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Electromyographic analysis of repeated bouts of eccentric exercise

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The repeated bout effect refers to the protective effect provided by a single bout of eccentric exercise against muscle damage from a similar subsequent bout. The aim of this study was to determine if the repeated bout was associated with an increase in motor unit activation relative to force production, an increased recruitment of slow-twitch motor units or increased motor unit synchronization. Surface electromyographic (EMG) signals were recorded from the hamstring muscles during two bouts of submaximal isokinetic (2.6 rad · s^{-1}) eccentric (11 men, 9 women) or concentric (6 men, 4 women) contractions separated by 2 weeks. The EMG per unit torque and median frequency were analysed. The initial bout of eccentric exercise resulted in strength loss, pain and muscle tenderness, while the repeated eccentric bout resulted in a slight increase in strength, no pain and no muscle tenderness (bout × time effects, $P < 0.05$). Strength, pain and tenderness were unaffected by either bout of concentric exercise. The EMG per unit torque and median frequency were not different between the initial and repeated bouts of eccentric exercise. The EMG per unit torque and median frequency increased during both bouts of eccentric exercise ($P < 0.01$) but did not change during either concentric bout. In conclusion, there was no evidence that the repeated bout effect was due to a neural adaptation.

Keywords: electromyography, median frequency, muscle damage, repeated bout effect.

Introduction

The repeated bout effect refers to the protective effect provided by a single bout of eccentric exercise against muscle damage from a subsequent eccentric bout (Nosaka and Clarkson, 1995). This effect has been demonstrated consistently with repeated bouts of isolated eccentric contractions of the elbow flexors and the knee extensors. Additionally, reduced damage has also been demonstrated with repeated bouts of downhill running, descending steps, downhill walking and eccentric cycling (for a review, see McHugh *et al.*, 1999). Despite studies demonstrating the repeated bout effect, there is little consensus as to the mechanism.

Several authors have suggested that a neural adaptation might explain the protective adaptation (Pierrynowski *et al.*, 1987; Golden and Dudley, 1992; Mair *et al.*, 1994; Nosaka and Clarkson, 1995). In general, these studies have pointed to potential adaptations in motor unit behaviour during a repeated bout that might serve to limit the stress on the activated fibres. High stress on a few active fibres has been proposed as a mechanism of damage (Armstrong *et al.*, 1983; Moritani *et al.*, 1988). Accordingly, Nosaka and Clarkson (1995) suggested that the neural adaptation may 'better distribute the workload among fibres'. This could occur with (1) an increase in motor unit activation relative to force production, as indicated by training studies (Komi and Buskirk, 1972; Hortobàgyi *et al.*, 1996a,b), (2) a change in the motor unit recruitment, as proposed by Golden and Dudley (1992), or (3) increased synchrony of motor unit firing, as suggested by Pierrynowski *et al.* (1987).

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Eccentric strength training studies have consistently demonstrated marked neural adaptations that were noticeably less with concentric or isometric training (Komi and Buskirk, 1972; Hortobàgyi *et al.*, 1996a,b). The eccentric EMG per unit force increased by approximately 20% with 6 weeks of eccentric quadriceps strength training (Hortobàgyi *et al.*, 1996b). In contrast, the EMG per unit force did not change with 6 weeks of concentric training. Similar findings were previously reported for the elbow flexors by Komi and Buskirk (1972), who noted that the greatest increases in EMG occurred after 3 weeks of training, the time at which symptoms of muscle damage had resolved. After 3 weeks of eccentric training, the EMG per unit force had increased by 22% but had decreased by 10% with concentric training. Although these adaptations to eccentric training indicate a decrease in neuromuscular efficiency, they suggest that contractile stresses are distributed among a greater number of active fibres. Such an adaptation to a single bout of eccentric exercise could explain the repeated bout effect.

Several authors have proposed that fast-twitch motor units are preferentially recruited for eccentric contractions (Nardone and Schieppati, 1988; Nardone *et al.*, 1989; Howell *et al.*, 1995; Enoka, 1996). Additionally, fast-twitch fibres appear to be more susceptible to damage with eccentric contractions (Fridèn *et al.*, 1983; Fridèn, 1984; Lieber and Fridèn, 1991; MacPherson *et al.*, 1996). The frequency content of the EMG signal has been used to provide an indirect measure of motor unit recruitment (Moritani and Muro, 1987; Solomonow *et al.*, 1990; Bernardi *et al.*, 1997). A shift towards greater recruitment of slow-twitch motor units may decrease stress on the susceptible fast-twitch fibres and reduce damage from a repeated bout of eccentric exercise. This may also require activating more motor units to match the force production of the fast-twitch motor units.

Motor unit synchronization is the tendency for two or more motor units to fire almost simultaneously more often than might be expected by chance alone (Nordstrom *et al.*, 1990; Kamen and Caldwell, 1996). Pierrynowski *et al.* (1987) suggested that increased synchronization may explain the repeated bout effect. It is possible that increased synchronization during a repeated bout of eccentric exercise would provide a more uniform distribution of contractile stress among the active fibres.

The aim of this study was to compare surface EMG activity between repeated bouts of eccentric exercise of sufficient intensity to demonstrate a repeated bout effect. Specifically, the EMG per unit force and the median frequency were compared between bouts. We hypothesized that the EMG per unit force would be increased during the repeated bout, indicating a greater number of active motor units. Additionally, we hypothesized that the median frequency would be lower during the repeated bout, indicating increased recruitment of slow-twitch motor units. The results of the initial bout were published in a previous issue of this journal (McHugh *et al.*, 2000).

Methods

Experimental design

Two groups of individuals performed repeated bouts of either eccentric (11 men, 9 women) or concentric (6 men, 4 women) hamstring contractions 2 weeks apart. On the first day of testing, baseline measures of isometric strength were recorded before a bout of isokinetic exercise (eccentric or concentric). The participants performed six sets of 10 contractions at a target intensity of 60% of isometric strength. Immediately after the isokinetic exercise, isometric strength was re-tested. On the subsequent 3 days (days 1, 2 and 3), isometric strength, pain and muscle tenderness were assessed. This 4-day protocol was repeated 2 weeks after day 0 (11 days after day 3). The exact time of day of testing was not recorded, but measurements on consecutive days were separated by a minimum of 16 h and a maximum of 32 h.

The procedures for measuring isokinetic exercise, isometric strength tests, pain and muscle tenderness have previously been described (McHugh *et al.*, 2000). Briefly, isometric strength was measured in the seated position with the knee flexed to 0.8 rad. Four maximum efforts of 5 s duration were performed. Peak torque and the total area under the torque–time curve were recorded. The participants then performed six sets of 10 submaximal isokinetic (2.6 $rad \cdot s^{-1}$) contractions (Biodex System 2, Shirley, NY) at 60% of maximum isometric strength. Mean torque and the total area under the torque–time curve were recorded for each set. The intensity for the repeated bout of isokinetic exercise was the same as that for the initial bout (60% of isometric strength before the initial bout, not the repeated bout).

On subsequent days (days 1–3), pain and muscle tenderness were recorded. Pain ratings were recorded on a scale of 0 = 'no discomfort' to 10 = 'walking with a limp'. Muscle tenderness was assessed by pressing a small (18 mm diameter) probe attached to a force transducer into the muscle bellies of the biceps femoris, semimembranosus and the semitendinosus. If the participants reported discomfort at forces less than 40 N, the value was recorded. The tenderness index was calculated as 40 N minus the force at which discomfort was reported. This value was summed for the three muscles.

EMG measurements

During isokinetic exercise, EMG signals were recorded from surface electrodes placed over the biceps femoris, semimembranosus and semitendinosus muscles as described previously. Briefly, three pairs of electrodes were placed 3 cm apart over the respective muscles. The EMG signal was recorded by telemetry, bandpass filtered from 12 to 500 Hz and sampled at a rate of 1000 Hz with a common-mode rejection ratio of 135 dB (Noraxon, Scottsdale, AZ). The amplitude of the raw EMG signal was quantified in two ways. First, the raw signals were fully rectified, integrated (iEMG), summed for the three muscles and then expressed relative to the integrated torque curve for the same contraction. This technique accounts for all activity throughout the contraction and is referred to as EMG per unit torque$_{INT}$ (μV/N·m) to indicate the method of calculation. The second technique for quantifying EMG amplitude was based on the peak EMG and torque values. The peak EMG values for each contraction were recorded by computing the root mean square (RMS) of the raw signal with a 50-ms time constant. The values from each muscle were summed and expressed relative to the respective peak torque values. Therefore, this technique only accounted for the peak values for each contraction and is referred to as EMG per unit torque$_{RMS}$ (μV/N·m). Previous studies have used both RMS and iEMG to quantify activity during eccentric contractions (Komi and Buskirk, 1972; Hortobàgyi *et al.*, 1996a,b). Based on the results of previous work (Komi and Buskirk, 1972), we estimated that a sample size of 20 would be sufficient to detect a 10% increase in iEMG at an alpha level of 0.05 and a beta level of 0.2.

The frequency content of the EMG signal was also quantified by two different techniques, one including whole contractions and the other including only peak values. For whole contractions, sequential 4096 point fast Fourier transforms (FFT) were performed on the raw EMG signals for each of 10 contractions (five FFTs per set) from the first and sixth sets, as described previously (McHugh *et al.*, 2000). The disadvantage of this technique is that low power noise entered into the frequency calculations and signal stationarity was clearly violated. The advantage was that all activity for each contraction was analysed. For analysis of peak EMG activity, 512 point FFTs were performed on the peak activity of the raw signal for each contraction of all six sets. Additionally, the data were windowed with a Hamming window function before performing the FFT to ensure that the first and last points were identical (Kamen and Caldwell, 1996). The median frequency was computed from all FFTs. The results are reported as median frequency$_{4096}$ or median frequency$_{512}$ to differentiate the technique used.

Statistical analysis

Evidence of a repeated bout effect was tested by group (eccentric *vs* concentric) × bout (initial *vs* repeated) × time (pre, post, days 1, 2 and 3) mixed-model analyses of variance on each of three markers of muscle damage (isometric strength, pain, muscle tenderness). Evidence of a neural adaptation was tested by group × bout × set (set 1 to set 6) mixed-model analyses of variance on EMG per unit torque and median frequency. Repeated-measures analyses of variance were used for separate within-group analyses. Greenhouse-Geisser corrections were applied to significant analyses of variance that did not meet Mauchly's sphericity assumption. Probabilities that have been corrected are denoted by the subscript$_{GG}$. All *post-hoc* pairwise comparisons were performed with Bonferroni corrections. Values reported in the figures are the mean ± the standard error of the mean ($s_{\bar{x}}$); those in the text and tables are the mean ± standard deviation (s).

Results

Evidence of a repeated bout effect

Changes in isometric strength clearly demonstrated a repeated bout effect with eccentric exercise but not with concentric exercise (group × bout × time, $P < 0.01_{GG}$; Table 1). There was a significant decline in isometric strength after the initial bout of eccentric exercise (11% on day 1, 8% on day 2, 7% on day 3; time effect, $P < 0.001_{GG}$) and a slight increase in strength after the repeated bout (5% on day 1, 5% on day 2, 8% on day 3; time effect, $P < 0.05_{GG}$). There was no change in isometric strength after the initial or repeated bouts of concentric exercise.

Changes in pain clearly demonstrated a repeated bout effect with eccentric exercise but not with concentric exercise (group × bout × time, $P < 0.01$; Table 2). The participants reported significant pain after the initial bout of eccentric exercise (time effect, $P < 0.001$) but minimal pain after the repeated bout ($P = 0.07$). Minimal pain was reported after the initial bout of concentric exercise ($P = 0.16$) and no participants reported any pain after the repeated bout.

Changes in muscle tenderness clearly demonstrated a repeated bout effect with eccentric exercise (bout × time, $P < 0.01$; Table 2). The participants had significant tenderness after the initial bout of eccentric exercise (time effect, $P < 0.05_{GG}$) but minimal tenderness after the repeated bout ($P = 0.16$). No participant had any muscle tenderness after the initial or repeated bouts of concentric exercise.

Table 1. Isometric knee flexion strength (N·m) before and for 3 days after the initial and repeated bouts of eccentric or concentric exercise (mean ± *s*)

	Eccentric group			Concentric group		
	Initial bout	Repeated bout	Change	Initial bout	Repeated bout	Change
Pre	104 ± 31	114 ± 32	10 ± 10*	117 ± 44	135 ± 57	18 ± 22
Post	107 ± 29	112 ± 31	5 ± 10	125 ± 50	134 ± 54	9 ± 14
Day 1	93 ± 29	114 ± 33	21 ± 17*	122 ± 39	129 ± 48	7 ± 18
Day 2	93 ± 25	119 ± 34	26 ± 22*	120 ± 45	131 ± 55	11 ± 17
Day 3	95 ± 30	123 ± 38	27 ± 25*	117 ± 40	132 ± 53	15 ± 21

Note: group × bout × time, $P < 0.01_{GG}$; * repeated bout > initial bout, $P < 0.01$.

Table 2. Pain and muscle tenderness after the initial and repeated bouts of eccentric or concentric exercise (mean ± *s*)

	Pain (subjective scale 1–10)				Muscle tenderness	
	Eccentric group		Concentric group		Eccentric group	
	Initial bout	Repeated bout	Initial bout	Repeated bout	Initial bout	Repeated bout
Pre	0	0	0	0	0	0
Day 1	2.2 ± 2.5	0.5 ± 1.2*	0.1 ± 0.3	0	1.9 ± 7.4	2.3 ± 7.5
Day 2	3.5 ± 2.8	0.6 ± 1.5*	0.3 ± 0.7	0	6.4 ± 7.4*	0
Day 3	2.6 ± 2.8	0.1 ± 0.4*	0.1 ± 0.3	0	4.7 ± 10.2	0

Note: Pain: group × bout × time, $P < 0.01$; Tenderness: bout × time, $P < 0.01$ (no participants in the concentric group had tenderness after either bout); * repeated bout < initial bout, $P < 0.05$.

EMG activity during repeated bouts

Eccentric contraction intensities, expressed as a percentage of maximum isometric torque, were similar for the initial and repeated bouts of exercise (64.3 ± 4.8% *vs* 66.0 ± 6.9%; $P = 0.11$). Concentric contraction intensities were also similar between the initial and repeated bouts (58.3 ± 1.8% *vs* 58.0 ± 3.8%; $P = 0.79$).

For most analyses, the method of computing EMG per unit torque did not affect the results. However, values calculated from EMG RMS were on average 2.6 times the values calculated from iEMG. There was no difference between the initial and repeated bouts of eccentric exercise (bout effect, $P = 0.73_{INT}$, $P = 0.91_{RMS}$; Fig. 1) or concentric exercise ($P = 0.34_{INT}$, $P = 0.49_{RMS}$). Overall EMG per unit torque was lower for eccentric than concentric exercise ($P < 0.05_{INT\ \&\ RMS}$). During both bouts of eccentric exercise, EMG per unit torque increased from set 1 to set 6 (set effect, $P < 0.01_{GG\ INT\ \&\ RMS}$) but did not change during concentric exercise ($P = 0.34_{GG\ INT}$, $P = 0.4_{GG\ RMS}$). However, the increase in EMG per unit torque$_{RMS}$ during the repeated bout of eccentric exercise was smaller than that during the initial bout (group × bout × set, $P < 0.05_{GG}$). EMG per unit torque$_{RMS}$ increased by 30 ± 37% during the initial eccentric bout but only by 9 ± 23% during the repeated bout; the respective values during concentric bouts were −10 ± 40% and −1 ± 25% (group × bout, $P < 0.05$).

The method of computing the median freqeuncy did not affect the results. However, median frequency$_{4096}$ was on average 9% higher than median frequency$_{512}$. There was no difference in median frequency between the initial and repeated bouts of eccentric (bout effect, $P = 0.73_{4096}$, $P = 0.48_{512}$; Fig. 2) or concentric ($P = 0.29_{4096}$, $P = 0.14_{512}$) exercise. Overall median frequency was higher for eccentric than concentric exercise ($P < 0.001_{4096}$, $P < 0.05_{512}$). During both bouts of eccentric exercise, the median frequency increased from set 1 to set 6 (set effect, $P < 0.001_{4096\ \&\ 512}$) but did not change during concentric exercise ($P = 0.51_{4096}$, $P = 0.65_{512}$). These effects were not different between bouts (group × bout × set, $P = 0.58_{4096}$, $P = 0.83_{512}$).

Discussion

Evidence of a repeated bout effect

Our results indicate that even a relatively low-intensity bout of eccentric exercise – resulting in only moderate symptoms of muscle damage – was sufficient to clearly

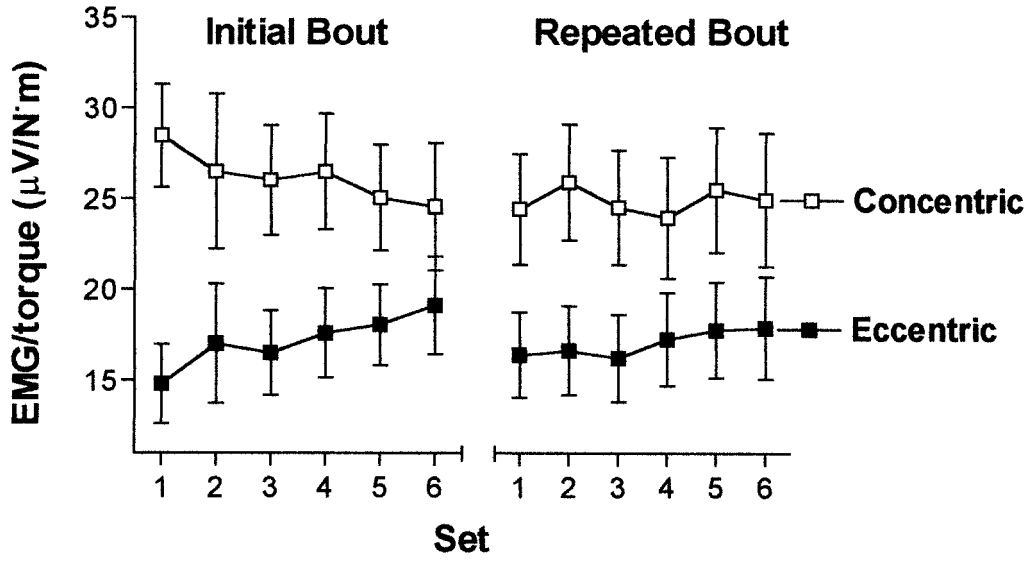

Fig. 1. EMG per unit torque_{RMS} for the initial and repeated bouts of eccentric and concentric exercise (group × bout × set, $P < 0.05_{GG}$). See text for details.

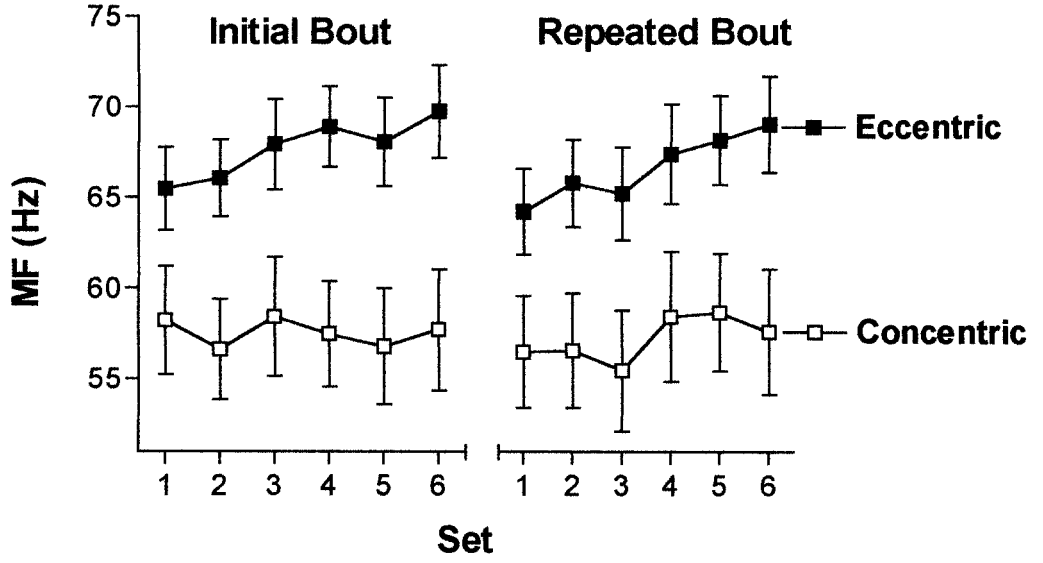

Fig. 2. Median frequency_{512} (MF) for the initial and repeated bouts of eccentric and concentric exercise (group × bout × set, $P = 0.83$). See text for details.

demonstrate a repeated bout effect. A 7–11% decrease in isometric strength after the initial bout of eccentric exercise contrasts with a 5–8% increase after the repeated bout. On the 3 days after the repeated bout, isometric strength was 23%, 28% and 29% higher, respectively, than on the equivalent days following the initial bout. Similarly, the participants had marked pain after the initial bout of eccentric exercise but minimal pain after the repeated bout. Although there was little muscle tenderness after the initial bout of eccentric exercise, no tenderness was reported by any participant on days 2 or 3 after the repeated bout. As expected, there were no symptoms of muscle damage after either bout of concentric exercise.

EMG activity

Despite marked differences in symptoms after the initial and repeated bouts of eccentric exercise, surface EMG activity was very similar between the bouts. The hypothesis that the repeated bout effect is due to an increase in motor unit activation relative to eccentric force production during the repeated bout was not supported by our results. Additionally, no shift to greater recruitment of slow-twitch motor units or increased motor unit synchronization was evident. Therefore, two possibilities exist: (1) the repeated bout effect was not due to any of these neural adaptations, or (2) the EMG techniques used were inadequate to detect such neural adaptations.

Changes in the amplitude of the EMG signal have been attributed to changes in motor unit activation in many studies. Specifically, increases in EMG amplitude with eccentric strength training have been attributed to increased motor unit activation (Komi and Buskirk, 1972; Hortobàgyi *et al.*, 1996a,b). The EMG amplitude has been quantified by RMS (Hortobàgyi *et al.*, 1996a,b) or integration of the rectified signal (Komi and Buskirk, 1972). All three of these studies demonstrated a disproportionate increase in EMG amplitude relative to force production. Hortobàgyi *et al.* (1996b) demonstrated a 50–70% increase in EMG relative to force with 6 weeks of eccentric quadriceps training. However, in the present study, no increase in EMG per unit torque was evident between the initial and repeated bouts of eccentric exercise. Based on the variance in the difference in EMG per unit torque between bouts, a difference of 18% could have been detected, at an alpha level of 0.05 with a beta level of 0.2 (80% power). This effect size was the same for values computed by the RMS and iEMG techniques. The observed difference in EMG per unit torque was less than 3%. Therefore, it is very unlikely that a type II error was committed.

Although there was no evidence of greater motor unit activation for the repeated bout of eccentric exercise, the pattern of EMG activity differed between the bouts. There was a large increase in EMG per unit torque during the initial eccentric bout ($28 \pm 29\%_{INT}$, $30 \pm 37\%_{RMS}$) and a much smaller increase during the repeated bout ($15 \pm 24\%_{INT}$, $9 \pm 23\%_{RMS}$). Typically, increases in EMG amplitude with repeated submaximal contractions indicate increased activation as previously active units become fatigued (Bigland-Ritchie *et al.*, 1986). There is usually a concurrent decrease in the median frequency and force-generating capacity (Hägg, 1992). However, in the present study, we found a concurrent increase in median frequency, while maximum voluntary isometric contractions were slightly higher (non-significant) immediately after eccentric exercise. Additionally, EMG amplitude, median frequency and maximum voluntary isometric contractions were unaffected by concentric exercise. In our previous paper (McHugh *et al.*, 2000), we attributed the concomitant increases in EMG and median frequency during eccentric exercise to additional recruitment of fast-twitch motor units. That a smaller increase in EMG amplitude occurred during the repeated bout indicates that the increase in the initial bout may be related to the damage process. To clarify this issue, *post-hoc* analyses were performed to determine if the increase in EMG per unit torque in the initial bout was related to the strength loss on subsequent days. There was a moderate positive correlation between percent increase in EMG per unit torque_{RMS} and mean percent strength loss ($r = 0.47$, $P < 0.05$), but pain and tenderness were unrelated. It is possible that, as muscle fibres were strained by repeated eccentric contractions, activation increased.

Analyses of the frequency content of the EMG signals also provided no evidence of a neural adaptation with the repeated bout. However, it is questionable whether median frequency is sensitive to changes in recruitment or synchronization between bouts of dynamic exercise. The relationships between recruitment, or synchronization, and the frequency content of the surface EMG signal have typically been based on analysis of isometric contractions (Moritani and Muro, 1987; Hägg, 1992; Bernardi *et al.*, 1997). The confounding effects of factors such as changing muscle length, signal stationarity and movement artefact can obscure analyses of the relevant neural or physiological processes (Kamen and Caldwell, 1996). In our previous paper (McHugh *et al.*, 2000), a large measurement error was reported for median frequency. Therefore, in the present study, median frequency was also computed from 512 point FFTs, which were windowed with a Hamming window function. The purpose of the shorter FFT was to reduce the effects of changing muscle length and the purpose of the Hamming window function was to improve signal stationarity. This technique was successful in reducing the 95% limits of agreement from 14 Hz to 6 Hz for the repeated bouts of concentric exercise. Based on the variance in the difference in median frequency between bouts, a difference of 4 Hz could have been detected at an alpha level of 0.05 with a beta level of 0.2 (80% power). The observed difference in median frequency between eccentric bouts was 1 ± 6 Hz, indicating that a type II error was probably not committed.

The increase in median frequency during eccentric exercise was consistent between repeated bouts, regardless of the method of computation (FFT_{4096} or FFT_{512}). In the initial bout, the median frequency increased by $9\%_{4096}$ and $8\%_{512}$ compared with $11\%_{4096}$ and $8\%_{512}$ in the repeated bout. Additionally, the median frequency was higher during eccentric than concentric exercise

despite lower EMG per unit torque. The differences between contraction types were consistent between bouts and between methods of computation. As we previously reported (McHugh *et al.*, 2000), it is important to note that median frequency and EMG per unit torque were not different between the eccentric and concentric groups during maximum voluntary isometric contractions. The 31–38% lower EMG per unit torque and 16–27% higher median frequency for eccentric contractions are consistent with the theory that fast-twitch motor units are selectively recruited for eccentric contractions (Nardone and Schieppati, 1988; Nardone *et al.*, 1989; Howell *et al.*, 1995; Enoka, 1996).

Conclusions

Our findings support previous work that described the EMG activity for an initial bout of eccentric exercise (McHugh *et al.*, 2000). The symptoms of muscle damage that were evident after the initial bout were absent or reduced after the repeated bout. The present EMG results indicate that the repeated bout effect was not due to a neural adaptation, as measured by surface EMG techniques. Alternative theories for the repeated bout effect include connective tissue (Lapier *et al.*, 1995) or cellular adaptations (Lynn and Morgan, 1994; Pizza *et al.*, 1996; Lynn *et al.*, 1998). However, there is no supporting clinical evidence for these theories.

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