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Effects of endurance training only versus same-session combined endurance and strength training on physical performance and serum hormone concentrations in recreational endurance runners

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ABSTRACT

This study investigated the effects of endurance training only (E, n=14) and same-session combined training, when strength training is repeatedly preceded by endurance loading (E+S, n=13) on endurance (1000 m running time during incremental field test) and strength performance (1RM in dynamic leg press), basal serum hormone concentrations, and endurance loading-induced force and hormone responses in recreationally endurance trained men. E was identical in the two groups and consisted of steady-state and interval running, 4-6 x wk^{-1} for 24 weeks. E+S performed additional maximal and explosive strength training (2 x $week^{-1}$) immediately following an incremental running session (35-45min, 65-85% HR_{max}). E and E+S decreased running time at week 12 (-8±5%, p=0.001 and -7±3%, p<0.001) and 24 (-13±5%, p<0.001 and -9±5%, p=0.001). Strength performance decreased in E at week 24 (-5±5%, p=0.014) but was maintained in E+S (btw-groups at week 12 and 24, p=0.014 and 0.011). Basal serum testosterone and cortisol concentrations remained unaltered in E and E+S but testosterone/SHBG-ratio decreased in E+S at week 12 (-19±26%, p=0.006). At week 0 and 24, endurance loading-induced acute force (-5 to -9 %, p=0.032 - 0.001) and testosterone and cortisol responses (18-47%, p=0.013 - p<0.001) were similar between E and E+S. This study showed no endurance performance benefits when strength training was performed repeatedly after endurance training compared to endurance training only. This was supported by similar acute responses in force and hormonal measures immediately post endurance loading after the training with sustained 1RM strength in E+S.

Key words: concurrent training, acute interference, testosterone, cortisol, endurance running, endocrine adaptations,

INTRODUCTION

High frequency and volume combined endurance and strength training has previously been shown to impair maximal strength development in untrained subjects (Hickson 1980). While detrimental effects on endurance performance are typically not observed in these subjects, heavy and explosive strength training added to endurance training of moderately and highly trained endurance runners may lead to beneficial adaptations in running economy, running speed at VO_{2max} and time to exhaustion, when adequate recovery between each training mode is provided (Beattie et al. 2014).

The superior effects of combined training on maximal and sub-maximal endurance running performance in endurance athletes are often attributed to improved neuromuscular efficiency, increased force generating capacity, and delayed recruitment of type II fibres as well as a conversion of fast-twitch type IIx fibres into more fatigue resistant type IIa fibers (Ronnestad and Mujika 2014). Although neuromuscular adaptations induced by combined training typically occur in conjunction with changes in endocrine function, only little is known regarding the contribution of the endocrine system when combining strength and endurance training (Kraemer et al. 1995; Taipale et al. 2010).

Previous studies have indicated that both endurance and strength exercises can transiently increase hormone concentrations (Kraemer et al. 1990; Häkkinen and Pakarinen 1993; Hackney et al. 2012), such as testosterone (T), growth hormone (GH) and cortisol (C). Among other physiological functions, it is likely that these acute alterations in anabolic and catabolic hormone concentrations directly affect the rates of protein synthesis, red blood cell production and energy restoration (Shahani et al. 2009; Vingren et al. 2010), facilitating biological adaptations to exercise training. As part of the adaptation process during prolonged endurance and/or strength training, exercise induced acute hormone responses (Keizer et al.

1987; Kraemer et al. 1995; Häkkinen et al. 2000) and basal hormone concentrations (Kraemer et al. 1995; Häkkinen et al. 2000; Ahtiainen et al. 2003; Hackney et al. 2003; Taipale et al. 2010) may be observed.

Although the hormonal responses to short term endurance and strength exercises are rather similar (Stokes et al. 2013), their physiological functions may differ due to the catabolic vs. anabolic nature of both types of exercises, possibly contributing to the beneficial effects of strength training on endurance performance. In previous studies, endurance and strength training have typically been performed on separate days allowing for prolonged recovery between subsequent training sessions (Ronnestad and Mujika 2014; Beattie et al. 2014). However, several studies have shown that heavy strength training sessions caused acute detrimental effects on subsequent running performance for 6-24 hours (Doma and Deakin 2013; Palmer & Sleivert 2003) owing to the possibility that strength training may in fact compromise endurance development for running performance. On the other hand, previous studies have also shown acute reductions in strength performance following endurance running (de Souza et al. 2007), possibly reducing the beneficial long-term effects of strength training on endurance performance. As decrements in endurance running performance may still be observed 24h following a strenuous strength loading (Doma and Deakin 2013) while recovery following endurance loadings of moderate duration and intensity is much shorter (Bentley et al. 2000, Millet & Lepers 2004), performing endurance training immediately prior to strength training may minimize acute interference and optimize strength training-induced endurance development.

The purpose of the present study was to investigate 1) the adaptations in endurance and strength performance as well as basal serum hormone concentrations and 2) the adaptations in acute force and hormone responses to endurance loading, following prolonged endurance training only versus same-session combined training when strength training is repeatedly

103 preceded by endurance loading. A secondary purpose of this study was to investigate whether
104 acute endurance loading-induced changes in force production and serum hormone
105 concentrations are associated with endurance and strength performance development.

106 MATERIALS AND METHODS

107 *Subjects*

108 Twenty-seven recreationally endurance-trained males participated in this study. The subjects
109 had performed endurance running for a minimum of 1 year with 2-6 sessions (at both
110 moderate and high intensity) per week prior to the start of the study. Before giving informed
111 consent, all subjects received information about possible risks of all study procedures. A
112 completed health questionnaire and resting ECG were reviewed by a cardiologist prior to the
113 first exercise testing. All subjects were free of acute and chronic illness, disease and injury
114 and did not report use of medications that would contraindicate the performance of intense
115 physical activity or would interfere with endocrine function. Demographic characteristics of
116 all subjects were as follows (mean±SD): age 33±7years, body height 179±6 cm and body
117 weight 78±7 kg. The study was conducted according to the Declaration of Helsinki and
118 ethical approval was granted by the Ethics Committee at the local University.

119 *Study design*

120 Following health-screening, subjects were assigned to an endurance only (E, n=14) or same-
121 session combined endurance and strength training (E+S, n=13) group. All subjects performed
122 identical endurance training for 24 weeks but additional strength training was added to the
123 E+S training program and was performed always immediately after a standardized endurance
124 running protocol.

Prior to the commencement of E and E+S training, baseline testing of endurance (incremental field test) and strength performance (dynamic leg press and counter movement jump [CMJ]) was conducted and concentrations of serum hormones (T, GH, and C) and sex hormone binding globulin (SHBG) were assessed. Acute force and hormone responses were determined by measuring maximal force (bilateral isometric leg press) and serum hormone concentrations before and after an incremental treadmill protocol (Fig 1). To ensure sufficient recovery, all tests were separated by at least 48 h of rest. The measurements of loading responses were repeated after 24 weeks, while the baseline measurements of endurance and strength performances as well as serum hormone concentrations were also conducted after 12 weeks. All post-training measurements (at week 12 or 24, respectively) were performed at the same time of day within ±1h of the testing time at week 0. To control the experimental conditions, subjects received both verbal and written instructions about the measurement preparation in order to minimize physical and mental stress and to allow for at least 7-8 h of sleep on the day before each testing. Basal concentrations of serum hormones were assessed in the morning (between 7:00 a.m. and 9 a.m.) after 12 h of fasting.

+++Figure 1 somewhere near here+++

Testing procedures

Strength and power performance

One repetition maximum (1RM) using the dynamic horizontal leg press device (David 210, David Health Solutions, Helsinki, Finland) was determined at week 0, 12 and 24, respectively. Following a warm up (1 set of 5 repetitions at 70% of estimated 1RM, 1 set of 2 repetitions at 80-85% of estimated 1RM, 1 set of 1 repetition at 90-95% of estimated 1RM), a maximum of 5 trials were allowed to obtain a true 1RM. The device was set up so that the knee angle in the initial flexed position was approximately 60 degrees (as measured by a

manual goniometer) and a successful trial was accepted when the knees were fully extended (~180 degrees). The greatest load that the subject could lift to full knee extension was accepted as 1RM.

Maximal power was determined by a counter movement jump (CMJ) on a force plate (Department of Biology of Physical Activity, Jyväskylä, Finland) at week 0, 12 and 24, respectively. Subjects were asked to keep their hands in contact with their hips throughout the movement and were instructed to jump as high as possible on command. Force data was collected and analyzed by Signal software (Signal 4.04, Cambridge Electronic Design Ltd., Cambridge, UK). Jumping height was calculated from the take-off velocity using the formula $h=v^2/2g$, in which h refers to jumping height and v refers to take-off velocity (Komi and Bosco 1978). The best trial in terms of jumping height measured in cm was used for statistical analysis.

To assess acute endurance loading-induced force responses at week 0 and 24, maximal isometric bilateral leg press force (MVC_{max}) was assessed by a horizontal leg press dynamometer (Department of Biology of Physical Activity, University of Jyväskylä, Finland). Subjects were seated with a hip and knee angle of 110 and 107 degrees, respectively and were instructed to produce maximal force as rapidly as possible on verbal command and to maintain the force plateaued for 3-4 seconds. Before the treadmill protocol three trials separated by 1 minute of rest were conducted, while after the exercise only two trials separated by 15 seconds were performed. The trial with the highest maximal force measured before or after the loading, respectively was used for statistical analysis. The force signal was low-pass filtered (20Hz) and analyzed (Signal software, version 4.04, Cambridge Electronic Design Ltd., Cambridge, UK).

Endurance performance measures and endurance loading

Running performance was determined by an incremental field test of 6x1000m (1 minute inter-set rest) performed on a 200 m indoor running track at week 0, 12 and 24, respectively. The initial speed for all subjects was 6 $min \cdot km^{-1}$ and the speed was increased by 30 seconds every 1000 m. The test was performed in small groups and velocity was controlled every 100 m. The final 1000 m were performed at individual maximal running speed and the time of this trial was used for statistical analysis.

At week 0 and 24, a graded protocol on a motorized treadmill was used to measure endurance loading-induced acute force and hormone responses. The initial velocity for all subjects was 9 $Km \cdot h^{-1}$ and increased by 1 $Km \cdot h^{-1}$ every 3 minutes, while the incline was kept constant at 0.5º. The treadmill was stopped every 3 minutes for 20 seconds in order to collect capillary blood samples from the fingertip for the determination of blood lactate concentrations. Twenty µl of blood were collected by small capillaries, inserted into reaction capsules containing a hemolyzing and anticoagulant agent and lactate concentrations were analyzed using a Biosen analyzer (C_line Clinic, EKF, Magdeburg, Germany). Time to exhaustion was used for statistical analysis and was defined as the maximal testing time until voluntary exhaustion. Furthermore, the velocity at a blood lactate concentration of 4 $mmol \cdot l^{-1}$ (V_4) was used as an indicator of running economy (Heck et al. 1985). Following voluntary exhaustion, a 5 minute cool down at the initial speed (9 $Km \cdot h^{-1}$) was performed. In order to determine acute force and hormone responses, MVC_{max} and serum hormone concentrations were assessed before the start of the treadmill protocol and after the cool down (Fig 1).

Venous blood sampling

Venous blood samples (10 ml) for the determination of serum hormone concentrations (basal concentrations at week 0, 12 and 24; endurance loading-induced acute responses at week 0 and 24) were collected by a qualified lab technician. Whole blood was centrifuged at 3,500

rpm (Megafuge 1.0 R, Heraeus, Germany) for 10 minutes after which serum was removed and stored at -80°C until analysis (approximately 4-8 weeks). Analysis of total serum testosterone, GH (22-kDa), sex hormone binding globuline (SHBG) and cortisol were performed using chemical luminescence techniques (Immunlite 1000, Simens, New York, USA) and hormone specific immunoassay kits (Siemens, New York, USA). The sensitivity for serum hormones were: T 0.5 $nmol \cdot l^{-1}$, GH 0.03 $mlU \cdot l^{-1}$, SHBG 0.2 $nmol \cdot l^{-1}$ and C 5.5 $nmol \cdot l^{-1}$. The intra-assay coefficients of variation for T, GH, SHBG, and C were 8.7±2.7%, 7.1±4.6 %, 6.4±1.7%, 6.0±0.5% and 7.1±1.1%, respectively. The inter-assay coefficients of variation for T, GH, SHBG and C were 10.6±3.2%, 11.1±4.3%, 5.8±0.3%, 7.6±1.4 and 7.9±1.2%, respectively. Basal T/C- and T/SHBG-ratios were also calculated. Plasma volume changes were estimated from changes in hematocrit and hemoglobin (Dill and Costill 1974) but were not used to correct obtained serum hormone concentrations (Kraemer and Ratamess 2005).

Endurance training

The subjects were required to maintain habitual physical activity throughout the study period. The prescribed endurance training program was identical in the two groups and consisted of both continuous and interval training sessions 4-6x per week (Table 1), based on the polarized training approach (Muñoz et al. 2014). The endurance exercises focused on running but alternative endurance types such as cycling and cross country skiing were occasionally permitted for specific exercises in order to minimize the risk of injuries (Table 1). While two training sessions per week were supervised, the remaining endurance training sessions were performed individually. In case of sickness, subjects were required to catch up missing training sessions to standardize training volume between subjects. The training intensity was based on heart rate zones calculated from maximal heart rate determined during the incremental treadmill protocol (except for short intervals for which intensities were calculated

based on the determined best 1000 m time, Table 1). Training intensity, duration and distance were consistently controlled and recorded by heart rate monitors (RS800cx, Polar Electro Oy, Kempele, Finland), using manually pre-programmed exercise files. The endurance training intensity and volume increased throughout the two 12-week periods (Table 1).

+++Table 1 somewhere near here+++

Strength training

In the E+S group, additional strength training was performed twice a week (once a week at week 12 and 24, respectively) and was conducted always after the incremental endurance run (35-45 min by progressively increasing intensity from 65-85%, Table 1), with at least 48 h in between two combined training sessions. Subjects were instructed to rest or perform a light run (35-40 min, 60-65%, Table 1) on the day before the combined E+S session. A maximum of 10 minutes was allowed between transitions from the running session to the strength training session.

The strength training consisted of mixed maximal and explosive (~20% of strength training volume) strength training sessions and was focused on the lower limbs, while additional exercises for the upper body were included. The loads of each exercise were determined by the number of repetitions and execution velocity which was progressively increased throughout the two 12-week periods. Exercises for the lower body included bilateral leg press, bilateral and unilateral knee flexion and calf raises. During weeks 1-12 and 13-20 jumping exercises commonly used to improve explosive force production were performed (loaded and unloaded squat jumps, drop jumps, leaps, step-ups). During weeks 21-24 hurdle jumps and resisted knee lifts were also incorporated into the strength training program. Exercises for the upper body included dynamic seated vertical press, biceps curls as well as exercises commonly used to improve core stability (crunches, torso rotation, and lower back extension).

As the subjects were not accustomed to strength training, low loads (15-20x 40-50% of 1RM, 1-2min inter-set rest) were utilized during weeks 1-4. Thereafter, strength training intensity progressed to heavier loads and a lower number of sets (5-12x 60-85% of 1RM, 1-3min inter-set rest). During the second 12-week period the major strength program structure was maintained, while both training volume and loads used were increased to maximize maximal and explosive strength improvements.

Statistical analyses

Data are presented as mean±SD and shown as relative changes from the pre-loading values unless otherwise indicated. The normality of distribution was assessed using the Shapiro-Wilk test and log transformation was performed when necessary. Within and between-group differences of basal measures were assessed by a mixed ANOVA design with repeated measures. Within and between-group differences of acute loading responses were assessed by a mixed ANCOVA design, using the pre-loading values as covariates. Bivariate correlations were computed using the Pearson product-moment correlation coefficient. The statistical significance for all tests was set at 0.05. Statistical analysis was conducted using IBM SPSS 20.0 (SPSS, Inc., Chicago, IL, USA).

RESULTS

The weekly average training time was 4.9±0.2 h and 4.7±0.5 h in E and E+S, leading to a total training time of 116.5±4.5 h and 111.8±10.8 h, respectively. The weekly average running distance was 36.6±5.6 km and 33.5±7.9 km in E and E+S leading to a total mileage of 879.5±133.3 km and 804±189.3 km, respectively.

Endurance and strength performance adaptations

1000 m running time (Fig 2) significantly decreased in E at week 12 (from 3.8±0.3 min·km^{-1} to 3.5±0.3 min·km^{-1}, p=0.001) and week 24 (to 3.3±0.2 min·km^{-1}, p<0.001). Similarly, significant reductions in 1000 m time were also observed in E+S at week 12 (from 3.6±0.4 min·km^{-1} to 3.4±0.4 min·km^{-1}, p<0.001) and week 24 (to 3.3±0.2 min·km^{-1}, p=0.001) and no significant between-group differences in the changes of 1000 m time were found.

Time to exhaustion determined during the incremental treadmill test significantly increased at week 24 in E (from 24.9±3±4 min to 27.2±3.1min, 10±7%, p<0.001) and E+S (from 27.3±2.8 min to 29.1±2.5 min, 7±7%, p=0.011), while no significant between-group differences in the changes of time to exhaustion were observed.

Similarly, V_4 significantly increased in E at week 24 (from 3.7±0.5 m·s^{-1} to 3.9±0.3 m·s^{-1}, 8±9%, p=0.013) and E+S (from 3.9±0.4 m·s^{-1} to 4.1±0.4 m·s^{-1}, 6±6%, p=0.003), while no significant between-group differences in the changes of V_4 were observed.

+++Figure 2 somewhere near here+++

Dynamic leg press 1RM strength (Fig 3a) remained statistically unaltered in E at week 12 but significantly decreased at week 24 (from 148±25 kg to 141±23 kg, p=0.014). In E+S 1RM strength remained significantly unaltered at both week 12 and 24 and the between-group difference in the changes of 1RM strength was significant at week 12 (p=0.014) and 24 (p=0.011). Baseline 1RM strength performance significantly correlated with the corresponding change in 1RM strength during the first 12-week period (r=-0.622, p=0.023) in E+S but not E.

CMJ height (Fig 3b) remained statistically unaltered in both E and E+S at week 12 and 24, while the change in CMJ height at week 12 was significantly greater in E+S than E (3±8 % vs. -4±7 %, P=0.025) .

+++Figure 3a and 3b somewhere near here+++

Basal hormone concentrations

No significant changes in basal serum concentrations (Table 2) of T, GH, cortisol, SHBG and the T/C-ratio were observed in either group at week 12 or 24. The T/SHBG-ratio (Fig 4) significantly decreased in E+S at week 12 (-19±26%, p=0.006) but was no longer significantly altered at week 24. The change in T/SHBG-ratio from week 12 to 24 was significantly larger in E+S compared to E (42±47% vs. -5±33%, p=0.006).

+++Table 2 somewhere near here+++

Acute force responses

In MVC_{max} (Fig 5) significant acute decreases occurred during the endurance loading in E and E+S at week 0 (-8±8%, p=0.001 and -9±9%, p=0.005, respectively) and 24 (-5±9%, p=0.03 and -6±10%, p=0.032, respectively). No significant training-induced changes in acute force responses to endurance loading at week 24 were observed.

+++Figure 4 somewhere near here+++

Acute hormone responses

Serum T (Fig 6a) significantly increased during the endurance loading in E and E+S at week 0 (18±22%, p=0.01 and 26±27%, p=0.012, respectively) and week 24 (32±46%, p=0.006 and 27±35%, p=0.013, respectively). No significant training-induced changes in acute serum T responses at week 24 were observed.

Serum C (Fig 6b) significantly increased during the endurance loading in E and E+S at week 0 (47±40%, p<0.001 and 37±28%, p<0.001, respectively) and week 24 (42±31%, p<0.001

and 35±29%, p<0.001, respectively) but no significant training-induced changes in acute serum C responses at week 24 were observed.

+++Figure 5a and 5b somewhere near here+++

Serum GH significantly increased during the endurance loading in E and E+S at week 0 (227 fold, p<0.001 and 208 fold, p<0.001, respectively) and week 24 (341 fold, p<0.001 and 210 fold, p<0.001, respectively). No significant training-induced changes in acute serum GH responses at week 24 were observed.

Plasma volume

No between-group differences in basal plasma volume changes at week 12 and 24 were observed. Basal plasma volume shifts in the two groups ranged from -1 to +4%. Similarly, no between-group differences in acute endurance loading-induced plasma volume shifts were observed at either measurement time. Loading-induced plasma volume shifts at week 0 and 24 ranged from -6 to -7% in the two groups.

DISCUSSION

The main findings of this study were: 1) both groups improved maximal and sub-maximal endurance performance to a similar extent; 2) 1RM strength was significantly decreased in E after the training period but was maintained in E+S, leading to the between-group difference at week 12 and 24; 3) the T/SHBG-ratio significantly decreased in E+S at week 12 and the change from week 12 to 24 was significantly larger in E+S than in E; 4) the endurance loading-induced acute force and hormone responses were similar in the two groups before and after the training period and no training-induced changes in acute loading responses were observed.

Previous studies have shown that maximal and explosive strength training added to endurance training improved running economy (Millet et al. 2002; Paavolainen et al. 2003; Storen et al. 2008), velocity at the lactate threshold (Mikkola et al. 2007; Guglielmo et al. 2009), maximal running speed (Millet et al. 2002) and running time over a given distance (Paavolainen et al. 2003; Spurrs et al. 2003), while only small or no effects on maximal oxygen consumption (VO_{2max}) were reported (Paavolainen et al. 2003; Spurrs et al. 2003; Storen et al. 2008; Taipale et al. 2010). Although added strength training may further induce endurance development due to greater training volume compared to endurance training alone, improvements in running performance have also been shown with reduced endurance training volume compensated by added strength training (Paavolainen et al. 2003). In general, however, beneficial adaptations in cardiorespiratory function and endurance performance following concurrent training have typically occurred with concomitant increases in maximal and/or explosive strength development, indicating that strength training-induced endurance development may occur as a result of enhanced neuromuscular performance (Paavolainen et al. 2003; Mikkola et al. 2007; Taipale et al. 2010).

In contrast to these studies, the present investigation found similar improvements in maximal and sub-maximal endurance performance for both training groups with no additional effects of the supplemented strength training in the E+S group. However, while the present E group significantly decreased 1RM strength after 24 weeks, maximal leg strength was maintained but not increased in the E+S group and a similar tendency was observed for CMJ height.

The present research design purposefully differed from previous studies since the strength loading was always performed immediately after an exhausting endurance running session, and therefore, every strength training session may have been affected by residual fatigue. While several cross-sectional studies have shown acute detrimental effects on strength performance (Leveritt and Abernethy 1999; de Souza et al. 2007) and anabolic hormone

responses (Goto et al. 2005) when strength loading was immediately preceded by endurance cycling or running, this has, to the best of our knowledge, only been supported by few longitudinal training studies. Craig et al. (1991) failed to observe lower body strength gains when strength training was repeatedly preceded by endurance running and, in line with our results, endurance performance increased to a similar magnitude in their combined and E training group. However, compared to the study of Craig et al. (1991), our E group significantly reduced maximal strength performance after 24 weeks of training, while our E+S group was able to maintain their basal strength performance which may in turn provide benefits over long-term.

Strength training-induced increases in maximal endurance running performance may in part be attributed to increased fatigue resistance allowing sustaining repeated cycles of stretch-shortening contractions over a prolonged period of time (Paavolainen et al. 1999). This greater level of resistance against neuromuscular fatigue in response to endurance loading would be expected after a prolonged period of combined endurance and strength training compared to endurance training only. However, the endurance loading-induced acute reductions in MVC_{max} were similar between E and E+S groups in the present study when compared before and after the 24-week training period, supporting the finding that both groups experienced similar improvements in endurance adaptations. However, caution should be taken since the endurance loading was performed with relative maximal loads (i.e. time to exhaustion based on current training status at weeks 0 and 24) and both training groups significantly increased time to exhaustion after 24 weeks of training. In light of the resulting increases in loading volume, the observed acute reductions in maximal force at week 24 may actually indicate a training adaptation. However, as the magnitude of reductions was similar in E and E+S, our findings indicate these positive adaptations to be induced by the prolonged endurance training rather than the added strength training.

Interestingly, in the E+S group a significant correlation was observed between the basal levels of 1RM strength and the corresponding changes in maximal strength performance during the first 12 but not 24 weeks. Although not statistically significant, after 12 weeks of training the E+S group had improved strength performance by 5±7% which was diminished at week 24, despite a progressive increase in training load. While the observed correlation indicates the importance of strength training especially for the weaker endurance runners, our results suggest a biphasic response to the performed strength training which may indicate the strength loading stimulus to be less effective during the second 12 weeks of training. As both the strength and endurance training volume and intensity progressively increased during the latter half of the training, it may be possible that the unfavorable effects of preceding endurance loading on the quality of the subsequent strength training session were intensified during the second 12-week period and, thus, resulting in further impairment of strength development.

However, this finding was not accompanied by significant changes in basal hormone concentrations. Typically, the training induced endocrine adaptations differ between the types of training performed (Kraemer et al. 1995). Prolonged strength training may lead to increases in basal levels of anabolic hormone concentrations at least in previously untrained subjects (Häkkinen et al. 2000; Ahtiainen et al. 2003), while the basal concentrations of these hormones may actually be decreased following endurance training only (Hackney et al. 2003). Combined endurance and strength training studies in which endurance and strength were performed on separate days, on the other hand, have shown small increases in basal serum testosterone concentrations in untrained (Kraemer et al. 1995) and significant increases in endurance trained subjects (Taipale et al. 2010). These previous findings indicate that the strength training-induced changes in basal hormone concentrations may counteract an endurance training-induced catabolic state, possibly contributing to the beneficial effects of

409 strength training for endurance athletes. In the present study, however, only small fluctuations
410 in basal hormone concentrations were observed, supporting the lack of effects of strength
411 training on endurance performance, when performed immediately after endurance running
412 sessions.

413 Interestingly, in our E+S group a significant reduction in the T/SHBG-ratio was observed
414 after 12 weeks of training and this initial decrease was followed by a large increase thereafter,
415 leading to a significant between-group difference in the magnitude of changes in the
416 T/SHBG-ratio from week 12 to 24. As the T/SHBG-ratio correlates with free available
417 testosterone concentrations (Selby 1990) and may therefore reflect an anabolic state, the
418 observed reductions in the E+S group during the first 12 weeks may indicate increased uptake
419 of testosterone by the target cells (Vingren et al. 2010). Although receptor content was not
420 assessed in this study, the observed reduction in the T/SHBG-ratio during the first 12 weeks
421 of training may reflect a positive response to a new training stimulus since the subjects were
422 recreationally endurance trained but not accustomed to strength training. Similar
423 improvements in endurance performance between the E+S and E groups in conjunction with
424 small increases in maximal strength for the E+S group at the 12-week time point suggests that
425 the present strength training method may be effective in inducing strength development for at
426 least 12 weeks. In a previous study (Taipale et al. 2010) the beneficial effects of strength
427 training on running economy were apparent not during the actual combined endurance and
428 strength training intervention but after a reduction in strength training volume. It may also be
429 possible that a reduction in strength training volume after the present 12 weeks of training
430 would have been necessary in order to induce improvements in maximal and sub-maximal
431 endurance performance.

432 Similar to the maintained serum hormone concentrations during the 24 weeks of training, no
433 training-induced changes in endurance loading responses of testosterone, growth hormone

and cortisol were observed. Both groups significantly increased endurance loading-induced acute anabolic and catabolic hormone concentrations before and after the training to a similar extent. While these findings are in contrast to a study by Kraemer et al. (1995) who found significantly larger testosterone responses to endurance loading after combined training in physically active subjects, our findings are in line with results of Craig et al. (1991). In their study, endurance running-induced growth hormone responses were examined between an endurance training only group and a combined strength and endurance training group, where endurance training always preceded strength training in the same session. In line with our results, they found no differences in growth hormone responses. However, similar to our observed acute force responses, the improvements in treadmill running time to exhaustion found in the present study may have blunted any potential changes in acute endurance loading-induced serum hormone concentrations. Furthermore, due to the length of the study where pre-training measurements were conducted in the Fall and post-training measurements carried out in the Spring, possible seasonal variations in serum hormone concentrations should be considered when interpreting the present findings (Svartberg et al. 2003).

Although training- or loading-induced alterations in serum hormone concentrations may be associated with chronic increases in strength performance, the possible role of neural interference in respect to the present findings should not be neglected. While a thorough investigation of neuromuscular mechanisms was beyond the scope of this study, it is possible that residual fatigue from the preceding endurance session affected neural activation of the exercised muscles during the subsequent strength training sessions, as shown in our previous study in untrained men (Eklund et al. 2014). Due to the high volume of endurance training performed in the present study, it is possible that such a neural inhibition may have contributed to the lack of expected increases in strength performance in the present E+S group.

In conclusion, the present study showed that same-session combined training where strength training was repeatedly preceded by endurance loading did not lead to superior endurance performance benefits in recreational endurance runners, when compared to endurance running only. It is likely that this was attributed to the impaired strength development, despite consistent progressive strength training in the E+S group. In support of this assumption, no between-group differences in training-induced changes in acute force and hormone responses to endurance loading were observed and basal hormone concentrations were maintained in the two groups. Although in the present design no group utilizing other combined endurance and strength training modes were included, these results suggest that endurance athletes should separate their endurance and strength training sessions in order to maximize benefits of the added strength training.

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573 TABLES

574 **Table 1.** Prescribed endurance training program for the two groups.

	Weeks 1-12	Weeks 13-24
Incremental run	2x/week, running only 35-45min/65-85%	2x/week, running only 40-45min/65-85%
Long run	1x/week, running, cycling or skiing 70-120min/60-65%	1x/week, running, cycling or skiing 85-125min/60-65%
Long intervals	1x/week, running only 4-5x5min/80-85%, rest 3min <65%	1x/week, running only 4-6x5min/85%, rest 3min <65%
Short intervals		1x/week, running only (on the track) 3-6x400m + 3-6x 800m/85%, rest 2min <65%
Light run	1x/week, running only 35-40min/60-65%	1x/week, running, cycling or skiing 40min/60-65%
Optional run	Optional 1x/week, running, cycling or skiing 35-40min/ 70-75%	

575 Intensity zones are % of HRmax except for short intervals (% of 1000m time).

576 In the E+S group, strength training was performed twice a week after the incremental

577 endurance run.

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583 **Table 2.** Basal concentrations of serum hormones throughout the 24 weeks of training.

	E			E+S		
	Week 0	**Week 12**	**Week 24**	**Week 0**	**Week 12**	**Week 24**
Testosterone ($nmol \cdot l^{-1}$)	16.9±6.4	15.3±4.1	15.6±5.2	19.7±7.9	15.9±5.3	19.1±5.9
GH (22-kDa) ($mlU \cdot l^{-1}$)	0.5±0.6	0.6±1.3	0.6±0.7	1.0±1.7	2.6±7.5	1.2±2.1
SHBG ($nmol \cdot l^{-1}$)	29.6±11.3	29.1±10.5	32.2±11.8	33.6±9.8	36.4±13.0	33.0±9.9
Cortisol ($nmol \cdot l^{-1}$)	499.9±85.2	469.2±92.8	514.0±44.2	504.4±130.9	466.4±104.7	498.2±93.2
T/SHBG-ratio ($nmol \cdot l^{-1}$)	0.6±0.2	0.6±0.2	0.5±0.1	0.6±0.3	0.5±0.2**	0.6±0.2
T/C-ratio ($nmol \cdot l^{-1}$)	0.034±0.012	0.033±0.010	0.030±0.010	0.040±0.013	0.034±0.008	0.040±0.016

584 GH=growth hormone; SHBG=sex hormone binding globulin; T/SHBG-ratio=testosterone/SHBG-

585 ratio; T/C-ratio=testosterone/cortisol-ratio; **p<0.01 compared to week 0.

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596 FIGURE CAPTIONS

597 **Figure 1.** Study design. Baseline tests included the determination of endurance (incremental
598 field test) and strength performance (1RM during dynamic leg press and CMJ height) as well
599 as the determination of basal hormone concentrations.

600 **Figure 2.** Changes in maximal 1000 m running time determined during an incremental field
601 test of 6x1000m. ***p<0.001 compared to values obtained at week 0.

602 **Figure 3.** Changes in 1RM strength (A) and CMJ height (B). *p<0.05, within bar compared
603 to values obtained at week 0; outside the bar as indicated.

604 **Figure 4.** Changes in T/SHBG ratio. **<0.01, inside the bar compared to values obtained at
605 week 0; outside the bar as indicated.

606 **Figure 5.** Endurance loading-induced acute reductions in isometric maximal force (MVC_{max})
607 at week 0 and week 24. *p<0.05, **p<0.01 compared to obtained pre-loading values at week
608 0 and 24, respectively.

609 **Figure 6.** Endurance loading-induced acute changes in serum testosterone (A) and cortisol
610 (B) concentrations. *p<0.05, **p<0.01, ***p<0.001 compared to obtained pre-loading values
611 at week 0 and 24, respectively.

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Fig. 1

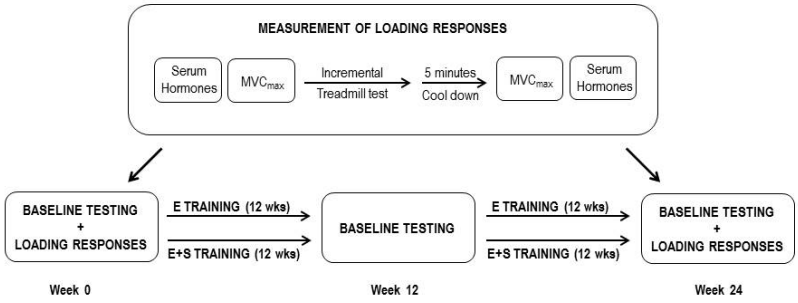

Fig. 2

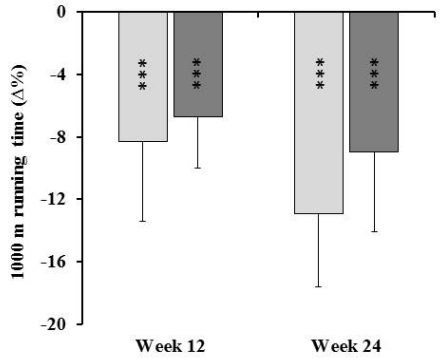

Fig. 3

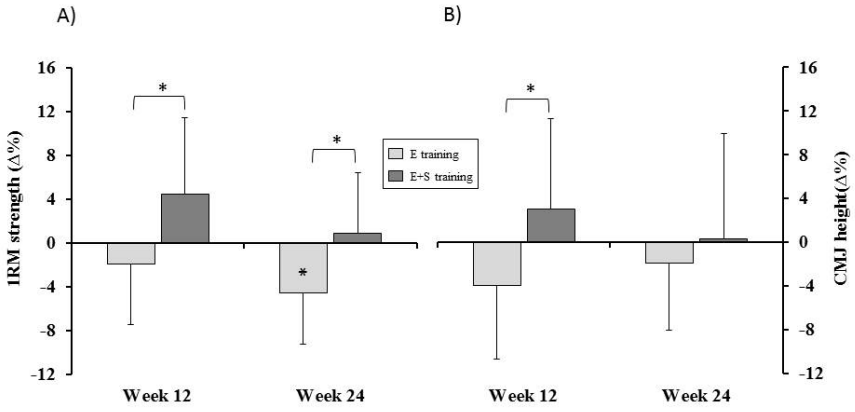

Fig. 4

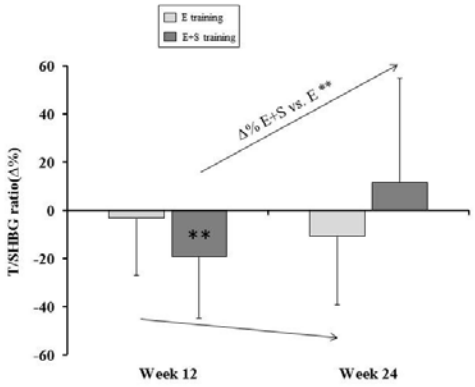

Figure 5

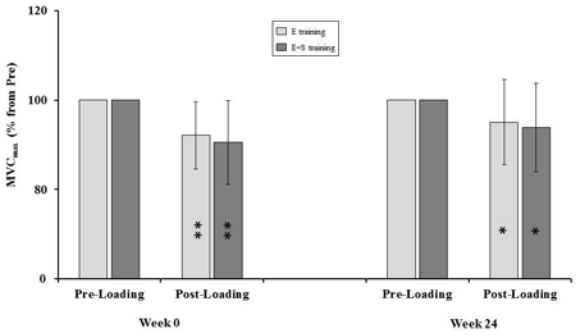

Figure 6