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Neuromuscular performance of explosive power athletes versus untrained individuals

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Abstract

Purpose: Electromechanical delay (EMD) and rate of force development (RFD) are determinants of explosive neuromuscular performance. We may expect a contrast in EMD and RFD between explosive power athletes, who have a demonstrable ability for explosive contractions, and untrained individuals. However, this comparison, and the neuromuscular mechanisms for any differences, has not been studied. **Methods:** The neuromuscular performance of explosive power athletes (n = 9) and untrained controls (n = 10) was assessed during a series of twitch, tetanic, explosive and maximum voluntary, isometric knee extensions. Knee extension force and EMG of the superficial quadriceps was measured in three 50 ms time windows from their onset, and normalised to strength and maximal M-wave (M_{max}), respectively. Involuntary and voluntary EMD were determined from twitch and explosive voluntary contractions, respectively, and were similar for both groups. **Results:** The athletes were 28% stronger and their absolute RFD in the first 50 ms was 2-fold that of controls. Athletes had greater normalised RFD (4.86 ± 1.46 vs. 2.81 ± 1.20 $MVC.s^{-1}$) and neural activation (mean quadriceps, 0.26 ± 0.07 vs. 0.15 ± 0.06 M_{max}) during the first 50 ms of explosive voluntary contractions. Surprisingly the controls had a greater normalised RFD in the second 50 ms (6.68 ± 0.92 vs. 7.93 ± 1.11 $MVC.s^{-1}$) and a greater change in EMG preceding this period. However, there were no differences in the twitch response or normalised tetanic RFD between groups. **Conclusion:** The differences in voluntary normalised RFD between athletes and controls were explained by agonist muscle neural activation, and not the similar intrinsic contractile properties of the groups.

Keywords: Rate of force development; electromechanical delay; neural activation; intrinsic contractile properties

Introduction

Explosive muscular contractions are fundamental to sports activities such as sprinting, jumping, and punching, and are important for preventing injury following mechanical perturbation (1, 27). During explosive contractions the time for the muscles to develop force is often limited to 50-250 ms (1, 15). Consequently, the electromechanical delay (EMD; the time delay between the onset of electrical activity at the muscle and the generation of force (46)) and the rate of force development (RFD; the slope of the force-time curve) are important descriptors of performance in explosive contractions (1, 5, 8, 14, 18, 27, 30). Explosive power athletes have a clear ability for explosive contractions, but their capabilities in relation to untrained individuals have been poorly documented, with few attempts made to investigate the physiological mechanisms for any differences. Such a comparison may enhance our understanding of explosive contractions and provide implications for enhancing athletic performance and reducing injury risk in all populations.

Whilst a shorter EMD would theoretically enhance explosive performance by decreasing the neuromuscular response time to a particular stimulus, it is not known whether EMD is shorter in explosive power athletes. Fibre type distribution and tendon stiffness are known to affect EMD (22, 39), and previous studies have reported differences in these factors when comparing explosive power athletes to untrained individuals (23, 26), suggesting that EMD discrepancies may also exist. Measurement of involuntary EMD, from electrically evoked twitch contractions, may reveal if aspects of muscle-tendon morphology and

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mechanics contribute to any differences in voluntary EMD. During explosive voluntary contractions EMD is ~100% (16-25 ms) longer than during involuntary contractions (27, 49, 51), indicating that neural activation may also play an important role in EMD. The difference between the EMD of voluntary and involuntary contractions may be an effective method of quantifying the voluntary neurological aspect of the delay.

Explosive power athletes would also be expected to have a greater RFD than other populations. A greater absolute RFD has been found in explosive power athletes when compared to endurance athletes (24). However, when RFD is normalised to maximal voluntary force (MVF) the difference between explosive power athletes and endurance athletes or untrained individuals is equivocal (17, 24, 42), perhaps because these studies made only a single observation of RFD at variable time points from force onset. Furthermore these reports did not investigate the specific causes for any differences in RFD.

Muscle-tendon morphology and mechanics are expected to affect the intrinsic contractile properties of the muscle-tendon unit, determined via electrically stimulated involuntary contractions (3, 19, 31). Assessment of involuntary contractions may therefore help to explain any enhanced RFD of explosive power athletes. However, the relationship between the intrinsic contractile properties of the muscle-tendon unit and voluntary RFD is equivocal (4, 8, 10). Alternatively, the level of neural activation during the initial phase of a contraction could also be responsible for differences in RFD. Recent cross-sectional studies have confirmed a relationship (r^2 = 0.75-0.83) between agonist muscle activation, assessed with EMG amplitude, and the torque-time integral in the first 40 ms of an explosive contraction (8-10). In addition to the level of neural activation, the degree of synchrony in the activation onset of all agonists/synergist muscles contributing to net force production is also likely to affect RFD, but this issue has not been investigated.

The aim of this study was to compare the neuromuscular function, of explosive power athletes and untrained individuals (controls) during voluntary and involuntary explosive isometric contractions of the knee extensors, with particular attention to EMD and the RFD throughout the ascending force-time curve. The comparison of voluntary and involuntary contractions, and the assessment of neural activation with EMG, may help to delineate the importance of neural factors vs. the intrinsic properties of the muscle-tendon unit in explosive muscular performance.

Methods

Participants

Nineteen male participants were recruited to form two groups; explosive power athletes (n = 9; age, 21 ± 3 yrs; height, 181 ± 7 cm; and mass 80 ± 8 kg) and controls (n = 10; age, 22 ± 4 yrs; height, 179 ± 5 cm; and mass 81 ± 13 kg). The athletes were sprinters or jumpers competing at a national/international level, and performing regular strength/power training (>3 x per week) for ≥2 years. The controls consisted of light to moderately active individuals (≤3 x aerobic activity a week), who were not involved in any strength or power training. All the participants were healthy, injury free and provided written informed consent prior to their involvement in this study, which was approved by the Loughborough University ethical advisory committee.

Overview

Participants visited the lab for 60-75 min at a consistent time of day on three occasions, to complete a familiarisation trial and two main trials in which measurements were recorded. Trials were separated by one week and consisted of the same protocol. Participants sat in an

isometric strength testing chair (5, 34) and completed a series of isometric voluntary and involuntary contractions of the knee extensors of their dominant leg. Specifically, twitch and tetanic electrically stimulated contractions, explosive voluntary and maximal voluntary contractions (MVC) were performed. In addition, knee flexor isometric MVCs were also completed at the end of each trial. Knee extension/flexion force and surface electromyography (EMG) of the superficial knee extensors and the biceps femoris were recorded throughout these contractions.

Force Measurements

Participants were firmly secured to the strength testing chair with a waist belt and shoulder straps. The hip and knee angle was fixed at 100° and 85°, respectively (180° was full-extension). An ankle strap was placed 2 cm proximal to the medial malleolus and was in series with a calibrated U-shaped aluminium strain gauge (linear response up to 1000 N; (21)). The strain gauge was positioned perpendicular to tibial movement during knee extension/flexion. The force signal was amplified (x500), interfaced with an analogue to digital converter (CED micro 1401, CED, Cambridge, UK), and sampled at 2000 Hz with a PC utilising Spike 2 software (CED, Cambridge, UK). The force signal was notch filtered at 100 and 200 Hz in both directions with an infinite impulse response digital filter (q-factor of 100), to remove any harmonics of the mains frequency. Real-time biofeedback of the force response was provided on a computer monitor.

Electromyography Measurements

Surface EMG was recorded from the rectus femoris (RF), vastus lateralis (VL), vastus medialis (VM), and the long head of the bicep femoris (BF), using a Delsys Bagnoli-4 EMG system (Delsys, Boston, USA). Following preparation of the skin (shaving, lightly abrading and cleansing with 70% ethanol), a single differential surface electrode configuration (1 cm inter-sensor distance, model DE-2.1, Delsys, Boston, USA) was attached over the belly of each muscle parallel to the presumed orientation of the muscle fibres, using adhesive interfaces. The reference electrode was placed on the patella of the same limb. The same investigator placed the electrodes in the same relative position on all participants, and marks left on the skin by the electrodes in the first main trial were used to relocate the electrodes in the second main trial. Each EMG signal was amplified (x100, differential amplifier 20-450 Hz), synchronised with the force signal using the same analogue to digital converter and PC software, sampled at 2000 Hz, and band-passed filtered in both directions between 6-500 Hz using a 2nd order Butterworth digital filter.

Electrical Stimulation

The femoral nerve was electrically stimulated with square wave pulses (0.1 ms duration) to elicit single pulse twitch contractions, and facilitate compound muscle action potentials (M-waves). The anode (carbon rubber electrode, 7 x 10 cm; Electro-Medical Supplies, Greenham, UK) was taped to the skin over the greater trochanter. The cathode, a custom adapted stimulation probe (1 cm diameter, Electro Medical Supplies, Wantage, UK) protruding 2 cm perpendicular from the centre of a plastic base (4 x 5 cm), was taped to the skin over the femoral nerve in the femoral triangle. The precise location of the cathode was determined as the position that evoked the greatest twitch response for a particular submaximal electrical current.

One second tetanic contractions (100 Hz) were evoked with a train of electrical impulses (square wave pulses, 0.1 ms duration), delivered via two carbon rubber electrodes (14 x 10 cm; Electro-Medical Supplies, Greenham, UK) taped securely to the anterior surface

of the thigh (the cathode 8 cm proximal from the patellar, and the anode 10 cm proximal from the cathode). The electrical impulses for all involuntary contractions were delivered with a constant current, variable voltage stimulator (DS7AH, Digitimer Ltd., UK), and triggered by the CED micro 1401. The stimulator output was also recorded by the analogue to digital converter and PC software.

Protocol

Measurements were completed in the following order.

Maximal M-wave and evoked Twitches

A series of twitch contractions at incremental electrical currents were elicited until a simultaneous plateau in M-wave and twitch tension response was observed. Thereafter, the electrical current was increased by 20% and three supramaximal pulses were elicited at 12 second intervals. The average M-wave response (peak-peak amplitude of the EMG signal) to these three supramaximal impulses was defined as the maximal M-wave (M_{max}) and used for EMG normalisation (see below). Peak tension, time to peak tension, and RFD in the first 50 ms from force onset (change in force divided by 0.05 s) was also averaged across the three supramaximal twitch contractions. Twitch RFD was measured in absolute terms and relative to peak twitch tension. In each twitch contraction the time difference between M-wave onset and force onset was determined for the superficial knee extensors, and the largest value of the three muscles was defined as involuntary EMD_{max} (49, 51). M-wave onset was identified manually (see below) and defined as a positive or negative deflection away from the baseline following the initial stimulation artefact. Involuntary EMD_{max} was averaged across the three supramaximal twitches.

Explosive Contractions

Participants completed a two minute warm-up of the knee extensors of their dominant leg with a series of sub-maximal contractions. They then performed 10 voluntary explosive isometric contractions, separated by a 20-s rest (pilot testing confirmed this recovery time was sufficient). For each contraction participants were instructed to relax, take a deep breath, and following an auditory signal, attempted to extend their knee as 'fast and hard' as possible for 1-1.5 s, with an emphasis on 'fast'. Participants were asked to avoid any countermovement prior to force onset. To provide biofeedback on whether a countermovement had occurred, the resting force level was displayed on a sensitive scale. The slope of the force-time curve (1 ms time constant) was also displayed throughout these contractions and the peak slope from each contraction used as biofeedback.

The three contractions with the largest peak slope and no discernible countermovement or pre tension (change of baseline force of < 0.5 N during the 100 ms prior to contraction onset) were used for analysis. Analysis consisted of measurement of force-time and EMG-time curves in three, 50 ms time windows (0-50, 50-100, and 100-150 ms) after their respective onsets (Fig. 1). RFD for each time window (change in force divided by 0.05 s) was measured in absolute terms and normalised to MVF (detailed below) i.e., RFD/MVF.
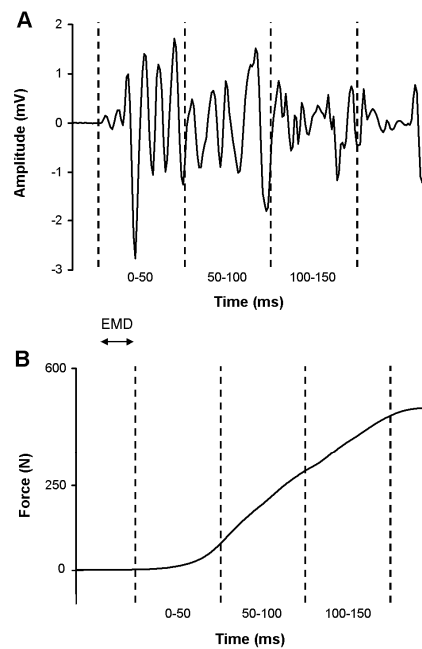
Fig. 1. VM filtered EMG (A) and knee extensor force (B) signals recorded during an explosive isometric contraction. Both signals were analyzed in three consecutive 50-ms time windows from their respective onsets (0–50, 50–100, and 100–150 ms). EMD was defined as the time difference between the onset of each signal.

The root mean square (RMS) of the EMG signal during each time window was calculated for each muscle (VM, VL, RF, BF). Antagonist (BF) EMG was normalised to peak BF activation during the knee flexor MVCs (detailed below). EMG amplitude for the agonists (VM, VL, RF) was normalised to M_{max} (i.e. EMG RMS/M_{max}). Normalised agonist EMG for each time window was also averaged across the knee extensors

to give a mean quadriceps value. The rate of change in normalised EMG (ΔEMG/Δtime) between rest and the first 50 ms time window (denoted by 0-25 ms, with 25 ms representing the midpoint of the first 50 ms window), the first and second time windows (25-75 ms), and the second and third time windows (75-125 ms), was also determined for each knee extensor and averaged across the three muscles. Median Frequency (MF) of each agonist EMG signal was calculated for the first 150 ms from onset, with a frequency resolution of 6.7 Hz, and was averaged across the three knee extensors. To determine synchrony in the activation onset of the three agonist muscles, the time difference between the EMG onset of the first and last muscle to be activated was calculated for each contraction. This time difference and all RFD and EMG variables were averaged across the three explosive contractions.

In each explosive contraction, the time difference between EMG and force onset (EMD) was determined for each of the superficial knee extensors and the largest EMD of the three muscles (true EMD; (49, 50)) was defined as EMD_{max}. The three contractions with the shortest EMD_{max} were averaged to give voluntary EMD_{max}, which is also reported as a percentage of involuntary EMD_{max}.

Maximal Voluntary Contractions (MVCs)

Participants completed four knee extensor isometric MVCs (separated by ≥30 s). In response to an auditory signal they were instructed to push as hard as possible for 3 seconds. Biofeedback and verbal encouragement were provided during and between each MVC. Knee extensor MVF was the greatest voluntary force achieved by a participant, in any of the knee extensor MVCs or explosive contractions during that laboratory visit.

Tetanic Contractions

A series of tetanic contractions at incremental electrical currents were elicited until 50% of MVF was evoked. Thereafter, three tetanic contractions of 50% MVF were evoked (20 s apart) and recorded. Absolute and normalised RFD variables, determined via the same methods as the explosive contractions, were averaged across the three tetanic contractions.

Knee Flexor MVCs

Following a 2-min warm-up of submaximal contractions participants performed three isometric MVCs of the knee flexors (separated by 30 s), at a knee joint angle of 105°. This knee joint angle was required as participants were not comfortable performing the knee flexor MVCs at an 85° knee joint angle. The highest RMS of the BF EMG signal was determined using a 100 ms time constant for each MVC, and was averaged to give peak BF activation.

Data Analysis and Statistics

For all voluntary and involuntary contractions identification of force and EMG onsets were made manually (visually). Manual identification of signal onsets is the 'gold standard' method (2, 20, 28, 33, 38, 40). Whilst it is considered more subjective than the mathematical algorithms (automated methods) typically used in exercise science (e.g., ≥2 SD of the baseline), manual identification is more sensitive and accurate (2, 20, 37), detecting onsets up to 60 ms earlier than automated methods (2, 33, 35). Accurately quantifying signal onsets and rapid changes during the initial phase of a contraction (50 ms) was integral to the aims of this investigation; therefore manual identification was employed. The same investigator analysed all signal onsets. Initially, signal recordings were viewed with a constant y-axis scale of ~1 N and 10 mV, for force and EMG respectively, and an x-axis scale of 500 ms. These scales provided a good resolution from which the pattern of the noise could be established and the signal onset (last peak/trough before the signal deflected away from baseline noise) interpolated. A vertical cursor was placed on signal onset and the signals were then viewed with a higher resolution (y-axis scale of ~0.5 N and 6 mV, for force and EMG respectively, and an x-axis scale of 25 ms), to verify that the vertical cursor was on the apex of the peak/trough (Fig 2). The instant of force onset was confirmed by also displaying the first derivative of the force-time trace. To determine the reliability of manually identifying force and EMG onsets, 19 explosive contractions (one from each participant) were chosen at random and re-analysed a week after the original analysis. From this repeat analysis the typical error of the measurement was calculated for force onset, EMG onset, and voluntary EMD_{max}.
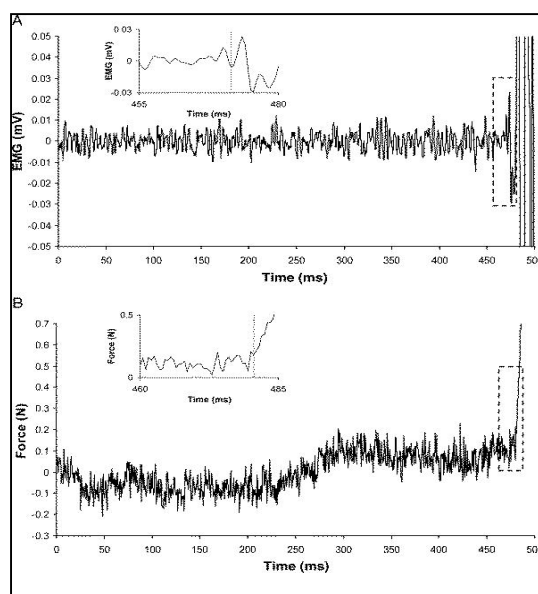
Fig 2. Typical RF EMG (A) and force (B) signals before the onset of an explosive voluntary isometric contraction of the knee extensors. The graphs are displayed on the scale by which the signal onset was manually detected, and the EMG has been converted to preamplification values. The inserts are magnifications of the signal within the dashed line box and illustrate the scale by which the investigator confirmed that signal onset (dashed line) had been placed on the apex of the last peak/trough before the signal deflected from the baseline noise.

For each dependent variable between trials coefficient of variation (CV) was calculated (across the two main trials) for each participant and averaged across all participants. Dependent variables for each participant were averaged across the main two trials before group values (mean ± SD) were generated. For variables measured at three time points (force, RFD, EMG and the rate of change in EMG during the explosive contractions; and RFD during the tetanic contractions) the influence of time and group was analysed with a two-way repeated measures ANOVA (2 groups x 3 repeated measures). A stepwise Bonferroni corrected Paired t-tests was then used to determine differences between groups at specific time points. All other dependent variables (median frequency, MVF and twitch parameters) were assessed using an independent samples t-test. Statistical analysis was completed using SPSS version 14, and the significance level was set at $P<0.05$.

Results

Electromechanical Delay

Involuntary EMD_{max} during the supramaximal twitches was similar for both groups ($P = 0.28$; Table 1). During the explosive contractions there was no consistent order of activation of the three knee extensors, even within an individual. Athletes and controls had similar voluntary EMD_{max} whether expressed in absolute terms or as a percentage of involuntary EMD ($0.14<P<0.19$; Table 1). The typical error of measurement for force and EMG (all three knee extensors) onset identification was 0.9 and 0.7-1.3 ms, respectively. The typical error of measurement of voluntary EMD_{max} was 1.2 ms. Between trials CV for voluntary and involuntary EMD_{max} were 12.6 and 10.0%, respectively.

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Table 1. Involuntary EMDmax, voluntary EMDmax, and voluntary EMDmax as a percentage of involuntary EMDmax for athletes (n = 9) and controls (n = 8).

	Athletes	Controls	*P*
Involuntary EMD$_{max}$ (ms)	7.3 ± 0.9	6.7 ± 1.1	0.282
Voluntary EMD$_{max}$ (ms)	13.5 ± 4.0	15.9 ± 3.7	0.188
Voluntary EMD$_{max}$ (%)	188 ± 63	243 ± 82	0.141
Data are mean ± SD.			

Voluntary Force

The athletes produced a greater MVF than the controls (742.6 ± 73.4 vs. 579.4 ± 131.6 N; P = 0.004). During the explosive contractions the athletes achieved a greater force at 50, 100 and 150 ms (P<0.001; Fig. 3A), but their absolute RFD was only greater during the 0-50 ms time window (3542 ± 887 vs. 1580 ± 605 N.s^{-1}; P<0.001; Fig. 4A).
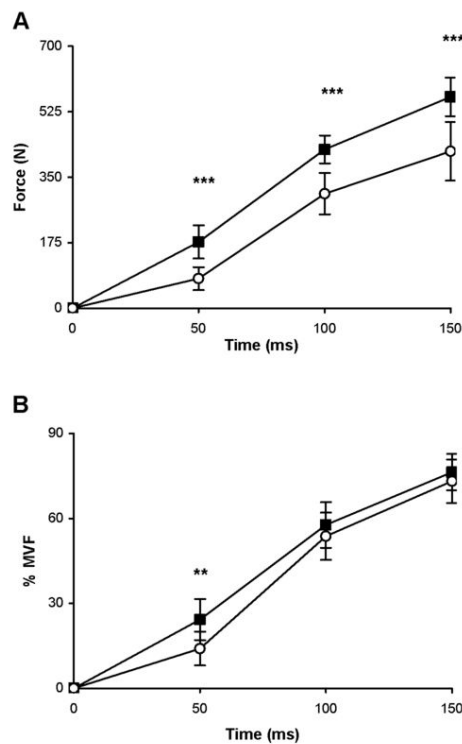

Fig. 3. Absolute (A) and normalized (B) force of explosive power athletes (filled squares; n = 9) and controls (clear circles; n = 10) during explosive isometric contractions of the knee extensors. Data are mean ± SD. **P < 0.01, ***P < 0.001.
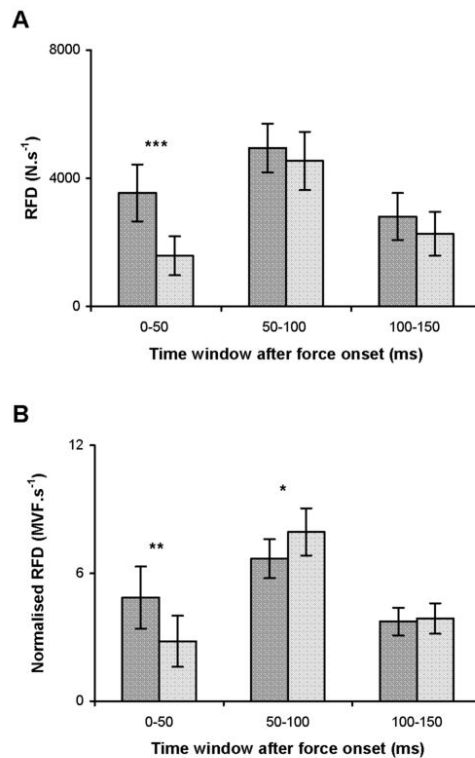
Fig. 4. Absolute (A) and normalized (B) RFD of explosive power athletes (dark bars; n = 9) and controls (light bars; n = 10) during explosive isometric contractions of the knee extensors. Data are mean ± SD. *P < 0.05, **P < 0.01, ***P < 0.001.

When RFD of the explosive contractions was normalised to MVF, the athletes had a greater normalised RFD in the 0-50 ms time window (4.9 ± 1.5 vs. 2.8 ± 1.2 MVF.s^{-1}; P = 0.004; Fig. 4B), and consequently, achieved a greater percentage of MVF after 50 ms (P = 0.004; Fig. 3B). In contrast, the controls had a greater normalised RFD during the 50-100 ms time window (athletes, 6.7 ± 0.9 vs. controls, 7.9 ± 1.1 MVF.s^{-1}; P = 0.02; Fig. 4B), and this resulted in no difference in the percentage of MVF achieved after 100 ms (P = 0.31; Fig. 3B). There was also no difference between the groups in normalised RFD during the 100-150 ms time window. Between trials CV for MVF was 2.3%, and for RFD was 12.8, 5.7, and 12.5% during the 0-50, 50-100, and 100-150 ms time windows, respectively.

Neural Activation during Explosive Contractions

The order of onset of the three agonist muscles was not consistent within or between participants of either group. Nevertheless, the time difference between the EMG onset of the first and last agonist muscle to be activated was shorter in the athletes (7.9 ± 2.4 ms) than the controls (14.4 ± 7.9 ms; P = 0.031), suggesting a greater synchronisation of agonist activation onset in the former.

Athletes had greater normalised EMG during the 0-50 ms time window for each of the three knee extensors (P<0.05) and the mean quadriceps (0.26 ± 0.07 vs. 0.15 ± 0.06 M$_{max}$; P = 0.003; Fig. 5). The mean quadriceps rate of change in EMG was greater in athletes over 0-25 ms (3.4 ± 1.0 vs. 2.0 ± 0.8 M$_{max}$.s^{-1}; P = 0.003; Fig. 6), but was greater in the controls over 25-75 ms (athletes, 0.3 ± 0.5 vs. controls, 1.0 ± 0.2 M$_{max}$.s^{-1}; P = 0.003; Fig. 6). There was no difference between the groups in normalised antagonist (BF) EMG during any of the 50 ms

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time windows (collapsed across the three time windows; athletes, 0.09 ± 0.07 vs. controls, 0.17 ± 0.23 peak RMS; P>0.33).

Athletes had a greater median frequency of the VL EMG signal (P<0.001), but not the RF or VM, during the first 150 ms of the explosive contractions (Fig. 7). Average median frequency of the three superficial knee extensors (mean quadriceps) was also greater in the athletes (P = 0.02; Fig. 7). The between trial CV for mean quadriceps EMG data and median frequency was 12.2% and 9.9%, respectively.

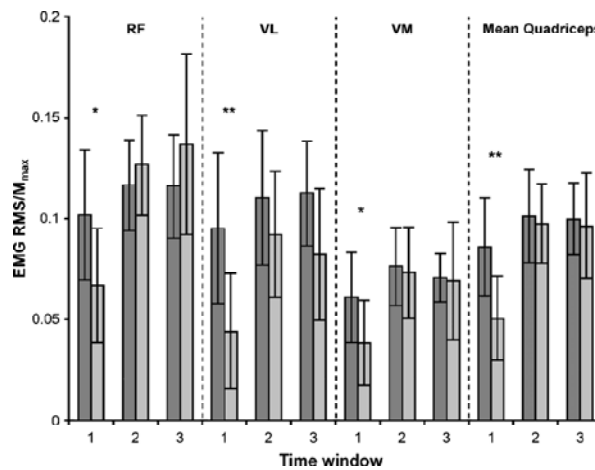

Fig. 5. Neural activation of the RF, VL, VM, and mean of the three muscles (mean quadriceps) of explosive power athletes (dark bars; n = 9) and controls (light bars; n = 10) during explosive isometric contractions of the knee extensors. Neural activation was measured with the RMS of the EMG signal normalized to maximal M-wave (EMGRMS/Mmax) for each muscle and analyzed over 0–50, 50–100, and 100–150 ms (represented with the numbers 1, 2 and 3, respectively) time windows from EMG onset. Data are mean ± SD. *P < 0.05, **P < 0.01.

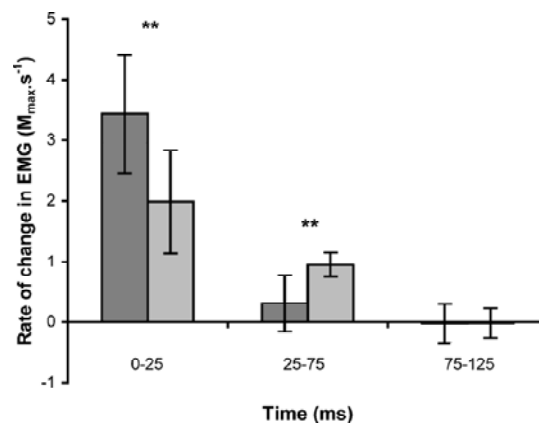Fig. 6. The rate of change in normalized EMG for explosive power athletes (dark bars; n = 9) and controls (light bars; n = 10) during explosive isometric contractions of the knee extensors. The rate of change in EMG was calculated during three time windows (0–25, 25–75, and 75–125 ms) for each muscle (VM, VL, and RF) and averaged for the superficial knee extensors. Data are mean ± SD. **P < 0.01.
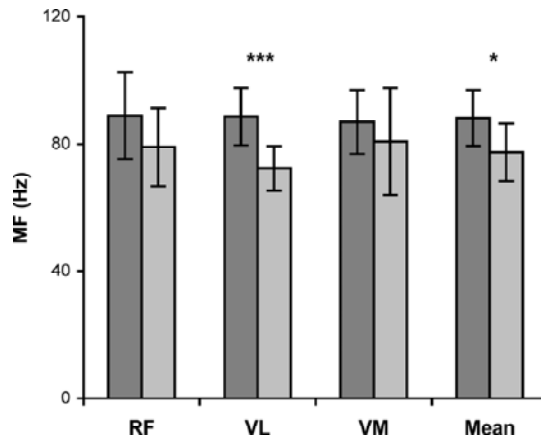
Fig. 7. MF of the EMG signal of the RF, VL, VM, and the mean of these three muscles for explosive power athletes (dark bars; n = 9) and controls (light bars; n = 10) during the first 150 ms of explosive isometric contractions of the knee extensors. Data are means ± SD. *P < 0.05, ***P < 0.001.

Tendon Properties

There was no difference between the groups in peak twitch tension (athletes, 152 ± 22 vs. controls, 131 ± 32 N), time to peak twitch tension (athletes, 90 ± 10 vs. controls, 84 ± 8 ms), absolute RFD (athletes, 2373 ± 285 vs. controls, 2115 ± 572 N.s^{-1}) or relative RFD (athletes, 15.7 ± 0.9 vs. controls, 16.1 ± 1.0 peak tension.s^{-1}) in the first 50 ms of the twitch. Tetanic peak tension was greater in the athletes (388 ± 37 vs. 288 ± 63 N; P<0.001), but was similar once normalised to MVF (athletes, 51.0 ± 2.6 vs. controls, $50.0 \pm 3.4\%$ MVF; P = 0.5). Athletes had a greater absolute RFD in the 0-50 and 100-150 ms periods (P<0.01, Fig. 8A), however, there was no difference between the groups in normalised RFD for any of the time windows (0.27<P<0.50; Fig. 8B). Between trials CV for twitch and tetanic force variables ranged from 2-7%.
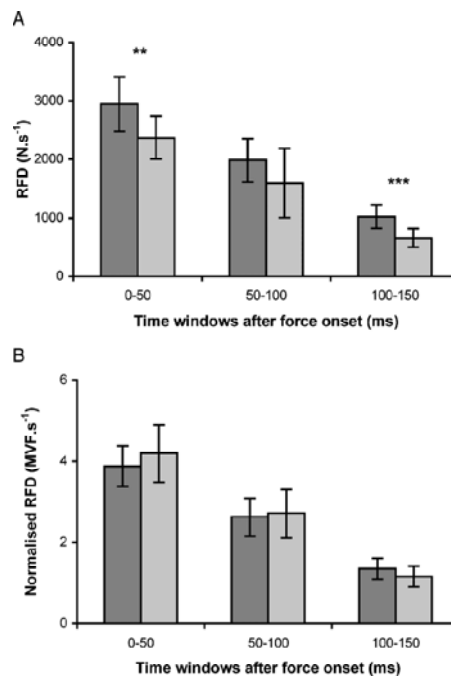

Fig. 8. Absolute (A) and normalized (B) RFD of explosive power athletes (dark bars; n = 9) and controls (light bars; n = 10) during titanic contractions of the knee extensors. Data are mean ± SD. **P < 0.01, ***P < 0.001.

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Discussion

The present study compared EMD, RFD, neural activation and the intrinsic muscle-tendon properties of the knee extensors in explosive power athletes and controls. There were no differences in EMD between the groups, but the athletes had a greater synchrony in the activation onset of the agonist muscles. During voluntary explosive contractions normalised RFD was greater in the athletes from 0-50 ms and higher in controls from 50-100 ms (Fig. 4B). Whilst normalised involuntary RFD was similar for the two groups, the athletes had a greater EMG amplitude in the first 50 ms, and the controls a greater change in EMG amplitude from the first to the second 50 ms time windows. This is the first study to show that discrepancies in voluntary RFD between explosive power athletes and controls appear to be due to differences in neural activation, and not the intrinsic contractile properties of the muscle-tendon unit.

The EMD values recorded in this study (6-15 ms) were relatively low compared to previous studies (14-90 ms, (22, 27, 29, 44, 48, 49, 51)). The long muscle-tendon length, low inertia of the shank, and limited mechanical compliance of our isometric model may explain these short EMD values. Additionally, manual identification of force/EMG onsets has been found more accurate, and detects onsets up to 60 ms earlier than automatic threshold methods (2, 33, 35).

Electromechanical delay is suggested to be an important factor in the performance of explosive sports activities (14). Explosive power athletes have a clear ability for explosive contractions, but no previous studies have assessed whether they have a shorter EMD than normal individuals. The present study observed no differences between the groups for voluntary (absolute and normalised) or involuntary EMD_{max} (Table 1). EMD is thought to be determined by multifactorial aspects of muscle-tendon morphology (e.g., fibre types (39)) and mechanics (e.g., tendon slack (29, 43) and compliance (22)). Type II muscle fibres and tendon compliance may exert opposing effects on EMD, yet both are reported to be greater in the knee extensors of explosive power athletes (23, 26), and could therefore explain the similar group values we have found. The employment of methods such as ultrasonography (7) and muscle biopsies in future studies may help to determine the contribution of morphological and mechanical properties to EMD. Neural activation may also influence voluntary EMD, but similar normalised voluntary EMD_{max} suggests that any differences in neural activation between the two groups did not affect the volitional aspect of the delay. Finally, the relatively high CV for voluntary (12.6%) and involuntary EMD_{max} (10.0%) appear to be an artefact of short EMD values, and may have reduced the likelihood of finding a small, but distinct difference between the groups.

Explosive power athletes had a greater absolute force at 50, 100, and 150 ms (Fig. 3A), due to differences in RFD during the 0-50 ms time window (Fig. 4A). The absolute RFD is known to be related to strength (4), and as athletes in the present study had a greater MVF, differences in absolute RFD could have been entirely due to this discrepancy in strength. However, this was not the case, and normalised RFD was greater in the athletes during the first 50 ms of contraction (Fig. 4B). Other studies have also reported a greater normalised RFD in explosive power athletes when compared to endurance athletes (17) or untrained individuals (42), although, these studies did not investigate the mechanism of any differences. Theoretically the discrepancies in normalised RFD that we observed could have been due to neural activation, and/or the intrinsic muscle-tendon properties.

The magnitude, RFD and time-course of the maximal twitch response were similar for the two groups, and there was no difference between the groups in any of the tetanic RFD variables once they were normalised to MVF (Fig. 8B). This may seem surprising, but whilst some studies have reported differences in maximal twitch characteristics between explosive

power athletes and untrained individuals or endurance athletes (25, 32), others have not (6, 13). We are not aware of any previous studies that have compared the tetanic RFD between explosive power athletes and controls. The similar responses of the groups to evoked twitch and tetanic contractions, suggest that the intrinsic muscle-tendon properties of the knee extensors did not explain the observed differences in voluntary normalised RFD.

In contrast, the athletes had a greater mean quadriceps (agonist) EMG amplitude during the 0-50 ms time window (Fig. 5) and a greater rate of change in EMG from 0-25 ms (Fig. 6). Therefore, whilst there was no difference in antagonist activation, greater agonist activation appears to account for the higher normalised RFD of athletes. In support of this neurological explanation, a number of cross-sectional studies have strongly correlated (r^2 0.75-0.83) agonist activation, measured by preceding surface EMG, with initial torque output (8-10). There is good evidence that explosive ballistic training (presumably similar in nature to the training performed by the athletes in the present study) increases single motor unit discharge frequency and incidence of double discharges in the early phase of an explosive contraction (41), and could well explain the enhanced neural activation we have found. Other mechanisms of greater neural activation might include increased motor unit recruitment (11) and/or synchronisation (47).

In addition to the level of neural activation, the synchrony in activation onset of the agonist muscles may also have influenced initial RFD. The athletes activated all three superficial quadriceps in half the time of the controls, and therefore had more muscles contributing to knee extension force earlier in the contraction. To our knowledge, this is the first study to document this effect, which could be the result of training or innate differences between the groups.

The median frequency of the mean quadriceps EMG signal was greater in the athletes (Fig. 7) during the first 150 ms of the contraction. This was despite use of a relatively broad frequency resolution (6.7 Hz); that was limited by the need to analyse the power spectrum in a short time window (150 ms). These results could be interpreted as greater recruitment of larger fibres and/or type II fibres in the athletes, as these factors are thought to increase conduction velocity and shift the frequency-power spectrum to higher frequencies (11, 36, 45). However, the frequency power spectrum is also influenced by the location of active motor units (relative to the EMG electrodes), and may be only weak-moderately related to conduction velocity during the ascending limb of the force-time curve (12). Nevertheless, the median frequency results of this study provide additional evidence for contrasting agonist innervation of athletes and controls (16).

Neural activation also appears to explain the surprisingly greater normalised RFD in the controls from 50-100 ms. The athletes achieved a near maximum level of voluntary activation in the first 50 ms, leaving less scope for further increases in agonist activation. In contrast the controls had a greater change in EMG from 25-75 ms (Fig. 6), and this appears to explain the greater relative change in force output, during the 50-100 ms time window (Fig. 3B). Therefore, despite the athletes achieving a greater percentage of their MVF at 50 ms (P<0.01; Fig. 3B), there was no difference in the percentage of MVF achieved at 100 ms (Fig. 3B). Past cross sectional studies have only investigated EMG amplitude and RFD over different time phases commencing from EMG/force onset (e.g., 0-30, 0-40, 0-50...0-200 ms, (1, 4, 5, 8-10)). Results of the present study suggest that analysing RFD and EMG over short, consecutive time windows may provide a better understanding of the factors that affect RFD.

The findings of the present study have practical implications for enhancing sports performance and reducing the risk of injury in all populations. The greater force produced by the athletes within 50 ms of contraction onset (absolute RFD, 2.3 fold) was primarily due to their ability to achieve a higher percentage of MVF (1.73 fold) than controls, and this

difference appeared to be due to neural activation. Therefore training for tasks requiring force production over this time period, including joint stabilisation for injury prevention, should focus on developing neural activation. On the other hand, the similarity of normalised RFD and neural activation over time periods greater than 100 ms suggests that tasks requiring force production over this time period should focus on developing strength to attain a greater absolute force.

In conclusion, whilst EMD was similar for both groups, the greater synchrony of activation onset and level of activation of the athletes' agonist muscles during the first 50 ms of explosive voluntary contractions explained their higher normalised RFD. In contrast the controls had a greater increase in activation from 25-75 ms after EMG onset, and higher normalised RFD during the second 50 ms. Coupled with their similar contractile response to involuntary twitch and tetanic contractions, these results suggest that neural activation was responsible for the different pattern of RFD in athletes and controls. Future studies should attempt to establish whether the same discrepancies exist under dynamic conditions.

Acknowledgements

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