

Fatigue-Induced ACL Injury Risk Stems from a Degradation in Central Control

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ABSTRACT

MCLEAN, S. G. and J. E. SAMOREZOV. Fatigue-Induced ACL Injury Risk Stems from a Degradation in Central Control. *Med. Sci. Sports Exerc.*, Vol. 41, No. 8, pp. 1661–1672, 2009. **Purpose:** Fatigue contributes directly to anterior cruciate ligament (ACL) injury via promotion of high risk biomechanics. The potential for central fatigue to dominate this process, however, remains unclear. With centrally mediated movement behaviors being trainable, establishing this link seems critical for improved injury prevention. We thus determined whether fatigue-induced landing biomechanics were governed by a centrally fatiguing mechanism. **Methods:** Twenty female NCAA athletes had initial contact (IC) and peak stance (PS) three-dimensional hip and knee biomechanics quantified during anticipated and unanticipated single-leg landings, before and during unilateral fatigue accumulation. To induce fatigue, subjects performed repetitive ($n = 3$) single-leg squats and randomly ordered landings, until squats were no longer possible. Subject-based dependent factors were calculated across pre-fatigue trials and for those denoting 100%, 75%, 50%, and 25% fatigue and were submitted to three-way mixed-design analyses of covariance to test for decision, fatigue time, and limb effects. **Results:** Fatigue produced significant ($P < 0.01$) decreases in IC knee flexion angle and PS knee flexion moment and increases in PS hip internal rotation and knee abduction angles and moments, with differences maintained from 50% fatigue through to maximum. Fatigue-induced increases in PS hip internal rotation angles and PS knee abduction angles and loads were also significantly ($P < 0.01$) greater during unanticipated landings. Apart from PS hip moments, significant limb differences in fatigued landing biomechanics were not observed. **Conclusions:** Unilateral fatigue induces a fatigue crossover to the contralateral limb during single-leg landings. Central fatigue thus seems to be a critical component of fatigue-induced sports landing strategies. Hence, targeted training of central control processes may be necessary to counter successfully the debilitating impact of fatigue on ACL injury risk. **Key Words:** NEUROMUSCULAR FATIGUE, KNEE BIOMECHANICS, LANDING MECHANICS, ACL INJURY MECHANISMS

Identifying and ultimately countering the mechanism(s) of noncontact anterior cruciate ligament (ACL) injury remains a popular yet elusive research endeavor. A large amount of work in this area continues to focus on neuromuscular contributions to injury (21,24,42) because this factor is, in essence, modifiable and thus amenable to training (23). It has been argued, however, that with neuromuscular risk factors typically derived within a controlled laboratory setting, prevention efficacies within the random sports environment will remain compromised (6,25,33). The fact that ACL injury rates and their associated sex disparity have not diminished in spite of ongoing prevention developments tends to support this tenet (1).

The need to develop more robust and applicable ACL injury prevention methods has resulted in sports-relevant factors being increasingly integrated within the *in vivo*

testing environment (3,37). One such factor gaining recent research attention is that of neuromuscular fatigue (6,25). Neuromuscular fatigue as it pertains to human performance can be simply defined as a decrease in the maximal voluntary force produced by a muscle or muscle group (5,19). With regard to ACL injury, fatigue is proposed to increase risk by promoting extreme lower limb biomechanics, stemming from inadequate active joint stabilization via a suboptimal muscle activation strategy (6,25,34). A more extended (hip and knee) landing posture (7,34), increased out-of-plane hip rotations (6), and resultant increases in three-dimensional (3D) knee motions and loads (7,25,34) are common biomechanical outcomes of fatigued landings. Considering that these profiles culminate in concomitant increases in ACL loading (31,36), countering neuromuscular fatigue effects within the ACL injury prevention modality seems well warranted.

Before the impact of fatigue can be successfully addressed within the ACL injury prevention strategy, understanding its precise contributions to high-risk movements is critical. Traditionally, fatigue has been defined as either central or peripheral, based primarily on which components of the neuromuscular control system are directly impacted (18,19,41,44). Specifically, peripheral fatigue refers to exercise-induced processes leading to a reduction in the force-generating capacity of the muscle, occurring at or distal to the level of the neuromuscular junction (19,44). It is

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generally accepted that peripheral fatigue effects are caused mainly by metabolic factors or muscle damage if eccentric contractions are prominent (39). Conversely, central fatigue relates to a gradual exercise-induced reduction in the level of voluntary muscle activation (19,41), being attributable to impairment at sites proximal to the neuromuscular junction (20). Such effects are more pronounced during prolonged bouts of submaximal exercise activity (9,19), where changes at the spinal (e.g., muscle spindle and tendon organ reflex inhibition) and supraspinal (e.g., suboptimal output from the motor cortex) level promote inadequate drive to the working muscles (19,27,38,44). Multifaceted tasks such as dynamic sports maneuvers require explicit force production and control at both the peripheral and central levels (6). It thus seems plausible to assume that fatigue-induced ACL injury risk stems from both peripheral and central fatiguing mechanisms. From an injury prevention standpoint, however, countering peripheral fatigue is difficult, as trained increases in muscle fatigue resistance typically mirror increased athlete effort, rendering such fatigue effects inevitable (34). Targeted training of central (spinal and supraspinal) control mechanisms, therefore, which is known to enhance motor performance (2,13), may ultimately provide a more effective means of negating a high-risk fatigued movement response (6). Before contemplating such action, however, the potential for fatigue-induced adaptations in landing biomechanics to be influenced explicitly via a central fatigue mechanism must be identified.

During submaximal tasks such as sports landings, the force-generating capacity of muscle-fibers and the voluntary drive to the motor neurons are in constant flux (44), making peripheral and central fatigue mechanisms difficult to delineate. The unconstrained nature of these high-impact tasks also renders elucidation of explicit fatigue effects within the exercising limb virtually impossible via state-of-the-art assessment modalities (e.g., transcranial magnetic stimulation) (43). There is considerable evidence, however, suggesting that fatigue-induced impairment of voluntary muscle activation also occurs in the uninvolved contralateral limb muscles (32,40,46). This crossover in the fatigued response, being most pronounced in the lower limbs (41), is posited to arise through an explicit central fatigue mechanism (32,41,46). Metabolic and/or mechanical changes in the exercising muscle, for example, are suggested to elicit an anticipatory down-regulation in central control to maintain biarticular coordination and, in doing so, avoid catastrophe (30). The increased central activation necessary to maintain adequate force production in the fatigued limb (19), particularly within supraspinal control centers (19,44), may similarly impede voluntary drive to homologous muscles (40,46). Currently, a crossover in central fatigue has been observed for isolated muscle groups only (32,41,46) and, although not tested, is considered habitually irrelevant for movement execution or postural control (40). Considering the complex muscle activation strategies governing sports landings, however (4,28), it seems feasible that subtle changes in these strategies

arising via a crossover in central fatigue could produce noticeable and potentially hazardous adaptations in the contralateral limb's biomechanical response (4).

Evidence of a crossover in fatigue during the repetitive execution of sports landings would immediately suggest such tasks are influenced by a dominant central fatiguing mechanism. Further, culmination of this influence within an altered contralateral joint biomechanical profile would highlight the potentially critical role of central fatigue within the noncontact ACL injury mechanism. Therefore, with these facts in mind, we sought to determine whether exposure to a unilateral lower limb fatiguing protocol would induce similar biomechanical adaptations in the contralateral limb during single-leg landings. In addition, we examined the sensitivity of this crossover effect to movement tasks of increasing cognitive complexity. To achieve these aims, we tested the following hypotheses, which were based on our previous observations (6,34).

1. Unilateral lower limb fatigue will induce significant increases in initial contact (IC) hip and knee extension and hip internal rotation angles and in peak hip internal rotation and 3D knee positions and loads for the fatigued limb during the first 50% of single-leg stance.
2. Fatigue-induced changes in the above biomechanical parameters will be more pronounced during unanticipated compared with anticipated landings.
3. Lower limb joint biomechanical adaptations will be consistent between the fatigued and contralateral non-fatigued limb for unanticipated but not anticipated single-leg landings.

METHODS

Subjects. On the basis of data from previous studies investigating combined fatigue and anticipatory effects on lower limb landing mechanics (3,6), a power analysis revealed that to currently achieve 90% statistical power with an α level of 0.05, a minimum of 18 single-sex subjects would be required. A total of 20 female NCAA Division 1 (volleyball, soccer, and basketball) athletes (19.2 ± 1.7 yr) were subsequently recruited to participate in the study. The subject exclusion criteria for the study were as follows: 1) a history of previous knee injury and/or surgery, 2) pain in lower extremity before testing, 3) any recent injury to the lower extremity (previous 6 months), 4) undertaking any exercise within 24 h of testing, and 5) a current pregnancy. Before conducting the study, research approval was gained through the Institutional Review Board of the Cleveland Clinic Foundation, and written informed consent was obtained for all subjects. All subjects wore spandex bike shorts, sports shoes, and a sport brassier during testing.

Experimental design. Subjects had bilateral 3D lower limb joint kinematic and kinetic data recorded during a series of single- and double-leg dynamic landing tasks, both before and during exposure to a generalized fatigue protocol.

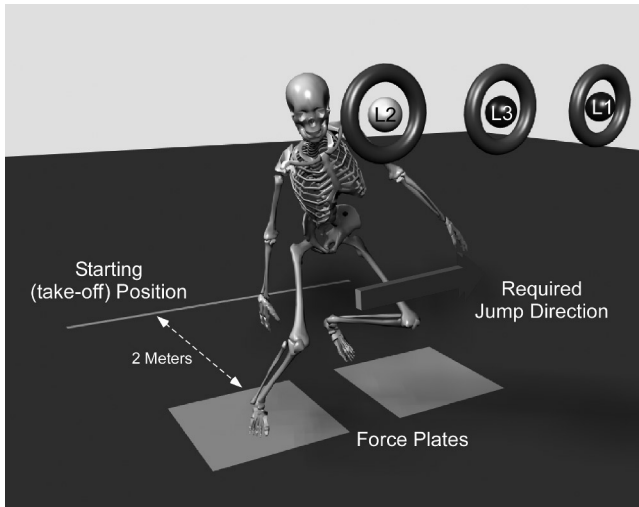


FIGURE 1—Subjects reacted to a random light stimulus and immediately cut in the appropriate direction on landing from a forward jump. Three lights were used, corresponding to a rapid (anticipated or unanticipated) land-and-cut to the right (L1) or left (L2), or vertical two-legged jump (L3).

Before fatigue, subjects were required to perform one of three randomly ordered jump landings, with the jump initiated from a stationary starting position located 2 m behind the force plates (Fig. 1). Landings were governed by an explicit light stimuli (L1, L2, or L3) activated before the landing phase (6,34). Specifically, activation of L1 required subjects to land on their left foot only and immediately cut laterally to the right. Conversely, activation of L2 necessitated a rapid cut off the right foot, laterally to the left. If L3 was activated, subjects landed on both feet and jumped vertically as high as possible. A successful jump in each instance required the respective foot (feet) to make complete contact with a separate AMTI force plate (OR6-5 nos. 4046 and 4048; Advanced Mechanical Technology, Inc., Watertown, MA), within the field of view of an eight-camera high-speed (240 fps) motion analysis system (Motion Analysis, Corp, Santa Rosa, CA).

Each single-leg landing trial was further discretized to incorporate either an anticipated or an unanticipated movement response, with this order again being randomized (6). For anticipated trials, the light stimulus was activated before (approximately 5 s) the subject initiating the takeoff phase of the movement. For unanticipated trials, the stimuli were automatically triggered via a light beam switch (42RLU-4000B; Allen Bradley, Anaheim, CA), which the subjects broke after takeoff, such that it was not received until approximately 400 ms before ground contact. The relationship between reaction time and movement complexity is well established within the literature, with the former necessarily increasing as the complexity of the task similarly increases (14,22). Considering reaction times typical of a choice reaction tasks therefore (17,22), in conjunction with current task complexity and our own experience in testing unanticipated movements (6), a 400-ms preland stimulus time was deemed adequate to challenge subjects while still

allowing successful task performance. Vertical (two-legged) jump trials were used to estimate and compare subject fatigue levels only (see below) and, hence, were always anticipated (6). Subjects were required to perform six successful landing trials for the five respective landing conditions (two legs \times two decisions; vertical), resulting in approximately 30 prefatigue landing trials being undertaken. A single experimenter with experience in these methods (S.G.M.) delineated between successful and unsuccessful trials, with the latter being repeated elsewhere along the randomly ordered trial sequence. Specifically, trials in which subjects moved in the wrong direction, or remained stationary upon landing, were deemed unsuccessful in anticipation and were subsequently removed from the analyses.

After the prefatigue trials, subjects again performed anticipated and unanticipated landings while simultaneously being exposed to a general fatigue protocol. Specifically, subjects performed a set of three single-leg squats immediately followed by a randomized landing trial, with this sequence repeated until maximal fatigue was attained. Maximal fatigue was defined as the point where subjects could no longer complete three sequential squats unassisted (6). Defining maximal fatigue in this manner afforded more reliable data comparisons across subjects (6,29). The fatigued limb was randomized because we have shown previously that limb dominance does not influence landing neuromechanics under a combined fatigue and anticipatory state (6). During squatting trials, the squatting limb was positioned on a base (scooter), which moved (medial/lateral) continuously over a range of 10 cm at 2 Hz (Fig. 2). This device was used to simulate game-induced fatigue progressions (6,34). Each

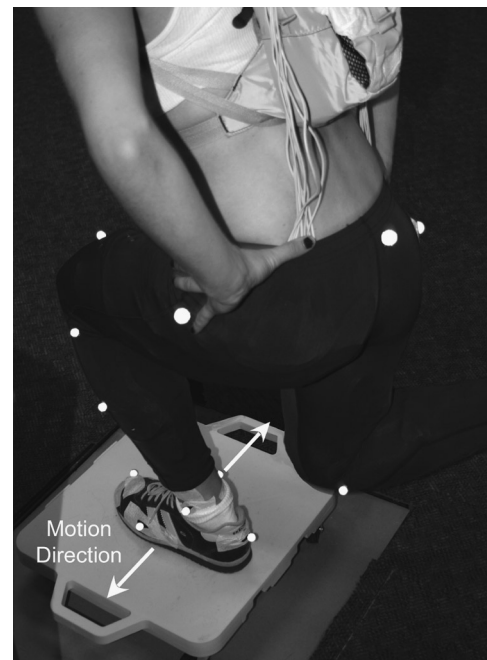


FIGURE 2—For squatting trials, the involved limb was positioned on a base that moved continuously over a range of 10 cm at 2 Hz to assist in simulating game-induced fatigue progressions.

squat was performed such that subjects necessarily reached a position with the femur parallel to the ground before returning to the upright position. Further, subjects were instructed to maintain the upper body as close to vertical throughout the squatting sequence.

Data processing and analyses. Lower limb (hip, knee, and ankle) 3D joint rotations were quantified for each landing trial on the basis of the 3D coordinates of 28 (12.5 mm in diameter) precisely attached reflective skin markers (6,34) (Fig. 3). Markers were secured to predetermined (shaved) anatomical landmarks via hypoallergenic, air-permeable cross elastic tape (Cover-Roll Stretch; BSN Medical GmbH, Hamburg, Germany). Ankle-length spandex tights were subsequently pulled-up over the markers, ensuring that their positions were maintained. Small incisions were made in the tights so that markers could be drawn through. This method was used with the intent of maximizing the disparity in contrast between the markers and background and to possibly minimize marker movement or loss, particularly when subjects began to sweat as they approached maximal fatigue (6).

After marker placement, a high-speed video recording was obtained with the subject standing in a stationary (neutral) position (6). A kinematic model was then defined on the basis of these data, consisting of nine skeletal segments

(foot, talus, shank, and thigh of each limb and the pelvis) and 24 degrees of freedom (DOF) using Mocap Solver 6.17 software (Motion Analysis, Corp.). We have used these methods extensively to successfully quantify lower limb joint rotations for these and similar movements (6,34,36). Specifically, the pelvis was assigned six DOF relative to the global (laboratory) coordinate system, with the hip, knee, and ankle joints of each limb defined locally and assigned three rotational DOF, respectively (6). Hip, knee, and ankle joint centers were also defined in accordance with our previous work (6). The 3D marker trajectories recorded during each landing trial were processed by the Mocap Solver software to solve for the 3D lower limb joint rotations at each time frame. Rotational data were expressed relative to each subject's standing (neutral) position data (34). These and the 3D ground reaction force (GRF) data were then low-pass-filtered with a cubic smoothing spline at a 12-Hz cutoff frequency (34,49).

Intersegmental 3D lower limb joint forces and moments were obtained by submitting filtered kinematic and GRF data to a conventional inverse dynamics analysis (34). Segment inertial characteristics were based on the work of de Leva (10). The 3D intersegmental forces (anterior-posterior, medial-lateral, and compression-distraction) at the hip, knee, and ankle were transformed to the femoral, tibial, and talar

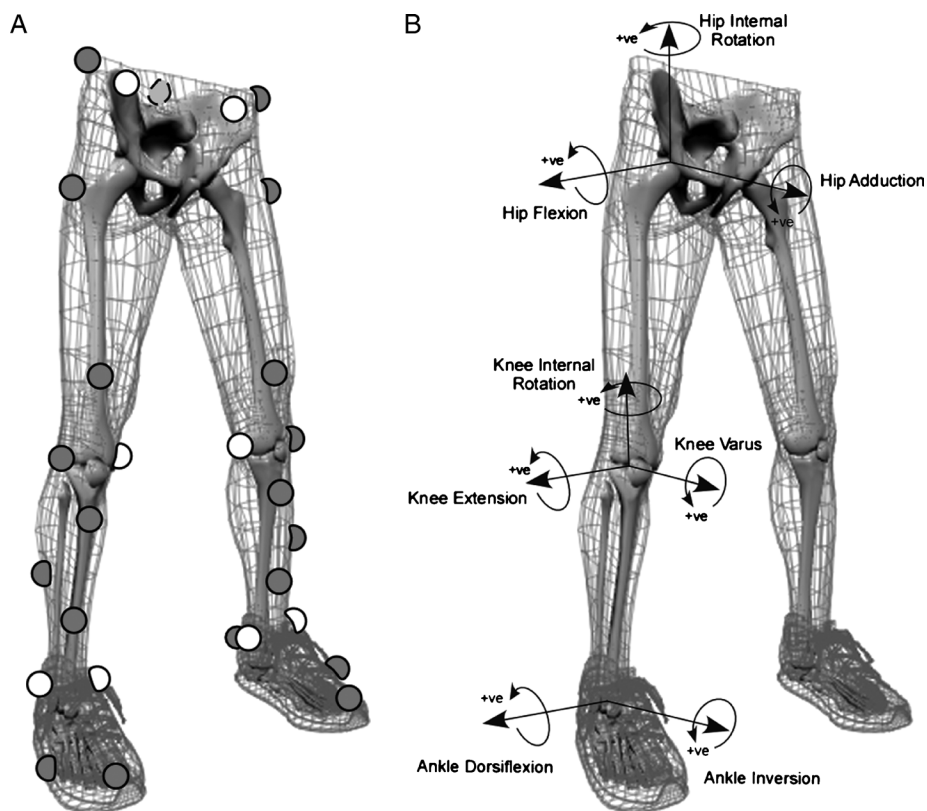


FIGURE 3—Marker locations used to define a kinematic model comprised of 9 skeletal segments (A). The *left* and *right* anterior superior iliac spine (ASIS) and bilateral medial femoral condyle and lateral and medial malleoli markers (*white*) were removed before the recording of movement trials. Pelvis (body) motion was described with respect to the global (laboratory) coordinate system via three translational and three rotational degrees of freedom (B). The hip, knee, and ankle joints were defined locally, and each assigned three respective rotational DOFs.

reference frames, respectively. Intersegmental hip and knee moments were defined as flexion–extension, adduction–abduction, and internal–external rotation with respect to the cardanic axes of their respective joint coordinate systems (34). Intersegmental ankle moments were expressed as plantar–dorsiflexion, internal–external rotation, and supination–pronation (34). Joint moments represented the external loads applied at each joint. Hence, a “knee abduction moment” described an external load that moved the knee into an abduction posture (34). For graphical purposes, kinematic and kinetic data were time-normalized to 100% of stance, with IC and toe-off being defined as the point when the vertical GRF first exceeded and fell below 10 N, respectively (6,34).

Dependent measures. Planned statistical comparisons of key kinematic and kinetic variables suggested previously to impact noncontact ACL injury risk (7,21,24,34) were undertaken to test the research hypotheses. Specifically, IC hip and knee flexion, hip internal–external rotation positions, peak hip internal rotation and knee flexion, and abduction and internal rotation angles between 0% and 50% of peak stance (PS) were obtained from each single-leg landing trial. PS (between 0% and 50% of stance) external hip flexion and internal rotation and knee flexion, abduction, and internal rotation moments were also calculated. These data were not normalized to body mass and/or height because this study did not incorporate any between-group data comparisons. Peak kinematic and kinetic values were only considered over the first 50% of stance because non-contact ACL injuries are viewed to occur early to very early in the stance phase (21). Ground contact times for each trial were also calculated, with IC and toe-off defined as above.

Statistical treatment. Mean subject-based values of each dependent factor were initially calculated from pre-fatigue jump trial data. For the remaining (fatigue) trials, data from the last trial for each of the four (two legs × two decisions) single-leg landing conditions were obtained and used to denote maximum (100%) fatigue values (6). Data for the 25%, 50%, and 75% fatigue trials were also recorded. To determine these specific trials, the total number of landing trials executed across the entire (Trial 1 to maximal fatigue) fatiguing protocol was first calculated. Trials corresponding closest (rounding down if necessary) to one fourth, half, and three fourths of this total were then determined and used for data analyses. Mean pre-fatigue maximum vertical (two-legged) jump height was calculated for each subject on the basis of the maximum height of the pelvis center of mass (6). Maximum jump heights were also calculated from the 100% fatigue vertical jump trial and represented as a percentage of the pre-fatigue baseline value (6). Between-subject variations in maximum fatigue level were subsequently examined by treating subject-based measures of this ratio as a covariate in the ensuing statistical treatment (6). The above data were then submitted to a multiple-factor mixed-design ANCOVA, testing for the main effects of and possible interactions among limb

TABLE 1. Combined effects of movement type, leg, and level of fatigue on resultant mean ± SD ground contact times (ms) during execution of single-leg landings.

Limb	Anticipated					Unanticipated						
	Pre	0%	25%	50%	75%	100%	Pre	0%	25%	50%	75%	100%
Fatigued	0.410 ± 0.059	0.418 ± 0.069	0.388 ± 0.061	0.421 ± 0.058	0.417 ± 0.061	0.419 ± 0.052	0.403 ± 0.063	0.409 ± 0.058	0.398 ± 0.064	0.389 ± 0.078	0.389 ± 0.081	0.420 ± 0.067
Nonfatigued	0.409 ± 0.037	0.400 ± 0.053	0.393 ± 0.056	0.396 ± 0.067	0.427 ± 0.062	0.401 ± 0.061	0.392 ± 0.082	0.399 ± 0.068	0.395 ± 0.087	0.386 ± 0.051	0.406 ± 0.052	0.401 ± 0.049

TABLE 2. Effect of decision and fatigue level on mean ± SD IC and PS lower limb rotations (°) during a dynamic single leg (fatigued and nonfatigued) landing.

Variable	Fatigued Limb									
	Anticipated					Unanticipated				
	Pre	25%	50%	75%	100%	Pre	25%	50%	75%	100%
IC hip flex ^a	30.6 ± 7.2	30.3 ± 6.5	28.7 ± 7.3	28.0 ± 9.5	27.8 ± 9.1	28.2 ± 6.3	28.9 ± 6.4	27.3 ± 7.9	28.0 ± 8.5	25.9 ± 9.3
IC hip int rot ^a	4.6 ± 3.2	6.7 ± 3.1	4.9 ± 2.5	5.8 ± 4.1	5.7 ± 3.0	6.6 ± 2.7	7.8 ± 2.0	8.7 ± 3.5	7.8 ± 4.1	6.6 ± 3.5
IC knee flex ^b	-16.0 ± 2.0	-13.5 ± 2.2	-12.1 ± 2.3	-11.3 ± 2.2	-10.0 ± 2.3	-16.5 ± 2.5	-14.9 ± 2.7	-13.8 ± 1.7	-13.1 ± 2.1	-11.7 ± 2.9
PS hip int rot ^{a,b,c}	8.8 ± 4.7	11.0 ± 3.6	10.1 ± 3.8	10.1 ± 3.5	9.6 ± 4.9	10.8 ± 4.7	12.4 ± 5.6	22.5 ± 3.7	19.7 ± 5.5	17.7 ± 4.8
PS knee flex ^a	-57.9 ± 8.6	-55.6 ± 10.8	-52.2 ± 11.7	-51.3 ± 8.5	-48.3 ± 11.2	-55.3 ± 7.6	-55.3 ± 10.0	-55.4 ± 10.4	-53.0 ± 11.4	-50.4 ± 8.7
PS knee abd ^{a,b,c}	-5.1 ± 3.6	-5.2 ± 3.8	-4.6 ± 3.9	-5.0 ± 3.6	-4.5 ± 3.2	-3.4 ± 2.8	-3.9 ± 3.8	-8.5 ± 2.4	-8.5 ± 3.9	-7.2 ± 3.5
PS knee int rot ^a	13.1 ± 6.1	13.7 ± 7.5	13.3 ± 6.9	14.5 ± 7.7	13.5 ± 7.4	14.6 ± 6.5	15.4 ± 6.7	15.1 ± 7.1	15.7 ± 7.2	14.8 ± 7.3

For the above rotations: hip flexion, hip internal rotation, knee extension, knee adduction, and knee internal rotation are positive.

^a Decision.

^b Fatigue level.

^c Fatigue level × Decision.

(fatigued or nonfatigued), fatigue level (pre, 25%, 50%, 75%, or 100%), and decision (anticipated or unanticipated). Decision and fatigue level were treated as a repeated measure within each subject. An initial α level 0.05 denoted statistical significance for all comparisons. In instances where a significant effect of fatigue, or interactions between main effects was observed, a Tukey *post hoc* analysis, with an adjusted α for our multiple statistical comparisons, determined precisely where they occurred.

RESULTS

Subjects completed an average of 64.5 ± 13.2 successful series of three single-leg squats and a randomized landing before reaching maximal volitional fatigue, which equated to approximately 18 ± 2 min. In addition, subjects also performed 5.2 ± 2.8 unsuccessful movements over the fatigue protocol, which were excluded from statistical treatment. Percentage maximum jump heights at 25% fatigue (96.09 ± 2.5, $P = 0.821$) were statistically similar to mean baseline values. Percentage height values at 50% (79.8 ± 14.3%), 75% (77.5 ± 15.6%), and 100% fatigue (74.7 ± 12.3%), however, were statistically significantly ($P < 0.001$) lower than the pre-fatigue and the 25% fatigue values. Furthermore, percentage maximum jump height values at 50%, 75%, and 100% fatigue were found to be statistically similar. Between-subject variations in maximum percentage jump heights also did not influence the remaining statistical outcomes, suggesting that the ensuing statistical comparisons could be made with confidence. Fatigue (observed power =

0.377), decision making (observed power = 0.394), or limb conditions (observed power = 0.422) did not statistically influence mean contact time data (Table 1). Considering the above results, therefore, it was felt that the remaining statistical treatments could be evaluated with confidence.

Several biomechanical parameter comparisons were influenced by the main effect of fatigue level (Tables 2 and 3). With regard to kinematics, statistically significant ($P < 0.01$) decreases in IC knee flexion and increases in PS hip internal rotation and knee abduction postures occurred as fatigue progressed through to maximum (100%). Specifically for each of these three parameters, values at 50%, 75%, and 100% fatigue were found statistically to be significantly ($P < 0.01$) different from pre-fatigue and 25% fatigue levels (Fig. 4). Differences were not observed, however, among 50%, 75%, and 100% values or between pre-fatigue and 25% fatigue values in each case. PS hip internal rotation and knee abduction moments significantly ($P < 0.01$) increased as fatigue progressed, with values at 50%, 75%, and 100% fatigue being significantly ($P < 0.01$) greater than pre-fatigue and 25% fatigue levels (Fig. 5). Differences were not observed, however, among 50%, 75%, and 100% or between pre-fatigue and 25% fatigue levels. PS knee flexion moments observed at 50%, 75%, and 100% fatigue were also significantly ($P < 0.01$) smaller than those at pre-fatigue values. In addition, 100% fatigue PS knee flexion moment values were significantly larger than the corresponding 25% fatigue measures.

Decision making impacted key hip and knee kinematic parameters during the single-leg landing tasks (Table 2).

TABLE 3. Effect of decision and fatigue level on mean ± SD PS lower limb external moments (N·m) during a dynamic single leg (fatigued and nonfatigued) landing.

Variable	Fatigued Limb									
	Anticipated					Unanticipated				
	Pre	25%	50%	75%	100%	Pre	25%	50%	75%	100%
PS hip flex ^a	-106.5 ± 46.5	-113.8 ± 40.2	-114.9 ± 48.4	-110.3 ± 34.4	-107.5 ± 32.0	-108.4 ± 37.4	-114.0 ± 34.3	-107.8 ± 31.2	-113.1 ± 40.4	-105.0 ± 46.1
PS hip int rot ^{a,b,c}	-8.7 ± 3.4	-14.5 ± 4.6	-15.6 ± 6.3	-15.5 ± 5.0	-15.9 ± 5.2	-11.2 ± 3.1	-16.4 ± 5.5	-20.0 ± 6.2	-17.5 ± 7.1	-16.6 ± 8.0
PS knee flex ^{b,c}	142.9 ± 18.1	123.3 ± 21.5	119.0 ± 21.0	110.7 ± 22.7	99.9 ± 23.8	147.5 ± 19.5	139.0 ± 21.6	129.3 ± 23.3	124.0 ± 27.7	115.8 ± 25.6
PS knee abd ^{b,c,d}	40.1 ± 7.5	37.5 ± 8.1	41.3 ± 5.0	38.5 ± 12.3	38.0 ± 5.1	34.1 ± 9.4	38.4 ± 11.0	60.1 ± 14.0	60.4 ± 10.7	61.5 ± 12.2
PS knee int rot ^b	-12.5 ± 5.8	-14.0 ± 6.9	-13.9 ± 5.3	-13.9 ± 6.1	-13.5 ± 6.5	-18.2 ± 7.5	-18.2 ± 6.6	-18.0 ± 8.2	-17.8 ± 7.1	-17.1 ± 5.9

For the above external moments: hip flexion, hip internal rotation, knee extension, knee adduction, and knee internal rotation are negative.

^a Limb.

^b Decision.

^c Fatigue level.

^d Fatigue level × Decision.

TABLE 2. (Continued)

Variable	Nonfatigued Limb									
	Anticipated					Unanticipated				
	Pre	25%	50%	75%	100%	Pre	25%	50%	75%	100%
IC hip flex ^a	30.8 ± 7.6	31.7 ± 7.0	30.7 ± 8.8	30.6 ± 8.9	30.9 ± 7.7	28.6 ± 6.8	29.5 ± 8.6	29.2 ± 7.5	27.6 ± 9.5	27.5 ± 10.3
IC hip int rot ^a	3.7 ± 3.8	5.2 ± 2.1	5.6 ± 1.7	6.3 ± 3.0	6.4 ± 2.1	6.9 ± 1.2	6.8 ± 2.3	7.1 ± 4.0	9.0 ± 3.4	6.9 ± 1.9
IC knee flex ^b	-16.5 ± 3.4	-12.8 ± 2.8	-12.5 ± 3.0	-12.4 ± 3.2	-13.2 ± 2.5	-16.9 ± 2.0	-16.3 ± 3.1	-14.5 ± 3.0	-12.5 ± 2.4	-12.4 ± 1.7
PS hip int rot ^{a,b,c}	8.0 ± 3.5	9.4 ± 4.0	10.1 ± 4.4	10.8 ± 3.9	10.4 ± 3.1	10.3 ± 4.7	11.7 ± 3.6	16.7 ± 4.8	19.3 ± 4.5	17.6 ± 3.9
PS knee flex ^a	-58.7 ± 7.4	-55.9 ± 8.0	-53.7 ± 7.8	-55.2 ± 8.2	-53.4 ± 8.5	-54.8 ± 6.2	-53.0 ± 9.4	-50.1 ± 8.0	-51.4 ± 7.3	-50.2 ± 9.0
PS knee abd ^{a,b,c}	-3.8 ± 2.9	-4.3 ± 2.9	-5.4 ± 3.2	-5.0 ± 3.6	-4.4 ± 3.0	-3.9 ± 2.6	-4.3 ± 2.7	-7.6 ± 3.4	-10.3 ± 3.6	-11.0 ± 2.6
PS knee int rot ^a	13.2 ± 5.4	13.7 ± 5.8	14.3 ± 6.4	14.8 ± 6.8	14.4 ± 6.5	14.7 ± 5.7	15.8 ± 6.2	15.6 ± 6.3	15.3 ± 6.5	15.1 ± 6.9

Unanticipated landings elicited statistically significant ($P < 0.01$) decreases in IC hip flexion and increases IC hip internal rotation compared with anticipated landings. Statistically significant ($P < 0.01$) increases were also observed in PS hip internal rotation and knee abduction and internal rotation postures during unanticipated compared with anticipated landings. Hip and knee kinetics were also influenced directly by the main effect of decision making (Table 3). PS hip internal rotation and knee flexion, abduction, and internal rotation moments were all observed statistically to be significantly ($P < 0.01$) larger during unanticipated compared with anticipated landings. Statistically significant ($P < 0.01$) interactions between the main effects of decision and fatigue level were also observed for PS hip internal rotation angles and knee abduction angles and moments (Figs. 4 and 5). Specifically, fatigue-induced increases in each of three parameters were statistically more pronounced during unanticipated compared with anticipated landings.

The main effect of limb affected specific hip kinetic parameters during the landing phase (Table 2). Statistically, PS hip flexion moments were significantly ($P < 0.01$) larger in the nonfatigued compared with the fatigued limb, whereas the reverse was true for PS hip internal rotation moments, with significantly ($P < 0.01$) larger values observed for the fatigued limb (Fig. 5). Limb was not found statistically to significantly influence any of the remaining kinematic or kinetic parameters. Further, statistically significant interactions between limb and either of the remaining two main effects were not observed for any dependent measures.

DISCUSSION

Perceived deficiencies in ACL injury prevention methods have promoted increased consideration of sports relevant factors within the laboratory-based testing environment

(3,6). Such efforts have highlighted the potentially important role of neuromuscular fatigue within the noncontact ACL injury mechanism and the equally important need to counter its debilitating effects (7,25,34). Limited insight exists, however, into how fatigue may manifest within the injury mechanism. In particular, the potential for central fatigue mechanisms, which may be trainable, to adversely impact the fatigued sports landing strategy was largely unclear. Current outcomes, however, suggest this may indeed be the case, with what seems to be a reasonable limb crossover in central fatigue evident for our chosen single-leg landing tasks.

The current fatigue model represents an evolving concept within our work in this area (6). Specifically, we examined lower limb mechanics in parallel with ongoing fatigue progressions (29) rather than using the typical pretest-fatigue-posttest approach (7,34). This method negates the potential for data comparisons to be adversely impacted by rapidly deteriorating fatigue effects (34). Further, it enables data to be examined at explicit points along the fatigue pathway, which may provide greater insights into fatigue contributions to ACL injury risk (6). In direct agreement with our previous work (6), lower limb joint biomechanical modifications occurred at fatigue levels well below maximum. The fact that these modifications did not change as fatigue progressed further suggests that fatigue-induced ACL injury risk may arise at a similar time point. Further work is necessary to determine the magnitude of fatigue effects necessary to compromise joint mechanics and resultant ligament integrity. As noted, trials in which an incorrect movement was executed were removed from analyses. There is of course a concern that if too many trials were deemed unsuccessful, then assertions pertaining to both resultant anticipation and/or fatigue effects may be compromised. The relatively small percentage of unsuccessful trials excluded, however, in conjunction with the equally small between-subject variability

TABLE 3. (Continued)

Variable	Nonfatigued Limb									
	Anticipated					Unanticipated				
	Pre	25%	50%	75%	100%	Pre	25%	50%	75%	100%
PS hip flex ^a	-113.3 ± 36.5	-131.6 ± 42.0	-130.8 ± 43.9	-137.8 ± 39.9	-129.6 ± 36.4	-122.2 ± 35.2	-125.8 ± 30.3	-121.4 ± 36.9	-129.6 ± 39.4	-124.2 ± 37.7
PS hip int rot ^{a,b,c}	-7.8 ± 4.3	-9.4 ± 3.9	-11.2 ± 5.5	-13.8 ± 6.0	-11.0 ± 5.8	-12.5 ± 3.9	-15.8 ± 5.8	-13.7 ± 6.1	-15.9 ± 5.4	-11.8 ± 6.0
PS knee flex ^{b,c}	136.6 ± 17.9	118.1 ± 19.2	111.8 ± 22.3	113.5 ± 21.3	111.2 ± 24.2	146.1 ± 19.6	135.6 ± 23.5	129.0 ± 20.8	123.1 ± 23.1	114.9 ± 24.5
PS knee abd ^{b,c,d}	41.2 ± 9.6	36.5 ± 12.2	37.2 ± 12.9	36.6 ± 14.1	38.4 ± 6.8	42.6 ± 8.6	45.5 ± 9.2	61.9 ± 12.7	60.5 ± 12.6	62.3 ± 13.1
PS knee int rot ^b	-14.9 ± 6.7	-13.0 ± 5.9	-13.5 ± 6.1	-15.1 ± 7.1	-14.2 ± 7.3	-20.2 ± 6.5	-21.9 ± 8.6	-19.7 ± 9.4	-18.9 ± 9.1	-17.6 ± 8.2

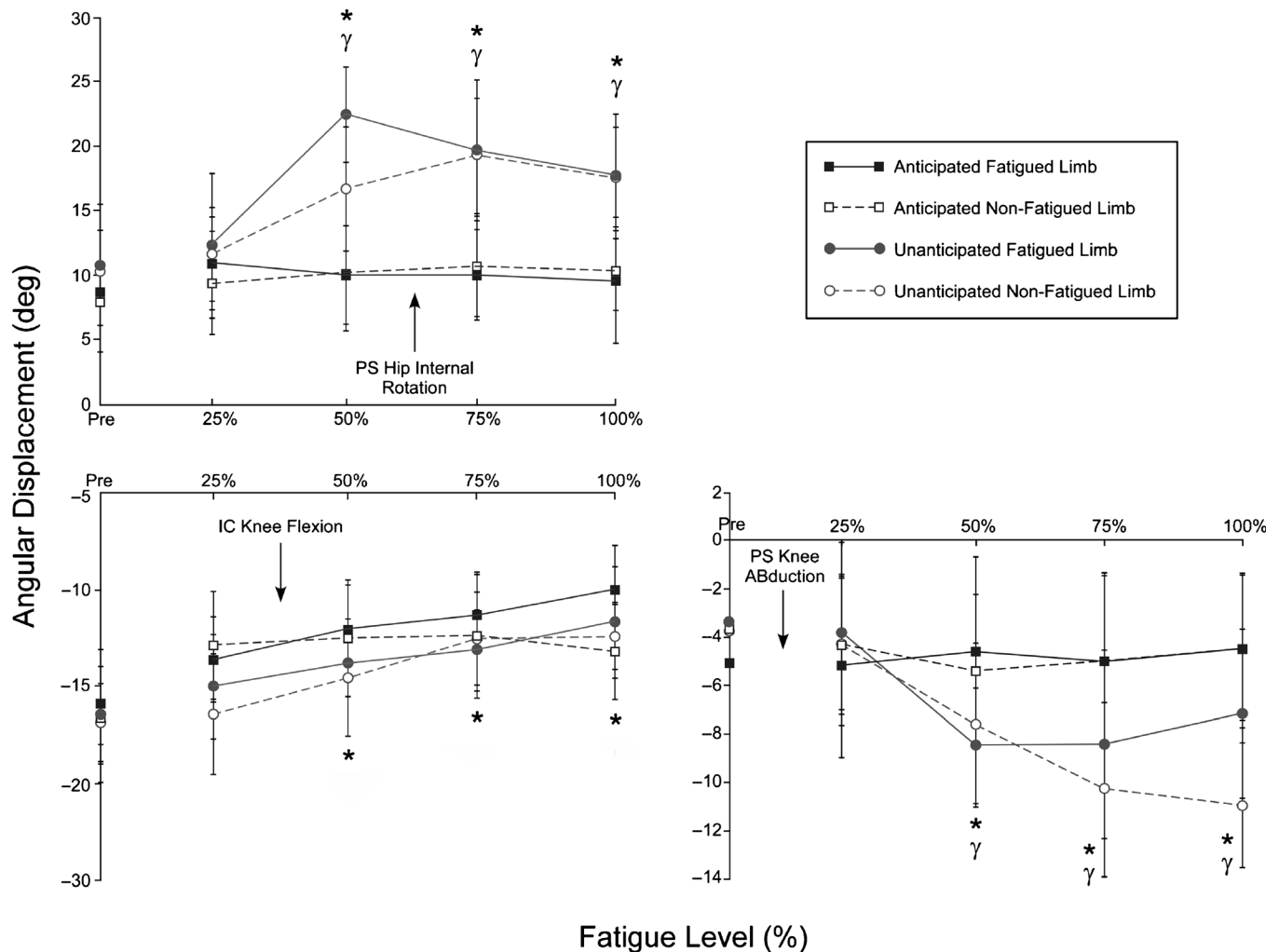


FIGURE 4—Combined effects of fatigue level and decision making on key kinematic parameters elicited during dynamic single-leg (fatigued and nonfatigued) landings. As fatigue progressed, statistically significant ($P < 0.01$) decreases in IC knee flexion, and increases in PS hip internal rotation and knee abduction postures were observed compared with prefatigue and 25% fatigue values (*). Fatigue-induced increases in PS hip internal rotation and knee abduction were also statistically more pronounced in unanticipated compared with anticipated landings (γ) at 50%, 75%, and 100% fatigue levels.

in this percentage, suggests that this is currently not the case. We tested an elite athletic population with notable experience in performing our chosen movements, which may explain the high consistency in successfully performing these tasks. Increased movement error, however, may occur within a recreationally active population, which may adversely impact data interpretation within a similar experimental design. Such possibilities should thus be considered within future work in this area.

As we initially hypothesized and similar to previous work (6,7,25,34), modifications in hip and knee landing mechanics existed within the fatigued limb. Increases in PS hip internal rotation angles and moments, for example, were a commonly observed outcome of fatigue. Our fatigue model was implemented with the intent of in addition fatiguing the out-of-plane hip stabilizers during the repetitive squatting tasks. Although we did not measure hip rotator muscle activations explicitly, these biomechanical outcomes suggest that we achieved this goal. Increased hip internal rotation

postures and loads during sports landings are purported to increase ACL injury risk through resultant increases in knee abduction load states (35,36). The fact that we saw concomitant increases in PS knee abduction moments may support this tenet. Increased hip internal rotation promotes suboptimal biarticular quadriceps and hamstring lengths (12), limiting their ability to successfully oppose the large extrinsic abduction loads often associated with dynamic landings (4). In addition, with lower limb muscle fatigue increasing knee laxity (48), a compromise in ligament mechanoreceptor feedback is likely (27), further inhibiting muscle stabilization against these extreme load states. It seems, therefore, that improving hip strength and control, particularly in the presence of fatigue, should remain a key focus of ongoing ACL injury prevention developments.

The tendency for the fatigued limb to land with the knee more extended in conjunction with a reduced PS knee flexion moment likely represents an adaptive strategy to ensure a successful landing. With the ability of the fatigued

knee extensors to eccentrically control center of mass deceleration after landing being compromised, a more extended knee would counter the excessive lower limb collapse that would otherwise occur (6). Although not statistically significant, a similar trend was evident at the hip joint, which seems intuitive considering its primary stabilizing role during the absorption phase of landing tasks (11). We are unsure as to why fatigue did not impact the IC hip flexion position more substantially. It may have been that our fatigue protocol compromised hip musculature to the point that adequate lower limb stabilization was impossible, regardless of the initial hip posture. Greater knee extension at landing, therefore, would accommodate for this ineffective hip response. Considering also the relatively difficulty of the single-leg landing task, a reasonable amount of hip flexion may have been necessary to maintain a stable center of mass position for successful task execution. Reasonable deviation in this hip posture may have thus rendered successful performance

of the landing task impossible. Although not critical to the outcomes of this study, an integrative assessment of lower limb biomechanical and muscle activation strategies would provide additional insights here.

Whereas the inclusion of fatigue within our testing model likely improves its sports efficacy, limiting assessments to safe landing movements renders the determination of explicit injury risk difficult. Inferences regarding potential causality may be made, however, when the fatigue-induced 3D knee biomechanical profile is considered. Landing with a more extended knee posture, for example, is proposed to increase ACL loading via concomitant increases in the quadriceps-driven anterior tibial shear force (7,21). The addition of a relatively large extrinsic knee abduction load, therefore, which itself increases ACL loading (31), likely presents a combined load state with reasonable potential for injury. Current outcomes thus tend to further support the contention that fatigue may be an integral component

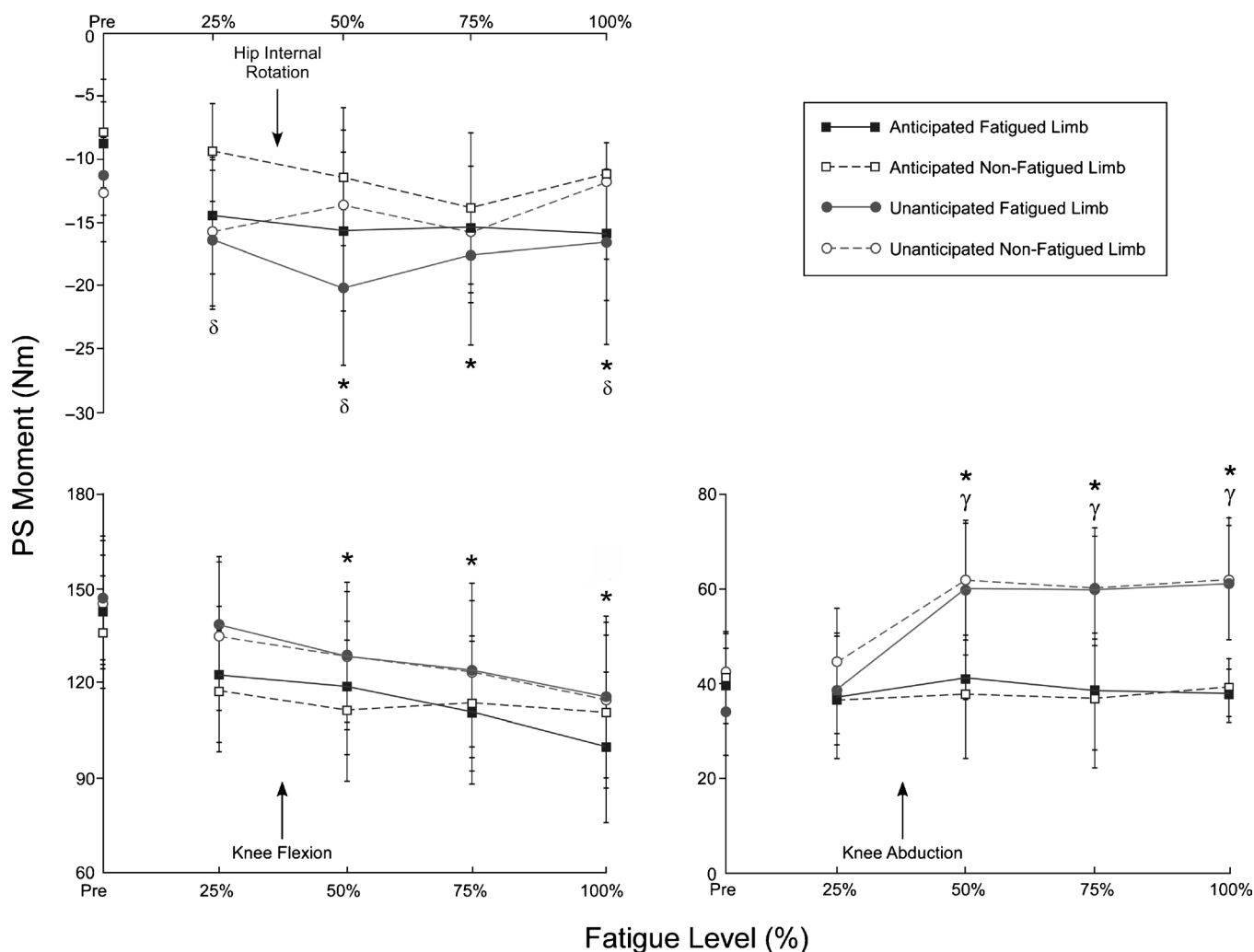


FIGURE 5—Combined effects of fatigue level and decision making on key kinetic parameters elicited during dynamic single-leg (fatigued and nonfatigued) landings. As fatigue progressed, statistically significant ($P < 0.01$) decreases in PS knee flexion, and increases in PS hip internal rotation and knee abduction moments were observed (*). Fatigue-induced increases in PS knee abduction moments were also more pronounced in unanticipated compared with anticipated landings (γ) at 50%, 75%, and 100% fatigue levels. In addition, PS hip internal rotation moments were significantly larger in the fatigued compared with the nonfatigued limb (δ) at 50% and 100% fatigue levels.

of the sports-related noncontact ACL injury mechanism (6,7,25,34).

Outcomes of this study add further strength to the claim that performing an unanticipated landing in a fatigued state may represent a worst-case scenario for noncontact ACL injury risk (6). Fatigue-induced increases in PS hip internal rotation angles and knee abduction angles and loads, for example, known neuromechanical risk factors (21,24), were even more pronounced during unanticipated landings. For anticipated movements, a preplanned movement strategy affords central (spinal and supraspinal) control mechanisms adequate time to stabilize lower limb joints via appropriate muscle action (3,6,26). For unanticipated movements, however, temporal constraints placed on these same mechanisms compromise the evoked motor response such that suboptimal muscle behaviors (8,16) and inadequate joint stabilization prevail (3,6). Fatigue is proposed to further impact the unanticipated movement response via additional compromise within these same governing central pathways (6). Of course, such assertions cannot be made via an isolated assessment of fatigued limb biomechanics because individual central and/or peripheral fatigue contributions are impossible to delineate within such a model (19). Further insights are gained, however, when the bilateral limb biomechanical responses to the combined fatigue–decision-making paradigm are considered.

The biomechanical changes observed in the nonfatigued contralateral limb provide direct evidence of a central fatigue mechanism within the fatigued sports landing strategy. Further, and contrary to previous thoughts (40), the magnitude of this crossover effect suggests isolated central fatigue mechanisms may be large enough to promote potentially hazardous postural adjustments. As noted earlier, several theories are proposed to explain the existence of a crossover in central fatigue (19,30,44,46). Inhibition of the α -motor neuron pool and hence altered central motor drive, for example, is suggested to arise from Groups III and IV afferents, considered sensitive to changes in muscle metabolism as fatigue progresses. In the current study, maximal fatigue was considered the point where subjects could no longer perform three consecutive squatting tasks. It seems plausible that the metabolic changes likely evident within the dominant muscle groups (e.g., quadriceps and gluteals) at this point would be large enough to elicit an anticipatory down-regulation in central control. That homologous muscle activity is impeded by the increased central activation necessary to maintain force production in the fatigued limb (46), in addition, may explain why a crossover in central fatigue was only evident for unanticipated landings. With unanticipated tasks already challenging central control pathways (3,6), only small additional increases in central activation may be necessary to promote an altered contralateral movement response. The preplanned control strategy associated with an anticipated landing, however, may still afford successful movement execution in spite of fatigue-induced central activation increases. Hence, whereas central fatigue may be a dominant mechanism within a

fatigued landing, its potential to cause ACL injury may only be realized for tasks that adequately compromise supraspinal and/or spinal control pathways. Injury may still prevail of course, if a centrally mediated fatigue response manifests in conjunction with substantial peripheral fatigue, where reduced muscle force and insufficient joint stabilization already exist.

Although we are confident that fatigued landing responses arose via a central fatigue mechanism, it is possible that biomechanical adaptations observed in the uninvolved limb may have also arisen via additional peripheral factors. Our increased focus on fatiguing the hip muscles, for example, may have induced peripheral fatigue in bilateral hip stabilizers. The increased PS hip internal rotation moment observed in the nonfatigued limb, which may have, in turn, impacted knee loading (35), may imply such an occurrence. Of course, this altered hip strategy may have simply arisen to compensate for the already dominant central fatigue adaptations experienced elsewhere along the kinetic chain. Delineating cause and effect within our *in vivo* experimental model, however, is impossible. Unintended muscle contractions in the uninvolved limb during fatigue and/or landing tasks may have also induced reasonable peripheral adaptations, although this does not seem likely (41). An included assessment of bilateral muscle activation behaviors would help answer this question. More innovative research models that extend beyond an isolated lower limb focus may also provide additional insights, and we intend to explore these ideas in our ongoing research efforts. Regardless, central fatigue seems to be a critical governing factor during fatigued sports landings and may thus play an equally important role within the resultant ACL injury mechanism.

Outcomes of the current study may have immediate implications for ongoing prevention methods. In particular, targeted training of supraspinal and spinal control mechanisms impacting dynamic landing strategies may provide an effective means to oppose debilitating central fatigue effects. Exposure to more complex (37) or cognitively demanding (3,6) movement tasks may facilitate improved perception and decision making within the random sports environment. Mental imagery (16), for example, may present as an effective means to develop central control strategies that successfully transfer to the fatigued movement environment. In addition, virtual reality technology affords realistic simulation of complex real-world scenarios, enabling individuals to be effectively “immersed” within and to demonstrate control over the inherent movement environment (45). Through this process, a detailed construction of knowledge and an advanced training and retention of cognitive skills linked to problem solving and rapid decision making is possible (45,47). Such benefits have already rendered virtual reality an increasingly important teaching/training tool within a variety of clinical, pathologic, military, and extreme workplace settings (45). It seems intuitive that similar technologies would afford equal benefit and success when applied to the prevention of injuries within a complex sports environment. It may also be

possible to train “hard-wired” spinal control mechanisms to further combat fatigue effects. Targeted functional electrical stimulation of specific muscle groups, for example, has been shown to improve reaction time through an enhanced reflex response and subsequent reduction in the muscle premotor phase (15). The longevity of these potential benefits, however, and their ability to be maintained in the presence of fatigue remain unclear. Furthermore, integrating any of these prementioned techniques within a large scale prevention program will be particularly challenging. Nevertheless, prevention methods that can successfully oppose the potentially devastating impact of central fatigue on lower limb landing biomechanics seem worthy of consideration and exploration.

CONCLUSIONS

This study has shown that exposure to a general unilateral lower limb fatiguing protocol induces a crossover in fatigue to the contralateral limb during dynamic single-leg

landings. In doing so, it has highlighted the likely role of central fatigue within the fatigue-induced sports landing strategy and its potential to precipitate high-risk hip and particularly knee joint biomechanics. The likelihood for ACL injury seems further enhanced when unanticipated landings are executed in a fatigued state. In this instance, additional compromise within already taxed supraspinal and spinal control pathways, likely arising through the inhibitory action of the fatigued muscles, increases the possibility of ineffective perception, decision, and movement execution strategies. Targeted training of central control mechanisms should thus necessarily be incorporated within the ACL injury prevention strategy to offset successfully the potentially catastrophic biomechanical outcomes associated with central fatigue.

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