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Physiological elevation of endogenous hormones results in superior strength training adaptation

Running head: ***"elevated hormone levels and strength training"***

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ABSTRACT

The purpose of this study was to determine the influence of transiently elevated endogenous hormone concentrations during exercise on strength training adaptations. Nine subjects performed four unilateral strength training session per week on the elbow flexors for 11 weeks. During two of the weekly sessions, leg exercises were performed to acutely increase the systemic anabolic hormone concentration immediately before the exercises for one of the elbow flexors (*L+A*). On the two other weekly training sessions, the contralateral elbow flexors were trained without prior leg exercises (*A*). By randomizing one arm of the subjects to serve as a control and the other as experimental, both conditions have the same nutritional and genetic environment. Serum testosterone and growth hormone was significantly increased during the *L+A* training session, while no hormonal changes occurred in the *A* session. Both *A* and *L+A* increased 1RM in biceps curl, peak power in elbow flexors at 30% and 60% of 1RM, and muscle volume of the elbow flexors ($p<0.05$). However, only *L+A* achieved increase in CSA at the part of the arm flexors with largest cross sectional area ($p<0.001$), while no changes occurred in *A*. *L+A* had superior relative improvement in 1RM biceps curl and favorable muscle adaptations in elbow flexors compared to *A* ($p<0.05$). In conclusion, performing leg exercises prior to arm exercises, and thereby increasing the levels of serum testosterone and growth hormone, induced superior strength training adaptations compared to arm training without acute elevation of hormones.

Key words: PLASMA TESTOSTERONE, GROWTH HORMONE, 1RM, PEAK POWER

INTRODUCTION

Strength training increases muscle size and strength due to combinations of multiple and integrating factors, i.e. mechanical stress, metabolic demands, endocrine activities, and neuromuscular control (Harridge 2007). A well known endocrine response to a bout of strength training is an acute and relatively short lasting increase in circulating levels of anabolic hormones (e.g. testosterone and growth hormone (GH)), and the catabolic hormone cortisol (Kraemer et al. 1990). The findings of transient increases in GH and testosterone during and after a strength training session has led to strength training strategies which maximizes the acute hormonal response to strength training by using multiple exercises, multiple sets, heavy loads, and relatively short rest periods (i.e. Fleck and Kraemer 2004; Kraemer and Ratamess 2004; ACSM 2009; Kraemer et al. 2002).

Although training regimes eliciting acute elevations in testosterone and GH are well adapted within some strength training milieus (e.g. “testosterone boosting exercise” used by bodybuilders), the documentation for a possible additive effect of training with transient increases in testosterone and GH are equivocal. The basal effects of androgens on muscle mass are, however, well documented; e.g. physiological levels of androgens are necessary for normal strength and muscle adaptations to strength training (Kvorning et al. 2006) and supraphysiological doses of testosterone induces muscle hypertrophy (Bhasin et al. 1996; 2005; Storer et al. 2008). Furthermore, androgen receptor antagonists, which inhibit testosterone from binding to the androgen receptor, impair muscle growth during synergist overload (Inoue et al. 1994). Consequently, the role of the acute hormone responses to exercise can theoretically be important because anabolic hormones, i.e. testosterone and GH, can increase protein synthesis in muscle cells (Fryburg and Barrett 1993; Ferrando et al. 1998). Therefore, exercise-induced stimulation of the endocrine system may be a trigger for additive adaptation processes in skeletal muscle cells, leading to increased content of contractile proteins. In fact, testosterone is considered the major promotor

of muscle growth and subsequent increase in muscle strength and in response to strength training in men (Vingren et al. 2010).

It has been suggested that the acute response of GH to strength training may be most prominent for tissue remodeling (Kraemer and Ratamess 2005). Nevertheless the acute increase of GH to strength training has been found to correlate with the magnitude of muscle fiber hypertrophy during a period of strength training (McCall et al. 1999). This relationship could indicate that the transient increase in GH during each exercise bout has a positive effect on the cellular adaptations to strength training. Strength training programs that elicit the greatest acute testosterone and GH response are however, similar to programs which elicit the greatest cortisol response (Kraemer and Ratamess 2005). In peripheral tissue, cortisol increases protein degradation and decreases protein synthesis in muscles cells resulting in greater release of amino acids into circulation. Furthermore, it has been observed that physiologically elevation of cortisol may reduce the activity of the hypertrophy promoting pathway protein kinase B (Akt) (Spiering et al. 2008). Because of its possible role in tissue remodeling, acute changes of cortisol during strength training is often examined (Kraemer and Ratamess 2005).

It has been suggested that the acute hormonal response induced by a single strength training session is an important contributor to muscle hypertrophy during long-term strength training (Kraemer et al. 1990; Kraemer and Mazzetti 2003; Vingren et al. 2010). However, there is no consensus regarding the long-term effects of the transient exercise induced increase in anabolic hormones on strength training adaptations. It has, however, been observed that acute increases in circulating factors have an additive effect on strength training adaptations (Hansen et al. 2001; Madarame et al. 2008) and that acute elevation in endogenous testosterone (by strength training) potentiates the androgen receptor response to a strength training session (Spiering et al. 2009). On the other hand, it has been observed increased CSA and strength after strength training without any acute elevations in circulatory hormones (Wilkinson et

al. 2006) and that exposure of loaded muscle to exercise-induced elevations in endogenous anabolic hormones do not enhance strength training adaptations (West et al. 2010). The latter may be related to the observations that physiological elevation of cortisol may reduce the activity of the hypertrophy promoting pathway protein kinase B (Akt) (Spiering et al. 2008). The reason to this discrepancy in the literature remains unclear. However, the results of West et al. (2010) may be affected by the order of the exercises, since the exercises which were supposed to acutely elevate the circulatory hormones were performed after the exercises of the target muscles (elbow flexors). Therefore, in the present study the exercise order was reversed, meaning that the legs are exercised first, and therefore the elbow exercises were performed simultaneously as the hormone levels were elevated.

Consequently, the purpose of this study was to investigate the influence of transient exercise induced elevations in endogenous hormone concentration preceding exercising elbow flexors on muscle strength, power, and hypertrophy responses to strength training. Due to the potent effect of testosterone and GH in stimulating protein synthesis, we hypothesized that an acute increase in the levels of anabolic hormones, induced by performing leg exercises before arm exercises would induce superior strength training adaptations in arm muscles compared to the effect when training arm exercises only.

METHODS

Subjects

Eleven untrained male subjects (20-34 yrs, weight: 79±3 kg, height: 181±3 cm) volunteered for the study, which was approved by the Southern Norway regional division of the National Committees for Research Ethics in Norway. All subjects signed an informed consent form prior to participation. None of the subjects had performed any strength training during the preceding 6 months. Two of the subjects did not complete the study due to illness during the intervention period and their data are excluded.

Experimental design

The tests were conducted at the start (pre-intervention) and the conclusion (post-intervention) of the 11-week intervention. The subjects performed four unilateral strength training sessions per week on the elbow flexors. During every second session three leg exercises were performed immediately before three exercises for one of the elbow flexors to acutely increase the systemic anabolic hormone concentration (*L+A*). On the two other weekly training sessions the contralateral elbow flexors were trained with the identical protocol as the *L+A*, but without prior leg exercises (*A*). Whether the dominant arm should be trained after the leg exercise or not was randomized. By randomizing one arm of the subjects to serve as a control and the other as experimental, both conditions have the same nutritional and genetic environment. Hormonal response to the *L+A* and *A* training program was measured in the 5th week of the training intervention.

Testing

The pre- and post-intervention tests were divided into two separate test days. Magnetic resonance tomography (MR) for determination of cross sectional area (CSA) and muscle volume of the elbow flexors was performed on one day. All subjects performed pre-and post-tests in strength and power on another day with the same test order: 1) maximal strength (1RM) in elbow flexors, 2) maximal strength

(1RM) in knee extensors, 3) peak power output in elbow flexors. The subjects were instructed to refrain from intense exercise the day preceding a test and to consume the same type of meal before testing. They were not allowed to eat during the hour preceding a test or consume coffee or other products containing caffeine during the last three hours before a test. The pre- and post-intervention tests were performed at the same time of the day to avoid circadian variance, and the post test was carried out 3-5 days after the last strength-training session.

Maximal strength

Maximal strength in right and left elbow flexors was measured as 1 repetition maximum (1RM) in biceps curl. Strength tests were always preceded by a 10-min warm-up on a cycle ergometer. Prior to the baseline test, two familiarization sessions were conducted with the purpose of instructing the subjects in proper lifting technique and testing procedure. Subjects were seated with the testing arm supported on a bench. The test was conducted using a cable pulley, and to get the lift accepted the weight had to be lifted from full extension in the elbow to full flexion of the elbow with supination of the forearm during the lift and without any compensatory movements from the body. This procedure was repeated to determine the 1RM to the closest 1.25 kg. The rest period between each attempt was 3 min. After testing 1RM in biceps curl, maximum strength in the leg was tested as 1RM in leg press. The model of leg press utilized was a 45 degree angled hip sled (Gym 80 International, Gelsenkirchen, Germany), in which plates were loaded on each side of a foot platform. The depth of leg press in the 1RM test was set to a knee angle of 90°. To ensure similar knee angles during pre- and post-tests, the subject's leg press depth was carefully monitored and marked on a scale on the leg press machine. Thus, each subject had to reach his individual depth marked on the scale for the lift to be accepted. Similarly, the placement of the feet was monitored for each subject to ensure identical test positions during all tests. Before testing 1RM in elbow flexors and leg extensors the subjects performed a standardized and specific warm-up protocol consisting of 3 sets with gradually increasing load (40%, 75%, 85% of predicted 1RM) and decreasing

number of repetitions (10, 7, 3). The first 1RM attempt was performed with a load approximately 5% below the predicted 1RM load. After each successful attempt, the load was increased by 2-5% until the subjects failed to lift the same load after 2-3 consecutive attempts. The pre- and post-intervention tests were conducted using the same equipment with identical positioning of the subject relative to the equipment and monitored by the same investigator. The coefficient of variation for test-retest reliability for the 1RM test has previously been shown to be ≤5% (Rønnestad et al. 2007) and was <3% in the present study.

Peak power output

After the 1RM test in leg press, peak power output in right and left elbow flexors was tested with the same equipment and positions as in the 1RM test. Peak power was measured using a load of 30% and 60% of 1RM biceps curl. Between each trial there was 3 minutes of rest. The best result out of 3-4 trials for each load was used in statistical analysis. The same absolute load as in the pre-test was used in the post-test. The subjects were instructed to execute a maximal contraction. The peak power output was assed using a Muscle lab (Ergotest Technology AS, Langesund, Norway). The Muscle lab measures power output by a linear position transducer. The time spent in the concentric phase, as well as the work distance, was measured. Using a similar method to measure peak power in our lab, the coefficient of variation has been reported to be <4% in repeated examinations in 17 subjects (Rønnestad 2009).

Elbow flexor cross-sectional area and muscle volume measurement

Magnetic resonance tomography (MR) (Magnetom Avanto 1.5 Tesla, Siemens AG, Munich, Germany) was used to measure CSA of the elbow flexors. Subjects were scanned in the supine position. The scanned arm was stretched behind head, and centred in the middle of the machine. Nine cross sectional images from caput humeri against elbow joint were taken with 35 mm interslice gap. Each image represented a 5 mm thick slice. The resolution of the MR machine was 320 pixels x 256 pixels. The

images were subsequently uploaded to a computer for further analysis. The analyser was blinded for what kind of training that had been performed. The CSA of the arm flexors was measured at the four most distal images (image 6-9), where image 6 and 7 was defined as the proximal part and image 8 and 9 was defined as the middle part. Using a similar method, the coefficient of variation has been reported to be 2% in repeated examinations in eight individuals (Moss et al. 1997) and was <2% in the present study. The volume *V* of the muscular portion between every two consecutive scans was calculated from the equation:

$$V = 1/3 \times [a + \sqrt{(ab + b)}] \times t$$

where *a* and *b* are the CSA of the elbow flexors in the two scans and *t* is the inter-scan distance (length) between the adjacent areas. The volume of the elbow flexors from scan 6 to scan 9 was calculated by summing up all of the inter-scan muscular volumes.

Hormonal analysis

The hormonal response was analyzed in serum samples drawn from an antecubital vein during two training sessions in the 5th week of the intervention. The decision of measuring the hormonal response in the middle of the intervention period and not pre and post was based on previous findings of no large changes in acute hormonal response during a short term strength training period (Athiainen et al. 2003). During the *L+A* session 8.5 ml blood was sampled before the session started, immediately after the leg exercises, immediately after the arm exercises, and 30 min after the session was finished. During the *A* session blood samples were taken before the start of the session, immediately after the arm exercises, and 30 min after the session. The blood samples were taken in the time interval from 10:00 to 14:00. Therefore, the blood samples were obtained at different times during the day among subjects, but at the same time of the day for each subject for the *L+A* and *A* condition to limit influence of diurnal

variations. Two hours before the first blood sample subjects ate the same meal for both the *L+A* and *A* condition.

All hormone analyses were performed at the Hormone laboratory at Aker University Hospital, Oslo, Norway. Total testosterone in serum was analysed with a commercial competitive radioimmunoassay (RIA) from Orion Diagnostica (Espoo, Finland). The coefficient of variation (CV) in this kit is less than 6%. Cortisol was analysed with a commercial competitive luminoimmunoassay (LIA, CV<8%) from Siemens Medical Solutions Diagnostics (Los Angeles, USA) and growth hormone was analysed with a commercial non-competitive immunofluorometric assay (IFMA, CV<5%) from PerkinElmer Life Sciences, Wallac Oy (Turku, Finland).

Training

At the start of each strength training session, subjects performed a ~10-min warm-up at self-selected submaximal intensity on a stationary cycle ergometer. In the *L+A* sessions, the general warm-up was followed by two warm-up sets in leg press with gradually increasing load. The leg exercise protocol was based on prior studies showing that involvement of a large muscle mass, high in volume, moderate to high in intensity, using short rest intervals; tend to produce the greatest acute hormonal elevations (Raastad et al. 2000; Smilios et al. 2003; Kraemer and Ratamess 2005). Leg exercises performed were: leg press, knee extension, and knee-flexion. Three sets with 10RM and 60-90 sec rest between sets were performed on all leg exercises. Subjects were instructed to relax their arms while performing leg exercises. The elbow flexor training exercises were similar for both the *L+A* and *A* sessions: two warm-up sets in biceps curl with gradually increasing load were followed by biceps curl, hammer curl, and biceps curls with pronated forearm. During the first five weeks, subjects trained with 10RM sets at the first weekly session and 8RM sets at the second weekly session. During the final six weeks, sets were adjusted to 8RM and 6RM for the first and second weekly sessions, respectively. Subjects were

encouraged to continuously increase their RM loads throughout the intervention period and they were allowed assistance on the last repetition. The number of sets in each exercise on the elbow flexors was always two. The heavy strength training on both arms and legs was conducted with the concentric phase and eccentric phase lasting around 2-3 s. Since the present study had a within-person design and thus both test conditions had the same nutritional environment, there was no strict control of the nutritional practice. However, the participant was encouraged to consume proteins and carbohydrates during and after each bout of exercise throughout the intervention. All subjects were supervised by an investigator at all workouts during the first two weeks, and thereafter at least once every week throughout the intervention period.

Statistics

All data in the text and figures are presented as mean±SE. Pre- and post-intervention measurements for each training mode were compared using paired Student's *t*-test. To test for differences between *A* and *L+A* at pre-intervention, paired Student's *t*-tests were used. To test for differences in relative changes (from pre- to post-intervention) between *A* and *L+A* in, paired Student's *t*-tests were performed.
For each training mode, changes in hormones were compared using one-way repeated ANOVA. If there was significant difference, a Tuckey´s HSD test was pre selected for post hoc analysis. In serum testosterone there was a statistical power of 80% to detect difference from before the leg exercises to after the leg exercises of 2.8 $nmol \cdot l^{-1}$, using a significance level (alpha) of 0.05 (two-tailed). Two-way repeated measures ANOVA (time of intervention period and section where CSA was measured as factors) with Bonferroni post hoc tests were performed to evaluate differences (post- vs. pre-values) in elbow flexor CSA in all measured sections (section 6-9) within training mode. In addition, CSA along the elbow flexors, values for each section were analyzed by a two-way repeated measures ANOVA (group and section where CSA was measured as factors) with Bonferroni post hoc tests for evaluation of differences in relative changes (post- vs. pre-values) between training modes. One-way ANOVA was performed using GraphPad InStat (GraphPad Software, Inc. CA, USA), Two-way ANOVA analyses

were performed in GraphPad Prism 5 (GraphPad Software, Inc. CA, USA), and Student's t-tests were calculated in Excel 2003 (Microsoft Corporation, Redmond, WA, USA). All analyses resulting in $p \leq 0.05$ were considered statistically significant, except for multiple comparisons (1RM, muscle volume, peak power at 30% and 60% of 1RM) where $p \leq 0.013$ was considered significant due to Bonferroni correction.

RESULTS

Baseline

There were no significant differences between the arms which performed the *L+A* and *A* training before the intervention period with respect to 1RM, CSA, peak power at 30% and 60% of 1RM (Table 1).

(Insert Table 1 approximately here)

Body weight and training load

There was no significant change in body weight from before to after the training intervention. Both *L+A* and *A* increased their training load in the elbow flexor exercises during the intervention period ($p<0.01$) with no significant difference between them (Figure 1).

(Insert Figure 1 approximately here)

Hormonal response

Immediately after the leg exercise, significant increases in serum testosterone and GH were observed in the *L-A* session ($p<0.05$), while these increases were not evident in the *A* session (Figure 2). Serum cortisol did not change significantly in either session, although there was a tendency of increased values

after the arm exercises in the *L+A* session (p=0.16). Hormone values approached pre session values 30 min after the *L+A* session with no significant difference between the *L+A* and *A* session (Figure 2).

(Insert Figure 2 approximately here)

Maximum strength and body weight

During the intervention period the 1RM in leg press was increased by 23±4% (p<0.001, Figure 3). Strength, measured as 1RM in biceps curl, increased in both *A* and *L+A* arm (from 39.2±2.1 kg to 44.7±2.7 kg, and from 37.5±2.8 kg to 45.3±2.2 kg, respectively, p<0.001). The *L+A* training resulted in superior relative improvement in 1RM biceps curl compared to *A* training (p<0.05, Figure 4).

(Insert Figure 3 and 4 approximately here)

Peak power

Peak power in elbow flexors at 30% of 1RM increased from before to after the intervention in both *A* and *L+A* (from 94±6 W to 107±9 W, and from 96±8 W to 112±7 W, respectively, p<0.05), with no difference between groups (Figure 5, upper panel). Both *A* and *L+A* increased peak power in elbow flexors at 60% of 1RM (from 115 ±10 W to 135±13 W, and from 112±9 W to 149±12 W, respectively, p<0.05). There was no statistical significant difference between *A* and *L+A* (p=0.11; Figure 5, lower panel).

(Insert Figure 5 approximately here)

Elbow flexor muscle cross-sectional area

Both *L+A* and *A* increased their volume of the elbow flexors from section 6 to 9 (from

144.9±7.0 cm^3 to 159.5±7.9 cm^3, and from 144.9±7.8 cm^3 to 158.0±8.9 cm^3, respectively, $p<0.001$). ANOVA analyses revealed that both groups increased the CSA of the two proximal sections, while only *L+A* increased the elbow flexors´ CSA at the two middle sections where the CSA of elbow flexors was largest ($p<0.05$, Figure 6).

(Insert Figure 6 approximately here)

Discussion

In agreement with our hypothesis, transient elevations in endogenous hormone concentrations during exercise potentiated adaptations to strength training. Specifically, performing leg training before arm training in the same session induced elevations of circulating testosterone and GH and resulted in superior relative improvement in 1RM biceps curl compared to the changes observed in the arm trained without preceding leg exercises. Furthermore, training the arms under exercise induced elevations in anabolic hormones augmented the increase in CSA at the part of the elbow flexors with the largest CSA.

The finding of acute increase in testosterone, GH, and tendencies for increased cortisol during the *L+A* training, which contained multiple exercises involving large muscle mass, multiple sets, heavy loads, and relatively short rest periods, is in line with previous studies (e.g. Kraemer et al. 1990; Häkkinen and Pakarinen 1993; Gotshalk et al. 1997; Raastad et al. 2000; Ratamess et al. 2005). Furthermore, the finding of no change in hormonal levels during the *A* training, which included low muscle mass and low training volume, is also in line with previous findings (Häkkinen and Pakarinen 1993; Gotshalk et al. 1997; Ratamess et al. 2005). We do not know exactly how long the trained muscles are in recovery mode and we can not exclude the possibility that the leg training-induced increase in hormones may affect the adaptations in the *A* arm as well. However, if that is the case we still find significant differences between the *L+A* arm and *A* arm, so at least the effect is larger in the *L+A* arm than in the *A* arm. Furthermore,

acute elevations in endogenous testosterone potentiate the androgen receptor expression to a single strength training session and thereby improve the testosterone-receptor interaction (Spiering et al. 2009). This may be an important factor in explaining why passive tissue does not get bigger when blood is being circulated through the body with higher hormones and has classically been shown in studies where upper body training does not impact lower body musculature (Kraemer et al. 1995).

The ~21% increase in 1RM biceps curl in *L+A* is in line with other studies using similar strength training intervention, while the ~14% increase in *A* is slightly below the expected improvement (McCall et al. 1996; Moss et al. 1997; Hansen et al. 2001; McBride et al. 2003). The observations of superior gains in 1RM biceps curl and CSA in the *L+A* arm indicates that the acute elevations in circulating hormones contributes to strength training adaptations. The findings of no difference between training modes in improvement of training loads was somewhat surprising, but are probably explained by central fatigue (due to residual fatigue from the leg exercises). The majority of subjects also expressed their subjective feelings of reduced abilities to exert force when the arm exercises where preceded by leg exercises. The latter is also supported by the findings of reduced training volume when exercises involving large muscle mass precede exercises involving small muscle groups (Bellezza et al. 2009). The present findings indicate that the effects of acute elevations of hormones are not solely systemic. Muscles ability to interact with the circulating levels of endogenous hormones seems to be important (Harridge 2003). The majority (~98%) of testosterone in blood is bound to either sex hormone binding-globulin or albumin (Hayes 2000), therefore only the unbound testosterone is considered to be biologically active and able to diffuse across the sarkolemma and interact with intracellular receptors. The increased testosterone level observed after strength training exercise is typically not associated with changes in sex hormone-binding globulin (Fry et al. 1998; Fahrner and Hackney 1998). Furthermore, acute increase in plasma testosterone has been observed to increase the level of biological active unbound testosterone (Kraemer et al. 1998; Durand et al. 2003) and thereby potentially increase the interaction between androgen hormones and androgen receptors.

Androgen receptor mRNA and protein have been observed to increase after 1-3 strength training sessions where levels of serum testosterone was acutely elevated (Bamman et al. 2001; Willoughby and Taylor 2004; Hulmi et al. 2008; Spiering et al. 2009). Furthermore, administration of testosterone (without strength training) increases the content of androgen receptors in animal muscles (Carson et al. 2002; Lee et al. 2003) and humans (Ferrando et al. 2002). This is in line with the suggestion that androgens increase their receptor expression (Bhasin et al. 2001; Esposito et al. 2002; Gobinet et al. 2002). Furthermore, a correlation has been observed between the content of androgen receptors in the m. vastus lateralis and 1RM (Ratamess et al. 2005). These findings indicate that the content of androgen receptors may contribute to strength changes during strength training. It has recently been found that acute elevation in endogenous testosterone (by strength training) potentiates the androgen receptor response to a strength training session compared to no acute elevation of endogenous testosterone (Spiering et al. 2009). It might therefore be speculated that the acute elevations of testosterone in *L+A* increased the androgen receptor expression and via improved testosterone-receptor interaction, increased protein synthesis, and consequently, superior strength training adaptations was achieved.

Administration of GH has not been shown to increase muscle growth and strength in normal exercising men (Yarasheski et al. 1992; Frisch 1999). However, it has been hypothesized that the brief rise in GH can cause interaction with muscle cell receptors and thereby aid in subsequent recovery and stimulates muscle hypertrophy (Kraemer et al. 1998). Furthermore, the acute increase of GH to strength training has been found to correlate with the magnitude of muscle fiber hypertrophy (McCall et al. 1999). It has been suggested that GH and cortisol are involved in the regulation of the mRNA expression of IGF-I and myostatin (Rennie et al. 2004). This is in line with the findings of increased IGF-IEa and mechano growth factor expression after GH treatment in older subjects (Hameed et al. 2004) and myostatin has been shown to be up regulated in response to elevated serum glucocorticoids (Lang et al. 2001; Ma et al.

2003). However, reviews of the metabolic effects of GH on human skeletal muscle concludes that the balance of evidence suggests there are no major anabolic effects of exogenous recombinant human GH in stimulating muscle protein accretion, muscle size, muscle strength, or muscle fiber characteristics in normal, healthy adult men or women, including the elderly (Rennie 2003; Liu et al. 2007; 2008). However, GH exists in many different isoforms other than the 22 kDa measured in this study and this is also the isoform used in most studies on GH effects (Kraemer et al 2010). Consequently, other isoforms of GH released in response to exercise might have more pronounced effects on muscle tissue. Furthermore, we should not rule out the possibility that the concurrent acute increase in testosterone and GH may lead to a synergetic positive effect on strength training adaptations.

Strength training programs that elicit the greatest cortisol response are similar to programs which elicit the greatest acute testosterone and GH response (Kraemer and Ratamess 2005). While chronic high levels of cortisol have adverse effects, acute elevations may be part of a larger remodeling process in muscle tissue (Kraemer and Ratamess 2005). It has been observed that physiological elevation of cortisol may reduce the activity of the hypertrophy promoting pathway protein kinase B (Akt) (Spiering et al. 2008). The latter could potentially negatively affect the hypertrophic response to the *L+A* training. However, in the present study we observed only a small and statistically non-significant elevation in cortisol.

The increase in volume of the elbow flexors by ~10% in both *L+A* and *A* is in agreement with the 10-12% increase in CSA of elbow flexors in comparable studies (McCall et al. 1996; Walker et al. 2004). However, the finding of no statistical significant increase in CSA at the part of the elbow flexors with the largest CSA in *A* was not expected and the reasons remain unknown. However, the findings of regional differences in CSA adaptations to strength training are not unusual (e.g. Roman et al. 1993; Häkkinen et al. 2001). The findings of increased CSA at the part of elbow flexors with largest CSA in

L+A could be due to the acute elevation of anabolic hormones during the strength training. Furthermore, it has been observed that the largest CSA of a muscle determines the maximum strength of the muscle (Bamman et al. 2000; Klein et al. 2001), and consequently the increase in CSA in the *L+A* arm is the likely mechanism behind the superior 1RM improvement in *L+A*. The findings of increased CSA and strength after strength training without any acute elevations in circulatory hormones are in agreement with the findings of Wilkinson et al. (2006). They observed that unilateral leg training induced local muscle hypertrophy only in the exercised limb, which occurred in the absence of changes in systemic hormones. This is probably due the fact that hormonal changes is not the solely mechanism inducing muscle hypertrophy, for example are mechanical tension and local growth factors important in hypertrophic signaling (e.g. Goldberg et al. 1975; Adams and Haddad 1996).

Both neural factors and factors related to increased muscle mass as well as transition in the quality of the contractile proteins could be involved in the observed adaptations to strength training. We can not address the impact of neural adaptations since no measurements of activation level were included in the present study. However, biceps curl is a coordinative easy exercise and by the twitch-interpolate technique it has been reported that untrained subjects have no or only minor activation deficits in simple movements (Shield and Zhou 2004). Thus, differences in neural adaptations should not explain the observed difference in 1RM biceps curl adaptations between *L+A* and *A* training.

In contrast, it has been observed that performing exercises for the larger muscle groups subsequent to the elbow flexors had no additive effect on muscle strength and muscle hypertrophy (Walker et al. 2004; West et al. 2010). The reason for this discrepancy is unclear. Unfortunately the study of Walker et al. (2004) did not investigate the acute effects of the different protocols on hormonal changes, thus it is difficult to tell whether there was a difference between groups in the hormonal milieu while exercising. Differences in exercise order may contribute to explain different findings, since the subjects in the

present study performed leg exercises before arm exercises, while in Walker et al. (2004) and West et al. (2010), the subjects performed leg exercises after the arm exercises. However, in the study by Hansen et al. (2001) legs were also trained after arm exercises and a somewhat additive effect on isometric strength, but not on isokinetic and isotonic strength was reported. Another difference from the study by Walker et al. is that in the present study, one arm was subjected to *L+A* training, while the other arm was subjected to the *A* training. There may be individual responses to strength training and individual differences in hormonal and nutritional level which may lead to a large variation in the effects of strength training. By randomizing one arm to the control and the other as experimental, both conditions have the same hormonal, nutritional, and genetic environment. Thus, if there is difference between the training modes it may be easier to detect with the method used in the present study as compared to the method of Walker et al. (2004), where two different groups of subjects performed different strength training. West et al. (2010) took the latter into account and had roughly the same methodological approach as the present study, but found no differences between *L+A* and *A* in neither hypertrophy nor strength adaptations. One important difference might however be that West et al. (2010) performed leg exercises after the arm exercises. Consequently, we speculate that performing the contractions simultaneously as the hormones are elevated may be critical to get this additive effect on the training adaptations. Although the mechanisms behind this speculation are unclear, it seems logical that an additive training effect may be achieved when blood with elevated levels of testosterone and GH is directed to the working muscles during the heavy strength training. The current findings leads support to the notion that testosterone is one of the major promoters of gains in muscle mass and strength in response to strength training (Vingren et al. 2010).

The present finding of increased peak power after strength training is in agreement with previous findings (i.e. Wilson et al. 1993; Young and Bilby 1993; McBride et al. 2002). It has been suggested that heavy strength training and slow velocities (like in the present study) leads primarily to improvements in

the high force/low velocity portion of the force velocity curve (Fleck and Kraemer 2004). This might explain why there were tendencies towards superior power development at 60% of 1RM in *L+A* compared to *A* (p=0.11) and no difference were observed on the peak power development at 30% of 1RM. The load at 60% of 1RM was closer to the training load and the 1RM load in which *L+A* had superior improvement compared to *A*.

In summary, subjects performed identical strength training exercises for the elbow flexors during which circulating endogenous testosterone and GH were experimentally manipulated via leg exercises. Herein, we have presented evidence suggesting that performing leg exercises and thereby increasing levels of serum testosterone and GH prior to arm exercises, induces superior strength training adaptations compared to arm training alone, without acute elevation of hormones. Specifically, superior gains in 1RM biceps curl as well as favorable muscle adaptations in the elbow flexors were demonstrated when arm exercises were performed with physiological elevated concentrations of serum testosterone and GH compared to the identical arm training without elevated hormone levels.

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FIGURE CAPTIONS

Figure 1 Mean training weight (kg) during 11 week training intervention in the elbow flexor exercises in the arm which performed leg exercises prior to the arm exercises (*L+A*) and the arm which performed no leg exercises prior to the arm exercises (*A*). *Different from pre in both *L+A* and *A* ($p<0.01$).

Figure 2 Plasma testosterone, growth hormone, and cortisol measured before the strength training session (T-0), immediately after the leg exercises in the *L+A* session (T-1), immediately after the arm exercises for both the *L+A* and *A* session (T-2), and 30 min after the arm exercises in both *L+A* and *A* session (T-3). * Significant different from values before the session started ($p<0.05$).

Figure 3 1RM in leg press before (Pre) and after (Post) the 11-week intervention period. *Different from Pre ($p<0.001$).

Figure 4 Relative changes in 1RM for the arm which was trained immediately after leg exercises during elevated concentrations of anabolic hormones (*L+A*) and the arm which was trained without leg exercises before and with no elevation in anabolic hormones (*A*). *Different from Pre ($p<0.001$). #Difference between groups in relative change from pre-test to post-test ($p<0.05$).

Figure 5 Relative changes in peak power at 30% and 60% of 1RM (upper and lower panel, respectively) for the arm which was trained immediately after leg exercises (*L+A*) and the arm which was trained without leg exercises before (*A*). *Different from Pre ($p<0.05$).

Figure 6 Changes in CSA for the arm which was trained immediately after leg exercises (*L+A*; upper panel) and the arm which was trained without leg exercises before (*A*; lower panel) in different regions

of the elbow flexors. Section 6 is most proximal and section 9 is most distal. The space between each section is 35 mm. *Different from Pre (p<0.01).

FIGURE 1

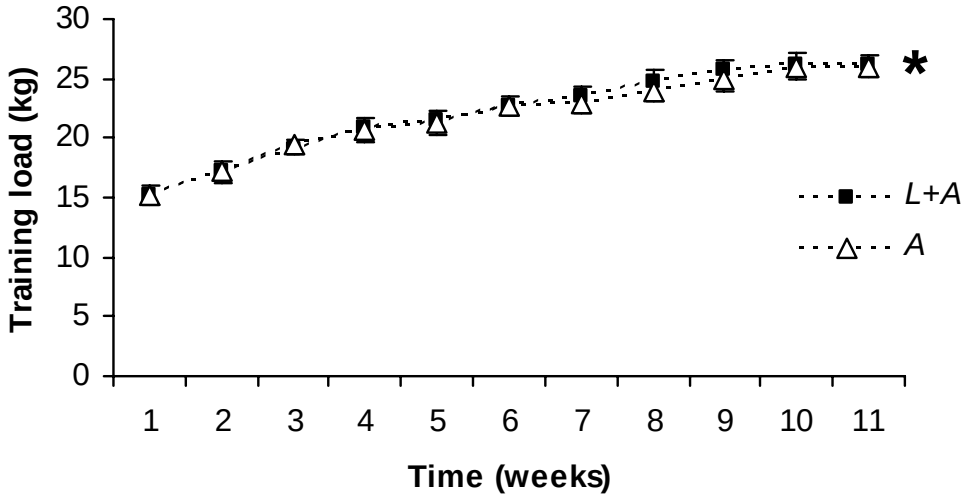

FIGURE 2

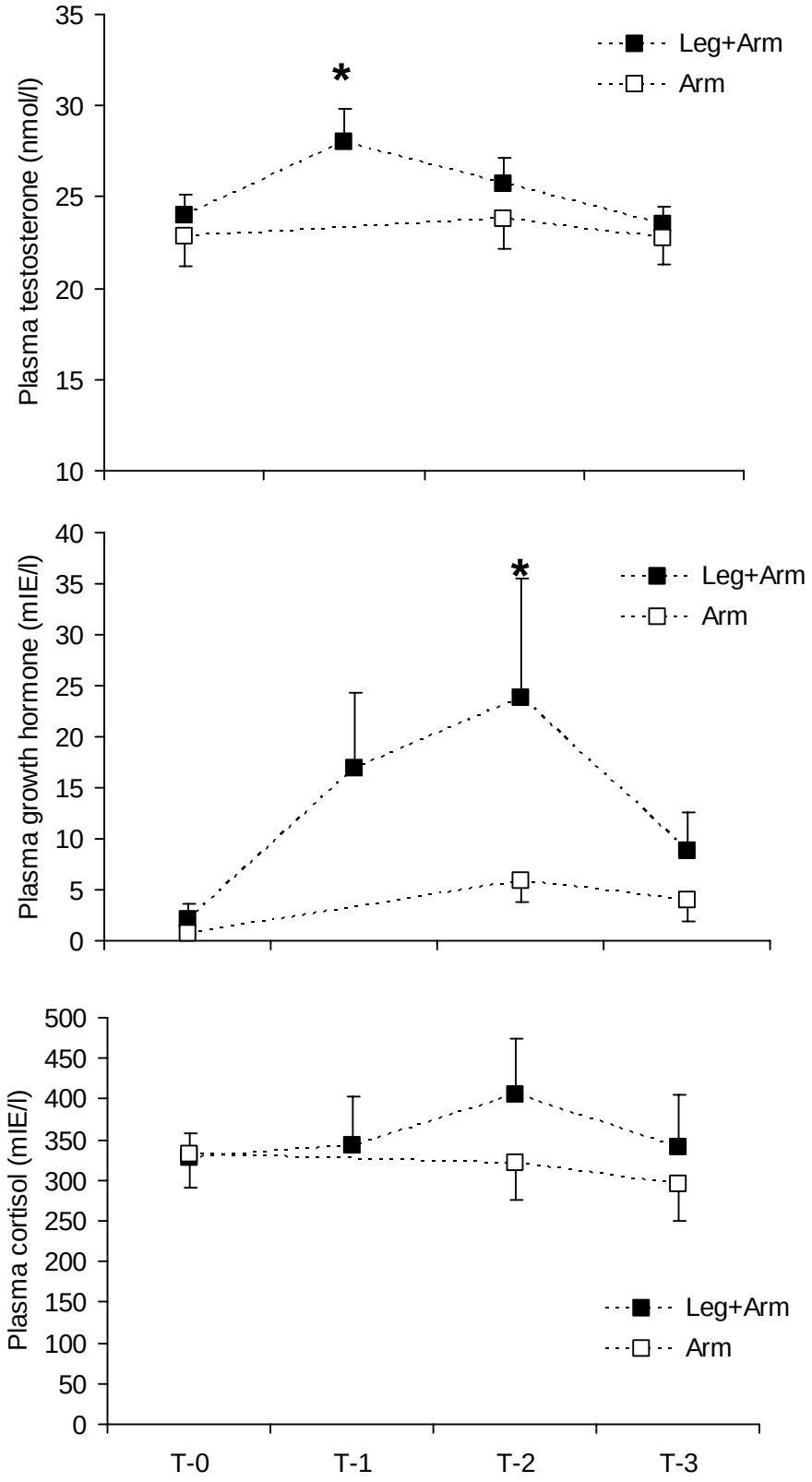
Plasma testosterone (nmol/l)
35
30
25
20
15
10
*
Leg+Arm
Arm
Plasma growth hormone (mIE/l)
40
35
30
25
20
15
10
5
0
*
Leg+Arm
Arm
Plasma cortisol (mIE/l)
500
450
400
350
300
250
200
150
100
50
0
Leg+Arm
Arm
T-0
T-1
T-2
T-3

FIGURE 3

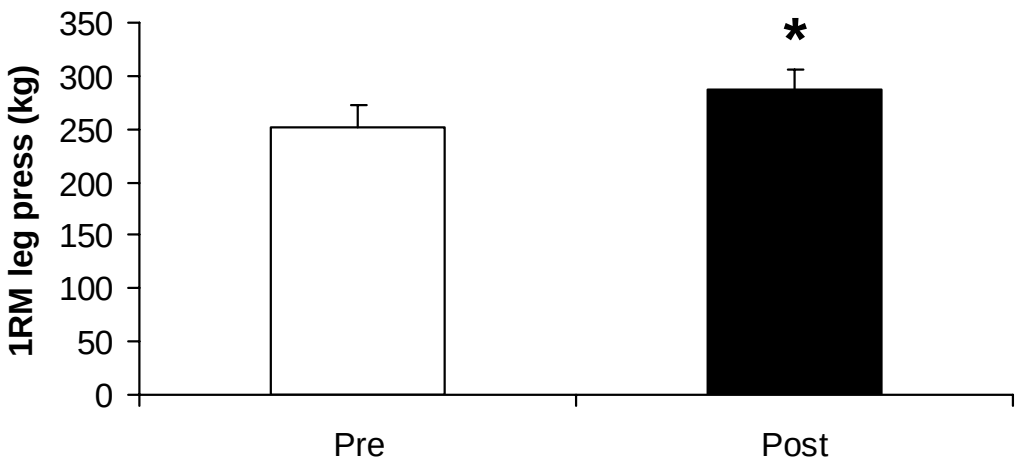

FIGURE 4

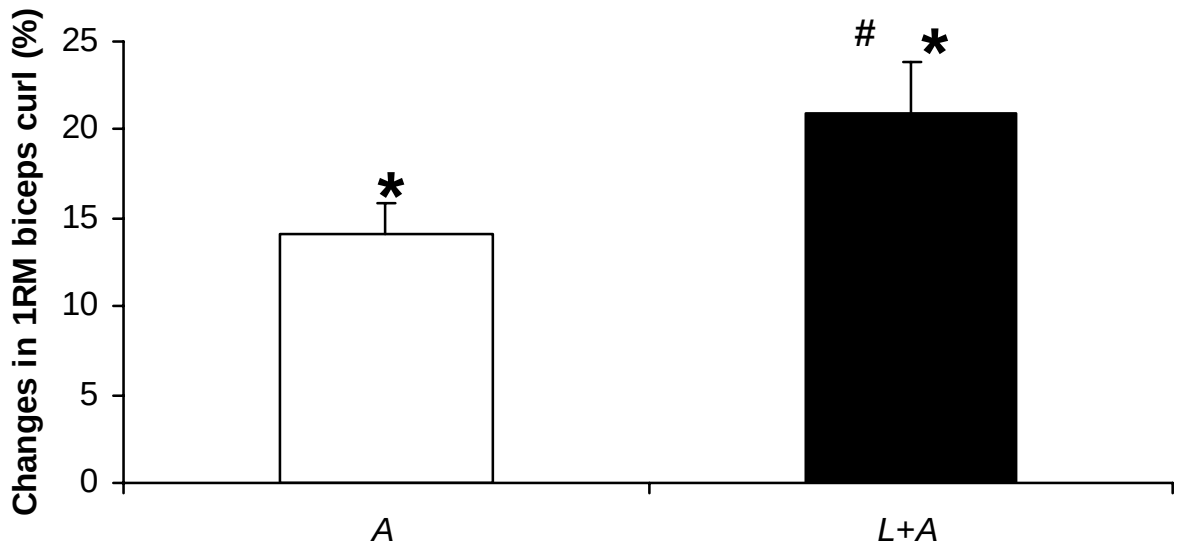

FIGURE 5

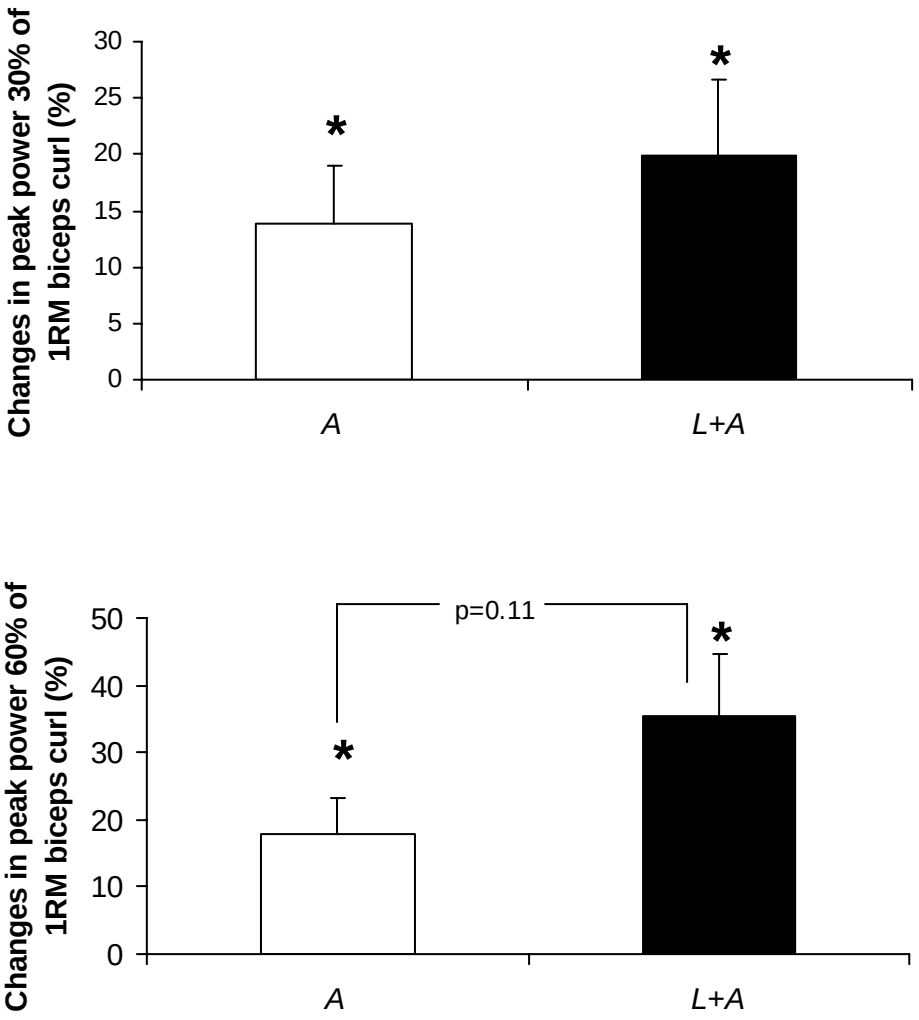
Changes in peak power 30% of 1RM biceps curl (%)
30
25
20
15
10
5
0
*
*
A
L+A
Changes in peak power 60% of 1RM biceps curl (%)
50
40
30
20
10
0
p=0.11
*
*
A
L+A

FIGURE 6

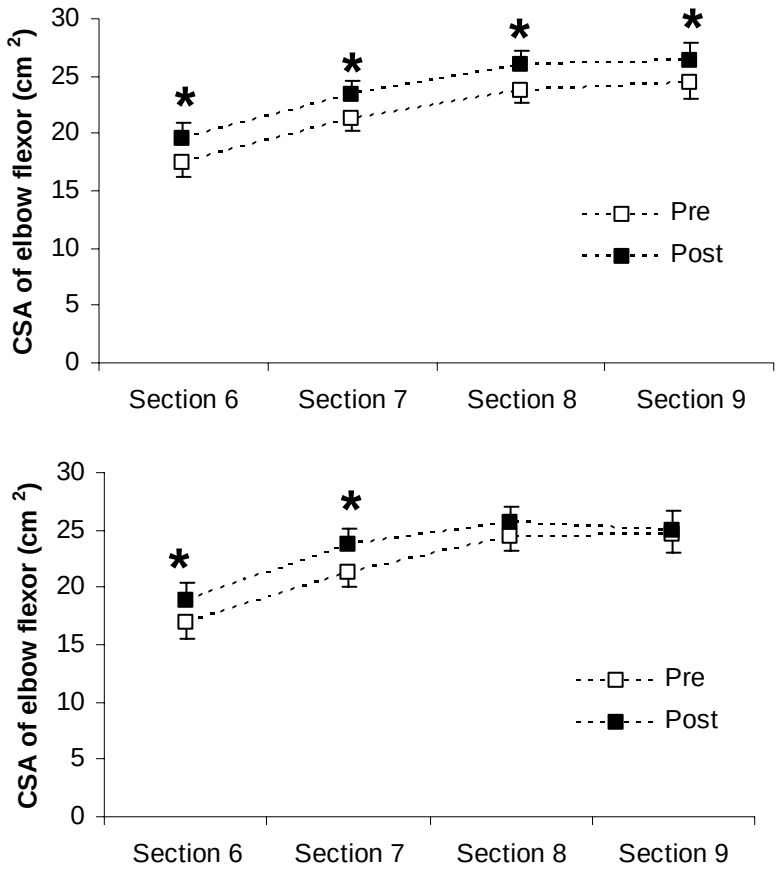
CSA of elbow flexor (cm 2)
30
25
20
15
10
5
0
Section 6
Section 7
Section 8
Section 9
Pre
Post
*
CSA of elbow flexor (cm 2)
30
25
20
15
10
5
0
Section 6
Section 7
Section 8
Section 9
Pre
Post
*

Table 1 Baseline values for the elbow flexors which performed leg exercises immediately before arm exercises (*L+A*) and the elbow flexors which performed arm exercises only (*A*).

L+A	*A*
37.5 ± 2.8	39.2 ± 2.1
96 ± 8	94 ± 6
112 ± 9	115 ± 10
19.4 ± 1.2	19.1 ± 1.3
24.2 ± 1.2	25.4 ± 1.4

Values are mean±SE.