature. While co-contraction of the QH is the primary stabilizer in the knee frontal plane (Lloyd & Buchanan, 2001), the hip abductors and adductors have been shown to play an important role in knee frontal plane stabilization (Lloyd & Buchanan, 2001). With these muscle groups not currently fatigued, therefore, they may have been able to generate enough force to maintain frontal plane postures and loads in spite of QH fatigue. Integrative assessment of muscle activation strategies would provide substantial insight into this and we intend to include such analyses in our ongoing research.

In accordance with our hypothesis, subject sex influenced landing biomechanics, with females demonstrating greater hip flexion and smaller abduction angles at both IC and peak vGRF as well as greater knee flexion angles at peak vGRF. The peak vGRF was also larger for females than males. Previous research has demonstrated sex-based differences in lower extremity kinematics regardless of fatigue state; however, the results in the sagittal plane are conflicting. At the hip, research shows that females land with either increased (Kernozek et al., 2008) or consistent (Decker et al., 2003; McLean et al., 2007; Benjaminse et al., 2008) flexion angles compared with males. Similar results have been demonstrated at the knee, with studies demonstrating females landing with increased (Fagenbaum & Darling, 2003), decreased (Chappell et al., 2005; Decker et al., 2003), and comparable (Benjaminse et al., 2008; McLean et al., 2007) flexion angles relative to males. The lack of consistency in sagittal plane lower limb landing kinematics are likely governed by the task performed and may be population or subject specific (McLean et al., 2005; McLean et al., 2004).

In the hip frontal plane, our female subjects landed in a more adducted position than males. This is in disagreement with previous research demonstrating no sex difference in frontal plane kinematics with fatigue (Benjaminse et al., 2008; Kernozek et al., 2008), but is in accordance with a study by McLean et al. (2004), demonstrating a more adducted hip position in females during a side-step cutting task. The fact that females displayed increases in hip adduction is potentially hazardous, given that hip adduction has been implicated as a component of the dynamic knee valgus position that is often associated with increased risk of noncontact ACL injury (Hewett et al., 2005). This difference in frontal plane posture may be explained by a sex difference in hip abductor muscle strength. Studies have demonstrated that males have stronger hip abductors than females (Leetun et al., 2004), which was likely the case with our subjects and could have resulted in the more adducted hip posture in the female subjects.

Subject sex also influenced peak vGRF during landing, with females demonstrating a larger peak vGRF than males both pre- and postfatigue, a finding consistent with that of Kernozek et al. (2008). Considering that the magnitude of the peak vGRF governs the magnitude of the moments experienced throughout the lower extremity during landing, a larger vGRF could be injurious as it could lead to larger moments placed on the joints. However, the fact that sex differences were not identified for the knee and hip moments suggests that the females were able to compensate for the larger peak vGRF.

Directly comparing our findings to the findings of others presents a challenge, as most previous work examining the effects of muscle fatigue on lower extremity biomechanics have methodological differences that likely contributed to the discrepancies observed in outcomes. We would argue that these disparities make comparisons across studies inaccurate and may lead researchers to draw incorrect conclusions. Therefore, we would like to caution the reader to keep the procedural distinctions in mind when interpreting this and other work. The first such distinction is that a variety of models have been employed to induce and define fatigue (Augustsson et al., 2006; Borotikar et al., 2008; Chappell et al., 2005; Madigan & Pidcoke, 2003; McLean et al., 2007). As noted above, researchers have often selected a general lower extremity fatigue model, which has been shown to alter limb proprioception differently than a localized fatigue approach (Miura et al., 2004). Considering the neuromuscular complexities associated with dynamic sports landings, therefore, general fatigue models may result in a wide variety of adaptive neuromuscular strategies to maintain joint stability and to prevent excessive forces from being transferred to passive joint structures (Simpson et al., 1988). It is also possible that with general fatigue models, exhaustion rather than neuromuscular fatigue is induced (Borotikar et al., 2008; Kernozek et al., 2008), especially in general fatigue models where fatigue is subjectively determined by the subject (Coventry et al., 2006; Madigan & Pidcoke, 2003). If this is indeed the case, then the additional effect on central processing and control mechanisms may potentiate an even greater disparity in the resultant neuromechanical strategy between these and studies using isolated fatigue models where fatigue is objectively defined (Borotikar et al., 2008). Second, the neuromechanical response to fatigue may be influenced by the tasks subjects are asked to perform. A variety of
activities—single- and double-leg hops and landings, crossover cutting, running, and so on—have been used to assess the body’s response to fatigue. It is probable that the body adopts varying strategies to adjust for the differing forces and loads that it will experience (Yu et al., 2006), in different situations, and thus we would expect varying limb positions and joint loads for subjects who performed different tasks. Third, the dependent variables chosen and the time points at which these variables are analyzed differ between studies. We chose to analyze hip and knee neuromechanics at IC and peak vGRF as discussed previously. It remains unclear exactly when ACL injuries occur (Griffin et al., 2006), though peak vGRF does correspond to a time in the stance phase where joint moments are similarly large. Given that ACL injury is likely governed by the combined magnitudes of the 3-D knee loading (McLean & Samorezov, 2009), we felt data extraction at this specific point was reasonable. Regardless, future research would benefit from additional exploration into how neuromechanical data extracted at different points or phases across stance may be linked to ACL injury risk. Fourth, the subject population researchers have used may affect study outcomes. Subject maturation, skill level, and training have been shown to play a role in neuromuscular control of the lower extremity and could help to explain differences in study outcomes (Hewett et al., 2004).

We have demonstrated that fatigue of the QH during dynamic single-leg landings results in hip and knee postures suggested by previous studies to occur at the time of ACL injury (Griffin et al., 2006; Ireland, 2002). At IC, for example, subjects displayed increased hip internal rotation, knee extension, and knee external rotation following fatigue, described by Ireland (2002) as the “position of no return.” The increased knee extension and external rotation postures postfatigue were maintained at peak vGRF, a time during the stance phase that may also correspond to the timing of the injury event (Ireland, 2002). Future studies further examining the effect of isolated neuromuscular fatigue on lower extremity neuromechanics would benefit from integrating EMG to provide greater insight into the effect of fatigue on relative muscle timings. Further, future work would also benefit from examining the role of neuromuscular fatigue of the lower extremity on ankle kinetics and kinematics.

While we do not discount the role of the ankle in stabilization of the lower extremity during dynamic activity, we chose to investigate the effects of QH neuromuscular fatigue on only the hip and knee joints as these are the primary joints affected by QH neuromuscular fatigue because the QH musculature only cross the hip and knee joints. Further, we acknowledge that fatigue effects are short-lived and that recovery may have occurred across the three postfatigue trials. However, subjects were encouraged to move as quickly as possible so that the estimated elapsed time between the final repetition of the fatiguing exercise and the completion of the final postfatigue forward hop was less than three minutes, a time reported in which recovery from submaximal fatigue occurs (Augustsson et al., 2006). In addition, we cannot rule out the possibility that muscles such as the gastrocnemius were also fatigued during the isokinetic exercise. However, based on subsequently collected data (Thomas et al., 2010) examining the effects of gastrocnemius fatigue on lower extremity neuromechanics, we believe the presence of any gastrocnemius fatigue in conjunction with QH fatigue would be negligible. Regardless, the outcome of the current study suggests that inclusion of QH fatigue-resistance training is indeed warranted within ongoing injury prevention modalities.

The results of this study suggest that fatigue of the QH musculature produces postfatigue increases in IC hip internal rotation angle and knee extension and external rotation angles at IC and peak vGRF. Quadriceps and hamstrings fatigue also resulted in decreases in knee flexion moment at IC and peak vGRF and knee external rotation moment at peak vGRF. These combined manifestations likely increase the risk of noncontact ACL injury during dynamic landing tasks. These results suggest that consideration of the effects of QH fatigue in current ACL injury prevention modalities is warranted.

**References**


