Journal of Human Kinetics volume 13, 2005, 17-26

The Influence of Eccentric and Concentric Muscle Contractions on Concentration of Growth Factors in Strength Trained Athletes

by

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The aim of this study was to compare the growth factors (testosterone, growth hormone, insulin growth factor and insulin growth factor binding protein-3) concentrations and physiological responses during eccentric and concentric muscle contractions in strength trained athletes. A group of nine subjects performed an incremental exercise test requiring concentric and eccentric muscle contractions. Serum growth hormone (GH), insulin-like growth factor (IGF-I) and insulin-like growth factor binding protein-3 (IGFBP-3) were assessed, before and after both types of exercise. The concentrations of IGF-I and GH tended to be higher during exercise than at rest and reached the level of statistical significance at maximal exercise intensity ($p<0.05$ and $p<0.01$). In summary, the data from this investigation demonstrates that the increase in serum IGF-I concentration occurs after incremental arm exercise but the changes are independent of endogenous growth hormone responses. Therefore this data supports the importance of local variables in the magnitude of the IGF response system. The direct mechanism is more pronounced during eccentric exercise what could stimulate muscle mass and strength after this type of muscle contractions.

Key words: *eccentric exercise, hormone, insulin-like growth factor-I*

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Introduction

Intense physical training exerts a sequence of molecular events that induce protein synthesis and muscle hypertrophy in athletes. However, recent research suggests that exercise with a predominance of eccentric muscle contractions can induce more profound adaptive changes in skeletal muscle than those obtained during concentric work (Dudley et al. 1991, Hather et al. 1991, Murayama et al. 2000). It seams that eccentric work stimulates greater increase of fast-twitch fibers and strength than that of concentric training. This muscle hypertrophy is caused by a long term increase in protein synthesis and a decrease of protein breakdown. It was hypothesized that in exercising muscles the number of myofibrils as well as actin and myosin filaments increased, what would provide more cross-bridges for force production during maximal muscle contraction (Newham et al. 1983, Tipton and Wolfe 1998, Sorichter 2002). On the other hand, current theories acknowledge that muscle injury after eccentric training leads to satellite cell activation and proliferation. These cells migrate to the damaged muscle where they are involved in myofibril regeneration. The repair process of cell membranes after intense exercise with an eccentric component, such as in weight lifting, can require more glucose availability what may impair glycogen resynthesis (Hawke and Garry 2001, Schwane et al. 1983). In addition, some evidence suggests that eccentrically exercised muscle is less sensitive to insulin, what could delay muscle glycogen storage (Hather et al. 1991, Willmore and Costill 2004).

As has been previously described, physical performance with eccentric muscle contractions is important in maximizing muscle strength and size. The growth promoting effect is also a result of direct biological stimulation of endogenous anabolic hormones and endocrine factors. One possible mechanism for exercise induced muscle hypertrophy is a high growth hormone secretion (GH), which promotes hepatic production of insulin-like growth factor (IGF-I) (Borst et al. 2001, De Vol et al. 1990). This factor stimulates muscle IGF type receptors to increase protein synthesis, but their activation depends on binding proteins, IGFBP, which prolongs the half life of IGF-I and modifies its interaction with their receptors (Di Luigi 2002, Schwarz et al. 1996). Throughout exercise, circulating levels of both anabolic and catabolic hormones increase with intensity (Kraemer et al. 1995). Testosterone (T) and growth hormone (GH) are known to promote hypertrophy, while increased concentration of corticosteroids (C) after exercise accelerate protein degradation (De Vries et al. 2003). Due to these effects, insulin and growth factors increase to antagonize the negative effects of cortisol on both synthesis and degradation pathways of protein turn-

over. Intense endurance training has been shown to elevate circulating GH and IGF-I in young subjects, but this mechanism is not well understood. It is difficult to explain the relationships between the type of muscular contraction (eccentric and concentric) and growth factor concentrations in trained athletes.

The aim of this study was to compare the growth factors (testosterone, growth hormone, insulin growth factor and insulin growth factor binding protein-3) concentrations and physiological responses during eccentric and concentric muscle contractions in strength trained athletes.

Material and Methods

Nine healthy, male subjects took part in the study. Their basic characteristics include (age 21,64 ± 1,39 years, body mass 74,87 ± 10,06 kg, body height 173,5 ± 5,25 cm, BMI 24,85 ± 3,19). All of the subjects were informed of the purpose and nature of the investigation and gave their written consent to participate. The study was approved by the Ethics Committee at the Academy of Physical Education in Katowice.

First the subjects performed an incremental arm cycling ergometer exercise with graded intensity (CEx). This test requires predominantly concentric muscle contractions (shortening of elbow flexors). In the second test the subjects performed an incremental arm exercise with eccentric contractions (EEx). The intensity of eccentric work was displayed on a digital indicator to motivate the subjects to generate the required force. Both tests started at a power output of 30 W with 15 W increments every three minutes until voluntary exhaustion which was determined when the subject could not maintain the required pedaling frequency. HR, BP, VO_2, $ExCO_2$ and VE were measured from the 6th min prior to exercise, and during each exercise load until the tests were completed. Gas exchange variables were measured continuously breath-by-breath using the Oxycon apparatus (Jaeger, Germany) and maximal work load was recorded. During the exercise, heart rate was continuously controlled using the PE-3000 Sport-Tester (Polar Inc. Finland).

Serum growth hormone (GH), insulin-like growth factor (IGF-I) and insulin-like growth factor binding protein-3 (IGFBP-3) were assessed using immunoradiometric assay (IRMA) kits. Cortisol (C) and testosterone (T) were measured by radioimmunoassay kits obtained from Diagnostic System Laboratories (Webster, Texas). Blood lactate concentration (LA) was measured by an enzymatic method using commercial kits (Boehringer, Manheim, Germany). Creatine kinase (CK) and lactate dehydrogenase (LDH) activity was measured using an enzymatic method (Analco).

At the time of blood draw, subjects had abstained from food and had not engaged in strenuous physical activity within the previous day. Blood samples were obtained from the finger tip and ulnar vein before and during exercise (at one minute intervals) for determination of lactate concentration. Serum GH, T, C, IGF-I, IGFBP-3 and CK, LDH activity were measured before exercise, at maximal intensity and one hour after the test.

The significance of differences for all variables (W_{max}, LA, VO_2, HR, GH, T, C, IGF-I, IGFBP-3, and CK, LDH activity) between eccentric and concentric exercise test protocols were determined using the Students-t test. The relationships between hormone concentrations and physiological variables were checked by Spearman correlation coefficient regression analysis in each exercise test. Statistical significance was set at $p<0.05$.

Results

Maximal power output and physiological variables changed significantly when different types of muscular contractions were applied (Tab. 1). Maximal oxygen uptake and power output were lower during eccentric muscle contractions in comparison to concentric arm exercise. Mean values of pulmonary ventilation and heart rate at maximal exercise intensity were higher during concentric exercise than in eccentric one ($p<0.01$).

The concentrations of IGF-I and GH tended to be higher during exercise than at rest and reached the level of statistical significances at maximal exercise intensity ($p<0.05$ and $p<0.01$) in both exercise tests (Tab. 2). A tendency for lower cortisol levels and higher testosterone concentration was found after eccentric exercise. There were significant differences in mean serum concentrations of testosterone ($p<0.05$) and growth hormone ($p<0.01$) after one-hour of recovery between eccentric and concentric exercise. A significant correlation was found between serum IGF-I concentration and oxygen uptake during exercise. The correlation coefficients for this relationship reached $r=0,66$ ($p<0.05$).

Blood lactate concentration increased significantly in both exercise tests but was higher during concentric exercise at maximum power output and at each exercise load ($p<0.001$). There was a tendency towards higher CK activity during eccentric exercise and significant differences were observed after one hour of recovery in relation to concentric exercise ($p<0.05$). Serum LDH activity increased significantly and this difference was more pronounced one hour after eccentric arm exercise (Fig.1). A significant correlation was found between serum IGF-I concentration and LDH activity ($r=0,67$; $p<0.05$) and the activity of LDH and GH concentration ($r=0,69$; $p<0.05$) in eccentric exercise and GH and CK activity in concentric exercise ($r=-0,80$; $p<0.01$).

Table 1

Maximal power output and physiological variables measured before exercise (BEx), at maximal intensity of concentric exercise (CEx) and eccentric incremental exercise (EEx)

Variable	BEX	CEx	EEx
W_{max} [W]	0	152,2 ± 16,0	145 ± 22.5*
VO_{2max} [ml·min^{-1}]	7,4±1,01	32,75 ± 7,09	19,2 ± 2,55***
HR_{max} [b·min^{-1}]	68,0 ± 8,0	188 ± 9,0	145 ± 18,0**
LA_{peak} [mmol·l^{-1}]	1,77 ± 0,32	9,15 ± 2,54	3,22 ±1,26***
VE [l·min^{-1}]	11,05 ± 1,06	101,5 ± 13,9	40,81 ± 11,06***
pH	7,4± 0,03	7,28 ± 0,04	7,42 ± 0,02**

* Significant differences between concentric and eccentric exercise

VO_{2max} = maximal oxygen uptake; W_{max} = maximal power output; HR_{max} = maximal heart rate; LA_{peak} = peak blood lactate concentration; VE = pulmonary ventilation.

Table 2

Changes in hormonal concentrations before exercise (BEx), at maximal intensity of concentric exercise (CEx) and eccentric incremental exercise (EEx)

Variable	BEx	CEx	BEX	EEx
T [nmol·l^{-1}]	15,94 ± 4,81	17,18 ± 5,36*	20,1 ± 3,38	19,02 ± 4,64*
GH [ng·ml^{-1}]	0,14 ± 0,09	9,73 ± 4,29***	0,16 ± 0,06	6,21 ± 2,29**
C [nmol·l^{-1}]	378,6 ± 178,0	618,2 ± 252,5	369,9 ± 65,9	438,8 ± 145,3
IGF-I [ng·ml^{-1}]	359,1 ± 67,9	460,9 ± 109,4*	417,0 ± 60,8	478,1 ± 72,6*

* Significant differences between rest hormone concentrations and at maximal intensity of concentric and eccentric exercise

T = testosterone; GH = growth hormone; C = cortisol; IGF-I = insulin-like growth factor.

Table 3

Differences in hormonal concentrations during maximal intensity of concentric (CEx) and eccentric incremental exercise (EEx) and after 1-hour of recovery (R)

Variable	CEx	EEX	p
T [nmol·l^{-1}]	17,18 ± 5,36	19,02 ± 4,64	n.s
T_R [nmol·l^{-1}]	13,06 ± 3,88	18,01 ± 4,97	0.05
GH [ng·ml^{-1}]	9,73 ± 4,29	6,21 ± 2,29	n.s
GH_R [ng·ml^{-1}]	9,98 ± 3,77	0,48 ± 0,15	0.01
C [nmol·l^{-1}]	618,2 ± 252,5	438,8 ± 145,3	n.s
C_R [nmol·l^{-1}]	506,9 ± 231,2	390,9 ± 84,2	n.s
IGF-I [ng·ml^{-1}]	460,9 ± 109,4	478,1 ± 72,6	n.s
IGF-I_R [ng·ml^{-1}]	346,0 ± 40,2	369,4 ± 86,0	0.05

* Significant differences between concentric and eccentric exercise

T = testosterone; GH = growth hormone; C = cortisol; IGF-I = insulin-like growth factor

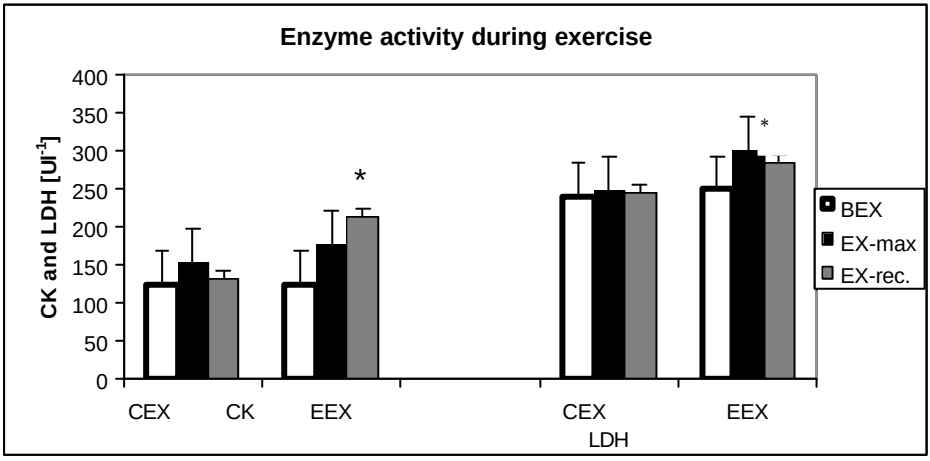

Fig. 1.

Changes in CK and LDH activity before exercise (BEx), during maximal intensity of concentric (CEx) and eccentric exercise (EEx) and after 1-hour of recovery (Ex-rec.)

** Significantly greater than in concentric exercise*

Discussion

The results of the presented study indicate that incremental physical exercise causes a significant increase in circulatory activity of IGF-I and GH concentrations. However, eccentric exercise has been shown to stimulate IGF secretion to a greater extent than concentric exercise. In contrast, GH concentrations were more pronounced after concentric exercise. The impact of incremental exercise on IGFBP-3 concentration was measured because it alters IGF-I level. A tendency to increase the IGFBP-3 concentration was observed in exercise without statistical significance. As previously reported, physical activity influences the growth-promoting effects of GH as a result of biological effects of the hormone on muscle metabolism and also from the impact of the insulin like growth factors and their binding proteins (Booth et al.1998, De Vries et al. 2003). As a result, cell proliferation and anabolism of tissue increases. Despite the fact that physical exercise stimulates IGF-I secretion, there is conflicting data about the role of this factor in exercise dependant muscle hypertrophy (Kraemer et al. 2000, Sorichter 2002). The discrepancies between different studies are probably due to the many factors (age, gender, training type and intensity, nutrition, hormonal statues and perhaps the type of muscle contraction) that influence the IGF system. In addition, recent findings suggest that the eccentric component of training stimulates an increase of muscle fiber area approximately ten times greater than that with only concentric contractions (Hortobagyi 1996, Murayama et al. 2000). The endocrine factors are thought to be responsible for these changes by promoting muscle growth (Leifke et al. 2000, Tipton and Wolfe 1998). However, the higher concentration of IGF-I after exercise and one hour recovery in strength trained athletes was not related to anabolic hormone concentrations. This demonstrated that the increase of IGF-I secretion during incremental exercise is independent of endogenous GH responses. Therefore, it was hypothesized that the increase of IGF-I secretion is more related to mechanical stimulation (damage of muscle fibers) and local factors than GH influence. Available data indicates a significant correlation between age-related changes of serum sex hormones and insulin-like growth factor and their binding globulin levels in men (Leifke et al. 2000). On the other hand, concentration of testosterone, growth hormone and IGF-I were unaffected by the training program in older men. The absence of such relationships during exercise is controversial. Additionally, the physiological responses for exercise induced increase in serum growth factors may be sensitive to lactate changes, high acidity or structural damage during high intensity training. Kraemer (2000) demonstrated that high intensity cycle and treadmill exercise (above the lactate threshold) stimulated the IGF system greater than lower intensity exercise. He

hypothesized, that the IGF-I and IGFBP-3 secretions are more sensitive to high lactate concentration than other measures of the acid base status, such as hydrogen ions, which are more related to post-exercise GH concentrations.

The eccentric cycling exercise used in this study produced lower maximal work capacity, lower values of VO_{2max} and blood lactate concentration. The greater serum CK and LDH activity suggested muscle membrane damage after this type of exercise. It has been documented that decreased muscle force and high plasma CK activity reflect structural damage to muscle fibers and accompanying muscle soreness (Evans and Cannon 1991, Schwane et al. 1983). The eccentric exercise applied in this study induced an increase in CK and LDH activity and reduced oxygen extraction from arterial blood. However, these changes were different from those of concentric arm contractions shown in previous studies (Hather et al. 1991, Hoffman et al. 1996). Upper limb exercise imposes much more stress on the cardiovascular system due to greater sympathetic activation and slow venous blood return from non-exercising muscles (Casabury et al. 1992, Faria and Faria 1998, Rostein and Meckel 2000). During eccentric muscle contraction physiological variables are determined mostly by local, not endocrine factors (Kraemer e al. 2000, Schwane et al. 1983). It has been also reported that after eccentric exercise, glycogen resynthesis decreases (Hather et al. 1991, Murayama et al. 2000). In addition, some evidence suggests that eccentrically exercising muscles are less sensitive to insulin, which would limit muscle fiber uptake of glucose (Willmore and Costill 2004). A decrease in aerobic power and lactate concentration during eccentric exercise in relation to concentric arm exercise was observed in this study. Possible causes of these phenomena include low resistance, greater increase in muscular utilization of O_2 and higher sympathetic activation during concentric arm exercise (Tordi et al. 2001, Sorichter 2002).

Conclusions

The data from this investigation demonstrates that the increase in serum insulin-growth factor concentration occurs after incremental arm exercise but the changes are independent of endogenous growth hormone responses. Therefore this data supports the importance of local variables in the magnitude of the IGF response system. The direct mechanism is more pronounced during eccentric exercise what could stimulate significant increases in muscle mass and strength after this type of muscle contractions.

References

Aminoff T., Smolander J., Korhonen O., Louhevaara V. 1998. Prediction of acceptable physical work loads based on responses to prolonged arm and leg exercise. Ergonomics 41(1), 109-120.

Booth F.W., Tseng B.S., Flük M., Carson J.A. 1998. Molecular and cellular adaptation of muscle in response to physical training. Acta Physiol. Scand. 162, 343-350.

Borst S.E. et al.2001. Effects of resistance training on insulin-like growth factor and IGF binding proteins. Med. Science. Sports Exerc.v.33, (4), 648-653.

Casaburi R., Barstow T.J., Robinson T., Wasserman K. 1992. Dynamic and steady-state ventilator and gas exchange responses to arm exercise. Med. Science Sports Exerc. 24 (12), 1365-1374.

De Vol D., Rotwein P., Sadow J.L., Novakovsky J., Bechtel P.J. 1990. Activation of insulin-like growth factor gene expression during work induced muscle hypertrophy. Am. J. Physiol. 259, E89-E95.

De Vries W.R., Schers T.J., Ait Abdesselam S. et al. 2003. Involvement of endogenous growth hormone-releasing hormone (ghrh) in the exercise-related response of growth hormone. Int. J. Sports Med. 24, 208-211.

Di Luigi L. 2002. IGF-I, IGFBP-2, and -3: do they have a role detecting rhGH abuse in trained men? Med. Science. Sports Exerc.v.34, (8), 1270-1278.

Dudley G.A., Tesch P.A., Miller B. J, Buchanan P. 1991. Importance of eccentric actions in performance adaptations to resistance training. Aviation, Space, and Environmental Medicine. 62, 543-550.

Evans W.J., Cannon J.G. 1991. The metabolic effects of exercise induced muscle damage. Exercise Sport Science Reviews. 19, 99-125.

Faria E.W., Faria I.E. 1998. Cardio respiratory responses to exercise of equal relative intensity distributed between the upper and lower body. J. Sports Science. 16 (4), 309-315.

Hather B.M., Tesch P.A., Buchanan P., Dudley G. A. 1991. Influence of eccentric actions on skeletal muscle adaptations to resistance training. Acta Physiol. Scand. 143, 177-185.

Hawke T.J. Garry D.J. 2001. Myogenic satellite cells: Physiology to molecular biology. J Appl. Physiol. 91, 534-551.

Hoffman M.D., Kassay K.M., Zeni A.I., Clifford P.S. 1996. Does the amount of exercising muscle alter the aerobic demand of dynamic exercise? Europ. J Appl. Physiol. Occup. Physiol. 74 (6), 541-547.

Hortobagyi T., Hill J.P., Houmard J.A., Fraser D.D., Lambert N.J., Israel R.G.1996. Adaptive response to muscle lengthening and shortening in humans. J Appl. Physiol. 80, 765-772.

Kraemer W.J. Aguilera B. A., Terada M. et al. 1995. Response of IGF-I to endogenous increases in growth hormone after heavy resistance exercise. J.Appl. Physiol. 79,1310-1315.

Kraemer W.J. et al. 2000. Effects of exercise and alkalosis on serum insulin-like growth factor I and IGF-binding protein-3. Can. J. Appl. Physiol. 25 (2), 127-138.

Leifke E., Gorenoi V., Wichers C., Mühlen A., Brabant G. 2000. Age-related changes of serum sex hormones, insulin-like growth factor-1 and sex-hormone binding globulin levels in men: cross-sectional data from a healthy male cohort. Clin. Endocrinol. 53, 689-695.

Murayama.M., Nosaka.K., Yoneda T., Minamitani K. 2000. Changes in hardness of the human elbow flexor muscles after eccentric exercise. Eur J Appl Physiol. 82, 361-367.

Newham D.J. MacPhail G., Mills K.R., Edwards R.H.T. 1983. Ultrastructure cha nges after concentric and eccentric contractions of human muscle. J Neurol Sci. 61, 109-122.

Nosaka K., Clarkson P.M. 1996. Variability in serum creatine kinase response after eccentric exercise of the forearm flexors. Int. J. Sports Med. 17, 120-127.

Rostein A., Meckel Y. 2000. Estimation of % VO2 reserve from heart rate during arm exercise and running. Eur. J. Appl. Physiol. 83(6), 545-550.

Schwane J.A. Johnson S.R., Vandenakker C.B. Armstrong R.B. 1983. Delayed-onset muscular soreness and plasma CPK and LDH activities after downhill running. Medicine Science Sports and Exercise. 15, 51-56.

Schwarz A.J, Brasel J., Hintz R.L, Mohan S., Cooper D.M. 1996. Acute effect of brief low- and high-intensity exercise on circulating insulin-like growth factor (IGF) I, II and IGH-binding protein-3 and its proteolysis in young healthy men. J. Clin. Endocrinol. Metab. 81, 3492-3497.

Sorichter S. 2002. Biochemical markers of skeletal muscle disease. Skeletal muscle pathology, diagnosis and management of disease. ed by V. Preedy, T. Peters. vol.44, 483-491.

Tipton K.D., Wolfe R.R. 1998. Exercise-induced changes in protein metabolism. Acta Physiol. Scand. 162, 377-387.

Tordi N., Belli A., Mougin F., Rouillon J.D., Gimenez M. 2001. Specific and transfer effects induced by arm or leg training. Int. J. Sports Med. 22 (7), 517-524.

Willmore J.H., Costill D.L. 2004. Physiology of sport and exercise. Human Kinetics, Champaign III. 92-110.