Neuromuscular adaptations to training

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CANNON, R. J., AND E. CAFARELLI Neuromuscular adaptations to training. J. Appl. Physiol. 63(6), 2396-2402, 1987.—The purpose of this experiment was to determine whether there is a central adaptation to resistance overload. The right adductor pollicis muscle of each subject was trained with either voluntary \((n = 9)\) or electrically stimulated contractions \((n = 7)\), the contralateral muscle acted as an internal control, and seven other subjects acted as a control group. Training was the same in both groups: 15 contractions at 80% maximal voluntary contraction \((MVC)\), 3 days/wk for 5 wk. Trained muscles in both groups increased \(MVC\) by \(\sim 15\%\) (voluntary, \(P < 0.01\); stimulated, \(P < 0.05\)). There was a small \((9.5\%)\) but significant \((P < 0.05)\) increase in MVC of the untrained muscles in the voluntary group. MVC did not change in the control group. Maximum electromyogram \((EMG)\) was highly reproducible pre- to posttraining in the trained groups. Sensory adaptation to training caused a reduction in force sensation in the stimulated group \((P < 0.05)\) but not in the voluntary group. Because there was a small increase in MVC of the untrained muscle of the voluntary group \((9.5\%, P < 0.05)\) but not in the stimulated group, it is possible that there is a central motor adaptation, but it is not manifested in increased neural drive \((EMG)\). Moreover, this central adaptation may be responsible for the decrease in force sensation that follows training.

Like many exercise-induced adaptations, those that occur in the motor systems are thought to occur both centrally and peripherally \((9, 13, 14, 17, 21, 29, 30)\). In the neuromuscular system it is convenient to think of everything distal to the neuromuscular junction as peripheral and everything proximal to it as central. The neuromuscular junction itself does not always fit conveniently into this scheme, but muscle tissue and the muscle mechanoreceptors are clearly peripheral. When challenged by a resistance overload the contractile proteins proliferate, and the muscle becomes capable of a greater response to any level of neural activity \((13, 14, 25)\). It has also been reported that the maximal surface electromyogram \((EMG)\) increases during training \((21, 30)\). This finding has been interpreted as an increase in central neural drive that enhances maximal-force production in some unspecified way.

It has further been suggested that sensory adaptations to training occur centrally and peripherally. Some of the earliest discussions of the so-called "muscle sense" refer to a central feedforward loop that sends a copy of the motor output directly to the sensory cortex \((1, 33, 34)\). There is considerable experimental evidence that is consistent with this hypothesis \((6, 9-12, 27)\). However, it is also apparent that because muscle is so well supplied with sensory receptors, it is quite capable of sending information back to the brain from the periphery \((5, 9, 16, 26)\). Although these central and peripheral pathways are available, little is known about the relative contribution to force sensation during static contraction and how each adapts to an increase in force-producing capacity.

The primary purpose of the present experiment was to determine whether central sensory adaptations occur in response to chronic resistance overload. Central and peripheral adaptations were uncoupled by training one group of subjects with normal voluntary contractions and another group with electrically stimulated contractions. Electrical stimulation does not involve the normal efferent pathways, particularly the higher centers for neuromuscular control where the central feedforward mechanism resides \((27, 34)\). Thus there would be no control adaptations of the sort that would occur from initiating voluntary movement.

Secondary purposes were to determine whether there is an increase in maximal neural drive after training or whether the frequency distribution of the surface \(EMG\) adapts to resistance training. Strength training increases the degree of synchrony in motor-unit recruitment patterns, and increased synchrony is a central adaptation that would increase the electrical energy in the low end of the frequency spectrum \((3)\). Muscle trained with electrical stimulation would lack a central adaptation in its neural activating mechanism.

METHODS

Subjects. Twenty-three paid volunteers gave their informed consent and were assigned to one of three groups. 1) VOL \((n = 9)\), trained by making \(15 \times 80\%\) maximal voluntary contractions \((MVC)\) 3 days/wk for 5 wk; 2) STIM \((n = 7)\), underwent the same training as VOL except that contractions were elicited by direct neural stimulation; and 3) CONT \((n = 7)\), control group, did not train.

Force measurement. All measurements were made from the adductor pollicis muscle of each hand. The dynamometer for measuring force in this muscle was adapted from Merton \((28)\). The hand was placed in the apparatus with the palmar surface facing medially, and
the limb was restrained as shown in Fig. 1. A loop was passed over the thumb and then attached to a rigid steel bar to which four strain gauges were bonded. Force applied to the bar produced a proportional voltage change that was amplified (×10) and then recorded simultaneously on FM tape (Vetter, model 1D) and on a chart recorder (Health/Schlumberger, model SR206). Contractile force was read from the paper records and expressed as a percent of pretraining MVC force (2, 28).

**Electromyography.** Electrical activity of the adductor pollicis muscle was picked up with a unipolar arrangement of silver-silver chloride electrodes (Beckman Instruments, mini-electrodes). The active electrode was attached over the muscle, and the indifferent electrode was on the dorsum of the second finger. A plate electrode on the upper arm served as ground. Electrode placement was constant throughout the experiment.

The EMG interference pattern was displayed on an oscilloscope and simultaneously full-wave rectified, smoothed (time constant = 200 ms), and then written out on a chart recorder. In addition, the EMG was recorded on FM tape for later frequency analysis. The averaged amplitude of the smoothed, rectified, EMG signal was read from the chart recordings and expressed as a percent of the EMG measured at MVC.

The EMG frequency distribution was estimated by passing the signal through band-pass filters appropriate to the general distribution of frequencies in the EMG (3, 24, 31). The taped signal was first high-pass filtered (16 Hz cut off), and then fed through a low-frequency band-pass filter (24-47 Hz at −3 dB) and a high-frequency band-pass filter (140-360 Hz at −3 dB). Roll off for each was −20 dB/octave. The output of the filters was rectified, and the amplitude was proportional to the electrical power contained within each bandwidth. This technique approximates the total frequency distribution of the EMG, and changes in the ratio between the high- and low-band-pass filter outputs indicate shifts in the distribution (3, 24, 31).

**Force sensation.** We used a technique to measure force sensation that was modified from Goodwin et al. (16) and has been described previously (5–7). Briefly, sensation was measured by matching force sensation in a reference muscle with a contraction of the contralateral muscle that felt like the same force. The right adductor pollicis was always the reference muscle, and the left adductor pollicis was always the matching muscle. The subjects were seated in front of the dynamometers as shown in Fig. 1, and a variable resistance meter driven by amplified output of right strain gauge. Subject’s task during sensation measurements was to drive display indicator to center mark and simultaneously make a contraction of opposite hand that felt like the same force. Force required to move indicator depended on input resistance of display, which was controlled by experimenter. Force produced by left muscle was measured for force sensation in right muscle. (Modified from Ref. 5.)

**Electrical stimulation.** A saline-soaked stimulating electrode (DISA) with the anode placed distally was located directly over the ulnar nerve in the olecranon notch. Shocks were delivered at 1 Hz and 50 μs duration, and the voltage was turned up until a discernable twitch appeared in the force record. Next, the position of the stimulating electrode was manipulated until the twitch was maximal for this stimulus voltage. The voltage was then gradually increased until the twitch amplitude stabilized and was then increased an additional 10% to ensure maximality (8). Training consisted of fusing this pulse at 50 Hz and producing a contraction of ~80% MVC for 3–4 s.

**Protocol.** Subjects practiced all procedures before beginning the experiment. To begin, each was positioned in the dynamometer and measurements of limb position were recorded. The thumb was extended and abducted so that muscle length and tension was the same on both sides. The angle between thumb and first finger was always 90°.

Once in the dynamometers, the subject made three or four brief MVC’s, the largest of which was recorded as MVC for that day. Contractions of 25, 50, and 80% MVC of the right (reference) adductor pollicis were then matched with contractions of the left adductor pollicis. Reference forces were repeated three times, and the average of the three matching forces was the control force sensation. Finally, subjects made a static contrac-
tion of 80% MVC and held it until it declined to 50% MVC. Both force and EMG were recorded continuously during this procedure and stored on FM tape. Pre- and posttraining measurements were identical.

During the training sessions the VOL group subjects made 80% MVC contractions lasting 3–4 s at ~5 min−1. A 2-min rest period followed after the first and second group of five contractions. The training procedure for the STIM group was identical except that contractions were electrically stimulated through the ulnar nerve as described above.

Descriptive statistics (means ± SE) were used to describe all of the data. A two-way analysis of variance with repeated measures was used to test the effect of training on each variable under all experimental conditions (20). Duncan's multiple range test was used for post hoc analysis when significant main effects were found. The probability level of at least 0.05 for statistical significance was chosen a priori.

RESULTS

Effect of training on MVC. Figure 2 shows the effects of 5 wk of resistance training on adductor pollicis MVC. Both VOL and STIM groups increased MVC by 15% (P < 0.05), but there was no significant difference in the CONT group MVC after training. The degree of adaptation was similar in the VOL and STIM groups because the intensity of the training stimulus was the same.

An interesting observation was made in the untrained contralateral muscles after training. There was a 9.5% increase in MVC (P < 0.05) in the untrained adductor pollicis of the VOL group (Fig. 3). This finding is reminiscent of the so-called cross-over effect that has been described previously (17). In contrast, the untrained muscle in the STIM group did not show a significant increase in MVC after the training period. There was no change in MVC in either muscle of the CONT group.

Effect of training on neural activation. To determine whether EMG increased after training, it was first necessary to demonstrate that it was repeatable before and after the training period. The absolute amplitudes of the smoothed rectified EMG at forces varying from 25 to 100% of the MVC in the CONT group before and after training are plotted in Fig. 4. Correlation and regression analysis of the difference between pre- and posttraining measures showed that the slope of the relation was not significantly different from 1.0 and the intercept was not
different from zero. This demonstrates that the EMG was satisfactorily repeatable under the conditions of the experiment (21, 30).

Figure 5 shows the absolute EMG plotted as a function of force relative to the pretraining MVC before and after the training period. There are no statistically significant differences in EMG amplitude between any of the groups that can be attributed to the effects of training. It took the same degree of neural activation to generate a given amount of force after 5 wk of resistance training, despite the fact that the VOL and STIM groups showed a 15% increase in MVC. Thus we have no evidence that neural drive to skeletal muscle increases as part of the adaptation to chronic resistance overload.

The length of time the subjects were able to maintain static force above 50% MVC was not altered by resistance training in any of the groups (pre = 41.8 ± 1.5 s, post = 44.6 ± 2.0 s). Because the contraction started out at such a high intensity, EMG did not increase after 10 s but decreased in parallel with force (2). Thus the ratio between EMG and force remained at ~1.0 for the duration of the contraction (Fig. 6).

The frequency distribution represented by the ratio of the outputs of the high- and low-band-pass filters during the maintained contraction is shown in Fig. 7. These data are collapsed across conditions because there were no significant differences in the ratio of high- to low-band-pass outputs (H/L) among the groups. The data shown in Fig. 7 have been adjusted for EMG amplitude, which fell throughout the contraction (1). The initial value for H/L was taken from the first 3 s of the pretraining-maintained contraction. There was no change in the time course of H/L after training.

Effect of training on force sensation. An adaptation of the muscle sensory system to an increase in effector capacity would be expected to cause a decrease in force sensation for any absolute force. To determine that this occurred, the difference in matching force between before and after the training period was plotted as a function of the relative pretraining force (Fig. 8). Expressing the data like this makes it possible to see the reduction in sensory intensity at the same proportion of the total force-producing capacity. The most dramatic change occurred in the STIM group where the matching force after training declined as a function of the actual force generated. There was no significant change in force sensation in the CONT group after the training period. The VOL group showed no decrease in force sensation despite the fact that training produced a 16% increase in MVC force. This contrary finding is undoubtedly the result of the 10% increase in MVC that occurred in the untrained hand. Because the matching muscle got stronger, there was a change in how it sensed force itself, and thus the instrument with which we measured force sensation changed calibration.


discussion

After 5 wk of training we found that although adductor pollicis MVC increased, the maximal EMG and submaximal force sensation were unchanged from control. The untrained adductor pollicis of the trained subjects showed a significant increase in MVC but no change in maximal EMG. When training contractions were elicited by neural stimulation rather than by voluntary effort,
MVC increased, force sensation decreased, and maximal EMG remained the same. Neither of the training methods used in this experiment had any effect on the frequency distribution of the EMG.

**Increased force-producing capacity.** The primary adaptation associated with chronic resistance overload is an increase in the size of muscle cells due to a proliferation of contractile proteins (10, 14, 15, 25). The increase in maximal force-producing capacity seen in a hypertrophied muscle is therefore secondary to an increase in cross bridges available for interaction (13). Aside from this there seems to be no mechanism in skeletal muscle that accounts for increased force. However, the relationship between muscle size and the maximal force that a muscle can produce has not been completely verified by soft-tissue-imaging techniques such as ultrasound or computer-assisted tomographic scanning. For example it has not been shown that in the early stages of training an increased MVC is associated with a proportional increase in cross-sectional area (21, 30). These data and the apparent cross-over effect observed by us and by others have been interpreted as a central adaptation within the neuromuscular system (21, 30). In contrast, others have concluded that there are no peripheral physiological changes that can be observed in untrained contralateral muscles after a period of unilateral training (14). In light of the present experiment, we would agree with this dissenting view because although we observed an increase in MVC of the untrained contralateral muscle in the VOL group, we did not see an accompanying increase in maximal EMG (see following section). Thus it is likely that when the so-called cross-over effect is observed it is either because the pretraining MVC's were not truly maximal or because learning has occurred proximal to the final common pathway.

**Maximal neural activation.** Several factors such as electrode placement, fatigue, and possibly fiber size can influence the amplitude of the surface EMG. In the present study we exercised considerable care to avoid these effects. Electrode positions were marked on the skin so that they could be exactly repeated between experiments. Fatigue is associated with numerous physiological adjustments such as changes in firing frequency as well as fluid and electrolyte shifts that can markedly change the ratio between force and electrical activity (2). We controlled for these effects by using contractions of very short duration with rest periods between each. The effectiveness of these measures was confirmed by our observations that the EMG amplitude for any force was repeatable before and after the training period. The question of how fiber hypertrophy might alter the surface interference pattern is not so straightforward. Although the sizes of an action potential from a single fiber may be proportional to the mass of the fiber, the pick-up field of the recording electrodes remains the same. Thus it is not clear exactly what would happen to the surface EMG. Our findings that there was no increase in maximal EMG when there must have been significant hypertrophy would suggest that hypertrophy alone does not influence the surface EMG.

Our EMG data suggest that there is no change in maximal neural activation as a result of training. Although some aspects of neuromuscular activity adapt to resistance overload (29) there is no evidence to suggest that increased force is due to increased neural drive. The amplitude of the smoothened rectified surface EMG is a function of the number of active motor units and their frequency of activation (2). All motor units are maximally active during MVC, and the number of fibers in a single muscle does not increase after the intense training (15, 25, 28). The only mechanism available that could account for the increase in both MVC force and the amplitude of the surface EMG is the activation frequency of the motor-unit pool. However, it is well known that increasing stimulation frequency does not alter MVC (28). Some recent findings showing changes in neural drive have been confounded by inconsistencies in the data collected from trained and untrained limbs of the same subjects. For example, Moritani and deVries (30) found a significant increase in hiceps EMG after only a few weeks of training. However, they reported an increase in maximal EMG in the untrained, contralateral limbs of trained subjects that was nearly twice the increase in maximal EMG of the trained muscle (see Tables 1 and 2 in Ref. 30). They also reported that the combined increase in maximal EMG and muscle girth measurements only accounted for half the increase in MVC (see Table 1 in Ref. 30). In a similar study Komi and colleagues (21) trained six pairs of twins. They reported a 20% increase in quadriceps MVC and a 38% increase in maximal EMG (see Table 2 in Ref. 21). This means that they are reporting a change in neural activation that is twofold greater than the increased force output. Moreover, the subjects of Komi et al. (21) were adolescent males and females who grew an average of 2.3 ± 0.6 cm during the 12-wk training period. These inconsistencies do not permit the conclusion that increased neural acti-
vation increases force production after training. We would therefore suggest that our data reveal not some unspecified increase in neural drive but rather a more responsive group of hypertrophied muscle cells.

**EMG frequency response.** Several investigators have argued that the downward shift in the frequency distribution of the EMG is due to a slowing of conduction velocity in the sarcolemma and the T-tubular system (4, 22, 24). Others have suggested that it is related to the metabolic state of the muscle or to the degree of synchrony of motor-unit firing (3, 21, 24, 30). Indeed, conduction velocity slows during fatigue, motor-unit firing frequency slows during submaximal and maximal contractions, and the degree of synchrony increases with fatigue (2, 24, 31). We assume that all these changes occurred during the maintained 80% MVC contractions, and hence it is not possible to make inferences about the specific reason for the decline of H/L. Nevertheless, our purpose was to determine whether resistance training had any effect on H/L behavior during a high-intensity contraction. Since the training regimen did not alter our measurement of H/L we conclude that this parameter does not adapt to resistance overload.

**Force sensation.** Although it is generally accepted that any given force feels like less in a trained muscle, there are no data in the literature that would verify this phenomenon. In the present study there was no change in force sensation for any given force in the VOL group. This occurred because there was a significant force increase in the contralateral muscle. Thus any change in force sensation that would have occurred after training was offset by a shift in sensation of the matching muscle; in short, there was a change in calibration. In comparison, there was no such increase in MVC in the untrained muscle of the STIM group, and force sensation declined for any force after the training period. We suggest that these data are evidence for a decline in force sensation after training. This conclusion is congruent with other findings showing that muscles weakened by disease are more sensitive to a given tension (10–12, 16). Similarly, when muscles are acutely weakened such as they are when fatigued (7, 10, 19, 27) or when contracting at nonoptimal starting lengths, force sensation is amplified (6). When the weakening agent is removed, or when the muscle recovers from fatigue, force sensation declines in complementary fashion (6, 10).

The mechanism responsible for these adjustments in force sensation is not known. Although it is generally accepted that force sensation depends on both central and peripheral inputs, the adaptation of these pathways to acute and chronic changes in force-producing capacity are not understood. However, it is clear that in a weakened muscle the sensation of force for any given force is increased and in a strengthened muscle the sensation of force for any given force is attenuated. There are apparently no physiological or morphological changes in muscle receptors after training (26), but the increase in total proteins would mean that the density of receptors per unit volume becomes less. Moreover, in acute muscle weakness there is no change in receptor density (26), but under some experimental conditions receptors such as muscle spindles may be excited so that force sensation is uncoupled from actual force production (5, 7). Thus, although muscle receptors are not totally necessary for changes in force sensation, they are clearly sufficient to signal it on their own.

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