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Hormonal Adaptation Determines the Increase in Muscle Mass and Strength during Low-Intensity Strength Training without Relaxation

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Abstract—The study was designed to test the hypothesis that, during strength training, a restricted blood supply to the working muscles stimulates the secretion of anabolic hormones and an increase in the muscle mass and strength can be achieved with significantly lower training loads. During eight weeks, three times a week, 18 young, physically active males trained their leg extensor muscles. Nine subjects (group I) worked at 80% of the maximal voluntary contraction (MVC), whereas the rest (group II) performed their exercise without relaxation and at a lower load (50% MVC). The total training load in group II was significantly lower than in group I (77 ± 5 vs. 157 ± 7 kJ, respectively). The eight-week training of both groups significantly increased the mean maximum strength (by 35 and 21% in groups I and II, respectively) and volume (by 17 and 9%, respectively) of the muscles trained (however, the differences between the groups with respect to these changes were nonsignificant). Group I displayed a higher increase in the blood level of creatine phosphokinase than group II, while group II showed a greater increase in the blood concentration of lactate. In contrast to group I, group II displayed a significant increase in the blood concentrations of growth hormone, insulin-like growth factor 1 (IGF-1), and cortisol. Hence, the suggestion that the secretion of metabolic hormones is triggered by a metabolic, rather than mechanical, stimulus from working muscles seems plausible.

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INTRODUCTION

Strength training, aimed at increasing the strength of muscles, leads to the hypertrophy of muscle fibers, which is expressed as an increase in the mass of the muscle trained. Furthermore, this training can rearrange the entire regulatory scheme of the locomotor system [1–3]. At least two factors may be involved in the development of this hypertrophy. First, it may be induced by a direct mechanical influence exerted upon the working muscles. In this case, higher external loads should produce stronger effects. On the other hand, it may be induced by some products of tissue metabolism abundant in working muscles. Therefore, it seems reasonable to suppose that a restricted blood supply to a muscle during its training should intensify the training effect even at a rather low intensity of mechanical activity.

It should be noted that both mechanical activity and metabolic factors can initiate muscle hypertrophy either by direct action on muscle fibers or indirectly, through induced secretion of anabolic hormones. Which of these two factors (i.e., mechanical or metabolic) is more important for triggering the hormonal response to the activity of the muscles? To answer this question, we should take into account the opinion of McCall et al. that the increase in the blood concentration of growth hormone (GH) is related to the activity of muscle Ia afferent receptors [4]. In addition, intense strength training, accompanied by a rather high muscle tension, is also associated with intense synthesis of autocrine insulin-like growth factor 1 (IGF-1) [5]. On the other hand, it should be taken into consideration that increased concentrations of anabolic hormones are frequently explained by certain metabolic changes in the working muscles [1, 6, 7]. For example, controlled ischemia of the extremities was shown to potentiate the hormonal response during strength [8] and aerobic exercises [9, 10].

This study was designed to test the hypothesis that, during strength training, a restricted blood supply to the working muscles stimulates the secretion of anabolic hormones and, therefore, an increase in the muscle mass and strength can be achieved with significantly lower training loads. Usually, to restrict the blood supply to working extremities, some sort of occlusion cuffs are used [8, 11–13] or an increased pressure is applied to the lower half of the body placed into a pressure chamber [9, 10, 14], which is extremely inconvenient for training. Therefore, to restrict the blood supply to working muscles, we decided to use low-intensity exercise with incomplete relaxation [15, 16].

Table 1. Volumes of muscles and maximal voluntary contraction before and after eight weeks of strength training

Parameter	Group I (classic strength training)		Group II (strength training without relaxation)	
	before	after	before	after
Volume of the m. quadriceps femoris, cm^3	2155 ± 1175	2467 ± 147*	2209 ± 113	2320 ± 75*
Volume of the m. gluteus maximus, cm^3	907 ± 57	1064 ± 64*	859 ± 48	979 ± 51*
MVC displayed in exercise, N	410 ± 18	553 ± 29*	397 ± 29*	481 ± 28*

* Significantly different from the pretraining value, $p < 0.05$.

METHODS

The study was performed with 18 young, physically active males (age, 21 ± 2 years; body weight, 75 ± 3 kg; body height, 1.81 ± 0.10 m). All subjects gave their informed consent before taking part in the study. The study was approved and supervised by the Ethics Commission of the Institute of Biomedical Problems (the commission is subordinate to the National Committee on Biomedical Ethics of the Russian Academy of Sciences).

During eight weeks, three times a week, these subjects trained the extensor muscles of their knee and hip joints with an exercise machine for leg presses. Nine subjects (group I) were trained according to the classic scheme of strength exercise; the rest (group II) performed low-intensity exercise without relaxation (their muscles remained strained throughout the exercise because they did not move the working platform of the machine to its highest and lowest positions).

The weekly schedule of the classic training cycle (group I) consisted of three exercise sessions. The first, high-load, session (on Monday) was a developing one. Two toning-up sessions (on Wednesday and Friday) were significantly lower in volume. The developing session consisted of seven bouts with 10-min pauses for recovery. Each bout was performed at 80% of the maximal voluntary contraction (MVC; determined before and after each training cycle) to exhaustion (6–12 repetitions, 50–60 min). A toning-up session consisted of three bouts with the same pauses.

Similarly, three sessions were included in the weekly schedule of the low-intensity training without relaxation (group II). The developing session (on Monday) consisted of four sets of three bouts at a load of 50% MVC. Each 50- to 60-s bout was performed to exhaustion; the pauses between bouts and between sets were 30 s and 10 min, respectively. Toning-up sessions (on Wednesday and Friday) consisted of single sets.

The volumes of the quadriceps muscle (m. quadriceps femoris) and gluteus maximus muscle (m. gluteus maximus) were calculated from magnetic resonance (MR) spin echo cross-sectional scans (at 1.5 T and 17-mm intervals between scans) performed at the Myasnikov Institute of Clinical Cardiology (Moscow) by means of a Magnetom 63SP apparatus (Siemens, Germany). To avoid aberrations caused by the redistribution of internal fluids, the subjects remained recumbent for 15 min before each MR scan. The results are presented as the muscle volumes averaged for both legs.

Tests for lactate, GH, IGF-1, testosterone, and cortisol were performed in blood samples taken at weeks 2 and 7 of the training course, 2 h after a standard breakfast, immediately before and after the developing exercise. Blood samples for creatine phosphokinase (CPK) and its myocardial isozyme (CPK MB) were taken before and 18 h after exercise. The level of CPK muscle isozyme (CPK MM, released from striated muscle fibers) was calculated as the difference between the total CPK activity and the activity of its MB isozyme. Concentrations of hormones and enzymes were determined with the use of DSL test systems (United States).

In this paper, all data are presented as mean ± standard error of the mean. The significance of differences was determined with the Wilcoxon and Mann–Whitney tests at $p = 0.05$. Interrelations between variables were described with Pearson correlation coefficients.

RESULTS

Eight-week strength training significantly increased the strength (by 35 and 21% in groups I and II, respectively) and volumes of the quadriceps (15 and 6%) and gluteus maximus (18 and 13%) muscles (Table 1). However, these data showed no significant differences between the groups. Nevertheless, the total work performed during the eight-week training course was significantly lower in group II (77 ± 5 vs. 157 ± 7 kJ in group I, Fig. 1).

At the beginning of the training course (week 2), an exercise session increased the blood lactate concentration by 6.2 mmol/l (group I) and 10.8 mmol/l (group II); by the end of the course (week 7), this between-group difference became weaker (Fig. 2).

During week 2, group I displayed a 59% greater postexercise increase in the activity of CPK MM than group II (258 ± 69 vs. 152 ± 38 IU/l, respectively). However, by the end of the training course, a low-intensity exercise session (group II) produced a significantly lesser increase in the activity of this isozyme (65 ± 6 IU/l; $p < 0.05$); in contrast, group I displayed only a nonsignificant decrease (Fig. 2). In other words, by the

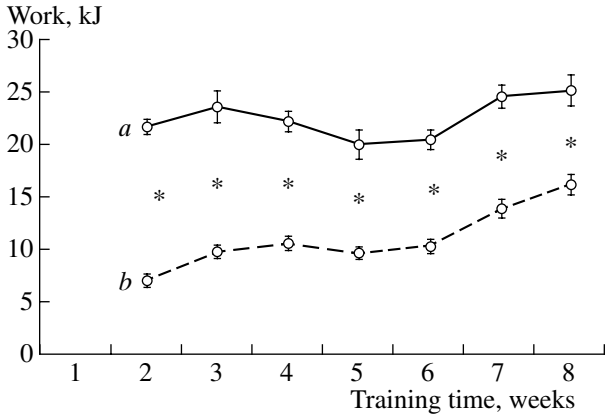

Fig. 1. Time course of the work performed in (*a*) group I (trained according to the classic scheme at a load of 80% MVC) and (*b*) group II (trained without relaxation at a load of 50% MVC). Asterisks indicate significant between-group differences ($p < 0.05$).

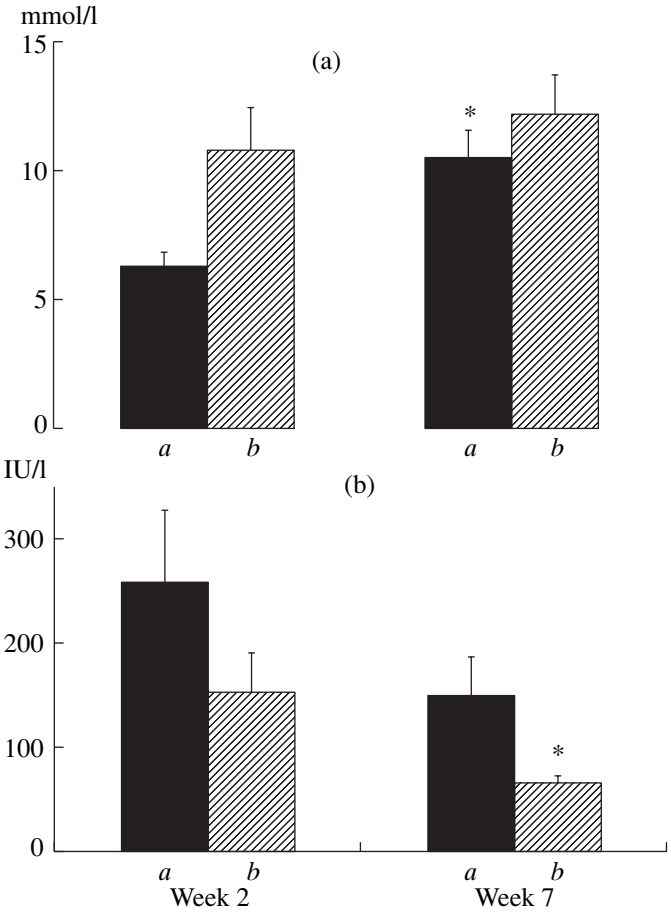

Fig. 2. Training-induced increases in the concentration of (a) lactate and (b) activity of creatine phosphokinase muscle isozyme at the beginning (week 2) and the end (week 7) observed in (*a*) group I (trained according to the classic scheme at a load of 80% MVC) and (*b*) group II (trained without relaxation at a load of 50% MVC). Asterisks indicate significant differences from the concentrations detected in the same group at week 2 ($p < 0.05$).

end of the training course, the classic training session (group I) resulted in a 129% higher increase in the activity of CPK MM than a low-intensity exercise session (group II).

Both at the beginning (week 2) and the end (week 7) of the training courses, exercise sessions resulted in an increased secretion of GH. This increase was more pronounced in group II (Table 2). Especially important is the fact that such a strong GH response persisted throughout the entire training course. Note that only in this group was the postsession increase in the blood IGF-1 level significant at weeks 2 and 7, whereas in group I it did not change at all (Table 2). The postsession changes in the concentrations of GH and IGF-1 recorded at week 2 showed a rather good correlation ($r = 0.88$, $p < 0.05$).

A significant end-of-the-course (week 7) postexercise increase in the total testosterone level was observed only in group I (Table 2).

A significant increase in the blood level of cortisol after the exercise was observed only in group II (throughout the training course, Table 2). By the end of the course, this group displayed a significant decrease in the testosterone/cortisol ratio, whereas group I displayed only a weak (nonsignificant) trend toward a decrease in this ratio.

DISCUSSION

Many researchers have shown that strength training performed at external loads below 60% MVC does not increase the MVC [11, 17]. In this study, however, we observed a significant increase in muscle mass and strength achieved via low-intensity (50% MVC) strength training performed without relaxation. These changes were somewhat lower than in the group trained according to the classic scheme of strength training (at 80% MVC) but did not differ significantly from them. This fact allows us to conclude that the training scheme with incomplete relaxation has a rather high hypertrophic potential.

These results can be explained by the assumption that the two training schemes produced different changes in the blood concentrations of lactate and CPK MM. Lactate is the end product of anaerobic glycolysis. Because the work performed during strength exercise (at 40- to 60-s maximum loads) is supplied with energy predominantly through the anaerobic pathway, the blood lactate level can serve as an indicator of the metabolic rates in the working muscles. As expected, a restriction of the blood supply was accompanied by a significant increase in the blood lactate concentration. At the beginning of the training course (week 2), an exercise session increased this concentration to 6.2 and 10.8 mmol/l (in groups I and II, respectively) (Fig. 2). By the end of the course, the difference between the groups became lower. Hence, in spite of the lower vol-

Table 2. Biochemical parameters of the blood at the beginning (week 2) and the end (week 7) of the training course before and after training sessions in groups I and II

Parameter	Group I (classic strength training)			
	2 week		7 week	
	before	after	before	after
Growth hormone, ng/ml	0.22 ± 0.02	3.47 ± 0.76*	0.20 ± 0.05	6.30 ± 2.82*
Insulin-like growth factor 1, ng/ml	277 ± 39	259 ± 29	276 ± 27	266 ± 30
Testosterone, ng/ml	4.0 ± 0.7	4.1 ± 0.8	3.5 ± 0.5	4.0 ± 0.6*
Cortisol, ng/ml	195 ± 19	259 ± 37	253 ± 22	341 ± 53
Testosterone/cortisol	0.021 ± 0.006		0.014 ± 0.002	
Parameter	Group II (strength training without relaxation)			
	2 week		7 week	
	before	after	before	after
Growth hormone, ng/ml	0.27 ± 0.03	9.09 ± 1.37*	0.20 ± 0.03	8.29 ± 1.41*
Insulin-like growth factor 1, ng/ml	320 ± 50	350 ± 47*	304 ± 17	372 ± 30*
Testosterone, ng/ml	2.8 ± 0.3	3.4 ± 0.6	2.3 ± 0.5	2.5 ± 0.6
Cortisol, ng/ml	154 ± 18	264 ± 29*	187 ± 14	358 ± 30*
Testosterone/cortisol	0.024 ± 0.006		0.013 ± 0.003**	

*Significantly different from the preexercise value, $p < 0.05$

**Significantly different from the preexercise value recorded at week 2, $p < 0.05$.

ume of work performed in the training with incomplete relaxation, the corresponding postexercise blood concentrations of lactate were higher than its concentrations after the classic training.

The presence of muscle CPK in the blood indirectly reflects the load-dependent damage to cell membranes in the working muscles. For example, any eccentric exercise produces higher blood levels of CPK than any concentric exercise [18–20]. At week 2 of our study, its increase was higher in group I (Fig. 2), which, most probably, is related to the higher training load than in group II, training at a low intensity without relaxation (80 vs. 50% MVC, respectively). Occlusion of the working extremity during low-intensity training does not increase the postexercise blood level of CPK MM [8]. By the end of the training course (week 7), the difference between the groups in the postexercise concentrations of CPK MM became even greater because of its significant decrease in group II, which was not accompanied by any significant changes in group I (Fig. 2). The lower postexercise increase observed at week 7 agrees very well with the fact that, in response to a standard load, trained subjects displayed a much lower increase in the concentrations of CPK MM than their untrained counterparts did [21]. A remarkable reduction in its increase after exercise was observed in the course of eccentric training, which can be explained by weaker damage to membranes (a consequence of adaptation of muscles to high loads) [22].

Hence, the changes in the blood levels of lactate and CPK MM observed for the two training schemes indicate that the classic scheme is associated with greater damage to muscle fibers, manifested through a higher blood level of this CPK isozyme. On the other hand, the exercise performed with incomplete relaxation is associated with a higher rate of glycolysis, which leads to higher blood concentrations of lactate. This means that the classic training scheme predominantly produces a mechanically dependent stimulus that can trigger anabolic processes in muscle fibers, whereas the training scheme with incomplete relaxation is associated with larger metabolic changes. Any explanation of the hypertrophic response observed in the latter case (if the restriction of the blood supply to the working muscles can be regarded as the main feature of this scheme) should necessarily involve the results of occlusion tests. For example, MR images showed that four weeks of aerobic bicycle ergometer training (45-min sessions at a load of approximately 20% MVC performed four times a week at an additional pressure of 50 mm Hg applied to the lower half of the body) increased the cross-sectional area (CSA) of a working muscle (predominantly, type I and IIB fibers) [9, 14]. The authors explain this hypertrophy by higher levels of contractile proteins and glycogen accompanied by hyperhydration. Similar results were obtained during eight-week ischemic (with a cuff pressure of 200 mm Hg) low-intensity strength training (twice a week, four sets of exercise at 50% MVC) of elite rugby players [11]. In

this case, the dynamic strength of the knee extensor muscles and their CSAs increased by 14 and 15%, respectively. Like the accompanying changes in the electromyographic activity, these phenomena are explained by a corresponding increase in the contractile protein content.

An increased secretion of GH and IGF-1 may be responsible for these effects. We found that the postexercise blood concentration of GH was higher in group II, although the mechanical effects exerted on the muscle fibers were significantly lower than in the classic scheme (Table 2). A similar increase in the concentration of GH was observed earlier during aerobic and strength exercises performed with controlled occlusion of veins [8, 10]. The authors of these papers attributed this effect to the activation of group III and IV afferents caused by the local intramuscular accumulation of some physiologically active metabolites that took place under the restricted blood supply (a phenomenon known as a metabolic reflex).

IGF-1 is the main mediator of the anabolic effects of GH. It is secreted both systemically (by GH-stimulated liver cells) and in the autocrine way (by skeletal muscle fibers during their high contractile activity) [5]. For example, intense synthesis of IGF-1 takes place in muscles after heavy strength training with a rather high mechanical tension [5]. Similarly, we detected increased blood concentrations of IGF-1 only after the exercise performed with incomplete relaxation. In contrast, the classic strength training performed with much higher mechanical loads caused no significant changes in its concentration. It should be noted that the postexercise increase in the IGF-1 concentration observed in the subjects trained with incomplete relaxation coincided with a higher secretion of GH (Table 2). The significant correlation ($r = 0.88$) between the postexercise increases in the concentrations of GH and IGF-1 observed during week 2 of the training course allows us to conclude that the increased concentration of IGF-1 resulted from the metabolic increase in the secretion of GH.

Remarkably, the low-intensity strength training stimulated not only the secretion of anabolic hormones but also the secretion of cortisol. Table 2 shows that, by the end of the training course, group II displayed a significant decrease in the testosterone/cortisol ratio compared to its level during week 2; a similar (although nonsignificant) trend toward a decrease was observed in group I. This indicates that the physiological cost of this training load is rather high [23].

CONCLUSIONS

This work provides the first evidence that low-intensity exercise (at 50% MVC) without relaxation leads to greater secretion of anabolic hormones than the more intensive classic strength exercise (80% MVC). Presumably, this phenomenon, like the effect produced by muscle ischemia, is caused by stronger effects of metabolic stimuli in the muscle during its training with incomplete relaxation necessarily associated with a restricted blood flow. The eight-week strength training performed according to the classic scheme or to the scheme with incomplete relaxation resulted in rather similar gains in muscle strength and volume. Our data allow us to attribute the positive effect of the low-intensity training performed without relaxation to metabolically stimulated secretion of anabolic hormones; the mechanism of this phenomenon requires further, more detailed analysis.

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