Acute and Chronic Hormonal Responses to Resistance Training Designed to Promote Muscle Hypertrophy

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Catalogue Data

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Abstract/Résumé
Acute and chronic hormonal responses to resistance training were evaluated in 11 college men who completed 12 weeks (33 sessions) of high volume resistance training. No differences in resting concentrations of growth hormone (GH), insulin-like growth factor-I, testosterone, or sex hormone-binding globulin occurred from pre- and posttraining in the trained vs. nontrained control group. However, cortisol (C) decreased 17% for both groups (p < 0.05). There were no differences in exercise-induced responses between Sessions 10 and 20, with all hormone concentrations increasing (p < 0.05) from pre- to mid- and postexercise session. However, after correction for plasma volume decreases, only C and GH showed differences, with C increased from mid- to postsession (48% 10th; 49% 20th), and GH increased from pre- to mid- and postsession for both sessions 10 (0.16 ± 0.42 pre; 4.77 ± 6.24 mid; 6.26 ± 5.19 post; µg · L⁻¹) and 20 (0.33 ± 0.85 pre; 5.42 ± 9.08 mid; 8.24 ± 7.61 post; µg · L⁻¹). Significant correlations (p < 0.05) existed only between absolute mean GH

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increases from presession and the degree of muscle fiber hypertrophy for type I (τ = 0.70 mid, 0.74 post) and type II (τ = 0.71 post) fibers. In conclusion, resistance training had no effect on resting serum hormone concentrations, whereas similar acute exercise responses occurred between the 10th and 20th training sessions.

Les ajustements et adaptations des hormones à l’entraînement à la force sont analysés chez 11 jeunes hommes ayant complété 12 semaines (33 séances) d’entraînement à fort volume. Tant chez le groupe entraîné que chez le groupe témoin, les concentrations de repos de l’hormone de croissance (GH), du facteur de croissance-I du type insulinique, de la testostérone, et de la globuline/protéine liant la testostérone ne diffèrent pas du début à la fin du programme d’entraînement. Dans les deux groupes, le niveau de cortisol (C) a chuté de 17% (p < 0,05). Les ajustements hormonaux ont été similaires entre la 10e et 20e séance; les niveaux de toutes les hormones ont augmenté de façon significative du début de la séance jusqu’à mi-chemin et jusqu’après. Néanmoins, seules les valeurs de GH et de C sont restées différentes après correction pour la baisse du volume plasmatique; les valeurs de C ont augmenté de mi-chemin à la fin de la séance (48% à la 10e; 49% à la 20e) et les valeurs de GH ont augmenté du début de la séance jusqu’à mi-chemin et jusqu’après tant à la 10e séance (pré, 0,16 ± 0,42; mid, 4,77 ± 6,24; post, 6,26 ± 5,19; μ g. L^-1) qu’à la 20e (pré, 0,33 ± 0,85; mid, 5,42 ± 9,08; post, 8,24 ± 7,61 μ g. L^-1). Les seules corrélations significatives (p < 0,05) sont entre l’augmentation moyenne du niveau de GH depuis du début de la séance et le niveau d’hypertrophie des fibres musculaires de type I (mid, τ = 0,70; post, τ = 0,74) et de type II (post, τ = 0,71). En conclusion, l’entraînement à la force n’a pas d’effet sur les niveaux sériques des hormones au repos alors que les effets sont similaires entre la 10e séance et la 20e séance d’entraînement.

Introduction

In an effort to determine a role for anabolic hormones in the muscular hypertrophy associated with resistance training, a number of studies have described the endogenous hormonal responses to acute bouts of resistance exercise (Hakkinen et al., 1993; Kraemer et al., 1990; 1995; Staron et al., 1994). Differences in the magnitude of the acute exercise-induced response of growth hormone (GH) were found to be dependent on the structure of the resistance training regimen (Hakkinen et al., 1993; Kraemer et al., 1990; 1991; Vanheder et al., 1984). For example, Kraemer et al. (1990) found that a high volume training regimen typical of that used by bodybuilders to promote maximal muscle hypertrophy, resulted in a greater GH response compared to a high intensity training regimen typical of that used by competitive weightlifters to promote maximal muscle strength and/or power.

These studies have raised questions about the potential role of acute exercise-induced hormone elevations in the physiological adaptations that accompany prolonged training. However, only two prior investigations were found that evaluated the effects of prolonged training on the acute exercise-induced hormonal responses (Craig et al., 1989; Hickson et al., 1994). Despite no apparent significant differences, Craig et al. (1989) suggested that the exercise-induced hormonal responses of GH, but not testosterone (T), may be altered by resistance training in young men. Hickson et al. (1994) reported attenuation of T and cortisol (C) responses following 7 weeks of training; however, the relative exercise intensity was also reduced because subjects trained with fixed absolute workloads.
In addition to acute exercise hormonal responses, the resting hormone responses to resistance training have also been investigated. The results for specific hormone concentrations are equivocal and appear to be dependent on training status (Hakkinen 1989a). In longitudinal investigations of elite competitive weightlifters, Hakkinen and colleagues (1987, 1988a, 1989b) have generally found that resting hormone levels are not significantly changed, although transient changes in T occurred during periods of intense training (1987, 1988b), and over longer time periods in the highest caliber weightlifters (1988a).

Resting hormone concentrations have also been measured during controlled resistance training studies. Of those studies using non-elite or previously non-resistance-trained subjects, no changes occurred for most resting hormones (Hakkinen et al., 1985; Kraemer et al., 1995), although increases in T (Staron et al., 1994) and decreases in C have been reported (Hakkinen et al., 1985; Kraemer et al., 1995; Staron et al., 1994). However, of the many investigations of resting hormone responses in men, only Staron et al. (1994) included a nontraining control group, and therefore it is unclear whether factors other than resistance training also affected the resting hormone concentrations.

Therefore, despite a well characterized acute hormonal response to resistance exercise and considerable speculation about its role in adaptation to training, there is little information as to the trainability of this response. The purpose of the present study was to investigate the effects of high volume resistance training on the acute exercise-induced hormonal response as well as on resting hormone concentrations.

Methods

Eleven college men (ages 18–25) with recreational resistance training experience completed 12 weeks of training at a frequency of 3 sessions a week. This subject population was chosen because they would have achieved significant initial neuromuscular adaptations, with further increases in strength attributed primarily to muscle hypertrophy (Hakkinen 1989a). Also, these subjects would be better conditioned to tolerate the rigorous resistance exercise regimen used in this study.

A high volume training regimen previously shown to cause significant acute exercise-induced hormone elevations was used (Kraemer et al., 1990). This training protocol was considered to provide the best prospects for evaluating potential adaptations in the acute exercise-induced hormonal responses and the importance of hormonal factors on other training responses. The training protocol and criteria for subject selection are described in more detail elsewhere (McCall et al., 1996). Briefly, training sessions were held on Monday, Wednesday, and Friday mornings. Each session consisted of three 10 repetition maximum (10-RM) sets for 8 exercises, with a 1-min rest between sets and exercises. Four exercises emphasized the biceps brachii while the remaining exercises incorporated all major muscle groups of the upper and lower body.

Free weights or weight machines were used for all exercises. Before and after training, subjects underwent testing sessions to evaluate the efficacy of the training for increasing strength, total muscle cross-sectional area (CSA), and the muscle fiber CSA of the biceps brachii. These procedures have previously been described in more detail (McCall et al., 1996). Informed consent was obtained
from each subject, and all procedures were approved by the university's human subjects committee.

Blood samples were collected (15 mls/sample) for determination of serum concentrations of growth hormone (GH), insulin-like growth factor-I (IGF-I), testosterone (T), cortisol (C), and sex hormone-binding globulin (SHBG). Resting blood samples were collected before and after the 12-week training period. To control for factors other than resistance training which could affect resting hormone concentrations, 8 college men (ages 19–29) not participating in resistance training served as controls. Following an overnight fast, subjects reported to the lab between 7 and 9 a.m., at which time an indwelling flexible catheter was placed in an antecubital vein and kept patent with a heparin saline solution. After the catheter was inserted, subjects rested 20 min prior to blood collection to minimize hormone fluctuations related to anticipatory responses (Kraemer et al., 1991). Subjects remained supine during all resting blood draw procedures.

Blood samples were also collected to evaluate hormonal responses during specific resistance exercise training sessions. The first exercise blood samples were obtained during the 10th session (4th week) because, despite their prior lifting experience, it took the subjects several sessions to tolerate the protocol's 1-min rest interval and establish the 10-RM training loads. Blood collection was repeated during the 20th session (8th week) to assess possible changes due to training. Blood was collected presession (pre), after completion of 4 exercises (mid), and 10 min postsession (post).

Exercise blood samples were collected following the same procedures as resting collections, except that the catheter was placed in a superficial forearm vein to allow the arm to bend without interference or discomfort from the catheter when performing the exercises. Subjects were supine for each collection period, with mid and post periods lasting 5 to 7 min. All blood collection periods occurred within (±) 1 hour for the different test sessions.

All blood samples were placed on ice and allowed to clot. The samples were centrifuged 10 min at 3,300 rpm and the serum was removed to microcentrifuge tubes and stored at -70 °C awaiting hormonal analyses. Double antibody 125I-radiomunoassays were used to determine serum concentrations of GH (Diagnostic Products Corp., Los Angeles), IGF-I (Incastor Corp., Stillwater, MN), T, and C (Diagnostic Systems Labs, Webster, TX). Immunoradiometric 125I-assays were used to determine serum concentrations of SHBG (Diagnostic Systems Labs). For GH, T, and SHBG, all samples were determined within a single assay, thereby eliminating interassay variance. For IGF-I and C, single assay determinations were not feasible, and interassay variances were 8.10% and 5.47%, respectively. Intra-assay variances were 1.36, 2.26, 3.56, 4.64, and 6.44% for GH, IGF-I, T, C, and SHBG, respectively.

To evaluate the impact of decreases in plasma volume (PV) during acute exercise on the serum hormone concentrations (Kraemer et al., 1993), changes in PV were estimated from Hb and Hct using the equation of Dill and Costill (1974). Hormone concentrations at mid and post were subsequently corrected for changes in PV. These PV corrections assumed that primarily plasma was lost from the circulation (Kraemer et al., 1993). After the training study was completed, 4 additional control subjects were recruited to evaluate the impact of postural changes on the PV changes. In these control experiments, subjects underwent body posi-
tion changes and blood collection periods comparable to those incurred during the exercise conditions. The results indicated there were significantly smaller decreases in PV during the postural control experiments (∼2.5% pre to mid; ∼3.9% pre to post) as compared to resistance exercise (∼6.5% pre to mid; ∼17.1% pre to post).

Two-factor ANOVA (time: within-subjects; group: between-subjects) were used to assess changes in resting hormone concentrations. For acute exercise responses of the trained group, two-factor (within subjects) repeated measures ANOVA were used to assess differences within and between the 10th and 20th training sessions. When significant main effects were observed, one-factor repeated measures ANOVA and Scheffé post hoc tests were used to identify differences within the acute session. Pearson product-moment correlations were calculated to determine the relationship between an individual’s relative degree of muscle hypertrophy resulting from training with either resting hormone concentrations or absolute hormonal increases (i.e., difference from pre; μg · L⁻¹) during acute resistance exercise sessions. Significance was established at $p < 0.05$.

**Results**

**Muscle Strength and Hypertrophy**

The average number of training sessions completed was $33.25 \pm 0.75$, with all subjects completing ≥32 sessions. The results for increased muscle strength and hypertrophy are presented in more detail elsewhere (McCall et al., 1996). Briefly, training resulted in a 25% increase in 1-RM forearm flexor strength ($36.04 \pm 7.89$ kg to $45.13 \pm 6.60$ kg). Muscle CSA measurements from MRI scans indicated hypertrophy of biceps brachii muscle CSA from $11.8 \pm 2.7$ cm² pretraining to $13.3 \pm 2.6$ cm² posttraining. Analysis of tissue obtained from muscle biopsies of the biceps brachii showed hypertrophy for the average fiber areas of both type I ($4,196 \pm 850$ vs $4,617 \pm 1,116 \mu m^2$) and II ($6,378 \pm 1,552$ vs $7,474 \pm 2,017 \mu m^2$) fibers as a result of training.

**Resting Hormone Concentrations**

Table 1 shows the values for resting hormone concentrations pre- and posttraining. No differences in resting hormone concentrations occurred between groups; however, C decreased 16.7% from pretraining for both groups. For the trained group, resting hormone concentrations prior to the 10th and 20th sessions were also unchanged, except for C which was decreased from pretraining for the 20th session (Table 2). There were no changes in resting T:C ratios throughout the study.

**Acute Exercise-Induced Hormone Responses**

Blood samples were not collected from one subject during the 10th exercise session due to problems with the catheter, therefore $n = 10$ for these results. Table 2 shows the results for acute exercise-induced hormonal responses. No differences in hormonal responses occurred between the 10th and 20th sessions for any of the hormones evaluated. Although the pattern of response varied between hormones, acute exercise increased all hormone concentrations. However, after correcting for the exercise-induced decreases in PV (∼6.5% to ∼17.1%), no changes occurred.
Table 1  Resting Serum Hormone Concentrations ($M \pm SD$) Before and After 12 Weeks of Resistance Training

<table>
<thead>
<tr>
<th></th>
<th>Trained ($n = 11$)</th>
<th>Control ($n = 8$)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
</tr>
<tr>
<td>hGH ($\mu g \cdot L^{-1}$)</td>
<td>1.20 ± 3.69</td>
<td>0.12 ± 0.26</td>
</tr>
<tr>
<td>IGF-I (nmol · L⁻¹)</td>
<td>26.1 ± 6.0</td>
<td>26.4 ± 6.1</td>
</tr>
<tr>
<td>Testosterone (nmol · L⁻¹)</td>
<td>18.3 ± 6.4</td>
<td>17.9 ± 5.2</td>
</tr>
<tr>
<td>Cortisol (nmol · L⁻¹)</td>
<td>622 ± 188</td>
<td>485 ± 163*</td>
</tr>
<tr>
<td>SHBG (nmol · L⁻¹)</td>
<td>26.7 ± 10.4</td>
<td>26.8 ± 9.7</td>
</tr>
</tbody>
</table>

*ANOVA main effect significantly different from pretraining, $p < 0.05$.

for IGF-I, T, and SHBG, while GH remained increased at mid and post, and C remained elevated post as compared to mid.

HORMONE CONCENTRATIONS AND MUSCLE HYPERTROPHY

For resting hormone concentrations, GH, IGF-I, T, and SHBG were not significantly correlated with either total biceps brachii hypertrophy or muscle fiber hypertrophy. For the acute exercise-induced GH increases (PV corrected), because no differences were found between the 10th and 20th sessions, and there were significant correlations for the absolute increases (i.e., difference from pre; $\mu g \cdot L^{-1}$) of GH at mid ($r = 0.81$) and post ($r = 0.86$), the mean of both sessions was used as the measure of GH response. There were no significant correlations between mean absolute acute exercise-induced GH elevations and hypertrophy of the biceps brachii muscle CSA. However, significant correlations were found between mean absolute acute exercise-induced GH increases and the relative degree of type I ($r = 0.70$ mid; 0.74 post) and II ($r = 0.62$ mid, 0.71 post) fiber hypertrophy (Figures 1 and 2). No correlations were found between acute exercise-induced changes of other hormones and the indices of muscle hypertrophy.

Discussion

In the present study, the acute hormonal responses to resistance training were similar between the 10th and 20th training sessions. Although Craig et al. (1989) reported that acute exercise elevation of GH increased after 12 weeks of training, the changes were not statistically significant. In contrast, using a high intensity/low volume
Table 2  Serum Hormone Concentrations \((n = 10; M \pm SD)\) Pre, Mid, and Postexercise

<table>
<thead>
<tr>
<th></th>
<th>Pre</th>
<th>Mid (PV corrected)</th>
<th>Post (PV corrected)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>10th Session</strong></td>
<td></td>
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</tr>
<tr>
<td>GH (µg · L(^{-1}))</td>
<td>0.16 ± 0.42</td>
<td>5.51 ± 7.13*</td>
<td>6.94 ± 5.85*</td>
</tr>
<tr>
<td>IGF-1 (nmol · L(^{-1}))</td>
<td>25.2 ± 6.5</td>
<td>28.8 ± 6.6*</td>
<td>27.6 ± 6.2*</td>
</tr>
<tr>
<td>Testosterone (nmol · L(^{-1}))</td>
<td>15.4 ± 5.5</td>
<td>18.6 ± 4.1</td>
<td>16.5 ± 5.2</td>
</tr>
<tr>
<td>Cortisol (nmol · L(^{-1}))</td>
<td>483 ± 131</td>
<td>482 ± 114</td>
<td>668 ± 239*</td>
</tr>
<tr>
<td>SHBG (nmol · L(^{-1}))</td>
<td>30.2 ± 6.4</td>
<td>31.9 ± 9.5</td>
<td>31.9 ± 9.2</td>
</tr>
<tr>
<td><strong>20th Session</strong></td>
<td></td>
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</tr>
<tr>
<td>GH (µg · L(^{-1}))</td>
<td>0.33 ± 0.85</td>
<td>6.27 ± 10.51*</td>
<td>8.71 ± 8.00*</td>
</tr>
<tr>
<td>IGF-1 (nmol · L(^{-1}))</td>
<td>24.7 ± 6.1</td>
<td>28.2 ± 6.9*</td>
<td>26.1 ± 7.4*</td>
</tr>
<tr>
<td>Testosterone (nmol · L(^{-1}))</td>
<td>17.1 ± 5.2</td>
<td>20.5 ± 7.5*</td>
<td>17.3 ± 5.1*</td>
</tr>
<tr>
<td>Cortisol (nmol · L(^{-1}))</td>
<td>438 ± 129(^{+})</td>
<td>473 ± 158</td>
<td>649 ± 290*</td>
</tr>
<tr>
<td>SHBG (nmol · L(^{-1}))</td>
<td>26.9 ± 16.1</td>
<td>33.9 ± 16.4*</td>
<td>31.0 ± 17.0</td>
</tr>
</tbody>
</table>

Significantly different, \(p < 0.05\): *from pre; \(^{+}\)from mid; \(^{+}\)from pretrained value reported in Table 1.

resistance exercise protocol, Hickson et al. (1994) found that repeated bouts of training attenuated the acute exercise-induced elevations of T and C.

The disparity between Hickson et al.’s results and those of the present study is likely due to the use of fixed absolute loads by Hickson et al., whereas we increased resistance to maintain a 10-RM load. Differences between the exercise regimen used by Hickson et al. and that of the present study (lower intensity/high volume) may also explain the results, given that prior studies have shown the acute-exercise hormone response to differ between these regimens (Kraemer et al., 1990; 1991).
Figure 1. Relationship between changes in acute exercise-induced GH concentration and relative type I muscle fiber hypertrophy.

Figure 2. Relationship between changes in acute exercise-induced GH concentration and relative type II muscle fiber hypertrophy.
Finally, adaptations in the acute exercise-induced hormone response may have occurred from recreational resistance training prior to beginning the present study and/or during the training sessions before the first exercise blood collection. Unfortunately, obtaining a true pretraining acute exercise response was impossible because, despite their prior training, subjects could not strictly adhere to the protocol’s 1-min rest interval at the outset. Yet despite this limitation, the present study provides some insight into the role played by hormonal factors when hypertrophy is stimulated by this type of training regimen.

To our knowledge, no prior study has correlated any acute exercise-induced changes in circulating hormone concentrations with morphological adaptations within the muscle. However, caution is warranted in the interpretation of correlational data from relatively small subject numbers. While the correlations between acute exercise GH elevations and fiber hypertrophy are not evidence of a causative role for GH in muscle hypertrophy, these relationships could be indicative of a role for repeated acute exercise-induced GH elevations on cellular adaptations in the trained muscle.

Although many cellular mechanisms of GH action are poorly understood with regard to regulation of muscle hypertrophy, several lines of evidence indicate that both circulating GH and mechanical loading promote anabolism in skeletal muscle and are interactive when combined. A recent study demonstrated a net uptake of GH in working muscle during dynamic exercise in humans (Brahm et al., 1997), whereas earlier human studies have shown that acute GH administration increases net skeletal muscle protein anabolism by increasing protein synthesis rates (Fryburg and Barrett, 1993). Moreover, studies on GH-deficient mice report dose-dependent increases in muscle protein synthesis rates during chronic GH administration (Pell and Bates, 1992).

Recent human studies have also shown a net protein uptake by skeletal muscle that persists for up to 48 hours after acute resistance exercise (Phillips et al., 1997). Finally, McCall et al. (1998) demonstrated that compensatory hypertrophy of the rat soleus was greatest when accompanied by exogenous GH and IGF-I treatment, and others have reported an interactive effect for GH and/or IGF-I and resistance exercise as countermeasures for muscle atrophy in hindlimb-suspended hypophysectomized (Grossman et al., 1997) and normal rats (Linderman et al., 1994).

Based on the previous and current data, we hypothesize that repeated acute exercise-induced elevations of circulating GH in combination with muscle overload during training resulted in a net anabolism of myofibrillar proteins. However, the absence of an increase in circulating IGF-I in the present study does not exclude the possibility that the proposed GH effects were also mediated locally by an autocrine and/or paracrine production of IGF-I (Adams and Haddad, 1996).

In the present study, only GH and C increased significantly at mid- or postsession after correction for estimated PV decreases. In line with the current study, Kraemer et al. (1992) reported comparable PV decreases which accounted for the acute exercise-induced elevations of T and IGF-I concentrations, while GH increases remained significant after correction for PV decreases. While some have argued against correction for PV changes because the uncorrected concentration is what the tissue “senses” (Cappon et al., 1994), accounting for PV losses is important when considering the responses of blood parameters to resistance exercise (Kraemer et al., 1993). Therefore, the significant elevation of GH and C after cor-
rection for PV decreases may be indicative of the release of these hormones into (and/or decreased clearance from) the circulation, whereas other hormones appeared to be increased by hemoconcentration.

Regardless of the mechanism for the increased circulating hormone concentrations, significant local effects in the tissues exposed to elevated hormones might be anticipated. In addition, multiple other factors (e.g., cell receptor density, circulating binding proteins, regional blood flow) influence the availability and capacity of hormones to affect target tissues.

Except for a decrease in C in both trained and untrained groups, the resting concentrations of T, SHBG, GH, and IGF-I were unchanged as a result of training. While several other longitudinal resistance training studies of men have reported no changes in resting T (Craig et al., 1989; Hakkinen et al., 1985; Hickson et al., 1994; Kraemer et al., 1995), increased T was reported by Staron et al. (1994) in previously untrained men. In agreement with the current study, other resistance training studies also found no changes in resting GH concentrations (Craig et al., 1989; Hakkinen et al., 1985; Staron et al., 1994).

In agreement with prior studies (Hakkinen et al., 1985; Kraemer et al., 1995; Staron et al., 1994), the present study found decreased resting C in the trained group; however, the decline was similar in the control group and therefore could not be attributed to resistance training. These results underscore the importance of including a control group when evaluating resting hormone concentrations. In contrast to our results, another resistance training study reported decreased C in trained men and no change in controls over a 9-week period (Staron et al., 1994).

While we cannot explain the decreased C in our subjects, we speculate that (a) anxiety from the catheter insertion diminished during the posttraining blood collection in spite of our efforts to minimize anticipatory hormonal responses to blood draw procedures, or (b) comparable seasonal decreases occurred in both groups. Previous resistance training studies on women reported similar changes over time in both trained and control groups for androstenedione (Westerling et al., 1987), testosterone, and estradiol (Stoessel et al., 1991) and speculated that seasonal changes might be responsible.

In conclusion, resting hormone concentrations and the patterns of acute exercise hormonal elevations during the 4th and 8th weeks were not changed by 12 weeks of resistance training in men who had prior recreational resistance training experience. Decreased PV accounted for the acute exercise elevations of T, IGF-I, and SHBG. However, GH and C remained significantly elevated when corrected for PV losses. Only the acute exercise-induced GH elevations were correlated with the magnitude of muscle fiber hypertrophy following training. These data indicate that the potential for GH to influence the attainment of muscle hypertrophy in recreationally resistance trained men may be attributable to repeated exercise-induced elevations.

References


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