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Muscle activation of the knee extensors following high intensity endurance exercise in cyclists

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Abstract This study was conducted to assess the effects in trained cyclists of exhausting endurance cycle exercise (CE) on maximal isometric force production, surface electromyogram (EMG) and activation deficit (AD) of the knee extensors. Ten male subjects made four isometric maximal voluntary contractions (MVC) of the knee extensor muscles immediately prior (pre), 10 min after (post) and 6 h after completion of CE. The CE consisted of 30 min of exercise on a stationary cycle ergometer at an intensity corresponding to 80% of maximal oxygen uptake ($\dot{V}O_{2max}$) followed by four × 60-s periods at 120% of $\dot{V}O_{2max}$. Two MVC were performed with recording of surface EMG from the knee extensors, whilst an additional two MVC were completed with percutaneous electrical muscle stimulation (EMS; 25 pulses at 100 Hz with the maximal tolerable current) superimposed over the maximal voluntary contraction force (MVF) but without EMG (to avoid interference). The MVF, integrated EMG (iEMG), and AD [calculated as the difference between MVF and the electrically stimulated force (ESF) during the EMS contractions] were statistically analysed. The MVF was significantly reduced ($P < 0.05$) post and 6-h post compared to pre-CE level. The iEMG was significantly reduced ($P < 0.05$) post and 6 h post CE. The ESF was also reduced, whilst AD was significantly increased ($P < 0.05$) post and 6-h post CE compared to the pre CE. These results suggest that the level of exercise stress administered in this study was sufficient to impair the central and peripheral mechanisms of force generation in knee extensors for a period of 6-h. Athletes engaged in concurrent training (strength and endurance) should consider this effect in exercise programming.

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Introduction

It has been reported that the residual effects of fatigue from a previous training session may disrupt exercise performance during subsequent training sessions (Gleeson et al. 1995; Häkkinen 1992). It has been suggested that improvements in muscle strength are reduced when the neuromuscular system cannot produce a maximal level of contraction due to previous fatigue (Abernethy 1993; Craig et al. 1991). This has been found to be a primary concern among professional and recreational athletes who wish to develop simultaneously their endurance capacity and muscle strength (Hickson et al. 1988; Paavolainen et al. 1999). Although concurrent training is an approach adopted among endurance athletes aiming to improve both cardiovascular-respiratory fitness and muscle strength, only a few studies have examined the effects of a previous endurance exercise session on subsequent strength training, or vice versa (Abernethy 1993; Bentley et al. 1998). There has been a paucity of information on the recovery period necessary between an endurance exercise session and a subsequent resistance training session to achieve an optimal training effect (Bentley et al. 1998; Häkkinen 1993).

It has been demonstrated that competitive cyclists require both a high level of cardiovascular-respiratory fitness for submaximal endurance as well as muscle strength and power for short periods of sprint activity (Schabort et al. 1998). Endurance exercise performed either below or at an exercise intensity representing maximal oxygen uptake ($\dot{V}O_{2max}$) has been found to result in substrate depletion, dehydration or damage to skeletal muscle (Gonzalez-Alonso et al. 1998; Goodman et al. 1997; Thomson et al. 1979). It has been shown that submaximal exercise depletes glycogen primarily among slow twitch (ST) fibres (Vollestad et al. 1984), whilst

supramaximal (above $\dot{V}O_{2max}$) exercise depletes glycogen in fast twitch (FT) fibres (Thomson et al. 1979). To examine fatigue in cyclists, a test protocol has been previously used that consisted of a 30-min cycle at 80% of $\dot{V}O_{2max}$ followed by four periods of 60-s supramaximal sprint cycle exercise (Bentley et al. 1998). Such exercise has been shown to deplete significantly muscle glycogen stores, and produce a high concentration of blood lactate (Sahlin et al. 1998). The purpose of this study was to examine the level of muscle fatigue induced by such exercise, the recovery of fatigue after the exercise, and possible mechanisms of fatigue.

The fatigue could be either central or peripheral in origin and it has been suggested that it may result from a disturbance in homeostasis, due either to accumulation of metabolic wastes, depletion of energy supply substrates, or a combination of both (Sahlin et al. 1998). The mechanisms of fatigue and recovery in specific concurrent training regimes should be identified for optimal training programming.

Electromyography (EMG) and percutaneous electrical muscle stimulation (EMS) are two experimental techniques that have been frequently used to study muscle activation during maximal voluntary contractions (MVC) as well as the location and mechanisms of neuromuscular fatigue. The level of force exerted by muscle is directly related to the level of EMG obtained during isometric contractions as has been shown by Bigland-Ritchie et al. (1980), and De Luca (1985). The development of muscle tension during MVC has been found to be influenced by processes including motor unit recruitment (De Luca 1985). It has also been suggested that the level of tension developed in muscle serves as a stimulus to induce adaptations in muscle strength (Atha 1981). Recent investigations involving resistance trained subjects have demonstrated a reduction in surface EMG accompanying a decrease in voluntary isometric force production in the early stages after weight lifting exercise (Häkkinen 1992; Linnamo et al. 1998).

The level of neuromuscular activation can be assessed using electrical stimulation techniques. In combination with EMG, additional force induced by superimposed EMS during MVC has been used to assist in identification of central and peripheral mechanisms of fatigue (McKenzie et al. 1992; Merton 1954; Thomas et al. 1989). A parallel decrease in maximal voluntary contraction force (MVF) and EMG may be due to reduced central motor drive or impaired neuromuscular conduction and transmission. However, if EMS produces an increase in contraction force, this may indicate that the fatigue is mainly due to the central and conductive mechanisms but not the muscle contractile mechanisms. So far EMS has not been used to evaluate the fatigue and recovery processes following exhausting cycle exercise (CE) in trained endurance athletes.

The effect of exhausting CE, which included submaximal and supramaximal intensity segments, on muscle force generating capacity has been previously investigated in trained cyclists (Bentley et al. 1998). It has been found that the isokinetic and isoinertial strength was significantly reduced 6-h after completion of the CE but recovered within 24 h of exercise. The present investigation used surface EMG and EMS during MVC of the knee extensor muscles to examine the variations in isometric contractions following cycle exercise in trained subjects.

Methods

Subjects

Ten male subjects, with a mean age of 25 (SD 7) years, body mass of 72.2 (SD 7.1) kg and $\dot{V}O_{2max}$ of 58.6 (SD 5.7) ml · kg^{-1} · min^{-1}, were recruited for the study. Nine subjects were triathletes, one was a road cyclist. At the time of the study, the subjects were completing between 140 and 320 km a week of cycle training varying in intensity and duration. The experiment procedures were explained to the subjects both verbally and on an information sheet. Each subject was then required to sign an informed consent document and complete a pre-test health status questionnaire. The experiment procedure was approved by the Human Research Ethics Committee of Southern Cross University.

Experiment

Three test sessions, separated by at least 48 h, were completed by each subject during a 2-week period. The test sessions included:

1. Familarisation with the procedures
2. An incremental exercise test on a cycle ergometer for determination of $\dot{V}O_{2max}$
3. Exhausting endurance CE.

Four maximal isometric contractions of the knee extensor muscles were performed before (pre), 10 min after (post) and 6 h after CE. Surface EMG was recorded during two MVC, whilst superimposed EMS was used during the two remaining MVC. The EMG was not monitored during the two contractions with EMS to avoid cross-instrument interference on the EMG signals.

The familarisation session was used to determine each subject's level of tolerance to EMS. The time (milliseconds) to MVF was also determined during MVC. This information was subsequently used to determine the time at which the electrical stimulus would be triggered during the isometric contractions.

The incremental exercise test was performed on a mechanically braked cycle ergometer (Monark 868, Sweden). The test commenced at an exercise intensity of 100 W and increased by 30 W every 3 min until the subject was unable to continue or could not maintain the required cadence of 80 rev · min^{-1}. The $\dot{V}O_{2max}$ was defined as the average of the two highest oxygen uptake values obtained at 15-s intervals during the final intensity of the incremental test (Hawley and Noakes 1992).

Cycle exercise

The CE was performed on a mechanically braked cycle ergometer (Monark, 868, Sweden) fitted with a racing saddle and the subjects' own pedals. The CE consisted of 30 min at an exercise intensity equivalent to 80% of $\dot{V}O_{2max}$ followed by four periods of 60 s at an intensity representing 120% of the intensity at $\dot{V}O_{2max}$. A 60-s recovery was allowed between each period. Cycling at 100 W for 5 min was allowed after the 30-min exercise period and before the four supramaximal efforts. Cycling cadence throughout the exercise remained at 80 rev · min^{-1}. Verbal encouragement was given to the subjects during the four periods of supramaximal exercise.

This protocol of submaximal and supramaximal exercise was similar to previous research that has demonstrated a reduction of glycogen concentrations in both FT and ST muscle fibres (Gollnick et al. 1974; Thomson et al. 1979). The intensity and duration of exercise was also selected to imitate a typical training session performed by competitive cyclists (see Stepto et al. 1999).

Isometric knee extension test

Maximal voluntary and electrically stimulated isometric contractions of the knee extensor muscles were performed pre, post and 6 h after CE. The pre and 6-h post-test sessions commenced with a standardised warm-up involving 5 min of cycle exercise at 100 W and a 5-min period of rest before the commencement of the knee extension test. As part of the warm-up, the subjects were also required to perform two submaximal voluntary contractions on the dynamometer.

During the test the subject sat in a chair modified from an isokinetic dynamometer (Cybex II, Lumex Inc., Ronkonkoma, USA), firmly strapped in place at the waist and at the quadriceps muscle of the right leg. A velcro strap was attached around the right leg just superior to the malleolus of the ankle joint. The strap was connected to a load cell (S1W, XTran, Australia) which was anchored to a lever arm (Custom-designed, Southern Cross University) bolted to the test chair. The position of the force transducer on the lever arm was recorded during familiarisation for accurate re-positioning during each subsequent test sessions. A goniometer was used to set the knee joint at a 90° angle. Force-time data was collected via the load cell calibrated to an Associative Measurement Laboratory (AMLAB) computer system (Associative Measurement, Sydney, Australia).

To avoid the baseline noise of EMG recording due to cross-instrument interference, two contractions were performed whilst surface EMG was recorded, and the other two contractions with superimposed EMS but without EMG. The two sets of contractions were performed in a randomised order to allow for possible order effects. Surface EMG signals were collected from the vastus lateralis (VL), vastus medialis (VM) and rectus femoris (RF) muscles for evaluation of voluntary neuromuscular activation. The EMS consisted of a train of 25 electrical pulses delivered to the muscle via surface electrodes during MVC after MVF was reached. The activation deficit (AD) of the knee extensors was determined as an increase in force induced by EMS during the MVC (Kent-Braun and Le Blanc 1996).

Electromyography

A pair of silver-silver chloride surface EMG recording electrodes were used with a reference electrode located at equal distance to the two recording electrodes (custom-built, Southern Cross University). The electrodes, pre-assembled in a small box, were attached over the belly of the VL, VM and RF muscles. Each recording electrode had a diameter of 6 mm. Each pair of recording electrodes, with an inter-electrode distance of 20 mm, was placed in a longitudinal direction to the muscle fibre (Basmajian and De Luca 1985). Before application of the electrodes, the skin surface was shaved and cleansed with alcohol and mildly abraded to reduce impact impedance. Electrode conduction gel was applied to the electrode contact face. To reduce artefacts due to movement, the electrodes were taped firmly in place. A bandage was applied to the thigh in an attempt to keep the electrodes in place during the CE and post-exercise test sessions. The outline of the electrode box was drawn with a marker pen on to the skin to allow accurate repositioning following the 6-h recovery period. A similar procedure had been used in previous work examining the recovery of the integrated EMG (iEMG) signal following weight training exercise (Häkkinen 1992; Linnamo et al. 1998).

The signals from three channels of EMG and one channel of force were displayed on a monitor and recorded continuously on a computer hard disk throughout each contraction using the AMLAB system. The raw EMG signals were recorded via a differential a.c. amplifier in the AMLAB system which possessed an input impedance of $> 10^{12}\ \Omega$ and a common mode rejection ratio of more than 120 dB. The force and EMG signals were A-D converted at 2 000 Hz and stored on computer hard disk for further analysis. The EMG data was band-pass filtered [5 Hz (−3 dB) to 958 Hz (−3 dB)]. A 1-s period of raw EMG from the point of MVF was analysed for each MVC. This data was full-wave rectified and integrated using the AMLAB system.

Electrical muscle stimulation

Two stimulation units associated with the AMLAB system operating in parallel elicited EMS. Each unit had a maximal current output of 100 mA. Two sets of 3 inch × 5 inch self adhesive surface electrodes (Uni-Patch, Wabasha, USA) were applied to the quadriceps muscle group. One set of electrodes was placed slightly to the lateral side of the thigh over the VL muscle, with one electrode just proximal to the patella and the other just distal to the hip joint over the anterio-lateral part of the quadriceps muscle. The other set of electrodes was placed in a similar way but slightly to the medial side of the thigh over the VM muscle. A pen was used to outline each electrode to enable easy re-positioning on each subsequent testing occasion.

A potential methodological problem when using EMS during MVC is that the superimposed electrical stimulus may not correspond to the maximal level of voluntary force produced during MVC. This has the effect of eliciting a larger increment in force because the subject is not voluntarily contracting maximally at the time of EMS. To eliminate this problem, the time to peak force was used to determine the point when *all* subjects had reached MVF and this level of force generation had not declined. Force data from the familiarisation trial showed that all the subjects had reached peak force 0.7 s after the onset of force generation (defined as reaching 100 N of force). The superimposed EMS train was therefore delivered at this time. The EMS was configured as 25 pulses at a frequency of 100 Hz (i.e. a total duration of 250 ms), with a square-wave pulse width of 0.5 ms and an intensity of current that depended on the subject's tolerance. The maximal current subjects could withstand as determined from the familiarisation trial was 80–90 mA from each stimulation unit. The MVF and the electrically stimulated force (ESF) were subsequently determined and the difference in these two values was deemed to be the AD of muscle as proposed by Kent-Braun and Le Blanc (1996).

Statistical analysis

Two factor (test time × force) analysis of variance (ANOVA) with repeated measures was used to compare pre, post, and 6-h post CE values for MVF obtained during MVC with and without EMS. The AD was expressed in both absolute terms and as a percentage of ESF. Single factor (test time) ANOVA with repeated measures was used to compare AD at each test time. A two-factor ANOVA was calculated to compare iEMG of the VL, VM or RF (test time × muscle) as well as MVF and ESF during MVC with EMS (test time × force) measured pre, post and 6-h post CE. In all cases significance was set at an α level of $P \leq 0.05$.

Results

All the subjects successfully completed the test procedures.

Significant reductions were found for MVF, obtained during MVC performed with and without EMS, post and 6-h post CE when compared to the pre CE level (Table 1, Fig. 1). There was a trend for MVF obtained during MVC with EMS to be lower than that obtained

Table 1 The integrated electromyogram of the knee extensor muscles and maximal voluntary contraction force (*MVF*) during isometric contractions without electrical muscle stimulation, [before (pre), 10 min after (post), and 6 h after (6 h) cycle exercise]

(mV · s)	Pre		Post		6 h	
	Mean	SD	Mean	SD	Mean	SD
MVF (N)	331	29	292	31*	310	26*
Vastus lateralis muscle	3555	1000	3146	1616	3126	760
Vastus medialis muscle	3462	964	2708	1065*	2994	1245
Rectus femoris muscle	3533	1416	2707	1351*	2905	1091*

* Significantly different from the pre values ($P < 0.05$)

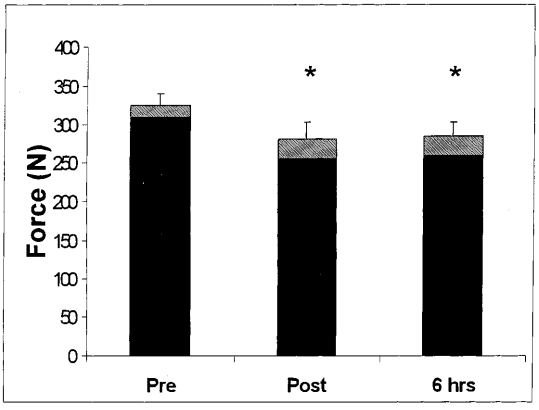

Fig. 1 Maximal voluntary contraction force (Force) and activation deficit (*grey area of each histogram*) during electrically stimulated isometric contractions before (*pre*), 10 min after (*post*), and 6 h after (*6 h*) cycle exercise. *Error bars* represent SD, *maximal voluntary contraction force and activation deficit both $P < 0.05$ compared to pre

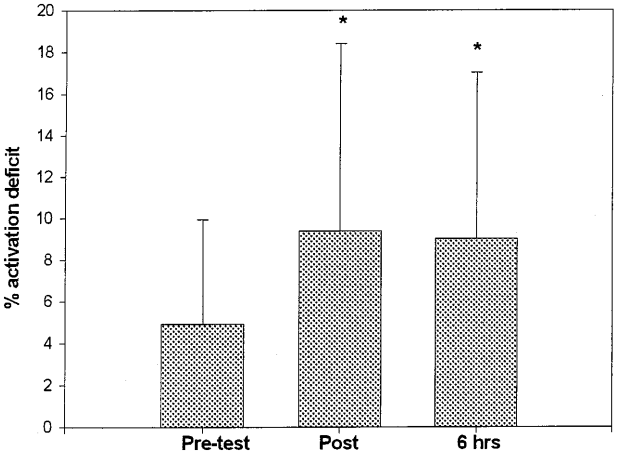

Fig. 2 The activation deficit as a percentage of electrically stimulated force before (*pre*), 10 min after (*post*) and 6 h after (*6 h*) cycle exercise. *Error bars* represent SD, *$P < 0.05$ compared to pre

in MVC performed without EMS at each test time, however this difference was not significant.

Significant reductions in iEMG were found in the RF and VM muscles post CE. However, no significant difference was evident post CE for the VL muscle (Table 1). A significant reduction in iEMG for the RF muscle was found following 6 h of recovery, however, no significant differences were found for iEMG of the VM or VL muscles after 6 h of recovery from CE (Table 1).

During the electrically stimulated contractions, ESF was significantly greater than MVF at each test (Fig. 1). The AD expressed in absolute terms as well as percentage of ESF was significantly greater both post CE and after 6 h of recovery (Figs. 1, 2).

Discussion

The EMG and EMS were used in the present investigation in conjunction with an isometric contraction before and in the early stages following exhausting CE. The CE protocol resulted in a significant reduction in MVF, post and after 6 h of recovery from CE. The reduction in MVF was also accompanied by decreases in iEMG. The AD significantly increased post and 6 h following CE compared to the pre levels.

It has been reported that the level of surface EMG in general reflects the degree of neural activity including the number of motor units recruited and the firing frequency to the muscle being investigated (Basmajian and De Luca 1985; Bigland-Ritchie 1981). A number of studies have investigated iEMG during recovery periods following a variety of exercise protocols (Kroon and Naeije 1988; Linnamo et al. 1998; Newham et al. 1983). However, no studies have examined the recovery of iEMG during isometric contractions in trained athletes following exhausting endurance cycle exercise. The reduction in iEMG shown in this study was comparable to that in previous work that has demonstrated a parallel reduction in iEMG and maximal voluntary strength during isometric contractions of the knee extensor muscles during recovery following resistance training (Häkkinen 1992; Linnamo et al. 1998).

Linnamo et al. (1998) have reported a reduction in iEMG 24 h following resistance exercise consisting of five sets of a ten repetition maximal load. Those authors have suggested the reduction in isometric force was possibly due to fatigue of central origin, as evidenced by the decreased iEMG level. The reduction in the iEMG level found in the present study may also have been evidence of a central mechanism associated with the reduction in MVF. However, other researchers have found no change in iEMG in the recovery period following concentric stepping exercise (Newham et al. 1983) or immediately after stretch-shortening cycle exercise (Strojnik and Komi 1998) despite reductions in maximal voluntary strength.

The iEMG of the VL, VM and RF muscles was lower post CE and after 6 h of recovery. However, significant reductions were only found in the iEMG of the VM muscle post CE and the iEMG of the RF muscle post

and 6 h following CE. A number of studies have examined EMG of the VL, VM and RF muscles during cycling activity (Eisner et al. 1999; Takaishi et al. 1996). It has been shown from these works that a similar level of recruitment occurs in the knee extensor muscles of the quadriceps during the "down phase of pedalling in cycling. Therefore, it is unlikely that differences in iEMG of the VL, VM and RF muscles found in the recovery stages in our study were due to differences in the recruitment of these muscles during the CE protocol.

Another possible explanation for the changes in iEMG of the VL, VM and RF muscles is the conditions surrounding the electrode placement post CE as well as the re-positioning of the electrodes after 6 h of recovery. Other researchers have used a similar methodology to the present study, in longitudinal investigations examining muscle fatigue or neurological changes with weight training or cycle exercise (Hanon et al. 1998; Linnamo et al. 1998). A limitation of these studies and the present investigation was that the possible variations in EMG signal following replacement of the electrodes and the changes in muscle temperature during the exercise protocol were not evaluated.

The method of EMS used in this investigation had been successfully included as part of research investigating the level of voluntary muscle activation of the quadriceps muscle group in untrained and resistance trained subjects (Shield et al. 1997, unpublished work, Southern Cross University). The 4.9% increment in force found in the present study was comparable to the work of Shield et al. (1997, unpublished work) who have observed a 2.6% increment in force evoked by EMS superimposed on MVC. By quantifying AD, some researchers have assessed whether central or peripheral mechanisms are responsible for the decline in voluntary force levels (McKenzie et al. 1992; Merton 1954; Thomas et al. 1989). For example, Thomas et al. (1989) have found electrically stimulated force levels declined in parallel with voluntary force levels following a 5-min sustained isometric MVC. For this reason, these authors have concluded that the reduction in force was not associated with decreased central drive, but rather from impairment of more peripheral processes of muscle contraction. However, other authors have concluded an increase in force induced by an electrical stimulus indicated impaired central activation (Gandevia 1992; McKenzie et al. 1992).

In the present study, an increase in AD was found as a consequence of CE. This finding may be interpreted as a reduction in central neural drive. However, the increase in AD was also accompanied by a reduction in *both* ESF and MVF, indicating that despite the superimposed electrical stimulus during MVC, the muscle was not able to contract at the level prior to CE. A reduction in the level of MVF and ESF has been postulated to indicate a disruption of the peripheral processes of muscle contraction such as altered nerve conduction, synaptic transmission, or excitation-contraction processes (Duchateau and Hainaut 1985).

Co-activation of the antagonist muscles has been identified during fatiguing isometric contractions (Psek and Cafarelli 1993). This has the effect of producing a force that opposes the force produced by the agonist muscle group. This in turn may reduce the force produced for a given action initiated by the agonist muscles. Although the present study identified possible central and peripheral mechanisms associated with the reduction in MVF, it was possible that co-activation of the hamstrings also contributed to the reduction in MVF following CE. Because EMG of the hamstrings was not assessed, it was impossible to conclude whether co-activation was associated with the reduction in MVF. At the same time, it is possible that the hamstring muscles were equally recruited and were fatigued to a similar extent following CE as has been proposed by Andrews (1987) and Eisner et al. (1999). Whether fatigue of the antagonist muscles prior to an isometric leg extension test would reduce the co-active effect remains to be investigated.

The results of the present study showed that isometric force production of the quadriceps was reduced for at least 6 h following exhausting endurance CE. This would suggest that it may not be possible for resistance training to be performed at an intensity that would otherwise be done without prior endurance training. Whether this would influence the number and intensity of weights lifted during a resistance training session and therefore efficacy of the training needs further longitudinal investigation.

In summary, MVF and ESF of the knee extensors was reduced for at least 6 h following the exhausting cycle exercise completed by trained endurance cyclists. The reduction in MVF was also associated with a reduction in surface EMG. It remains to be examined whether the reduction in force generating capacity observed in this study following endurance exercise would affect long-term gains in muscle strength with resistance training.

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