

Effects of power training on muscle structure and neuromuscular performance

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Accepted for publication 17 December 2003

The present study examines changes in muscle structure and neuromuscular performance induced by 15 weeks of power training with explosive muscle actions. Twenty-three subjects, including 10 controls, volunteered for the study. Muscle biopsies were obtained from the gastrocnemius muscle before and after the training period, while maximal voluntary isometric contractions (MVC) and drop jump tests were performed once every fifth week. No statistically significant improvements in MVC of the knee extensor (KE) and plantarflexor muscles were observed during the training period. However, the maximal rate of force development (RFD) of KE increased from $18\,836 \pm 4\,282$ to $25\,443 \pm 8\,897$ N ($P < 0.05$) during the first 10 weeks of

training. In addition, vertical jump height (vertical rise of the center of body mass) in the drop jump test increased significantly ($P < 0.01$). Simultaneously, explosive force production of KE muscles measured as knee moment and power increased significantly; however, there was no significant change ($P > 0.05$) in muscle activity (electromyography) of KE. The mean percentage for myosin heavy chain and titin isoforms, muscle fiber-type distributions and areas were unchanged. The enhanced performance in jumping as a result of power training can be explained, in part, by some modification in the joint control strategy and/or increased RFD capabilities of the KE.

High intensity strength training typically induces clear neural and/or muscular adaptations (Moritani & DeVries, 1979; Häkkinen et al., 1981; Häkkinen & Komi, 1983; Staron et al., 1990; Sharman et al., 2001). However, the impact of training with explosive movements for power (power training) on the neuromuscular system is less clear (Häkkinen et al., 1990; McBride et al., 2002). Moritani and DeVries (1979) have demonstrated that in high intensity strength training the neural factors dominate in strength development at the three first weeks of training, while the hypertrophic factors start to play a major role after this time. In power training, increases in muscle power capabilities have been observed with coincidental changes in neural factors (McBride et al., 2002); however, possible muscle structural adaptations have not been clearly identified (Häkkinen et al., 1985; McBride et al., 2003; McGuigan et al., 2003).

The adaptability of skeletal muscle fiber size in response to altered loading patterns is well documented (e.g., Saltin & Gollnick, 1983). Fitts and Widrick (1996) have concluded that “both slow and fast-

twitch fibers have capacity to hypertrophy when overloaded”. In addition, training can cause changes in muscle fiber-type composition, which can be observed as a shift in expression between myosin heavy chain (MHC) isoforms. While myosin is directly involved in the force production processes of muscle contraction, titin, which is a large protein acting as an anchor between the z-lines and myosin within the sarcomere, is thought to have effects on the elastic characteristics of muscle fibers (Tskhovrebova & Trinick, 2002). It is possible that improvements in muscle power could be related to shifts in MHC or titin isoforms, because both of them contribute to the force production (Staron et al., 1990; McBride et al., 2003). Andersen and Aagaard (2000) have shown that the 3 months of heavy resistance training decreases the amount of MHC IIX content, while MHC IIA content increases. Williamson et al. (2000) have found that a lower-intensity resistance training, lasting 12 weeks, caused a reduction in the expression of fibers expressing two MHC isoforms and overall increase in MHC I among elderly subjects. Thus, skeletal muscle is a highly plastic tissue

being capable of altering its contractile proteins. It is not known how exercise affects muscle elasticity expressed as different titin isoforms and whether changes in MHC isoforms are followed by changes in titin isoforms in human skeletal muscle. A 6-month study using both strength and power training combined demonstrated a shift in the expression of MHC isoforms from MHC IIX to MHC IIA (Sharma et al., 2001). However, a subsequent study showed no change in MHC or titin isoforms following a power-only training protocol (McGuigan et al., 2003).

Power training has been shown to cause shifts in isometric force–time and/or force–velocity curves simultaneously with quantitative changes of the neuronal input to the muscle (Moritani & deVries, 1979; Komi et al., 1982; Häkkinen et al., 1985). Power training has been shown to increase jumping height measured as increase in vertical rise of center of body mass (Bobbert, 1990) simultaneously with qualitative shifts in the electromyographic (EMG) patterns (Schmidtbleicher & Gollhofer, 1982). However, typical power training programs are often characterized by a lower volume and muscle time under tension in comparison with strength training. Therefore, it is possible that the amount of volume (associated with hypertrophic response of muscle) in power training is inadequate to stimulate muscle structure changes over a relatively short training period. As shown above, previous studies typically have focused on either neural or muscular adaptation of power training adaptation independently. The present study was designed to combine these two methods when examining effects of long-term power training on muscle structure, neuromuscular function, and physical performance. This investigation has attempted to re-address the issues related to possible mechanisms responsible for increases in muscle power following power training. Specific points addressed in response to power training were (1) the effects of power training on MHC isoforms, muscle fiber areas, and the elastic structure (titin), and (2) neuromuscular adaptations and improvements in performance. It was hypothesized that improvements in neuromuscular performance could be related to changes in EMG patterns and/or shifts in MHC and/or titin isoforms.

Methods

Subjects

Twenty-three recreationally active men, who were randomly divided into two groups, volunteered as subjects for the study. The training group consisted of 13 subjects (age 24 ± 4 years, height 1.78 ± 0.05 m, body mass 77.9 ± 12.5 kg, fat% 11.0 ± 4.6 determined by the methods of Jackson & Pollock, 1985), while the control group consisted of 10 subjects (age 25 ± 2 years, height 1.78 ± 0.07 m, body mass 77.2 ± 11.7 kg, fat%

8.6 ± 6.0). All the subjects were informed of the possible risks of the experiment and they gave their written consent to participate. The ethical committee of the University of Jyväskylä, Finland, approved this study.

Training

The experimental group trained twice a week for 15 weeks, which was preceded by a 2-week preparatory phase consisting of light loads of squats, deadlifts, as well as abdominal and calf exercises. Each training session was controlled by an experienced supervisor. The training of leg extensor muscles included various types of stretch-shortening cycle (SSC) exercises such as jumping performances with a special sledge apparatus, drop jumps from the heights of 20–70 cm, jump squats (30–60% of one repetition maximum), one leg and two leg hopping, and hurdle jumps. The jumping performances (five to 10 repetitions per set) were performed with a maximal effort to develop explosive force production of lower limb muscles. The overall number of muscle actions increased progressively from 80 to 180 actions per training session throughout the whole training period. Training diaries of the subjects revealed that other physical activities such as cycling, walking, and ball games lasted almost 6 h per week for the experimental group and 4 h 30 min per week for the control group ($P < 0.27$ between the groups). However, this extra training was their normal weekly activity, and it did not change during the experimental period.

Protocol

The experimental group was tested before, after 5, 10, and 15 weeks of training, while the control group was tested only before and after the entire training period of the experimental group. Testing included two to three maximal bilateral isometric voluntary contractions (MVC) for knee extensors (KE) and plantarflexors (PFs), and three to five maximal SSC exercises (drop jumps). For the MVC testing subjects were instructed to exert maximal force against the footplate as quickly as possible and to maintain the maximal force for 2–3 s. In the sitting position (see Komi, 1973), the angle of the knee joint was kept as 107° for testing the KEs and 180° for testing the PFs. The respective value of the ankle joint was 90° in both conditions.

In the drop jump performances the constant dropping height of 50 cm (from the bench to the floor) was used for each subject, and its subsequent rebound height was recorded. The instruction for the subjects was designed so that they had to perform three to five drop jumps with maximal effort and with minimal displacements of the knee joints. This technique was expected to load more PFs compared with KEs and, furthermore, the performance would have been more powerful (reactive).

Before and after the training period, needle biopsies (a sample size of 100–150 mg) were obtained from the middle portion of the lateral gastrocnemius (GA) muscle. This muscle was selected, because the present training was planned to mainly load the triceps surae muscle group. Local anesthetics (2 mL lidocaine–adrenalin, 1%) were administered subcutaneously prior to incision of the skin. An ultrasound scanner (Aloka SSD-280 LS, Tokyo, Japan) fitted with 7.5 MHz transducer was used to evaluate the site and depth for taking the sample. The muscle sample was mounted on Tissue-TEK (Miles Inc., Elkhart, IN, USA) and frozen rapidly in isopentane, which was cooled to -160°C in liquid nitrogen. The samples were stored at -80°C until analyzed.

Measurements and analysis of drop jumps and maximal isometric forces (MVC)

In the drop jumps, ground reaction forces (GRFs) were measured with a force plate. Surface electromyography (EMG) activity from the vastus lateralis (VL), vastus medialis (VM), GA, soleus (SOL) and tibialis anterior (TA) muscles were recorded telemetrically (MESPEC 4000, Mega Electronics Ltd, Finland) with surface electrodes (Beckman miniature skin electrodes, Beckman Instruments, Inc. 650437, IL, USA) having the interelectrode distance of 20 mm. The electrodes were placed longitudinally over the muscle bellies between the center of the innervation zone and the distal tendon of each muscle. The EMG signal amplification was 1000 times (Biotel 99, Glonner, Germany; bandwidth 20–640 Hz/–3 dB; CMRR 110 dB), and it was digitized and synchronized with the force records at a sampling frequency of 1 kHz.

All performances were videotaped at the frame rate of 200 Hz. Reflective markers were placed on the following points: the distal head of the fifth metatarsal bone, the lateral malleolus, the lateral epicondyle of the femur, the greater trochanter, and the tragus. These points were digitized for 2D video analysis (Peak Performance Technologies, Inc., Motus software, Denver, CO, USA). After filtering with the fourth-order Butterworth conditioner with a cut-off frequency of 8 Hz, the digitized and scaled co-ordinates were synchronized with 2D GRF data for calculating joint moments and powers (Belli et al., 2002). In the synchronization of video and other recorded data, a circuit to introduce a trigger signal (GRF value of 100 N) to the computer and a flash for a video image was used. Anthropometric data provided by the standards of Dempster (1955) were used to determine inertia and mass of the segments.

The raw EMG signals were first full-wave rectified, then integrated and finally time normalized in four different phases: pre-activation from 100 to 50 ms before the ground contact, pre-activation from 50 to 0 ms before the contact, braking phase, and push-off phase. The onset of the GRF of each jump was used as a reference point to identify the beginning and the end of the contact. The force and velocity records were used to identify the end of the braking phase. In addition, the full-wave-rectified EMG signals were averaged for obtaining muscle activity patterns both individually and by groups. This phase-dependent averaging method (see Moritani et al., 1991) allows repeated bursts of EMG activity during jumping to be aligned in time with respect to the mechanical data. The averaging was started 200 ms before the onset of the GRF and finished 600 ms after that point.

In the MVC condition, according to the maximal force the best performance of each subject was chosen for further analysis. The sampling rate for the force signal was 1 kHz. The maximal rate of force development (RFD) was calculated by determining the steepest application point of the force curve by each 5 ms. In addition, average force produced during nine consecutive (half overlapping) absolute time periods (100 ms in duration) was calculated from the start of the force production (see e.g., Viitasalo et al., 1980). The EMG signals were full-wave rectified and the maximal voluntary average EMG (aEMG) values were calculated during 1 s period when the force was kept on the maximal level.

Analysis of muscle biopsies

For histochemical analysis, serial sections (10 μ m) of the muscle biopsy samples were cut in a cryostat (–24 °C), and routine ATPase analysis was performed after pre-incubation at pH of 4.37, 4.60, and 10.30 (Brooke & Kaiser, 1970). Four different fiber types were defined (types I, IIA, IIAX, and IIX)

according to (Staron et al., 1990). In order to localize the muscle cell borders, the sections were incubated with 1:500 dilution of mouse monoclonal anti-dystrophin (dys 2, Novocastra Laboratories, Newcastle upon Tyne, UK) overnight at +4 °C. After washing with Tris-buffered saline (TBS, pH = 7.5) the bound primary antibody was visualized by avidin–biotin peroxidase kit (Vectastain PK-4002, Vector Laboratories, Burlingame, CA, USA) using diaminobenzidine (Sigma, St. Louis, MD, USA) as a chromogen.

The serial sections were visualized and analyzed using an Olympus BX50 microscope (Olympus Optical Co., Tokyo, Japan), a Sanyo Hi-resolution Color CCD camera (Sanyo Electronic CO., Osaka, Japan), and an eight-bit Matrox Meteor Framegrabber (Matrox Electronic Systems, Quebec, Canada), combined with image-analysis software (Tema, Scan Beam, Hadsund, Denmark). A fiber mask was automatically applied by the computer along the cell borders of dystrophin immunostained sections. All incorrect borders were excluded and only horizontally cut fibers were used in the determination. A fiber number was assigned by the computer to each individual fiber. Images of ATPase stainings were fitted into the fiber mask. The staining of the each individual fiber was divided into three groups: light, intermediate, or dark, and four different fiber types were defined (types I, IIA, IIAX, IIX). The relative proportion of the various fiber types and fiber type areas were determined from 225 ± 47 (mean \pm standard deviation (SD), range 150–290 cells) representative cells.

From each biopsy 10–20 serial cross-sections (10 μ m) were cut and placed in 100–200 μ L of lysing buffer and heated for 3 min at 90 °C in order to analyze MHC isoform composition using SDS-PAGE gel electrophoresis. Between 5 and 20 μ L of the myosin-containing samples were loaded on a SDS-PAGE gel containing 6% polyacrylamide and 30% glycerol. Gels were run at 70 V for 42 h at 4 °C (Andersen & Aagaard, 2000). After fixation the gels were Coomassie-stained. Three different MHC bands can be separated in normal adult human skeletal muscle with the present electrophoresis procedure. These bands correspond to the MHC isoforms I, IIA, and IIX (Klitgaard et al., 1990). Percentage of different MHC isoforms was determined using a densitometric system (Cream 1D, Kem-En-Tec aps, Copenhagen, Denmark).

For titin analysis, 10–20 serial cross-sections (10 μ m) were cut from each biopsy and placed in 700 μ L of lysing buffer and heated for 10 min at 60 °C. Between 5 and 10 μ L of samples were loaded on a SDS-PAGE gel containing 3.3–12.0% linear gradient gel with Fairbanks buffer (Granzier & Wang 1993). Gels were run at 70 V for 24 h at room temperature and silver stained (Silver Stain Plus, Bio-Rad Laboratories, Hercules, CA, USA). For identification of the titin bands, the gels were blotted at 25 V overnight using a Mini PROTEAN II Trans-Blot Cell system to a PVDF membrane. The membranes were incubated with mouse monoclonal anti-titin (1:500, Sigma, St. Louis, MD, USA) overnight at 4 °C. After washing with buffer, the bound primary antibodies were visualized by avidin–biotin peroxidase kits for mouse using diaminobenzidine as a chromogen.

Statistical analysis

Repeated measures ANOVA was utilized to test the main effects of experimental conditions on selected variables. When significant *F*-values were found, a *post hoc* test was applied to determine the specific condition means that differ from one another. Mean and SD were calculated by conditions. Finally, Pearson's correlation analysis was utilized to study relationships between the changes in different variables.

Results

Maximal isometric force (MVC)

MVC of the PF muscles increased until the 10th week of training (3910 ± 1106 vs. 4336 ± 1003 N) parallel with the maximal aEMGs of the GA and SOL muscles in the training group (Fig. 1). The changes of aEMGs of those muscles correlated positively with the respective changes of MVC ($r = 0.77$, $P < 0.01$ for GA and $r = 0.59$, $P < 0.01$ for SOL). During the last 5 weeks of training, however, MVC (4241 ± 1071 N) and EMGs of the PFs did not increase. The maximal RFD remained unchanged ($21\,657 \pm 7696$ vs. $20\,158 \pm 7728$ N s⁻¹) during the entire training period. In the control group, MVC values of the PF muscles did not change significantly (4080 ± 910 vs. 4239 ± 793 , $P > 0.05$) during the training period. The respective aEMGs and RFD did not change either.

MVC of the KE muscles did not increase significantly in any experimental condition (before: 3598 ± 897 N vs. after 5 weeks: 4478 ± 2092 N vs. after 10 weeks: 4366 ± 1948 N vs. after 15 weeks: 4497 ± 1780 N), and this was also the case for the corresponding maximal aEMGs of KE. aEMG values of the VL muscle, for example, were 317 ± 179 μ V (in the beginning), 378 ± 212 μ V (after 5 weeks), 406 ± 242 μ V (after 10 weeks), and 374 ± 185 μ V (after 15 weeks). The maximal RFD of KE increased from $18\,836 \pm 4282$ to $25\,443 \pm 8897$ N s⁻¹ ($P < 0.05$) during the first 10 weeks of training and did not change ($P > 0.05$) during the last 5 weeks of training ($22\,213 \pm 6828$ N s⁻¹). Figure 1 demonstrates that the beginning of the force–time curve shifted to the left and up during the entire training period. In the control group, MVC and the maximal RFD of the KE were the same before and after the

Fig. 1. Mean (\pm SD) maximal isometric force and aEMGs of the gastrocnemius and soleus muscles in the course of 15-week training period (left). Mean force–time curves of the knee extensors before the training and after 5, 10, and 15 weeks of it (right). The x-axis represents nine overlapping time periods, and the lines were drawn between these nine points. The mean forces were obtained from the individual values of each subject.

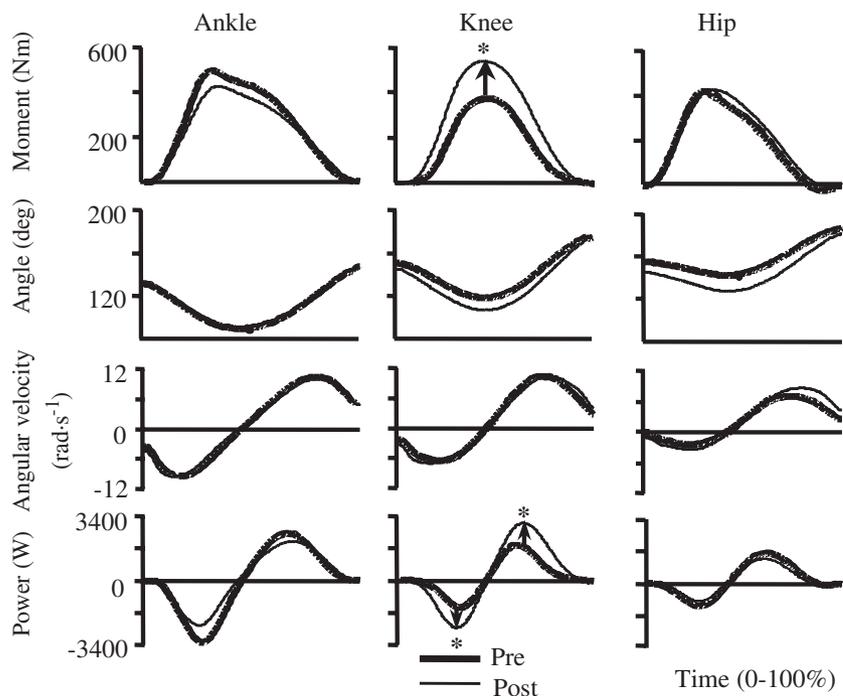
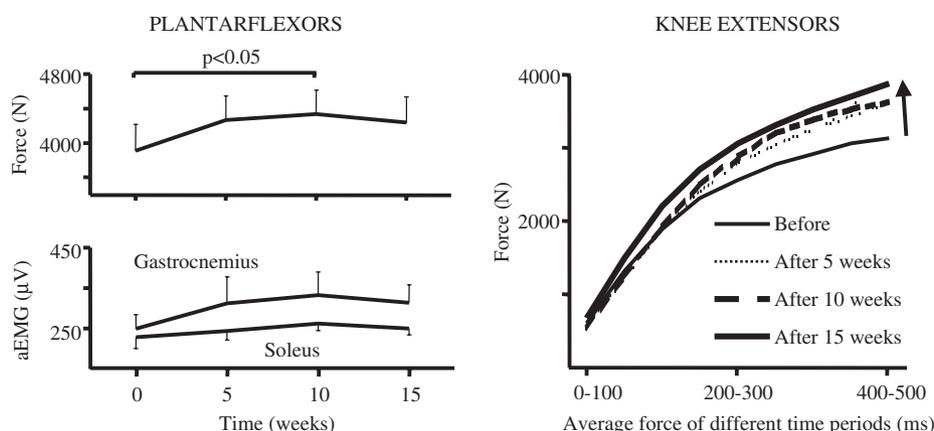


Fig. 2. Time normalized, joint moment, angle, angular velocity, and power curves in the ankle, knee and hip joints during the contact phase in drop jump exercises measured before the training (thick line) after the 15 weeks of training period. * $p < 0.05$

Table 1. Characteristics of myosin heavy chain (MHC) composition, muscle fiber distribution and muscle fiber size before and after the 15 weeks of training period in the experimental and control groups

	Experimental group		Control group	
	Before	After	Before	After
MHC I	62.6 ± 13.1	65.0 ± 14.4	73.1 ± 13.2	67.6 ± 14.5
MHC IIA	32.6 ± 10.5	30.6 ± 11.5	23.7 ± 11.6	29.8 ± 11.6
MHC IIX	4.9 ± 7.1	4.4 ± 5.4	3.3 ± 3.5	3.0 ± 13.6
Fiber type I	59.6 ± 13.1	63.2 ± 16.5	65.0 ± 11.1	65.2 ± 10.3
Fiber type IIA	27.4 ± 7.1	23.7 ± 6.7	23.7 ± 9.8	27.5 ± 8.4
Fiber type IIAX	9.3 ± 6.4	10.0 ± 7.0	6.3 ± 5.4	6.0 ± 5.5
Fiber type IIX	3.8 ± 4.8	3.2 ± 4.9	5.0 ± 8.0	1.3 ± 2.3
Fiber size I	57.5 ± 3.7	61.6 ± 16.3	63.7 ± 11.3	64.4 ± 11.3
Fiber size IIA	29.7 ± 7.5	25.5 ± 7.2	25.0 ± 10.0	28.8 ± 8.9
Fiber size IIAX	9.3 ± 7.1	9.9 ± 6.8	6.1 ± 5.2	5.6 ± 5.2
Fiber size IIX	3.5 ± 4.2	3.1 ± 4.9	5.3 ± 8.6	1.3 ± 2.0

training period (4355 ± 1359 vs. 4556 ± 1334 N, and $26\,719 \pm 8444$ N s⁻¹ vs. $25\,395 \pm 4915$ N s⁻¹, respectively).

Drop jumps

In the drop jump, the rising height of the center of gravity increased from 0.30 ± 0.06 to 0.37 ± 0.05 m ($P < 0.01$) in the experimental group (0.22 ± 0.05 vs. 0.22 ± 0.04 m in the control group) during the entire training period, while the contact time did not change (0.226 ± 0.090 vs. 0.231 ± 0.049 s). At the same time, knee moment (M_{peak} : 377 ± 133 N m vs. 544 ± 130 N m, $P < 0.05$) and power (P_{peak} : 2093 ± 788 vs. 3227 ± 919 W in the push-off phase, $P < 0.01$) increased, while changes in moment and power values of the hip and ankle joints were insignificant ($P > 0.05$) (Fig. 2). The changes in the knee moment correlated positively with the changes of rising height of the gravity ($r = 0.70$, $P < 0.01$), while the ankle and hip joint powers did not relate ($P > 0.05$) to the drop jump performance. No changes in aEMGs or in muscle activity patterns of the acting lower limb muscles were noticed in drop jump.

Muscle structure

During the training period, the mean percentage of myosin heavy chain isoforms (MHC I, MHC IIA, and MHC IIX) remained the same: $62.59 \pm 13.13\%$ vs. $65.01 \pm 14.39\%$, $32.56 \pm 10.48\%$ vs. $30.64 \pm 11.48\%$, and 4.86 ± 7.10 vs. $4.35 \pm 5.40\%$, respectively. The muscle fiber distributions and areas were also unchanged in the both groups as well as MHC isoforms of the control group. Titin gel electrophoresis analysis showed that all the subjects expressed a faster-mobility titin band, and furthermore, eight subjects from training group and three subjects from control group expressed a lower-mobility titin band. The content of the lower-mobility titin ranged from 1% to 6%, except one subject from control

group, who expressed almost 20% lower-mobility titin. There were no changes in titin isoform compositions either in training group or control group (Table 1).

Discussion

The results of the present study demonstrated that MVC of KE muscles did not change during the training period, while PF muscles produced higher forces after 10 weeks of training. The maximal RFD of KE increased, however, significantly ($P < 0.05$) during the first 10 weeks of training. In the drop jumps, the increase in the rising height of the center of gravity were highly related with the changes in the knee power but only non-significant changes were observed in EMGs during the training. The mean percentage for MHC and titin isoforms, muscle fiber distributions, and muscle fiber areas were unchanged.

The present study confirms earlier findings that prolonged power training results in improvements in neuromuscular performance during the earlier weeks of training (e.g., Moritani & DeVries, 1979; Häkkinen et al., 1985). This may mainly be caused by neural factors, which play a major role at early stages of strength gain (Moritani & DeVries, 1979). Komi and Bosco (1978) have noticed that there is a very close relationship between the changes in EMG and the changes in force during the course of 12 weeks of training. Furthermore, Häkkinen et al. (1981) have observed that during the first 8 weeks of training, force increased parallel with increase in EMG but without muscle fiber hypertrophy. In the present study, the EMG increases of the PF muscles were related to the respective increases in force during the course of the first 10 weeks of training. However, during the last 5 weeks no additional increases in EMGs or force were observed. This may be due to too small or too similar training stimuli or

overreached adaptation processes. In other words, the present subjects reached their limit in improving their neuromuscular performance with the power-only training protocol utilized in this investigation. Previous studies (Lyttle et al., 1996; Newton et al., 2002) have suggested that heavy resistance strength training together with power training may better affect improvements in performance than power training alone. Newton et al. (2002) demonstrated that mixed-methods resistance-training increased both muscle strength and power, while Lyttle et al. (1996) found that a combination of plyometric and weight training was superior in comparison with ballistic resistance training for increasing jumping performance.

The present power training protocol of 15 weeks did not result in any significant changes in muscle fiber type, muscle fiber size or MHC isoform composition. Progressive resistance training has been shown to increase muscle strength and size simultaneously with alterations in fiber contributions (e.g., Andersen & Aagaard, 2000). Furthermore, increase in the MHC IIA proportions induced by resistance training have been demonstrated but, on the other hand, our earlier study in women support the present finding that prolonged power training did not cause muscle hypertrophy (Häkkinen et al., 1990). Titin isoform composition was not altered in the present experiment as well. All the subjects and controls expressed the faster-mobility titin band, while the lower-mobility titin band was detected in half of the subjects in quite a low percentage. These results suggest that jumping exercise did not change titin isoform expression. Fry et al. (1997) partly support this suggestion showing that each studied subject expressed the same titin isoforms in VL, GA, and SOL, and both a 16-week aerobic rowing and a 6-week sprint cycling training protocols did not appear to affect titin isoform composition. Torsell et al. (2002) showed, however, that changes in passive tension could occur without changes in titin isoform expression in single fibers in unloaded rat SOL muscles. Interestingly one of subjects in the present and in our previous study (Kyröläinen et al., 2002) expressed the lower-mobility titin isoform considerable more than others did. Kyröläinen et al. (2002) observed that the runner, who expressed two titin isoforms, were the most economical compared with those runners, who expressed only the fast mobility titin isoform. Thus, further studies are needed to compare skeletal muscle elastic characteristics of individuals with two titin isoforms to other individuals, who express only one titin isoform. Regardless, in the longitudinal analysis performed in this study no titin isoform changes occurred and thus a potential influence or contribution of titin to the performance changes cannot be made.

The observed performance enhancement in jumping as a result of power training cannot conclusively be explained by changes in muscle structure and in neural adaptation of PF. Additional factors for explaining the performance enhancement in this study could be sought from some modifications in the joint control strategy and/or RFD capabilities of KE. In jumping, the increased use of KE may have resulted in an increase in absorbed negative energy during the braking phase and may have enhanced the respective positive energy in the following push-off phase leading to increased jumping performance. In addition, an improvement in muscle coordination (agonist vs. antagonist muscles) could play a role in increased joint power production. While EMGs of the VL and VM muscles increased slightly, the respective values of their antagonist muscle (BF) slightly decreased. Although all EMG changes were statistically insignificant, their combined effect may explain increased joint power around the knee joint.

Perspectives

Despite considerable improvements in jumping performance, MVC and RFD, the results from this investigation do not reveal any further information into possible neuromuscular mechanisms. It was surprising that despite a well-controlled exercise program the true changes in the most of measured parameters were difficult to detect. The present study confirms that mixed strength training methods could affect more clearly neuromuscular performance and its explanatory mechanisms than power training alone as Newton et al. (2002) and Lyttle et al. (1996) have demonstrated. Thus, future training studies should focus on developing new parameters besides the conventionally employed MVC and RFD. More detailed analysis of motor unit firing patterns in combination with attempts to clarify modification in the fine structure of the contractile and elastic muscle components may be the way to find true mechanistic explanations.

Key words: electromyography, force, jumping, titin.

Acknowledgements

The authors would like to express their gratitude to Mr. Seppo Seppälä, Mr. Markku Ruuskanen, Mr. Petri Järvinen, Mr. Tommi Tikka, Ms. Marja-Liisa Romppanen, Ms. Pirkko Puttonen, Ms. Aila Ollikainen and Ms. Riikka Kivelä for their assistance during the measurements and analysis. This study was supported by grant nos. 173/722/98 and 66/722/99 from the Ministry of Education, Finland. Finally, the authors declare that the experiments comply with the current laws in Finland.

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