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ORIGINAL ARTICLE

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Effects of power training on mechanical efficiency in jumping

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Abstract The present study investigates the effects of power training on mechanical efficiency (ME) in jumping. Twenty-three subjects, including ten controls, volunteered for the study. The experimental group trained twice a week for 15 weeks performing various jumping exercises such as drop jumps, hurdle jumps, hopping and bouncing. In the maximal jumping test, the take-off velocity increased from 2.56 (0.24) $m \cdot s^{-1}$ to 2.77 (0.18) $m \cdot s^{-1}$ ($P < 0.05$). In the submaximal jumping of 50% of the maximum, energy expenditure decreased from 660 (110) to 502 (68) $J \cdot kg^{-1} \cdot min^{-1}$ ($P < 0.001$) while, simultaneously, ME increased from 37.2 (8.4)% to 47.4 (8.2)% ($P < 0.001$). Some muscle enzyme activities of the gastrocnemius muscle increased during the training period: citrate synthase from 35 (8) to 39 (7) $\mu mol \cdot g^{-1}$ dry mass$\cdot min^{-1}$ ($P < 0.05$) and β-hydroxyacyl CoA dehydrogenase from 21 (4) to 23 (5) $\mu mol \cdot g^{-1}$ dry mass$\cdot min^{-1}$ ($P < 0.05$), whereas no significant changes were observed in phosphofructokinase and lactate dehydrogenase. In the control group, no changes in ME or in enzyme activities were observed. In conclusion, the enhanced performance capability of 8% in maximal jumping as a result of power training was characterized by decreased energy expenditure of 24%. Thus, the increased neuromuscular performance, joint control strategy, and intermuscular coordination (primary factors), together with improved aerobic capacity (secondary factor), may result in reduced oxygen demands and increased ME.

Keywords Economy · Electromyography · Enzyme · Force · Muscle

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Introduction

Human movement is economical when the submaximal oxygen uptake per unit of body mass required to perform a given task is small (Cavanagh and Kram 1985). Mechanical efficiency (ME) is high when energy expenditure is small compared with the mechanical work done (Kyröläinen et al. 1990). In jumping, ME was reportedly 38.7% in conditions where the amplitude of knee bending during the braking phase was small (Bosco et al. 1982). The respective value for jumping with a large amplitude of knee bending was 30.1%, and without any prestretch of knee extensor muscles the net ME was 19.7%. The ME has been demonstrated to be highest in pure negative (eccentric) work (49–59%), while in the pure positive (concentric) work ME is lowest (15–19%) (Aura and Komi 1986a; Kyröläinen et al. 1990). Where eccentric and concentric actions are combined, as in stretch-shortening cycle (SSC) exercises, the ME of its positive work was 35.5 (6.9)% (Aura and Komi 1986b), and the total ME varied from 37 to 54% (Kyröläinen and Komi 1995).

Studies on the effects of power training on movement economy, ME and muscle metabolism are limited. Earlier studies have demonstrated that endurance athletes have higher oxidative muscle enzyme activities as compared to their sprint type counterparts (Costill et al. 1976). In addition, mitochondrial enzymes, such as succinate dehydrogenase, have been shown to be highly correlated with maximal oxygen uptake ($\dot{V}O_{2max}$)(e.g., Costill et al. 1976) but, in contrast, individuals with

nearly identical $\dot{V}O_{2max}$ values can have a two-fold range in mitochondrial enzymes (Holloszy and Coyle 1984). Dawson et al. (1998) have shown that 6 weeks of short sprint training can improve endurance resulting in some alterations in muscle enzyme activities in fit subjects.

Jumping economy and mechanical efficiency have been shown to be closely associated, and power training has been demonstrated to increase economy, possibly due to increased tendomuscular stiffness when metabolic demands may decrease (Kyröläinen et al. 1991). In SSC exercises, such as jumping, the fast motor control of human movement is essential for powerful performance. Before the ground contact the extensor muscles are activated under the influence of a central motor program (Melvill-Jones and Watt 1971). The preactivity appears to be a preparatory requirement, both for the enhancement of EMG activity during the eccentric phase of the take-off, and for the timing of muscular action with respect to the ground contact (Moritani et al. 1991). Power training may increase preactivity, thereby creating better conditions for powerful reflex functions (Kyröläinen and Komi 1995). This leads to increased tendomuscular stiffness, where regulation of stretch reflexes play an important role (Houk 1979).

It can be hypothesized that if power training could be demonstrated to result in neural adaptations and improvements in motor control, it could then lead to decreased oxygen consumption (improved economy) and increased ME. At the same time, some changes in muscle structure (e.g., muscle fiber area and collagen) may appear but, however, no changes in muscle enzymes can be expected. Therefore, the purpose of the present study was to investigate the effect of power training on economy in jumping. The mechanisms for possible changes in economy were sought through observations of neuromuscular function and muscle enzymes.

Methods

Subjects

Thirteen untrained men [age 24 (4) years, height 1.78 (0.05) m, fat% 11.0 (4.6)] and ten untrained controls [age 25 (2) years, height 1.78 (0.07) m, fat% 8.6 (6.0)] volunteered as subjects for the study. All the subjects were informed of the possible risks of the experiment and they gave 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 squats, deadlifts, and abdominal and calf exercises. The training of leg extensor muscles included various types of SSC exercises such as jumping performances with a special sledge apparatus, drop jumps from the heights of 20–70 cm, jump squats (30–60% from the maximum), one-leg and two-leg hopping, and hurdle jumps. The jumping performances (5–10 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.

Protocol

Both groups were tested before and after the 15-week training period. Testing included jumping tests on a special sledge apparatus (Kyröläinen and Komi 1995; Kaneko et al. 1984) at maximal and submaximal intensities. The mass of the sledge was 33.2 kg and its inclination was 22.5° with respect to the horizontal line. In the sledge-jump performances the optimal dropping height was determined individually for each subject. This means that they were dropped from different heights to find out their best rising height (Komi and Bosco 1978). Thereafter, they performed 3–5 jumps with maximal effort from the individually predetermined optimal dropping height. Each condition involved 3–5 maximal SSC exercises (sledge jumps), in which the lowest knee angle was about 90°. Finally, 20 submaximal jumps were performed once in every 3 s (total duration 3 min) from the optimal dropping height, at an intensity of 50% of the single SSC maximum. Blood samples were drawn from a fingertip for blood lactate (B-La) analysis (Biochemica Boehringer, Mannheim, Germany) at rest, immediately after, and 5 min after the jumping bout.

Before and after the training period, needle biopsies (a sample size of 100–150 mg) were taken from the middle portion of the lateral gastrocnemius muscle. This muscle was selected because the present training was planned to load mainly the triceps surae muscle. 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 biopsy for analysis was mounted on Tissue-TEK (Miles, Elkhart, Ind., 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

In the sledge jumps, ground reaction forces (GRF) were measured with a force plate, which was placed perpendicularly to the sliding surface. Surface electromyography (EMG) activity from the vastus lateralis (VL), vastus medialis (VM), gastrocnemius, soleus and tibialis anterior muscles were recorded telemetrically (MESPEC 4000, Mega Electronics, Kuopio, Finland) with surface electrodes (Beckman miniature skin electrodes, 650437, Beckman Coulter, Ill., 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 (Biotel 99, Glonner, Germany; bandpass 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.

In the submaximal SSC exercises on the sledge, the expired gases were analyzed (SensorMedics Vmax 229, Yorba Linda, Calif., USA) continuously utilizing the breath-by-breath method. The instrument was regularly calibrated with known gas mixtures, and the measured values were corrected in STPD.

Analysis of sledge jumps

The raw EMG signals were first fullwave rectified, then integrated, and finally time normalized for preactivation, braking and push-off phases. The onset of the ground reaction force of each jump was used as a reference point to identify the beginning and the end of

the contact. The velocity records were used to identify the end of the braking phase. In addition, the fullwave rectified EMG signals were averaged to obtain muscle activity patterns by both individual and group. This phase-dependent averaging method (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 ground reaction force and finished 1,300 ms after that point.

Enzyme activity

A 10–20 mg sample of muscle tissue was freeze dried, dissected free of all visible blood, adipose and connective tissue, and viewed under a stereomicroscope. Approximately 2 mg of the dissected tissue was homogenised in 800 µl 0.3 M K_2HPO_4, 0.05% BSA, pH 7.7 and stored at −80°C preceding analyses. Spectrometric determination of NADH changes at 340 nm (Lowry and Passonneau 1972) was used for measuring citrate synthase (CS), short chain β-hydroxyacyl-CoA dehydrogenase (HAD), phosphofructokinase (PFK) and lactate dehydrogenase (LDH) activity. LDH activity was measured at 25°C and ×50 dilution in 1 mM pyruvat, 172 µM NADH, 0.02% BSA, 0.02 M imidazole, pH 7.0. CS activity was measured at 25°C and ×50 dilution in 100 µM acetyl-CoA, 0.5 mM NAD (free acid), 1 mM sodium malate, 8 $\mu g \cdot ml^{-1}$ malate dehydrogenase (1200 $U \cdot mg^{-1}$, Boehringer Mannheim), 2.5 mM EDTA, 10 mM Tris-HCl, pH 8.0. HAD activity was measured at 25°C and ×50 dilution in 50 µM acetoacetyl-CoA, 35 µM NADH, 0.06 mM EDTA, 40 mM imidazole, pH 7.0. PFK activity was measured at 25°C and ×20 dilution in 0.9 mM fructose-6-phosphate, 0.3 mM NADH, 0.9 mM ATP, 0.9 mM AMP, 18 $\mu g \cdot ml^{-1}$ aldolase (20 $U \cdot mg^{-1}$, Boehringer Mannheim), 7 $\mu g \cdot ml^{-1}$ glycerol-3-P-dehydrogenase (170 $U \cdot mg^{-1}$, Boehringer Mannheim), 7 $\mu g \cdot ml^{-1}$ triosephosphate isomerase (5000 $U \cdot mg^{-1}$, Boehringer Mannheim), 0.9 mM Na_2HPO_4, 1.8 mM $MgCl_2$, 0.9 mM mercaptoethanol, 0.045% BSA, 45 mM Tris-HCl, pH 8.1. Enzyme activities are expressed as µmol substrate$\cdot min^{-1} \cdot g^{-1}$ dry mass muscle tissue.

Statistical analysis

A repeated measures ANOVA was utilized to test interactions of the selected variables between the subject and control groups in different experimental conditions. Mean and standard deviations (SD) were calculated by conditions. Finally, Pearsońs correlation analysis was utilized to study relationships between the changes in different variables.

Results

In the maximal sledge jump, the performance of the experimental subjects, determined by their take-off velocity, increased from 2.56 (0.24) to 2.77 (0.18) $m \cdot s^{-1}$ ($P < 0.05$), while the contact time remained the same [0.362 (0.056) vs 0.363 (0.059) s]. In the control group, the take-off velocity was 2.56 (0.32) $m \cdot s^{-1}$ before the training period and exactly the same [2.56 (0.27) $m \cdot s^{-1}$] after that. Fig. 1 demonstrates, in addition, that only insignificant changes ($P > 0.05$) in muscle activity patterns of the investigated lower limb muscles were noticed in the maximal jumping condition. This was also true with respect to quantitative values of EMGs, both for the experimental group [e.g., mean of VL and VM during contact: 0.414 (0.132) vs 0.448 (0.165) mV] and for the control group [0.377 (0.169) vs 0.315 (0.134) mV].

In the submaximal jumping condition, the absolute mechanical work remained constant in a before–after comparison. Simultaneously, oxygen consumption decreased from 33.0 (5.0) to 23.7 (3.0) $ml \cdot kg^{-1} \cdot min^{-1}$ ($P < 0.001$) corresponding to energy expenditure changes from 660 (110) to 502 (68) $J \cdot kg^{-1} \cdot min^{-1}$ ($P < 0.001$), respectively. Thus, mechanical efficiency increased from 37.2 (8.4)% to 47.4 (8.2)% ($P < 0.001$) in the experimental group, while no statistically significant changes were observed in the control group [39.9 (6.3)% vs 36.6 (4.7)%]. In addition, no changes in GRFs or EMGs were observed in either group (Fig. 2), and the B-La values were also unchanged [2.18 (1.09) $mmol \cdot l^{-1}$ vs 2.56 (1.50) $mmol \cdot l^{-1}$].

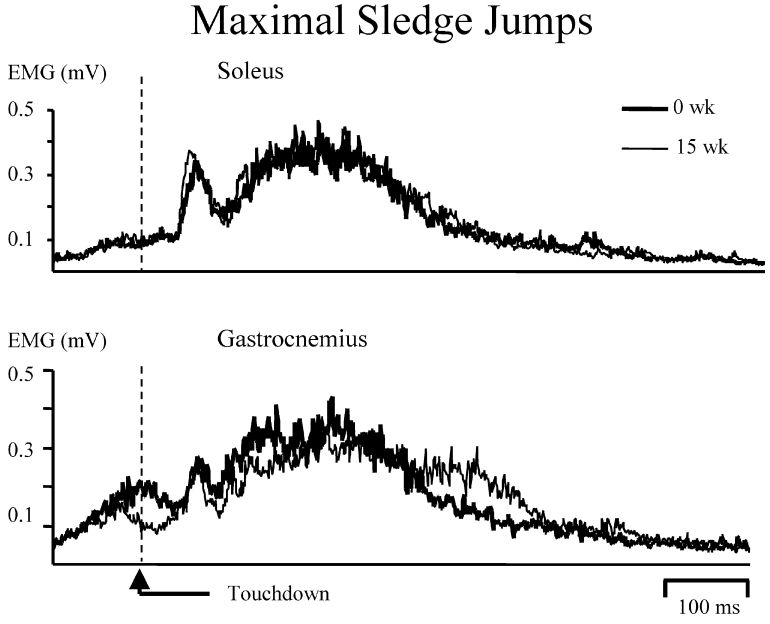

Fig. 1 Effects of power training on muscle activity patterns in the maximal sledge jumps before (*thick line*) and after 15 weeks of training (*thin line*). The *vertical dotted line* represents the beginning of the contact

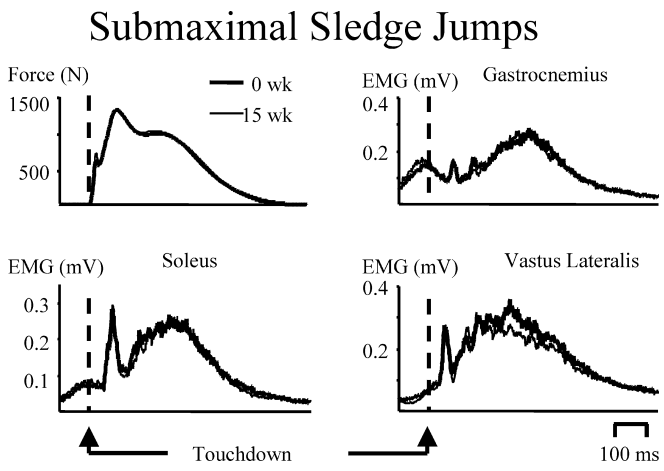

Fig. 2 Effects of power training on ground reaction force and on muscle activity patterns in the submaximal sledge jumps before (*thick line*) and after 15 weeks of training (*thin line*). The *vertical dotted line* represents the beginning of the contact

Some enzyme activities (Fig. 3), however, changed during the training period: CS from 35 (8) to 39 (7) μmol·g^{-1} dry mass·min^{-1} ($P < 0.05$) and HAD from 20 (4) to 23 (5) μmol·g^{-1} dry mass·min^{-1} ($P < 0.05$), while no statistically significant changes were observed in PFK and LDH. In the control group, no changes in muscle enzyme activities were observed.

Discussion

The enhanced performance capability of 8% in maximal jumping as a result of power training led to the improved ME of 24%. The activity of the muscle enzymes CS and HAD increased at the same time. However, no statistically significant relationships between changes in oxygen consumption or net energy expenditure and changes in those muscle enzyme activities induced by prolonged power training were observed.

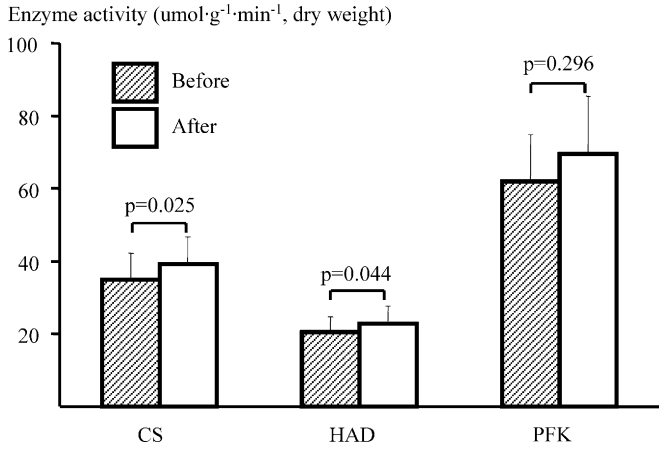

Fig. 3 Mean (SD) enzyme activities of citrate synthase (*CS*), short chain β-hydroxyacyl-CoA dehydrogenase (*HAD*) and phosphofructokinase (*PFK*) before and after the training period in the experimental group

The enhanced performance capabilities in jumping as a result of power training cannot be explained by changes in the measured muscle activity patterns or increased neural input to these muscles (Fig. 1). Any changes in muscle structure were noticed, and slow fiber types (Type I) dominated both before the training [59.6 (13.1)%] and after it [63.2 (16.5)%] (Kyröläinen et al., to be published). Thus, the most important factor for explaining the performance enhancement in this study seems to be some modification in the joint control strategy and / or an increased rate of force development by the knee extensor muscles (Kyröläinen et al., to be published). This may also partly explain improvements in jumping economy and ME. In addition, the present minor or negligible neural and muscular modifications may be due in part to the considerable (mean 6 h week^{-1}), primarily aerobic type, extra physical activity performed weekly by the subjects. However, its amount did not increase during the training period but was maintained on the same level as before. Thus, its role as an explanatory factor is minimal. Quite similar lack of physiological adaptation to power training has also been observed previously (McBride et al. 2001).

Although the jumping economy and ME improved, no changes were observed in force production or muscle activity patterns (Fig. 2), or in ground contact times. The possible stretch reflex peaks are well visible both before and after the entire training period. This is in contrast to our earlier findings where those peaks almost disappeared in VL and VM muscles induced by power training in females (Kyröläinen et al. 1991). Thus, because reflexes occurred similarly before and after the training period, it is likely that there was a significant change in tendomuscular stiffness. This suggestion is based on the study by Houk (1979) and the review article by Dietz (1992), in which reflexes from the muscle spindles and tendon organs are thought to play an important role in adjustments to external disturbances and thus regulating tendomuscular stiffness during the initial ground contact. In SSC exercises, these proprioceptive reflexes are functioning simultaneously with the voluntary neural system. In addition, other possible explanation for improved jumping economy could be sought by examining possible changes in the passive elastic structures of skeletal muscle, such as collagens.

In the present study, it is interesting to see that additional power training twice a week, combined with normal daily activities, was sufficient stimulus to increase the activities of the oxidative enzymes (CS and HAD) by 13–15%. The activities of PFK and LDH, glycolytic enzymes, did not change significantly ($P > 0.05$) in either of the groups. However, individual differences in the enzyme activities between the subjects were high (ranging from −26 to +61%). No changes in the analyzed muscle enzyme activities were noticed in the control group. These results are in slight contrast to the study of Dawson et al. (1998) who found that

6 weeks of short sprint training decreased CS activity and improved endurance. Linossier et al. (1993) reported, however, no change in CS activity after the repeated maximal 5 s sprint training. The present subjects, with high distribution of slow twitch fibers, improved their jumping economy simultaneously with increased oxidative enzyme activities (CS and HAD), suggesting enhancements in the aerobic capacity of the muscle fibers. Thus, it seems that the subjects learnt to better utilize their muscle fibers and this resulted in reduced oxygen demands and improved movement economy.

It seems that the rise in the levels of activity of the mitochondrial enzymes underlies the increase in the capacity to oxidize carbohydrates and fatty acids, and to generate adenosine triphosphate (ATP) in oxidative phosphorylation. Further support for the proposition of enhanced energy metabolism is provided by the observed increase in activity in HAD, a marker of fatty acid oxidative capacity. This means that the power training, together with other physical activity, caused enhancements in fatty acid oxidative capacities. It is likely that the changes in those oxidative muscle enzyme activities would have correlated positively with the respective changes in $\dot{V}O_{2max}$ but, unfortunately, this was not measured. In the submaximal jumping conditions the respective relationships were not statistically significant.

In conclusion, despite considerable improvement in jumping economy the results did not reveal true mechanism factors to explain this adaptation. Although the measured muscle oxidative enzymes increased their activities, the findings of improved ME may be more likely explained by the improvements in neuromuscular performance, joint control strategy, and intermuscular coordination.

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