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Hamstring-Dominant Strategy of the Bone–Patellar Tendon–Bone Graft Anterior Cruciate Ligament–Reconstructed Leg Versus Quadriceps-Dominant Strategy of the Contralateral Intact Leg During High-Intensity Exercise in Male Athletes

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Hamstring-dominant strategy of the bone-patellar tendon-bone graft ACL reconstructed leg vs. quadriceps-dominant strategy of the contra-lateral intact leg during high intensity exercise in male athletes

ABSTRACT

Purpose: The purpose of the present study was to investigate the effect of the ACL reconstruction on the quadriceps-dominant strategy as a parameter associated to the neuromuscular control of the knee joint.

Methods: Fourteen ACL reconstructed competitive soccer players with bone-patella tendon-bone autograft and fourteen healthy competitive soccer players performed two 10-min treadmill runs, one at a moderate and one at a high intensity. Electromyographic recordings were acquired using a telemetric system at the 3rd, 5th, 7th, and 10th minute of the runs from the vastus lateralis and the biceps femoris bilaterally. The dependent variable examined was the peak EMG amplitude during the stance phase. ANOVAs were used to examine significant main effects and interactions.

Results: Vastus lateralis electromyographic activity during high intensity running increased for both the control and intact leg ($F=4.48$, $p<0.01$) while it remained unchanged for the reconstructed leg ($p>0.05$). Biceps femoris electromyographic activity during high intensity running increased for the reconstructed leg only compared to both the control ($F=3.03$, $p<0.05$) and intact leg ($F=3.36$, $p<0.03$).

Conclusions: There is no presence of a quadriceps-dominant strategy in ACL reconstructed athletes during moderate intensity exercise. During high intensity exercise, the intact contra-lateral leg develops a quadriceps-dominant strategy, whereas the reconstructed leg does not. The reconstructed leg increases instead biceps femoris activity developing a “hamstring-dominant” strategy and this “asymmetry” may theoretically be in favor of the reconstructed knee.

Level of Evidence: III, retrospective comparative study of two groups

INTRODUCTION

After anterior cruciate ligament (ACL) reconstruction several alterations at the neuromuscular control of the knee joint may develop including selective muscle fiber atrophy in the involved quadriceps [1, 2], altered motor unit activation following surgery and subsequent retraining [3] and loss of joint afferent information which may lead to suboptimal muscle fiber activation [4]. These neuromuscular response perturbations of ACL-reconstructed knees may affect the amount of stress that is applied on the ACL graft postoperatively due to selective muscle activation, and thus may have important implications for the graft integrity. Moreover, since relevant neuromuscular control strategies have been previously considered as potential risk factors for native ACL injury [5, 6, 7] they most likely have dual interest in the case of an ACL-reconstructed individual, namely their reconstructed and contralateral intact knee.

Even if pure muscular response is measured, the combined recordings from both anterior and posterior thigh muscles activity may provide important information for the amount of stress that is applied to the ACL (either native ACL or a graft substitute). For instance, the quadriceps-to-hamstring ratio has been considered a parameter associated to neuromuscular control that can affect the ACL integrity [7]. Since exercise intensity has been related to the muscle activity [8, 9, 10] it would be reasonable to observe such neuromuscular response parameters with reference to the exercise intensity. During moderate intensity activities such as walking and jogging, ACL reconstruction re-establishes the extensor electromyographic (EMG) activity of the operated leg towards normative values [11, 12, 13, 14]. On the contrary, no relevant information exists regarding high intensity exercise of ACL-reconstructed individuals. High intensity exercise represents a particular condition where metabolic fatigue is accumulated and special neuromuscular demands evolve. The quadriceps-dominant strategy that has been described for healthy subjects while performing high intensity exercise consists of an increase in agonist (extensor) EMG activity without a concomitant increase in antagonist (flexor) EMG activity [8, 15, 16]. This response is considered to represent an optimization strategy to compensate for the deleterious effects of fatigue on joint neuromuscular control [8, 15, 16].

However, the literature lacks of information about the neuromuscular response of the ACL-reconstructed leg during high intensity exercise. In addition, it is unknown what the neuromuscular response behavior of the intact contra-lateral knee of an individual with unilateral ACL reconstruction is during high intensity activities.

The purpose of the present study was to investigate the effect of the ACL reconstruction on the quadriceps-dominant strategy as a parameter associated to the neuromuscular control of the knee joint during moderate and high intensity exercise. We hypothesized that (a) during moderate intensity exercise there will be no evidence of quadriceps-dominant strategy for of any the control, intact and reconstructed leg, (b) during high intensity exercise the quadriceps-dominant strategy will be evident for the control and intact contra-lateral but not the reconstructed leg.

METHODS

Two groups of athletes participated in the study. The first group consisted of a consecutive series of fourteen ACL-reconstructed competitive male soccer players[mean (SD) age, body mass and height, 24.8 (5.3) years, 77.3 (7.5) kg and 177 (5.3) cm with ACL-reconstructed knees and the second group consisted of fourteen healthy competitive male soccer players who had never suffered any kind of orthopaedic or neurological condition [mean (SD) age, body mass and height, 21.7 (4.4) years, 72.2 (8.3) kg and 180 (9.0) cm]. The operated athletes had undergone ACL reconstruction with bone-patella tendon-bone (BPTB) autograft, on average 18.5 (SD 4.3) months before testing. ACL reconstruction was performed sub-acutely within 6 months after the injury from the same surgeon (range 1 to 4 months). All subjects had a unilateral ACL tear confirmed by MRI and arthroscopy.

All subjects underwent the same rehabilitation protocol, starting from the first post operative day with the use of passive exercises. Return to sports was permitted 6 months after reconstruction provided that the athletes had regained stability and full functional strength, according to the following criteria [17]: (1) Full range of motion, (2) KT-1000 side-to side difference <3mm, (3) quadriceps strength >85% compared to the contralateral side, (4) hamstrings strength 100% compared to the contralateral side, (5) hamstrings-to-quadriceps strength ratio >70% and (6) functional testing >85% compared to the contralateral side. Their strength was determined with the BIODEX System-3 isokinetic dynamometer (Biodex Corp., Shirley, NY, USA), revealing acceptable symmetry in quadriceps and hamstrings strength, as well as acceptable hamstring-to-quadriceps-ratio. All subjects agreed with the testing protocol and gave their consent to participate in accordance with the Institutional Review Board policies of our Medical School.

Prior to any data collection, a clinical evaluation was performed on all subjects by the same clinician. During this evaluation, the Tegner and Lysholm scores were obtained, while anterior tibial translation was evaluated using the KT-1000 arthrometer (MEDmetric Corp., San Diego, California) [18]. These measurements were performed using 134N posterior-anterior external force at the tibia, as well as maximum posterior-anterior external force until heel clearance. Repeated anterior tractions were performed until a constant reading on the dial was registered.

The athletes reported to the laboratory on three different occasions, separated by 48 hours, within a two weeks period. For their first visit to the laboratory, athletes performed an incremental treadmill (Technogym Runrace 1200, Italy) running test to volitional exhaustion with 3 minute-stages, to determine maximal aerobic power (VO_2max) and lactate threshold (LT) [19]. A computerized system was used for all metabolic measurements (CPX Ultima, Medical Graphics, St Maul, MN, USA). At the end of each stage, capillary blood samples were collected and analyzed for lactate (Accutrend, Roche Diagnostics, Germany). Prior to each test, all analyzers were calibrated according to the manufacturer instructions. Attainment of VO_2max was verified according to criteria established by the American College of Sports Medicine [18]. Lactate threshold was determined according to Cheng et al [20]. The high intensity running was set at ~85-88 of VO_2max (HI) and the moderate intensity running was set at ~80% of the lactate threshold (MOD) [21].

In each of the two subsequent visits to the laboratory, athletes were required to perform a 10-minute run at the pre-selected intensities. We only tested one intensity at each visit and the test order was randomly assigned for every athlete. During running, EMG data were collected for 15 seconds at the 3rd, 5th, 7th and 10th minute. Gas exchange data were recorded simultaneously breath-by-breath, heart rate was measured throughout the test and blood lactate was measured prior to running and immediately after termination of exercise.
EMG traces were obtained from the vastus lateralis (VL) and biceps femoris (BF) muscles bilaterally using bipolar, circular, pre-amplified, pre-geld Ag/AgCl electrodes with 10 mm diameter and fixed inter-electrode spacing of 20 mm (Noraxon Inc, Scottsdale, AZ, USA).

EMG data were recorded with a wireless 8-channel EMG system (Telemyo 2400T, Noraxon Inc, Scottsdale, AZ, USA) and displayed real-time on a personal computer using dedicated software (MyoResearchXP, Noraxon Inc, Scottsdale, AZ, USA). The surface of the skin was prepared by shaving hair, rubbing it with abrasive paper and cleaning it with alcohol. The electrodes were fixed longitudinally over the muscle belly. For the VL the electrodes were placed at the antero-lateral muscle bulge at 2/3 of the proximo-distal thigh length, while for the BF the electrodes were placed at the dorso-lateral side of the thigh at 1/2 of the proximo-distal thigh length [22, 23]. The visually largest area of muscle belly was selected using a contraction against manual resistance. The ground electrode was placed on lateral femoral condyle of the right leg. Electrodes and cables were secured with surgical tape, in order to avoid any interference with the running pattern of the subjects.

Footswitches (Noraxon Inc, Scottsdale, AZ, USA) placed under the heel and big toes of both legs were used to denote heel-strike and toe-off. Prior to the running, subjects performed a "zero offset" function to establish a zero baseline for each of the EMG channels. EMG was acquired at a sampling rate of 1500 Hz. The raw EMG was measured in a band of 10 to 500 Hz, was full-wave rectified, was high pass filtered (cut-off frequency at 20 Hz) with an 8th order Butterworth filter to remove movement artifacts and was smoothed with a 100 ms RMS algorithm. Values from 20 strides were averaged to calculate the mean peak amplitude during stance for each of the four time intervals (FIGURE 1). The stance period was selected for analysis because the ACL is stressed maximally during this portion of the gait cycle [24].

Statistical analysis

Based on our hypotheses, the dependent variable examined in the present study was the peak EMG amplitude during the stance phase. A 2-way fully repeated ANOVA within the control group, with time (four levels) and leg (two levels) as within-subjects factors, revealed no time*leg interactions for the EMG amplitude for either the moderate or high intensity running (data not shown). Thus, the left leg was selected as the control leg.

We compared the control with the intact contra-lateral and with the reconstructed leg using a 3-way mixed ANOVA with muscle (two levels) and time (four levels) as within-subjects and groups (two levels) as between-subjects factors. Finally we compared the intact and reconstructed leg using a 3-way fully repeated ANOVA with muscle (two levels), time (four levels) and leg (two levels) as within-subjects factors. Significant main effects and interactions were investigated with a Fisher least significant differences post hoc test. The level of significance was set at a=0.05.

RESULTS

Clinical results: At the time of data collection no clinical evidence of knee pain and effusion was found in the ACL-reconstructed subjects. All subjects in the ACL-reconstructed group were satisfied with the outcome of the surgery and resumed their pre-injury level of sports participation. Negative Lachman and pivot-shift tests indicated that the knee joint stability was regained clinically for all ACL-reconstructed subjects. For the subjects with ACL reconstruction, the median Lysholm score was 95 (range 94-100) and the Tegner score was 8 (range 7-9) at the time of examination. KT-1000 results revealed that the mean difference between the anterior tibial translation of the reconstructed and intact contra-lateral sides was 1.6 mm (range 1 to 2 mm) for the 134N test and 1.8 mm (range 1-2 mm) for the maximum manual test, respectively.

Physiological results: Moderate intensity running was performed at an average intensity 63.9% (4.1) and 64.2% (4.6) of their predetermined VO_2max for the control and reconstructed group respectively. Pre-exercise blood lactate values were 2.1 (0.3) and 2.1 (0.2) mM and post-exercise blood lactate values averaged 2.3 (0.3) and 2.4 (0.6) mM for the control and reconstructed group respectively. High intensity running was performed at an average intensity 88.7% (3.1) and 87.6% (4.4) of their predetermined VO_2max for the control and reconstructed group respectively. Pre-exercise blood lactate values were 2.1 (0.3) and 2.1 (0.3) mM and post-exercise blood lactate values averaged 7.9 (1.6) and 7.6 (1.7) mM for the control and reconstructed group respectively.

Electromyographic results: During moderate intensity exercise there was a main effect of muscle since significantly higher activity was found for VL compared to BF ($p<0.05$). However this result will not be considered further since the EMG data were not normalized (see below in Discussion section). There was not any other significant main effect or interaction. EMG amplitude remained unchanged for all legs for both the VL and BF.

INSERT TABLE 1 ABOUT HERE

During high intensity exercise there was a main effect for muscle since significantly higher EMG activity was found for VL compared to BF ($p<0.05$). However this result will also not be considered further since the EMG data were not normalized. When comparing the control and intact contra-lateral leg during high intensity exercise, we found a main effect for time since EMG activity increased from the 3rd to 10th of exercise for both legs ($F=10.89$, $p<0.001$, power=0.99) and a muscle* time interaction since EMG activity increased in time for the VL but not for the BF ($F=4.48$, $p<0.01$, power=0.87). Furthermore when comparing the intact contra-lateral with the reconstructed leg, we found a main effect for time ($F=7.96$, $p<0.001$, power=0.98) since EMG activity increased between the 3rd and 10th minute of exercise and a muscle*time*leg interaction ($F=3.36$, $p<0.05$, power=0.72) since EMG activity increased between the 3rd and 10th minute for the VL of the intact leg while remained unchanged for the

VL of the reconstructed leg and increased between the 5th and 10th minute for the BF of the reconstructed leg while remained unchanged for the BF of the intact contra-lateral leg. Finally when comparing the control with the reconstructed leg, we found a main effect for time (F=6.79, $p<0.001$, power=0.97) since EMG activity increased between the 3rd and 10th minute of exercise and a muscle*time*groups interaction (F=3.03, $p<0.05$, power=0.70) since EMG activity increased between the 3rd and 10th minute for the VL of the control group while remained unchanged for the VL of the reconstructed group and increased between the 5th and 10th minute for the BF of the reconstructed group while remained unchanged for the BF of the control group.

INSERT TABLE 2 ABOUT HERE

DISCUSSION

The purpose of the present study was to investigate the effect of ACL reconstruction on the quadriceps-dominant strategy during moderate and high intensity running. We hypothesized that (a) during moderate intensity exercise there will be no evidence of quadriceps-dominant strategy for any of the control, intact contra-lateral and ACL-reconstructed leg, (b) during high intensity exercise the quadriceps-dominant strategy will be evident for the control and intact contra-lateral but not the ACL-reconstructed leg.

The first hypothesis was confirmed by our results. During 10 minutes of moderate intensity running, the EMG amplitude of VL and BF remained unchanged with time for the control, intact contra-lateral and reconstructed leg (TABLE 1). The second hypothesis was also verified by our results. VL EMG activity increased for the control and intact contra-lateral but not the reconstructed leg. Furthermore BF EMG activity showed an opposite trend and increased for the reconstructed and not for the control or intact leg (TABLE 2). Collectively we observed that during high intensity exercise the development of the quadriceps-dominant strategy is evident for the control and intact contra-lateral but not the reconstructed leg.

Our results are in agreement with previous studies indicating that in individuals performing moderate intensity exercise, EMG amplitude of the exercising muscles remains unchanged with time [9, 10, 25, 26, 27]. Thus, our results verify that under low demand activities such as moderate intensity running there is no presence of a quadriceps-dominant strategy in either control or ACL- reconstructed athletes.

Regarding high intensity activities, previous studies indicate that fatiguing exercise is associated with increased activation of the agonist muscles [8, 9, 10, 15, 16, 26, 27]. Concomitant with the increased agonist muscle activity there has been demonstrated unaltered antagonist muscle activity [8, 15, 16]. This preferential increase in agonist activity during high intensity exercise has been characterized as quadriceps-dominant strategy and is considered to reflect the physiological response to the accumulation of metabolic fatigue [9, 10, 26, 27, 28] as well as a biomechanical consequence that is associated with better neuromuscular control of the joint during fatigue [8, 15, 16]. Our results are in agreement with the development of quadriceps-dominant strategy during fatiguing exercise showing that both the control knee of uninjured athletes, as well as the intact contra-lateral knee of ACL-reconstructed athletes, exhibit an increased EMG activity of the vastus lateralis muscle concomitantly with unaltered biceps femoris activity. Thus, our results demonstrate that the intact contra-lateral knee of an ACL-reconstructed patient shows the exact same neuromuscular response with a “normal” knee during high intensity exercise. This suggests that no compensations are observed on the intact contra-lateral side during high intensity exercise regarding the response to accumulating fatigue.

Our results further demonstrated that the ACL-reconstructed leg deviated from the normal quadriceps-dominant strategy showing no increase in VL activity coupled with increased BF activity. This deviation from the quadriceps-dominant strategy pattern that was noted for the control knees may reflect a “protective” mechanism in the case of ACL-reconstructed knees, where the quadriceps-dominant strategy has been replaced by a “hamstrings-dominant” strategy. This modification in the knee musculature activity during high intensity exercise, leads to a decreased anterior stress applied to the ACL graft as compared to the corresponding situation observed for control knees.

Our results may offer a reasonable explanation in certain cases of ACL re-injury after a unilateral ACL reconstruction. We believe that the quadriceps dominant strategy per se can not be responsible for contra-lateral injury since this strategy is the “normal” condition. Reconstructed subjects are more prone to (re)-injury compared to controls because their previous injury [29]. However within the reconstructed group there is a neuromuscular “asymmetry” with one leg demonstrating quadriceps dominant-strategy and the other one a more “knee-protective” hamstring-dominant strategy. Thus the intact contra-lateral leg has theoretically greater risk for injury compared to the operated leg. This neuromuscular “asymmetry” may offer a possible explanation in the case of contra-lateral injuries but in cases of re-ruptures/graft failures other mechanisms must be considered. Although there is controversy in the literature regarding the exact incidence rates for contralateral ACL rupture and for re-rupture/graft failure after a unilateral ACL reconstruction [30, 31, 32, 33], these situations are both significant issues, especially for young athletic and active population after the index operation. In addition, since previous studies have noted that the incidence of injury to the contralateral intact knee after unilateral ACL reconstruction is associated with higher activity level [30, 31, 33], our results may have special clinical value, by offering a potential explanatory mechanism for such injuries at high intensity exercise.

Several explanations can be offered for the absence of the quadriceps-dominant strategy at the ACL-reconstructed knee. These include selective muscle fiber atrophy in the involved quadriceps [1, 2], altered motor unit activation following surgery and subsequent retraining [3] and loss of joint afferent information which may lead to suboptimal muscle fiber activation [4]. These neuromuscular alterations following ACL reconstruction may be responsible for the unaltered agonist EMG activity during high intensity exercise. Regarding antagonist EMG activity, increased BF EMG activity has been shown in ACL deficient subjects during low demand activity, such as walking and jogging [12, 13, 34, 35], but surgical reconstruction seems to re-establish biceps femoris activity towards normative values under non-fatiguing activities [12, 13, 14]. This was also verified in the present study showing unaltered BF activity during the moderate intensity running. Thus the increased BF activity following ACL reconstruction is only evident during high intensity fatiguing exercise.

The reason for the increased antagonist EMG activity in the reconstructed leg is not clear from the present study. Interestingly the activation ratio in the reconstructed leg has shifted towards the antagonist (biceps femoris) and this may “mimic” the quadriceps avoidance gait pattern seen in ACL deficient subjects [36, 37]. Furthermore the reciprocal activation pattern seen in the control and intact contra-lateral leg [38] is no longer present and this may favour increased antagonist activity.

To the best of our knowledge this is the first study that investigated EMG activation patterns during intense exercise in ACL reconstructed athletes. Previous studies on ACL reconstructed athletes have compared EMG levels under moderate intensity activities and no study has investigated EMG activity with time during high intensity activities [11, 13, 14]. Our approach enabled us to extend our findings to intense running which represents a highly functional activity for the ACL reconstructed athlete. Furthermore ACL injuries and re-injuries are common during high intensity exercise [39, 40] and thus low demand activities such as walking or light jogging may have limited value regarding the efficiency of the neuromuscular function following ACL reconstruction. Strength of this study was that by monitoring cardiorespiratory data we were able to assign the subjects exercised at a comparable level. Pre- and post- exercise measurements of blood lactate demonstrated that lactate was not significantly elevated, further demonstrating the mild physiological strain imposed on the subjects during the moderate intensity exercise. Similarly, our cardiorespiratory data indicated that both our groups exercised at a comparable high fraction of their VO_2max during high intensity exercise bouts. Blood lactate values increased from baseline (~2mM) to a similar high level (~7-8mM) indicating the accumulation of significant metabolic fatigue. Thus we are confident that similar levels of fatigue occurred in our groups and that the presence of the quadriceps-dominant strategy is a consequence of fatigue accumulation.

Our study has some limitations. Our sample consisted of male patients with BPTB graft which does not allow for generalization of our findings to female patients. Also, since no data for the ACL-deficient knee were collected it is not clear whether the data observed in the ACL-reconstructed knee is secondary change after surgery or preexisting abnormality caused by ACL deficiency. It should also be acknowledged that EMG recordings should be performed with great care and the results should be interpreted with caution during dynamic muscle contractions. Signal capturing, recording and processing was performed according to established guidelines [22, 23, 41]. We examined EMG activity developed solely during the stance period, thereby reducing to some extent the role of the signal non-stationarities with respect to other effects being studied [41]. Furthermore, the activity of many (successive) steps was averaged providing a reasonable estimation of peak EMG amplitude. Normalisation of EMG data (for example to maximum voluntary contraction) was not performed due to the additional error introduced by this process and the fact that our study design involved repeated measures, thereby overcoming influences of electrode positioning and inter-electroded distance on the signal value [42]. We assumed that because the same instrumentation was use for all subjects, the level of measurement noise would be consistent for all subjects and that any differences could be attributed to changes within the system itself. Finally our dependent variable was examined across a “control” condition (moderate exercise) as well as two “control” legs (control and intact contra-lateral).

Conclusions:

There is no presence of a quadriceps-dominant strategy in the ACL reconstructed athletes during moderate intensity exercise. During high intensity exercise, the intact contra-lateral leg develops a quadriceps-dominant strategy, whereas the ACL-reconstructed leg does not. The reconstructed leg increases instead biceps femoris activity, developing a “hamstring-dominant” strategy and this “asymmetry” may theoretically be in favor of the reconstructed knee.

REFERENCES

1. McHugh MP, Tyler TF, Nicholas SJ, et al. Electromyographic analysis of quadriceps fatigue after anterior cruciate ligament reconstruction. *J Orthop Sports Phys Ther* 2001;31:25-32.
2. Snyder-Mackler L, Binder-Macleod SA, Williams PR. Fatigability of human quadriceps femoris muscle following anterior cruciate ligament reconstruction. *Med Sci Sports Exerc* 1993;25:783-789.
3. Drechsler WI, Cramp MC, Scott OM. Changes in muscle strength and EMG median frequency after anterior cruciate ligament reconstruction. *Eur J Appl Physiol* 2006;98:613–623.
4. Konishi Y, Fukubayashi T, Takeshita D. Mechanism of quadriceps femoris muscle weakness in patients with anterior cruciate ligament reconstruction. *Scand J Med Sci Sports* 2002;12:371–375.
5. Eiling E, Bryant AL, Petersen W, Murphy A, Hohmann E. Effects of menstrual-cycle hormone fluctuations on musculotendinous stiffness and knee joint laxity. *Knee Surg Sports Traumatol Arthrosc* 2007;15:126–132
6. Friden T, Roberts D, Ageberg E, Walden M, Zatterstrom R. Review of knee proprioception and the relation to extremity function after an anterior cruciate ligament rupture. *J Orthop Sports Phys Ther* 2001;31:567–576
7. Ahmad CS, Clark AM, Heilmann N, Schoeb JS, Gardner TR, Levine WN. Effect of gender and maturity on quadriceps to-hamstring strength ratio and anterior cruciate ligament laxity. *Am J Sports Med* 2006;34:370–374

8. Kellis E, Liassou C. The effect of selective muscle fatigue on sagittal lower limb kinematics and muscle activity during level running. *J Orthop Sports Phys Ther* 2009;39:210-20.

9. Sabapathy S, Schneider DA, Morris NR. The VO_2 slow component: relationship between plasma ammonia and EMG activity. *Med Sci Sports Exerc* 2005;37:1502-1509.

10. Shinohara M, Moritani T. Increase in neuromuscular activity and oxygen uptake during heavy exercise. *Ann Physiol Antropol* 1992;11:257-262.

11. Bulgheroni P, Bulgheroni MV, Andrini L, et al. Gait patterns after anterior cruciate ligament reconstruction. *Knee Surg Sports Traumatol Arthrosc* 1997;5:14-21.

12. Ciccotti MG, Kerlan RK, Perry J, et al. An electromyographic analysis of the knee during functional activities. II. The anterior cruciate ligament-deficient and -reconstructed profiles. *Am J Sports Med* 1994;22:651-658.

13. Knoll Z, Kiss RM, Kocsis L. Gait adaptation in ACL deficient subjects before and after anterior ctuciate ligament reconstruction surgery. *J Electromyogr Kinesiol* 2004;14:287-294.

14. Lewek M, Rudolph K, Axe M, et al. The effect of insufficient quadriceps strength on gait after anterior cruciate ligament reconstruction. *Clin Biomech* 2002;17:56-63.

15. Kellis E, Zafeiridis A, Amiridis IG. Muscle coactivation before and after the impact phase of running following isokinetic fatigue. *J Athl Train* 2011;46:11-19.

16. Padua DA, Arnold BL, Perrin DH, Gansneder BM, Carcia CR, Granata KP. Fatigue, vertical leg stiffness, and stiffness control strategies in males and females. *J Athl Train* 2006;41:294-304.

17. DeLee J, Drez D, Miller M. *DeLee & Drez's orthopaedic sports medicine: principles and clinical practise.* Philadelphia, PA: Saunders, 2003.

18. Daniel DM, Malcom LL, Losse G, et al. Instrumented measurement of anterior laxity of the knee. *J Bone Joint Surg Am* 1985;67:720–726.

19. Whaley MH, Brubaker PH, Otto RM. *ACSM's guidelines for exercise testing and prescription*. Philadelphia, PA: Lippincott Williams & Wilkins, 2005.

20. Cheng B, Kuipers A, Snyder AC, et al. A new approach for the determination of ventilatory and lactate thresholds. *Int J Sports Med* 1992;13:518–522.

21. Carter H, Pringle JSM, Jones AM, et al. Oxygen uptake kinetics during treadmill running across exercise intensity domains. *Eur J Appl Physiol* 2002;86:347–354.

22. Freriks B, Hermens H, Disselhorst-Klug C, et al. The recommendations for sensors and sensor placement procedures for surface electromyography. In: H.J. Hermens, Eds. *European recommendations for surface electromyography*. Enschede: Roessingh Research and Development, 1999.

23. Merletti R, Hermens HJ. Detection and conditioning of the surface EMG signal. In: R. Merletti and P. Parker, Eds, *Electromyography: physiology, engineering and non-invasive applications*, Wiley-IEEE, New Jersey, 2004.

24. Zhang LQ, Minorik JM, Lin F, Koh JL, Makhsous M, Bai Z. ACL strain during simulated free-speed walking. In: *Proceedings of the 25th annual meeting of the American Society of Biomechanics*. San Diego, 2001.

25. Ciccotti MG, Kerlan RK, Perry J, et al. An electromyographic analysis of the knee during functional activities. II. The anterior cruciate ligament-deficient and -reconstructed profiles. *Am J Sports Med* 1994;22:651-658.

26. Jones AM, Pringle JSM, Carter H. Influence of muscle fiber type and motor unit recruitment on VO_2 kinetics. In: A.M. Jones and D.C. Poole, Eds, *Oxygen uptake kinetics in sport, exercise and medicine*, Routledge, London, 2005.

27. Saunders MJ, Evans EM, Arngrimsson SA, et al. Muscle activation and the slow component rise in oxygen uptake during cycling. *Med Sci Sports Exerc* 2000;32:2040-2045.

28. Gaesser GA, Poole DC. The slow component of oxygen uptake kinetics in humans. *Exerc Sport Sci Rev* 1996;24:35–71.

29. Waldén M, Hägglund M, Ekstrand J. High risk of new knee injury in elite footballers with previous anterior cruciate ligament injury. Br J Sports Med. 2006 Feb;40(2):158-62

30. Salmon L, Russell V, Musgrove T, Pinczewski L, Refshauge K. Incidence and risk factors for graft rupture and contralateral rupture after anterior cruciate ligament reconstruction. *Arthroscopy* 2005;21:948–957

31. Shelbourne KD, Gray T, Haro M. Incidence of Subsequent Injury to Either Knee Within 5 Years After Anterior Cruciate Ligament Reconstruction With Patellar Tendon Autograft. *Am J Sports Med* 2009;37:246-251

32. Wright RW, Dunn WR, Amendola A, et. al. Risk of Tearing the Intact Anterior Cruciate Ligament in the Contralateral Knee and Rupturing the Anterior Cruciate Ligament Graft During the First 2 Years After Anterior Cruciate Ligament Reconstruction : A Prospective MOON Cohort Study. *Am J Sports Med* 2007;35:1131-1134

33. Sward P, Kostogiannis I, Roos H. Risk factors for a contralateral anterior cruciate ligament injury. *Knee Surg Sports Traumatol Arthrosc* 2010;18:277–291.

34. Kalund S, Sinkjaer T, Arendt-Nielsen L, et al. Altered timing of hamstring muscle action in anterior cruciate ligament deficient patients. *Am J Sports Med* 1990;18:245-248.

35. Limbird TJ, Shiavi R, Frazer M, et al. EMG profiles of knee joint musculature during walking: changes induced by anterior cruciate ligament deficiency. *J Orthop Res* 1988;6:630-638.

36. Beard DJ, Soundarapandian RS, O' Connor JJ, et al. Gait and electromyographic analysis of anterior cruciate ligament deficient subjects. *Gait Posture* 1996; 4:83–88.

37. Berchuck M, Andriacchi TP, Bach BR, et al. Gait adaptations by patients who have a deficient anterior cruciate ligament. *J Bone Joint Surg Am.* 1990;72:871–877.

38. De Luca CJ, Mambrito B. Voluntary control of motor units in human agonist and antagonist muscles: coactivation and reciprocal activation. *J Neurophysiol* 1987;58:525-542.

39. Hawkins RD, Hulse MA, Wilkinson C, et al. The association football medical research programme: an audit of injuries in professional football. *Br J Sports Med* 2001;35:43-47.

40. Alentorn-Geli E, Myer GD, Silvers HJ, et al. Prevention of non-contact anterior cruciate ligament injuries in soccer players. Part 1: Mechanisms of injury and underlying risk factors. *Knee Surg Sports Traumatol Arthrosc* 2009;17:705-29.

41. De Luca CJ. The use of surface electromyography in biomechanics. *J Appl Biomech* 1997;13:135–163.

42. Rahnama N, Lees A, Reilly T. Electromyography of selected lower-limb muscles fatigued by exercise at the intensity of soccer match-play. *J Electromyogr Kinesiol* 2006;16:257-263.

FIGURE LEGENDS

FIGURE 1: Bilateral recording for a representative ACL reconstructed subject during high intensity running. Vertical lines indicate right footswitch. The time between heel strike and toe-off corresponds to the stance phase.

TABLE 1. Main effects and interactions during moderate intensity exercise (n=14).

CON vs. REC				**CON vs. INT**				**INT vs. REC**			
	F ratio	**p value**	**power**		**F ratio**	**p value**	**power**		**F ratio**	**p value**	**power**
muscle	24.9	<0.001	0.99	**muscle**	26.64	<0.001	0.99	**muscle**	9.594	0.008	0.91
groups	1.24	0.276	0.46	**groups**	0.03	0.854	0.12	**leg**	1.39	0.286	0.33
time	1.71	0.171	0.29	**time**	0.15	0.926	0.09	**time**	0.133	0.94	0.12
muscle*groups	3.3	0.081	0.48	**muscle*groups**	2.57	0.121	0.22	**muscle*leg**	0.039	0.847	0.22
muscle*time	0.36	0.779	0.23	**muscle*time**	1.03	0.386	0.15	**muscle*time**	1.13	0.351	0.35
time*groups	0.7	0.554	0.16	**time*groups**	1.38	0.256	0.28	**leg*time**	0.87	0.467	0.19
muscle*time*groups	1.73	0.168	0.31	**muscle*time*groups**	0.88	0.455	0.17	**muscle*leg*time**	0.375	0.771	0.23

F ratios, p values and the corresponding power for every main effect and interaction during the moderate intensity exercise.

TABLE 2. Main effects and interactions during high intensity exercise (n=14).

CON vs. REC				**CON vs. INT**				**INT vs. REC**			
	F ratio	**p value**	**power**		**F ratio**	**p value**	**power**		**F ratio**	**p value**	**power**
muscle	13.641	<0.001	0.95	**muscle**	27.122	<0.001	0.99	**muscle**	6.26	0.026	0.64
groups	0.39	0.537	0.148	**groups**	0.02	0.879	0.07	**leg**	0.799	0.39	0.13
time	6.79	<0.001	0.97	**time**	10.89	<0.001	0.99	**time**	7.96	0.001	0.98
muscle*groups	3.81	0.062	0.47	**muscle*groups**	0.947	0.34	0.16	**muscle*leg**	1.131	0.31	0.17
muscle*time	0.63	0.6	0.176	**muscle*time**	4.48	0.006	0.87	**muscle*time**	0.189	0.903	0.08
time*groups	3.32	0.024	0.74	**time*groups**	1.44	0.239	0.37	**leg*time**	4.76	0.006	0.87
muscle*time*groups	3.03	0.034	0.70	**muscle*time*groups**	0.662	0.58	0.18	**muscle*leg*time**	3.36	0.028	0.72

F ratios, p values and the corresponding power for every main effect and interaction during the high intensity exercise.