Change in intramuscular and intermuscular neural adaptation after resistance training in trained college athletes

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Abstract: The purpose of this study was to investigate the intramuscular and intermuscular neural adaptation after resistance training in trained college athletes. The results indicated that, progressive resistance training significantly ($P<0.01$) increased MVC and integrated electromyography (IEMG) of MG muscle. There were, however, a significant decrease ($P<0.01$) in integrated electromyography (IEMG) of TA muscle. It was concluded that, with strength improvement in trained limb agonist muscle activation increased whereas antagonist muscle coactivation decreased. Consequently intermuscular neural adaptation (decrease of muscle coactivation) and intramuscular neural adaptation include increase of motor unit recruitment, firing rate and firing duration, does occur in trained athletes when a new training stimulus is implemented.
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1. Introduction

It is well established that physical activity that incorporates high muscle tensions, i.e., heavy-resistance strength training, can lead to an increase in maximal contractile muscle force. However, the specific mechanisms responsible for this adaptation are not fully known [1]. The increase in maximal contraction force may not solely be explained by increases in muscle cross-sectional area or volume. Rather, an increased "neural drive" to the muscle fibers contributes to the training-induced increase in maximal contractile force, even in the absence of increases in muscle size. Thus not only muscle size and muscle phenotype but also neural innervations are important determinants of maximal contractile muscle strength in vivo [1-2]. Physical training can stimulate both neurological and muscular adaptation [3-4], which can result in an increase in muscular force. Of the two, neurological adaptations to training are less understood especially in trained athletes. Although the effect of resistance training on muscle morphology has received considerable examination, less is known about the specific neural mechanisms responsible for the training-induced increase in maximal muscle strength [1]. Nonetheless, support for such change was demonstrated [3-5]. The specific neural adaptation that occurs is thought to be the result of improvements in intramuscular and intermuscular coordination. The intramuscular adaptations include motor unit recruitment, firing rate, synchronization of firing, and stretch reflex input; and the intermuscular adaptations include activation of synergists and co-contraction of antagonists [3]. Numerous reports exist of the morphological changes in human skeletal muscle induced by resistance training. Such changes include increases in anatomical muscle cross-sectional area [6-7], steeper muscle fiber pen nation angles [6], physiological muscle fiber area and increased percentage 2A fibers with a corresponding decrease in 2X fibers [8-9]. Likewise, the neural adaptation induced by resistance training has been addressed with the use of integrated electromyography (EMG) as an indicator for a change in efferent neural drive. Several investigators have reported increases in integrated EMG after resistance training and reported more synchronous motor unit impulses on electromyography (EMG) after resistance training when compared with pre-training patterns [1-2,7], although not consistently demonstrated in all studies [10-11]. In addition, Rutherford and Jones [12] suggested that training establishes new neural pathways that increase the coordinated activation of the muscle groups involved in a particular muscle action. Furthermore, Carolan and Cafarelli [13] proposed that reduced antagonistic co-contraction after isometric training of the leg extensors may be responsible for the greater torque-producing capabilities of the agonist muscle. It has also been proposed that training elicits alterations in the excitatory and/or inhibitory input, so that a greater inflow of impulses reaches the motor neuron of the working muscle [2,14]. Recent investigations have attempted to determine whether training induces greater motor neuron activation by monitoring EMG activity over the course of a resistance training program. Some studies have indicated that EMG activity increases with training [2,7,14-16],

supporting the hypothesis of increased neural activation and Others, however, have reported no such change [10-11,17]. Aagaard et al [1] reported that some of this disparity may be explained by the inherent methodological constraints associated with the recording of surface muscle EMG during maximal voluntary contraction (MVC). It is commonly thought that these neurological improvements occur during the initial stages of training, after which a gradual crossover to muscular adaptation occurs [18-19]. This would lead to the assumption that neurological change does not occur in the trained athlete, at least not to any significant degree. However, it is also logical to assume that, when a different training stimulus is introduced, additional neurological and muscular adaptations may occur. Because of lake and discrepancy in the research literature to evaluating such a possibility therefore, the purpose of this study was to investigate the intramuscular and intermuscular neural adaptation after resistance training in trained college athletes.

2. Methods

In order to determine if a new training stimulus would stimulate neural adaptation in previously trained men college athletes, the subjects were divided into 2 groups, experimental (EXP) and control (CON). The only alteration between CON and EXP group was that the experimental group engaged in a form of resistance training on plantar flexor muscles, whereas the control group did not. Various techniques have been employed to identify whether neural mechanisms are involved during a training program; however, we chose surface EMG as a simple, noninvasive measure of neural activation. Data were collected prior to initiating the new training protocol and at the end of 8 weeks.

Thirty male college student athletes(body mass 74.03 ± 5.41 kg, height 175.80 ± 6.31cm, age 22.33 ± 1.26 yr and sport history =3.8 1.31years, means ± SD) with no neuromuscular dieses history served as subjects for this experiment. The subjects were randomly assigned to either CON (n=15) or EXP (n=15) in an even distribution. The subjects were carefully informed about the design of the study with special information on possible risks and discomfort that might result, and subsequently signed an informed consent document prior to the start of the study. Before the first test, limb dominance was noted by the subject's preference in kicking a ball and taking off in a single-legged hop [20].

The young men were football athletes who were in an off-season. Subjects in the training group trained for 8 weeks. During training, they trained the plantar flexor muscles. Two types of progressive strength training, standing one-leg calf raises and Donkey calf raises, were performed 3 times a week for 8 weeks. One-leg calf-raises were carried out with each subject standing with their knee and hip joints secured in a neutral position. Each subject raised the heel of the training side from the neutral position to 30° plantar flexion in the sagittal plane, while supporting a barbell on their shoulders. In order to performance of Donkey calf raises exercise the subject stood with toes on the edge of a calf board (approximately 3-5 inches in height). The subject bent forward at the hips until the torso was parallel to the floor, and he stabilized the body by holding onto a piece of equipment. In this position he allowed the heels to drop as far as comfortably possible below the level of the toes. Then he raised the torso as high as possible on the balls of the feet. Once the top of the movement is reached he slowly lowered the heels as far below the level of the toes as possible, returning to the starting position [21]. Weight bag according to each subject 1RM put onto their back. Strength-training exercise consisted of 3 sets of 10-12 repetitions at 70-75% of the one-repetition maximum (1-RM) with a rest period of 1-2 min between sets. The 1-RM was tested by the formula (1RM = Bar Wtkg ÷ 1-(0.02 × Reps) [22] on the 1st day of every week during training for 8 weeks, and the intensity was adjusted to maintain a progressive resistance training stimulus. Before testing, each subject warmed up for 5 min with aerobic, low-resistance ergometer cycling and static stretching of the leg muscles [20]. One specific warm-up set of 15 repetitions was performed for each exercise at an intensity of 45% of the 1 RM. For control of the repetition speed compensatory acceleration technique was used [22]. A single investigator supervised each training session. The total time for carrying out the training program was 30 to 45 min for each session.

Each subject completed three trials of MVC, with each trial separated by a minimum of 180 s. In the MVC test, the subject was asked to increase force gradually for 2 s, and thereafter to try to keep the maximal force for approximately 2 s [23]. Before the pre-training measurements, each subject was familiarized with the equipment used and the procedures involved in the experiment. The force of plantar flexion was measured by a load cell (Lafayette, USA) placed between the metal base plate and force lever plate. The force signal from the load cell was amplified through a DC amplifier (32528, Lafayette, USA). Each subject sat on a seat, and was positioned at 80° hip flexion and 10° plantar flexion, with the knee at the neutral position. A belt was used as a support to keep the hip joint, knee joint, and thigh unchanged during testing [23]. The foot was also tightly secured by two straps to keep the ankle joint unchanged. Arms were folded in front of the

chest [20]. Verbal encouragement was given during the exercises. Hip, knee and ankle postures were measured using a SG110 and SG150 twin axis goniometer, (DataLog, Model P3X8, Biometrics, UK). The electrogoniometer was attached with double-sided (medical) adhesive tape and secured with adhesive medical tape. Goniometry recordings were analyzed using the datalog ver.2.0a software analysis package.

The measurements of each parameter were performed at pre-training (PRE) and post-training (POST) in both the training and the control groups.

The surface EMG signals of muscle were recorded during MVC by way of surface bipolar electrodes. The skin surface was cleaned with alcohol and rubbed with sand paper. The index of good skin impedance condition was that the skin gets a light red color and for fixed skin impedance condition 5 minutes time was used [24]. The room temperature fixed at 25 ºC. Surface bipolar electrodes Ag-AgCl (Medicotest blue sensor, M-oo-s), with 6 mm contact diameter, and 1.5 cm inter-electrode space were placed at the tibialis anterior ~10 cm below the caput fibulae, and at the gastrocnemius medial heads ~7 cm below the caput fibulae (2). For avoid cable movement artifacts and minimized the risk of separating the electrodes from skin, the cable, pre-amplifier and electrodes fixed on the skin by regular tape and net bandages. In order to cooperate between MVC test and EMG measurement we used alarm of EMG device. A circle was drawn with a permanent marker around all the electrodes to ensure consistent relocation of the electrodes. The EMG signals were amplified differentially with an AC amplifier (gain 375), and band-pass filtering was set at both low pass (500 Hz) and high pass (10 Hz) cutoff filters (ME3000p8, Mega Electronic, Finland) and sent to a personal computer via an analog to digital (A/D) board with Sensivity 3 mV and Resolution: 2.95 mV (12-bit, 8 channels, Mega Electronic, Finland).

All signal processing was performed using markers of Megawin software (version 2.0; Mega Electronic, Finland). The signals were band-pass filtered from 10-500 HZ. The EMG data of the MVC trial was used to calculate the IEMG for 1- second time between 800 ms before and 200 ms after the peak.

Data are presented as mean (SD), and these were computed by standard methods. The changes to each variable during the experimental periods (PRE and POST) were analyzed using paired students t-test. The independent t-test was used to determine a significant differences between trained and control groups. A Statistical significance was set at the $P<0.05$ level. These statistical analyses were performed with SPSS software (SPSS 11.5, SPSS).

3. Results

The changes in MVCs, IEMG values obtained from the medial gastrocnemius (MG) (Agonist) and tibialis anterior (TA) muscle (Antagonist) at PRE and POST in the EXP and CON group are shown in Tables 1 and 2.

Table 1- Changes in variables at pre-training (*PRE*) and post-training (*POST*) in the EXP group, Values are means (SD). (*MVC:* Maximum isometric voluntary contraction, I*EMG:* integrated electromyography).

Variable	EXP group	
	PRE	POST
MVC (Kg)	2.61±22.31	2.7*±24.84
IEMG of (MG) muscle (μV·s)	66.89±1321.13	68.36*±1406.26
IEMG of (TA) muscle (μV·s)	52.23±917.51	49.04*±823.59

*Significantly different from PRE at $P<0.01$

Table 2- Changes in variables at pre-training (*PRE*) and post-training (*POST*) in the CON group, Values are means (SD). (*MVC:* Maximum isometric voluntary contraction, I*EMG:* integrated electromyography)

Variable	CON group	
	PRE	POST
MVC (Kg)	2.63±22.34	2.59±22.29
IEMG of (MG) muscle (μV·s)	69.1±1319.53	67.98±1317.26
IEMG of (TA) muscle (μV·s)	51.12±916.38	50.89±917.11

*Significantly different from PRE at $P<0.01$

Figures 1-3 show the percentage changes in plantar flexor MVC, IEMG of the MG muscle and TA muscle values at pre-training (PRE) and post-training (POST), in the EXP and CON group.

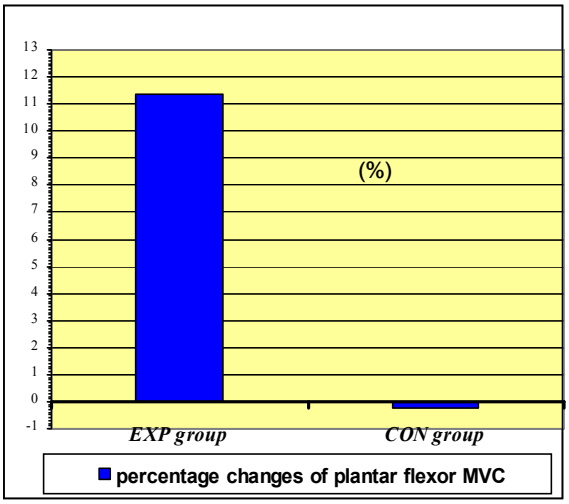

Fig 1- percentage changes in plantar flexor MVC at pre-training (*PRE*) and post-training (*POST*) in the EXP and CON group

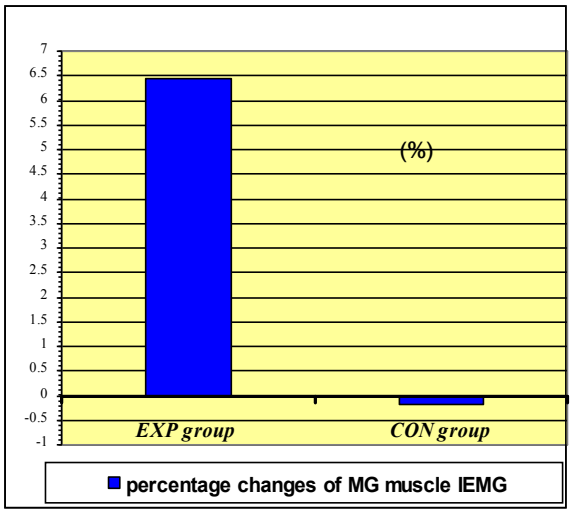

Fig 2- Percentage changes in MG muscle IEMG at pre-training (PRE) and post-training (POST) in the EXP and CON group

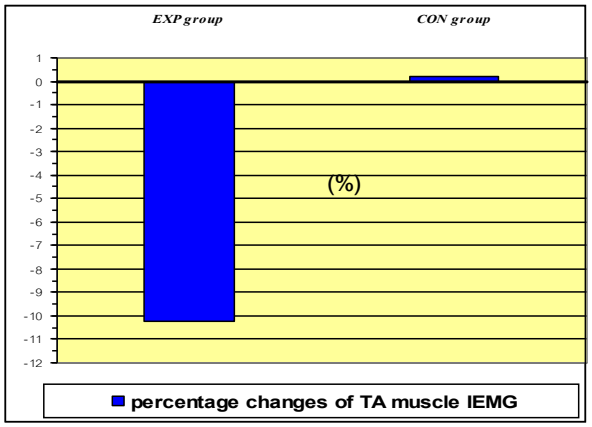

Fig 3- Percentage changes in TA muscle IEMG at pre-training (*PRE*) and post-training (*POST*) in the EXP and CON group

After training, MVC was significantly increased (11.34%, P<0.000) in EXP group but no significant change in the CON group. Percentage changes in plantar flexor MVC at pre-training (PRE) and post-training (POST) in the EXP and CON group was shown in figure 1.

The IEMG of the MG muscle significantly increased (6.44%, P<0.000) in EXP group after training whereas there was no significant change in the CON group. Percentage changes in MG muscle IEMG at pre-training (PRE) and post-training (POST) in the EXP and CON group was shown in figure 2.

The IEMG of the TA muscle significantly decreased (10.23%, P<0.000) in EXP group after training. For the CON group there was no significant difference in this variable. Percentage changes in TA muscle IEMG at pre-training (PRE) and post-training (POST) in the EXP and CON group was shown in figure 3.

The control subjects showed no significant changes for all variables throughout the experimental period, indicating that the changes observed in the training group were due to the new progressive resistance training programmer.

4. Discussions

The results of this study indicated a significant increase in plantar flexor MVC, MG muscle IEMG and a significant decrease in TA muscle IEMG after a new progressive resistance training in trained athletes. Some studies reported that resistance training increased agonist muscle EMG [1-2,7] although the others showed no significant change in this factor [10-11]. The reason for the discrepancies between the results of the present investigation and those of others examining EMG responses to training may be a function of differences in the mode, intensity, period of training and procedures used to analyze and quantify the EMG signal [11]. The present study used different contraction modes for isometric testing and for isotonic strength training. Isometric strength was assessed in order to avoid any positive learning effect, as subjects did not train with isometric contractions. Thus, differences in the muscle action used for testing purposes and the methods used to quantify EMG amplitudes could partly explain the conflicting results between the present study and other studies. Evetovich et al [11] reported that after training as individual muscle fibers enlarge; their positions under surface electrodes are altered. Therefore, it is possible that hypertrophy alone could have influenced the EMG signal. Garfinkel and Cafarelli [25], however, hypothesized that if electrode placement is constant, then the electrodes are detecting EMG over the same area of muscle membrane and, therefore, hypertrophy would not alter the EMG. Maximal iEMG changes after training may reflect the degree of electrical excitation of the underlying muscles and is affected by the number and size of motor units recruited, frequency of stimulation, and the synchrony of firing [15]. The interpretation of the increases in IEMG during maximal muscle actions after training is uncertain. Increases in IEMG can reflect increases in motor unit recruitment and/or motor unit firing rates [15]. Some studies that used the twitch interpolation technique with isometric muscle actions [26] have suggested that motor unit activation during maximal voluntary contractions before training is maximal. If this were the case, the increase in IEMG after training should reflect increased motor unit firing frequency, which may or may not cause greater force [25]. It is also

possible that increased surface area of hypertrophied muscle fibers could contribute to increased IEMG after training, but the relatively small muscle hypertrophy that occurred and the fact that muscle hypertrophy is not always accompanied by increased maximal IEMG [25] suggest that this is unlikely. Higbie et al [15] reported that the significant changes in strength after resistance training resulted from a combination of muscle hypertrophy and increased neural activation. However, it was not possible to precisely determine the relative importance of the two adaptations. They noted that based on the magnitude of the mean changes, and the correlations between changes in torque and changes in muscle hypertrophy and maximal IEMG, muscle hypertrophy and neural adaptations appeared to contribute approximately equally to the changes in strength after training. However, a substantial part of the strength change could not be accounted for by these two factors. Other studies have found that changes in muscle size or maximal IEMG after heavy resistance training are only moderately or poorly correlated with strength changes [27-29]. In the present study there was a significant decrease in TA muscle IEMG (antagonist) after training in trained athletes. Some studies found that after 8 weeks of training there was an increase isometric torque during resistance training that was not associated with a change in maximal activation of the agonist muscle but a decrease in EMG activity in the antagonist [10,12-13]. It is possible that the increase in maximal agonist muscle strength was due, in part, to a training-related decrease in co-activation of the antagonist muscles. It has been suggested, however, that this should result in greater agonist activation, and thereby cause increased agonistic EMG activity [10]. Rutherford and Jones [12] have suggested that changes in antagonist co-contraction are learned adaptations, whereas Carolan and Cafarelli [13] stated that the level of co-activation changes with training. The magnitude and the time course of the changes in the antagonist co-activation may be related to the types of action used, to the exercises utilized in the training and to the initial physical status of the subjects in terms of experience and skill in strength training. Nevertheless, the present result support the concept that strength training can lead not only to the increased activation of the agonist muscle in trained athletes but training-induced learning effects in terms of reduced co-activation of the antagonist muscle also plays a role enhancing the net force production of the agonists. The extent to which reduced co-activation of the antagonist is mediated by mechanisms in the central nervous system or associated also with peripheral neural control, especially during various dynamic actions, is difficult to establish [30]. Changes in co-activation could be a learned adaptation manifested as an improvement in coordination or skill. Reducing antagonist co-activation requires no conscious effort and therefore is likely mediated by mechanisms in the central nervous system. It has been suggested that co-activation is facilitated by Renshaw cell firing, which inhibits the Ia inhibitory inter-neurons by excitation of the Ib inter-neurons from the Golgi tendon organs or by direct descending motor pathways. Attenuation of any or all of these pathways would reduce co-activation. Because adaptations in antagonist co-activation do not account for all the nonhypertrophic increases in quadriceps MVC, the possibility of additional changes occurring elsewhere in the neuromuscular system must be entertained [13].

Pucci et al [31] noted that an increase in motor unit recruitment may have contributed to the increase in surface EMG activity and may have occurred independently of increases in mean motor unit firing rates. Reductions in co-activation have been proposed to be a contributing factor to the increase in agonist MVC with training. They reported that the small changes in activation and co-activation observed in their studies are not likely to be sufficient to solely account for the large increases in MVC force during training. It is possible that other neural adaptations may also have occurred. Such adaptations include changes in the control of the synergistic muscles [12,32] and motor unit firing rate synchronization [33]. The argument for neural factors being involved in strength increases hinged on increases in muscle activation observed in the surface electromyogram. At maximal efforts this adaptation was interpreted to mean a more complete recruitment of the entire motor unit pool or increased motor unit firing rates [31]. Also it has been suggested that training establishes new neural pathways that increase the coordinated activation of the muscle groups involved in a particular muscle action.

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