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High intensity running results in an impaired neuromuscular response in ACL reconstructed individuals

Kostas Patras
University of Ioannina

Giorgos Ziogas
University of Ioannina

Stavros Ristanis
University of Ioannina

Elias Tsepis
Technological Educational Institution of Patras at Aigion

Nicholas Stergiou
University of Nebraska at Omaha, nstergiou@unomaha.edu

See next page for additional authors

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Authors

Kostas Patras, Giorgos Ziogas, Stavros Ristanis, Elias Tsepis, Nicholas Stergiou, and Anastasios D. Georgoulis

1 **HIGH INTENSITY RUNNING RESULTS IN AN IMPAIRED NEUROMUSCULAR**

2 **RESPONSE IN ACL RECONSTRUCTED INDIVIDUALS**

3 Kostas Patras[1], Giorgos Ziogas[1], Stavros Ristanis[1], Elias Tsepis[1,2], Nicholas Stergiou[1,3],

4 Anastasios D. Georgoulis[1]

5

6 [1]Orthopaedic Sports Medicine Center, Department of Orthopaedic Surgery, University of

7 Ioannina, Greece

8 [2]Physical Therapy School, Technological Educational Institution of Patra at Aigion, Greece

9 [3]Nebraska Biomechanics Core Facility, College of Education, University of Nebraska at

10 Omaha and College of Public Health, University of Nebraska Medical Center, USA

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12

13

14 **<u>Address for Correspondence:</u>**

15 Anastasios D. Georgoulis, M.D

16 Georgiou Papandreou 2

17 Ioannina 45444, GREECE

18 Tel/Fax: +302651064980

19 P.O BOX 1330

20 Email:oaki@cc.uoi.gr

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1   **ABSTRACT**

2   **Introduction:** Anterior cruciate ligament reconstruction reestablishes electromyographic

3   activity during moderate activities such as walking but is unclear if this is also the case in

4   sports activities such as heavy intensity running. **Methods:** Telemetric electromyographic

5   recordings from vastus lateralis and biceps femoris muscles were performed bilaterally in nine

6   bone-patella tendon-bone anterior cruciate ligament reconstructed athletes during two 10

7   minute treadmill runs, one at a heavy intensity and one at a moderate intensity. **Results:**

8   During the high intensity run, electromyographic activity increased significantly

9   [294.2(120.6) µV to 317.1(140.5) µV, p= 0.03] for the vastus lateralis of the intact leg while

10   did not change for the vastus lateralis of the anterior cruciate ligament reconstructed leg

11   [267.8(142.8) µV to 263.8(128.9) µV, p>0.05]. During the moderate intensity run

12   electromyographic activity did not change for either leg. **Conclusions:** High intensity exercise

13   results in an impaired neuromuscular response for the vastus lateralis muscle of the anterior

14   cruciate ligament reconstructed leg.

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16   Keywords: neuromuscular performance, EMG, fatigue, running

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INTRODUCTION

Anterior cruciate ligament (ACL) function is associated with coordination of the muscles surrounding the knee joint [1, 2]. As a result, rupture of the ACL leads to alterations in muscle recruitment including diminished electromyographic (EMG) activity of the quadriceps and the gastrocnemius and increased EMG activity of the biceps femoris during walking [3]. In addition, rupture of the ACL generates earlier EMG activity at the biceps femoris during uphill walking [4], reduced EMG activity of the vastus lateralis and the hamstrings during running and reduction in EMG activity of the biceps femoris and hamstrings after 10 minutes of walking [5].

It has been found that reconstruction of the ACL (ACLR) re-establishes EMG activity of the operated leg towards normative values during walking and jogging [6-9]. However, previous studies have demonstrated that the ACLR quadriceps muscles exhibit reduced ability to recruit high-threshold motor units due to reduced neural drive and/or selective type II muscle fibers hypotrophy [10-14]. Thus, although the reconstructed leg may exhibit similar EMG levels with the intact contralateral leg during low demand activities such as walking, it is unclear if ACL reconstruction may affect the behavior of EMG activity during the course of a sustained high intensity running, where the need to recruit high-threshold motor units is more apparent.

Previous studies in healthy individuals demonstrated that during high intensity exercise above the lactate threshold (the point at which lactate levels accumulate in blood), the working muscles increase their EMG activity as a physiological compensatory mechanism [15-19]. However, during moderate exercise performed below the lactate threshold, muscle EMG levels remain constant across time [15, 18]. The underlying mechanism of the increased EMG activity during high intensity exercise is considered to be the enhanced recruitment of high threshold motor units as exercise progresses [16-18]. In this context, ACLR subjects may

1 not increase their EMG activity of the involved muscles due to their above-mentioned

2 diminished ability to recruit high threshold motor units. This may have important clinical

3 implications for the ACLR athlete since a lack of increased EMG activation in the

4 reconstructed muscles during high intensity exercise may indicate premature muscle fatigue

5 and lead to decreased performance. It has been reported that the knee joint provides the major

6 energy-absorption function during the landing phase of single-leg landing tasks,[20] and such

7 diminished neuromuscular response of the surrounding muscles may increase the potential for

8 re-injury, especially during the latter parts of a game when a player is fatigued [21].

9   Therefore, the purpose of the present study was to investigate the effect of ACLR on the

10 muscle activation levels over time during running at two different intensities, a moderate and

11 a high intensity. The moderate intensity was defined as running at a speed corresponding to

12 80% of the intensity at the lactate threshold (80%LT). The high intensity was defined as

13 running at a speed corresponding to 40% above the intensity at the lactate threshold [22]. We

14 hypothesized that (a) in both the intact contralateral and ACLR legs the EMG activity will not

15 increase during ten minutes of running at moderate intensity, while (b) ten minutes of running

16 at high intensity will increase the EMG activity at the intact contralateral leg but not at the

17 ACLR.

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1   **METHODS**

2   **Subjects**

3   Nine amateur male soccer players [mean (SD) age, body weight and height, 27.7 (3.5)

4   years, 79.5 (7.3) kg and 178 (5.9) cm] were recruited for the present study. The athletes had

5   undergone ACL reconstruction with bone-patella tendon-bone (BPTB) graft, 19.2 (5.7)

6   months before testing. ACL reconstruction was performed sub-acutely within 6 months after

7   the injury. All subjects had a unilateral ACL tear confirmed by MRI and arthroscopy. All

8   patients underwent the same rehabilitation protocol, starting from the first post operative day

9   with the use of passive exercises. Return to sports was permitted 6 months after

10   reconstruction as it is recommended in the literature [23]. At the time of data collection no

11   clinical evidence of knee pain and effusion was found in the ACL reconstructed subjects. All

12   subjects had resumed their sports activities and agreed with the testing protocol by giving

13   their consent to participate in accordance with the Institutional Review Board policies of our

14   Medical School.

15

16   **Clinical evaluation**

17   Prior to any data collection, a clinical evaluation was performed in all subjects by the

18   same clinician. During this evaluation, the Tegner and Lysholm scores were obtained, while

19   anterior tibial translation was evaluated using the KT-1000 knee arthrometer (MEDmetric

20   Corp., San Diego, California) [24]. These measurements were performed using 134N

21   posterior-anterior external force at the tibia, as well as maximum posterior-anterior external

22   force until heel clearance. Repeated anterior tractions were performed until a constant reading

23   on the dial was registered. Negative Lachman and pivot shift tests indicated that the static

24   knee joint stability was regained. The median Lysholm score was 95 (94-96) and the Tegner

25   score was 8 (range, 7-9). KT-1000 results revealed that the mean difference between the

1 anterior tibial translation of the reconstructed and intact sides was 1.6 mm (range, 1 to 2mm)

2 for the 134-N test and 1.8 mm (range, 1 to 2 mm) for the maximal manual test.

3

4 **Torque measurements**

5 For all patients, torque measurements were performed on an isokinetic dynamometer

6 (Biodex, System-3, Biodex Medical Systems Inc., New York, USA). Approximately 45

7 minutes after termination of the VO_2max and lactate threshold test (at a time where blood

8 lactate levels had returned to baseline values), subjects where tested for maximum torque

9 output of the quadriceps and hamstrings. The subjects sat on the dynamometer chair and were

10 secured with body straps while the hip and knee joints were flexed at 90°. For a warm-up they

11 performed submaximal isokinetic concentric contractions by flexing and extending the knee

12 joint. During testing five maximal concentric reciprocal knee extensions-flexions were

13 performed at angular velocities of 60°/sec and 180°/sec with one minute rest interval between

14 velocities. Angular velocities within this range are typically used for strength testing of ACLR

15 subjects [27-30]. Peak torque was identified as the highest value during the five repetitions. A

16 strength index was calculated for each angular velocity as the % ratio of

17 reconstructed/contralateral peak torque [28]. This muscle torque testing was performed to

18 verify that there were no persistent gross deficits that may have influenced the neuromuscular

19 response of the subjects. We observed no such deficits.

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21 **Data collection**

22 Subjects reported to the laboratory having abstained from caffeine or food consumption

23 for 4 hours and without vigorous training for 24h. During warm-up the subjects performed 3

24 minutes walking at self-selected pace and 5 minutes jogging on a treadmill (Technogym

25 Runrace 1200, Italy) at a speed of 1.94 or 2.22 m⊙sec^{-1} where heart rate and lactate was

1   measured. Then, subjects performed an incremental exercise test with expired gas and heart

2   rate analysis (CPX Ultima Series, Medical Graphics, USA) to volitional exhaustion to

3   determine maximal oxygen uptake (VO_2max) and lactate threshold (LT). Prior to each test, all

4   analyzers were calibrated according to manufacturer instructions. The initial speed was set at

5   2.5 or 2.78 m⊙sec^{-1} depending on the subject fitness and was increased by 0.56 m⊙sec^{-1} every

6   3 minutes until volitional exhaustion. At the end of each stage, capillary blood samples were

7   collected and analyzed for lactate (Accutrend, Roche Diagnostics, Germany). Running was

8   recommenced within 20 sec. Attainment of VO_2max was verified according to criteria

9   established by the American College of Sports Medicine [25]. Lactate threshold was

10   determined according to the Dmax method proposed by Cheng et al [26]. Velocities for the

11   moderate and heavy intensity bout were determined using extrapolation from VO_2-velocity

12   individual plots. The moderate intensity was set at 80% of the intensity at the lactate threshold

13   and the heavy bout was set 40% above the intensity at the lactate threshold (40%D) [22].

14   In two subsequent visits to the laboratory, subjects were required to perform a 10-min

15   run, one at the moderate and one at the high intensity [FIGURE 1]. The two runs were

16   presented to the subjects in a random order. Prior to running, the subjects performed 3

17   minutes of walking at 1.39 m⊙sec^{-1} and 3 minutes of jogging at 1.81 m⊙sec^{-1} where baseline

18   EMG data where collected at the first 15 seconds of each minute. During running, EMG data

19   was collected for 15 seconds at the 3rd and 10th minute (last minute of the exercise test)

20   [FIGURE 2]. Gas exchange data was recorded simultaneously breath-by-breath. Heart rate

21   was measured throughout the test and blood lactate was measured at baseline (prior to

22   running, after the completion of walking and jogging), and immediately after termination of

23   exercise.

24   **INSERT FIGURE 1 ABOUT HERE**

25   **INSERT FIGURE 2 ABOUT HERE**

1   Surface electromyography was obtained from the vastus lateralis (VL) and biceps

2   femoris (BF) muscles bilaterally using bipolar, circular, pre-amplified, pre-geld Ag/AgCl

3   electrodes with 10 mm diameter and fixed inter-electrode spacing of 20 mm (Noraxon, USA).

4   VL was selected on the basis that is the primary force-producing muscle during running.

5   Furthermore the VL acts as a shock absorber during the first part of stance, thus protecting the

6   graft from high impact forces [31]. BF was chosen due to its role in preventing anterior tibial

7   translation and protecting the knee from the pivoting phenomenon [7]. EMG traces were

8   recorded with a wireless 8-channel EMG system (Telemyo 2400T, Noraxon, USA) and

9   displayed on-line on a personal computer using dedicated software (MyoResearchXP,

10   Noraxon, USA).

11   The surface of the skin was prepared by shaving the hair, rubbing the skin with abrasive

12   paper and cleaning the skin with alcohol. The electrodes were fixed longitudinally over the

13   muscle belly. For the VL the electrodes were placed at the antero-lateral muscle bulge at 2/3

14   of the proximo-distal thigh length. For the BF the electrodes were placed at the dorso-lateral

15   side of the thigh at 1/2 of the proximo-distal thigh length [32, 33]. The largest area of muscle

16   belly was identified using a contraction against manual resistance. The ground electrode was

17   placed on the lateral femoral condoyle of the right leg. Electrodes and cables were secured

18   with surgical tape to avoid any interference with the running pattern of the subjects.

19   Footswitches (Inline Foot Contact Sensor, Noraxon, USA) were placed under the heel

20   and big toe of both legs and were used to denote the events of the gait cycle (heel-strike and

21   toe-off). Prior to the bout, subjects performed a "zero offset" function to establish a zero

22   baseline from each of the EMG channels. EMG data was acquired at a sampling rate of 1500

23   Hz. The Root Mean Squared (RMS) amplitude was calculated for each muscle burst.

24   Specifically, the raw EMG data measured in a band of 10 to 500 Hz, was full-wave rectified,

25   high pass filtered with an 8th order Butterworth filter to remove movement artifacts (with a

1 cut-off frequency of 20 Hz), and smoothed with a 100 ms RMS algorithm. Only the stance

2 period of running was considered in the analysis and the highest value for the RMS amplitude

3 (peak amplitude) was recorded. Values from 15 strides were averaged to calculate the mean

4 peak amplitude during stance for each recording period. The stance period was selected for

5 analysis because the ACL is stressed maximally during this portion of the gait cycle [34].

6

7 **Statistical analysis**

8 Based on our hypotheses, the dependent variable examined was the mean peak EMG

9 amplitude during stance. First, the Kolmogogov-Smirnov test was calculated to ensure

10 normality of our datasets, Subsequently, our first hypothesis was tested using paired Student's

11 t-tests to compare the mean peak EMG amplitude between the 3rd minute of the moderate

12 intensity bout and the mean peak EMG amplitude during the 10th minute of the moderate

13 intensity bout. This test was performed for both the intact contralateral and ACLR legs.

14 Similarly, our second hypothesis was tested using paired Student's t-tests to compare the

15 mean peak EMG amplitude between the 3rd minute of the high intensity bout and the mean

16 peak EMG amplitude during the 10th minute of the high intensity bout. Again this test was

17 performed for both the intact contralateral and ACLR legs. The level of significance was set

18 at a=0.05. Paired Student's t-tests were also used to compare the VO_2 at minute 3 with the

19 VO2 at minute 10 and the blood lactate values at baseline with the blood lactate values at

20 minute 10.

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1 **RESULTS**

2 *Electromyographic and physiological responses during the moderate intensity test*

3   EMG amplitude of the VL did not increase for neither the intact nor the ACLR leg

4 (p=0.43; and, p=0.222, respectively] (Table 1). For the BF, the EMG amplitude also did not

5 increase for neither the intact nor the ACLR leg [p=0.637; and p=0.316, respectively]. The

6 paired t-test comparisons did not reveal significant differences for VO_2 and blood lactate

7 values between the 3rd and 10th minute (p=0.444 and p=0.161 respectively) verifying that this

8 exercise was indeed of moderate intensity (Table 2).

9 *Electromyographic and physiological responses during the high intensity test*

10   The EMG amplitude of the VL for the intact leg increased significantly (p= 0.03), while

11 for the ACLR leg remained unchanged (p=0.684) (Table 1). For the BF, the EMG amplitude

12 did not increase for neither the intact nor the reconstructed leg (p=0.325; and p=0.107,

13 respectively). The paired t-test comparisons revealed significant differences for VO_2 and

14 blood lactate values between the 3rd and 10th minute (p=0.026 and p<0.001, respectively)

15 verifying that this exercise was indeed of high intensity (Table 2).

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17 **INSERT TABLE 1 and 2 ABOUT HERE**
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1   **DISCUSSION**

2   The purpose of the present study was to investigate the effect of ACLR on the muscle

3   activation levels over time during running at two different intensities, a moderate and a high

4   intensity. In our study we recruited amateur competitive ACLR soccer players. Their physical

5   characteristics revealed that had good physical conditioning but were not highly trained. To

6   the best of our knowledge only one study has tested ACLR athletes during high intensity

7   running and their physical characteristics were very similar to those of the athletes in the

8   present study [35]. We hypothesized that (a) in both the intact contralateral and ACLR legs

9   the EMG activity will not increase during ten minutes of running at moderate intensity, while

10   (b) ten minutes of running at high intensity will increase the EMG activity at the intact

11   contralateral leg but not at the ACLR. EMG activity was measured bilaterally at the 3rd and

12   the 10th minute of the running tests for the VL and the BF muscles.

13   Regarding our first hypothesis, the EMG amplitude for the VL and BF muscles did not

14   increase for both the intact contralateral and ACLR leg. Therefore, the results supported our

15   hypothesis. Furthermore and during the moderate running test, VO_2 stabilized around the

16   value obtained at minute 3. Blood lactate also did not increase above the baseline values,

17   averaging 2.4 mM. These results verified the relatively mild physiological strain during the

18   moderate running test [36] [FIGURE 3a]. Previous studies utilizing cycling at intensities

19   similar to our moderate bout, have demonstrated that under moderate physiological strain,

20   EMG activity of the quadriceps muscles did not increase during the course of the exercise [15,

21   17, 18]. Furthermore, previous research indicates that during moderate intensity exercise, the

22   physiological strain imposed on the athlete is of similar profile to our data with no increase in

23   the VO_2 and the lactate values [17, 36]. Thus, we are confident that the lack of increase in

24   EMG during the moderate intensity running is a result of the low energetic requirements of

25   the task.

1   Regarding our second hypothesis the increased physiological strain imposed on the

2   subjects [FIGURE 3b] was accompanied with a significant (7.2%) increase in the EMG

3   activity of the VL muscle for the intact contralateral leg. No significant change was found in

4   the EMG activity of the VL muscle for the ACLR leg [Figure 4]. Pilot testing in healthy

5   subjects revealed similar increases in the EMG amplitude that were in the range of 6 to 8%.

6   The 7.2% in the ACLR group is within the expected range. Thus, the results supported our

7   second hypothesis. During the high intensity running test, VO_2 rose well above the values at

8   the 3rd minute. The blood lactate values at end-exercise averaged 7.2mM. These resulted

9   indicated great physiological strain [36]. These values also verified that the high intensity

10   exercise taxed significantly both the aerobic and anaerobic energetic pathways of the subjects

11   in approximating "field" situations. Furthermore, the continuous monitoring of the

12   physiological data allowed each subject to exercise at the same intensity. Previous studies that

13   examined the relationship between EMG activity and exercise intensity indicated that the

14   EMG progressively increases during constant exercise performed at high intensities [16-19].

15   The progressive increase in the EMG activity during the high intensity exercise reflects the

16   physiological response of the muscles to the accumulating fatigue [19, 37].

17   **INSERT FIGURE 3 ABOUT HERE**

18   **INSERT FIGURE 4 ABOUT HERE**

19   Several explanations can be given for the lack of increase in the EMG amplitude for

20   the VL of the reconstructed leg. These may include selective muscle fiber atrophy in the

21   involved quadriceps [10, 13, 14], altered motor unit activation following surgery and

22   subsequent retraining [11], and loss of joint afferent information which may lead to selective

23   muscle fiber hypothophy [12]. These neuromuscular alterations following ACL reconstruction

24   may have a negative impact on performance at high intensities where the need to activate high

25   threshold motor units is more apparent [16-18, 36, 37] and may, at least in part, be responsible

1   for the lack of increase in the EMG amplitude. Therefore, it appears that although

2   reconstruction of the ACL re-establishes EMG activity of the operated leg towards normative

3   values during walking and jogging [6-9], under high intensity running (that generates high

4   levels of fatigue) the involved leg has an altered response showing no increase in EMG

5   activity. In this context our EMG results are in accordance with recent studies suggesting that

6   decrements of functional and neuromuscular performance after ACL reconstruction are more

7   pronounced under fatiguing test conditions [35, 38]. Indeed, Augustsson et al reported that

8   although ACLR subjects had similar hopping performance in the involved and intact legs

9   under non-fatigued conditions, the performance decrements were more pronounced on the

10   involved side as compared to the intact side when the subjects were fatigued [38].

11   In the present study the BF muscle of the intact leg was not affected by fatigue,

12   showing no increase in the EMG activity during the high intensity running. Large variability

13   in the individual behaviour was evident for this muscle. Similarly, large variability in the

14   EMG activity of the BF has also been reported for an incremental running exercise, where the

15   EMG amplitude of the VL increased linearly with running velocity while the EMG activity of

16   the BF did not display any such relationship with the running velocity [39]. Therefore, it

17   seems that during the stance phase of running the energetic requirements remain relatively

18   stable for the BF.

19   Importantly and under the fatigued condition, the ACLR subjects demonstrated

20   "asymmetry" in terms of the EMG time-course between the ACLR and contralateral intact

21   leg. This neuromuscular discrepancy under high demand activities may potentially overload

22   the knee joint which has a major energy-absorption contribution during the landing phase of

23   single-leg landing tasks [20]. This may increase the possibility for re-injury, especially during

24   the latter parts of a sporting event when fatigue rapidly accumulates [21]. In this regard,

1   endurance training may delay fatigue occurrence and prevent the development of such

2   neuromuscular "asymmetry" in ACLR athletes.

3   In the present study only ACL reconstructed athletes with bone-patellar tendon-bone

4   autograft were recruited. Thus, it is unknown if a similar response pattern will be observed in

5   athletes with a different graft such as hamstrings. It should also be acknowledged that EMG

6   recordings should be performed with great care and the results should be interpreted with

7   caution when it comes to dynamic muscle contractions and especially whole body exercises

8   such as running. With that in mind, signal capturing, recording and processing was performed

9   according to established guidelines [32, 33]. We selected a fixed epoch for the period of

10   contraction in our study. Thus, we examined electrical activity developed solely during the

11   stance period, thereby reducing to some extent the role of the signal non-stationarities with

12   respect to other effects being studied [40]. Furthermore, the peak activity of many

13   (successive) steps was averaged providing a reasonable estimation of peak electrical activity

14   during every recording period and minimizing within subject variability. In addition our

15   study design involved repeated measures, that is, the value of the EMG activity of every

16   muscle was compared with its original value while the electrodes remained attached during

17   the whole task, thereby overcoming the between subject variability of EMG amplitude. It is

18   well accepted that EMG amplitude indicates the overall recruitment of motor units but does

19   not provide direct information about the recruitment pattern of type I and II muscle fibers.

20   However, in a recent study, McDonald et al have shown that the RMS amplitude is the most

21   reliable and sensitive EMG variable for a fatiguing cycling exercise at a power output similar

22   to our high intensity [19]. Finally, a limitation of the present study is the small sample size

23   utilized which may affect statistical decisions (Type I and II errors). However, the complexity

24   of the experiment with the long visits in the laboratory by each subject revealed problems

25   with respect to recruitment and affected our sample size. Despite these problems we feel

1   confident with our results since in all statistically significant decisions the effect sizes were

2   quite large.

3   In conclusion, the present study demonstrated that at the end of a 10-minute run at high

4   intensity, the VL muscle of the intact leg had significantly increased EMG activity by 7.2%

5   (presumably as a response to compensate for the induced metabolic fatigue), while the VL of

6   the reconstructed leg showed no such increase. It appears that under high physiological strain

7   that simulates metabolic fatigue as in "real" conditions, the operated and the intact leg

8   respond in different ways. Future studies should identify whether the lack of increased EMG

9   in the VL muscle of the operated leg is accompanied with other electromyographic alterations

10   (i.e. timing or pre-activity)

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1   **ACKNOWLEDGEMENTS**

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1   **TABLE LEGENDS**

2   **Table 1:** EMG amplitude (μV) for each muscle during the running tests. Values are given as

3   mean (SD). Asterisks (*) denotes significantly higher than VL EMG amplitude at 3rd min for

4   the intact leg.

5

6   **Table 2:** Metabolic variables during the moderate and high intensity running tests. Values are

7   mean (SD). LA refers to blood lactate values. Asterisks (*) denotes significantly higher than

8   VO_2 3rd minute for the heavy intensity bout, $p<0.001$. Cross (†) denotes significantly higher

9   than LA baseline for the heavy intensity bout, $p<0.0001$.

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1 **FIGURE LEGENDS**

2 **Figure 1:** Subject running on the treadmill during the high intensity test. Respiratory data

3 were measured breath-by-breath with simultaneous EMG recordings. The EMG electrodes

4 were secured with tape to reduce movement artifacts. The configuration was kept exactly the

5 same for the moderate and the high intensity running.

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7 **Figure 2:** Typical EMG bursts of activity for the vastus lateralis muscle of the involved leg at

8 the beginning (a) and at the end (b) of the high intensity running.

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10 **Figure 3:** (a) VO_2 response during moderate running at a velocity of 8 km/h (80%LT). Note

11 that after the 3rd minute of exercise, the VO_2 remains stable.

12 (b) VO_2 response during high intensity running at a velocity of 13.2 km/h (40%D). Note that

13 the VO_2 continues to rise beyond minute 3.

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15 **Figure 4**: EMG amplitude for the VL muscle. Values are mean (SD) for the ACLR and intact

16 leg during the 3rd and 10th min.

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1   Table 1: Mean values ± SD of the EMG amplitude (µV) for each muscle during the running

2   tests. (N=9). SD refers to one standard deviation. VL refers to vastus lateralis and BF refers to

3   biceps femoris.

	Moderate bout VL		Heavy bout VL		Moderate bout BF		Heavy bout BF	
	3rd min	10th min	3rd min	10th min	3rd min	10th min	3rd min	10th min
ACLR	227.0 (145.3)	210.2 (118.2)	267.8 (142.8)	263.8 (128.9)	158.3 (57.6)	165.4 (53.3)	222.4 (92.4)	233.9 (94.2)
INTACT	235.6 (103.2)	243.7 (114.8)	294.2 (120.6)	317.1* (140.5)	201.4 (155.6)	205.9 (171.4)	208.7 (110.4)	217.1 (106)

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1   Table 2: Mean values ± SD of the metabolic variables during the moderate and high intensity

2   running tests. (N=9). SD refers to one standard deviation. LA refers to blood lactate values.

	Velocity (m·sec^{-1})	VO$_2$ 3rd minute (ml·min^1·kg^{-1})	VO$_2$ 10th minute (ml·min^{-1}·kg^{-1})	LA Baseline (mM)	End-exercise LA (mM)
Moderate intensity bout	2.33 (0.19)	31.6 (2.8)	32.5 (2)	2.1 (0.2)	2.4 (0.6)
Heavy intensity bout	3.5 (0.25)	43.9 (2.9)	47.8[*] (3.7)	2.2 (0.3)	7.2[†] (1.8)

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3   Figure 1: Subject running on the treadmill.

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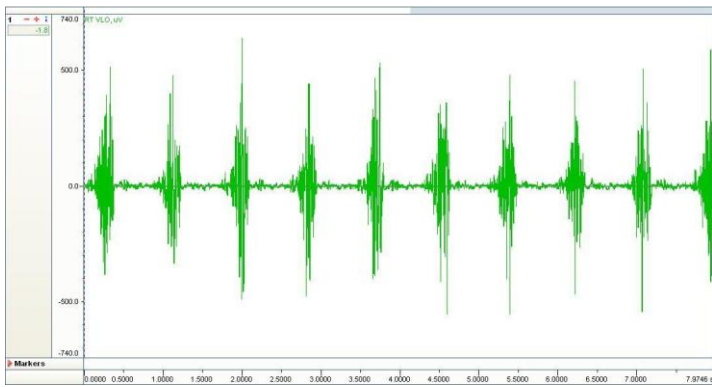
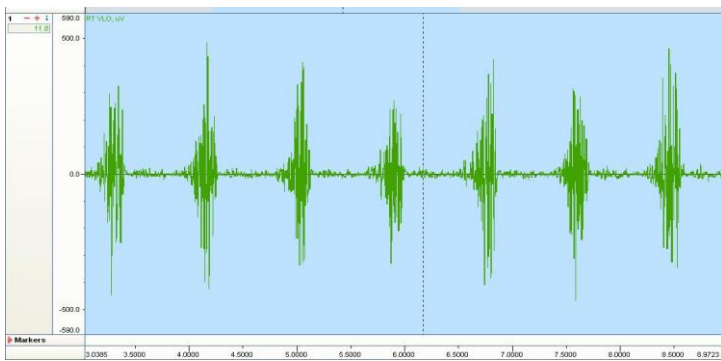2   (a)

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4   (b)

5   Figure 2: EMG signal of the vastus lateralis at the beginning (a) and at the end (b) of the high

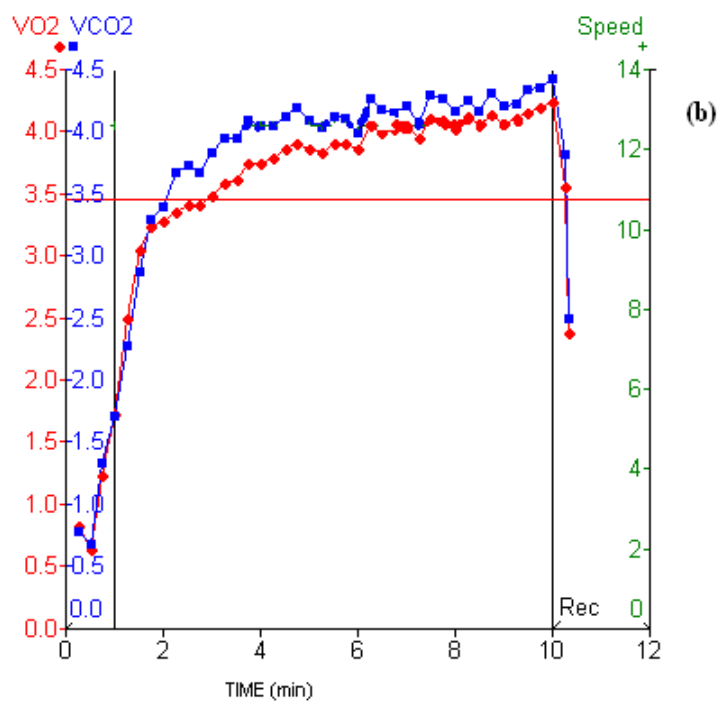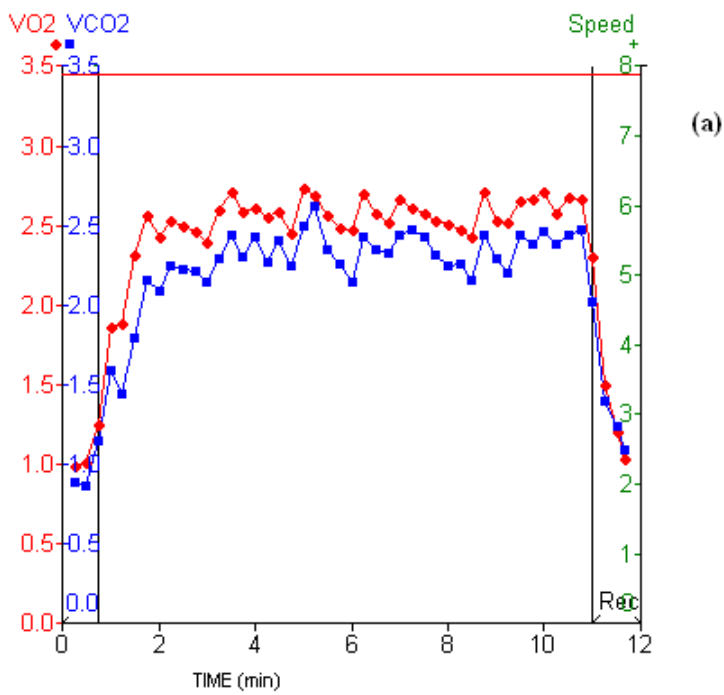6   intensity running test treadmill running test in a typical subject.
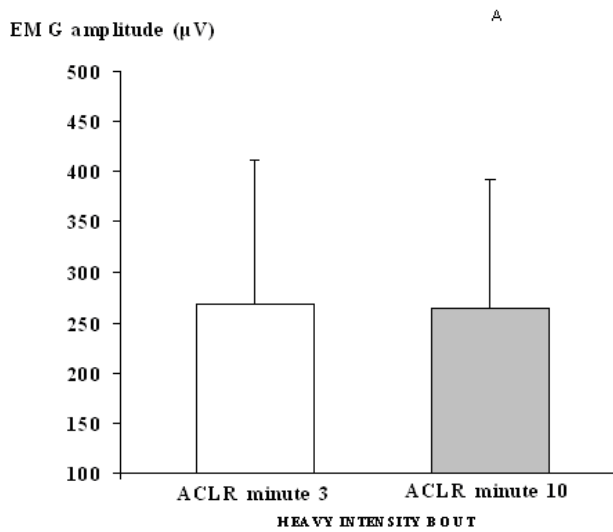
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3   Figure 3: VO$_2$ response during a moderate running at a velocity of 8 km/h in a typical subject

4   (a) and VO$_2$ response during a heavy running at a velocity of 13.2h in a typical subject (b).
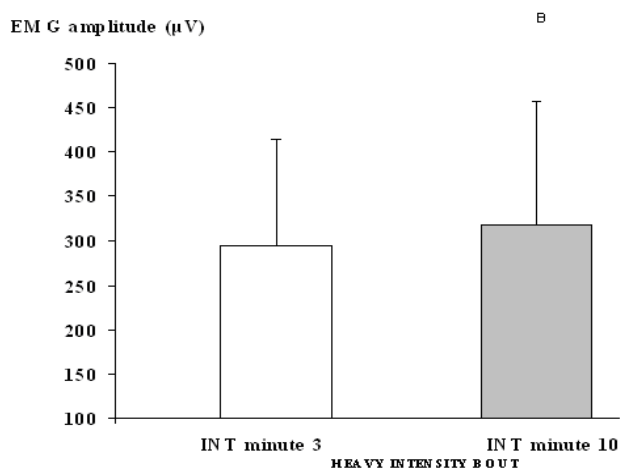
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5 Figure 4: EMG amplitude for vastus lateralis during the heavy intensity running test for (a)

6 the ACL reconstructed leg and (b) the contralateral intact leg. Values are presented as mean ±

7 SD during the 3rd and 10th minute. (N=9).