Adaptations in skeletal muscle following strength training

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Costill, D. L., E. F. Coyle, W. F. Fink, G. R. Lesmes, and F. A. Witzmann. Adaptations in skeletal muscle following strength training. J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 46(1): 96–99, 1979. Five men were studied before and after 7 wk of isokinetic strength training to determine its effects on muscle enzyme activities and fiber composition. One of the subject's legs was trained using 10 repeated 6-s maximal work bouts, while the other leg performed repeated 30-s maximal knee extension exercises. The total work accomplished by each leg was constant. Training 4 times/wk achieved similar gains in peak torque for both legs at the training velocity (3.14 rad/s) and at slower speeds. Fatigability of the knee extensor muscles, as measured by a 60-s exercise test, was similar in both legs after training. Biopsy specimens showed significant changes in the % of the muscle area composed of type I and IIa fibers as a result of both strength training programs. In terms of muscle enzymes, only the 30-s exercise program resulted in elevated glycolytic, ATP-CP, and mitochondrial activities. Despite these changes, none of the parameters measured were found to be related to the gains in either muscle strength or fatigability during maximal isokinetic contractions.

ADAPTATIONS IN SKELETAL MUSCLE following endurance training are well documented (6, 10) and demonstrate an enhanced capacity for oxidative metabolism. Adjustments to strength training, on the other hand, are not so well defined. The intent of the present investigation is to assess the effects of two modes of maximal isokinetic training on muscle enzyme activities and fiber composition. A comparison has been made between training programs that required 6- and 30-s bouts of maximal isokinetic contractions of the knee extensor muscles. The muscle enzymes selected for study are representative of the metabolic systems responsible for energy derivation during anaerobic (i.e., creatine phosphokinase, myokinase, phosphorylase, phosphofructokinase, and lactate dehydrogenase) and aerobic (i.e., succinate dehydrogenase and malate dehydrogenase) muscular effort.

METHODS

Five healthy males served as subjects in this investigation. The age, height, and weight for the men averaged (±SE) 23.6 ± 1.3 yr, 177.2 ± 2.6 cm, and 77.3 ± 4.7 kg, respectively. All subjects were fully informed of the risks and stresses associated with this research before giving their written consent to participate.

Measurements. Testing and training of the knee extensor muscles were conducted with the aid of an isokinetic dynamometer (Cybex II, Lumex, Inc.) as previously described by Thorstensson (17). Before and after training, each of the subject's legs was tested for peak torque at knee extension velocities ranging from 1.05 to 5.23 rad/s. In addition, an anaerobic performance test was done with each leg. This test consisted of maximal knee flexions and extensions at 3.14 rad/s for 60 s. Work output was summed every 10 s during this test.

Four biopsies were obtained from the vastus lateralis muscle of each leg before and after training. One portion of the specimen was positioned in a mounting medium and frozen in isopentane cooled to the temperature of liquid nitrogen for histochemical analysis. The remaining biopsy material was weighed, frozen in liquid nitrogen, and later assayed for muscle enzyme activities.

The myofibrillar ATPase method (4, 16) was used for muscle fiber classification. Tissue slices (10 μm thick) were preincubated at a pH 4.3 and 4.6, and the staining reaction carried out at a pH 9.4. Muscle fibers were subsequently classified as types I (slow twitch oxidative), IIa (fast twitch, oxidative), and IIb (fast twitch, glycolytic). Fiber areas were determined from serial cross sections of each muscle sample after staining for NADH-diaphorase (15). The mean muscle fiber area for each sample was determined by averaging the cross-sectional areas of 20 fibers that had been photographically reproduced and measured by planimetry.

Enzyme analyses. Muscle samples analyzed for succinate dehydrogenase (SDH), total (cytoplasmic plus mitochondrial) malate dehydrogenase (MDH), lactate dehydrogenase (LDI), and total phosphorylase were first homogenized at 4°C in an 0.1 M triethanolamine (TEA) buffer (pH 7.6; 0.5% bovine serum albumin (BSA) 5 mM 2-mercaptoethanol). We have noted that the use of this buffer results in SDH activity that is 0.5 times that obtained with a PO4 homogenizing buffer. The proportionality of the SDH activity produced with the TEA and PO4 buffers is constant and does not alter the interpretation of the findings in the present study. The reaction media and fluorometric methods for these enzyme assays have been detailed earlier (3, 11).

Homogenates for phosphofructokinase (PFK) were prepared in 100 mM potassium phosphate buffer (pH
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8.2), containing 10 mM glutathione, 0.5 mM ATP, 5 mM MgSO₄, and 30 mM NaF (1). The fluorometric determination of NADH disappearance was performed at 25°C using a reaction cocktail described by Mansour et al. (12).

Creatine phosphokinase (CPK) and myokinase (MK) activities were determined fluorometrically on whole muscle homogenized in a TEA-BSA buffer (see SDH method above), containing 10 mM cysteine or 1.0 mM glutathione. The MK reaction was performed in a cocktail containing ADP (0.2 mM), MgCl₂ (5 mM), glucose (2 mM), NADP + (0.1 mM), 5 μg/ml hexokinase, and 1 μg/ml glucose-6-phosphate dehydrogenase. After determining the MK reaction rate, creatine phosphate (CP) was added to the mixture to measure the activity of CPK (11).

Training. The subjects trained each leg 4 times/wk for a period of 7 wk. One leg (6 s) was trained using 10 repeated 6-s maximal knee extensions, while the other leg (30 s) performed repeated 30-s maximal knee extension exercise. The rest intervals between the 6- and 30-s bouts was 114 s and 20 min, respectively. The 6-s leg was exercised first during each training session, and the work bouts for the 30-s leg were repeated until the total work performed was equal to that produced by the 6-s leg. All training bouts were executed at a velocity of 3.14 rad/s (180°/s). The rationale for selecting training bouts of 6 and 30 s was based on our efforts to stress both systems of anaerobic ATP generation (ATP-CP and glycolytic systems). In preliminary studies we noted little or no muscle lactate increase following 6 s of maximal isokinetic exercise (unpublished observations). The 30-s maximal work, however, increased muscle lactate from a resting level of 0.9 to 19.4 mmol/kg wet wt. These data suggest that the 6 s of maximal exercise was achieved principally at the expense of the ATP-CP systems, while the 30 s bout stressed both the ATP-CP and glycolytic systems.

Differences between means were tested for significance with a t test for paired observations.

RESULTS

As a result of training, the subjects showed a significant (P < 0.05) increase in peak torque for both legs (6 and 30 s) at the velocity used during training (3.14 rad/s) and at slower speeds. Improvements in peak torque ranged from a high of 14% (0 rad/s) to a low of 4% at 3.14 rad/s. No differences, however, were found between the strength gains of the 6-s and 30-s legs.

Figure 1 illustrates that during the 60-s maximal exercise test, both the 6-s and 30-s legs increased (P < 0.05) their mean power output as a result of training. This apparent training advantage was noted only during the first 30 s of the test. Thereafter, the slope of this "fatigue" curve and the mean power values were the same in both legs before and after training.

Table 1 presents the mean changes in muscle fiber characteristics measured before and after training. The percentage of type I, IIa, and IIb fibers did not change with either form of training. The muscle cross-sectional area represented by each fiber type, however, the type I fibers showed a decrease (P < 0.05) of 5.0 and 7.8% in the 6-s and 30-s legs, respectively. At the same time, the type IIa fibers in both legs increased significantly (P < 0.05). The type IIb fibers remained unchanged with training. Neither the 6 s nor 30 s training
in the 30-s leg, the muscle’s resistance to fatigue was the same as the 6-s-trained leg which showed no increase in muscle enzymes (Fig. 1 and Table 2). Although was that, while the muscle enzyme activities increased work performed by the muscle.

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LDH activity failed to increase with the 30-s training program.

DISCUSSION

There are 3 major findings of this research. The first is that two 30-s maximal exercise bouts/day (4 days/wk) are sufficient stimulus to increase the activities of muscle phosphorylase, PFK, CPK, MK, MDH, and SDH. When the same quantity of work, however, was divided into repeated 6-s exercise bouts, only PFK activity increased. Thus, with the exception of PFK activity, the stimulus responsible for increasing muscle enzyme activities seems to be related to the duration of each maximal exercise bout rather than the quantity of work performed by the muscle.

The second major observation in this investigation was that, while the muscle enzyme activities increased in the 30-s leg, the muscle’s resistance to fatigue was the same as the 6-s-trained leg which showed no increase in muscle enzymes (Fig. 1 and Table 2). Although an increase in glycolytic and ATP-CP enzymes is generally interpreted to reflect an enhanced anaerobic potential, the present data demonstrate that fatigue during repeated maximal contractions is independent of the muscle’s capacity to generate ATP via anaerobic metabolism. Despite the greater strength exhibited in the early seconds of the 60-s anaerobic performance test, mean power declined so rapidly that the trained legs exhibited the same mean power as the untrained legs after 30–40 s of effort (Fig. 1). One possible explanation for this greater rate of decline in the trained muscles is a more rapid depletion of muscle ATP and CP as a result of the increased energy requirements imposed by the higher power output in the trained muscles during the first half of the performance test. Recent studies (13), however, have demonstrated an increase in muscle ATP and CP concentrations with strength training, which could compensate for the greater energy demands at these higher power outputs.

Another possible explanation for the more rapid decline in mean power by the trained muscles might be attributed to the influence of pH on maximal muscular force. Hill (9) in 1940 demonstrated that the formation of lactic acid in response to stimulation stopped when muscle pH dropped to roughly 6.3. More recently Hermansen and Osnes (8) have measured the pH in exercising human skeletal muscle and have reported values as low as 6.35 at exhaustion. Because lowering cellular pH is known to slow the rate of glycolysis, it might be argued that the decrease in muscle pH which accompanied the anaerobic performance test could have limited ATP synthesis by reducing the effectiveness of the glycolytic enzymes. If this were the case, then one might expect to see a lesser rate of fatigue in the 30-s-trained leg, which shows the greatest enzymatic potential for glycolysis. The fact that both the 30- and 6-s-trained legs showed similar rates of fatigue suggests that the factor responsible for muscular exhaustion may be independent of ATP resynthesis.

Fuchs et al. (5) have suggested that increased hydrogen ion concentration in muscle reduces the binding capacity for Ca\(^{2+}\) through an inactivation of the fibrillar protein, troponin. Recent unpublished studies by Hermansen (personal communication) have demonstrated that in skinned rabbit muscle fibers, a decrease in muscle pH results in a marked loss of contractile tension, that persists despite the availability of ATP. Thus, the loss of mean power illustrated in Fig. 1 may be due, in part, to the effects of increasing hydrogen ion concentration on the muscle’s contractile mechanism, rather than on the glycolytic ATP resynthesis, per se.

It is interesting to note that during the final 30 s of the anaerobic performance test both the trained and untrained muscles produced the same mean power values. If we adhere to the concept that a decrease in pH was responsible for the decline in mean power, then we might speculate that its influence was greatest in the pH sensitive fast twitch (type II) muscle fibers (2). Thus, the rapid fatigue noted in the first 30 s of the test after training may be due to a decline in tension generated by the type II fibers. The mean power produced in the final 30 s of the test both before and after training may simply reflect the tension and lesser fatigability of the slow twitch (type I) fibers. This concept is supported by fatigue and muscle glycogen depletion studies conducted during repeated fast voluntary contractions in man (14, 19). These studies demonstrated that the decline in peak torque which accompanied repeated maximal contractions was positively correlated (r = 0.86) with the percentage of type II fibers in the contracting muscle.

The third principal finding of this research was that the biopsy specimens showed a significant (P < 0.05) change in the fiber composition of the vastus lateralis

**Table 2. Muscle enzyme activities before and after training**

<table>
<thead>
<tr>
<th>Enzyme</th>
<th>6-s Leg</th>
<th>30-s Leg</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
</tr>
<tr>
<td>Phosphorylase</td>
<td>18.3</td>
<td>17.4</td>
</tr>
<tr>
<td></td>
<td>±2.0</td>
<td>±1.6</td>
</tr>
<tr>
<td>Phosphofructokinase</td>
<td>64.5</td>
<td>66.1*</td>
</tr>
<tr>
<td></td>
<td>±3.2</td>
<td>±2.8</td>
</tr>
<tr>
<td>Lactate dehydrogenase</td>
<td>±22</td>
<td>±28</td>
</tr>
<tr>
<td>Creatine dehydrogenase</td>
<td>611</td>
<td>609</td>
</tr>
<tr>
<td></td>
<td>±14</td>
<td>±27</td>
</tr>
<tr>
<td>Myokinase</td>
<td>309</td>
<td>308</td>
</tr>
<tr>
<td></td>
<td>±18</td>
<td>±14</td>
</tr>
<tr>
<td>Malate dehydrogenase</td>
<td>41.0</td>
<td>46.0</td>
</tr>
<tr>
<td></td>
<td>±2.3</td>
<td>±2.5</td>
</tr>
<tr>
<td>Succinate dehydrogenase</td>
<td>4.0</td>
<td>4.1</td>
</tr>
<tr>
<td></td>
<td>±0.7</td>
<td>±0.6</td>
</tr>
</tbody>
</table>

Values are means ± SE, in μmol/g·min, for legs exercised with repeated 6-and 30-s maximal isokinetic contractions. * Significant (P < 0.05) difference between means of pre- and posttraining.
These studies of strength training demonstrate significant enzymatic changes and modifications in the composition (fiber area ratios) of skeletal muscle. Despite these adaptations, none of the parameters measured was found to be related to either muscle strength or fatigability during maximal isokinetic contractions. These data suggest that fatigue in maximal muscular effort is not dependent on the muscle's anaerobic potential as measured by glycolytic and ATP-CP enzyme activities.

This research was supported by National Institutes of Health Grant HL-20408; Lumex Inc.; and the Ball State University Research Committee Grant Res. 79-1976.

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Received 4 April 1978; accepted in final form 8 August 1978.

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