Fatigue Mechanisms in Trained and Untrained Plantar Flexors

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Reference Data

ABSTRACT
The effect of an isometric intermittent submaximal fatigue protocol on the voluntary and evoked contractile properties of 14 trained and 14 untrained plantar flexor muscles was investigated at pre-, postfatigue, and recovery. The greater force output of trained subjects at rest was not attributed to differences in muscle activation or evoked contractile properties. At prefatigue, trained subjects had 40.3% less antagonist EMG activity than untrained subjects, which may have contributed to their increased strength. They also performed more contractions till fatigue and had significantly (p < 0.01) smaller decrements in maximum voluntary contraction force immediately postfatigue. The greater fatigability of untrained subjects was accompanied by significant decreases in M-wave amplitude and peak twitch torque immediately postfatigue. Fatigue-induced decreases in untrained agonist EMG, combined with similar decreases in antagonist EMG for both groups, resulted in relatively less antagonist activity for the trained subjects. It appears that trained-state differences in plantar flexor fatigability are related to peripheral and motor control changes rather than to central impairments.

Key Words: evoked contractile properties, training, muscle activation, co-contractions

Introduction
The response to fatigue can be modified by a variety of factors including muscle type, trained state, duration and intensity of contractions, and whether the contractions are sustained or intermittent (3, 18, 19, 31). Although most everyday activities involve intermittent submaximal contractions, a greater proportion of fatigue studies have focused on the effects of sustained maximal, and to a lesser extent sustained submaximal, contractions. The few submaximal intermittent studies provide conflicting reports on the effects of fatigue on various neuromuscular properties. While Dolmage and Cafarelli (15) reported no muscle inactivation in the vastus lateralis, McKenzie and Gandevia (37) indicated a small degree of central fatigue in both the elbow flexors and diaphragm following a submaximal intermittent fatigue protocol.

Bigland-Ritchie et al. (7) reported no central fatigue in the quadriceps and adductor pollicis muscles, but some central fatigue (maximum voluntary contraction [MVC] less than tetanic force) in the soleus muscle following submaximal intermittent fatigue. Evoked contractile properties such as the compound muscle action potential (M-wave) amplitude have been reported to both potentiate (17), and not change (7) with an intermittent fatigue protocol of the adductor pollicis. Using intermittent submaximal fatigue protocols, an initial potentiation, followed by a depression in twitch amplitude reported by Dolmage and Cafarelli (15), contrasted with the lack of change in twitch amplitude found by Bigland-Ritchie et al. (7). Given the lack of conclusive information in the literature, more research is needed to clarify the fatigue mechanisms associated with intermittent submaximal contractions.

Unlike cardiovascular endurance training adaptations (22), strength training can lead to a relative decrease in the oxidative potential of the muscle (35), potentially diminishing resistance to fatigue (11). Although strength training may increase the number of contractions one can perform at an absolute load (25, 28, 48), the same studies also have reported no change in the relative fatigue or the ability to sustain a percentage of the maximal load.

Other studies have found that strength training can have a positive influence on the muscle's resistance to repeated maximal contractions (36, 45). Increases in time-to-fatigue, smaller losses in force, electromyography (EMG), motor unit discharge frequency, and rates of tension development/relaxation, as well as increases in M-wave amplitude, have been reported following a variety of strength training protocols (16, 26, 52). Some training related fatigue studies, however, have based their results on short-term training programs of 5 weeks to 3 months (16, 45), with the impact of longer training not investigated. Second, most training related fatigue studies have investigated maximal contractions (16, 26, 28, 45).

An investigation of the effects of long-term (years) resistance training may more clearly elucidate the effects of strength training on the ability to sustain a submaximal percentage of the maximum load (relative fatigue). Therefore it was the objective of this cross-sectional study to compare voluntary and evoked contrac-
tile mechanisms associated with the recovery from submaximal intermittent fatiguing contractions of untrained and prolonged-resistance-trained individuals.

**Experimental Design and Methodology**

**Subjects and Experimental Set-up**
The study had 14 resistance trained (21.5 ± 4 yrs, 169.3 ±6.3 cm, 65.4 ± 5.2 kg) and 14 untrained (24.4 ± 5 yrs, 167.9 ± 6.3 cm, 68.1 ± 7.1 kg) subjects with equal gender representation in each group. Trained subjects were college varsity athletes who had been consistently resistance training for at least 2 years and represented a long-term training group (3.8 ± 2.3 yrs of training). All trained subjects had been participating in their off-season training program for at least 3 months, which included maximal strength training techniques (3–5 sets of 3–10 reps at 75–90% of 1-RM 3 days a week) for the plantar flexors. Subjects were recruited from the McGill University staff and student population, were fully informed of the procedures, and signed a consent form prior to the study, which was approved by McGill University’s Ethics Committee.

Subjects were seated in a straight-back chair with hips and knees at 90°. Their legs were secured in a modified boot apparatus with their ankles at 90° (6). All voluntary and evoked torques were detected by a custom-designed force transducer, amplified (recording amplifier and AC-DC differential amplifiers from Neurolog Systems model NL900A), and monitored on an oscilloscope (Tektronix model 2220). All data were stored on computer (Seanim ASI 9000 486 DX) at a sampling rate of 2000 Hz after being directed through an analog-digital board (Lab Master). Data were recorded and analyzed with a commercially available software program (Actran; Distributions Physiomonitor Ltd., Montreal).

Bipolar surface stimulating electrodes were secured to the superior aspect of the gastrocnemius. Stimulating electrodes were crafted in the laboratory from tin-foil, cheesecloth, and paper coated with conduction gel (Aquasonic) and immersed in a saline solution. The electrode length was sufficient to wrap the width of the muscle belly with an electrode width of approximately 4–5 cm. The electrodes were placed in approximately the same positions for each subject. Surface EMG recording electrodes (silver-silver chloride, input impedance 2000 ohms) were placed 3–5 cm apart over the distal segment of the tibialis anterior (TA) and soleus. A ground electrode was secured superficially to the head of the tibia.

Thorough skin preparation for all electrodes included sanding the skin around the designated areas followed by cleansing with an isopropyl alcohol swab. Agonist and antagonist EMG activities were analyzed during MVCs. EMG activity was amplified (isolated head stage 830 amplifier, Biomedical amplifier 830 CWE, Ardmore, PA), filtered (10–1000 Hz), monitored on oscilloscope, and stored on computer. The computer software program rectified and integrated the EMG signal (IEMG) over a 500-ms period during a MVC. M-wave amplitudes elicited by the twitch were measured under the same conditions prior to MVCs at pre- and postfatigue.

**Pre- and Postfatigue Measurements**
Peak twitch torques were evoked with electrodes connected to a high-voltage stimulator (Digitimer stimulator model DS7H+). The amperage (10 mA–1A) and duration (50–100 µs) of a 100-volt rectangular pulse was progressively increased until a maximum twitch torque was achieved. Supramaximal currents were used to elicit the maximal response from the muscle. At prefatigue, the average of 3 trials was used to measure twitch amplitude, time to peak twitch torque (TPT), and half-relaxation time (1/2 RT).

The interpolated twitch technique (ITT) was administered with a series of 3-sec submaximal (80, 60, 40, 20% of MVC) and maximal contractions. Three doublets (2 max evoked stimulations with a 10-µs interval) interspersed at 900-ms intervals were evoked and superimposed on the voluntary contractions to obtain an average response. The doublet utilized the amperage, duration, and voltage of the maximum twitch torque. Only the smallest or occluded superimposed signal was recorded with MVCs (3 trials). Superimposed doublets were used to ensure a large signal-to-noise ratio. Two potentiated doublets were also recorded at 1-sec intervals following the voluntary contractions. Torque signals were sent through both a low- and high-gain amplifier. The resident software program offset and amplified by a magnitude (10x) the high-gain signal for improved resolution.

An interpolated twitch (IT) ratio was calculated comparing the amplitudes of the superimposed doublets with the potentiated doublets to estimate the extent of inactivation during a voluntary contraction. Since the potentiated evoked doublet represents full muscle activation, the superimposed torque using the same intensity of stimulation would activate those fibers left inactivated by the voluntary contraction. The percentage of muscle fibers activated from a single IT ratio can be calculated by subtracting the ratio from a value of 1 and multiplying by 100 to represent an index of muscle activation during a voluntary contraction.

**Fatigue**
After voluntary and evoked testing, the subjects proceeded with the fatigue test. Contraction intensity was gradually increased for 3 sec until 50% of the predicted MVC, calculated from the index of muscle activation, was attained. This intensity was maintained for 10 sec, followed by a 3-sec gradual decrease to a resting state. The sequence was resumed after a 4-sec rest. Contraction cycles (work : rest ratio of 16:4 sec) continued until the effects of fatigue disrupted the subject’s ability to maintain the desired force for the 10-sec period. Volun-
tary and evoked properties were monitored immediately postfatigue and at 30 sec and 1, 2, 5, and 10 min of recovery.

Statistical Analyses
Data were analyzed using a two-way ANOVA with repeated measures on the second factor. The two factors (2 × 7) included trained state and testing period (pre-, postfatigue, and recovery periods of 30 sec and 1, 2, 5, and 10 min). F-ratios were considered significant at \( p < 0.05 \). If significant main effects (see tables) or interactions (see figures) were present, a Tukey post hoc test was conducted. Descriptive statistics in the text include means ± standard deviations (SD). Data in the figures include means ± standard errors (SE). Both average and specific recovery values are presented to highlight general recovery trends and specific differences in the time course of recovery.

Results
At prefatigue, trained subjects exerted significantly more torque than untrained subjects (Table 1). The greater torque output of trained subjects was not accompanied by significant differences between groups in muscle activation (IT ratio), peak twitch torque, and 1/2 RT (Table 1). However, TPT was 11.5% longer in trained subjects. In addition, trained subjects exhibited significantly less antagonist activity than untrained, as evidenced by their 41.3% lower antagonist/agonist IEMG ratio (Table 1).

At postfatigue there was a tendency (\( p = 0.07 \)) for trained subjects to perform more contractions than untrained subjects (75.8 ± 24.9 vs. 51.4 ± 15.8). Furthermore, the untrained subjects had significantly (\( p < 0.01 \)) more decrease in MVC immediately following fatigue (45.9 ± 8.5% vs. 32.3 ± 10.0%) (Figure 1). Although the decrement in MVC continued throughout the recovery period, no significant differences between groups were observed after 30 sec of recovery (Table 2). Fatigue related differences in the number of contractions and torque immediately following fatigue were not associated with differences in the index of muscle activation, antagonist IEMG, TPT, or 1/2 RT. However, both groups had an average 9.9% decrease in the index of muscle activation throughout recovery (Table 2).

In contrast, the IEMG of trained subjects was relatively unchanged from prefatigue following 30 sec of recovery while that of untrained subjects had a mean decrease of 25.9% over the entire recovery period (Table 2). To ensure that changes in soleus IEMG represented the activity of the triceps surae, gastrocnemius IEMG activity was calculated in 5 untrained subjects. Similarly,

![Figure 1. Mean % changes from prefatigue values in MVC of trained (squares) and untrained (triangles) subjects postfatigue and during 10 min of recovery. Vertical arrow = signif. diff. between groups. Horizontal arrow = signif. diff. from prefatigue values. In both figures, statistically signif. diff. calculated with ANOVAs from raw data. Vertical bars = ±SE.](image)

### Table 2
Percent Difference Between Prefatigue & Average Recovery Values in Plantar Flexor Voluntary & Evoked Contractile Properties

<table>
<thead>
<tr>
<th></th>
<th>Trained</th>
<th>( p )</th>
<th>Untrained</th>
<th>( p )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Max voluntary contraction (Nm)</td>
<td>107.1 ±97.7</td>
<td>0.001</td>
<td>91.1 ±21.4</td>
<td></td>
</tr>
<tr>
<td>Index of muscle activation (IT ratio)(^1)</td>
<td>99.5 ±1.1</td>
<td>11 of 14</td>
<td>98.7 ±2.1</td>
<td>11 of 14</td>
</tr>
<tr>
<td>Occurrence of full activation (ms)</td>
<td>12 of 12</td>
<td>11 of 14</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Antagonist/agonist IEMG ratio</td>
<td>0.17 ±0.01</td>
<td>0.001</td>
<td>0.29 ±0.02</td>
<td></td>
</tr>
<tr>
<td>Twitch torque (Nm)</td>
<td>9.6 ±3.7</td>
<td></td>
<td>7.7 ±1.7</td>
<td></td>
</tr>
<tr>
<td>Time to peak twitch torque (ms)</td>
<td>141.9 ±16.7</td>
<td>0.001</td>
<td>125.4 ±19.3</td>
<td></td>
</tr>
<tr>
<td>Half relaxation time (ms)</td>
<td>93.1 ±16.9</td>
<td></td>
<td>92.7 ±12.9</td>
<td></td>
</tr>
</tbody>
</table>

Note. Mean values (±SD) averaged over entire recovery period (30 sec, and at 1, 2, 5, & 10 min of recovery); IT ratio = 1 × 100; \(^1\)Represents a ratio and not a % of prefatigue values.

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\(^1\)IT ratio = 1 × 100, prediction of muscle activation derived from equation: \( y = a + bx + cx^2 \).
medial and lateral gastrocnemius IEMG activity had an average decrease over the entire recovery period of 22.9% (±8.1) and 20.7% (±11.6), respectively.

Although there were no group differences in the 31.3% decrease in antagonist IEMG activity, trained subjects had a 58% lower antagonist/agonist IEMG ratio (p < 0.001), indicating relatively less antagonist activity in trained subjects throughout the recovery period.

Similar to IEMG findings, untrained subjects had an average 31.2% decrease in M-wave amplitude during the recovery period whereas no change in the trained M-wave was observed (Table 2) (p < 0.02).

Significant differences between trained and untrained (IEMG, M-wave) subjects continued (p < 0.0001), with trained subjects experiencing a twitch potentiation of 14.7% in the testing procedure immediately postfatigue while untrained had a decrease of 10.6% (Figure 2). There were no significant differences between groups for the remainder of the recovery period. Nor were there any differences during recovery in TPT or 1/2 RT (Table 2).

In order to further specify the locale of fatigue in the plantar flexors, 5 more untrained subjects were subjected to 100-Hz max tetanic stimulation pre- and postfatigue. The 5.4% (±3.9) decrease in tetanic torque postfatigue was less than the decrements in twitch torque (10.6%) or MVC (45.9%).

Discussion

The greater torque of trained subjects at prefatigue was not accompanied by significant differences in muscle activation (IT ratio), peak twitch torque, or 1/2 RT. Sale et al. (47) reported no difference in knee extensor motor unit activation with the ITT after 19 weeks of weight training. Some short-term training studies (5–8 weeks) have failed to demonstrate increases in neural drive as evidenced by a lack of change in EMG with isometric (12, 20), eccentric, and concentric resistance training (32). Rutherford and Jones (46) suggested that strength improvements in the early part of a training program were largely due to alterations in the activity of antagonist muscle groups and an increased ability to coordinate other synergistic and stabilizing muscle groups. Although the greater strength of trained subjects could not be attributed to a greater neural drive or improvements in evoked contractile properties, trained subjects did exhibit 41.3% less antagonist activity.

Antagonist co-contractions function to provide protection from the inertial forces of the agonist (4, 50, 51) and improve the ability to target forces (23). Decreased antagonist activity would result in less resistance to the intended agonist forces. Trained individuals may have a greater ability to inhibit excessive antagonist contractions contributing to the net force output of the agonist muscle. Training-induced decreases in antagonist activity have been confirmed both in cross-sectional studies involving dynamic contractions (44) and longitudinal isometric training studies (13). Although muscle hypertrophy probably exerts a more significant influence, changes in motor control by decreasing antagonist activity could contribute to the greater agonist forces of trained individuals.

Although trained subjects showed a trend toward superior fatigue resistance as evidenced by the greater number of contractions to fatigue and a significantly smaller decrease in MVC immediately postfatigue, there was no significant difference between groups in the index of muscle activation. Both groups had significant muscle inactivation following fatigue (9.9%). Fatigue-induced muscle inactivation is not a consistent finding in the literature. Studies that have examined fatigue-related neural adaptations with the IT ratio following fatigue have reported either no (38, 53) or some evidence of muscle inactivation (37, 43). Inconsistent findings in the literature may be related to the sensitivity of the interpolated twitch technique (5), type of muscle, or population tested.

Surprisingly, although both groups had declines in muscle activation, only the untrained group had significant decreases (25.1%) in agonist IEMG activity. The more significant decreases in the IEMG of untrained subjects is similar to the findings of Rube and Secher (45), who reported smaller decreases in EMG activity following isometric MVC leg extension training. Other researchers have reported unaltered IEMG activity and smaller changes in frequency and duration of motor unit spikes following fatigue in endurance-trained individuals (26, 34, 52). The decrease in untrained IEMG activity may be related to changes in the M-wave amplitude.

Indeed, decreases in the M-wave amplitude (32.1%) were observed in the untrained group whereas no significant change occurred in the trained subjects. A reduction in M-wave amplitude or area may signify im-

![Figure 2. Mean % changes in peak twitch torque of trained (squares) and untrained (triangles) subjects pre/postfatigue and during 10 min of recovery. Vertical arrows = signif. diff. between groups. Vertical bars = ±SE.](image-url)
pairment in neuromuscular propagation and/or muscle membrane excitability (9). Many studies using repetitive MVC to induce fatigue have reported decreases in M-wave amplitudes (10, 33, 39, 40). Bigland-Ritchie et al. (7) did not find any significant change in the M-wave amplitudes of the adductor pollicis in their intermittent submaximal fatigue study. Decreases were reported by Garland et al. (21) with 15-Hz tetanic fatigue of the dorsiflexors. Duchateau and Hainaut (17) examined continuous and intermittent 30-Hz tetanic fatiguing contractions of the adductor pollicis, reporting an increase in the amplitude of the surface action potentials.

Potentiation of the M-wave may signify that presynaptic and/or end-plate potentials are facilitated possibly by a reduction in the dispersion of fiber action potentials (17). Thus one factor contributing to fatigue related differences would be the greater impairment in neuromuscular propagation in untrained subjects. Since both trained and untrained subjects had similar muscle inactivation, trained-state differences in IEMG activity would more likely be related to the reduction of untrained M-wave than to decreases in neural drive.

Another factor contributing to fatigue related trained-state differences would be alterations in evoked contractile properties. The peak twitch torque of untrained subjects decreased 10.6% immediately following fatigue, while for trained subjects it increased 14.7%. Both potentiation and impairment of the twitch amplitude following fatigue have been documented in the literature. Studies have demonstrated potentiation of twitch amplitudes with submaximal (15), short-term (10-sec), maximal (24, 27), and electrically induced contractions (49). Depression of twitch torque is evident with sustained (24, 27, 37) and intermittent maximal (8) as well as intermittent submaximal contractions (53).

In order to ascertain whether excitation-contraction (E-C) coupling was actually impaired in the untrained subjects, 5 more untrained subjects were subjected to 100-Hz maximal tetanic stimulation before and after the fatigue protocol. A smaller deficit in tetanic (5.4%) vs. twitch torque (10.6%) in addition to decrements in M-wave amplitude following the submaximal fatigue protocol would suggest that the trend for greater muscle fatigue in untrained subjects could be related to impairments in E-C coupling.

There were no trained-state differences in the relative drop in antagonist IEMG activity following fatigue. With data collapsed, tibialis anterior IEMG activity significantly decreased 31.3% following the fatigue protocol. However, the ratio of antagonist/agonist IEMG activity was 58% lower in trained individuals. Although trained and untrained subjects had similar decreases in antagonist EMG activity, the 25.9% decrease in untrained agonist activity (Table 2) would result in relatively greater antagonist activity for the untrained. Similar to prefatigue findings, motor control changes in the form of relatively lower antagonist activity could contribute to the greater fatigue resistance of the trained subjects.

Summary

Similar to a myriad of other fatigue studies, this one illustrated that a diversity of factors can contribute to the decrease in performance associated with fatigue. In the present study, trained subjects experienced less fatigue (MVC decreased 32%) with only a small decrement in muscle activation (9.4%) and a lack of impairment in EMG, neuromuscular propagation, or E-C coupling. In addition, a lower ratio of antagonist to agonist IEMG activity both pre- and postfatigue could have contributed to the greater endurance of trained subjects.

Although the postfatigue drop in MVC was less for trained subjects, the energy demands of sustaining greater absolute forces would still place considerable stress on the trained muscle. On the other hand, untrained subjects experienced a spectrum of impairments affecting muscle activation, EMG activity, membrane potentials, and E-C coupling. This would suggest that trained subjects may not experience the same extent of fatigue related widespread disruptions of voluntary and evoked contractile properties as untrained individuals.

The significance of the present study lies in its investigation of fatigue related trained-state differences. Although a number of studies have documented trained-state differences under resting conditions, there is a dearth of research examining trained-state differences postfatigue. The present study provides new or corroborating evidence in the following areas:

1. Whereas two studies (30, 46) found an improved ability to fully activate muscles after training, the present study found no trained-state differences in fatigue-induced muscle inactivation.
2. The present study further substantiates the findings of greater decreases in postfatigue EMG (29, 34) and M-wave amplitude (16) of untrained subjects.
3. Although other studies have reported either increases (16) or no change (1, 2, 14, 36) in peak twitch amplitude with training under resting conditions, the present study found a greater depression of peak twitch amplitude at postfatigue in untrained subjects.
4. Similarly, while some studies found lower antagonist EMG activity in untrained individuals under resting conditions (13), the present study found dramatically less antagonist activity in trained subjects following fatigue.

Practical Applications

A lack of trained-state differences in muscle activation and greater relative antagonist activity in untrained individuals at pre- and postfatigue provides a physiological basis for strength training recommendations for untrained individuals. The lack of differences in muscle activation indicates that the use of specific training strategies to enhance muscle activation early in the training
program are not of primary importance. Maximal strength training techniques that emphasize neural adaptations (54) may not provide a significant advantage over lower intensity training to support their inclusion in the early stages of a resistance training program.

Eccentric contractions have been reported to alter the orderly recruitment of motor units (slow to fast twitch), resulting in the activation of higher threshold motor units prior to lower threshold motor units (41). Considering the increased incidence of delayed onset muscle soreness with eccentric contractions (42), the discomfort of primarily eccentric or negative training would not provide added benefits in terms of increased muscle activation early in a training program.

However, this study does indicate that motor learning strategies to decrease antagonist co-contractions could be an important component of strength adaptations. The use of compound muscle exercises calls for the coordination of agonist, antagonist, and synergistic muscles. The inclusion and emphasis of compound exercises with free weights would enhance the motor learning component of strength development through the efficient activation, coordination, and balance of multiple muscle groups.

References
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