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Reply

Does maximal neural activation of muscle increase after resistance training?

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Sirs:

Our recent article (Häkkinen et al. 1992) examined in women the effects of short-term intensive strength training on the neuromuscular system as well as on serum concentrations of endogenous hormones. In that study we repeatedly measured our subjects on five identical occasions during the course of the 3-week experimental period. The measurements included the recording of electromyographic activity (EMG) during maximal voluntary bilateral isometric contraction from the vastus medialis, vastus lateralis and rectus femoris muscles of the right leg. Bipolar surface EMG recording was employed. The positions of the electrodes were marked on the skin by small ink tattoos. These dots ensured the same electrode positioning in each test over the 3-week experimental period. This method has been utilized in several of our other strength training studies since the paper by Häkkinen and Komi (1983). Maximal bilateral isomeric force and force-time curves of the leg extensor muscles were measured by an electromechanical dynamometer.

The cross-sectional area (CSA) of the quadriceps femoris muscle of the right thigh was measured with a compound ultrasonic scanner. Serum samples for hormone and sex hormone binding globulin determinations were analysed with the standard methods (see Häkkinen et al. 1992). A significant increase ($P<0.01$) from 2212 (SD 558) to 2431 (SD 664) N occurred in maximal isometric force corresponding to a relative increase of 9.7 (SD 8.4)%. An increase of 15.8 (SD 20.9)% took place also in the maximal integrated EMG (iEMG) of the trained muscles (averaged for the three muscles), while the relative enlargement in the CSA of the quadriceps femoris muscle was only 4.6 (SD 7.4)%. We concluded that these "findings in women indicated that the increases in maximal strength during short-term but intensive strength training were primarily due to increased voluntary activation of the trained muscles, while muscle hypertrophy remained limited in magnitude." Secondly, the hormone data obtained led us to indicate that "large interindividual differences in women in serum testosterone concentrations would indicate corresponding differences in muscle hypertrophy and strength development even during short term but intensive strength training" (Häkkinen et al. 1992).

In the criticism by Cafarelli and Fowler, the conclusions drawn by us from the EMG and force data seem questionable to them for several reasons. We thank them for their comments but we disagree with them and we would like to respond as follows.

Firstly, the data in Fig. 1 does illustrate that the change in maximal force after 2 weeks of intensive training as well as after a 3rd week of reduced training volume was significant as presented. Cafarelli and Fowler regard this as questionable. It is true that the SEM do overlap, when the *absolute* mean force values and their SEM are given. Female subjects are well-known to differ interindividually with regard to their absolute muscle forces. However, the female subjects in the present study could increase their force after the training so that the mean relative increase in force was as much as 9.7%. The SD (and SEM) of the *change* of the force was actually relatively small (only 8.4%) giving therefore a statistical significance (for a paired comparison) as stated. This is a question of how systematic the changes are, and not necessarily a question of how large the changes are, if the individual changes differ considerably. Of course, the sensitivity of various statistical tests differ slightly but in the present study this was not the primary question, because the changes were systematic. However, the interindividual differences in absolute force values naturally remained large also after training so that the weaker women became slightly stronger but the stronger women also became slightly stronger than before the training.

Concerning the request by Cafarelli and Fowler to send the individual data to them, we have a simple answer. The data for our study was collected in 1988. As mentioned in our paper the force and EMG data were recorded on magnetic tapes. At that time our old labora-

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tory building contained too many of the large tapes used, because there were also several other active researchers in our department (and in other departments as well) who also had collected a large number of tapes over the years, not to mention the data in printed form which was stored on bookshelves. So it was not "a surprising turn of events" to us that in response to the request by the Faculty we had to reduce the amount of stored data not actively needed as well as to reuse the old tapes, whenever possible. Of course, this was unfortunate, because we would normally have stored the data for some years so that there would have been the possibility of our utilizing the data for various other purposes.

Secondly, Cafarelli and Fowler state that "the investigators did not verify maximality of either the control or posttraining contractions with superimposed shocks. Thus, the data point at day 0 and day 15 may actually reflect submaximal contractions." We disagree completely with this argument. First of all, the subjects were carefully familiarized with voluntary contractions by performing several warm-up and a couple of maximal contractions before the actual testing contractions. We then recorded for each test 3–6 maximal test contractions to ensure that we had obtained the *voluntary* maximum of that particular test. It has been our experience that we obtain voluntary maximum during the first 3–4 contractions - however, we usually (as also in the present study) continue to record more test contractions, to see if the subject can improve her/his maximum. We then stop the test, when the subject unable to improve her/his maximum (usually no more than six contractions are needed on these rare occasions). More important, in the present study we actually did a test and retest comparison by performing a retest after 2 days from our data point at day 0. The mean maximal force for this retest day was 2185 (SD 500) N which is slightly smaller than our value of 2212 (SD 558) N at day 0. At day 15 the mean force value had increased *due to training* up to 2377 (SD 622) N. We have performed this type of evaluation of the reproducibility of our test procedures frequently over the years and we have published some of the data. So when there is no special reason, we do not find it necessary to publish this type of data every time. If the force values differed from each other, then we would naturally report all the data to demonstrate this difference.

Further to the second comment of Cafarelli and Fowler that "There are similar problems in trying to interpret the data in Fig. 2". We have a similar answer to this statement as we had to the first question with regard to Fig. 1. First of all, the SEM of the mean at day 0 is rather large, because it represents (as mentioned in the figure legend) the averaged absolute value for the vastus lateralis, vastus medialis and rectus femoris muscles. The SEM of the mean at day 15 and day 21 are also large for the same reason. However, these averaged iEMG values increased due to the training so that at the end at day 21 the relative increase was as much as 15.8 (SD 20.9)%. The SD (or SEM) of the change of the mean remained within reasonable limits so that the change (for paired comparison) was statistically significant as presented. It is true that there was also "an unexpected change in averaged maximal iEMG of the three muscles during the particular period of reduced training" which refers to day 17 and day 19. However, we considered this in the discussion of our paper as follows: "Differences may exist in the neural activation of separate muscles even in the same muscle group". Actually, the data of the present study showed that, for example, the maximum iEMG of the vastus lateralis (VL) muscle (which had the highest absolute mean value of the three muscles) was at these points at a relatively low level (at day 17 the VL was 0.55 (SD 0.21) $mV \times s$); while, on the other hand, the maximal iEMG of the rectus femoris (RF) muscle (which had its lowest absolute mean value) was also higher at this point (at day 17 the RF was 0.20 (SD 0.03) than at day 0 (0.19 (SD 0.04) $mV \times s$). So, when the averaged iEMG data of the three muscles are given, it can therefore partly (as was the case in the present study) mask the actual data and lead to misinterpretation. Secondly, we also pointed out in the Discussion that "EMG is such a complex measurement that one should always be careful with the interpretations".

Thirdly, Cafarelli and Fowler point out that "the authors have been selective in citing only literature that supports the notion of an increase in maximal voluntary activation (EMG) after resistance training. Reports from investigators who have not found an increase in maximal EMG after resistance training have not been cited - etc." Because this comment would also seem to include a *disagreement as to principles* we would like to give a longer answer to this as follows.

Nowadays it is a very *common* belief that a major part of the large initial increases observed in maximal strength during the very first weeks of heavy resistance strength training can be contributed to the training-induced increase in the maximal voluntary activation of the trained muscles (e.g. Moritani and DeVries 1979; Komi et al. 1978; Häkkinen and Komi 1983; Roy et al. 1984; Narici et al. 1989; Komi 1986; Sale 1986; Enoka 1988; Häkkinen 1989; Häkkinen et al. 1989; Moritani 1991) although some slightly different data is also available (Thorstensson et al. 1976; Cannon and Cafarelli 1987). Apparently, there seems to be a functional activation reserve, especially in previously untrained subjects, which can be mobilized during proper strength training (Sale 1986). This type of neural adaptation may therefore be responsible for several experimental findings of strength gains in the absence of measurable hypertrophy. It is very unlikely that muscle adaptation (hypertrophy) could account for these rapid initial gains in strength of the trained muscles. A gradually increasing contribution of muscular hypertrophy usually accompanies improved muscle strength during the later weeks of heavy resistance strength training (e.g. Moritani and De Vries 1979; Sale 1986; Komi 1986; Enoka 1988; Häkkinen 1989).

The increased maximal voluntary neural activation of the muscles during heavy resistance strength training is usually based on the increases noted in the maximal iEMG of the trained muscles. These types of EMG stud-

ies do not themselves reveal the detailed mechanisms of neural adaptation. The EMG is a complicated and summated signal that represents the extracellular voltage-time measure of the level of the excitation on the muscle (e.g. Enoka 1988). However, it is obvious that the early part of strength training might, especially among initially untrained subjects, be associated with increased synchronization of the prime mover motor units (MU). The increase in the magnitude of EMG (iEMG) during strength training results from the increased number of the active MU and/or increase in their firing frequency (Sale 1991; Enoka 1988). These findings would further suggest that an increased net excitation of the prime mover motoneurons could result from increased excitatory input, reduced inhibitory input or both (Sale 1986, 1991; Komi 1986; Enoka 1988; Häkkinen 1989). A degree of the training effect resides therefore in the facilatory and/or inhibitory neural pathways acting at the various levels in the nervous system (Moritani and DeVries 1979).

The role of the higher motor centres in increasing their descending activity might be of great importance (Milner-Brown et al. 1975) with a plausible simultaneous decreased coactivation of the antagonist muscles (Carolan and Cafarelli 1992). In addition to adaptation within the nervous system in voluntary contractions, effects of strength training may take place also in reflex responsiveness as reviewed recently in detail by Sale (1991). However, the reflex potentiation method – such as the iEMG method – cannot distinguish the degree of the contributions of increased recruitment and increased firing rates to the increase in MU activation (Sale 1991). But, both of these methods can demonstrate that strength training can change activation so that

1. The activation of the prime movers is increased and/or that
2. There is an improved co-contraction of synergists and/or that
3. There is an increased inhibition of the antagonist muscles.

Definitive evidence of the mechanisms underlying the neural adaptations awaits future studies. Further experimental research is needed, for example, utilizing a demanding and invasive technique, in which needle or fine wire electrodes are inserted into the muscle allowing single MU recordings to be made to clarify in more detail specific mechanisms of neural adaptation caused by strength training (see also Enoka 1988; Moritani 1991).

Moritani (1991) has pointed out in his recent review that one approach to the study of training is to obtain changes in electrically evoked muscle forces. "These forces are obviously independent of volition and should represent the force generating capacity of the muscle fibres themselves, not the level of neural drive" (McDonagh and Davies 1984). It has been shown by Davies et al. (1985) that isometric strength training for 8 weeks resulted in a 33% increase in maximal strength, but electrically evoked tetanic tension increased by only 11%. Recent work from Moritani's laboratory (Ishida et al. 1990) has also demonstrated a similar discrepancy between increases in maximal strength and electrically evoked twitch parameters. So Moritani (1991) has concluded that these data fit well with well-known data of others which indicate a discrepancy between increases in the CSA of muscle tissue and the increases in maximal strength during strength training. He further states that "these findings again strongly support the hypothesis that the increase in maximal voluntary strength in the early stages of training is due to a change in voluntary neural drive to the muscle tissue."

Moritani (1991) has further pointed out that there are two studies (Thorstensson et al. 1976; Cannon and Cafarelli 1987), which have failed to show an increase in iEMG after strength training. "Thorstensson et al. (1976) employed the barbell squat exercise and showed a large increase in weightlifting strength while observing no increase in isometric knee extension strength. It was just for this last movement that iEMG was recorded and showed no change. Cannon and Cafarelli (1987) have shown an approximately 15% increase in maximal voluntary strength of adductor pollicis muscle after 5 weeks of training and have observed no change in neural drive (EMG). They also observed a significant increase in maximal voluntary strength of the untrained contralateral muscles with no change in EMG amplitude. They suggested that their data did not reveal some unspecified increase in neural drive but rather a more responsive group of hypertrophied muscles. However, their argument seems to fail to explain the significant strength gain observed in the untrained contralateral muscles, as these muscles are unlikely to possess responsive groups of 'hypertrophied' muscles." It is also important to point out that the training intensity in the study by Cannon and Cafarelli (1987) was only 80% of maximal voluntary contraction. It has been shown (Häkkinen et al. 1985) that it should be, at least in some cases, between 80% and 90% of maximum or even more before a considerable increase might be obtained in the maximal voluntary iEMG of the trained muscles. Moritani (1991) has further pointed out that the adductor pollicis muscle is largely composed of type I fibres (Johnson et al. 1973) and the rate coding (MU firing frequency modulation) might be the only mechanism for increasing force above 50% maximal voluntary strength, as no MU are recruited above this level of force (Kukulka and Clamann 1981; Moritani et al. 1986). "One would, therefore, expect a greater degree of synchronization of MU activity for this small hand muscle, which in turn should result in increasingly large oscillations in the surface EMG." Finally, Moritani (1991) has concluded as follows: "On this basis and a considerable amount of evidence for neural adaptations presented, it is very difficult to explain the dissociation between the increase in maximal strength and the surface EMG amplitude. Further studies with intramuscular MU spike recordings are definitely needed for elucidating the nature of strength gain without accompanying increased neural drive."

Finally, we hope that our answers to the criticism of Cafarelli and Fowler have clarified the matter of neural and hypertrophic adaptations to heavy resistance strength training. Our recent paper (Häkkinen et al. 1992) was an attempt to study the problem in female

subjects during short-term but intensive strength-training as objectively as possible. We hope that this paper and others published by us and by our colleagues over the years have been of some value in examining the mechanisms taking place both in the neuromuscular and in the endocrine systems. We also hope that our studies give constructive stimuli to others in the field.

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