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Effects of Muscular Fatigue on Knee Joint Laxity and Neuromuscular Characteristics of Male and Female Athletes

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Objective: To elucidate the effects of muscular fatigue on knee joint laxity and the neuromuscular characteristics of male and female athletes. We were particularly interested in determining whether such effects would be more pronounced in female athletes than in males participating in the same sport.

Design and Setting: Subjects were assessed on 4 dependent variables during a rested and an isokinetically induced muscular fatigue state. We ensured that posttesting measurements were obtained in the fatigued state by testing only 2 dependent variables after each exercise bout.

Subjects: We recruited male (n = 17) and female (n = 17) subjects from a population of healthy collegiate basketball and soccer players.

Measurements: Measured dependent variables were as follows: anterior tibial translation, kinesthesia determined by assessing the threshold to detection of passive motion moving into knee flexion and extension; lower extremity balance ability quantified through a stability index value; and the electromyography-measured muscle activity of 6 knee-stabilizing muscles.

Results: In response to muscular fatigue, subjects demonstrated an overall decrease in the ability to detect joint motion moving into the direction of extension, an increase in the onset of contraction time for the medial hamstring and lateral gastrocnemius muscles, and an increase in the first contraction area of the vastus medialis and vastus lateralis muscles. Additionally, the increase in area of the vastus lateralis was greater for the males compared with the females.

Conclusions: Our results suggest that both male and female athletes exhibit decrements in proprioceptive ability and alterations in muscular activity subsequent to muscular fatigue.

Key Words: electromyography, anterior cruciate ligament, proprioception

As the number of females participating in high school and collegiate sports has increased, so has the incidence of trauma to the anterior cruciate ligament (ACL). Epidemiologic injury surveillance has demonstrated and continues to show a high and disproportionate number of ACL injuries occurring to female athletes participating in the sports of soccer and basketball, compared with their male counterparts.[1–10] While numerous factors have been suggested and investigated as the underlying causes of the disproportionate rate of ACL injury,[11–20] research has increasingly focused on the impact of excessive joint laxity, joint proprioception, and muscle activation patterns on functional knee joint stabilization.

In healthy athletes, functional joint stabilization is routinely achieved as the body gathers visual information, proprioceptive information from articular and musculotendinous receptors, and vestibular system information from the vestibules and semicircular canals of the ears. Once gathered, this information is processed by the central nervous system and results in the maintenance of posture and balance, the conscious appreciation of joint motion and position sense, and protective spinal-mediated reflexes. In addition to protective reflexive muscular activity, athletes appear to achieve functional joint stabilization by relying on some form of preactivated muscle tension in anticipation of expected joint load, whereby previously experienced muscle activation patterns and joint motions preprogram or "feed-forward" muscle activity.[21–26] This combination permits athletes to routinely stabilize the knee joint against potentially damaging joint forces.[21–26] However, it has been suggested that female athletes, who injure the ACL at a greater rate than males, may inherently possess alterations in joint proprioception and aberrations in muscle activation that may affect their ability to stabilize the joint and subsequently may predispose them to ACL injury.

Therefore, we initially conducted and published an investigation aimed at elucidating the sex differences in knee joint laxity and neuromuscular characteristics of collegiate-level soccer and basketball players.[27] We were particularly interested in determining whether previously uninjured male and female basketball and soccer players achieve functional joint

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stabilization through similar means. Interestingly, our results revealed that, compared with males, females who participate in basketball and soccer inherently possess greater knee joint laxity and demonstrate a longer time to detect knee joint motion moving into extension, rendering the knee less sensitive to potentially damaging forces. Additionally, the results of this investigation revealed that these female athletes exhibit greater electromyography (EMG)-measured peak amplitude and area of the lateral hamstring when landing, suggesting that they may have adopted a compensatory muscle activation pattern to routinely achieve functional joint stabilization. We concluded that female and male athletes, specifically those participating in soccer and basketball, differ in their means of routinely achieving knee joint functional stabilization.[27]

Since ACL injuries in females often occur during routine noncontact activities, female athletes may be able to routinely rely on compensatory muscle activation patterns to perform potential injury-causing activities without sustaining trauma to the ACL. Theoretically, a female athlete could perform many run-and-stop maneuvers or landings while rebounding a basketball and continually maintain joint stabilization against potentially damaging forces. However, interruption of the compensatory mechanism relied on for dynamic stability may produce a joint unable to sense and respond to imparted joint forces, resulting in ligamentous trauma.

Muscular fatigue is considered a viable culprit to interrupt the compensatory stabilizing mechanism, as research has demonstrated its deleterious effects on knee joint laxity,[28–31] as well as both the afferent and efferent neuromuscular pathways.[29,32–36] In response to various exercise protocols designed to induce muscular fatigue, increases in knee joint laxity have been documented in male and female athletes.[28–31,37] Even though researchers have studied the sexes in the investigation of muscular fatigue's effects on joint laxity, exercise protocols between the sexes have varied, making comparisons by sex virtually impossible. Knee joint proprioception, determined by examining joint kinesthesia (the ability to sense joint motion) and joint position sense (the perception of joint position), appears to be both directly and indirectly affected by muscular fatigue. Directly, muscular fatigue appears to worsen or impair expected learning improvements in joint position sense,[29,34] while having no apparent effect on joint kinesthesia.[29,36] Indirectly, muscular fatigue affects proprioception in that alterations in kinesthesia and joint position sense have been demonstrated secondary to increased knee joint laxity.[38–40] The efferent pathway, assessed by measuring balance or by examining muscle activity with EMG, appears to also be affected by muscular fatigue. After an isokinetic exercise program designed to induce muscular fatigue, healthy individuals have demonstrated decreased balance ability, suggesting that fatigue results in motor control deficits.[33] EMG studies suggest that, in addition to balance, muscular fatigue affects muscle activity by extending the latency of muscle firing[35] and by resulting in less efficient muscular processes.[32]

For female basketball and soccer players, the effects of muscular fatigue may be more deleterious than those occurring to their male counterparts and, therefore, may lead to a greater risk for ACL injury. These female athletes may be affected to a greater extent since they are inherently different from their male counterparts. As our previous study[27] suggests, female basketball and soccer players inherently possess greater knee joint laxity and diminished joint kinesthesia and rely on an apparently adopted pattern of increased muscle activation to achieve functional joint stabilization. Therefore, even if muscular fatigue affects the joint laxity and neuromuscular characteristics of male and female athletes to a similar degree, the females' ability to achieve functional joint stabilization may be affected to a greater extent.

Understanding the impact of muscular fatigue on knee joint laxity, as well as its impact on joint proprioception and muscle activity, we conducted a study to elucidate the effects of muscular fatigue on knee joint laxity and the neuromuscular characteristics of male and female athletes. We were particularly interested in determining whether such effects would be more pronounced in female athletes than in males participating in the same sport. Therefore, the purpose of our study was to examine and compare, by sex, the effects of muscular fatigue on the knee joint laxity, joint proprioception, balance, and EMG-assessed muscle activity of male and female athletes.

METHODS

Subjects

This study recruited male (n = 17; age = 20.4 ± 1.7 years, height = 181.5 ± 7.2 cm, weight = 80.3 ± 10.3 kg) and female (n = 17; age = 18.9 ± 0.9 years, height = 168.5 ± 4.9 cm, weight = 65.6 ± 8.3 kg) subjects from a population of male and female collegiate basketball players and soccer players at the University of Pittsburgh and surrounding colleges and universities. Subjects were healthy, had no history of knee joint ligamentous trauma to either lower extremity, and possessed a self-proclaimed functionally stable test limb ankle. Additionally, no subject reported suffering from any systemic or vestibular disorders known to impair cutaneous sensation or balance. All subjects gave written consent to participate in this study, which was approved by the University of Pittsburgh Investigatory Review Board for Biomedical Research. For all subjects, the dominant lower extremity limb served as the exclusive data collection limb. For this investigation, dominance was established by ascertaining which lower extremity the subject would prefer to land on when dropping from a 25.4-cm high step.

Instrumentation

Anterior tibial translation. To quantify knee joint laxity we utilized the KT-1000 (MEDmetric, San Diego, CA) instrumented knee arthrometer to measure anterior tibial translation

during the application of a 133-N (30-lb) anterior displacement force. Subjects were tested in the supine position with the knee joint in 20° of flexion, as confirmed by a goniometric reading. We employed the manufacturer's specifications for placement of the KT-100 onto the test limb as well as for all data collection. Three trials were performed, with the millimeters of anterior tibial translation recorded for each. From the 3 test trials, a mean test score was calculated and used for data analysis.

Although questioned,[41] the reliability of the KT-1000 has been established, even though it appears to be dependent upon the knee joint test angle.[42–44] Conducted at 20° of knee flexion, interexaminer and intraexaminer reliability studies have reported Pearson product-moment correlation coefficients of $r = 0.85$ and $r = 0.83$, respectively.[42–43] McLaughlin and Perrin,[44] investigating the intraexaminer reliability at 20° of knee joint flexion, reported an intraclass correlation coefficient of $r = 0.92$. We enhanced the reliability of this test device by ensuring that data collection was performed by a single researcher.

Knee joint proprioception. The assessment of the perception of knee joint proprioception was conducted by measuring knee joint kinesthesia, the ability to sense joint motion. We determined kinesthesia clinically by establishing the threshold to detection of passive motion (TTDPM), an assessment of the ability to detect relatively slow passive joint motion. For this investigation, TTDPM was established by using a proprioception testing device, which moves the knee joint into flexion or extension, while a rotational transducer interfaced with a digital microprocessor counter provides angular displacement values. We gathered TTDPM data, moving into both knee flexion and extension, by following a well-established research protocol.[45–48] Six random trials, 3 moving into flexion and 3 moving into extension, were performed, and the degrees of angular motion were recorded for each. A mean test score for each direction (into flexion and into extension) was calculated from the 3 test trials and used for data analysis. Reliability of the proprioception testing device has previously been established, with correlation coefficients of $r = 0.92$.[47]

Single-leg balance assessment. To assess lower extremity balance, we used a commercially available balance device, the Biodex Stability System (Biodex Inc, Shirley, NY). The Biodex Stability System consists of a movable balance platform that is interfaced with computer software (Biodex, version 3.1), which enables the device to perform as an objective assessment of balance. Reliability of the Biodex Stability System has been established with an interclass correlation coefficient ranging from 0.6 to 0.95.[49] For all data collection, the platform was set and remained at stability level 2, indicating a degree of platform instability that ranges from 1 to 6, with 1 representing the greatest amount of instability.

For all single-leg balance data collection, we employed an established laboratory protocol whereby subjects were asked to single-leg stand on the balance platform with both arms folded across the chest and maintain the unsupported limb in a position of 0° of hip flexion and 90° of knee flexion while slightly abducting, so as not to contact the test limb.[27,49] For 20 seconds, subjects attempted to maintain this unstable platform in a level position. For each test of balance the software program generated a stability index value, which was calculated by assessing the time and degree the platform was out of level. Three practice and 3 test trials were performed. From the 3 test trials, a mean stability index score was calculated and used for data analysis.

Muscle activity in response to landing. We collected muscle activity data in response to a landing task with the use of surface EMG, which is the study of the motor unit action potential. Muscle activity of the test limb was measured simultaneously in 6 knee joint muscles, as subjects, using only the test limb, dropped from a 25.4-cm high step and landed on the floor. The task of dropping and landing was selected based on epidemiologic injury data, which suggests that the primary mechanism of ACL injury in basketball and soccer players is a noncontact-type mechanism, such as landing from a jump.[1]

Before data collection, the electrodes were affixed to the skin in accordance with a previously established protocol.[27] To prepare the skin, the area of electrode placement was abraded with a pumice stone and cleaned with isopropyl alcohol, which ensured adequate surface contact for the electrodes. Ten-millimeter self-adhesive silver/silver electrodes (Multi Bio Sensors, Inc, El Paso, TX) were placed in pairs, 5 mm apart, over the muscle bellies of the following muscles: vastus medialis, vastus lateralis, medial hamstring, lateral hamstring, gastrocnemius (medial head), and gastrocnemius (lateral head). Additionally, a ground electrode was mounted on the patella.

Before determining the EMG activity during the single-leg landing task, we assessed the EMG activity of each of the 6 test muscles during a maximal isometric voluntary contraction (MVC). The MVC of the quadriceps and hamstring muscles was established using the Biodex Isokinetic Dynamometer (Biodex, Inc, Shirley, NY). After the application of the surface electrodes, the subject was seated in the Biodex testing chair with the distal lower limb secured to the test arm of the dynamometer. The knee was locked in 60° of flexion when the quadriceps muscle group was assessed and in 30° of flexion when the hamstring muscle data were collected. With the lever arm speed set at 0°/s, we instructed the subject to maximally push against the distal lower leg pad for a 5-second data-collection period. The MVC of the gastrocnemius muscle was obtained as the subject performed 1 5-second-long, manually resisted maximal isometric contraction in the direction of ankle plantar flexion.

Once MVC data were collected, the subject was asked to perform the single-leg landing test. The landing test required the subjects to use the test limb to single-leg stand atop a 25.4-cm high bench and drop from the bench to the floor, landing on the test limb. To synchronize muscle activity with landing, a footswitch, which was connected to an EMG channel, was secured to the floor within the landing

area. Landing on the footswitch signified contact with the floor. Testing required the subject to begin the landing task assessment single-leg standing and motionless on the bench, in order to establish the baseline EMG activity; the subject then dropped from the bench and landed on the floor footswitch. EMG activity was sampled from the time the subject single-leg stood on the bench until approximately 5 seconds after floor contact. Each subject performed 2 practice trials and 3 test trials. A trial was not considered for data collection if the subject landed on the contralateral limb, failed to land on the footswitch, or was unable to maintain balance upon landing.

EMG data management. For each landing task test trial, the muscle signal activity was collected by the surface electrodes and passed to a battery-operated FM transmitter worn by the subject. From the transmitter, the signal was passed to the computer, where the raw EMG data were sampled with a frequency of 2500 Hz. For each test trial, we employed Myoresearch software (Noraxon USA, Inc, Scottsdale, AZ) to calculate the onset time, amplitude, and area of the first contraction subsequent to landing, for each of the 6 muscles. From the 3 test trials, a mean score was calculated and used for data analysis. For this investigation, onset time was defined as the time in milliseconds from landing, indicated by contact with the footswitch, until the first muscle contraction. A muscle signal was considered a contraction if it exceeded the set trigger level of 10% of the MVC.

Induction of Muscular Fatigue

Using the Biodex Isokinetic Dynamometer, the test leg knee joint flexor and extensor musculature performed maximal effort concentric contractions until fatigue of the extensor musculature was quantified. However, before the initial induction of muscular fatigue, concentric peak torque of the knee joint extensors was determined. Securely fastened into the test chair of the Biodex with the knee joint of the test limb aligned with the axis of rotation of the dynamometer and the distal lower limb secured to the dynamometer's test arm, subjects initially completed 5 maximal repetitions of knee joint extension and flexion at a constant angular velocity of 180°/s. After the subject's extension peak torque was established, we conducted the first treatment intervention. This treatment consisted of 3 bouts of knee joint flexion and extension exercises with a 40-second interbout rest interval. The first and second bouts were composed of 40 maximal effort repetitions; however, subjects performed repetitions in the third set until the torque value of 3 consecutive repetitions fell below 25% of the initial knee extensor peak torque value. The number of repetitions permitted in the third bout was truncated at 90. To ensure identical treatment interventions, the number of repetitions required by the subject to meet the established criteria of the third bout was recorded and used in the second treatment intervention.

Testing Procedure

Upon arrival at the laboratory, subjects completed a detailed questionnaire designed to ensure compliance with the subject inclusion criteria and gather demographic and sport participation experience data. Once selected for the study, each subject was assigned a randomized order for pretreatment data collection of the 4 dependent variables. After pretesting, subjects performed the first exercise bout to induce muscular fatigue. To ensure that posttesting measurements were obtained in the fatigued state, only 2 dependent variables were assessed after the first exercise bout, while the other 2 were tested immediately after the second, identical exercise session. Posttesting session A measured knee joint kinesthesia and then anterior tibial translation, while posttesting session B measured muscle activity in response to landing followed by the single-leg balance assessment. To negate any compounding fatigue effects, posttesting sessions A and B were counterbalanced within sex.

Data Analysis

Twenty-two balanced analyses of variance (ANOVAs), taking into account variations due to the individual, the treatment, and sex, were performed to determine whether the treatment intervention of muscular fatigue had a significant effect on the dependent variables. Based on quantities obtained from the result of the ANOVAs, linear contrasts were performed to determine whether the response in males due to exercise was equal to the response in females due to exercise. A preset α level of $P < .05$ was selected to determine statistical significance.

RESULTS

The pretreatment and posttreatment mean test scores, by group, for the dependent measures of anterior tibial translation, TTDPM moving into extension, TTDPM moving into flexion, single-leg balance, and EMG-obtained muscle activity of 6 test muscles in response to landing are presented in Tables 1 through 6, respectively. In order to accurately and effectively present the data and results of this investigation, Tables 1 through 6 contain, with permission, the previously published pretreatment data.[27]

While results of the data analysis revealed that isokinetically induced muscular fatigue did not significantly affect anterior tibial translation, lower extremity balance, or TTDPM moving

Table 1. Effect of Muscular Fatigue on Anterior Tibial Translation (Mean ± SD)

Group	Pretreatment* (mm)	Posttreatment (mm)
Female	6.05 ± 1.46	6.06 ± 1.23
Male	4.80 ± 1.53	5.53 ± 1.66

* Pretreatment data reprinted with the permission of the *American Journal of Sports Medicine.*[27]

Table 2. Effect of Muscular Fatigue on Joint Kinesthesia (Mean ± SD) in Degrees of Angular Motion

Test	Group	Pretreatment*	Posttreatment
TTDPM† into Flexion	Female	2.81 ± 2.54	3.72 ± 2.47
	Male	1.89 ± 0.57	2.45 ± 1.14
TTDPM into Extension	Female	2.95 ± 1.47	4.48 ± 3.20‡
	Male	2.11 ± 0.63	2.82 ± 1.29

* Pretreatment data reprinted with the permission of the *American Journal of Sports Medicine.*[27]
† TTDPM indicates threshold to detection of passive motion.
‡ Indicates significant overall treatment effect ($P \leq .05$).

Table 3. Effect of Muscular Fatigue on Single-Leg Balance (Mean ± SD) in Degrees of Angular Motion

Group	Pretreatment*	Posttreatment
Female	3.27 ± 1.44	3.37 ± 1.46
Male	6.00 ± 3.06	6.57 ± 2.74

* Pretreatment data reprinted with the permission of the *American Journal of Sports Medicine.*[27]

into knee flexion, muscular fatigue significantly increased TTDPM moving into extension ($F_{1,49} = 6.50$, $P = .014$). However, this decrease in the ability to detect passive knee joint motion moving into extension was not significantly different between the groups.

Results of the EMG data analysis revealed that, overall, isokinetically induced muscular fatigue significantly increased the onset of contraction time of the medial hamstring ($F_{1,49} = 13.10$, $P = .001$) and lateral gastrocnemius ($F_{1,49} = 5.62$, $P = .022$) muscles, although there were no significant group differences. Muscular fatigue did not significantly affect the onset of contraction time of any of the other tested muscles or the amplitude of the first contraction of any of the 6 sampled muscles. For both males and females, muscular fatigue significantly increased the first contraction area of the vastus medialis ($F_{1,49} = 6.35$, $P = .015$) and vastus lateralis ($F_{1,49} = 13.10$, $P = .001$) muscles. Additionally, the vastus lateralis area increase in response to fatigue was significantly greater for the males compared with the females.

Table 4. Effect of Muscular Fatigue on Onset Time of First Contraction (Mean ± SD)

Sampled Muscle	Group	Pretreatment (ms)*	Posttreatment (ms)
Vastus medialis	Female	39.20 ± 56.66	51.97 ± 113.53
	Male	30.60 ± 51.98	24.18 ± 36.46
Vastus lateralis	Female	40.51 ± 28.21	36.27 ± 19.85
	Male	52.94 ± 70.59	29.77 ± 32.17
Medial hamstring†	Female	175.57 ± 108.56	308.60 ± 179.47
	Male	182.44 ± 91.88	277.65 ± 146.14
Lateral hamstring	Female	187.01 ± 133.19	243.36 ± 184.09
	Male	217.63 ± 108.95	298.94 ± 227.28
Medial gastrocnemius	Female	241.10 ± 141.57	120.85 ± 111.48
	Male	289.09 ± 177.96	300.28 ± 163.79
Lateral gastrocnemius†	Female	193.90 ± 155.33	265.82 ± 128.22
	Male	144.19 ± 98.58	242.79 ± 189.57

* Pretreatment data reprinted with the permission of the *American Journal of Sports Medicine.*[27]
† Indicates significant overall treatment effect ($P \leq .05$).

Table 5. Effect of Muscular Fatigue on First Contraction Amplitude (Mean ± SD)

Sampled Muscle	Group	Pretreatment (mV)*	Posttreatment (mV)
Vastus medialis	Female	361.65 ± 255.49	369.90 ± 203.78
	Male	290.87 ± 173.62	444.17 ± 277.55
Vastus lateralis	Female	315.82 ± 162.24	307.73 ± 99.25
	Male	298.00 ± 231.27	421.47 ± 287.83
Medial hamstring	Female	163.49 ± 84.45	133.83 ± 88.63
	Male	134.20 ± 66.33	124.75 ± 69.71
Lateral hamstring	Female	156.00 ± 72.59	165.86 ± 103.72
	Male	84.84 ± 43.47	97.74 ± 85.40
Medial gastrocnemius	Female	225.86 ± 223.35	233.93 ± 155.49
	Male	134.13 ± 74.70	141.68 ± 91.36
Lateral gastrocnemius	Female	131.72 ± 64.90	140.00 ± 130.98
	Male	161.45 ± 73.82	183.78 ± 150.95

* Pretreatment data reprinted with the permission of the *American Journal of Sports Medicine.*[27]

Table 6. Effect of Muscular Fatigue on the First Contraction Area (Mean ± SD)

Sampled Muscle	Group	Pretreatment (mV · s)*	Posttreatment (mV · s)
Vastus medialis†	Female	35.36 ± 18.93	44.39 ± 25.36
	Male	36.05 ± 33.53	63.47 ± 47.80
Vastus lateralis†‡	Female	27.75 ± 14.00	35.50 ± 10.89
	Male	28.59 ± 27.80	63.47 ± 51.48
Medial hamstring	Female	7.91 ± 6.04	6.37 ± 6.28
	Male	7.39 ± 6.20	4.75 ± 4.86
Lateral hamstring	Female	10.78 ± 8.34	12.52 ± 9.52
	Male	2.82 ± 2.66	1.85 ± 1.39
Medial gastrocnemius	Female	12.63 ± 15.43	12.72 ± 15.90
	Male	6.47 ± 5.42	6.72 ± 7.53
Lateral gastrocnemius	Female	6.24 ± 5.80	5.77 ± 5.20
	Male	9.76 ± 10.43	6.15 ± 4.88

* Pretreatment data reprinted with the permission of the *American Journal of Sports Medicine.*
† Indicates significant overall treatment effect ($P \leq .05$).
‡ Indicates significant sex-by-treatment interaction.

DISCUSSION

We conducted this research investigation to examine the effect of muscular fatigue on the knee joint laxity, kinesthesia, balance, and muscular activity of male and female athletes participating in the collegiate sports of soccer and basketball. Since epidemiologic data have established that females participating in these sports are sustaining ACL injuries 2 to 8 times more frequently than their male counterparts, we were particularly interested in determining whether the effects of fatigue would be more pronounced in female athletes than in males participating in the same sport.[1–10] It was hypothesized that, as a direct result of muscular fatigue or secondary to increased joint laxity, subjects would demonstrate aberrations in joint proprioception and alterations in joint-stabilizing muscle activity. We also hypothesized that these effects would be

significantly greater for the female subjects compared with the males.

For all subjects, muscular fatigue was induced in the knee joint musculature by having subjects perform multiple concentric flexion and extension contraction exercises using the Biodex Isokinetic Dynamometer. We selected an isokinetic dynamometer as the means of inducing muscular fatigue because of its ability to quantify muscle force production. Since the degree of fatigue was quantified by the subject's completing knee flexion and extension exercises until the knee extensors were able to produce only 25% of their initial peak torque, data comparisons could be made between subjects and groups. Traditionally, muscular fatigue has been defined as the inability to generate force and has been characterized not only by a loss of force-production capability, but also by localized discomfort and pain. Based on this definition, we feel our subjects' knee joint musculature was fatigued, since the quadriceps musculature demonstrated an inability to produce a preset amount of force. Unfortunately, we did not note the quantity of force produced by the hamstring musculature at the time the quadriceps musculature attained our predefined point of fatigue.

Before and after the treatment of muscular fatigue, the 4 dependent variables of anterior tibial translation, kinesthesia, balance, and muscular activity during a landing task were measured. In response to muscular fatigue, subjects demonstrated an overall decrease in the ability to detect joint motion moving into the direction of extension, an increase in the onset time of contraction for the medial hamstring and lateral gastrocnemius muscles in response to landing a jump, and an increase in the EMG area of the first contraction of the vastus medialis and vastus lateralis muscles when landing a jump. In addition, the increase in EMG area of the vastus lateralis after fatigue was greater for the male athletes compared with the females.

While muscular fatigue failed to significantly alter knee joint laxity in the subjects in our study, other authors agree that knee ligamentous structures probably undergo some increase in laxity during exercise, thereby placing athletes at risk for ligamentous injury.[28–31,37] The exercise protocols selected for many studies of knee joint laxity subject the knee joint to repetitive stresses at a high strain rate, such as occurs while running, cutting, jumping, and performing other sport participation skills. Skinner et al[29] documented a significant increase in anterior knee laxity in males as a result of an exercise protocol designed to produce muscular fatigue. Similar changes in ligamentous laxity as a result of exercise have been demonstrated by other researchers.[31,37] In response to running 5.635 km (3.5 miles), Stoller et al[37] demonstrated increases in torsional knee joint laxity measurements that remained above baseline 52 minutes after cessation of exercise. Female athletes have demonstrated increases in anterior, posterior, and total anterior-posterior laxity after sport participation.[28,30] Investigating the effect of playing basketball, running, and performing squat power lifts on knee joint laxity, Steiner et al[30] demonstrated an increase in knee joint laxity of female basketball players and runners subsequent to sport participation, but failed to showed increased laxity in subjects who performed squat power lifts or in the sedentary control group. Sakai et al[28] periodically measured the anterior knee laxity of female semiprofessional basketball players during the course of a typical day. Anterior laxity did not change during the sedentary working hours of the subjects but increased significantly with game-style basketball participation and remained above baseline 90 minutes after exercise.

Increases in joint laxity subsequent to exercise are suggested to be primarily due to the fact that joint structures, particularly the ligaments, exhibit viscoelastic characteristics.[50] Ligaments are composed of collagen and other structural proteins, and, therefore, when stressed, respond in a time-dependent and stress-dependent manner.[30,31,51] When subjected to cyclic elongation, as occurs with walking or running, the viscoelastic properties of a ligament display a time-dependent decrease in load. This decrease in load, which essentially protects the ligament from failure by continually decreasing stress, is demonstrated clinically with increased ligamentous laxity.[50]

Based on this concept, we proposed that an exercise protocol to induce muscular fatigue, which subjected the knee joint to repetitive or cyclic stress, would increase joint laxity. However, our study showed that knee joint laxity did not significantly increase after muscle-fatiguing exercise. This lack of a significant increase in joint laxity following exercise is consistent with the results of Steiner et al,[30] who demonstrated no significant increase in joint laxity after physical activities that placed high compressive loads on the joint. Even though isokinetic exercise, such as we used in this investigation, subjects the knee to repetitive stress, the resultant joint forces may be more compressive than shearing in nature. The dynamic forces occurring to the knee joint during isokinetic exercise have been investigated and related to activities of daily living, such as walking, stair climbing, and rising from a chair.[52,53] Kaufman et al[52] determined the peak tibiofemoral joint compression force and the peak anterior shear force during isokinetic knee flexion and extension exercises at 180°/s, the same angular velocity used in our study. They concluded that isokinetic exercise resulted in a compressive joint force that is roughly equivalent to ascending or descending stairs and an anterior shear force approximately equal to that occurring during walking. These findings suggest that isokinetic flexion and extension exercises do not simulate the joint forces occurring during activities of sport participation. Therefore, this method of inducing muscular fatigue may effectively create fatigued musculature without replicating the joint forces associated with sport activities such as running, cutting, and jumping, which appear to be necessary to induce alterations in ligament laxity.

After muscle-fatiguing exercise, our subjects demonstrated a significant decrease in knee joint kinesthesia, specifically the ability to detect joint motion when moving into the direction of knee extension. This decrease in joint kinesthesia contradicts

the work of Skinner et al,[29] who, in their study investigating the primary receptors for joint position sense, concluded that muscular fatigue did not significantly alter knee joint kinesthesia. Since this observed decrease in joint kinesthesia occurred in the absence of increased joint laxity, a mechanism other than increased joint laxity is responsible. However, such a mechanism is not currently explainable and appears to be as yet unsupported in the literature. More importantly, our findings suggest that muscular fatigue alters kinesthesia, the ability to sense joint motion, in both male and female basketball and soccer players. Since deficits in knee joint kinesthesia may reflect a joint unable to sense, and perhaps respond to, joint forces, these athletes may be at increased risk of ligamentous trauma. While these inferences are purely speculative, the results of our study do demonstrate that male and female basketball and soccer players experience deficits in proprioception, secondary to muscular fatigue, to a similar degree.

While the ability to sense joint motion was altered as a result of muscular fatigue, the ability to respond to joint motion, as quantified through balance assessment, was not significantly affected. Our subjects' lack of balance ability change as a result of muscular fatigue contradicts the results reported by Johnston et al,[33] who used another commercially available balance training device (KAT, Breg, Inc, San Marcos, CA) to demonstrate a significant decrease in lower extremity balance ability subsequent to an isokinetic exercise program designed to induce muscular fatigue. One plausible explanation for our findings of no significant decrease in balance ability after fatigue may relate to our method of measuring and qualifying single-leg balance ability. We chose to use a commercially available balance testing and training device, which provided us a means of objectively quantifying balance ability, but the computer-generated value we selected for quantifying balance may not have been sensitive enough to detect subtle changes. For our investigation, each balance test was qualified with a computer-generated stability index value, which was calculated by assessing the time and degree the platform was out of level, without consideration to the direction of platform displacement. Therefore, subjects could demonstrate 2 markedly different means of maintaining balance without demonstrating changes in the computer-generated stability index value. For example, a subject attempting to maintain balance could waver medially and laterally for the entire time during 1 test trial and then waiver exclusively anteriorly and posteriorly for the second trial, and the device would generate very similar stability index values for these 2 trials. This testing device, however, calculates and displays a medial-lateral tilt value, which is calculated by assessing the time and degree the platform is medially and laterally tilted, as well as an anterior-posterior tilt value, which is calculated by assessing the time and degree the platform is anteriorly and posteriorly tilted. Therefore, we might have been able to detect any existing alterations in balancing as a result of muscular fatigue had we extracted the computer-generated medial-lateral tilt and anterior-posterior tilt data from the testing device.

Even though muscular fatigue did not significantly alter the subjects' ability to respond to joint forces when measured with lower extremity balance, EMG-assessed muscle activity was significantly affected by the muscular fatigue protocol. Previously documented EMG changes resulting from muscular fatigue have primarily been the product of investigations of EMG activity in response to sustained or isometric-type contractions.[54–58] However, our investigation used EMG to measure muscle activation during a functional task. Results demonstrated that muscular fatigue significantly increased the onset of contraction time for the medial hamstring muscle and the lateral gastrocnemius muscle and significantly increased the EMG area of the vastus medialis and vastus lateralis muscles as subjects performed a landing task. These findings appear to suggest that the muscular fatigue protocol used in this investigation alters the muscle activity of the knee's dynamic stabilizers.

For both males and females, muscular fatigue significantly increased the onset of contraction time for the medial hamstring muscle and the lateral gastrocnemius muscle, 2 muscles that function to control anterior tibial translation. The underlying mechanism for this increase may be a decrease in conduction velocity, which has previously been shown to occur during fatiguing isometric-type contractions.[54–56] Our findings are similar to those of Nyland et al[35] and Wojtys et al.[59] Nyland and associates[35] used EMG to investigate the effect of lower extremity fatigue on ground reaction force production, lower extremity kinematics, and muscle activation during the landing phase of a run-and-rapid-stop maneuver. Employing a controversial and nontraditional probability level of $P < .10$, the authors concluded that, in the fatigued state, the onset of muscle activation of the quadriceps and hamstring muscles tended to occur later than was demonstrated in the unfatigued condition.[35] Wojtys et al[59] also demonstrated significant slowing of gastrocnemius muscle activity and quadriceps and hamstring muscle group activity in response to an anterior tibial translation force after fatigue.

The increase in contraction area of the vastus medialis and vastus lateralis demonstrated by our subjects, as a result of muscular fatigue, reflects the findings of investigations of the EMG characteristics of muscular fatigue. It has previously been established that, compared with resting muscle, fatigued muscle changes the EMG signal.[54–58] Muscular fatigue appears to result in a reduction in membrane conduction velocity, while amplitude remains constant. This decrease in conduction increases the width of the muscle signal and, therefore, increases the area under the muscle signal curve. Mathematically, this is interpreted as an increase in the mean area of the muscle contraction. Interestingly, our study also revealed that the increase in contraction area of the vastus lateralis in response to muscular fatigue was significantly greater for the male athletes compared with the female athletes. Although the underlying physiologic explanation for this difference in response to muscular fatigue is unknown, it does suggest that muscular fatigue affects female and male athletes differently.

Additionally, this significant difference encourages further investigations to elicit not only the physiologic mechanism responsible for this sex difference but also the clinical implications as they relate to ACL injury.

CONCLUSIONS

We conducted this study to investigate the effects of muscular fatigue on knee joint laxity and the neuromuscular characteristics of male and female basketball and soccer players. Moreover, we were particularly interested in determining whether such effects would be more pronounced in female athletes than in males participating in these sports. When subjected to muscular fatigue, both males and females demonstrated alterations in muscular activity and decrements in proprioceptive ability, without an increase in joint laxity. Therefore, muscular fatigue appears to affect knee joint proprioception in addition to preactivated, or compensatory, muscle firing patterns and, thus, may predispose both sexes of athletes to an increased risk for ligamentous injury.

Future research should continue to focus on inherent and postfatigue characteristics of male and female athletes participating in sports where a disproportionate number of ACL injuries are occurring. In addition to joint laxity and neuromuscular aberrations, other suggested causative factors, such as the influence of hormonal changes on ligamentous tissue properties and the reaction rate of the dynamic stabilizers after knee joint perturbation, should be investigated. Furthermore, future research should investigate knee joint laxity, neuromuscular responses, and functional ability subsequent to fatigue protocols that subject the knee to sport-specific forces.

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